Cohen's Pathways *of the* PULP

TENTH EDITION



Kenneth M. Hargreaves ■ Stephen Cohen

Web Editor: Louis H. Berman

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Editors

Kenneth M. Hargreaves, DDS, PhD, FICD, FACD

Professor and Chair
Department of Endodontics;
Professor
Departments of Pharmacology, Physiology (Graduate School), and Surgery (Medical School);
President's Council Endowed Chair for Research
University of Texas Health Science Center at San Antonio
San Antonio, Texas;
Diplomate, American Board of Endodontics

Stephen Cohen, MA, DDS, FICD, FACD

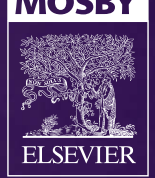
Clinical Professor (Adjunct)
Department of Endodontics
The Arthur A. Dugoni School of Dentistry
University of the Pacific;
Clinical Professor of Endodontics
Department of Preventive and Restorative Dental Sciences
University of California
School of Dentistry
San Francisco, California;
Consultant
Endodontic Postgraduate Program
Riyadh Colleges of Dentistry and Pharmacology
Riyadh, Saudi Arabia;
Diplomate, American Board of Endodontics

Web Editor

Louis H. Berman, DDS, FACD

Private Practice of Endodontics
Annapolis, Maryland;
Diplomate, American Board of Endodontics

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3251 Riverport Lane
St. Louis, Missouri 63043

COHEN'S PATHWAYS OF THE PULP, Tenth Edition ISBN 978-0-323-06489-7

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Library of Congress Control Number: 2010922576

Vice President and Publishing Director: Linda Duncan
Executive Editor: John Dolan
Senior Developmental Editor: Courtney Sprehe
Publishing Services Manager: Catherine Jackson
Senior Project Manager: Karen M. Rehwinkel
Design Direction: Jessica Williams

Printed in China

Last digit is the print number: 9 8 7 6 5 4 3 2 1

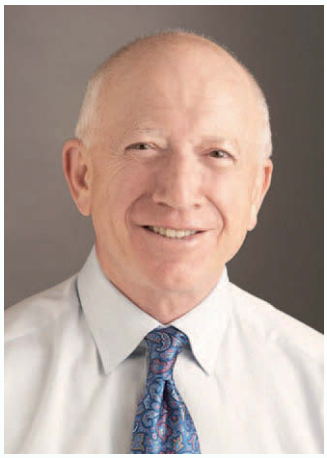
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About the Editors



KENNETH M. HARGREAVES

Dr. Kenneth M. Hargreaves is professor and Chair of the Department of Endodontics at the University of Texas Health Science Center at San Antonio. He completed his endodontic residency at the University of Minnesota in 1993. Dr. Hargreaves is a Diplomate of the American Board of Endodontics and maintains a private practice limited to endodontics. He is an active researcher, lecturer, and teacher and serves as the Editor of the *Journal of Endodontics*. He is principal investigator on several nationally funded grants that combine his interests in pain, pharmacology, and regenerative endodontics. He has received several awards, including a National Institutes of Health MERIT Award for pain research, the AAE Louis I. Grossman Award for cumulative publication of research studies, and two IADR Distinguished Scientist Awards.



STEPHEN COHEN

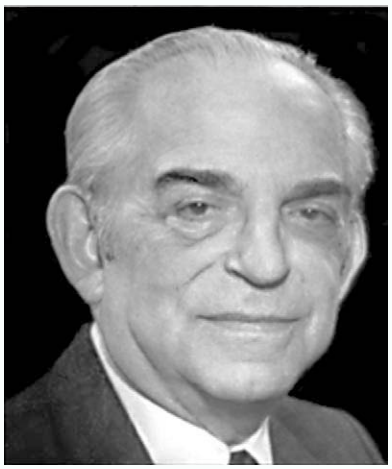
Dr. Stephen Cohen is one of the foremost endodontic clinicians in the country and a worldwide lecturer on endodontics. Dr. Cohen completed his studies in the Endodontic Postgraduate Program at the University of Pennsylvania in 1969 and began his private practice. From 1970 until 1988, Dr. Cohen served as Chairman of the Department of Endodontics at The Arthur A. Dugoni School of Dentistry of the University of the Pacific, and he has continued his involvement with the university as an Adjunct Clinical Professor of Endodontics. He is also a Clinical Professor of Endodontics in the Department of Preventive and Restorative Dental Sciences at the University of California School of Dentistry, San Francisco. Dr. Cohen was senior editor of the first nine editions of *Pathways of the Pulp* and continues to serve as editor of the tenth edition. He is a Diplomate of the American Board of Endodontics, and has held leadership positions in many of the major professional and academic organizations in endodontics. He maintains a full-time endodontic practice in San Francisco, where he has practiced since 1969.



LOUIS H. BERMAN

Dr. Louis H. Berman has been in full-time private practice limited to endodontics in Annapolis, Maryland since 1983. Dr. Berman completed his endodontic residency at the Albert Einstein Medical Center in Philadelphia. He was formerly a Clinical Instructor and Assistant Professor of Endodontics at the University of Maryland School of Dentistry. An active lecturer and author, he is a past president of the Maryland State Association of Endodontics, and is a member of the *Journal of Endodontics* Scientific Advisory Panel. Dr. Berman is the senior editor of the textbook *Dental Traumatology* and is a Diplomate of the American Board of Endodontics and a Fellow of the American College of Dentistry.

Dr. I.B. Bender



For a man of short stature, I.B. Bender was a giant in shaping the field of endodontics. Dr. Bender started his remarkable career as an educator in 1942 at the Albert Einstein Medical Center in Philadelphia, and continued with his passionate pursuit of endodontic teaching for the next 61 years. He authored more than 130 articles in medicine and dentistry and was co-author of the classic textbook, *The Dental Pulp*. Recipient of the most prestigious awards given to endodontists, he also served as President of the American Association of Endodontists and as a Director of the American Board of Endodontics. In 1989, the endodontic residency program at the Albert Einstein Medical Center honored him by dedicating their program as the I.B. Bender Division of Endodontics and established the I.B. Bender Research Endowment Fund.

A true Renaissance man, I.B. was internationally known not just for his boundless knowledge of endodontic literature and extraordinary insight into the biological aspects of our specialty, but also for his quick wit, enjoyable sense of humor, and persistent quest to challenge "conventional" beliefs and let science lead us into the future. I.B. was a wise man who once stated, "The clinical practice of yesterday's endodontics becomes the heresy of today, and today's endodontic practice becomes the heresy of tomorrow. So don't be so rigid in your techniques or beliefs."

I.B. took much pride in inspiring and imbuing his students with a passion for endodontic excellence. During lectures he was well known to compliment their correct answers with his wide smile, a tilt of his head while pointing his finger, and a statement that they had been "Bender-ized." He always welcomed a challenge to the intellectual status quo, and took great pleasure in a duel of intellect. I.B. referred to himself as "the oldest living functional endodontist … who has helped to move the status of endodontics from the 'outhouse' to the 'penthouse'." He valued his close friendships and always finished his personal notes, in his uniquely recognizable penmanship, with "Azever, I.B."

At a 1995 lecture at the I.B. Bender Conference, "The Biological Basis and Clinical Practice of Endodontics," I.B. closed by stating: "The implementation of extended research and teaching is a means by which endodontics will be able to meet the future demands for its service. Service itself in a specialty cannot make progress. Other developments in our discipline will come from people who can touch tomorrow." And I.B. touched tomorrow by touching the lives of all of us.

I.B. was instrumental in shaping our past, directing our present, and providing the foundation and vision for our ever-changing future. His inspiring passion for knowledge and excellence set the standards for the legions of students who will always remain indebted to him for role-modeling what a healer and teacher does for others. To commemorate this iconic role model of our specialty, we are pleased to dedicate this tenth edition of *Pathways of the Pulp* to Dr. I.B. Bender.

Kenneth M. Hargreaves
Stephen Cohen
Louis H. Berman

Contributors

Bettina Basrani, DDS, PhD
Specialist in Endodontics
Assistant Professor
Program Co-Director MSC in Endodontics
Faculty of Dentistry
University of Toronto
Toronto, Ontario, Canada

Ellen Berggreen, DDS, PhD
Associate Professor
Department of Biomedicine
Faculty of Medicine and Dentistry
University of Bergen
Bergen, Norway

Louis H. Berman, DDS, FACD
Private Practice of Endodontics
Annapolis, Maryland;
Diplomate, American Board of Endodontics

Serge Bouillaguet, DMD, PhD
Head of Endodontic Unit
Department of Cariology and Endodontology
School of Dental Medicine
University of Geneva
Geneva, Switzerland

B. Ellen Byrne, DDS, PhD
Senior Associate Dean
Professor of Endodontics
Virginia Commonwealth University
Richmond, Virginia

Joe H. Camp, DDS, MSD
Adjunct Professor
Department of Endodontics
School of Dentistry
University of North Carolina
Chapel Hill, North Carolina;
Private Practice of Endodontics
Charlotte, North Carolina

Noah Chivian, DDS, FACD, FICD
Clinical Professor
Department of Endodontics
University of Medicine and Dentistry
New Jersey
Newark, New Jersey;
Adjunct Professor
Department of Endodontics
University of Pennsylvania
School of Dental Medicine
Philadelphia, Pennsylvania;
Attending in Endodontics
Newark Beth Israel Medical Center
New York, New York;
Private Practice
Limited to Endodontics
West Orange, New Jersey;
Diplomate, American Board of Endodontics

Stephen Cohen, MA, DDS, FICD, FACD
Clinical Professor (Adjunct)
Department of Endodontics
The Arthur A. Dugoni School of Dentistry
University of the Pacific;
Clinical Professor of Endodontics
Department of Preventive and Restorative Dental Sciences
University of California
School of Dentistry
San Francisco, California;
Consultant
Endodontic Postgraduate Program
Riyadh Colleges of Dentistry and Pharmacology
Riyadh, Saudi Arabia;
Diplomate, American Board of Endodontics

Jeffrey M. Coil, DMD, MSD, PhD, FRCD(C), FADI
Director, Graduate Endodontics Program
Department of Oral Biological and Medical Sciences
Faculty of Dentistry
University of British Columbia
Vancouver, British Columbia, Canada;
Diplomate, American Board of Endodontics

Arthur W. Curley, BS, JD
Assistant Professor
The Arthur A. Dugoni School of Dentistry
University of the Pacific;
Adjunct Faculty
University of California
San Francisco, California;
Associate, American Board of Trial Advocates
Associate California Association of Oral and Maxillofacial Surgeons

Didier Dietschi, DMD, PhD, Privat-Docent
Senior Lecturer
School of Dental Medicine
Department of Cariology and Endodontics
University of Geneva
Geneva, Switzerland;
Adjunct Professor
School of Dentistry
Department of Comprehensive Care
Case Western Reserve University
Cleveland, Ohio

Paul D. Eleazer, DDS, MS
Professor and Chair
Department of Endodontics and Pulp Biology
School of Dentistry
University of Alabama
Birmingham, Alabama;
Diplomate, American Board of Endodontics

Mohamed I. Fayad, DDS, MS, PhD
Assistant Clinical Professor
Department of Endodontics
College of Dentistry
University of Illinois at Chicago
Chicago, Illinois;
Diplomate, American Board of Endodontics

Ashraf F. Fouad, BDS, DDS, MS
Professor and Chair
Department of Endodontics, Prosthodontics, and Operative Dentistry
Director, Postgraduate Program in Endodontics
University of Maryland
Baltimore, Maryland;
Diplomate, American Board of Endodontics

Jared C. Frisbie, DDS
Private Practice Limited to Endodontics
Portland, Oregon

Inge Fristad, DDS, PhD
Professor
Head and Director
Department of Clinical Dentistry
Faculty of Medicine and Dentistry
University of Bergen
Bergen, Norway

Anna B. Fuks, CD
Professor Emeritus
Department of Pediatric Dentistry
Hadassah Faculty of Dental Medicine
The Hebrew University
Jerusalem, Israel

Bradley H. Gettleman, DDS, MS
Private Practice of Endodontics
Glendale, Arizona;
Diplomate, American Board of Endodontics

Gerald N. Glickman, DDS, MS, MBA, JD
Professor and Chairman
Director, Graduate Program in Endodontics
Department of Endodontics
Baylor College of Dentistry
Dallas, Texas;
Diplomate, American Board of Endodontics

Harold E. Goodis, DDS
Professor Emeritus
Division of Endodontics
Department of Preventive and Restorative Dental Sciences
School of Dentistry
University of California
San Francisco, California;
Professor of Endodontics
Senior Research Mentor
Boston University Institute for Dental Research and Education/Dubai
United Arab Emirates;
Diplomate, American Board of Endodontics

James E. Haddix, DMD
Associate Professor and Director of Predoctoral Endodontics
College of Dentistry
Department of Endodontics
University of Florida
Gainesville, Florida

Kenneth M. Hargreaves, DDS, PhD, FICD, FACD
Professor and Chair
Department of Endodontics;
Professor
Departments of Pharmacology, Physiology (Graduate School) and Surgery (Medical School);
President's Council Endowed Chair for Research
University of Texas Health Science Center at San Antonio
San Antonio, Texas;
Diplomate, American Board of Endodontics

Gary R. Hartwell, DDS, MS
Professor and Chair
Department of Endodontics
New Jersey Dental School
Newark, New Jersey;
Diplomate, American Board of Endodontics

Gunnar Hasselgren, DDS, PhD
Professor and Director
Postdoctoral Endodontics
College of Dental Medicine
Columbia University
New York, New York

George T-J Huang, DDS, MSD, DSc
Chair and The Herbert Schilder Professor in Endodontics
Director, Advanced Specialty Education Program in Endodontics
Henry M. Goldman School of Dental Medicine
Boston University
Boston, Massachusetts;
Diplomate, American Board of Endodontics

Bradford R. Johnson, DDS, MHPE
Director of Postdoctoral Endodontics
Associate Professor
Department of Endodontics
College of Dentistry
University of Illinois at Chicago
Chicago, Illinois;
Diplomate, American Board of Endodontics

William T. Johnson, DDS, MS
Richard E. Walton Professor and Chair
Department of Endodontics
College of Dentistry
University of Iowa
Iowa City, Iowa;
Diplomate, American Board of Endodontics

Karl Keiser, DDS, MS, FICD, FACD
Clinical Associate Professor
Department of Endodontics
University of Texas Health Science Center
San Antonio, Texas;
Diplomate, American Board of Endodontics

David G. Kerns, DMD, MS
Professor and Director of Graduate Periodontics
Department of Periodontics
Baylor College of Dentistry
Texas A & M Health Science Center
Dallas, Texas

Päivi Kettunen, DDS, PhD
Professor
Group Leader, Craniofacial Developmental Biology Group
Department of Biomedicine
Faculty of Medicine and Dentistry
University of Bergen
Bergen, Norway

James C. Kulild, DDS, MS
Professor and Program Director
Advanced Specialty Education Program in Endodontics
School of Dentistry
University of Missouri–Kansas City
Kansas City, Missouri;
Diplomate, American Board of Endodontics

Alan S. Law, DDS, PhD
Private Practice
Lake Elmo, Minnesota;
Diplomate, American Board of Endodontics

Linda Levin, DDS, MS, PhD
Adjunct Associate Professor
Department of Endodontics
University of North Carolina School of Dentistry
Chapel Hill, North Carolina;
Diplomate, American Board of Endodontics

Martin D. Levin, DMD
Adjunct Associate Professor
Department of Endodontics
University of Pennsylvania
School of Dental Medicine;
Adjunct Associate Professor
Department of Postgraduate Endodontics
Nova Southeastern University
College of Dental Medicine
Ft. Lauderdale, Florida;
Private Practice
Chevy Chase, Maryland;
Diplomate, American Board of Endodontics

Roger P. Levin, DDS
Chairman and Chief Executive Officer
Levin Group, Inc.
Owing Mills, Maryland

Louis M. Lin, BDS, DMD, PhD
Professor and Director of Advanced Education in Endodontics
New York University
College of Dentistry
New York, New York;
Former Chairman of Division of Endodontics
University of California at Los Angeles
School of Dentistry
Los Angeles, California;
Diplomate, American Board of Endodontics

Henrietta L. Logan, MA, PhD
Professor
Department of Community Dentistry and Behavioral Science
University of Florida
Gainesville, Florida

Keijo Luukko, PhD, DDS
Professor
Department of Biomedicine
Faculty of Medicine and Dentistry
University of Bergen
Bergen, Norway

Donna Mattscheck, DMD
Private Practice of Endodontics
Billings, Montana;
Diplomate, American Board of Endodontics

Thomas V. McClammy, DMD, MS, PC
Private Practice of Endodontics
Scottsdale, Arizona

Zvi Metzger, DMD
Associate Professor, Chair
Department of Endodontology
The Goldschleger School of Dental Medicine
Tel Aviv University
Tel Aviv, Israel

Dale A. Miles, BA, DDS, MS, FRCD(C)
Adjunct Professor
University of Texas Health Science Center at San Antonio
San Antonio, Texas;
Adjunct Professor
Arizona School of Dentistry and Oral Health
Mesa, Arizona;
CEO
Digital Radiographic Solutions
Fountain Hills, Arizona

Carl W. Newton, DDS, MSD, FACD, FICD
Professor of Endodontics
Department of Endodontics
Indiana University
School of Dentistry
Indianapolis, Indiana;
Diplomate, American Board of Endodontics

Donald R. Nixdorf, DDS, MS
Assistant Professor
Division of TMD and Orofacial Pain
School of Dentistry;
Adjunct Assistant Professor
Department of Neurology
University of Minnesota
Medical School
Minneapolis, Minnesota

John M. Nusstein, DDS, MS
Assistant Professor and Chair
Division of Endodontics
The Ohio State University
College of Dentistry
Columbus, Ohio;
Diplomate, American Board of Endodontics

Christine I. Peters, DMD
Associate Professor
Department of Endodontics
School of Dentistry
University of the Pacific
San Francisco, California

Ove A. Peters, DMD, MS, PhD, FICD
Professor
Department of Endodontics
Arthur A. Dugoni School of Dentistry
University of the Pacific
San Francisco, California

Al W. Reader, DDS, MS
Professor and Program Director,
Advanced Endodontics
Department of Endodontics
College of Dentistry
The Ohio State University
Columbus, Ohio;
Diplomate, American Board of Endodontics

Isabela N. Rôças, DDS, MSc, PhD
Adjunct Professor
Department of Endodontics and Laboratory of Molecular Microbiology
Faculty of Dentistry
Estácio de Sá University
Rio de Janeiro, Brazil

Robert S. Roda, DDS, MS
Adjunct Assistant Professor
Department of Endodontics
Baylor College of Dentistry
Dallas, Texas;
Private Practice Limited to Endodontics
Scottsdale, Arizona;
Diplomate, American Board of Endodontics

Paul A. Rosenberg, DDS
Professor and Chair
Ignatius N. and Sally Quartararo
Department of Endodontics
New York University
College of Dentistry
New York, New York;
Private Practice Limited to Endodontics
New York, New York;
Diplomate and Former Director, American Board of Endodontics

Louis E. Rossman, DMD
Clinical Professor
Department of Endodontics
University of Pennsylvania
School of Dental Medicine;
Private Practice
Philadelphia, Pennsylvania;
Diplomate, American Board of Endodontics

Avishai Sadan, DMD
Dean, G. Donald and Marian James
Montgomery Professor of Dentistry
Herman Ostrow School of Dentistry
University of Southern California
Los Angeles, California

Asgeir Sigurdsson, Cand Odont, MS
Adjunct Associate Professor
Department of Endodontics
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina;
Honorary Clinical Teacher in Endodontology
UCL Eastman Dental Institute
London, United Kingdom;
Private Practice
Reykjavik, Iceland;
Diplomate, American Board of Endodontics

José F. Siqueira, Jr., DDS, MSc, PhD
Professor and Chairman
Department of Endodontics and Laboratory of Molecular Microbiology
Faculty of Dentistry
Estácio de Sá University
Rio de Janeiro, Brazil

Martin Trope, DMD, FICD, FACD
Private Practice
Philadelphia, Pennsylvania;
Diplomate, American Board of Endodontics

Frank J. Vertucci, DMD
Professor and Chairman
Department of Endodontics
College of Dentistry
University of Florida
Gainesville, Florida;
Diplomate, American Board of Endodontics

Merlyn W. Vogt, DDS
Assistant Professor
Department of Endodontics
College of Dentistry
University of Nebraska Medical Center
Lincoln, Nebraska

Richard E. Walton, DMD, MS
Professor
Department of Endodontics
University of Iowa
College of Dentistry
Iowa City, Iowa;
Diplomate, American Board of Endodontics

Paula J. Waterhouse, BDS (Hons), PhD, FDSRCS (Ed), FDS (Paed) RCS, FHEA
Clinical Senior Lecturer in Child Dental Health
School of Dental Sciences
Newcastle University;
Honorary Consultant in Pediatric Dentistry
Newcastle Dental Hospital
Newcastle upon Tyne, Tyne and Wear
United Kingdom

John M. Whitworth, PhD, BChD, FDS RCSEd, FDS RCS (Rest Dent)
Newcastle University;
Honorary Consultant in Restorative Dentistry
Newcastle Dental Hospital
Newcastle upon Tyne, Tyne and Wear
United Kingdom

Anne E. Williamson, DDS, MS
Director, Advanced Education in Endodontics;
Associate Professor
Department of Endodontics
University of Iowa
College of Dentistry
Iowa City, Iowa;
Diplomate, American Board of Endodontics

David E. Witherspoon, BDSc, MS
Private Practice
Dallas, Texas;
Diplomate, American Board of Endodontics

James Wolcott, DDS
Clinical Assistant Professor
University of Colorado
School of Dentistry;
Private Practice
Colorado Springs, Colorado;
Diplomate, American Board of Endodontics

Edwin J. Zinman, DDS, JD
Private Practice of Law;
Editorial Board
Journal of American Academy of Periodontology;
Former Lecturer, Department of Stomatology
University of California–San Francisco
School of Dentistry
San Francisco, California

Preface

The co-editors and publisher are pleased to present the tenth edition of a textbook that has been continuously evolving since 1976. We are also proud to announce the renaming of this text to ***Cohen's Pathways of the Pulp*** to reflect the leadership, passion, and expertise of Dr. Stephen Cohen, who has guided the production of all 10 editions. This current edition has been completely updated and expanded to provide the latest clinically relevant, evidence-based knowledge, while also introducing exciting new chapters on subjects such as Regenerative Endodontics, Cone Beam Technologies, and Endodontic Practice Management. The companion Expert Consult version of the book has also been greatly enhanced with supplemental content available only online. With its exceptional capacity to provide continuous updates, the Expert Consult site is reflective of the dynamic nature of our specialty, empowering our readers with not only the most current clinical information available, but also with an invaluable feature that permits the downloading of any figure in the book as a PowerPoint or jpeg image for the reader's personal study or lectures. Moreover, the online Challenge section provides self-assessment of knowledge with a unique collection of hyperlinks that connects all of the multiple choice questions to relevant sections of the text. As editors, we feel privileged and honored to offer this thoroughly updated and expanded version of the first interactive textbook in dentistry that enables the reader to continuously stay on the "cutting edge" of endodontics.

We welcome your feedback at any time.

Kenneth M. Hargreaves
Stephen Cohen
Louis H. Berman

Acknowledgments

Working with such a talented, patient, and highly professional team at Elsevier has been an enriching experience. In the future, when we look back to the time that was devoted to developing this tenth edition, we'll always remember fondly the special people with whom we've forged a bond through hard work and how they helped us at every level. Some of the many people that we'd like to express our gratitude to include:

- *John Dolan, Executive Editor*
- *Courtney Sprehe, Senior Developmental Editor*
- *Karen Rehwinkel, Senior Project Manager*
- *Jill Norath, Multimedia Producer*
- *Jessica Williams, Designer*
- *Jaime Pendill, Freelance Editor*

And to the many people who helped those named above we extend a heartfelt "Thank You"!

About the Tenth Edition

With the publication of this milestone tenth edition, the staff at Elsevier would like to thank Dr. Stephen Cohen for his many years of dedication to *Pathways of the Pulp*. In recognition of Dr. Cohen's leadership in making this a world-renowned text and for his many contributions to the field of endodontics, the new edition has been renamed *Cohen's Pathways of the Pulp*.

Pathways of the Pulp has a longstanding reputation of providing high-level, comprehensive content that conveys the most current endodontic information, materials, and techniques to an international audience. The tenth edition builds on this solid reputation with a book that delivers more than ever! Renowned contributors from across the country and around the world offer expert perspectives and expand on observations made by previous contributors. Full-color photographs, illustrations, and a wide range of radiographs clearly demonstrate core concepts and reinforce the essential principles and techniques unique to the specialty of endodontics.

CHAPTERS NEW TO THIS EDITION

Chapter 14: Pathobiology of the Periapex by Louis M. Lin and George T-J Huang. This chapter looks at topics such as apical periodontitis, infection, asymptomatic apical periodontitis (apical granuloma), the relationship between apical periodontitis and systemic diseases, and wound healing of apical periodontitis.

Chapter 16: Regenerative Endodontics by Kenneth M. Hargreaves and Alan S. Law. This chapter describes the biological basis for regenerating the pulp-dentin complex of teeth with necrotic pulps and reviews the clinical procedures reported to date on this newly emerging procedure.

Chapter 22: Restoration of Endodontically Treated Teeth by Didier Dietschi and Serge Bouillaguet. Topics covered in this chapter include a description of the unique physical characteristics of endodontically treated teeth, restorative materials, pretreatment evaluation and treatment strategies, and a variety of clinical procedures, including direct composite restorations, veneers, selection of posts and core materials, and much more.

Chapter 29: Cone Beam Imaging for Endodontics (online) by Dale A. Miles and Thomas V. McClammy. With the recent advent of cone beam computerized tomography's use in the dental office, this chapter elaborates on the enhanced radiographic capabilities now available for endodontic diagnosis and treatment planning.

Chapter 30: Key Principles of Endodontic Practice Management (online) by Roger P. Levin. Providing insight on marketing, staff development, patient management, and referral relationships, this chapter describes endodontic management systems and the goals necessary for the development of a successful endodontic practice.

ORGANIZATION

As in the previous edition, the tenth edition is divided into three parts, Part I: The Core Science of Endodontics, Part II: The Advanced Science of Endodontics, and Part III: Related Clinical Topics.

Part I begins with chapters that focus on the process of developing an endodontic diagnosis, how to effectively diagnose and manage acute dental pain, and how to identify pain of nonodontogenic origin. The next chapter reviews the process of case selection and treatment planning, which is followed by chapters that prepare the clinician for endodontic treatment, including a thorough review of armamentarium, sterilization, and disinfection, where appropriate. The next chapter describes tooth morphology and how it relates to techniques crucial for achieving access to the entire root canal system. Following are three chapters that extensively describe the latest instruments, materials, techniques, and devices used for canal cleaning, shaping, and obturation. The concluding chapter in Part I reviews the legal responsibilities of the clinician and gives guidance for avoiding potential legal issues pertaining to patient care.

Part II starts with a chapter that brings together what is known about the development, structure, and function of the dentinal-pulpal complex and is followed by a chapter on how the pulp reacts to different stimuli, materials, and dental procedures. Following are chapters on the pathobiology of the periapex and the diverse aspects of endodontic microbiology, with special emphasis placed on the underlying principles for sound antimicrobial treatment of endodontic infections.

Part III begins with a new chapter on the rapidly developing field of regenerative endodontics and continues with a chapter that details the endodontic management necessary for optimizing favorable healing for the various types of traumatic dental injuries. The next chapter looks at the relationship between pulpal and periodontal tissues, and the following chapter presents insight into the medications used in endodontics, especially as it relates to the pharmacologic management of odontogenic pain. The chapter on periradicular surgery reviews how it has evolved into a precise, biologically-based adjunct to

nonsurgical root canal therapy and is followed by a new chapter designed to help clinicians make informative decisions about the restorative options available to endodontically-treated teeth. Part III concludes with a chapter that looks at the special considerations that must be addressed when treating geriatric, pediatric, and medically compromised patients, followed by a chapter that provides the information necessary for proper treatment-planning considerations and management of nonsurgical endodontic retreatment.

In addition to the 25 chapters in the text there are also five new chapters included on *Expert Consult*, the book's online component. These chapters address topics such as management of the fearful dental patient, the law and dental technology, the role of digital technology in practice, cone beam computerized tomography, and endodontic practice management. *Expert Consult* also provides access to a wide range of essential educational and clinically-based topics, which are further described on the next page.

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  - Chapter 26: Understanding and Managing the Fearful Dental Patient
  - Chapter 27: The Law and Dental Technology: Focus on Endodontics
  - Chapter 28: Digital Technology in Endodontic Practice
  - Chapter 29: Cone Beam Imaging for Endodontic Procedures
  - Chapter 30: Key Principles of Endodontic Practice Management
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- **Drug database:** Drug information accessible by indications, contraindications, and adverse reactions. Powered by Gold Standard—the leading developer of drug information.
- **Challenge:** Multiple-choice questions stemming from chapter-specific discussions or case scenarios, including answers and references to pages in the book.
- **Glossary and SORT:** Articles from *The Journal of Evidence-Based Dentistry—The Glossary of Evidence-Based Terms* and *JEBDP Improves Grading System and Adopts Strength of Recommendation Taxonomy Grading (SORT) for Guidelines and Systematic Reviews*—provided in their entirety.

Introduction

Welcome reader, to this tenth edition of *Pathways of the Pulp,* a reconceptualized textbook that blends form, content, print, and digital formats to describe and detail the transformative future of endodontics. The first edition of *Pathways of the Pulp* was published in 1976, with the aim of describing in a single print volume the basic and clinical science of what was then the emerging specialty of endodontics. Nine editions later, we have continued to refine and redefine the scope and organization of our textbook to mirror and represent the changing science of endodontics and the evolving landscape of learning and communication. Now offered in 14 different languages, *Pathways of the Pulp* is the most comprehensive and innovative evidence-based endodontic textbook available.

Beginning with the ninth edition, *Pathways of the Pulp* has been available anytime, anywhere to readers in an online version, enhanced with instructional images and graphics and periodic content updates. The new format of the tenth edition has expanded the use of digital resources to achieve a seamless blending of print and online content through the addition of narrated video clips and animations, and quarterly content updates drawn from the peer-reviewed literature.

Although the evolving form and format of *Pathways of the Pulp* defines its commitment to education, the steady and selfless dedication of the more than 150 contributors to the 10 editions honors and extends our tradition of collaborating with the most qualified and highly regarded authors, academicians, and researches in our field. Working with all of the dedicated teachers and scientists who author our chapters continues to be the signature honor and privilege for the editors. We are especially grateful to Dr. Lou Berman, who in his capacity as Webmaster has made our promise of online availability and digital features a reality. We are all indebted as well to Elsevier for its willingness to explore and support ever more dynamic and engaging ways of imparting knowledge and communicating with our readers.

The nine prior editions of *Pathways of the Pulp* have begun with a chapter on the history of endodontics and the inspiring story of the intense interplay between basic science and clinical care that established and certified our specialty. The vision and dedication of our founders who lived that history are now embedded in the field and practice of endodontics, and to call them out separately would seem to distance them rather than bring them closer to us. What now connects us more productively and most effectively with our storied past is our commitment to redefining that critical interplay between basic science and clinical care in terms of new technologies, new possibilities, and new expectations.

In this tenth edition, through evidence-based discussions in all of our 30 chapters, including five that are only available online, we have meticulously detailed that what was once regarded as science fiction in clinical care is now rapidly becoming clinical endodontic fact. The quality of care available to patients has never been higher, from the administrative efficiency and privacy protection afforded to patients through electronic medical records, to the greatly enhanced effectiveness of infection management through real-time PCR. A sampling of the innovative and paradigm shifting developments outlined in this edition include the following:

- More accurate devices to determine pulp vitality and inflammation
- Greater control of the duration of local anesthesia on a case-by-case basis
- Cleaning and shaping root canals in less than 5 minutes with a single device
- Regeneration of the pulp to replace canal obturation
- Nonsurgically accelerating the remineralization of periapical lesions through the apical foramen

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.

As I conclude this Introduction I also bring to a close my 35 years as editor of *Pathways of the Pulp*. It is with complete confidence and great excitement that I pass the editorship of our book to Dr. Kenneth Hargreaves, a uniquely qualified and accomplished scientist, educator, and clinician.

I am deeply grateful for the privilege of editing and contributing to *Pathways of the Pulp*. It has been a true labor of love, which has allowed me to indulge my passion for science and my love of teaching. Perhaps most importantly, however, my editorship has taught me what it means to practice endodontics, a true healing art.

Stephen Cohen

CHAPTER 1

Diagnosis

LOUIS H. BERMAN and GARY R. HARTWELL

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
- Root Fractures and Cracks
- Referred Pain

CLINICAL CLASSIFICATION OF PULPAL AND PERIAPICAL DISEASES
- Pulpal Disease
- Apical (Periapical) Disease
- Pain of Nonodontogenic Origin

ART AND SCIENCE OF DIAGNOSIS

Almost a century ago, Dr. Hermann Prinz wrote: "The object of the practice of clinical dentistry … is to institute preventive measures, to relieve suffering, and to cure disease. These purposes are not achieved by the haphazard utilization of a few therapeutic formulas or of certain mechanical procedures, but they are based on a thorough knowledge of clinical pathology."[75] This statement has truly withstood the test of time and remains a cornerstone in diagnosis. But the gathering of scientific data is not enough to formulate an accurate clinical diagnosis. The data must be interpreted and processed in order 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 dental pathosis is the culmination of the art and science of making an accurate diagnosis.

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 a planned, methodical, and systematic approach to this investigatory process is crucial.

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

1. The patient tells the clinician why the patient is 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 differential diagnosis.
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. The diagnostic tests that are used become the science behind the creation of 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 11 for more information).

The reasons patients give for consulting with a clinician are often as important as the diagnostic tests performed. These remarks serve as important clues for the clinician that will help in formulating a correct diagnosis. Without these direct and unbiased comments, objective findings may lead to an incorrect diagnosis. The clinician may find dental pathosis, but it

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 the modality of treatment, or change the treatment plan.

may not be the pathologic condition that mediates the patient's chief complaint. Investigating these complaints may indicate that the patient's concerns are secondary to a medical condition or possibly a result of recent dental treatment. 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. Therefore, the clinician must pay close attention to the actual expressed complaint, determine the chronology of events that led up to this complaint, and question the patient as to any 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 individual 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 indicate that this review has been done by initialing the medical history form. Any patient "of record" should be questioned at each treatment visit to determine any changes in the patient's medical history or medications. A more thorough and complete update of the patient's medical history should be completed if the patient has not been seen for over a year.[35,56]

Baseline blood pressure and pulse should be recorded for a patient at each treatment visit. Elevation in blood pressure or a rapid pulse rate may indicate an anxious patient who may require a reduced stress protocol, or it may indicate that the patient has hypertension or other cardiovascular health problems. 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.[42,56,78]

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 drug allergies or allergies to dental products, an artificial joint prosthesis, or organ transplants or is taking medications that may negatively interact with common local anesthetics, analgesics, and antibiotics.

The previous listing may seem overwhelming, but it emphasizes the importance of attaining a thorough and accurate medical history before any dental treatment is provided. A multitude of textbooks and journal articles are available to keep the dental community current on the appropriate ways to provide dental care for patients with medical problems (e.g., *The Merck Manual* at http://www.merck.com/mrkshared/mmanual/home.jsp). These sources provide the clinician with details about the various medical conditions and the dental treatment modifications that must be made in order to provide the appropriate care.

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[42,56,78]

Pulmonary: Chronic obstructive pulmonary disease, asthma, tuberculosis[56,95]

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[17,21,22,33,56]

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[22,28,53,59,64,73,99]

Neurologic: Cerebrovascular accident, seizure disorders, anxiety, depression and bipolar disorders, presence or history of drug or alcohol abuse, Alzheimer's disease, schizophrenia, eating disorders, neuralgias, multiple sclerosis, Parkinson's disease[23,29]

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 than the medical condition itself. More common examples of medication side effects are stomatitis, xerostomia, petechiae, ecchymoses, lichenoid mucosal lesions, and bleeding of the oral soft tissues.[56]

In arriving at an accurate dental diagnosis, a clinician must also be aware that some medical conditions can have clinical presentations that mimic oral pathologic lesions.[91] Tuberculosis involvement of the cervical and submandibular lymph nodes can lead to a misdiagnosis of lymph node enlargement as a result of an odontogenic infection. Lymphomas can also involve these same lymph nodes.[56] 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.[28,53,56,59] 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 also 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.[56] Trigeminal neuralgia, referred pain from cardiac angina,

and multiple sclerosis can also mimic dental pain (see also Chapter 3). 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 will 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 the diagnosis of dental pathosis; this 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 etiology, then consideration must be given that an existing medical problem could be the true etiology. In such instances a consultation with the patient's physician is always appropriate. As noted previously, there are many current textbooks and journal articles that can serve as reference materials for clinicians who encounter some of these medical problems on an infrequent basis.

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 an S.O.A.P. format is used, designating the *S*ubjective *O*bjective *A*ppraisal *P*lan for the diagnostic workup. There are also built-in features within some practice management software packages that allow digital entries into the patients' 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 up to the chief complaint. The conversation should be directed by the clinician in order to produce 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 for the resolution of 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 promulgated the request for an evaluation. The dental history is divided into five basic directions of questioning: localization, commencement, intensity, provocation, 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 may remember when these symptoms started. Sometimes, the patient will even remember the initiating event: It may be spontaneous in nature, it may have begun after a dental visit for a restoration, trauma may be the etiology, or biting on a hard object may have initially produced the symptoms. 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.

Provocation and Relief of Pain: *"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. Some symptoms may be relieved by nonprescription pain relievers, and others may require narcotic medication for the reduction of symptoms (see Chapter 19 for more information). Note that patients who are using narcotic pain relievers may respond differently to questions and diagnostic tests, which may alter the objectivity of the diagnostic results. These provoking and relieving factors may help 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 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 as to how long the sensation is felt by the patient, and documented in terms of seconds or minutes.

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 be helpful in facilitating complete and accurate documentation. *(Courtesy Dr. Ravi Koka, San Francisco, Calif.)*

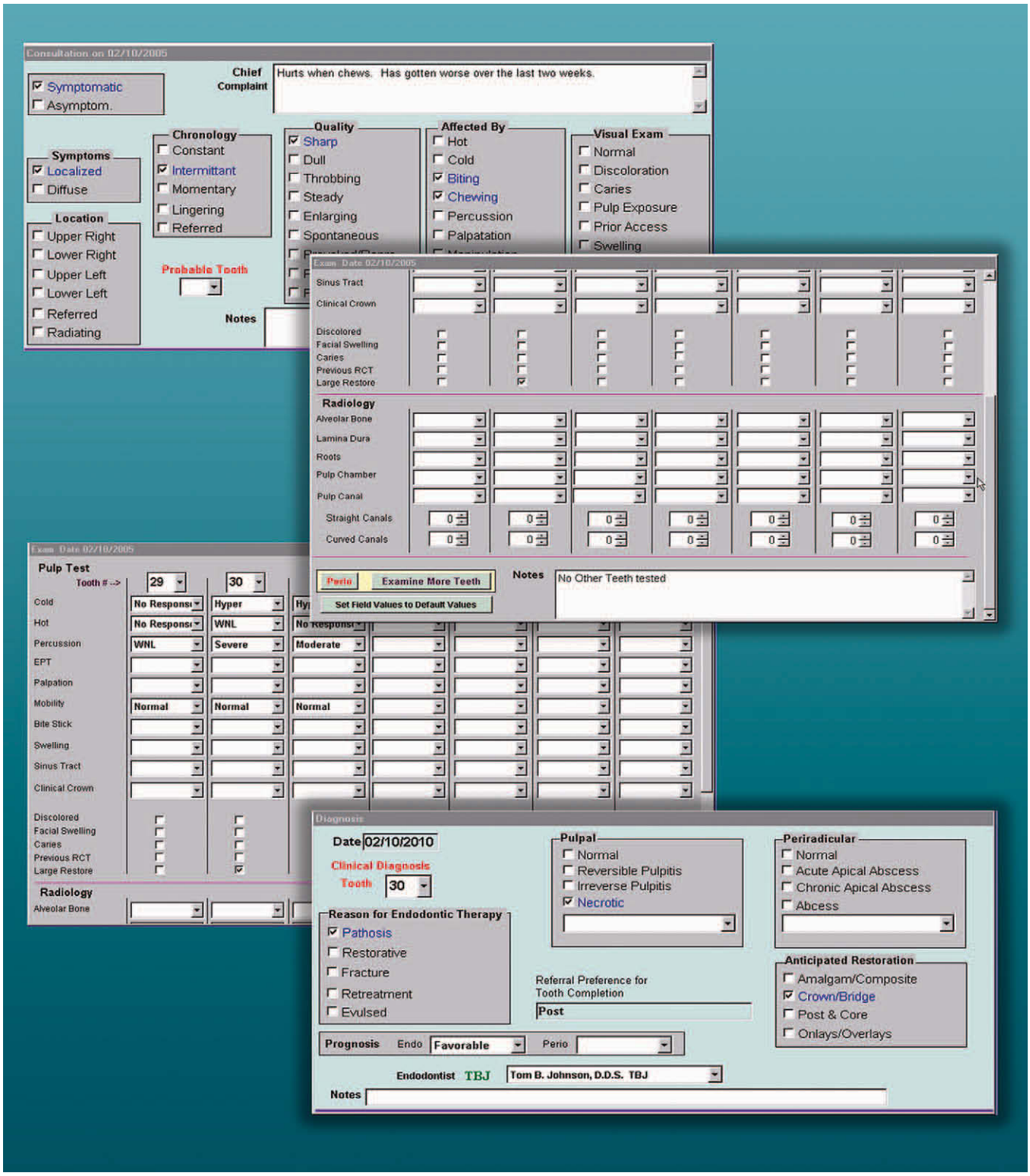


FIG. 1-4 Several practice management software packages have features for charting endodontic diagnosis using mouse-driven inputs, user-defined drop-down menus, and areas for specific notations. Note that for legal purposes, it is desirable that all recorded documentation have the ability to be locked, 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, Texas.)*



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 is indicative of an early canine space infection.

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, 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 indicative of 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 etiology is not found.[54] This 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 (Fig. 1-5) may be the earliest sign of a canine space infection.[55,94] 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. Further discussions of fascial space infections may be found in Chapter 15.

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 have as much likelihood to exit to the lingual where other spaces would be involved. For infections associated with these teeth, the root apices of the maxillary teeth must lie superior



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,** Swelling in this case is also 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.

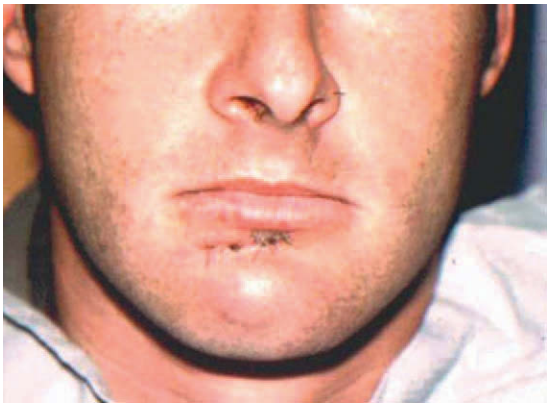


FIG. 1-8 Swelling of the submental space associated with periradicular disease from the mandibular incisors.

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.[55,94]

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.[55,94] There is a complete review of fascial space infections in Chapter 15.

Sinus tracts of odontogenic origin may also open through the skin of the face (Figs. 1-9 and 1-10). 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. 1-11; see also Fig. 1-9). Many patients with extraoral sinus tracts will give a history of being treated by general physicians and dermatologists with systemic or topical antibiotics and/or surgical procedures in attempts to heal the extraoral stoma. In these particular cases, only after multiple treatment failures are the patients finally referred to a dental clinician to determine whether there is a dental etiology.[46]

Intraoral Examination

The extraoral examination may give the clinician insight as to which intraoral areas may need a more focused evaluation. Extraoral swelling, localized lymphadenopathy, or an extraoral 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

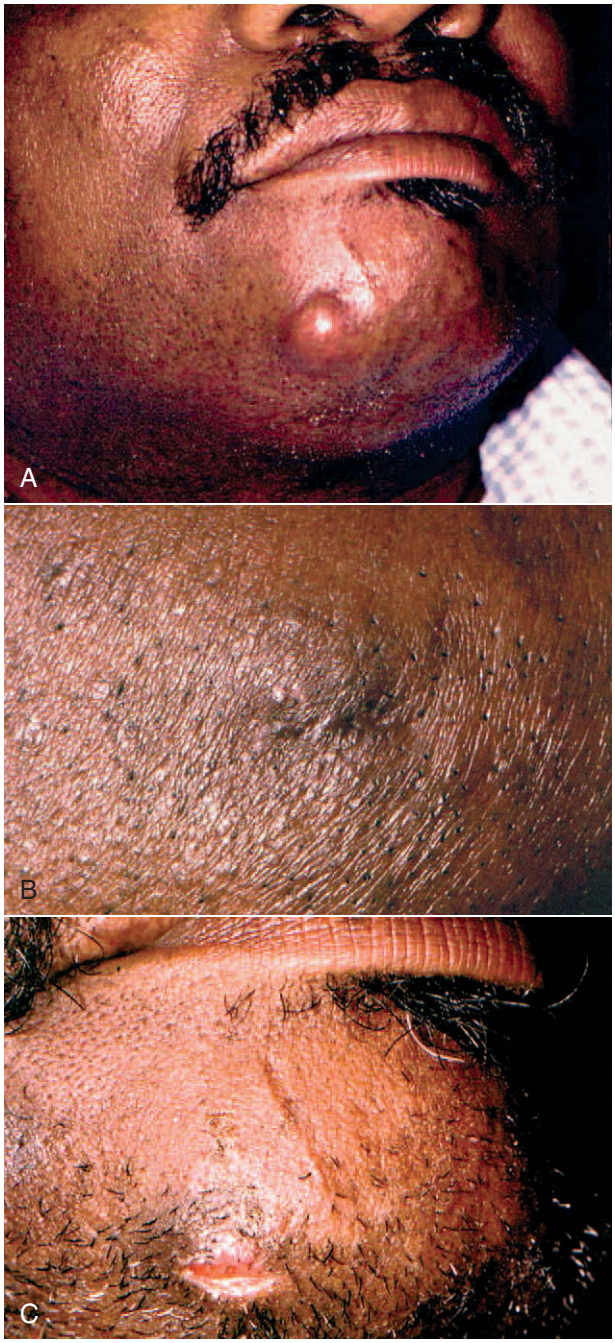


FIG. 1-9 A, Note the parulis on the right anterior side of the face. The extraoral drainage was found to be associated with periradicular disease from the mandibular right canine. **B,** Note the initial scar associated with the extraoral drainage incision after the parulis was drained and root canal therapy was performed on the canine. **C,** The healed incision area 3 months after drainage was achieved. Note the slight inversion of the scar area.

mucosa should be dried, either with an air syringe or a 2 × 2-in. gauze pad. By retracting the tongue and cheek, all of the soft tissue should be examined for any abnormalities in color or texture. Any raised lesions or ulcerations should be documented and, when necessary, evaluated with a biopsy or referral.[58]

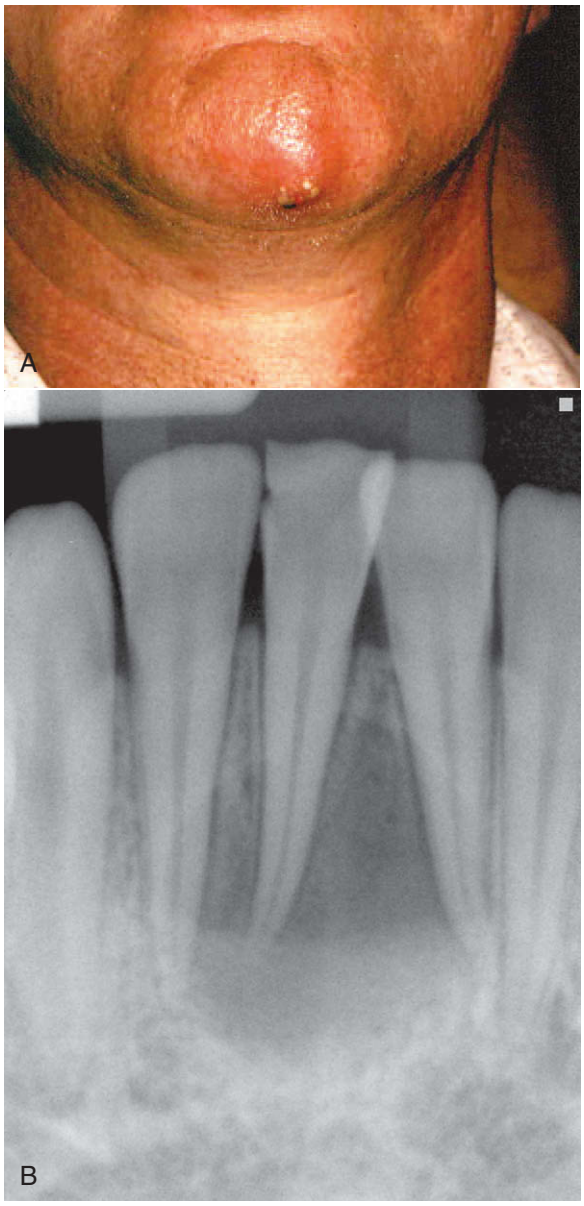


FIG. 1-10 A, Extraoral sinus tract opening on the skin in the central chin area. **B,** Radiograph of mandibular incisors and canine after root canal therapy.

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 etiology 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. 1-12) 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



FIG. 1-11 A, A culture is obtained from the drainage from the extraoral sinus tract. **B,** The healed opening of the extraoral sinus tract 1 month after root canal therapy was completed. Note the slight concavity of the skin in the area of the healed extraoral opening.



FIG. 1-13 Fluctuant swelling in the posterior palate associated with periradicular disease from the lingual root of the maxillary first molar.



FIG. 1-12 Fluctuant swelling in the anterior palate associated with periradicular disease from the lingual root of the maxillary first premolar.



FIG. 1-14 Fluctuant swelling in mucobuccal fold associated with periradicular disease from the maxillary central incisor.

in the posterior palate (Fig. 1-13) is most likely associated with the palatal root of one of the maxillary molars.[55,94]

Intraoral swelling present in the mucobuccal fold (Fig. 1-14) 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 15). 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.[55,94]

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*. This pathway,[7] 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 between two internal organs or a pathway between two epithelium-lined surfaces.[1]

Histologic studies have found that most sinus tracts are not lined with epithelium throughout their entire length. Harrison and Larson[40] found only 1 of the 10 sinus tracts they studied were lined with epithelium. The other nine specimens were lined with granulation tissue. In a study with a larger sample size, Baumgartner and colleagues[7] found 20 of 30 specimens did not have epithelium that extended beyond the level of the surface mucosa rete ridges. The remaining 10 specimens had some epithelium that extended from the oral mucosa surface to the periradicular lesion. 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 will necessitate further diagnostic procedures to determine whether other etiologic factors 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 magnitude before the 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 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 termination of the sinus tract is determined by following the path taken by the gutta-percha cone (Fig. 1-15). This will direct the clinician to which tooth is involved, and more specifically, which root of that tooth is the source of the pathosis. Once the causative factors related to the development of the sinus tract are removed, the stoma and the sinus tract will close within a few 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 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

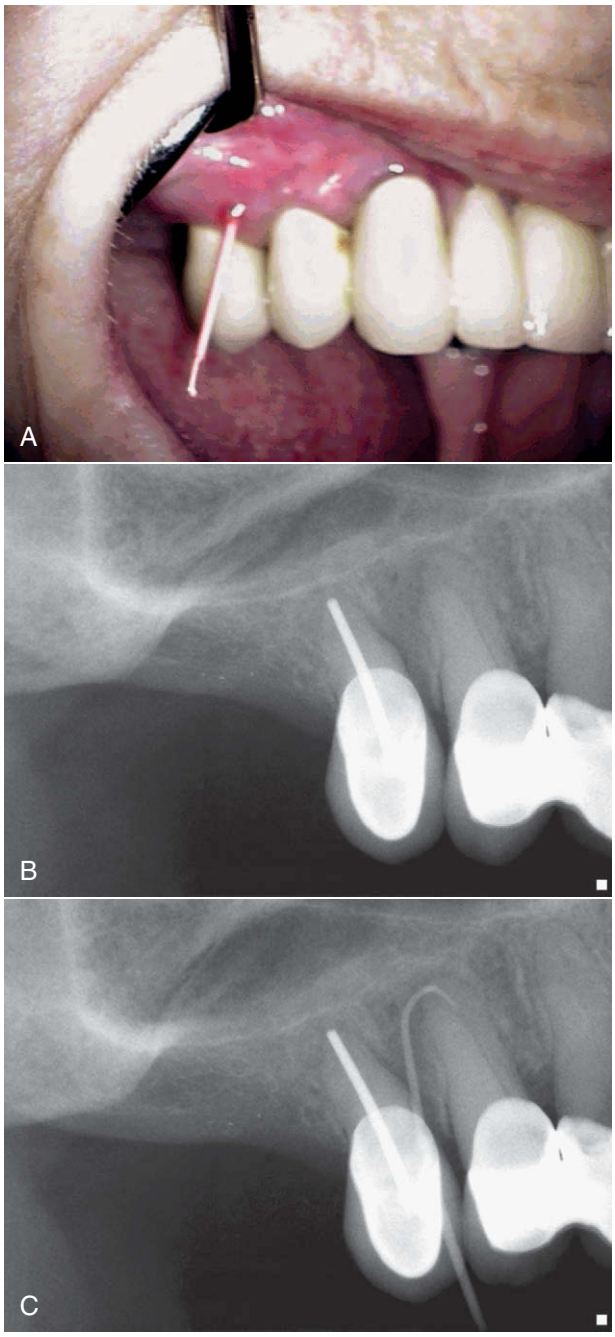


FIG. 1-15 A, To locate the source of an infection, the sinus tract can be traced by threading the stoma with a gutta-percha point. **B,** The radiograph of the area shows an old root canal in tooth #4, and a questionable radiolucent area associated with tooth #5, with no indication of the etiology of 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 tooth #5.

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 will assist in verifying the etiology.[39,90,93]

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 boney 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.

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 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 is coming from is interpreted by the mesencephalic nucleus, receiving its information from proprioceptive nerve receptors. Although subject to debate, the general belief is that there are few, if any, proprioceptors in the dental pulp; however, they are prevalent in the periodontal ligament spaces.[16] 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 give more accurate results. The contralateral tooth should first be tested as a control, as well as 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. The testing should initially be done gently, with light pressure being applied digitally with a gloved finger-tapping. If the patient cannot detect any 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. 1-16). The teeth 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.



FIG. 1-16 Percussion testing of a tooth, using the back end of a mirror handle.

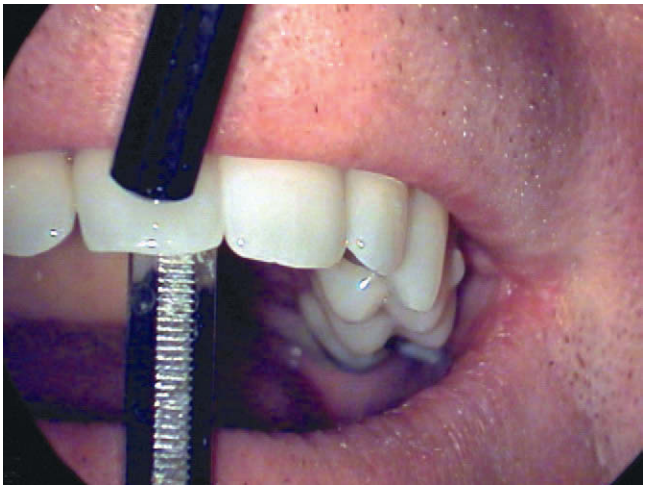


FIG. 1-17 Mobility testing of a tooth, using the back ends of two mirror handles.

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. 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 of the tooth, and one on the lingual aspect of the tooth (Fig. 1-17). Any mobility over +1 mobility should be considered abnormal (Box 1-2). However, the teeth should be evaluated on the basis of how mobile they are relative to the adjacent and contralateral teeth.

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.

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 etiology and is typically more generalized in other areas of the mouth. However, isolated areas of vertical bone loss may be of an endodontic etiology, 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 elsewhere in this chapter, 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).

Pulp Tests

Pulp testing involves attempting to make a determination of the responsiveness of pulpal sensory neurons. 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 objectively 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.[91,92]

Thermal

Various methods and materials have been used to test the pulp's response to thermal stimuli. The baseline or normal response to either hot or cold is a patient's report that a sensation is felt but disappears immediately on removal of the thermal stimulus. Abnormal responses include a lack of response to the stimulus, the 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.

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 any onset of symptoms.

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.

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. In these cases the patient may present with cold liquids in hand just to minimize the pain (Fig. 1-18). In these 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.

Cold is the primary pulp testing method for many clinicians today. To be most reliable, cold testing should be used in conjunction with an electric pulp tester so that the results

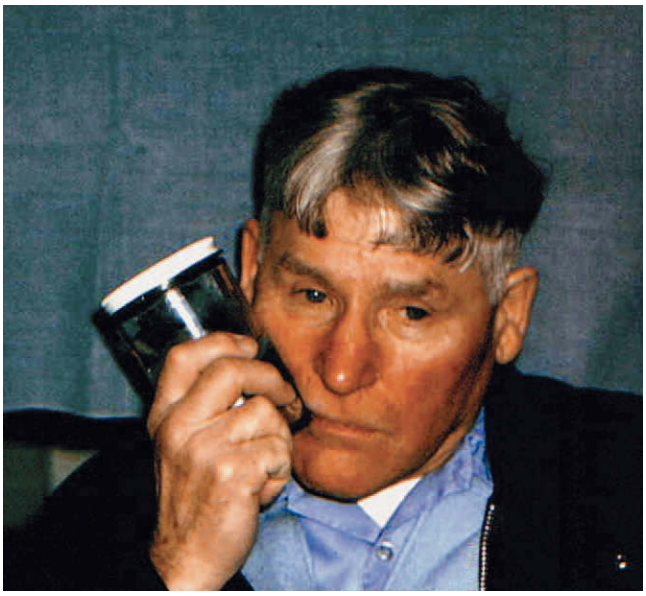


FIG. 1-18 Patient has irreversible pulpitis associated with the mandibular right second molar and has found that the only way to alleviate the pain is to place a jar filled with ice water against the right side of his face.

from one test will verify the findings of the other test. If a mature, untraumatized tooth does not respond to both electric pulp test and cold test, then the pulp should be considered necrotic.[71] However, a multirooted tooth, with at least one root containing vital pulp tissue, may respond to a cold test even if one or more of the roots contain necrotic pulp tissue.[71] Cold testing can be accomplished similarly to heat testing, by individually isolating teeth with a rubber dam. This technique for cold testing is especially useful for patients presenting with porcelain jacket crowns or porcelain-fused-to-metal crowns where there is no natural tooth surface (or much metal) accessible. Another benefit of this technique for cold testing is that it requires no armamentarium except for a rubber dam. If a clinician chooses to perform this test with sticks of ice, then the use of the 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,* has been found to be reliable in eliciting a positive response if vital pulp tissue is present in the tooth.[32,71,72] One study found that vital teeth would respond to both CO_2 and skin refrigerant, with skin refrigerant producing a slightly quicker response.[48] Carbon dioxide has also been found to be effective in evaluating the pulpal response in teeth with full coverage crowns for which electric pulp testing is not possible.[6] For testing purposes a solid stick of CO_2 is prepared by delivering CO_2 gas into a specially designed plastic cylinder (Fig. 1-19). 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 × 2-in. gauze or cotton roll so the CO_2 will not come into contact with these structures. Because of the extremely cold temperature of the CO_2 (−69° F to −119° F; −56° C to −98° C) burns of the soft tissues can occur. Investigators[6] have demonstrated on extracted teeth that CO_2 application resulted in a significantly greater intrapulpal temperature decrease than either skin refrigerant or ice. Studies[45,77] have also shown 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.

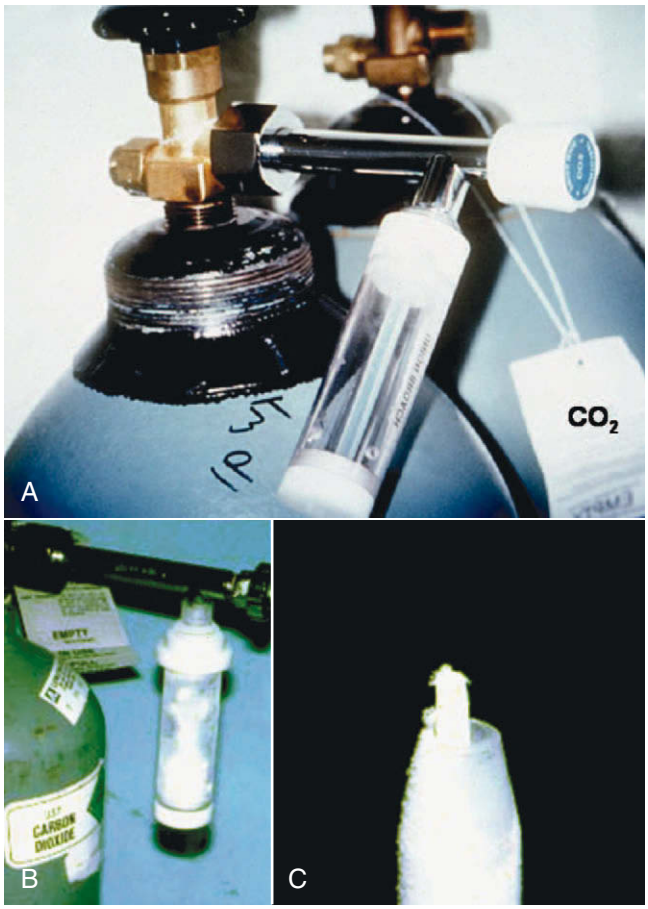


FIG. 1-19 A, Carbon dioxide tank with apparatus attached to form solid CO_2 stick/pencil. **B,** CO_2 gas being formed into a solid stick/pencil. **C,** CO_2 stick/pencil being extruded from the end of the plastic carrier.

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 CO_2.[32,48] One of the current product contains 1,1,1,2-tetrafluoroethane, which has zero ozone depletion potential and is environmentally safe. It has a temperature of −26.2° C.[48] The spray is most effective for testing purposes when it is applied to the tooth on a large #2 cotton pellet (Fig. 1-20). In one study[47] 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 be tested to establish a baseline response. It appears that 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.[6,32]

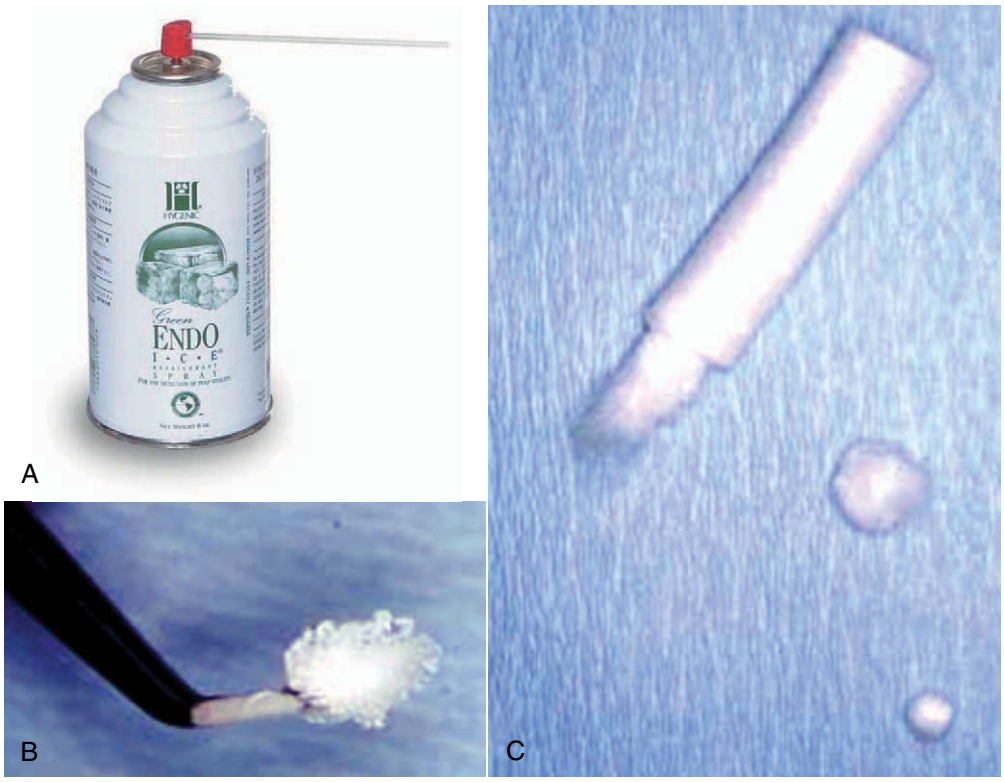


FIG. 1-20 A, Refrigerant spray container. **B,** A cotton roll can be used to form a large cotton pellet, or a size #2 (large) cotton pellet can be used to apply the refrigerant spray to the tooth surface. The small #4 cotton pellet does not provide as much surface area as the #2 cotton pellet, and therefore it should not be used to deliver the refrigerant to the tooth surface. **C,** A large cotton pellet that has been sprayed with the refrigerant and is ready to be applied to the tooth surface. (***A,*** *Courtesy Coltène/Whaledent, Cuyahoga Falls, Ohio.)*

One study[72] compared the ability of thermal and electric pulp testing methods to register the presence of vital pulp tissue. 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%.

Electric

Assessment of pulp vitality is most frequently accomplished by electric pulp testing and/or cold testing. The vitality of the pulp is determined by the intactness and health of the vascular supply, not the status of the pulpal nerve fibers. Even though advances are being made with regard to determining the vitality of the pulp on the basis of the blood supply, this technology is not accurate enough at this time to be used on a routine basis in a clinical setting.

The electric pulp tester has limitations in providing 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.[91,92] 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. Numerical 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[91,92] 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. The electric pulp tester will not work unless the probe can be placed in contact with or be bridged[69] to the natural tooth structure. With the advent of universal precautions for infection control, the patient may be required to place a finger or fingers on the tester probe to complete the electric circuit for some models; however, lip clips are an alternative to having patients hold the tester. The use of rubber gloves prevents the clinician from completing the circuit.[3] Proper use of the electric pulp tester requires that the teeth to be evaluated be 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 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.[62] 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.[10] Once the probe is in contact with the tooth, the patient is asked to touch or grasp the tester probe (Fig. 1-21, *A*).[3] This completes the circuit and initiates the delivery of electric current to the tooth. 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

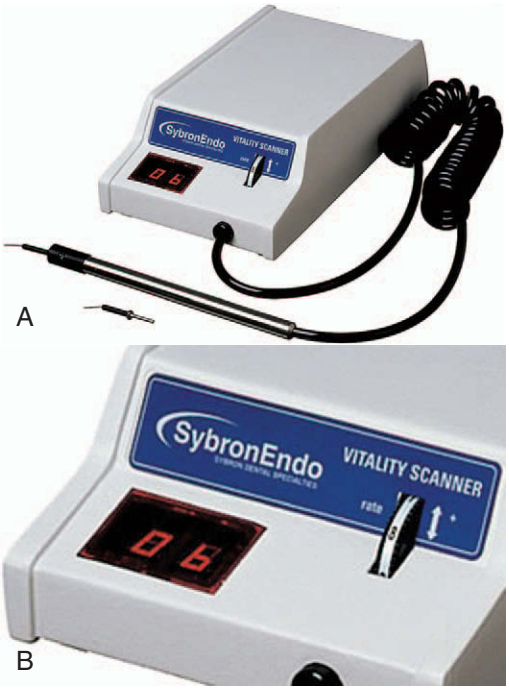


FIG. 1-21 A, View of an electric pulp tester with probe. The probe tip will be coated with a medium such as toothpaste and placed in contact with the tooth surface. The patient will activate the unit by placing a finger on the metal shaft of the probe. **B,** View of the electric pulp tester control panel: The knob on the right front 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, Calif.)*

readings from the pulp tester are recorded (Fig. 1-21, B) and will be evaluated once all the appropriate teeth have been tested and the results obtained from 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.[69] 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.

As noted previously, studies[32,71,72] have shown that there does not appear to be any significant difference between the pulp testing results obtained with the electric pulp tester and those obtained by the thermal methods, although cold tests have been shown to be more reliable than electric pulp tests in younger patients with less developed root apices.[71] However, unlike electric pulp testing, cold testing can reveal the health and integrity of pulp tissue (i.e., no response, a momentary response, or a prolonged, painful response after the thermal stimulus is removed). This is why it is a good rationale to verify the results obtained by one testing method by comparing them with the results obtained by the other method. 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.



FIG. 1-22 Nellcor OxiMax N-600× pulse oximeter. *(Courtesy Nellcor Puritan Bennett [Boulder, Colo]; part of Covidien.)*

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 will be frequency-shifted by 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.[81]

Several studies[25,44,61,81-83] have found LDF to be an accurate, reliable, and reproducible method of assessing pulpal blood flow. Even with these positive findings, the technology is not advanced enough for this method to be used on a routine basis in a dental practice. In one clinical trial[25] fabricating the individualized stabilization jig, making the LDF recordings took approximately 1 hour, a finding not unique to this study. If technology can be developed whereby testing by LDF can be accomplished in minutes, it will likely replace the thermal and electric pulp testing methods.

As is discussed in Chapter 16, 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.[96]

Pulse Oximetry

One of the great advantages of pulp testing with devices such as the laser Doppler flowmeter is that the collected data are based on objective findings rather than subjective patient responses. The pulse oximeter is another such noninvasive device (Fig. 1-22). 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; how much is 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, and 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.[84] 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.

Attempts to use the pulse oximeter to diagnosis pulp vitality have met with mixed results. Some studies[84,103] have reported that pulse oximetry is a reliable method for assessing pulp vitality. Others[103] have stated that in its present form the pulse oximeter is not of predictive diagnostic value for diagnosing pulp vitality. Most of the problems appear to be related to the currently available technology. Some investigators have concluded that these devices used for pulp testing are too cumbersome and complicated to be used on a routine basis in a dental practice.[50,84,103] However, Gopikrishna and colleagues[37] developed a sensor that can be directly applied to a tooth for assessing human pulp vitality and found it more accurate than electric and thermal pulp tests. This device has been especially useful in evaluating teeth that have been subjected to traumatic injuries,[38] because these teeth tend to present, especially in the short term, with questionable vitality using conventional pulp testing methods.[4]

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 *periradicular 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 elicit pain only when the percussion or bite test is applied in a certain direction to one cusp or section of the tooth.[15,80]

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 applicators, toothpicks, orangewood sticks, and rubber polishing wheels. Today several devices are specifically designed to perform this test. The Tooth Slooth (Professional Results, Laguna Niguel, CA) (Fig. 1-23) 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 teeth should 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 on release of biting pressure.



FIG. 1-23 To determine which tooth is sensitive to mastication and which part of that tooth is sensitive, having the patient bite on a specially designed bite stick is often helpful.

Test Cavity

The test cavity method for assessing pulp vitality is seldom used today. 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 might 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, it 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 the tooth, the application of a stain to the area is often of great assistance. Applying a bright fiberoptic light probe to the surface of the tooth is also helpful. This will be elaborated on later in this chapter in the discussion of the detection of cracks and fractures.

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, after an appropriate period of time, the pain is not eliminated, the clinician should similarly repeat this technique on the mandibular teeth below. It should be understood that periodontal ligament injections may inadvertently anesthetize an adjacent tooth, and thus are more useful for identifying the arch rather than the specific tooth.

Radiographic Examination and Interpretation

Intraoral Radiographs

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 this diagnosis, the potential for inappropriate treatment may be great if the radiograph alone is used for making this diagnosis. The clinician should not subject the patient to unnecessary multiple radiation exposures; often two pretreatment images from different angulations are sufficient. But in extenuating circumstances, especially when the diagnosis is difficult, multiple exposures may be necessary in order to determine the presence of multiple roots, 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,[34] there was agreement for the presence of pathosis in only 50% of the radiographically evaluated cases, as interpreted by two endodontists, three second-year residents, and an associate professor in radiology. In addition, when the cases were evaluated several months later, the evaluators agreed with their own original diagnosis only 75% to 83% of the time. Again, this emphasizes the necessity for other 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. Therefore, 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



FIG. 1-24 Radiograph of what appears to be a periapical lesion associated with a nonvital tooth; however, the tooth is vital. The appearance of apical bone loss is actually secondary to a cementoma.

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 infection in the pulpal space transgresses through the pulp canal space and into the adjacent alveolar bone. The pathosis may present merely as a widening or break in the lamina dura—the most consistent radiographic finding when a tooth is nonvital[49]—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 canal. On occasion there may be no radiographic change at all, even in the presence of an acute periradicular abscess.

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

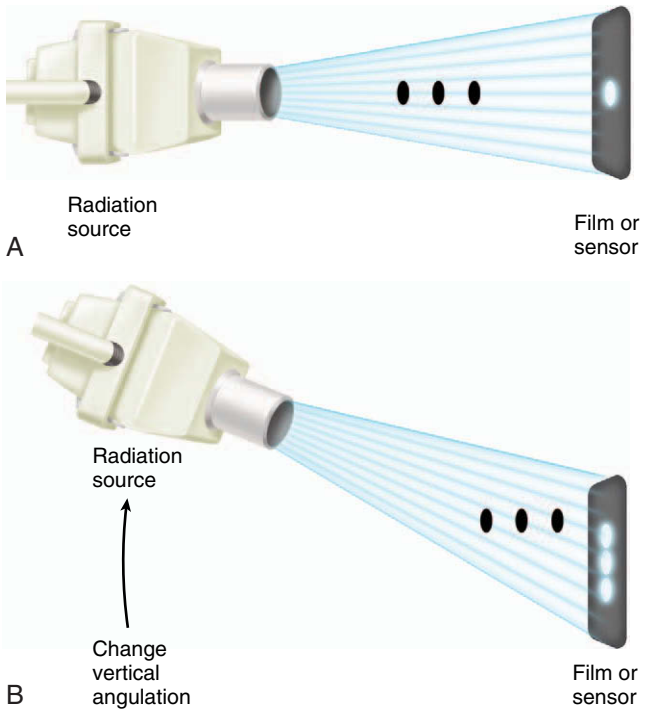


FIG. 1-25 Radiographic images are only two-dimensional, and often it is difficult to discriminate the relative location of overlapping objects. **A,** When the source of the radiation is directly perpendicular to overlapping objects, the image is captured without much separation of the objects. However, 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.

not be detected if the bone loss is only in cancellous bone. However, radiographic evidence of pathosis will be observed once this bone loss extends to the junction of the cortical and cancellous bone, as was illustrated by Bender and Seltzer,[11] who created artificial lesions in cadaver bone and evaluated them radiographically. As a follow-up to this study, the authors reported why certain teeth are more prone to exhibit radiographic changes than others, depending on their anatomic location.[12] Their findings revealed that 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. Most anterior and premolar teeth are located close to the cortical–cancellous bone junction. For this reason, periapical pathosis from these teeth is exhibited sooner. 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.

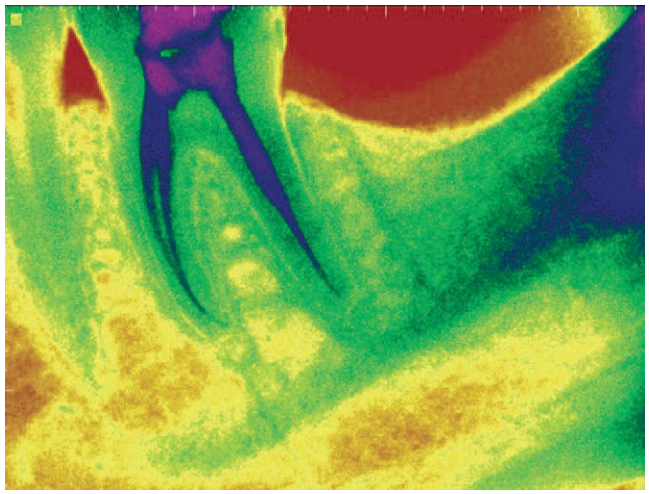


FIG. 1-26 Digital radiography has an advantage over conventional film in that the image can be enhanced and colorized, a useful tool for patient education.

Digital Radiography

One technique for controlling many of the variables in the diagnostic quality of conventional radiography has been the advent of *digital radiography*. This technology has been available for approximately 20 years but has more recently been refined with better hardware and more user-friendly software. Digital radiography has the ability to capture, view, magnify, enhance, and store radiographic images in an easily reproducible format that does not degrade over time.

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 remotely 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 and enhanced. 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 5 and online Chapters 28 and 29.

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, because there is none. The clinician can zoom in to different areas on the x-ray image, digitally enhance the image in order to better visualize certain anatomic structures, and in some cases the image can even be colorized, a useful tool for patient education (Fig. 1-26).

Until recently, x-ray film has had a slightly better resolution than most digital radiography images, at about 16 line pairs per millimeter (lp/mm).[63] However, some sensor manufacturers are now claiming to have resolutions beyond that of film and up to 22 lp/mm. 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. The 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 acceptability 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.[24,52,70] However, digital radiography has the advantage over conventional film in that there is no diminution in diagnostic quality caused by developing and processing errors, and it has the ability to enhance, magnify, store, and electronically send the images, as well the ability to duplicate the original radiograph as a perfect copy. In 1998, the American Association of Endodontists (Chicago, IL) predicted that "digital radiography will rapidly replace conventional dental X-rays."[2] The reader is referred to Chapters 28 and 29 for more information about digital radiography.

Cone-Beam Volumetric Tomography

The radiographic interpretation of a potential endodontic pathosis is an integral part of endodontic diagnosis and prognosis assessment. Until about 10 years ago, routine dental radiography consisted of only two-dimensional imaging. But limitations in conventional radiography promulgated a need for three-dimensional imaging, known as *cone-beam volumetric tomography* (CBVT) (also known as *cone-beam volumetric imaging* [CBVI] or cone-beam computerized tomography [CBCT]). Although a form of this technology has existed since the early 1980s,[79] these devices first appeared specifically for use in dentistry in 1998.[65] Most of these machines are similar to a dental panoramic radiographic device, whereby the patient stands or sits as a cone-shaped 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). The survey of the scans can be directly 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 interactively used by another clinician.

In general, the dental clinician will desire a limited field of vision, confining the study to the maxilla and mandible. 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



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



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, Calif.)*

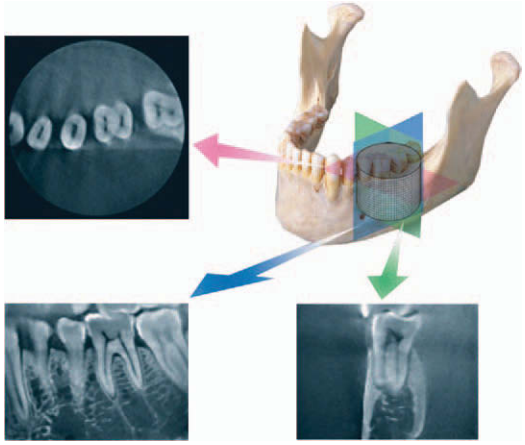


FIG. 1-29 Cone-beam volumetric tomography has the advantage of being able to detect pathosis in the bone or associated with the teeth without the obstruction of anatomic structures. The planes of vision may be axial, sagittal, or coronal. *(Courtesy J. Morita USA, Irvine, Calif.)*

intracranial aneurysms, that when undetected could be life-threatening.[66] The radiation source of CBVT 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 CBVT, 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, providing for a more accurate interpretation of anatomic structures. 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.[27]

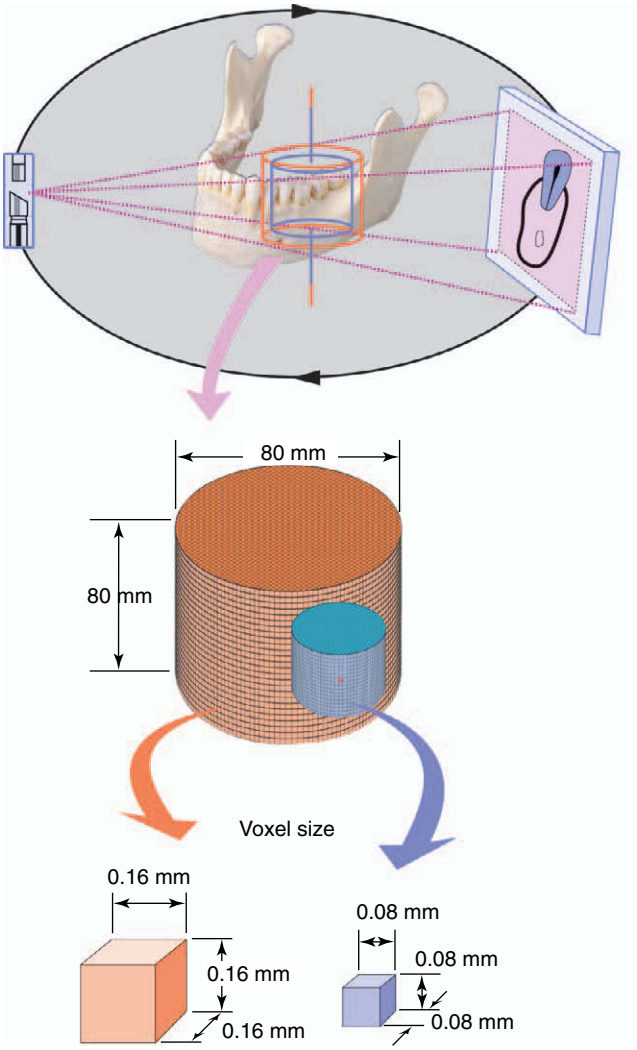


FIG. 1-30 The radiation source in cone-beam volumetric tomography is conical. The receiving sensor captures the image as "voxels," or three-dimensional pixels of information, allowing digital interpretation.

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. In 1961, Bender and Seltzer[11,12] showed that artificially created "lesions" in cancellous bone were radiographically undetected unless the bone loss extended to the junction between the cancellous and cortical bone. Therefore, an infection that is present only in the cancellous bone will typically not be radiographically visible. Consistent with this finding, if the root of a nonvital infected tooth is closer to the cortical bone (e.g., the *mesial* root of a mandibular first molar), the pathosis will be detected sooner than if the infected root is more centered within the cancellous bone (e.g., the *distal* root of a mandibular first molar). Because CBVT can clearly visualize the interior of the cancellous bone without the superimposition of the cortical bone, these boney defects may be more easily detected. In 2007, Lofthag-Hansen and colleagues[57] studied 48 teeth with "endodontic problems," using both standard periapical radiographs and CBVT. Three different oral radiologists reviewed the scans. They found demonstrable lesions associated with 32 of these teeth, using standard periapical radiographs, but found pathosis associated with 42 teeth when using CBVT. In addition, 53 roots from these teeth were found to have pathosis associated with them, using standard periapical radiographs, whereas when using CBVT, 86 roots were found to have associated pathosis. Nakata and colleagues[67] showed how periapical bone loss associated with a palatal root was detected by CBVT when it was not visible in conventional periapical radiographs. 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 radiographic interpretation sometimes difficult or impossible. Cotton and colleagues[19] presented seven case reports in which CBVT was invaluable in ascertaining information essential for making a diagnosis related to alveolar pathoses. These scans revealed an incomplete root canal, a large incisive foramen suspicious of being a lesion of endodontic origin, a root fracture, internal resorption, cervical resorption, root canal cement extruded with symptoms associated with the mental nerve, and a lesion associated with a failed implant that was originally thought to have been of endodontic origin from an adjacent tooth. In 2001, Velvart and colleagues[102] found 100% accuracy with CBVT scans predicting 78 periapical lesions that were then confirmed by surgical biopsy. However, when applying conventional radiographic imaging to these same cases, they found that only 78% of these lesions could be detected.

Cone-beam volumetric 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. CBVT, although it can show great detail in many planes of vision, 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 CBVT for endodontic diagnosis. It has already proven invaluable in the detection of dental and nondental pathoses (Fig. 1-32).

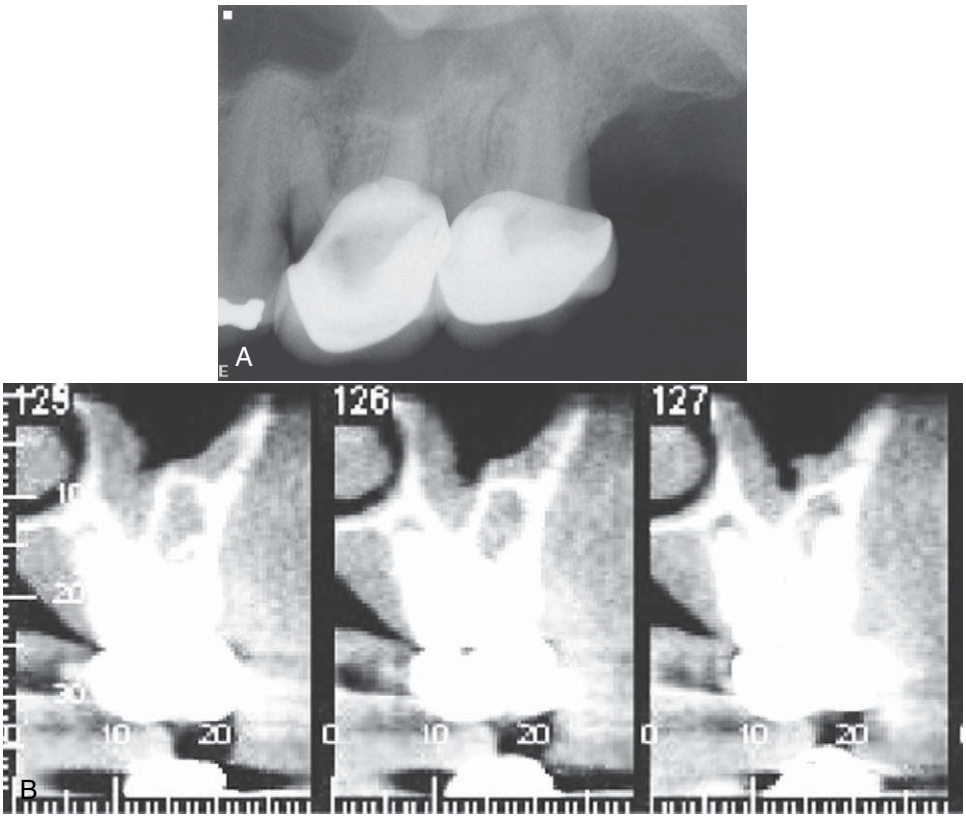


FIG. 1-31 A, This standard two-dimensional radiographic image reveals recurrent caries under the mesial margin of the maxillary first molar. However, the patient localized pain to mastication on the maxillary second molar. **B,** Cone-beam volumetric tomography revealed an apical abscess associated with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

Root Fractures and Cracks

Because of the wide variety of different types of cracks in teeth, there may be a myriad of symptoms and presentations, often making the diagnosis of a crack difficult. The extensiveness of a crack may directly alter the prognosis assessment for a given tooth. Therefore, any possible crack should be examined before dental treatment. These 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 even split the entire tooth into two separate pieces. 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. Because of the high prevalence of fractures and cracks in teeth and how they can directly alter the prognosis for a tooth, an extensive review is presented.

Crack Types

There have been many attempts in the literature to classify cracks in teeth, trying to differentiate the extent to which the crack has progressed into the tooth structure. By defining the type of crack present, an assessment of the prognosis may be determined and treatment alternatives may be planned as fully described in Chapter 17. Unfortunately, it is often impossible to determine how extensive a crack is until the tooth is extracted. Therefore, the determination of a crack is often more of a prediction, rather than a definitive diagnosis.

Cracks in teeth can be divided into three basic categories:

- Craze lines
- Fractures (also referred to as *cracks*)
- Split 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. 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 in 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, but left alone, especially with provocations such as occlusal prematurities, the fracture may progress to a split root. A fractured tooth may be treated by a simple restoration, endodontics, or even extraction, depending on the extent and orientation of the fracture, the degree of symptoms, and whether or not the symptoms can be eliminated. This makes the clinical management of fractured teeth difficult and sometimes unpredictable.

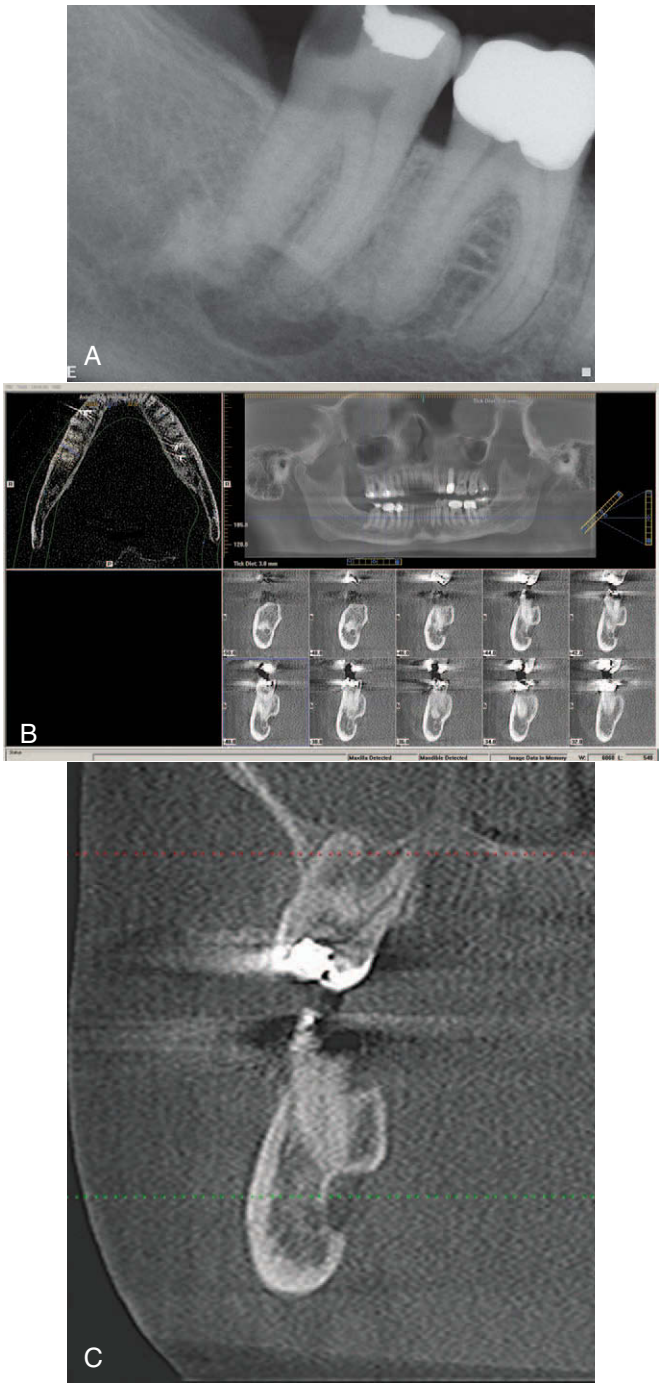


FIG. 1-32 A, Periapical radiograph revealed apical bone loss on this mandibular second molar, suspicious of apical pathosis. **B,** Cone-beam volumetric tomography revealed salivary indentation of the mandible in the area apical and lingual to the mandibular second molar, consistent with a Stafne defect. **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*[14] should be avoided.[1] 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 patients must be properly informed as to any potential decrease in the 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.[5,20,51,68,85,88,97]

Split 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 or involves the pulp, the tooth may not be restorable and endodontic treatment may not result in a favorable prognosis.

Proper prognosis assessment before any dental treatment is imperative, 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 the decision to continue with treatment and should avoid treating cases of definitive split roots.

Vertical Root Fractures

Certainly, of the previous three categories, fractured teeth are the most variable in presentation, in that the extent of the fracture is often difficult to determine. 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. 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 they may develop after endodontic treatment has been completed. Because diagnosing these vertical root fractures may be difficult, they often go unrecognized. Typically, these cracks lead to a split root, leaving the tooth with a poor prognosis. Therefore, diagnosing the existence and extent of a vertical root fracture is imperative before any restorative or endodontic treatment because these cracks can dramatically affect the overall success of treatment. Because the presence of vertical root fractures plays such an important role in the prognosis assessment of teeth, a detailed analysis of vertical root fractures is presented here.

ETIOLOGY OF A VERTICAL ROOT FRACTURE

Vertical root fractures may arise from a physical traumatic injury, occlusal prematurities,[14] repetitive parafunctional habits of heavy stressful chewing,[104] or resorption-induced pathologic root fractures.[8] However, the most common cause of vertical root fractures may be iatrogenic dental treatment.[8,14,74] Dental procedures such as the placement of posts and pins[31,60] or the tapping into place of a tightly fitting post or intracoronal restoration may induce a vertical root fracture (Fig. 1-33). The most common dental procedure contributing to vertical root fractures is endodontic treatment.[8] Before treatment, teeth that are about to undergo endodontic treatment may be predisposed

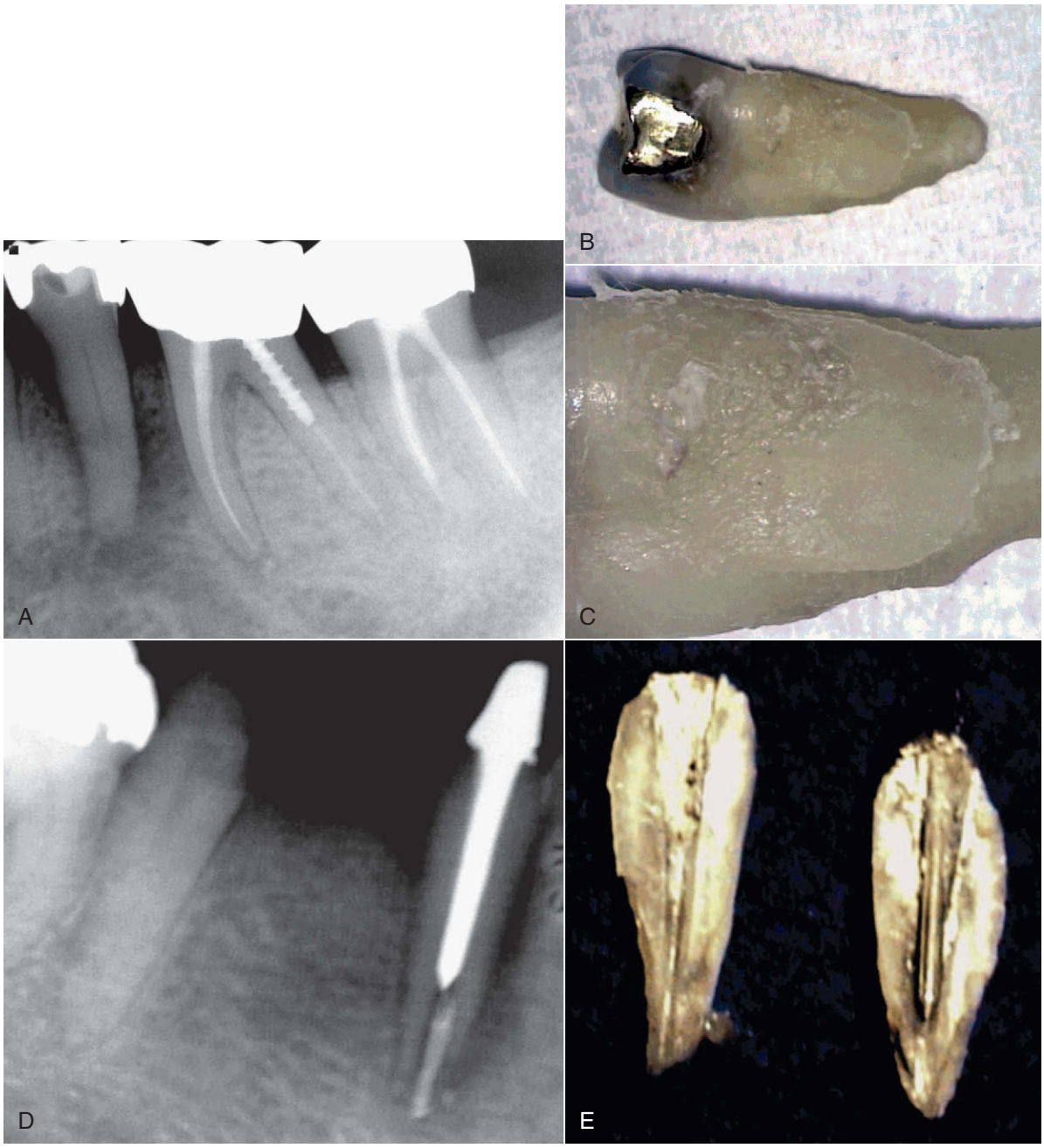


FIG. 1-33 Poorly fitting intracoronal restorations can place stresses within the tooth that can cause a vertical root fracture. **A,** This radiograph of a mandibular second premolar (with a gold inlay) reveals extensive periapical and periradicular bone loss, especially on the distal aspect. **B,** 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.

to vertical root fractures because quite often the tooth is already compromised from extensive coronal restorations, caries, resorption, or trauma. Teeth were once thought to be more susceptible to fracturing after endodontic treatment because of a decrease in hydration.[41] However, later studies found no difference in the dentin properties after endodontic procedures.[43,87] Although the physical characteristics of the dentin may not be compromised by endodontic treatment, the overenlarging of an endodontic access and excessive canal shaping will result in an increased amount of dentin removal. Consequently, the root may become weaker and may be more predisposed to vertical root fractures. Intracanal forces from excessive compaction pressure during obturation may also contribute to an increased incidence of vertical root fractures.[8,60]

DETERMINATION OF A VERTICAL ROOT FRACTURE

The assessment of a potential vertical root fracture may be based more on subjective than on objective findings, making the diagnosis more like an art than a science, but early detection is crucial. Many subtle findings can lead the clinician to suspect a vertical root fracture.[18] Medical and dental histories are important. Clinical, periodontal, and radiographic examinations may be at best only suggestive of a vertical root fracture. The tooth is typically painful, with symptoms ranging from mild to severe in intensity. When the tooth is painful, patients will typically complain of symptoms when they occlude or release on the tooth in a specific direction. Also noteworthy is the anatomic location of the tooth in question. Mandibular second molars have a higher incidence of vertical root fractures, followed by maxillary first molars and maxillary premolars.[14] Prominent cusps, balancing interferences, and occlusal prematurities may all be factors in this predisposition to fracturing.

MEDICAL HISTORY

The patient's medical history may seem like an unlikely place to discover any suggestion of a vertical root fracture. But a history of facial trauma may add information to help in the creation of a differential diagnosis. For example, patients with seizure disorders may be prone to dental trauma, either from severe seizure-induced clenching, or from physical injuries sustained secondary to a grand mal seizure (Fig. 1-34). In addition, a patient who has had a stroke, heart attack, or any other ailment that might have resulted in lack of consciousness could have traumatized a tooth. This could result in a vertical root fracture if the trauma is directed accordingly.



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.

DENTAL HISTORY

Various comments made by a patient when a dental history is taken may direct the clinician to suspect a vertical root fracture. A patient might report ice chewing or other parafunctional habits. A patient might also describe a symptom by stating "the tooth only hurts when I bite a certain way." Comments like "it only started hurting after I accidentally bit down on a cherry pit" might also be suggestive of a vertical root fracture. Other comments concerning recent dental treatment may also be significant. Localized pain in tooth after the placement of a cast post or cast intracoronal restoration could also implicate a vertical root fracture. The repeated falling out of a coronal restoration could be due to a fracture between the axial walls of the preparations; as the fractured segments flex or move apart, the restoration between these segments may lose its resistance form, become loose, and dislodge. Similarly, a retrograde restoration that has become dislodged could be secondary to a vertical root fracture apically.[26,74] As the apical fracture opens up, the retrograde restoration may come out (Fig. 1-35). An endodontic procedure that was performed well but does not result in healing may also be suggestive of a vertical root fracture, especially if the tooth does not heal after retreatment or apical surgery.

CLINICAL EVALUATION OF A VERTICAL ROOT FRACTURE

Crack Probing A vertical root fracture can occasionally be observed on clinical examination. The use of a dental operating microscope may be invaluable in detecting cracks and fractures in the clinical crown and root surfaces. Probing the crack may elicit pain or even reveal moving segments on either side of the crack line, indicating a split tooth. To determine the extent of the fracture, it is important to apply some pressure to the cusps that are adjacent to any visible crack (Fig. 1-36). On occasion, pressure to the cusp or actual probing of the crack will reveal a split tooth that would not have otherwise been found.

Selective Sensitivity Vertical root fractures may manifest as a selective sensitivity when the tooth is percussed in a particular direction. The back end of a dental mirror handle is a useful instrument. There are also specially designed bite sticks that when applied to a certain part of the tooth may elicit pain that would not be present when biting on another location of the tooth's occlusal surface (see Fig. 1-20).

Restorative Treatment Highly suspect of a vertical root fracture is a tooth with minimal or no restoration or caries that is nonvital. Except for luxation injuries, systemic disease (e.g., intraoral herpes zoster),[36] or surgical procedures that may accidentally devitalize a tooth (e.g., sinus surgery, orthognathic surgery, accidental extraction of a tooth with subsequent replantation), a vertical root fracture is one of the few reasons for such a tooth to become nonvital (Fig. 1-37). A tooth with recurrent endodontic pathosis and a post that is an abutment to a cantilever bridge should also be suspected of having a

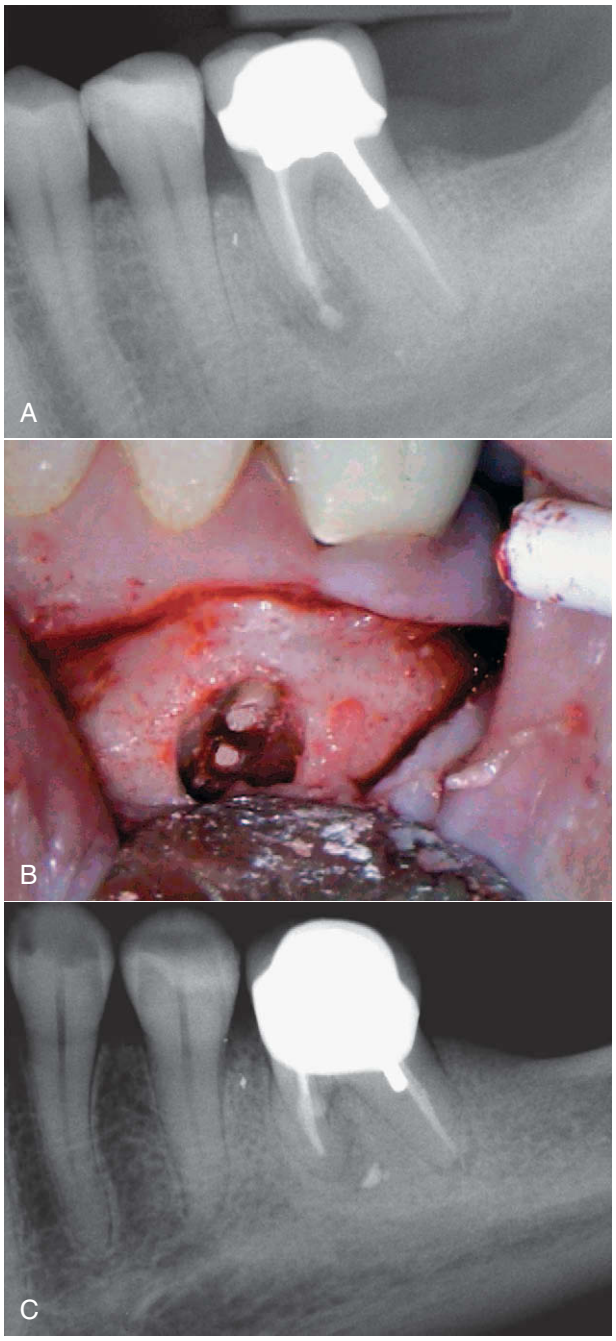


FIG. 1-35 Pulp infections that are refractory to endodontic treatment may often result from vertical root fractures. **A,** This radiograph shows the placement of retrograde restorations in the apex of the mesial roots of this lower left first molar. **B,** This photograph shows that the restorations are directly in the root's apex. **C,** After a little more than 1 year, the retrograde restorations are observed to have been dislodged, suggesting the possibility of a vertical root fracture.

vertical root fracture (Fig. 1-38). The torque created during mastication on the cantilevered pontic and the subsequent flexing of the bridge can cause stresses in the root that lead to vertical root fracture. Persistent symptoms from a tooth with a conservative cast intracoronal restoration should also make the clinician suspicious of a vertical root fracture. Generally speaking, there must be a reason for a tooth becoming nonvital and/or causing pain. The clinician should evaluate the restorative treatment, or lack thereof, and try to make an assessment as to whether or not there is sufficient cause for necrosis or symptoms; differential diagnosis should always include *vertical root fracture* under these circumstances.

Multiple Sinus Tracts Also indicative of a vertical root fracture is the presence of multiple sinus tracts adjacent to the tooth in question. Because the fracture may be present on at least two surfaces of the tooth, the infected area may drain to multiple sites, creating multiple sinus tracts.[26]

Dental Operating Microscope Used in medicine for years, the dental operating microscope (DOM) has become an invaluable tool when doing endodontic treatment. With magnification capabilities exceeding ×25, and with superb illumination, the clinician is now capable of observing intracoronal and extracoronal details with great precision. Sometimes a fracture may be observed extracoronally before endodontic treatment; its depth can be visualized intracoronally with the DOM after an endodontic access has been created (Fig. 1-39).

Surgical Exposure Often the only definitive way to determine whether there is a vertical root fracture or split tooth is by direct surgical exposure. For optimal visualization of a potential fracture, a full-thickness mucoperiosteal flap incision should be created at the level of the sulcus and reflected apically. Typically, only a small flap is necessary, because once the flap is reflected, if there is a vertical root fracture, it can usually be seen after the overlying granulation tissue is removed. Many times there is an associated boney dehiscence directly over the fracture. The use of a DOM is especially helpful in maximizing the illumination and visualization of these defects (Fig. 1-40).

Transillumination and Dyes Often it may be necessary to remove the restoration in the tooth to better visualize the 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 a crack. Transillumination may be more helpful (Fig. 1-41). Directing a high-intensity light directly on the exterior surface of the tooth at the cementum–enamel junction (CEJ) may indicate 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 be gray by comparison.[74] Although the presence of a fracture may be evident using dyes and transillumination, the depth of the fracture cannot always be determined.

RADIOGRAPHIC EVALUATION OF A VERTICAL ROOT FRACTURE

On occasion, a vertical root fracture or a split tooth may be an obvious diagnosis based on radiographic findings (Fig. 1-42). However, most of the time the fracture is in a plane that is not perceptible from a periapical radiograph. In one study[82] using extracted teeth, investigators determined that a fracture

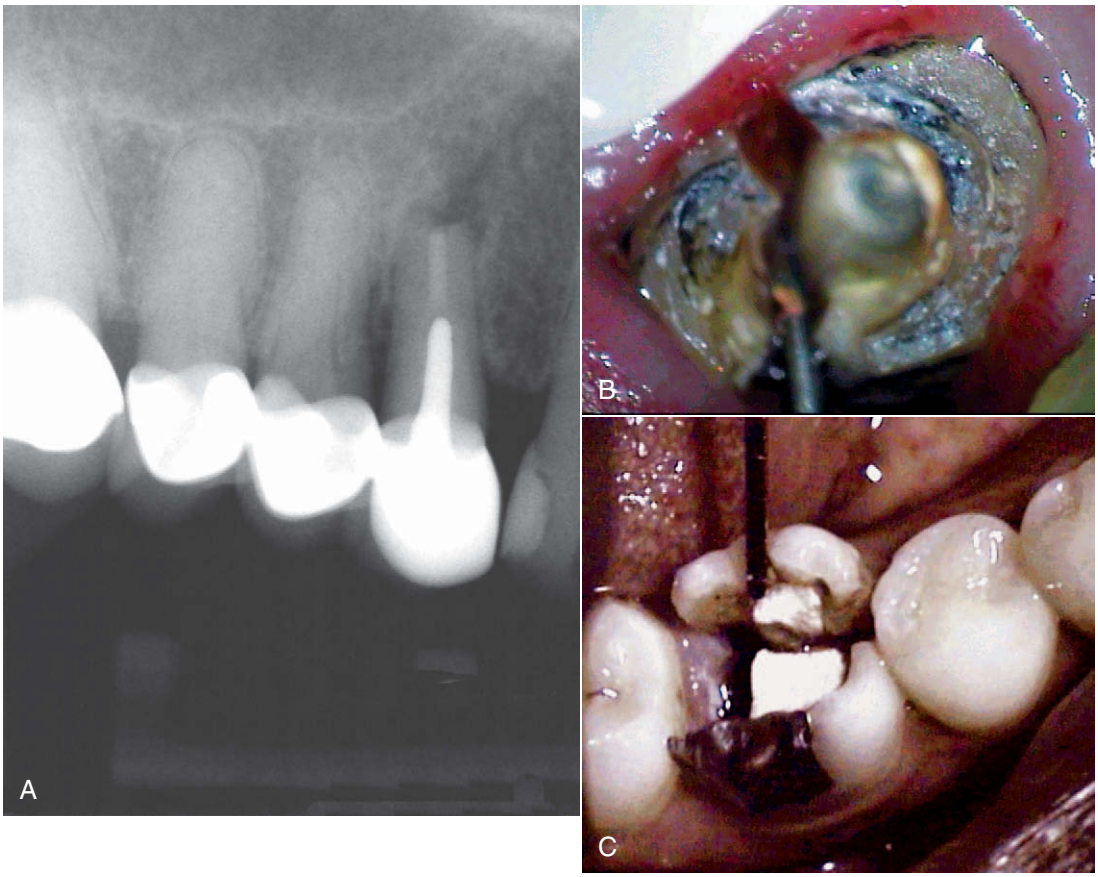


FIG. 1-36 Often it is difficult to diagnose a root fracture unless the coronal restoration is removed. **A,** This radiograph shows no evidence of a fracture. **B,** However, once the crown and post are removed, the fracture is easily visualized. **C,** Sometimes it is helpful to physically push on the axial walls of the clinical crown in order to observe the existence of a fracture.

becomes visible when the x-ray beam is directed within 4 degrees of the fracture plane. Outside of this horizontal angulation, a fracture may not be discernible. They also found that when a vertical root fracture is present, it is observed in a radiograph only 35.7% of the time (Fig. 1-43).

Another interesting way to detect vertical root fractures or split teeth is from a CAT scan or by cone-beam volumetric tomography. This technique has been shown to be superior to dental radiography in the detection of vertical root fractures.[105] However, this type of imaging is typically not available in dental offices, other than certain specialty practices.

To assist the clinician, other radiographic signs may be helpful in the detection of vertical root fractures.

Cement Trail When the vertical root fracture or split root extends from the mesial surface to the distal surface of the tooth, often this crack can be "interpreted" after endodontic treatment has been performed. Sometimes a "cement trail" can be seen up or across the root. This is the cement extruded through and out of the fracture site, after which it becomes visible radiographically. It can be confused with the obturation passing through accessory canals, but the appearance is more diffuse in cases of vertical root fractures, with no observation of a symmetrical lateral canal passing from a main canal (Fig. 1-44).

Halo-like Bone Loss Typically, when a nonvital tooth causes radiographic changes, the bone loss occurs apically. However, often when there is a vertical root fracture or split root, the bone loss has a tendency to give a "halo-like" appearance, traversing circumferentially around the root. The radiolucent area may also travel almost completely up one side of the root, with this pattern of bone loss often termed a "J-type" lesion[98] (Fig. 1-45).

Isolated Bone Loss The clinician should be aware of unusual radiographic changes revealing extensive bone loss that is isolated to just one tooth in the absence of advanced periodontal disease (Fig. 1-46). This could be suggestive of a vertical root fracture or split tooth.

When attempting to make a diagnosis of a vertical root fracture or split tooth, the clinician should also look for the following:

- A widened canal space that is inconsistent with the canal spaces of the adjacent roots (Fig. 1-47)
- A radiolucent space presenting between the long axis of the obturation material (or post) and the canal wall (Fig. 1-48)
- An associated bone loss mesial and distal to the root. When a vertical root fracture or split root extends from the mesial surface to the distal surface of the tooth, there is often an atypical widening of the entire periodontal ligament space

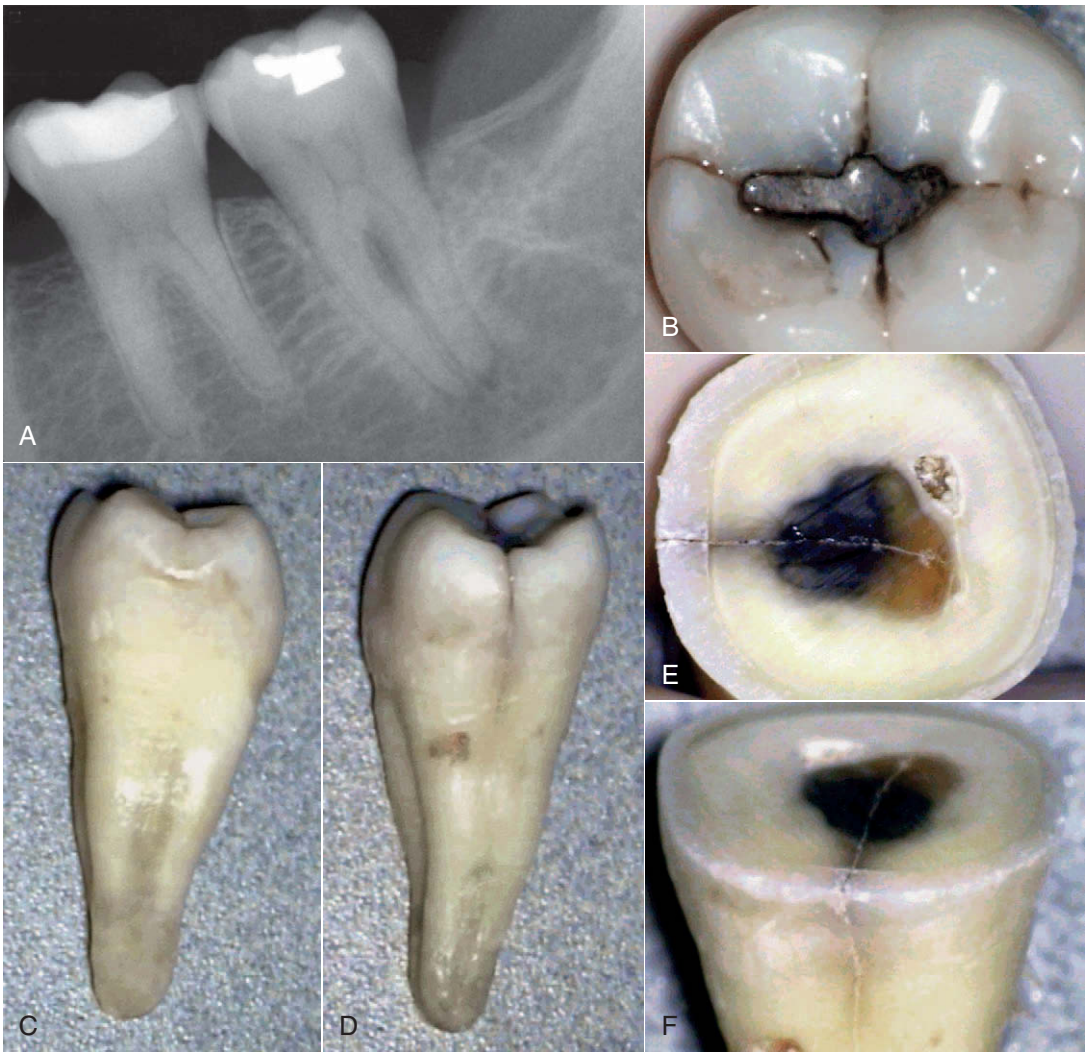


FIG. 1-37 A tooth with minimal or no restoration or caries is unlikely to become nonvital. **A,** This radiograph of a mandibular second molar shows a restoration that is distant from the pulp chamber, yet the tooth is nonvital and symptomatic. **B,** On occlusal examination, a slight crack is observed 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** and **F,** When the crown is sectioned, the crack can be observed to extend well into the pulp chamber.

When these features are visualized on the radiograph, a vertical root fracture or split tooth should be strongly suspected.

PERIODONTAL EVALUATION OF A VERTICAL ROOT FRACTURE

Typically, a vertical root fracture or split tooth has associated bone loss contiguous with the fracture line. Left undetected, this creates a dehiscence in the bone and a V-type pattern of bone loss extending apically. The periodontal pocket associated with this bone loss is generally isolated, narrow, and deep (Fig. 1-49); a similar defect may occur 180 degrees opposite to the defect (i.e., the other side of the fracture on the other side of the root). When the periodontal probe is inserted into this type of periodontal pocket, it is *tight* within the pocket, and the periodontal probe's movement from side to side is restricted. This is a classic periodontal presentation and is practically pathognomonic for a vertical root fracture or split tooth.

PROGNOSIS OF A VERTICAL ROOT FRACTURE

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 must convey these suspicions to the patient. The prognosis assessment should be described as follows.

Good Prognosis If a coronal crack is observed with nonmovable segments and the patient does not have symptoms, notwithstanding any other adverse parameters the tooth has a good prognosis.

Fair Prognosis If the tooth is sensitive on probing the occlusal crack, with the opposing segments nonmovable, then the prognosis is more guarded. The patient should understand that the pending endodontic treatment may not resolve the symptoms

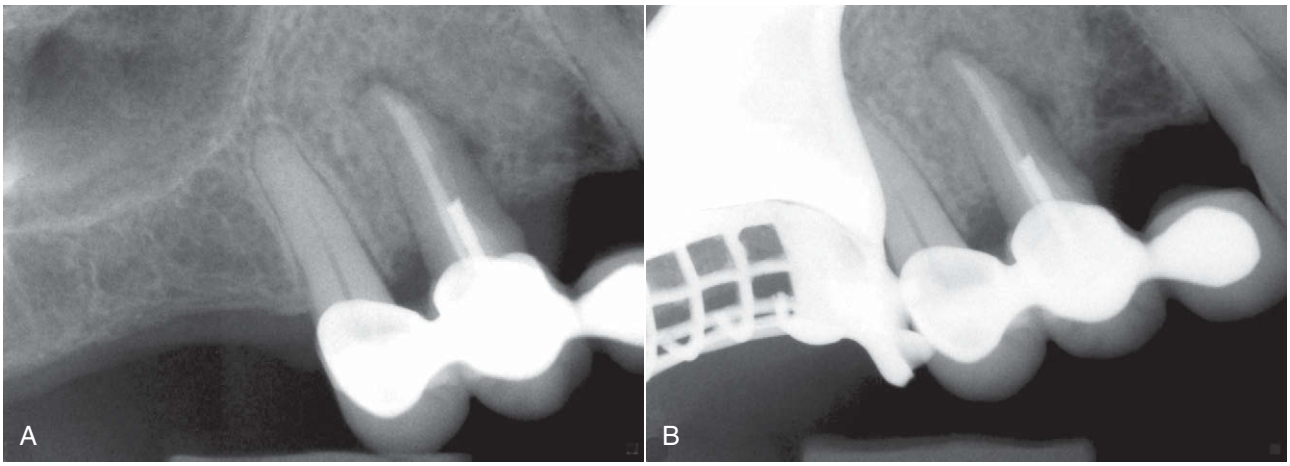


FIG. 1-38 A, Cantilever bridges can create oblique forces on an abutment tooth, especially if that tooth has endodontic treatment with a post. **B,** A distal-extension removable partial denture can compound the stresses on the abutment tooth, setting up a situation that can predispose that tooth to a vertical root fracture.

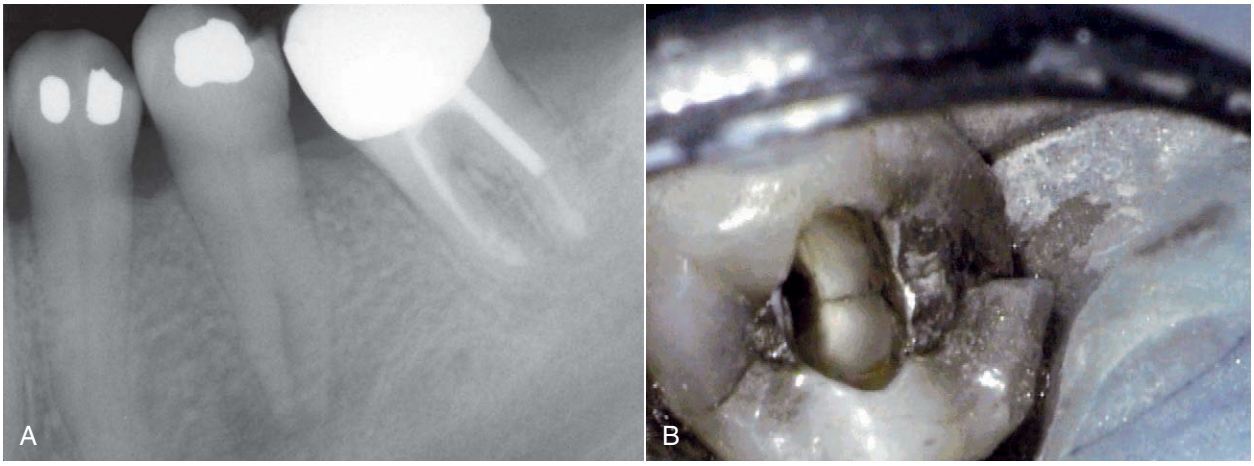


FIG. 1-39 Often a radiograph does not give a good indication of a vertical root fracture. **A,** This mandibular second premolar is nonvital, with a restoration that is not significantly close to the pulp chamber. **B,** The interior walls of the pulp chamber are examined under high magnification (×12) to reveal a vertical root fracture.

and that the prognosis is only fair. If endodontic treatment is performed, the interior walls of the access should be carefully examined, preferably with the use of a DOM along with transillumination, to determine whether the crack has traversed into the canal space (see Fig. 1-39). If this is observed, the patient should again be advised of the potential for a more compromised prognosis. The tooth should be restored with a bonded intracoronal core in the access, the use of a post should be avoided, and a full-coverage restoration with cuspal reinforcement should be placed.[86,100]

Poor Prognosis If there are movable segments on either side of the occlusal crack, then the prognosis is poor. Often the movement of the segments is difficult to visualize, so magnification is essential. If the pulp of this tooth is diagnosed as being necrotic, with a minimal caries and restorative history, and has a deep, narrow, isolated periodontal pocket with normal periodontium otherwise, then a vertical root fracture or a split root should be highly suspected, and extraction should be considered.

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 Chapter 3). This may create a diagnostic challenge, in that the patient may insist that the pain is from a certain tooth or even from an earache 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 location of pulpal pain.[30]

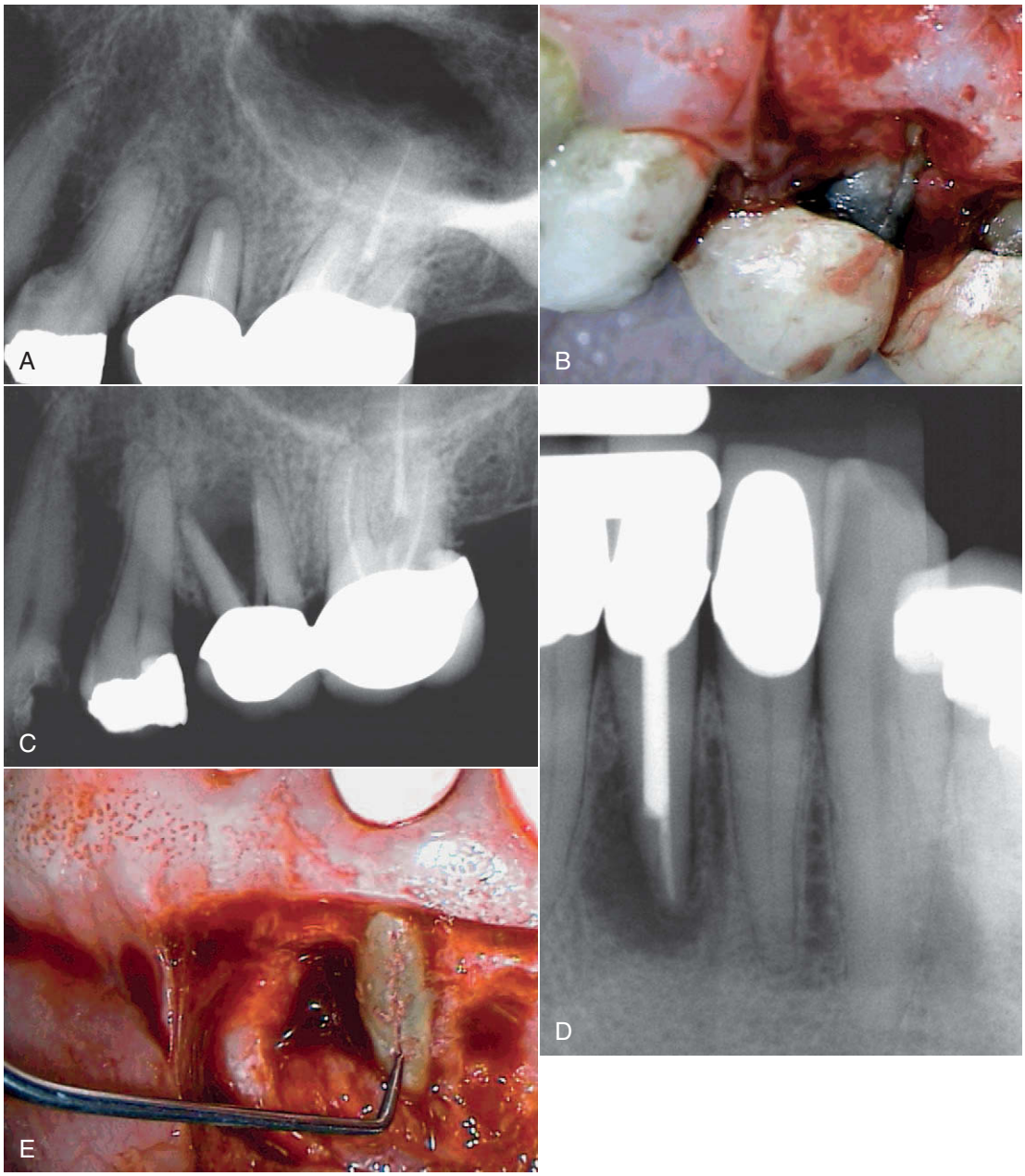


FIG. 1-40 When a vertical root fracture is suspected, sometimes the reflection of a surgical flap over the root may allow better visualization. **A,** The radiograph here shows no indication of a root fracture. **B,** However, after surgical exposure, the fracture can be seen. **C,** Unfortunately, this tooth was never extracted and is observed with a prominent split root 1 year later. **D,** Similarly, the radiograph of this mandibular anterior tooth shows no indication of a root fracture. **E,** However, when the root is surgically exposed, the fracture is easily seen.

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.[9,89] 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.[101] The authors 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.

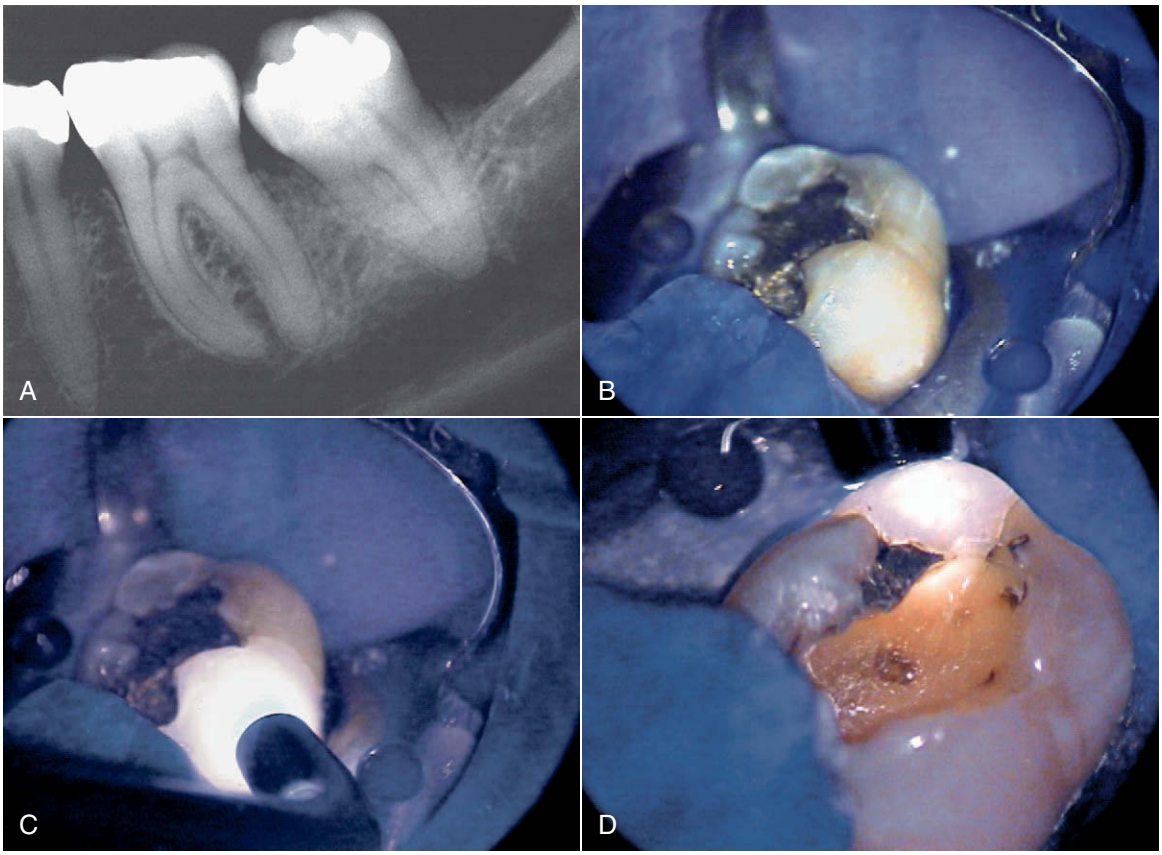


FIG. 1-41 Sometimes there is no clear indication of why a tooth is symptomatic. This radiograph shows a mandibular second molar with a moderately deep restoration **(A);** the pulp tests nonvital. Without any transillumination a fracture cannot be detected **(B).** However, by placing a high-intensity light source on the tooth surface, a root fracture can be observed on the buccal surface **(C)** and the distal-lingual surface **(D).**

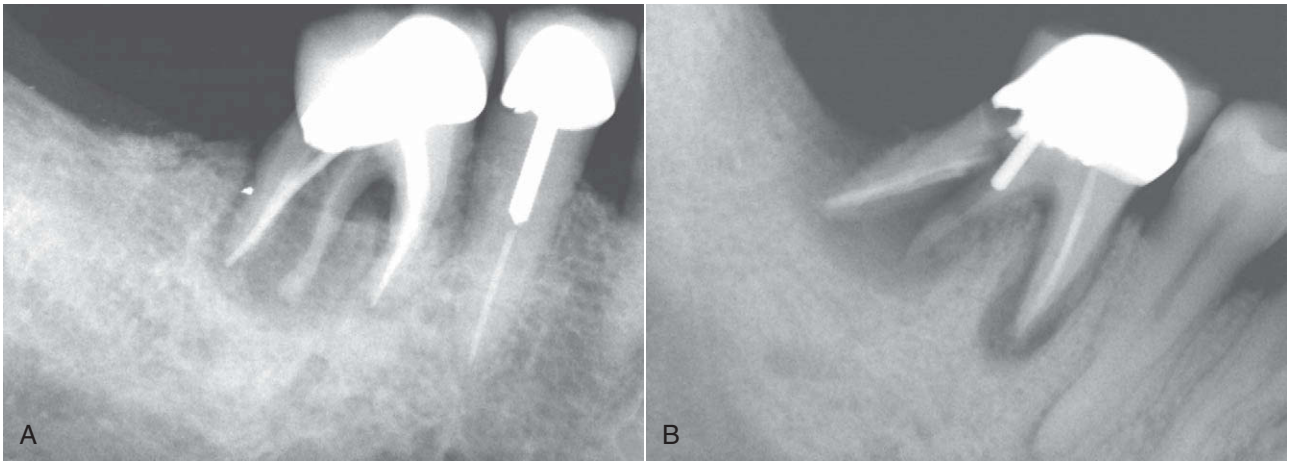


FIG. 1-42 Vertical root fractures that develop into split roots are sometimes easy to diagnose from a radiograph.

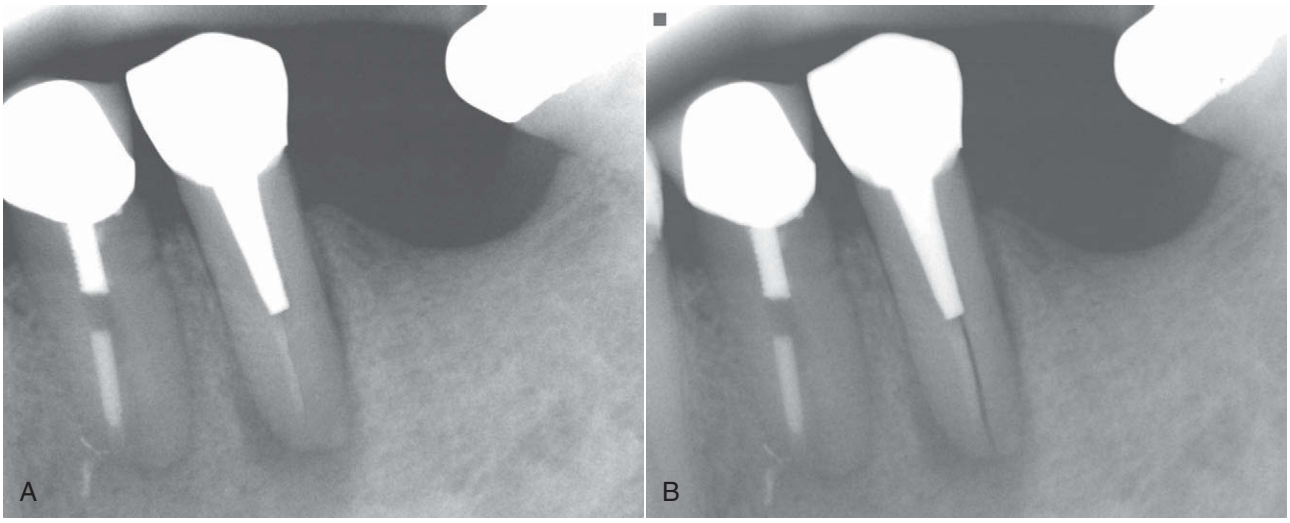


FIG. 1-43 Although split roots are not always radiographically visible, the precise angulation of the radiation source is necessary in order to observe them. **A,** A root fracture is not visible in the radiograph of this mandibular first premolar. **B,** However, if exposed with only a small change in the horizontal angulation, the fracture becomes visible.



FIG. 1-44 After obturation, obturating material may extrude through accessory canals. However, sometimes an extensive "cement trail" of material up the side of the root extrudes through a fracture space, with no demarcation of accessory canals. This is suggestive of a vertical root fracture.



FIG. 1-45 Often the radiographic presentation of a vertical root fracture is the pattern of bone loss occurring in a "J-shaped" radiolucency, with the bone loss originating apically and progressing coronally up one side of the root.



FIG. 1-46 Extensive periodontal bone loss around an isolated tooth, with the adjacent teeth within normal limits, is suggestive of a vertical root fracture.



FIG. 1-47 When there is a disproportionate widening of a canal space compared with the canals in the same tooth or adjacent teeth, as seen in this mandibular second molar, a vertical root fracture should be suspected.

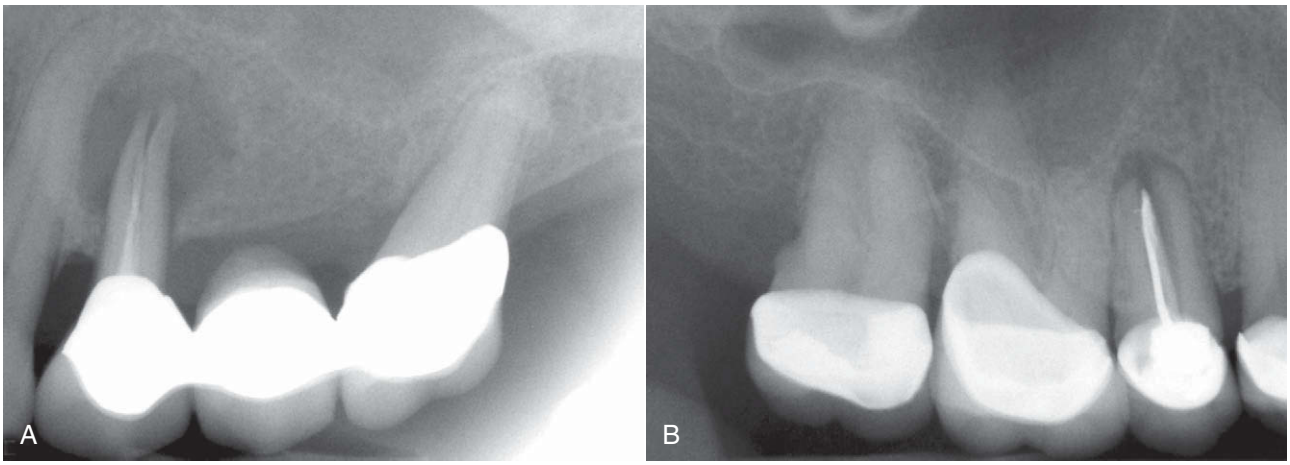


FIG. 1-48 A space between the obturation and the canal wall is highly suggestive of a split tooth.

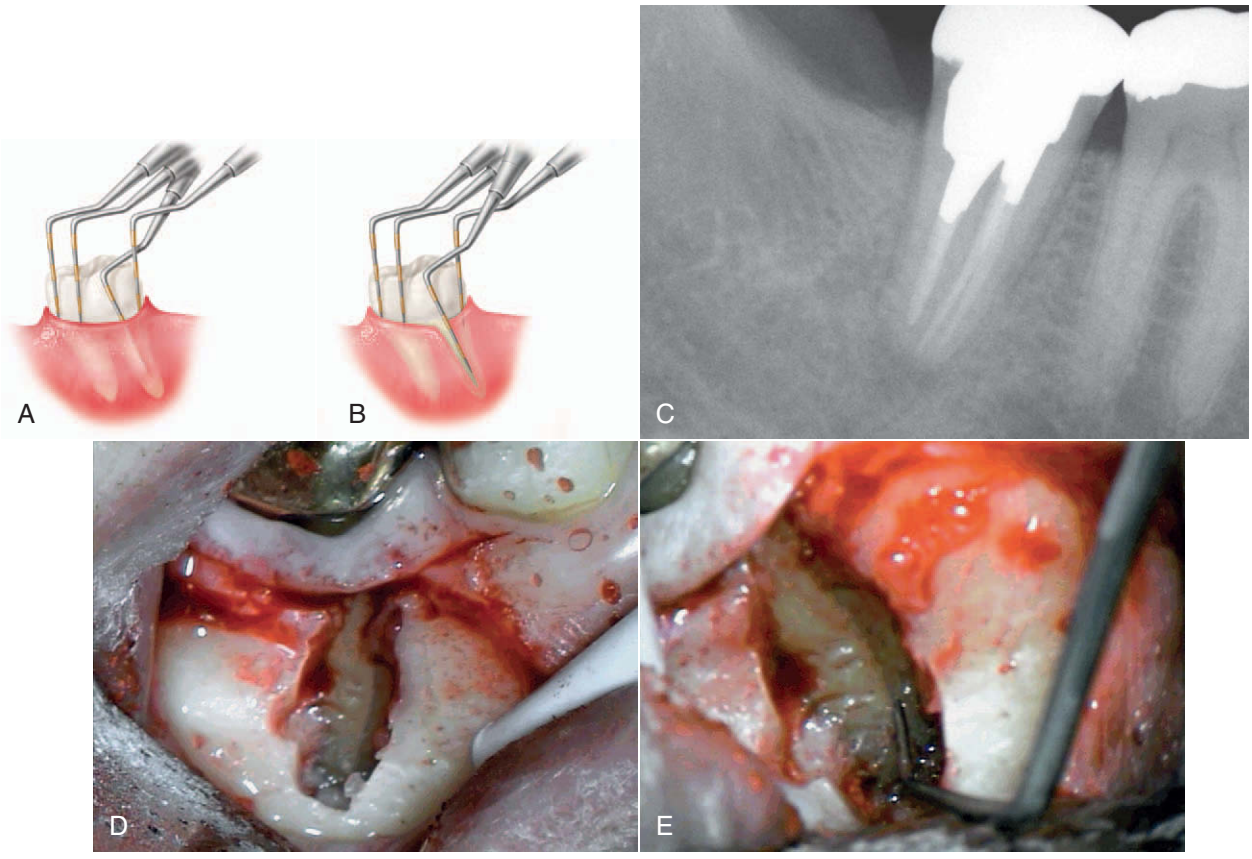


FIG. 1-49 When a narrow, isolated periodontal defect is present, with the adjacent periodontal structures within normal limits, there is typically an associated boney dehiscence with an underlying vertical root fracture beneath **(A-B).** This mandibular second molar showed this type of defect, as observed on surgical exploration **(C-E).**

CLINICAL CLASSIFICATION OF PULPAL AND PERIAPICAL DISEASES

Many attempts have been made over the years to develop classifications of pulpal and periapical disease. However, many studies have shown that there is not a great correlation between clinical signs and symptoms and the histopathology of a given clinical condition.[91,92] The terminology that has been historically used are biologic terms that may not accurately depict the actual histologic condition of the pulp and supporting periodontal tissues. Because removal of the questionable pulpal tissue for histologic examination is not practical, *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.

On October 3, 2008, the American Association of Endodontists convened a *Consensus Conference on Diagnostic Terminology* in an attempt to standardize the terminology used for classification of pulpal and periapical disease conditions. Terms such as acute and chronic, symptomatic and asymptomatic, reversible and irreversible, nonvital and necrotic, and apical, periapical, and periradicular are all used to describe similar clinical presentations. The conference was organized to establish uniformity in diagnostic terminology for textbook authors, practicing clinicians, and dental school faculty teaching endodontic diagnostic classifications. Although standardized diagnostic classifications remain a topic of active research, it is hoped that this first step in reaching a consensus will result in uniform diagnostic terminology in the near future. Until such a time that a consensus is reached, histologic terms such as "pulpal inflammation" or "infected pulp" should be avoided. These terms only describe the histopathology of these tissues subjectively because it is not possible to objectively determine their true status without the removal and direct microscopic examination of the affected tissues. The terminology and classification that follows are based on those suggested by the American Board of Endodontics (ABE) in 2007.

Pulpal Disease

Normal Pulp

Teeth with normal pulp do not exhibit any spontaneous symptoms. The pulp will respond to pulp tests, and the symptoms produced from such tests are mild, do not cause the patient distress, and result in a transient sensation that disappears 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.

Reversible Pulpitis

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.[76]

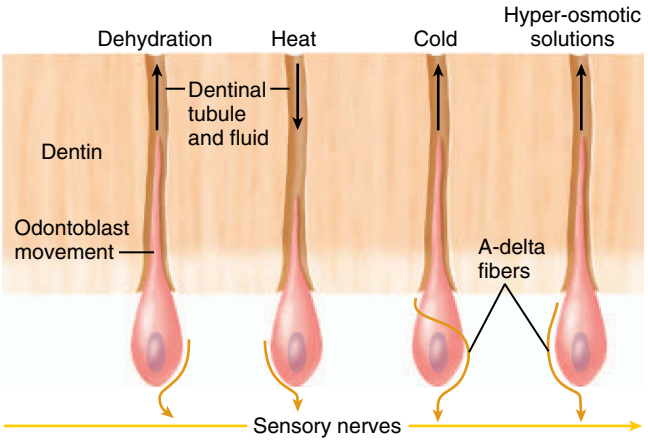


FIG. 1-50 Dentinal tubules are filled with fluid that, when stimulated, will cause sensation. Temperature changes, air, and osmotic changes can provoke the odontoblastic process to induce the stimulation of underlying A-delta fibers.

As is described in later chapters, the fluid movement within dentinal tubules stimulates the odontoblasts and associated fast-conducting A-delta nerve fibers in the pulp, which in turn produce sharp, quickly reversible dental pain (Fig. 1-50). 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, not to mention 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.[13]

Irreversible Pulpitis

As the disease state of the pulp progresses into the classification of *irreversible pulpitis,* treatment to remove the diseased tissue will be necessary. The ABE has suggested dividing this classification into the subcategories of symptomatic and asymptomatic irreversible pulpitis.

SYMPTOMATIC IRREVERSIBLE PULPITIS

Teeth that are classified as having *symptomatic irreversible pulpitis* exhibit intermittent or spontaneous pain. Rapid exposure of teeth in this category 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 evident on the radiograph, and there may be some suggestion of pulpal irritation by virtue of extensive pulp chamber and 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, may be seen radiographically or clinically, or may be suggested from a complete dental history. Typically, when symptomatic irreversible pulpitis remains untreated, the pulp will eventually become necrotic.

ASYMPTOMATIC IRREVERSIBLE PULPITIS

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 does not take place and result in severe pain and patient distress.

Pulp Necrosis

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 that symptoms develop as a result of extension of the disease process into the periradicular tissues. With pulp necrosis, the tooth will 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. As previously discussed, a traumatic injury to a tooth may prevent the lack of a response to pulp tests and mimic that of pulpal necrosis; therefore, a good dental history is imperative. 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 and where pulp testing over one root may give no response and over another root may give a vital response. The tooth may also exhibit symptoms of symptomatic irreversible pulpitis.

After the pulp becomes necrotic, bacterial growth can be sustained within the canal. When this infection (or the bacterial toxins from this infection) 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 when the pain is referred or not localized.

Previously Treated

The ABE has suggested a clinical diagnostic category for those cases that present where the tooth has already had nonsurgical root canal therapy performed and the root canal system has been filled with some type of root canal obturating material. 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 of these situations there will no longer be any vital or necrotic pulp tissue present to respond to pulp testing procedures.

Previously Initiated Therapy

The ABE has also suggested another clinical diagnostic category for those cases that present with partial endodontic therapy has been performed. Teeth in this category would include cases where only a pulpotomy or pulpectomy had been performed before presenting for root canal therapy. In most instances the pulpotomy or pulpectomy 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 the root canal therapy it would not be possible to make an accurate pulpal diagnosis since 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.

Symptomatic Apical Periodontitis

A tooth with *symptomatic apical periodontitis* will have an acutely painful response to biting pressure or percussion. This tooth may or may not respond to pulp vitality tests, and the radiograph or image of this tooth will generally exhibit at least a widened periodontal ligament space and may or may not have an apical radiolucency associated with one or all of the roots.

Asymptomatic Apical Periodontitis

A tooth with *asymptomatic apical periodontitis* generally presents with no clinical symptoms. This tooth does not respond to pulp vitality tests, and the radiograph or image will exhibit an apical radiolucency. This tooth is generally not sensitive to biting pressure but may "feel different" to the patient on percussion.

Acute Apical Abscess

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 will exhibit tenderness to palpation.

Chronic Apical Abscess

A tooth with a *chronic apical abscess* will not generally present with clinical symptoms. This tooth will not respond to pulp vitality tests and the radiograph or image will exhibit an apical radiolucency. The tooth is generally 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.

Pain of Nonodontogenic Origin

If after all of the testing procedures are completed and the decision is reached that the pain is not of odontogenic origin, then the patient should be referred to an orofacial pain clinic for further testing. This diagnostic classification is covered in detail in Chapter 3.

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 DOMs, 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 recent 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 appropriate form of treatment?

Testing, questioning, and reasoning are combined in order 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 treatment.

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CHAPTER 2

Management of Endodontic Emergencies

JAMES WOLCOTT, LOUIS E. ROSSMAN, and GUNNAR HASSELGREN

CHAPTER OUTLINE

EMERGENCY CLASSIFICATIONS
EMERGENCY ENDODONTIC MANAGEMENT
Vital Teeth
Pulpal Necrosis With Acute Apical Abscess
ANTIBIOTICS
ANALGESICS
LABORATORY DIAGNOSTIC ADJUNCTS
FLARE-UPS
CRACKED AND FRACTURED TEETH

EMERGENCY CLASSIFICATIONS

The proper diagnosis and effective management of acute dental pain is possibly one of the most rewarding and satisfying aspects of providing dental care. An endodontic emergency is defined as pain and/or swelling, caused by various stages of inflammation or infection of the pulpal and/or periapical tissues. The cause of dental pain is generally from caries, deep or defective restorations, or trauma. Bender[9] 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.[32,47] It has also been estimated that about 12% of the U.S. population experienced a toothache in the preceding 6 months.[44]

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 taken among endodontists in 1977,[18,19] 1990,[25] and 2009[43] there are seven clinical presentations that are considered endodontic emergencies:

1. Irreversible pulpitis and normal periapex
2. Irreversible pulpitis and acute apical periodontitis
3. Necrotic pulp with acute apical periodontitis, with no swelling
4. Necrotic pulp, fluctuant swelling, with drainage
5. Necrotic pulp, fluctuant swelling, with no drainage
6. Necrotic pulp, diffuse facial swelling, with drainage through canals
7. Necrotic pulp, diffuse facial swelling, with no drainage

There are other endodontic emergencies that were not discussed in these surveys. These emergencies pertain to traumatic dental injuries, as discussed in Chapter 17, to teeth that have had previous endodontic treatment, as discussed in Chapters 16 and 25, 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 3.

In the decades between the previously cited surveys, there have been several changes as 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 psychologic and biologic entity, as discussed in Chapters 19 and online Chapter 26, the management of acute dental pain must take into consideration both the physical symptoms as well as the emotional state of the 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.[9,24,35,64]

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 11 and 27, 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.

Vital Teeth

As described in Chapter 1, vital teeth 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 and/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 the irritant 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 19.

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 with 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,[18,19] 187 board-certified endodontists were surveyed 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 have been any procedural changes in how these emergencies are managed.[25] The emergency treatment of a tooth with irreversible pulpitis with or without a normal periapex seemed to be managed fairly similarly. In the same survey conducted in 2009,[43] 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. 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.[20,54,56,61,63,69] However, this has not come without controversy, with some studies showing otherwise,[23,77] 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 difficult.[3] If root canal therapy is to be completed at a later date, medicating the canal with calcium hydroxide probably is indicated to reduce the chances of bacterial growth in the canal between appointments[13]; however, controlled studies have not substantiated this concept.[10,13,31] 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.[31] Sources of infection, such as caries and defective restorations, should be completely removed to prevent recontamination of the root canal system between appointments.[31] The concept of single- versus multiple-visit endodontics is described in greater detail in Chapter 4.

For emergency endodontic treatment of vital teeth that are not initially sensitive to percussion, occlusal reduction has not been shown to be beneficial.[15,25] 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.[25,53,62]

Antibiotics are not recommended for the emergency management of irreversible pulpitis[40] (see Chapter 19). Moreover, placebo-controlled clinical trials have demonstrated that antibiotics have no effect on pain levels in patients with irreversible pulpitis.[51]

On the basis of several surveys of board-certified endodontists as well as other recommendations in the literature,[13,25,30,43,71] the emergency management of a symptomatic irreversible pulpitis involves initiating root canal treatment, 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.[30,71]

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. 2-1). 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,[18,19] 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. Ten 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.[43]

Care should be taken not to allow necrotic debris to be pushed beyond the apex, because this has been shown to promote more posttreatment discomfort.[10,25,59,67] 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. These devices are now used by an increased number of clinicians.[17,43]

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 tissue exudate that causes pain. Its use has been historically advocated to provide pain relief in patients with severe and recalcitrant periradicular pain.[18,19] The technique involves an engine-driven perforator entering through the cortical bone and into the cancellous bone, often without the need for an incision.[12] This provides 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 acute periapical periodontitis[48] or symptomatic necrotic teeth with radiolucencies,[53] there are still some advocates who recommend trephination for managing acute and intractable periapical pain.[33] The clinician should understand that local anesthesia may be difficult.[36] Also, extreme care must be taken to guard against inadvertent and possibly irreversible injury to the tooth root or surrounding structures, such as the mental foramen, intraalveolar nerve, or maxillary sinus.

NECROSIS AND SINGLE-VISIT ENDODONTICS

Although single-visit endodontic treatment for teeth diagnosed with irreversible pulpitis is not contraindicated,[1,56,58,63,78] performing single-visit endodontics on necrotic and previously treated teeth is not without controversy. In cases of necrotic teeth, research[20] 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. Although some more recent studies[68,72] have questioned the long-term prognosis of such treatment, especially in cases of acute periodontitis, several studies,[21,42] including a CONSORT (Consolidated Standards of Reporting Trials) clinical trial,[57] have shown no difference in outcome between single-visit and two-visit treatments. The concept of single- versus multivisit endodontics is further discussed in Chapter 4.

Swelling

Tissue swelling may be associated with an acute periradicular abscess at the time of the initial emergency visit, or may occur as an interappointment 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, dissecting tissue spaces along fascial planes.[37]

Swelling may be controlled by establishing drainage through the root canal or by incising the fluctuant swelling. As discussed later, and in Chapter 15, antibiotics are also an integral part of the management of swelling. The principal modality for managing swelling secondary to endodontic infections is to achieve drainage and remove the source of the infection.[27,37] When the swelling is localized, the preferred avenue is drainage through the root canal. Complete canal debridement and disinfection[29,74] are paramount to 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.[45] In the presence of persistent swelling, gentle finger pressure to the mucosa overlying the swelling may help facilitate drainage. Once the canals have been cleaned and allowed to dry, the access should be closed.[13,25,30] In these cases, there has been a trend to use calcium hydroxide as the intracanal medicament.[43]

INCISION FOR DRAINAGE

Often it becomes necessary to establish drainage from a localized soft tissue swelling. This can be accomplished through the *incision for drainage* of the area.[52] Incision for drainage is indicated whether the cellulitis is indurated or fluctuant.[37] A pathway for drainage is needed to prevent further spread of infection. An incision for drainage allows decompression of the increased tissue pressure associated with edema and can provide significant pain relief for the patient. The incision

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.

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FIG. 2-1 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. 2-1, cont'd

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:

- Make the 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 very 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.[27,37]

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. 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.[31,58] Gaining access to the periapical tissues through the root canals may require removal of posts and obturation, as well as negotiating blocked or ledged canals. Failure to complete root canal debridement and achieve periapical drainage may result in continued painful symptoms. The ability, practicality, and feasibility to adequately re-treat 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 254.

Leaving Teeth Open

On rare occasions, canal drainage may continue from the periapical spaces (Fig. 2-2). In these cases, the clinician may opt to step away from the patient for some time to allow for the drainage to continue and hopefully resolve on the same treatment visit.[71]

Historically, in the presence of acutely painful necrotic teeth with no swelling to diffuse swelling, 19.4% to 71.2% of surveyed endodontists would leave the tooth open between visits.[18,19] However, the more current literature makes it clear that this form of treatment would impair uneventful resolution and create more complicated treatment.[6,8,79] For this reason, *leaving teeth open between appointments is not recommended.* 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.[66] However, leaving teeth open between visits to allow for drainage or to manage intractable pain is not without controversy. August in 1977[4] and again in 1982[5] suggested that the problem with leaving teeth open had more to do with how they are later closed. He found that total instrumentation before the closing of open teeth had a 96.7% success rate.

ANTIBIOTICS

The prescription of antibiotics should be adjunctive to appropriate clinical treatment (see Chapters 15 and 19 for details). Because of potential risk factors such as allergies, drug interactions, and systemic complications, antibiotics should be prescribed judicially. They are indicated when signs and symptoms suggest systemic involvement such as high fever, malaise, cellulitis, unexplained trismus, and persistent and progressive infections, and for patients who are immunologically compromised.[7,22,28,34,74] The objective is to aid in the elimination of infection from the tissue spaces. The use of antibiotics alone, without properly addressing the source of the endodontic infection, is not appropriate treatment.[27,37]

ANALGESICS

Because a more thorough description of pain medications can be found in Chapter 19, 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 antiinflammatory drugs (NSAIDs).[43] However, no pain medication can replace the efficacy of thoroughly cleaning the root canal to rid the tooth of the source of infection.[26]

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[14]; its analgesic and antipyretic effects are equal to those of acetaminophen, and its antiinflammatory effect is more potent.[16] However, aspirin's side effects include epigastric 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. The maximal dose of 4 g in a 24-hour period should not be exceeded.

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.[14,39] 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.[46] These patients would gain more relief by taking a selective cyclooxygenase (COX)-2 inhibitor, such as diclofenac or celecoxib.

Because of their antiinflammatory effect, NSAIDs can suppress swelling to a certain degree after surgical procedures. The good analgesic effect combined with the additional

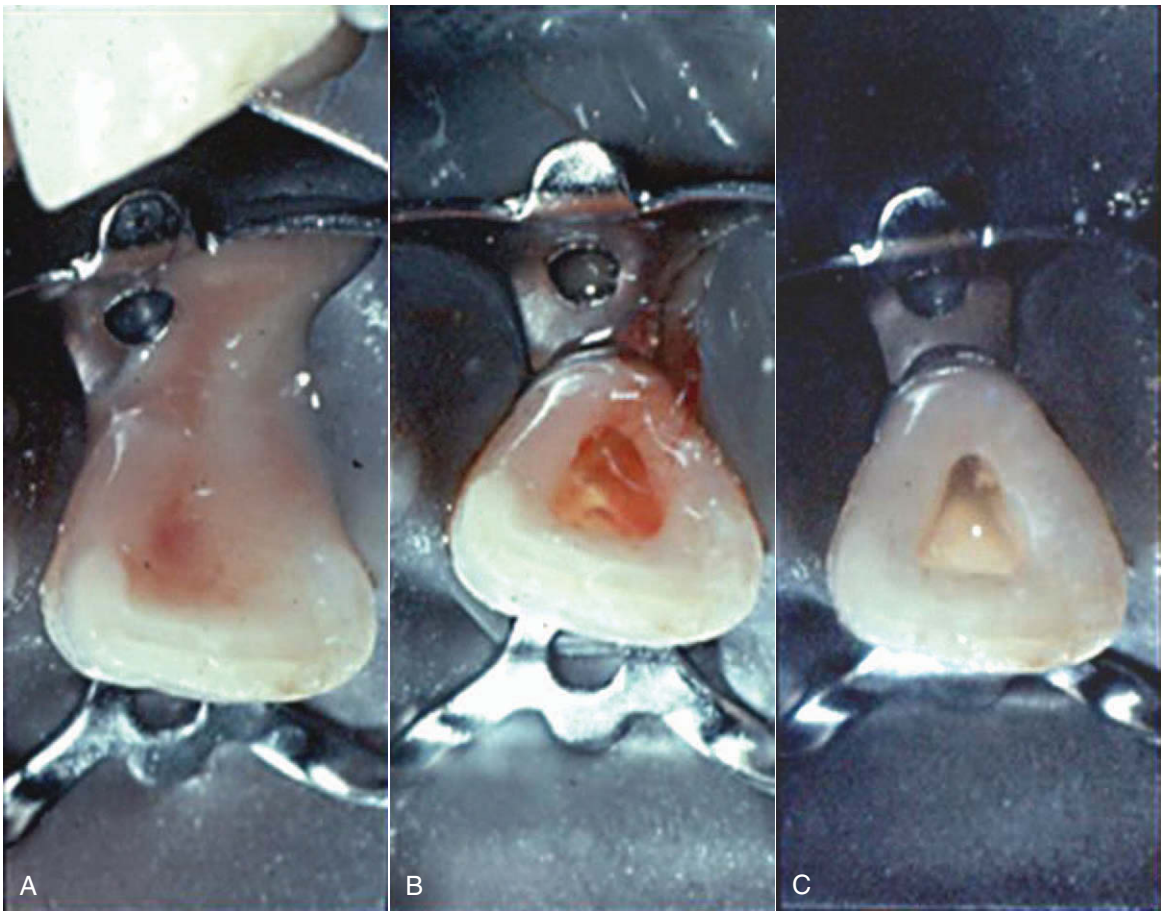


FIG. 2-2 Nonvital infected tooth with active drainage from the periapical area through the canal. **A,** Access opened and draining for 1 minute. **B,** Drainage after 2 minutes. **C,** Canal space dried after 3 minutes.

antiinflammatory 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.[16] 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 15 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 15), 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.[2] The incidence may be from 2% to 20% of cases.[38,49,55,76] A meta-analysis of the literature, using strict criteria, showed the flare-up frequency to be 8.4%.[73] Endodontic flare-ups seem to be more prevalent among females under the age of 20 years, and may occur more in maxillary lateral incisors; mandibular first molars, when there are large periapical lesions; and in the re-treatment of previous root canals.[70] The presence of pretreatment pain may also be a predictor of potential posttreatment flare-ups.[38,70,76] Fortunately, there does not seem to be a decrease in the successful conclusion of cases that had a treatment flare-up.[41]

Endodontic flare-ups may occur because of a variety of reasons, including preparation beyond the apical terminus, overinstrumentation, pushing dentinal and pulpal debris into the periapical area,[27] 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.[65] Although many of these cases can be pharmacologically managed (see Chapter 19), recalcitrant cases may require reentry into the tooth, the establishment of drainage either through the tooth or via trephination, or, at a minimum, adjustment of the occlusion.[15,62,65] The prophylactic use of antibiotics to decrease the incidence of flare-ups has been met with some controversy. Whereas earlier investigators[50] found that antibiotic administration before treatment of necrotic teeth decreased the incidence of flare-ups, a more recent study[70] found antibiotic use less effective than analgesics in reducing interappointment

emergencies, and other, more current studies[60,75] concluded that the prophylactic use of antibiotics had no effect on post-treatment symptoms.

CRACKED AND FRACTURED TEETH

Described in detail in Chapter 1, cracks and 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[11] 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 the necrosis. 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.

ACKNOWLEDGMENT

The editors acknowledge Dr. Louis Berman for his extraordinary contribution to preparing this chapter for publication.

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CHAPTER 3

Diagnosis of Nonodontogenic Toothache

DONNA MATTSCHECK, ALAN S. LAW, and DONALD R. NIXDORF

An unthinking dentist is a bad dentist. Perfect technique misapplied is at least as unconscionable as sloppy work.
MARJORIE JEFFCOAT, DMD

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

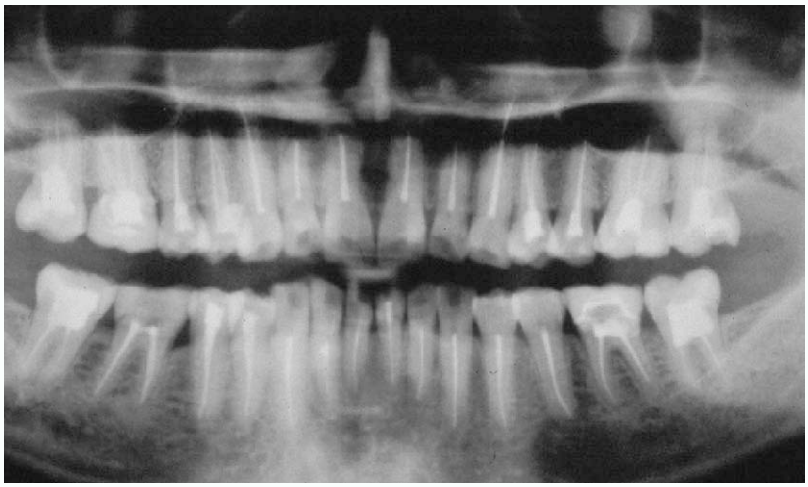


FIG. 3-1 Pantomogram of a patient who has undergone several endodontic procedures without resolution of her chief complaint. *(Courtesy Dr. Jeffrey Okeson, Lexington, Kentucky.)*

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 the person's perception 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 "referred pain phenomenon" and is known to occur in multiple areas of the body. Thus, a nonodontogenic toothache has a source of pain that is not the tooth indicated by the patient, thereby clearly demonstrating the diagnostic challenge being presented.

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."[16] 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.[104] One investigator reported that over a 6-month period of time, 22% of Americans experienced at least one of five types of facial pain. Of these pains, the most common type (12.2%) was toothache.[71]

Although toothache is the most common pain entity occurring in the facial region,[71] it is clear that 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 have an understanding of 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. Structures of the orofacial region can be divided 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. Pain from these deep structures is typically poorly localized and diffuse.

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. Transmission of nerve impulses from orofacial structures to the brain is via the peripheral nervous system, whereas modulation and interpretation of these impulses into what we feel as pain occur 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 proprioception. Such fibers can be found in the periodontal ligament, skin, and oral mucosa and include the A-beta fibers.

PRIMARY AFFERENT NEURONS

Detection and encoding of noxious stimuli for the orofacial region are performed primarily by the trigeminal, or fifth cranial, nerve. 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, gingiva, and 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 vast majority of dental nerves are C fibers that innervate the central pulp, and most of which terminate beneath the odontoblasts.[20]

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[109] or "prepain."[20] A-beta fibers have been shown to undergo phenotypic changes that allow them to encode painful stimuli under inflammatory conditions.[84]

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)[10] or respond only to cold/mechanical[68] or hot/mechanical noxious stimuli.[34]

In the tooth pulp, A-delta fibers traverse the odontoblastic layer and terminate in the dentinal tubules.[22] Because of their location and their sensitivity to mechanical stimulation, A-delta fibers are believed to respond to stimuli that result in movement of fluid within the dentinal tubules (e.g., osmotic, mechanical probing or thermal stimuli applied to the external surface of the tooth).[15] 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.[81] 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.[34] 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. 3-2).

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,[34,50,120] which anatomically and functionally resembles the dorsal horn of the spinal cord and has been referred to as the medullary dorsal horn.[50] 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.[62] Within the subnucleus caudalis, the A-delta and C fibers terminate primarily in the outer laminae

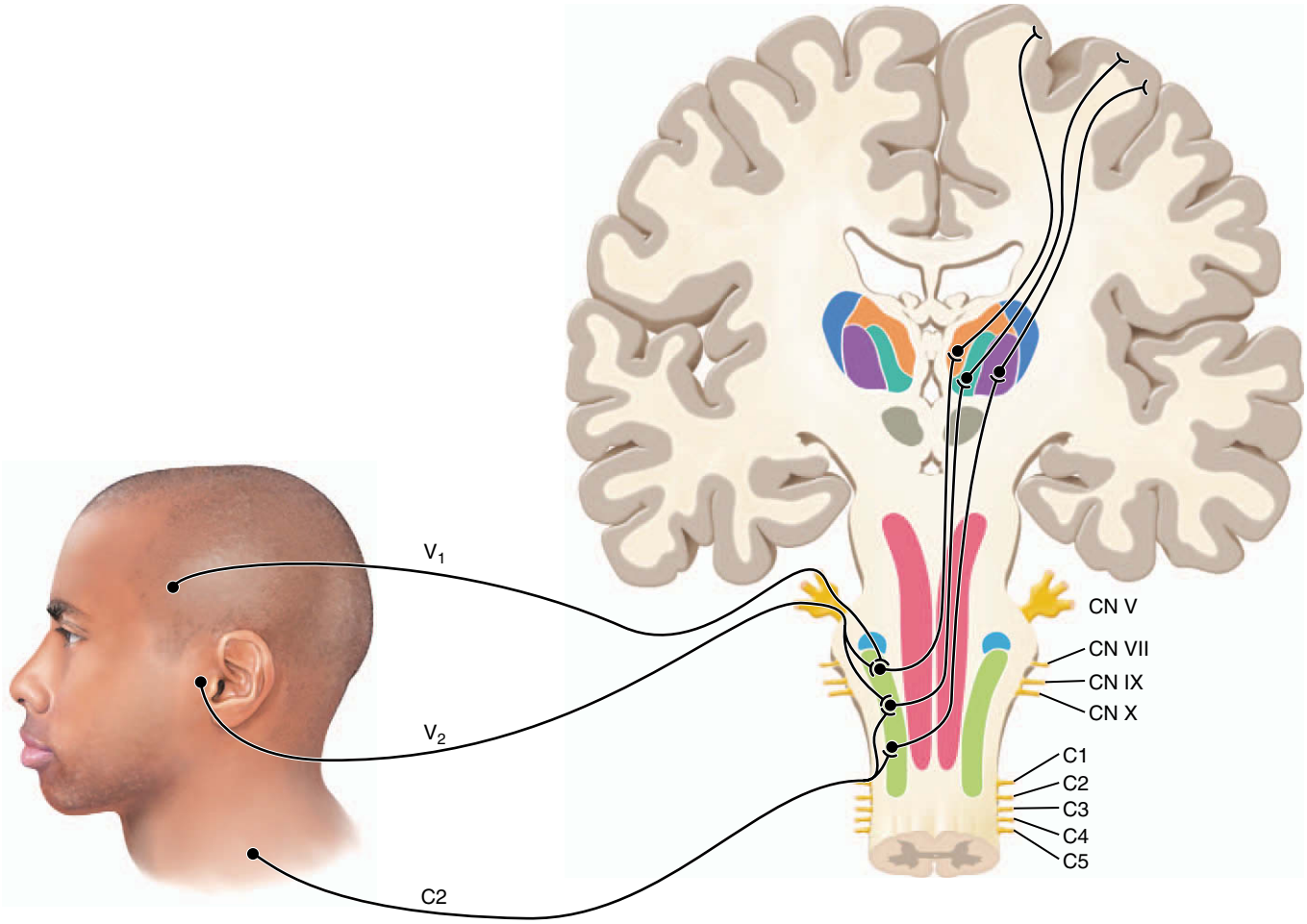


FIG. 3-2 A graphic depiction of the trigeminal nerve entering the brainstem. The primary afferent neuron synapses with a second-order neuron in the trigeminal nucleus. The second-order neuron carries pain information to the thalamus from which it is sent to the cerebral cortex for interpretation. (*Redrawn from Okeson JP*: Bell's orofacial pains, *ed 5, Chicago, 1995, Quintessence Publishing.*)

(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).[33] Combined, the local circuit neurons may modulate nociceptive transmission from the primary afferents to the projection neurons.

The fourth component of the dorsal horn is 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) and/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 results in increased pain transmission and reduced 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.[69]

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.[64] 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.[99] 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 entire sympathetic innervation of the orofacial region is supplied by the stellate ganglia, 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.[61] Whether the effects of sympathetic nerve fibers on pain transmission are direct (via homeostatic regulation) or indirect remain 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 conditions 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 and/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 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 19 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.[13] 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 we 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 3-1).

BOX 3-1

Types of Pain

Pain
An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in term of such damage[73]

Nociceptive Pain
Pain arising from activation of nociceptors[73]

Neuropathic Pain
Pain arising as a direct consequence of a lesion or disease affecting the somatosensory system[73,111]

Peripheral Sensitization
Increased responsiveness and reduced thresholds of nociceptors to stimulations of their receptive fields[73]

Central Sensitization
Increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input[73]

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.[87] 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. 3-3). 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. 3-4). 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

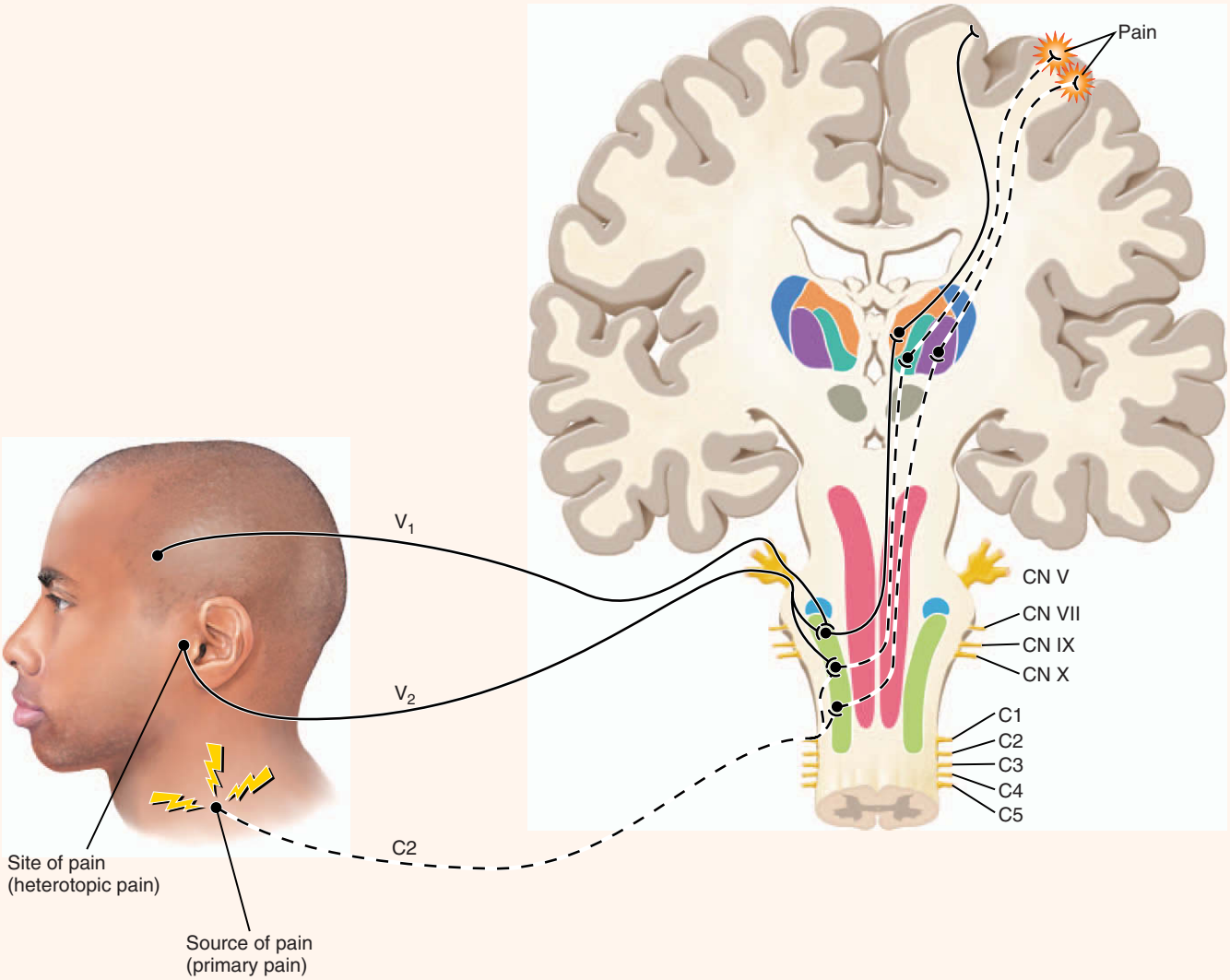


FIG. 3-3 Illustration of pain that is referred from an area innervated by one nerve *(C2)* to an area innervated by a different nerve *(V2)*. Note that this phenomenon occurs secondary to the convergence of different neurons onto the same second-order neuron in the trigeminal nucleus. The sensory cortex 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.)*

Continued

BOX 3-1

Types of Pain—cont'd

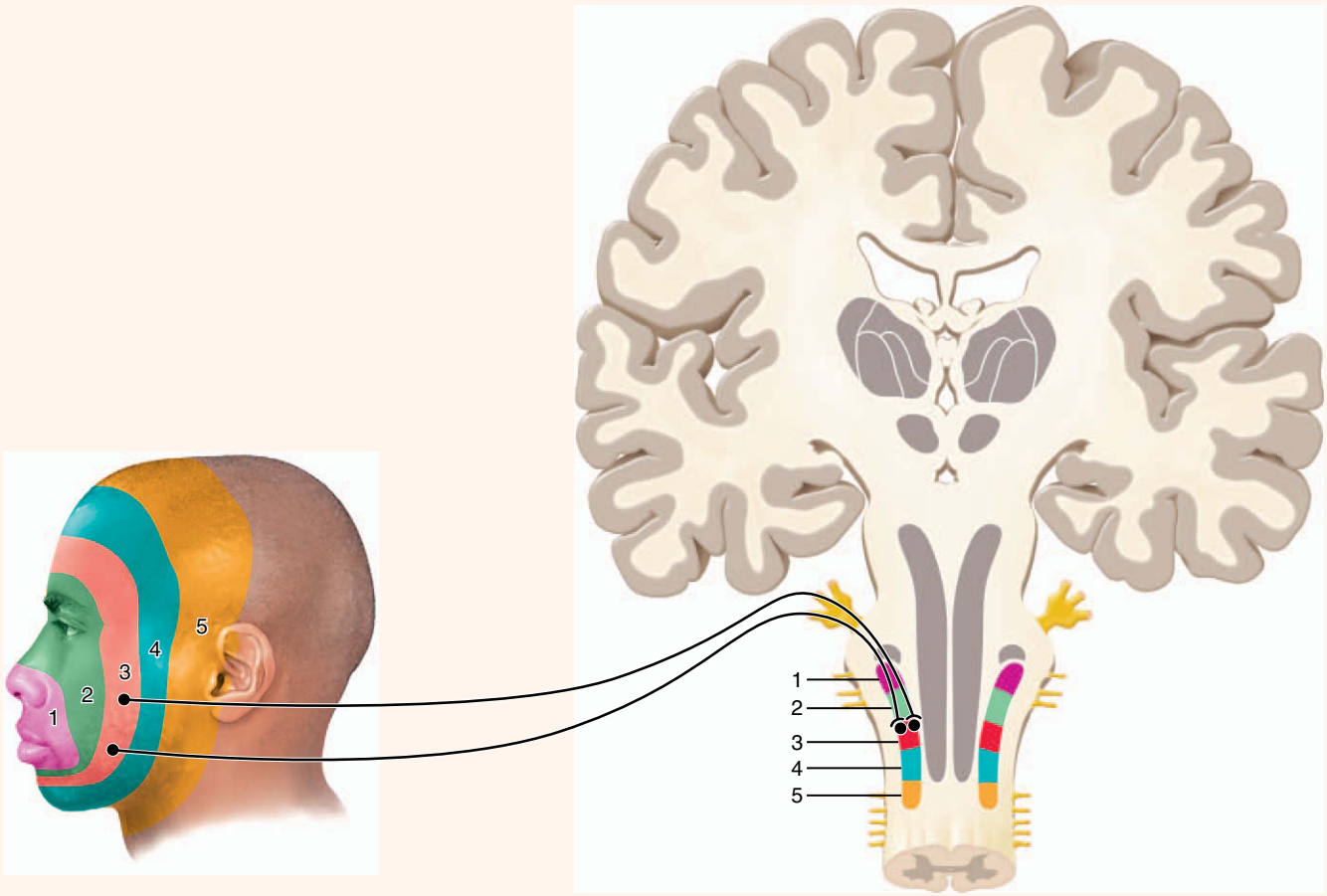


FIG. 3-4 Illustration of the laminated pattern of innervation from orofacial structures into the trigeminal nucleus. These laminated patterns commonly reflect the patterns of referred pains felt in the orofacial structures. *(Redrawn from Okeson JP:* Bell's orofacial pains*, ed 5, Chicago, 1995, Quintessence Publishing.)*

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. There are only two structures that serve as sources for primary odontogenic pain. These structures are 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 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, 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 of "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,[81] 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.

Typical of 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.[19] In addition, the dental pulp lacks 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 there is convergence of neurons from the pulps of mandibular teeth with those of maxillary teeth can result in pain from a mandibular pulpitis being referred to the maxillary arch. Because pulpal pain may be poorly localized by the patient, 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.[74] 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 their periradicular pain is dependent 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 (vs. "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, spontaneous or provoked with various stimuli applied at various intensities. The quality can vary between sharp and dull, 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 play a large role in adding 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 for the dental clinician to aid in the identification of 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 will prevent misdiagnosis and allow 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 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.[111] Our classification scheme uses this framework to enhance clarity of communication and follow consensus, even though the application of these criteria to pains that present in the orofacial region is known to be associated with misclassification.[30]

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.[40] 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.[110] 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.[118]

Although the definitive pathogenesis of MFP is unknown, authors have theorized that muscles may become disturbed through injury or sustained contraction such as clenching.[39,88] 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 and/or palpation sensitivity, and/or failure to resolve symptoms with local anesthetic blockade. In contrast, jaw function and palpation of the masticatory muscle(s) will elicit toothaches of myofascial origin. Typically, local anesthetic infiltration into the trigger point(s) will lead to resolution of symptoms.

Some 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 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 later section Additional Tests for further information about trigger point injections.

PAIN OF SINUS AND/OR NASAL MUCOSAL ORIGIN

Sinus/nasal mucosal pain is another source of pain that can mimic toothache.[2,3,25,114] 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.[32] 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 and/or nasal drainage) should be noted in the patient history.

Typical of deep visceral 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, will result 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 and/or erythema in the area, which could be suggestive of a dental abscess. However, when an etiology for pulpal and therefore periradicular pathosis is absent, sinus/nasal mucosal disease should be suspected. 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 mucosa 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 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.[31] Treatment of sinus/nasal mucosa 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; this has not been encountered in clinical practice by the authors, but has been reported to present as a nonodontogenic toothache.[70,94] Because the primary somatosensory innervation of the 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.[49] 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, the dental clinician 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 comprise 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, with about 18% of females and 6% of males experiencing this type of headache.[72,105] 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.[107] Migraine has been reported to present as toothache[4,23,29,46,82,86] and is likely the most common neurovascular disorder to do so. In addition to this, people with migraine headaches are thought of as having increased regional pain sensitivity that has diagnostic and treatment implications for the clinician.[85]

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 and/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.[79] Of note: Migraine headaches may partially or fully abate with the use of nonsteroidal antiinflammatory 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%).[96,100] 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,[106] 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).[48]

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,[49] thus the name. Elimination of pain after 10 minutes' inhalation of 100% oxygen is diagnostic for cluster headache[43]; sublingual ergotamine and sumatriptan are also effective acute treatment for cluster headache.[36] 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 lasting 2 to 30 minutes.[49] This headache disorder has a 100% response to indomethacin but is refractory to other treatments,[56] which underscores the need for obtaining an accurate diagnosis from an experienced clinician.

From a nonodontogenic perspective, cluster headache[4,11,18,45] and almost all the other TACs have been reported in the literature to present as nonodontogenic toothache.[4,8,9,27,77,91,98] 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.[78] 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. 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 visualized. 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 that the different subcategories 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, it 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. While 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 result in a diminution of 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 related to their pain,[51,119] such as brain tumors and multiple sclerosis.

Trigeminal neuralgia typically presents in individuals more than 50 years of age. The etiology is thought to be inflammation in one of the trunks of the gasserian ganglion, possibly as a result of carotid artery pressure. Individuals with multiple sclerosis will develop trigeminal neuralgia more frequently than the general population. For this reason, a person less than 40 years of age who develops trigeminal neuralgia should also be screened for multiple sclerosis[122] or other intracranial pathosis.[51] 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.[6] 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 suggested by the name, 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 yr), take on the classical 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.[42] The subsequent onset of true neuralgic pain may be quite sudden or may appear several years later,[88] 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's sign) similar to trigeminal neuralgia. In contrast to trigeminal neuralgia, there should be a zone of anesthesia peripheral to the area of the neuroma[92] that can be identified by checking for loss of pinprick sensibility, such as with the use of an explorer. Treatment involves pharmacologic management, often via local measures, and surgical coaptation of the nerve with prognosis being variable and dependent on adequate distal nerve tissue and time interval between injury and reconstruction.[123] Therefore, early recognition and referral are of key importance to prevent significant distal nerve degeneration.[66] 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.[60] 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.[92] 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 later section Neuropathy.

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, will be associated with skin or mucosal lesions (Fig. 3-5). This presentation does not result in much of a diagnostic challenge, but pain can precede the vesicular outbreak by many days or even weeks.[41] 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[41,53,58] 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 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 nonnoxious stimulation such as light brushing of the skin. Oral acyclovir has become the most common treatment for acute herpetic outbreaks and 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.[103] 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.[116]

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. 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. 3-6).[80] Mechanical compression in addition to thermal trauma may be a factor when thermoplasticized material is overextended, using an injectable[44] or carrier-based technique. Mechanical nerve trauma is more commonly associated with oral surgical procedures such as orthognathic surgery and third molar extraction.

Neuropathic complications have also been documented after mandibular implant surgery at a rate of 5% to 15% with permanent neuropathies resulting in approximately 8% of these cases.[57] It is unfortunate that traumatic neuritis is often misdiagnosed as a posttreatment chronic infection and that the area is re-entered 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.

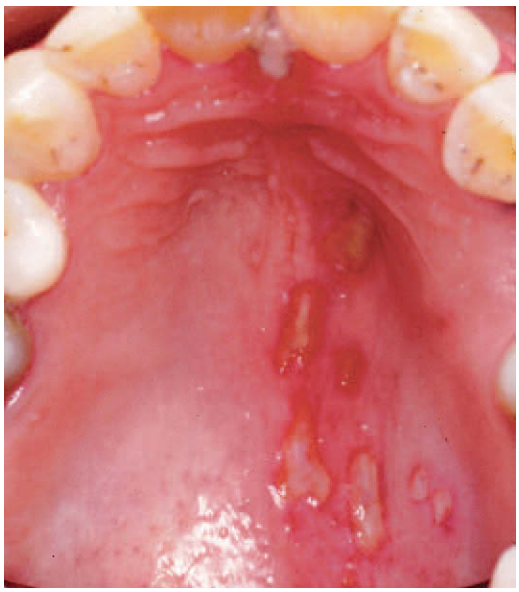


FIG. 3-5 Herpes zoster involving the maxillary division of the left trigeminal nerve of the palate of a 45-year-old male. He complained of a deep, diffuse dull ache of his maxillary left quadrant for 1 week before this vesicular outbreak.



FIG. 3-6 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.

Neuritic pain typically is a persistent, nonpulsatile burning and is 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 there is obvious irritant present, surgical debridement of the nerve to remove any substance that can continue to irritate the nerve is an important aspect of treatment. With a 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).

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. If pain of an unknown source is perceived in a tooth it may be labeled *atypical odontalgia*. If the pain persists after the tooth has been extracted it 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 their pathophysiology. Although each of these terms has been extensively described in the literature,[75,76] probably none of them actually represents one discrete condition but rather a collection of various conditions. At present, a subcommittee of the International Association for the Study of Pain (IASP, Seattle, WA) has put on hold the development of diagnostic criteria and terminology for orofacial pain because the research evidence does not clearly indicate preferred terminology based on presumed mechanisms. Until these criteria are published the use of old terms, and the confusion they bring, will persist.

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 or sympathetic nervous systems. *Peripheral* sensitization, *central* sensitization, and *sympathetic* enhancement all 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, intensity level. 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 worse peripheral sensitization, a new-onset central sensitization, or a sympathetic component to their pain. This statement is not meant to refer to situations with concurrent nerve trunk compression or other type of physical or chemical irritation present.

A diagnosis of neuropathy is based primarily on history and examination. 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. Typically, the examination is grossly unremarkable with no evidence of local tissue damage. However, constant pain of varying degrees of intensity will be reported in a focal area. This area may be hyperalgesic or allodynic. That is, noxious stimulation to the area will be perceived as more painful or nonnoxious stimulation will now be perceived as painful. Neuropathic disorders have a predilection for women rather than men but can affect both genders. These patients are usually more than 30 years of age and may have a history of migraine.[102] In the orofacial region neuropathies are most commonly seen in the maxillary premolar area and molar region.[54]

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. Some topical medications include topical anesthetics and capsaicin-containing compounds as well as transdermal preparations of nonsteroidal antiinflammatory drugs (NSAIDs), sympathomimetic agents, and *N*-methyl-D-aspartate (NMDA) receptor-blocking agents.[90]

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.

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 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.[117] 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 the block is effective in decreasing 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.

Psychogenic Toothache

Psychogenic toothache falls within a group of mental disorders known as somatoform. 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 will also present without local tissue changes. Patients with somatoform disorder are not fabricating the symptoms, nor are they seeking conscious benefit. It is important to make a distinction between somatoform disorders and factitious or malingering disorders.[5] In factitious disorders there are physical and/or psychologic symptoms that are produced by the individual and are under voluntary control. Malingering is similar to factitious disorder with the added characteristic that the symptoms are presented for obvious and recognizable benefit. This diagnostic 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, and neurovascular and neuropathic 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 physiologic patterns. The pain may be felt in multiple teeth and the pain 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 their condition. Their response to therapy is variable, including a lack a response or an unusual or expected 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 different pathologies that seem to be unrelated have been reported to present as nonodontogenic toothache.[89,94] The only common link that can be identified is that the involved tissues are innervated by branches of cranial nerves, and hence nociceptive input is processed in the trigeminal nucleus. Therefore, conceivably, any somatic structure with cranial nerve innervation has the potential to cause pain that is perceived by the patient 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.[7,35,55,67,83,112] 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.[14] 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.[7] 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, on both the ipsilateral and contralateral sides of the tumor,[21,52] as well as diaphragmatic pain mediated via the phrenic nerve.[12]

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.[1,113] 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.[26] 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 non-odontogenic pain being associated with smooth muscle tumors of a similar location.[115] 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.[97]

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[24,37,121] and jaw,[108] as well as metastatic disease, particularly within the mandible,[28,47,93,101] 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), especially 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,[17] 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.[59,63] These pains have been described as a continuous dull pain that can sometimes be made worse with jaw function. The stereotypical presentation include 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 and, because permanent blindness is a possible sequela if cranial arteries are left unmanaged, immediate referral to the appropriate medical colleague.

TAKING A PATIENT'S HISTORY

Pain diagnosis is largely based on the patient's subjective history; however, it is rare that patients will 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. Figure 3-7 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 though 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. 3-8), 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 serve to 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 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 their pain complaint then it may be helpful to have them keep a pain history, detailing the aspects of their 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.

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. 3-7 Example of a form to evaluate odontogenic pain.

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 their own words is a medical legal necessity but falls short of comprising 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.

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. 3-8 Example of a form to evaluate pain history.

Understanding the specific components of the complaints makes it possible to discern the relationship between the complaints. That is, either the complaints are wholly separate and there are two types of pathosis 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 also 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 to 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 question should be asked: 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 either 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 be more indicative of A-delta fiber-mediated dentinal pain, or whether it is electrical in nature, which would be more indicative of a neuralgia. Some common examples of pain descriptors and their respective pain types are listed in Table 3-1.

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 is an indication that the pain is not of odontogenic origin.

Alleviating factors can provide insight as to the nature of the pain. If the pain is relieved by a medication 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, a pain that is mid-level in intensity yet completely unresponsive to antiinflammatory drugs suggests a pain that is not inflammatory in origin.

TABLE 3-1

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 is suggestive of 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 there is more than one pain complaint present, 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 be helpful in narrowing down a differential. Any treatment that was performed should be ascertained and 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 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 detail 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 (see Fig. 3-9, *A* and *B*). 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 to the patient's pain. 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 25-gauge needle and a minimally myotoxic anesthetic such as 2% lidocaine or 3% mepivacaine without a vasoconstrictor. Myofascial trigger point injections may result in temporary relief from 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. In patients in whom there is suspicion of a sympathetic component to their pain, a stellate ganglion block can be used to provide diagnostic insight. This is usually done by a trained anesthesiologist. An effective block delivered to the stellate ganglion will interrupt sympathetic outflow to the ipsilateral side of the face, resulting in a partial Horner's syndrome. This is evidenced by flushing, congestion, lacrimation, miosis of the pupil, ptosis, and anhidrosis.[65] A sympathetic blockade that diminishes or eliminates a pain state may serve to guide future treatment such as repeated blocks or systemic treatment with sympathetically active drugs (e.g., clonidine and prazosin).[95]

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. Patients with 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–xii should be performed. For details on cranial nerve examination, please refer to other sources.[38] Investigation of sharp/dull differentiation as well as light touch discrimination between the different branches of the trigeminal nerve can provide insight as to location and etiology of 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." His medical history reveals a history of angina secondary to 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 having two different qualities: an

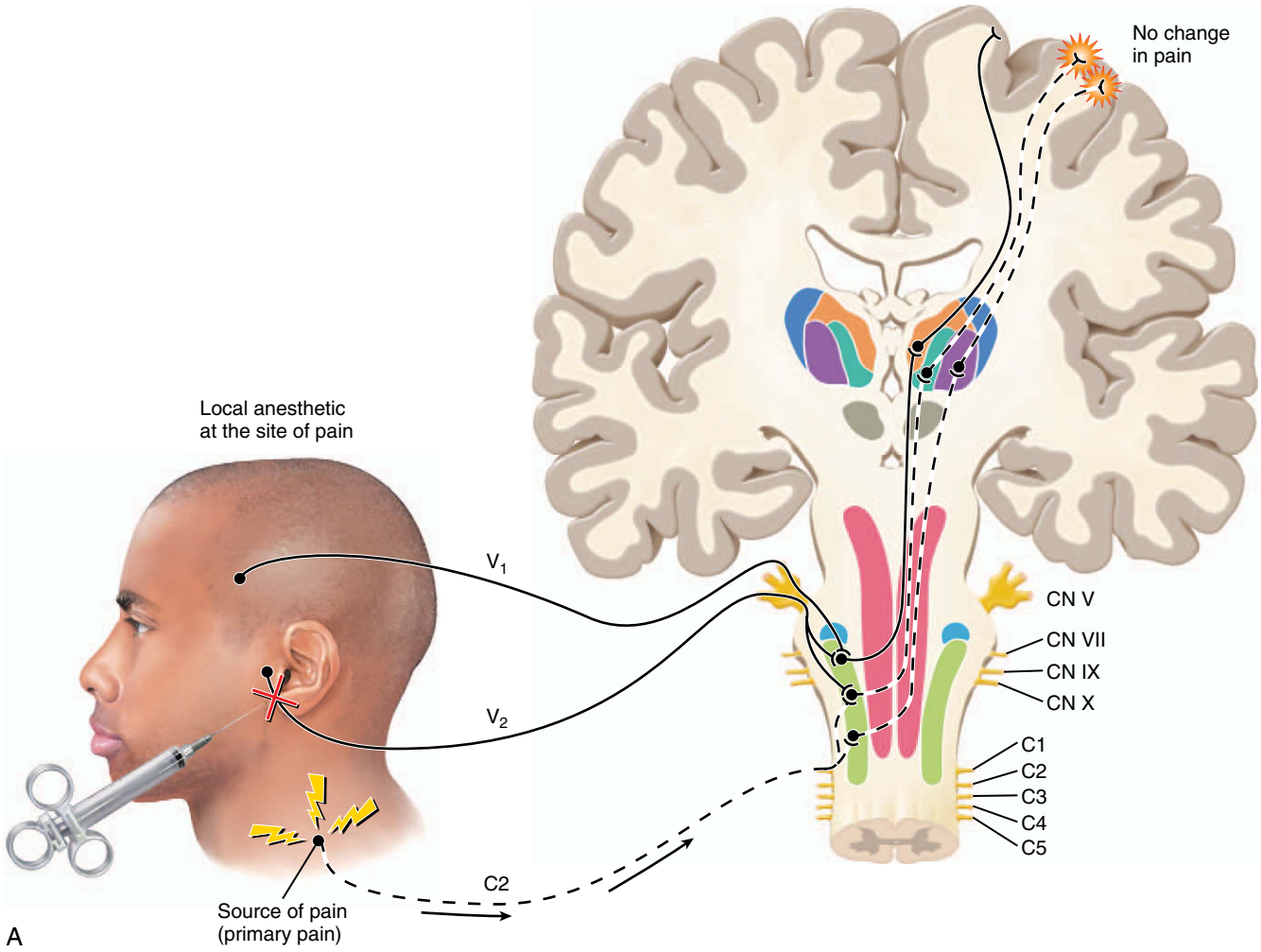


FIG. 3-9 A, Local anesthesia at the site of pain fails to reduce the pain.

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 last 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 "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 3-2.

TABLE 3-2

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.

A periapical radiograph (Fig. 3-10) 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.

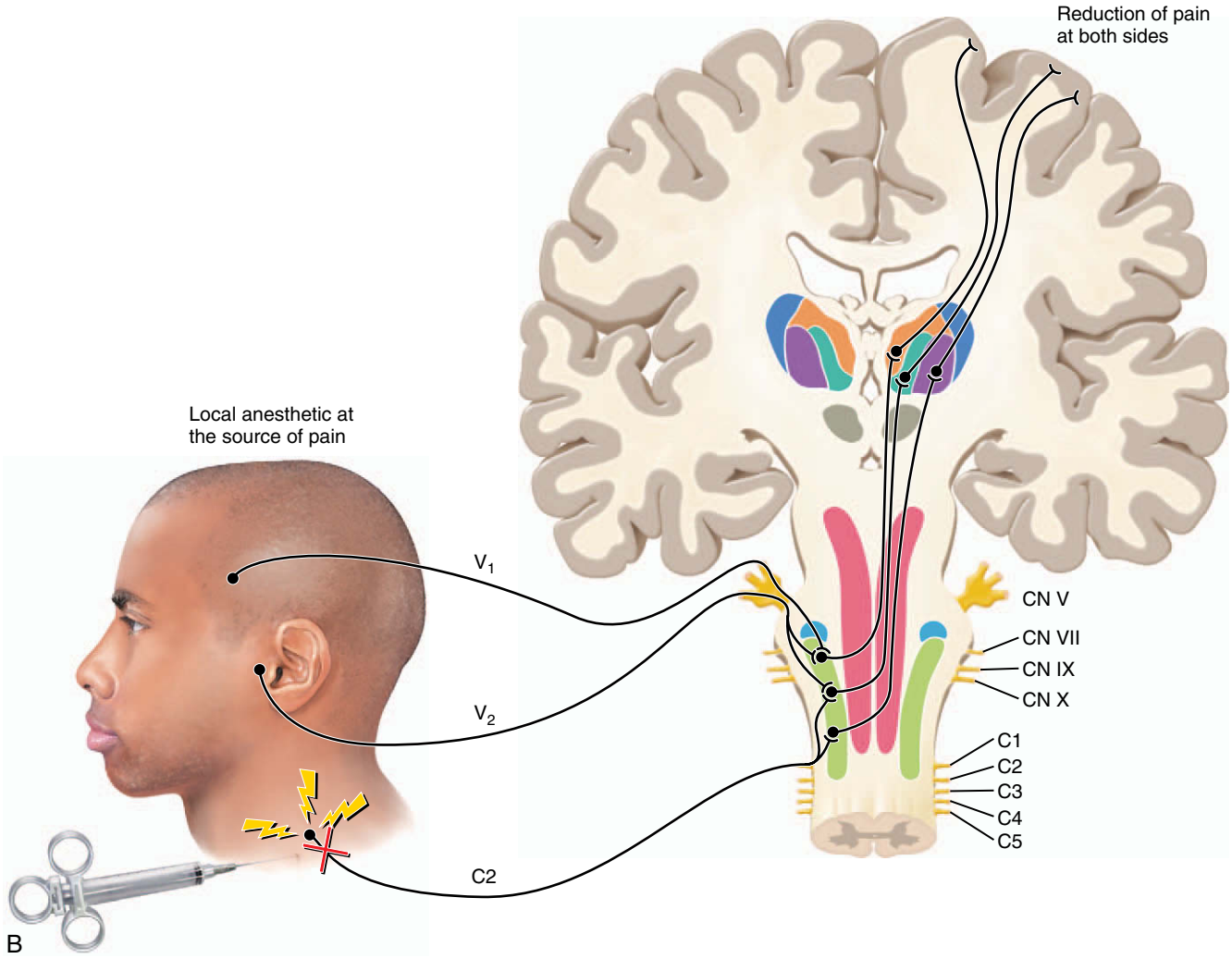


FIG. 3-9, cont'd B, Local anesthesia at the source of pain reduces the pain at the source as well as at the site. *(Redrawn from Okeson JP:* Bell's orofacial pains, *ed 5, Chicago, 1995, Quintessence Publishing.)*



FIG. 3-10 Periapical radiograph showing prior nonsurgical root canal treatment and mesiobuccal root amputation of tooth #3.

Additional tests: In the absence of a clear etiology, a more extensive extraoral examination is performed. Cranial nerves ii–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 past 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 no attempts have been made by the patient 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 ML cusp of tooth #30. After the bite test he reports an intensifying of his dull ache. For the results of clinical testing see Table 3-3.

Thirty seconds after pulp testing has been completed the patient again reports an intensifying of his dull ache.

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 his right deep masseter. Palpation of this trigger point results in immediate intensifying of 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.

Diagnosis 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 3-3

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 odontogenic and nonodontogenic causes of orofacial pain. 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 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

4

Case Selection and Treatment Planning

PAUL A. ROSENBERG and JARED C. FRISBIE

CHAPTER OUTLINE

COMMON MEDICAL FINDINGS THAT MAY INFLUENCE ENDODONTIC TREATMENT PLANNING
- Cardiovascular Disease
- Diabetes
- Pregnancy
- Malignancy
- Bisphosphonate Therapy
- Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome
- End-Stage Renal Disease and Dialysis
- Prosthetic Implants
- Behavioral and Psychiatric Disorders
- Psychosocial Evaluation

DENTAL EVALUATION AND DEVELOPMENT OF THE ENDODONTIC TREATMENT PLAN
- Periodontal Considerations
- Surgical Considerations
- Restorative Considerations
- Endodontic Therapy or Dental Implant
- Vital Case
- Nonvital Case
- Single-Visit versus Multiple-Visit Treatment
- Retreatment Case
- Immature teeth
- Other Factors That May Influence Endodontic Case Selection

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 could not have been treated. 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, nonsteroidal antiinflammatory drug (NSAID) premedication, profound local anesthesia, appropriate occlusal adjustment, and biologically based clinical procedures enables clinicians to complete endodontic procedures without intra- 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.[32,40,78] The American Academy of Oral Medicine (Edmonds, WA) has an excellent website (http://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. Figure 4-1 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 4-1).

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 needs to 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 medically complex patients treated by clinicians as Americans live longer.[31] Regardless of the classification system used, these

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. 4-1 Sample medical consultation letter.

BOX 4-1

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

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
- Anxiety (past experiences and management strategy)
- 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)

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. Patients who have had a myocardial infarction (i.e., "heart attack") within the past 6 months should not have elective dental care. Such patients have increased susceptibility to repeat infarctions and other cardiovascular complications and may be taking medications that could potentially interact with the vasoconstrictor in the local anesthetic. In addition, a vasoconstrictor should not be administered to patients with unstable angina pectoris or to patients with uncontrolled hypertension, refractory arrhythmias, recent myocardial infarctions (less than 6 mo), recent stroke (less than 6 mo), recent coronary bypass graft (less than 3 mo), uncontrolled congestive heart failure, and uncontrolled hyperthyroidism. Vasoconstrictors may interact with some antihypertensive medications and should be used only after consultation with the patient's physician. For example, vasoconstrictors should be used with caution in patients taking digitalis glycosides (e.g., digoxin) because the combination of these drugs could precipitate arrhythmias. Local

anesthetic agents with minimal or no vasoconstrictors are usually adequate for nonsurgical endodontic procedures (see also Chapter 20).[40] A systematic review of the cardiovascular effects of epinephrine concluded that, although the quantity and quality of pertinent articles were problematic, 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.[6] 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."[12]

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. The American Heart Association (AHA; Dallas, TX) amended their recommendations in 2007 and recommends antibiotic prophylaxis in patients with prosthetic heart valves, a history of infective endocarditis, and a number of congenital heart abnormalities.[77] The specific recommendations are summarized in a reference guide by the American Association of Endodontists (AAE; Chicago, IL) found online at http://www.aae.org/dentalpro/guidelines.htm. 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. A low compliance rate exists 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.[77]

Patients with artificial heart valves are considered highly 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. The coronary artery bypass graft is a common form of cardiac surgery. Ideally, vasoconstrictors should be minimized during the first 3 months after surgery to avoid the possibility of precipitating arrhythmias. Ordinarily these patients do not require antibiotic prophylaxis after the first few months of recovery unless there are other complications.[77]

A clinician may be the first to detect elevated blood pressure if he/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.

Few conditions exist in which there is a possibility that dental treatment could seriously injure the patient or result in death. However, 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.[67] 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 dental clinicians 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.[35,40] 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., international normalized ratio [INR] values for patients receiving warfarin therapy).
- Be familiar with methods used to obtain hemostasis both intra- and posttreatment.
- 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's 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. Therapeutic irradiation of the chest results in the inadvertent inclusion of the heart within the irradiation field. Some patients may experience pathologic changes of the heart valves that could predispose them to endocarditis, accelerated atherosclerosis of the coronary artery that increases their risk of experiencing a fatal myocardial infarction, or both. Clinicians must identify patients who have received irradiation to the chest and consult with patients' physicians in order 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.[27]

Diabetes

The Centers for Disease Control and Prevention (CDC, Atlanta, GA) in 2006 reported a 6% increase in the incidence of diabetes mellitus in the United States in 1 year.[30] This dramatic change has been linked to a remarkable increase in obesity among Americans during the last 10 years. Diabetes is the third leading

cause of death in the United States, and it is estimated that nearly 20 million Americans, representing 6.5% of the population, have diabetes.[45] Even more alarming is the fact that approximately 6 million of those cases have not been diagnosed. It is likely that diabetic patients requiring endodontic treatment will be increasingly common.

Diabetes mellitus appears to have multiple causes and several mechanisms of pathophysiology.[40] 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 diabetes who is well controlled medically and 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.[57] 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 diabetic patients who self-monitor their glucose levels to bring a glucometer to each visit. If pretreatment glucose levels are below normal fasting range (80-120 mg/dl), it may be appropriate to take in a carbohydrate source.[72] 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.[40] Signs and symptoms of hypoglycemia include confusion, tremors, agitation, diaphoresis, and tachycardia.[72] The clinician can avoid a hypoglycemic emergency by taking a complete, accurate history of time and amount of the patient's insulin and meals.

Appointments should be scheduled with consideration given to the patient's normal meal and insulin schedule.[57] 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.[45] However, patients with diabetes who have serious medical complications may need a modified dental treatment plan.[32] Moreover, studies suggest that diabetes is associated with a decrease in the success of endodontic treatment in cases with pretreatment periradicular lesions.[11,26] These patients may require referral to an endodontist for alternative treatment considerations.

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.[7,76] Although drug administration during pregnancy is a controversial subject, Box 4-2 presents commonly used dental drugs usually compatible with both pregnancy and breast-feeding.[40] 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 difficult, then the clinician should review the appropriate current literature and discuss the case with the physician and patient.[10,43,47]

BOX 4-2

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

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. There are limited data available on drug dosages and the effects on breast milk.[40]

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. 4-2). A panoramic radiograph is 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 is indicative of 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.

A useful website for the differential diagnosis of radiographic lesions (Oral Radiographic Differential Diagnosis [ORAD] II) was created by Dr. Stuart White and is available online at http://www.orad.org/index.html. A definitive diagnosis of a periradicular osteitis 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. Treatment should be initiated only after the patient's physician has been consulted. Resolving the question of endodontic treatment or

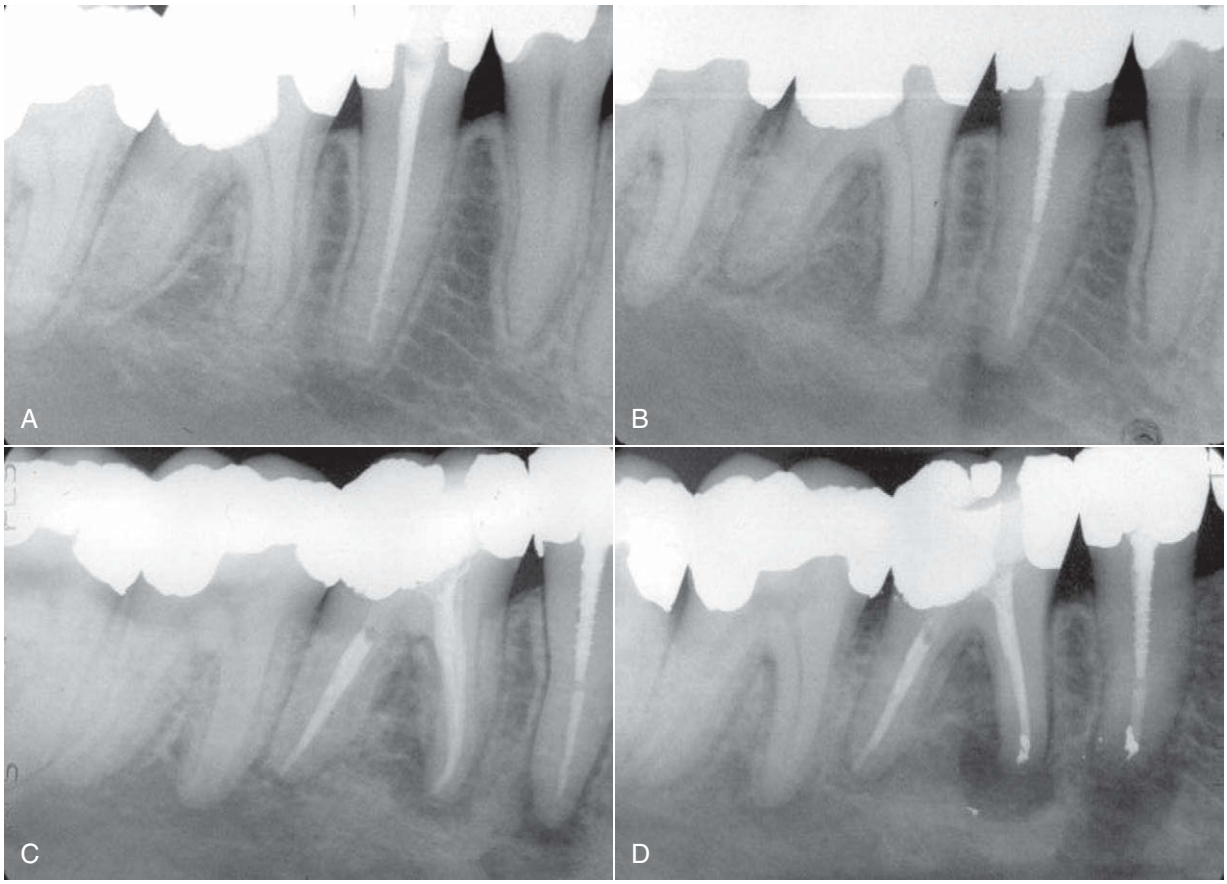


FIG. 4-2 A, Periapical view of tooth #29 after endodontic treatment by a general dental clinician. The diagnosis was irreversible pulpitis. **B,** Patient was referred to an endodontist 4 months later to evaluate radiolucencies of teeth #29 and #30. Symptoms indicated irreversible pulpitis of tooth #30, 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.)*

extraction for preradiation patients often requires a dialogue between the dental clinician and the physician.

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.[40] 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.[40] 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.[59]

Bisphosphonate Therapy

Bisphosphonates offer great benefits to patients at risk of bone metastases and in the prevention and treatment of osteoporosis. 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 bisphosphonate-associated osteonecrosis (BON) (Box 4-3).[3,4]

Because there are no studies that appropriately present the incidence of BON and treatment is unpredictable, prevention strategies are extremely important.[3,4] For patients at higher risk of BON, surgical procedures such as extractions, endodontic surgery, or placement of dental implants should be avoided, if possible. Sound oral hygiene and regular dental care may be the best approach to lowering the risk of BON.[3] Patients taking bisphosphonates and undergoing endodontic therapy should sign an informed consent form, inclusive of the risks, benefits, and alternative treatment plans.[4] In case of any infection in a patient taking bisphosphonates, aggressive use of systemic antibiotics is indicated.[40] Discontinuing bisphosphonate therapy may not eliminate any risk of developing BON.[41,42,44]

BOX 4-3

Risk Factors for Development of Bisphosphonate-Associated Osteonecrosis

- History of taking bisphosphonates for more than 2-3 years, especially with intravenous therapy
- History of cancer, osteoporosis, or Paget's 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

There is currently no valid diagnostic technique available to determine whether a patient is at risk of BON,[3] although 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 BON. For patients who have developed BON, close coordination with an oral maxillofacial surgeon or dental oncologist is highly recommended.

An astute awareness of the potential risk of BON in patients receiving bisphosphonate therapy is critical. Increased attentiveness to the prevention, recognition, and management of BON will allow the clinician to make the best treatment decisions. Our knowledge of BON is developing rapidly and it is essential that the clinician monitor published recommendation changes.[3,4,41,42,44]

Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome

It is important, when treating patients with acquired immunodeficiency syndrome (AIDS), that the clinician understand the patient's level of immunosuppression, drug therapies, and potential for opportunistic infections. Although the effect of human immunodeficiency virus (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.[56] 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 (for details see Universal Precautions for Prevention of Transmission of HIV and Other Bloodborne Infections, available at http://www.cdc.gov/ncidod/dhqp/bp_universal_precautions.html). Although saliva has not been demonstrated to transmit the virus in a dental situation, the potential for it to do so exists.[14,40] 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.[40]

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 400 mm^3 may receive all indicated dental treatment. Patients with a $CD4^+$ cell count less than 200 mm^3 will have increased susceptibility to opportunistic infections and may be effectively medicated with prophylactic drugs. Consultation with the patient's physician is advisable before performing surgical procedures and before initiating complex treatment plans.[40,65]

End-Stage Renal Disease and Dialysis

Consultation with the patient's physician is suggested 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 disease and preserve the patient's quality of life.[40,55]

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.[5] Although controversy exists, antibiotic prophylaxis should be provided for patients receiving hemodialysis and who have known cardiac risk factors. When prophylaxis is used, the standard regimen of the American Heart Association is recommended.[5]

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.[55] 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, because on the day of dialysis patients are generally fatigued and could have a bleeding tendency.[40]

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 (Chicago, IL) and the American Academy of Orthopaedic Surgeons (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 I) 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. It should be noted that

although endodontics has been shown to be a possible cause of bacteremia,[17,66] the risk is minimal in comparison with extractions, periodontal surgery, scaling, and prophylaxis.[52] Consultation with the patient's physician on a case-by-case basis is advisable to establish 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.[40] 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 necessary sophistication 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.[13,48] 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.[13]

DENTAL EVALUATION AND 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, must be considered. The alternative of a dental implant offers another choice when the endodontic prognosis is poor.

Periodontal Considerations

Extensive periodontal lesions frequently complicate the endodontic procedure being considered. Such lesions may necessitate consultation with an endodontist and/or periodontist in order to gather more information about the tooth's prognosis. A tooth with a poor periodontal prognosis may have to be sacrificed, despite a favorable endodontic prognosis.

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 18).

In primary endodontic disease the pulp is nonvital (Figs. 4-3 and 4-4), whereas in primary periodontal disease the pulp retains vitality. True combined endodontic–periodontal disease occurs less frequently. The combined lesion is found 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.[61] 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 for 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.[61]

Pathogenesis of the lesion can be better understood after vitality 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. 4-5).

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 correct failure of nonsurgical therapy. Before surgery, it is essential that the clinician attempt to determine the cause of failure. For example, a deficient restoration or recurrent decay will result in microleakage into the root canal space. Unless that issue is addressed, apical surgery will not be successful (Fig. 4-6).

Endodontic surgery (see Chapter 21) 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. 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.

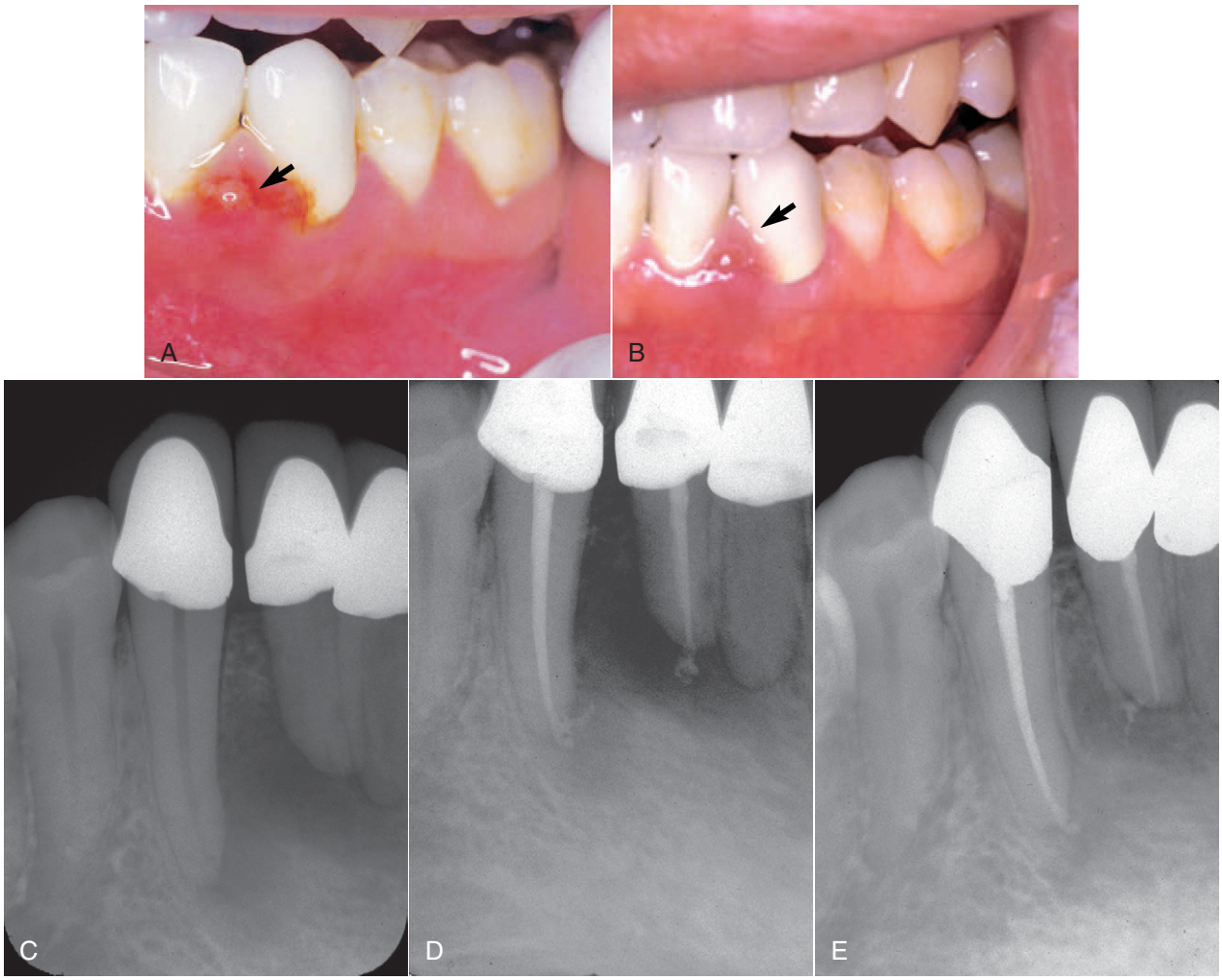


FIG. 4-3 A, Inflamed, edematous interproximal tissue *(arrow)* caused by acute endodontic pathosis. **B,** Soft tissue healing *(arrow)* 3 days after initiation of endodontic treatment. **C,** Periradicular pathosis. **D,** Completed endodontic therapy. **E,** Periradicular healing at 1-year recall.

The primary reason for apical surgery is to improve the quality of the apical seal. Dramatic changes in surgical technique and materials have occurred: The advent of microscopy, endoscopy, and ultrasonics, as well as of new retrograde materials such as mineral trioxide aggregate, represent important modifications of surgical technique.

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.[28] Numerous studies have examined the outcome of apical surgery and the results vary considerably.[73,74,80] This variability may reflect actual outcome differences or reflect variations in case selection techniques, recall periods, and methodology.

A study prospectively[74] assessed the 4- to 8-year outcome of apical surgery on 155 teeth in 138 patients. The recall rate was 85% and the overall healed rate was 74%. The healed rate was significantly higher for teeth with small (≤5 mm) than larger pretreatment lesions (χ^2, $P = 0.02$). Analysis indicated an increased odds ratio for disease persistence for teeth with larger pretreatment lesions and pretreatment root canal filling of adequate length. Pretreatment lesion size and root-filling length were significant predictors of the outcome of apical surgery.

One study assessed various predictors for healing 1 year after periapical surgery.[73] The study evaluated pretreatment, surgical, and posttreatment variables as possible predictors for a 1-year healing outcome in periapical surgery. Only one parameter, pain at initial examination, reached a 0.05 level of significance in a logistic regression analysis, indicating 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 having no pain at the initial examination.[73]

Restorative 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

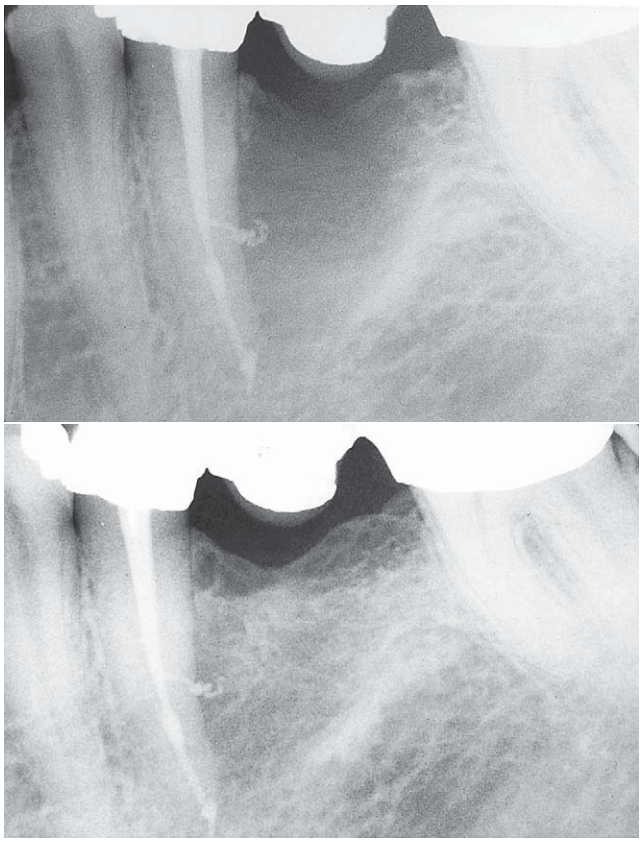


FIG. 4-4 A large, bony defect associated with tooth #20 healed after endodontic therapy. The tooth was nonvital, and no significant periodontal probing depth indicated pulpal disease.

initiating endodontic treatment. 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. 4-6). Whenever possible, restorations should be removed before endodontic treatment.



FIG. 4-5 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.

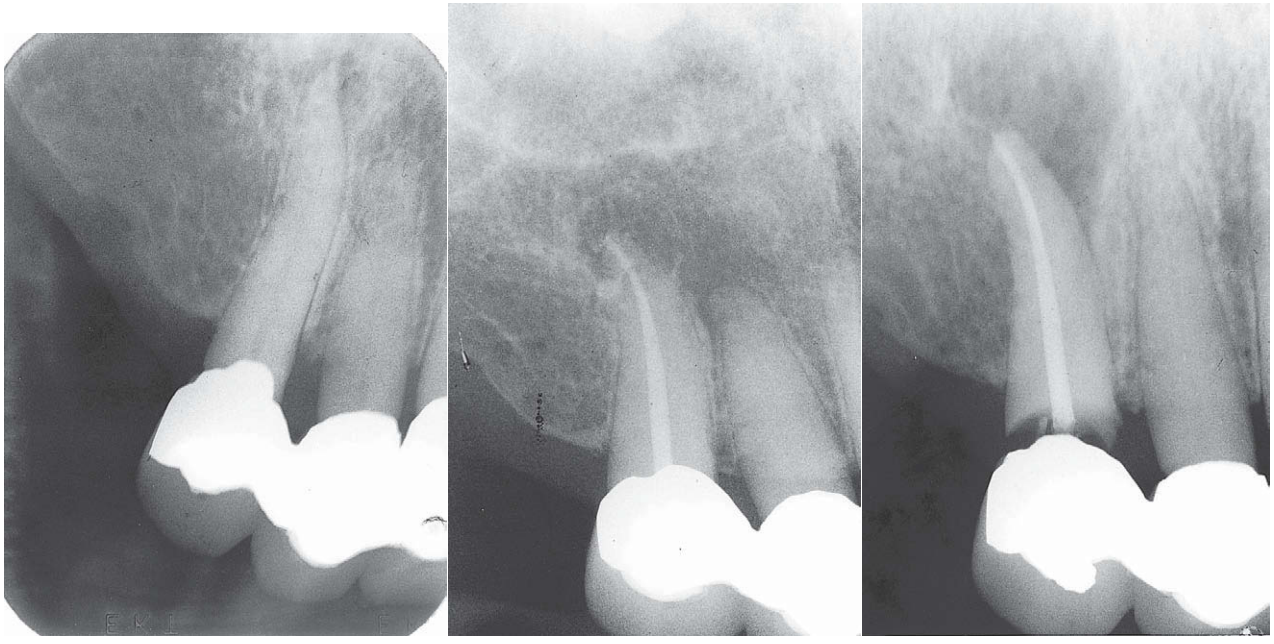


FIG. 4-6 Four years after endodontic therapy, the patient complained of pain and swelling associated with tooth #6. The initial impression was that apical surgery was indicated. However, further radiographs revealed the true cause of the endodontic failure. The initial endodontic access through the crown or caries damaged the coronal seal and recurrent decay followed.

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 pursue endodontics for a tooth with a questionable prognosis or extract and use a single-tooth implant as a replacement. At this time, there are no randomized controlled outcome studies comparing endodontic therapy with single-tooth implants. Numerous studies have evaluated both nonsurgical endodontic therapy[16,50,51,62,70,71] and endosseous dental implants.[2,15,34,39]

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 systematic review of the literature examined the success and failure of nonsurgical endodontic therapy. The authors concluded that four conditions (pretreatment absence of a periapical radiolucency, root filling with no voids, root filling extending to 2 mm within the radiographic apex, and satisfactory coronal restoration) were found to improve the outcome of primary root canal treatment. They went on to note that the results of the review "should be interpreted with caution and cannot be considered to give definitive conclusions because of the retrospective and heterogeneous nature of the data. It does, however, provide strong clues about the factors likely to dominate outcomes and inform the design of future randomized trials."[50]

Another study[16] 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. Absence of pretreatment apical periodontitis, single-rooted teeth, and the absence of intratreatment complications were significant predictors of better outcome in initial endodontic treatment.[1] In 2004, a study considered the outcomes of endodontic treatment on 1,462,936 teeth. More than 97% of teeth were retained after 8 years. It should be noted that most of these cases were completed by general dental clinicians.[62]

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 best available evidence. Most current studies indicate no significant difference in the long-term prognosis between restored endodontically treated teeth and single-tooth implants.[34]

A retrospective cross-sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants compared 196 implant restorations and 196 matched initial nonsurgical root canal–treated teeth. The results suggested that restored endodontically treated teeth and single-tooth implant restorations have similar failure rates, although the implant group showed a longer average and median time to function and a higher incidence of posttreatment complications requiring subsequent treatment intervention.[19]

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, esthetic concerns, cost-to-benefit ratio, systemic factors, potential for adverse effects, and patient preferences. 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."[34]

It seems clear that the patient is 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 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.

Interestingly, some endodontic advanced education programs are now including implant training in their curriculum. This training will enable the endodontist to provide more value to the patient and referring 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.

The scope of modern endodontics has been enhanced by the use of ultrasonics and microscopy as well as improved instruments and new materials. Teeth can be retained today that could not have been treated in the past. Biologically, it is clear that elimination of intraradicular infection is the key to endodontic success.

Vital Case

The acute vital case is best managed by a biologically based approach. Pain in such cases may be due to increased intrapulpal pressure and/or the release of inflammatory mediators and extension of the pathogenic process into the periodontal membrane. The challenge for the clinician is to treat inflamed and well-innervated tissue without causing additional pain. Performing a complete pulpotomy or, if time permits, establishing measurement control and completing a pulpectomy has a high degree of predictability in alleviating pain.[8,33] If a canal has been entered, the clinician should be committed to removing all tissue. Partial instrumentation (i.e., leaving tissue remnants in the canal) may result in increased posttreatment pain.[8,24,58] Teeth should be closed with a temporary filling at the conclusion of the visit in order to prevent bacterial contamination from saliva. After instrumentation of vital teeth with a history of pain and percussion sensitivity, occlusal

reduction has been shown to be important in preventing posttreatment pain.[60]

Nonvital Case

The acute nonvital case represents a microbiologic challenge for the clinician (see also Chapters 9 and 15). A tooth that has had an asymptomatic nonvital pulp for some time may suddenly become acutely symptomatic. The cause of this dramatic change is due to an imbalance in the host–parasite relationship. This can be due to an increase in the virulence of bacteria, a change in the flora, or reduced host defense mechanisms.[8] These changes can be initiated simply by opening the tooth and changing the environment of the bacterial flora. The therapeutic goals in such cases are to reduce the bacterial content in the root canal system and to promote decompression of the periradicular tissues by instrumentation and irrigation of the canal. Calcium hydroxide should be delivered into the root canal system as an intervisit dressing. Teeth should be closed with a cotton pledget in the chamber covered by a temporary seal. Endodontic treatment should be completed as soon as possible in order to prevent continued bacterial penetration into the canal. When a fluctuant swelling exists, incision and drainage may be performed in conjunction with canal instrumentation (Figs. 4-7 and 4-8).

Single-Visit versus Multiple-Visit Treatment

Vital cases are often 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 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 and/or swelling. The second visit allows the clinician to determine the effect of the therapy on the inflamed tissues. Deferring the filling of the canal(s) also leads to a shorter initial visit for the emergency patient. Although some researchers have reported less posttreatment pain in single-visit cases,[24,58] 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.[25] Neither single-visit nor multiple-visit root canal treatment can prevent 100% of short-term and long-term complications.[25] The review found that the incidence of postobturation discomfort was similar in the single- and multiple-visit approaches although analgesic use was significantly less



FIG. 4-7 Incision and drainage should be performed on this fluctuant swelling *(arrow)* in conjunction with canal instrumentation.

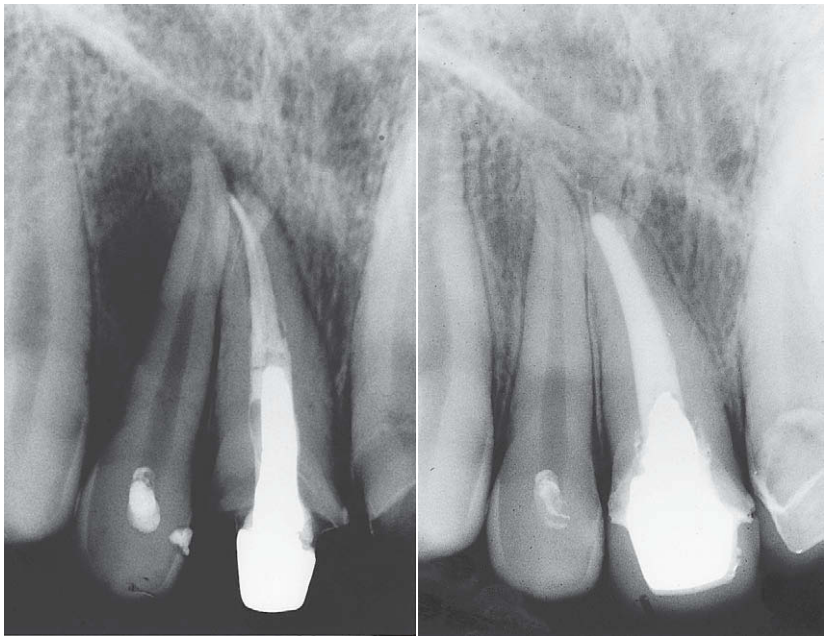


FIG. 4-8 Two years after endodontic therapy of tooth #8, the patient returned with pain and swelling. A clinician mistakenly began endodontic access on tooth #7, without confirming the apparent radiographic diagnosis by vitality testing. Tooth #7 was vital, and tooth #8 was successfully retreated after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

common in patients undergoing multiple-visit root canal treatment.

A systematic review examined the prevalence of posttreatment pain and flare-up among patients undergoing single- and multiple-visit endodontic treatment.[63] The heterogeneity among the included studies was far too great to conduct a meta-analysis and yield meaningful results. The authors 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.

Teeth with nonvital pulps and apical periodontitis are often more complex than vital cases. 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.[68,69] In contrast, other researchers have found no statistically significant difference in success when using the single-visit or multivisit approach to the nonvital tooth with apical periodontitis.[25,29,46,53,54,75] Two randomized controlled trials compared radiographic evidence of periapical healing after root canal treatment of necrotic teeth completed in one or two visits. The authors found no statistically significant difference in healing between the two groups.[46,53]

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. A swelling associated with an abscess, cellulitis, or presence of a stoma represent signs of biologic processes. The biologic significance of these conditions should be considered before determining specific goals for each visit. 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.

Agreement is lacking concerning long-term success rates associated with single-visit and multivisit procedures. Sjögren and associates[68] investigated the influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. Periapical healing was observed 5 years after completion of treatment. They concluded that "Complete periapical healing occurred in 94% of cases that yielded a negative culture. When the samples were positive before root filling, the success rate of treatment was just 68%—a statistically significant difference." They concluded that the objective of eliminating bacteria from the root canal system "cannot be reliably achieved in a one-visit treatment because it is not possible to eradicate all infection from the root canal without the support of an interappointment antimicrobial dressing." However, the findings of Friedman,[29] Weiger and colleagues,[75] and Peters and Wesselink[54] contrast with those of Sjögren and colleagues.[68] Those studies found no statistically significant differences in healing between teeth, with apical periodontitis, treated in one visit and two visits with the inclusion of calcium hydroxide as an intravisit medication.[54,75] Sathorn and colleagues completed a systematic review of single- versus multiple-visit endodontic treatment of teeth with apical periodontitis.[64] They found that, on the basis of current best available evidence, 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.[64] 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.[38]

A study concerning the outcome of initial treatment noted the complexity of treating apical periodontitis.[29] The author commented that "… 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."

Retreatment Case

Retreatment cases offer a particular set of challenges to the clinician, and this topic is covered extensively in Chapter 26. Important questions to be considered before retreatment include the following:

- Why did the treatment fail?
- 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. 4-9 to 4-12). Retreatment cases may require surgical endodontics in combination with nonsurgical retreatment. Referral to a specialist is often helpful when planning treatment for complex cases.

Immature Teeth

Primary and immature permanent teeth may have 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 self-esteem of the patient. See Chapters 16 and 24 for further information.

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. 4-13). The inability to isolate a tooth is

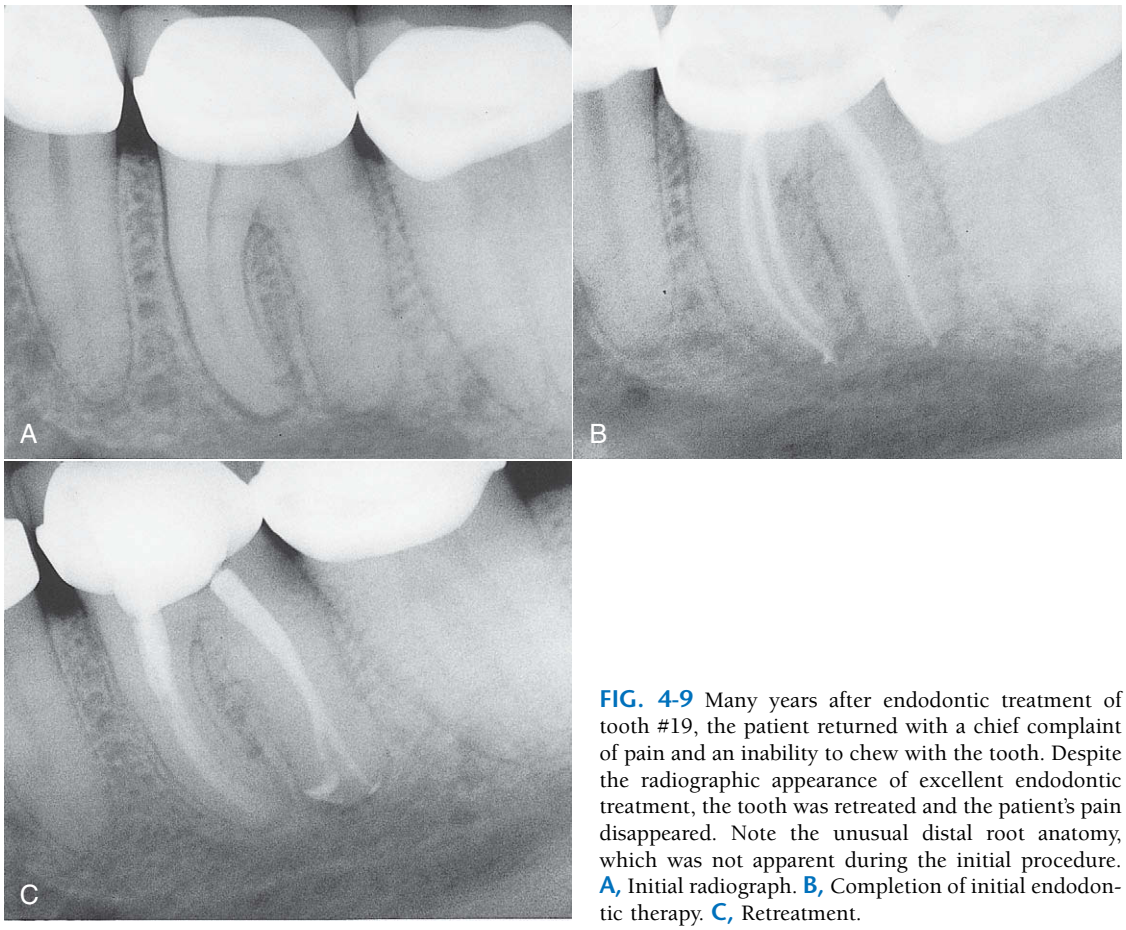


FIG. 4-9 Many years after endodontic treatment of tooth #19, the patient returned with a chief complaint of pain and an inability to chew with the tooth. Despite the radiographic appearance of excellent endodontic treatment, the tooth was retreated and the patient's pain disappeared. Note the unusual distal root anatomy, which was not apparent during the initial procedure. **A,** Initial radiograph. **B,** Completion of initial endodontic therapy. **C,** Retreatment.

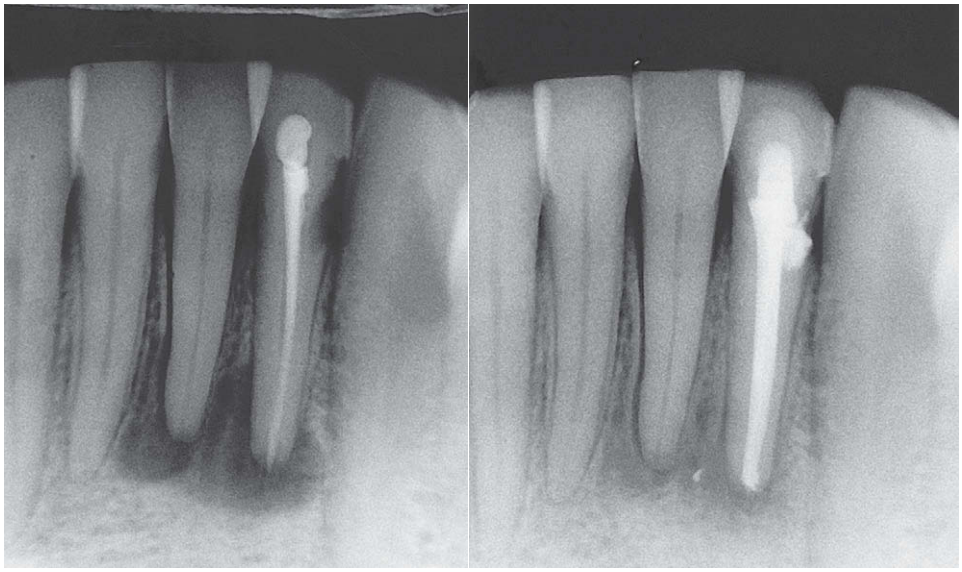


FIG. 4-10 Initial radiograph was misleading and implicated tooth #23 and tooth #24. Pulp testing indicated a vital pulp in tooth #24, and it was not treated. Retreatment of #23 resulted in healing of the periradicular lesion. *(Courtesy Dr. Leon Schertzer.)*

also a problem and may result in bacterial contamination of the root canal system. Extra roots and canals pose a particular anatomic challenge that radiographs do not always reveal (Fig. 4-14). A bite wing radiograph is useful in providing an accurate image of the pulp chambers of posterior teeth. Retreatment cases offer particular mechanical challenges and are discussed in detail in Chapter 25. Ledges, perforations, or a post may be present, all of which complicate treatment and alter the prognosis. 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.

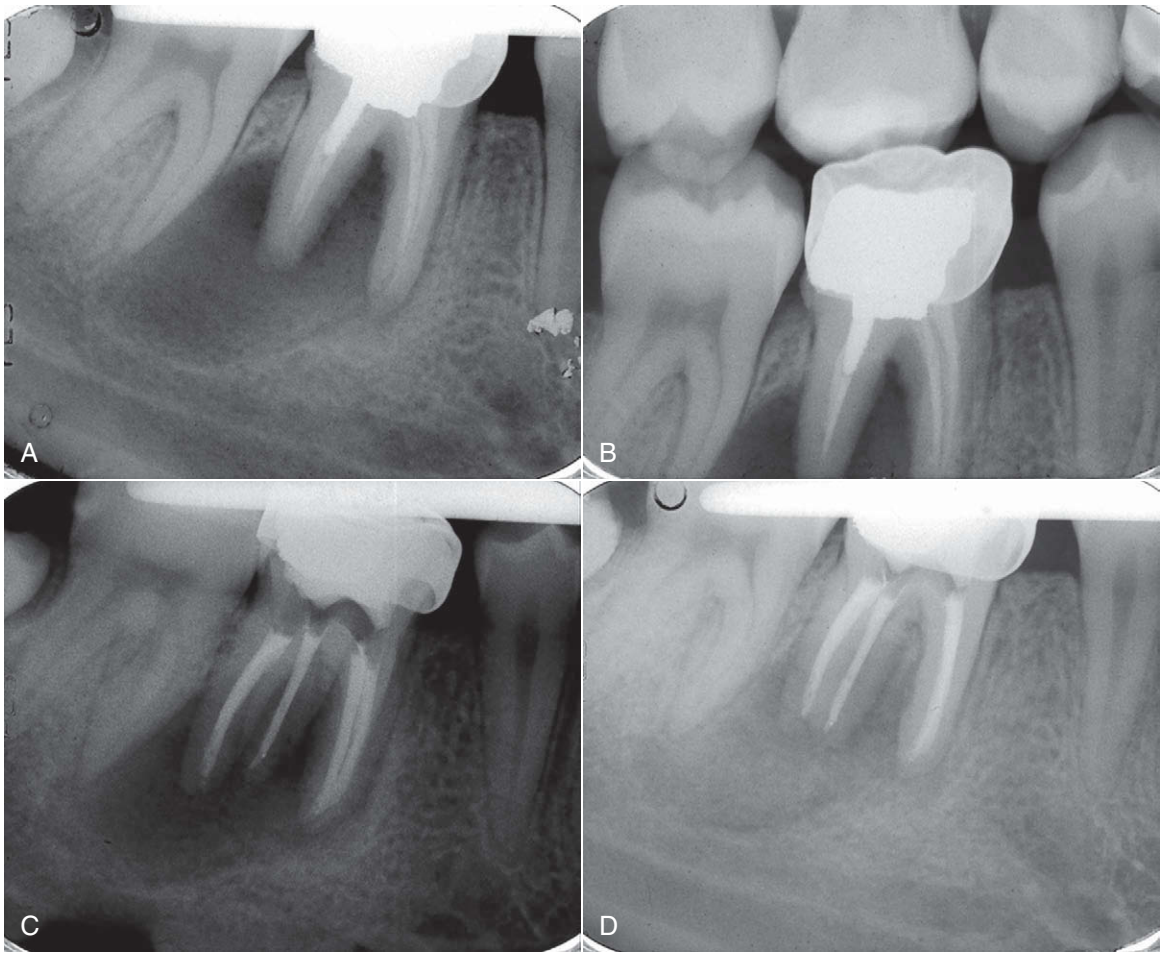


FIG. 4-11 Nonsurgical retreatment of tooth #30. An additional root was located and treated. **A,** Note inadequate endodontic treatment and large periapical lesion. **B,** Bite wing radiograph. **C,** Retreatment after post removal. **D,** Eighteen-month recall radiograph indicates periapical healing.

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 http://www.aae.org/dentalpro/CaseAssmtReferral.html) (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 of 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.

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.

Anxiety

Anxiety presents a problem at many levels of dental care (see also online Chapter 26). Avoidance of dental treatment due to anxiety appears to be associated with significant deterioration of oral and dental health.[79] Even at the diagnostic stage, severe anxiety may confuse the process.[21] 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.[37,79] Also,

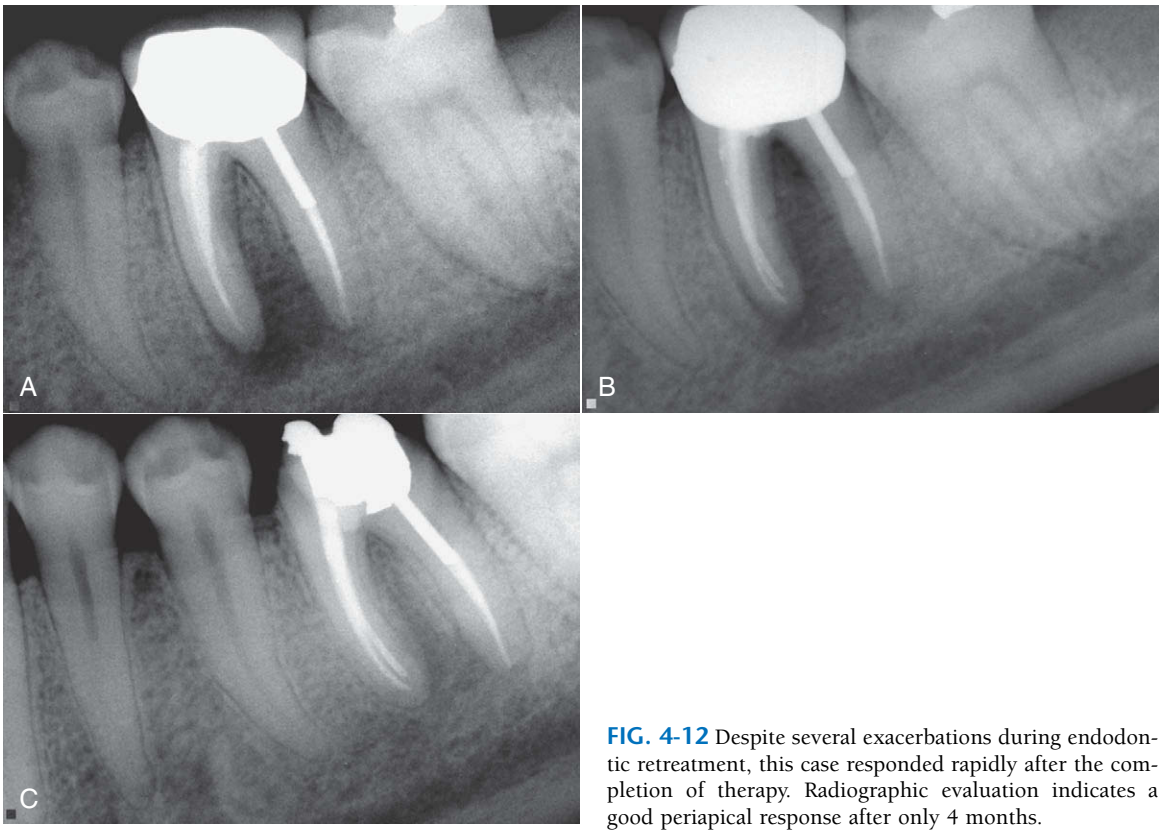


FIG. 4-12 Despite several exacerbations during endodontic retreatment, this case responded rapidly after the completion of therapy. Radiographic evaluation indicates a good periapical response after only 4 months.

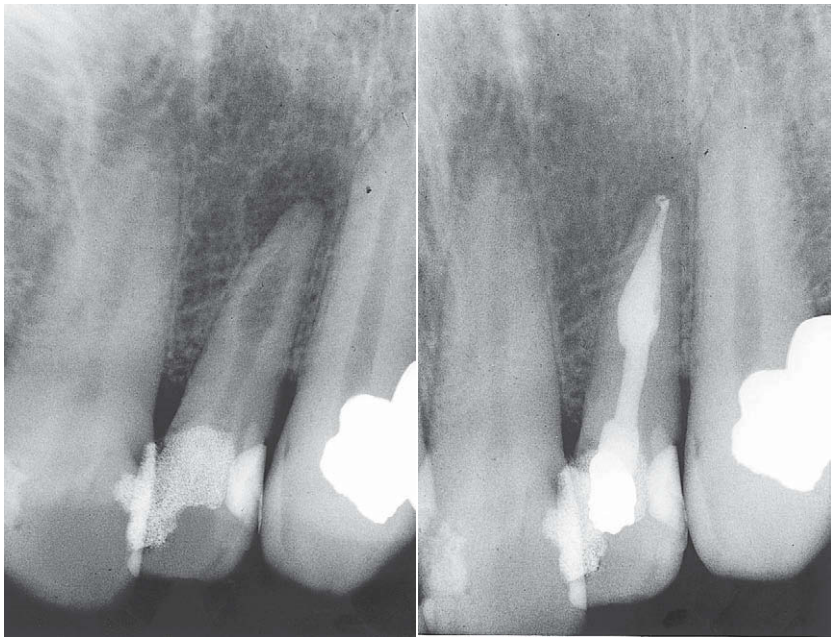


FIG. 4-13 Resorptive defects can be successfully treated. Early intervention, before there is perforation of the root, increases the chance of success. *(Courtesy Dr. Leon Schertzer.)*

highly anxious patients appear to be more sensitive to pain.[22,36] High levels of anxiety have been found to negatively affect clinical procedures including local anesthesia.[48]

It has been demonstrated that dental anxiety and expectation of pain had a profound effect on a patient's ability to understand information provided.[23] A person's cognitive ability to process information is significantly affected by stress.[23] 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.

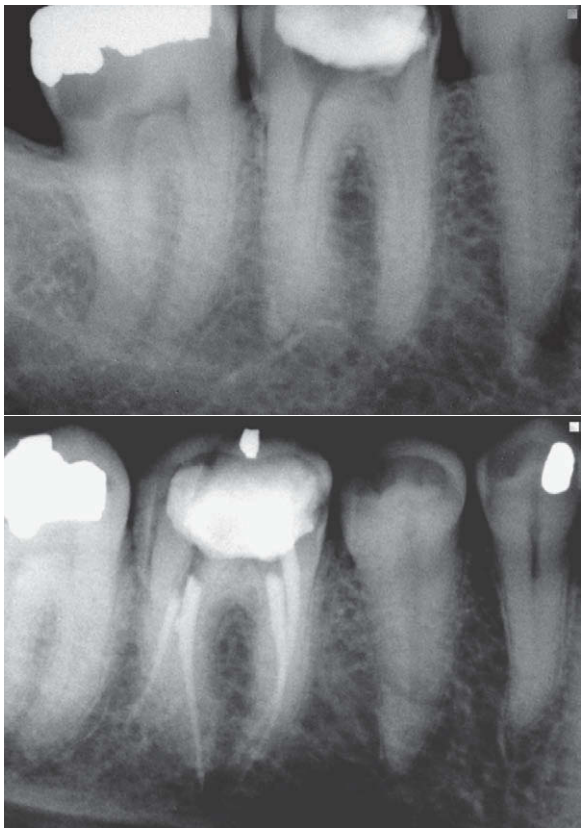


FIG. 4-14 The presence of curved roots and multiple canals is a complicating factor.

Unfortunately, the impact that a high level of anxiety can have on patient's cognition, local anesthesia, and intra- and posttreatment experiences is not always recognized. Existing research has focused primarily on the effect of pretreatment information on reducing anxiety and stress during surgery.[23] It was determined, using an anxiety virtual analog scale (AVAS), that higher AVAS scores were correlated with a greater number of injections needed to eliminate pain and that the higher the score, the less likely it was that pain would be eliminated during endodontic treatment.[18]

In a landmark medical study it was found that pretreatment discussion of surgical treatments and associated discomfort reduced by 50% the need for posttreatment morphine and reduced the time to discharge.[20] More than 200 studies indicate that pre-emptive behavioral intervention decreases anxiety before and after surgery, reduces posttreatment pain intensity and intake of analgesics, and accelerates recovery.[13] 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.[49] Written 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.

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 in order 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. It is wise to schedule patients who require mandibular block anesthesia to arrive 15 to 20 minutes before their treatment visit. This avoids the frustration of "losing treatment time" while the anesthetic agent becomes fully effective (see also Chapter 20).

Appointments to fill nonvital cases should be scheduled approximately 1 week after instrumentation in order to maximize the antimicrobial effect of the intracanal dressing when calcium hydroxide is used.[8,68,69] Acute (pain and/or swelling) nonvital cases should be seen every 24 to 48 hours in order 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

5

Preparation for Treatment

GERALD N. GLICKMAN and MERLYN W. VOGT

CHAPTER OUTLINE

PREPARATION OF THE OPERATORY
- Infection Control
- Health Insurance Portability and Accountability Act

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- Aids in Rubber Dam Placement
- Problem Solving in Tooth Isolation

Before nonsurgical root canal treatment can begin, a number of issues related to treatment considerations, along with clinician and patient needs, must be addressed. These include proper infection control and occupational safety procedures for the entire health care team and treatment environment; appropriate communication with the patient, including case presentation and informed consent; premedication, if necessary, followed by effective administration of local anesthesia; a quality radiographic or digital image survey; and thorough isolation of the treatment site.

PREPARATION OF THE OPERATORY

Infection Control

Because all dental health care personnel (DHCP), including those not directly involved in patient care, are at risk for exposure to a host of infectious organisms (e.g., influenza; upper respiratory disease; tuberculosis [TB]; herpes; hepatitis B, C, and D; and acquired immunodeficiency syndrome [AIDS]), effective infection control procedures must be used to minimize the risk of cross-contamination in the work environment.[21-23,25,54,71,73] These infection control procedures must not only protect patients and the dental team from contracting infections during dental procedures, but also reduce the number of microorganisms in the immediate dental environment to the lowest level possible.

As the AIDS epidemic continues to expand, it has been established that the potential for occupational transmission of human immunodeficiency virus (HIV) and other fluid-borne pathogens can be minimized by enforcing infection control policies specifically designed to reduce exposure to blood and other infected body fluids.[12,13,16,54,73] Because HIV has been shown to be fragile and easily destroyed by heat or chemical disinfectants, the highly resistant nature of the hepatitis B virus, along with its high blood titers, makes it a good model to evaluate infection control practices in order to prevent transmission of a large number of other pathogens via blood or saliva. Because all infected patients are not readily identifiable through the routine medical history and many are asymptomatic, the American Dental Association (ADA, Chicago, IL) recommends that each patient be considered potentially infectious; this means that the same strict infection control policies or universal precautions apply to all patients.[22,73] In addition, the Occupational Safety and Health Administration (OSHA) of the U.S. Department of Labor, in conjunction with both the ADA and the Centers for Disease Control and Prevention (CDC, Atlanta, GA), has issued detailed guidelines on hazard and safety control in the dental setting.[2,3,11-14,41,73] In 1992, laws specifically regulating exposure to blood-borne disease became effective through the OSHA standard 29 CFR 1910.1030, Occupational Exposure to Bloodborne Pathogens.[25] Primarily designed to protect any employee who could be "reasonably anticipated" to have contact with blood or any other potentially infectious materials, the standard encompasses a combination of engineering and work practice controls, as well as recommendations for the use of equipment and protective clothing, training, signs and labels, and hepatitis B vaccinations. It also authorizes OSHA to conduct inspections and impose financial penalties for failure to comply with specific regulations.[25]

In 1993 the ADA, CDC, and OSHA recommended or mandated specific infection control guidelines for dental practice.

In 2003 the CDC updated these guidelines to help further reduce the potential for disease transmission from patient to DHCP, from DHCP to patient, and from patient to patient. The document now emphasizes the use of standard precautions rather than universal precautions for the prevention of exposure to and transmission of not only blood-borne pathogens but also other pathogens encountered in oral health settings.[15] The major recommendations including updates are as follows:

1. The ADA and CDC recommend that all DHCP with potential occupational exposure to blood or other potentially infectious material be vaccinated against hepatitis B. The OSHA standard requires that employers make the hepatitis B vaccine available to occupationally exposed employees, at the employer's expense, within 10 working days of assignment to tasks that may result in exposure. An employee who refuses the vaccine must sign a declination form that uses specific language approved by OSHA. In addition, postexposure follow-up and evaluation must be made available to all employees who have had an exposure incident. In addition, all DHCP must be educated about tuberculosis; baseline tuberculin skin tests (TSTs) must be done for all DHCP who might have contact with persons suspected or confirmed to have active TB.[15]
2. A thorough patient medical history, which includes specific questions about hepatitis, AIDS, current illnesses, unintentional weight loss, lymphadenopathy, and oral soft-tissue lesions, must be taken and updated at subsequent appointments. All patents should be screened for latex allergies, and they should be referred if an allergy is suspected.[15]
3. Dental personnel must wear protective attire and use proper barrier techniques. The standard requires the employer to ensure that employees use personal protective equipment (PPE) and that such protection is provided at no cost to the employee.
   a. Disposable latex or vinyl gloves must be worn when contact with body fluids or mucous membranes is anticipated or when touching potentially contaminated surfaces; they may not be washed for reuse. OSHA requires that gloves be replaced after each patient contact, when torn, or when punctured. If their integrity is not compromised, sturdy, unlined utility gloves for cleaning instruments and surfaces may be decontaminated for reuse. Polyethylene gloves may be worn over treatment gloves to prevent contamination of objects, such as drawers, light handles, or charts.
   b. Hands, wrists, and lower forearms must be washed with either a nonantimicrobial soap or antimicrobial soap and water when hands are visibly soiled, after bare-handed touching of inanimate objects likely to be contaminated, before and after treating each patient, before donning gloves, and immediately after removing gloves.[15] An antimicrobial surgical hand scrub should be used for surgical procedures before donning sterile gloves. The standard requires that any body area that has contact with a potentially infectious material, including saliva, must be washed immediately after contact. It is strongly recommended that hand lotions that do not affect glove integrity be used to prevent dryness associated with hand-washing.[15] Sinks should have electronic, elbow-, foot-, or knee-action faucet controls for asepsis and ease of function. Employers must provide washing facilities (including an eye wash) that are readily accessible to employees.
   c. Masks and protective eyewear with solid side shields or chin-length face shields are required when splashes or sprays of potentially infectious materials are anticipated and during all instrument and environmental cleanup activities. When a face mask is removed, it should be handled by the elastic or cloth strings, not by the mask itself. It is further suggested that the patient wear protective eyewear.
   d. Protective clothing, either reusable or disposable, must be worn when clothing or skin is likely to be exposed to body fluids, and it should be changed when visibly soiled or penetrated by fluids. OSHA's requirements for protective clothing (i.e., gowns, aprons, laboratory coats, and clinic jackets) are difficult to interpret, because the "type and characteristics [thereof] will depend upon the task and degree of exposure anticipated." The ADA and CDC recommend long-sleeved uniforms. However, according to OSHA, long sleeves are required only if significant splashing of blood or body fluids to the arms or forearms is expected. Thus endodontic surgery would likely warrant long-sleeved garments. OSHA requires that the protective garments not be worn outside the work area. The standard prohibits employees from taking contaminated laundry home to be washed; it must be washed at the office or by an outside laundry service. Contaminated laundry must be placed in an impervious laundry bag that is colored red and labeled "BIOHAZARD." Although OSHA does not regulate nonprotective clothing (e.g., scrubs), such clothing should be handled like protective clothing once fluids have penetrated it.
   e. Patients' clothing should be protected from splatter and caustic materials, such as sodium hypochlorite, with waist-length plastic coverings overlaid with disposable patient bibs.
   f. High-volume evacuation greatly reduces the number of bacteria in dental aerosols and should be employed when using the high-speed handpiece, water spray, or ultrasonics.
   g. Use of the dental dam as a protective barrier is mandatory for nonsurgical root canal treatment, and failure to use this barrier is considered to be below standard care.[16,17,29]
4. OSHA regulates only contaminated sharps. Contaminated *disposable* sharps (e.g., syringes, needles, and scalpel blades) and contaminated *reusable* sharps (e.g., endodontic files) must be placed into separate, leak-proof, closable, puncture-resistant containers. These containers should be colored red or labeled "BIOHAZARD," and they should be marked with the biohazard symbol. The standard states that before decontamination (i.e., sterilization), contaminated reusable sharps must not be stored or processed in a way that requires employees to use their hands to reach into the containers to retrieve the instruments. The OSHA ruling allows picking up sharp instruments by hand only after they are decontaminated.[21,41,54]
   a. The clinician should take the following steps when handling contaminated endodontic files: With tweezers, place used files in glass beaker containing a nonphenolic

disinfectant and detergent holding solution. At the end of the day, discard the solution and rinse with tap water. Add ultrasonic cleaning solution, and place the beaker in the ultrasonic bath until thoroughly clean (i.e., 5 to 15 min). Discard the ultrasonic solution, and rinse with tap water. Pour the contents of the beaker onto a clean towel, and use tweezers to place the clean files into a metal box for sterilization. Files with any visible debris should be separately sterilized. Once sterilized, these files can be picked up by hand and debrided, using 2 × 2 inch sponges. Once cleaned, files should be returned to the metal box for sterilization. (As indicated in Chapter 8, all files should be regarded as disposable.)

b. In general, the standard prohibits bending or recapping of anesthesia needles. However, during endodontic treatment, reinjection of the same patient is often necessary, so recapping is essential. Recapping with a one-handed scoop method and using a mechanical device are the only permissible techniques; needles must not be recapped by using both hands or directing the needle point toward any part of the body.[15] Shearing or breaking of contaminated needles should never be permitted.

5. Countertops and operatory surfaces, such as light handles, radiograph unit heads, chair switches, and any other surface likely to become contaminated with potentially infectious materials, can be either covered or disinfected. Protective coverings (e.g., clear plastic wrap, special plastic sleeves, and aluminum foil) can be used. These coverings should be changed between patients and when they become contaminated. OSHA mandates, however, that work surfaces be decontaminated or recovered or both at the end of each work shift and immediately after overt contamination. The coverings should be removed by gloved personnel, discarded, and then replaced with clean coverings after gloves are removed. Alternatively, countertops and operatory surfaces can be wiped with absorbent toweling to remove extraneous organic material and then sprayed with an Environmental Protection Agency (EPA)–registered and ADA-accepted tuberculocidal disinfectant (e.g., a 1 : 10 dilution of sodium hypochlorite, iodophor, or synthetic phenol). With the advent of endodontic microscopy, appropriate barriers should be placed on the handles and controls of the microscope, or the entire unit can be draped to prevent cross-contamination. If the system becomes contaminated, disinfection should be performed according to the microscope manufacturer's guidelines.
6. Contaminated radiographic film packets must be handled in a way that prevents cross-contamination. Contamination of the film (when it is removed from the packet) and subsequent contamination of the processing equipment can be prevented either by properly handling the film as it is removed from the contaminated packet or by preventing the contamination of the packet during use.[70] After exposure, "overgloves" should be placed over contaminated gloves to prevent cross-contamination of processing equipment or darkroom surfaces.[40] For darkroom procedures, films should be carefully manipulated out of their holders and dropped onto a disinfected surface or into a clean cup without being touched. Once the film has been removed, gloves should be removed and discarded; the film can then be processed. All contaminated film envelopes must be accumulated (after film removal) in a strategically positioned impervious bag and disposed of properly. For daylight loaders, exposed film packets should be placed into a paper cup; gloves should be discarded and hands washed. Next, a new pair of gloves should be donned, and the paper cup with films and an empty cup should be placed into the chamber. Using gloved hands, the chamber should be entered and packets should be carefully opened, allowing the film to drop onto a clean surface in the chamber. Empty film packets should then be placed into an empty cup and gloves removed and discarded in the cup; films can then be processed.[41] Plastic envelopes, such as the ClinAsept barriers (Kodak Dental Systems/Carestream Health, Rochester, NY), have simplified the handling of contaminated, exposed films by protecting films from contact with saliva and blood during exposure. Once a film is exposed, the barrier envelope is easily opened and the film can be dropped into a paper cup or onto a clean area before processing. The barrier-protected film, however, should be wiped with an EPA-approved disinfectant as an added precaution against contamination during opening.[70] For digital radiography sensors, U.S. Food and Drug Administration (FDA)–cleared barriers should be used.
7. In conjunction with the previously mentioned guidelines for infection control, a mouth rinse of 0.12% chlorhexidine gluconate, such as Peridex (3M, St. Paul, Minn), is suggested before treatment. This rinse will minimize the number of microbes in the mouth and, consequently, in any splatter or aerosols generated during treatment.[21,54,73] It should be noted, however, that such mouth rinses have not proved to reduce the incidence of clinical infections.
8. After treatment, all instruments and burs must be cleaned and sterilized by sterilizers monitored with biologic indicators. Cassettes, packs, or trays should be rewrapped in original wrap, and individually packaged instruments should be placed in a covered container. Air and water syringes must be flushed, cleaned, and sterilized. Antiretraction valves (i.e., one-way flow check valves) should be installed to prevent fluid aspiration and to reduce the risk of transfer of potentially infective material. Heavy-duty rubber gloves must be worn during cleanup. The ADA and CDC recommend that all dental handpieces and "prophy" angles be heat sterilized between patients.[3,21,41,54] Before sterilization, all handpieces should be wiped with an EPA-registered disinfectant. In addition, high-speed handpieces should be run for a minimum of 30 seconds to discharge water and air, with spray directed into a high-volume evacuation system. Dental unit water lines should be periodically flushed with water or a 1:10 dilution of 5.25% sodium hypochlorite (NaOCl) to reduce biofilm formation. All regulated infectious waste must be immediately disposed of in containers that meet specific criteria. Disposal must be in accordance with applicable federal, state, and local regulations.

In 1987 the infection control decision-making process was transferred to the U.S. government through OSHA.[73] The ongoing goal of OSHA is to establish a routine and practical program of enforcing infection control standards (based on published CDC guidelines) to ensure the health and safety of all members of the dental health team. According to OSHA,

clinicians must classify personnel and tasks in the dental practice according to levels of risk of exposure and must establish "standard operating procedures" to protect the patient and staff from infection transmission.[41,54,73] OSHA requires the clinician to provide infection control training for all employees and to maintain records of such training; all hazardous substances that employees are exposed to on the job must be properly labeled; and a written hazard communications program with manufacturers' material safety data sheets (MSDSs) is needed for all hazardous substances. With the enactment of the OSHA Occupational Exposure to Bloodborne Pathogens standard, employers must make exposure determinations and develop an exposure control plan. As mentioned previously, the rule encompasses a number of critical areas (e.g., universal precautions, engineering and work practice controls, employee training, and specific recordkeeping) designed to protect employees from exposure to blood-borne pathogens, particularly HIV and hepatitis B virus. Although the OSHA standard was written principally to protect employees, it does not encompass all the infection control practices recommended by the ADA and CDC to protect clinicians and patients.

In 1994 the CDC issued its position statement on the prevention of transmission of TB in dental settings. The statement suggested that elective dental treatments for patients suspected of having TB be deferred until it has been confirmed that they are free of the disease. The CDC also stated that emergency care for a patient with TB should be provided only in facilities with appropriate respirators, negative-pressure treatment areas, and other respiratory engineering controls.[11] Compliance with OSHA regulations and with evolving infection control policies of the ADA and CDC will help provide a safer workplace for the entire dental treatment team.[21,41,54,73]

Health Insurance Portability and Accountability Act

The Health Insurance Portability and Accountability Act (HIPAA) was passed into law on August 21, 1996; the original purpose of this law was to make health care insurance "portable" so that an individual's insurance could be passed from one employer to another employer. Because of additions to help fight fraud and abuse, ensure the security of medical records, protect the privacy of a patient's confidential health information, and a worthwhile goal to replace paper transactions with electronic transactions, the HIPAA standard has become one of the most widespread and complicated regulations ever passed.[33] On April 14, 2003, HIPAA launched a whole new era of medical/dental care because it is now the responsibility of the clinician–employer to make sure employees conduct themselves in a manner that supports the provisions of the standard. Two of the rules under HIPAA that affect clinicians are the Transactions Rule and the Privacy Rule. For the purposes of this chapter, only the Privacy Rule is discussed (see Chapter 27 [online] for other information about HIPAA).

The Privacy Rule controls what is called *protected health information (PHI)*. PHI is individually identifiable health information that is held or released by a practice regardless of how it is communicated (oral, paper, or electronic). The entire Privacy Rule was created to ensure that a patient's PHI is not used or disclosed to those individuals or parties who do not need to know such information. In general, the Privacy Rule requires dental practices to take reasonable steps to limit use of disclosure of PHI to the *minimum necessary* to achieve an intended purpose. This rule does not apply to uses or disclosures made to the patient, another provider (for treatment purposes), or governmental authorities.

The clinician must employ or appoint a privacy officer to help bring the office into compliance. The privacy officer oversees all ongoing activities related to maintaining the privacy of PHI consistent with state law. It is the officer's duty to report to the clinician–employer the status of the office's compliance efforts and to develop appropriate documents such as patient request forms, acknowledgment forms, and patient authorization forms. When such documents are ready for disposal, it is the privacy officer's responsibility to see that documents containing PHI are shredded and destroyed.[33]

The Notice of Privacy Practices is a kind of "reverse" informed consent for the patient.[33] Any clinician who has a direct treatment relationship with an individual must provide that individual with a Notice of Privacy Practices. Further, the dental practice must provide a copy of the current notice to anyone who asks for it (whether a patient or not). The notice must explain how PHI may be used or disclosed by the practice, the patient's privacy rights as to PHI, and the practice's obligations as to PHI. The final Privacy Rule only requires the clinician who has a direct treatment relationship with a patient to make a "good faith" effort to obtain a patient's written "acknowledgment" that he or she has received the Notice of Privacy Practices.[33] The patient cannot be "made" to sign such a statement. In addition, the patient can file a complaint against the practice for alleged violations of privacy policies. Thus, under HIPAA, the patient has the following rights: the right to receive a copy of the practice's Notice of Privacy Practices, the right to request a restriction on uses and disclosures, the right to request receipt of confidential communications by alternative means or at alternative locations, the right to request access to inspect and copy the dental record, the right to request an amendment to the dental record, and the right to request an accounting of disclosures. With the exception of the first right, these rights are all followed by the phrase "to request." The practice does not have to grant all requests; as with any law, there are exceptions to the rules. However, if the clinician agrees to a request, any violation of the request is a violation of the privacy regulations. These issues are covered extensively in Chapter 11 and online Chapter 27.

PATIENT PREPARATION

Treatment Planning

Aside from emergencies that require immediate attention, endodontic treatment usually occurs early in the total treatment plan for the patient. Therefore any asymptomatic but irreversible pulpal and periradicular problems are managed before they become symptomatic and more difficult to handle. The most important rationale for the high priority of endodontics is to ensure that a sound, healthy foundation exists before further treatment is attempted. A stable root system within sound periradicular and periodontal tissues is paramount to the placement of any definitive restorations.

Regardless of the specifics of the case, the clinician is responsible for explaining the nature of the treatment and informing the patient of any risks, the prognosis, and other pertinent facts. Because of bad publicity and hearsay, root

canal treatment is reputed to be a horrifying experience. Consequently, some patients may be reluctant, anxious, or even fearful of undergoing root canal treatment. Thus it is imperative that the clinician educate the patient before treatment (i.e., "informing before performing")[17] to allay concerns and minimize misconceptions.

Good clinician and patient relationships are built on effective communication. Sufficient evidence suggests that clinicians who establish warm, caring relationships with their patients through effective case presentation are perceived more favorably. These clinicians also have a more positive impact on the patient's anxiety, knowledge, and compliance than those who maintain impersonal, noncommunicative relationships.[20] Most patients experience increased anxiety while in the dental chair. However, a simple but informative case presentation that answers all questions reduces patient anxiety and solidifies the patient's trust in the clinician.

Case Presentation

The ADA and the American Association of Endodontists (AAE, Chicago, IL) publish brochures (e.g., *Endodontics: Your Guide to Endodontic Treatment*[1]) to help patients understand root canal treatment. Valuable educational aids of this nature should be made available to the patient either before or immediately after case presentation. This supportive information addresses the most frequently asked questions concerning endodontic treatment. The following sections review these questions. Accompanying each question is an example of an explanation that patients should be able to understand. In addition, the clinician will find it useful to have a set of illustrations or drawings to help explain the procedure. The AAE offers specialized case presentation forms with carbonless copies for record keeping and patient use.

What Is Endodontic (Root Canal) Treatment?

Endodontics is the specialty in dentistry concerned with the prevention, diagnosis, and treatment of diseases or injuries to the dental pulp. The pulp, which some people call "the nerve," is the soft tissue inside the tooth that contains the nerves and blood vessels and is responsible for tooth development. Root canal treatment is a safe and effective means of saving teeth that otherwise would be lost.

What Causes the Pulp to Die or Become Diseased?

When a pulp is injured or diseased and unable to repair itself, it becomes inflamed and eventually dies. The most frequent causes of pulp death are extensive caries, deep restorations, trauma (e.g., severe blow to a tooth), cracks in teeth, and periodontal or gum disease. When a pulp is exposed to bacteria from caries or saliva that has leaked into the pulp system, infection can occur inside the tooth and, if left untreated, can cause infection to build up at the tip of the root, forming an abscess. Eventually the bone supporting the tooth will be destroyed, and pain and swelling will often accompany the infection. Without endodontic treatment, the tooth will eventually have to be removed.

What Are the Symptoms of a Diseased Pulp?

Symptoms may range from momentary to prolonged, mild to severe pain on exposure to hot or cold or on chewing or biting. In some cases the condition may produce no symptoms at all. The patient should be informed that the radiographic examination may not demonstrate abnormal conditions of the tooth. The clinician should also make it clear that sometimes in the absence of pain, radiographic evidence of pulpal or periradicular disease or both may be present.

What Is the Success Rate of Root Canal Therapy?

Endodontics is one of the few procedures in dentistry that has a predictable prognosis if treatment is performed properly. Studies indicate that root canal treatment is usually 90% to 95% successful. Those in the failure group may still be amenable to retreatment or surgical treatment to save the tooth, although no treatment's success can be guaranteed. In addition, patients must understand that the prognosis may vary depending on the specifics of each case and that, without good oral hygiene and a sound restoration after endodontic therapy, there may be an increased chance for failure. The need for periodic follow-up must be addressed to assess the long-term status of the tooth and periradicular tissues.

Will the Endodontically Treated Tooth Discolor After Treatment?

If the treatment is done correctly, discoloration seldom occurs. In the unlikely event that discoloration should occur, bleaching with heat or chemicals can be used to restore the natural color and shade of teeth. Some endodontically treated teeth appear discolored because they have been restored with tooth-colored restorations that have become stained or with amalgam restorations that leach silver ions. In these instances the restorations may be replaced, but often the placement of crowns or veneers is indicated.

What Are the Alternatives to Root Canal Treatment?

The only alternative is to extract the tooth, which often leads to shifting and crowding of surrounding teeth and subsequent loss of chewing efficiency. The patient should understand that often, extraction is the easy way out and, depending on the case, may prove to be more costly for the patient in the long run. The patient always reserves the right to do nothing about the problem, provided the clinician has explained the associated risks of this decision.

Will the Tooth Need a Crown or "Cap" After the Treatment?

If there is no previously existing crown, the need for a crown or "cap" depends on the amount of sound tooth structure remaining after endodontic treatment. In addition, the need for a crown or cap depends on the type of tooth and the amount of chewing force to which the tooth will be subjected. Loss of tooth structure significantly weakens the tooth and renders it more susceptible to fracture; as a result, it may be necessary to protect what is left with a restoration, such as a crown. Significant loss of tooth structure with a concomitant loss of retentive areas for coronal buildups may necessitate the placement of a metallic, resin, or ceramic post in a canal to retain the buildup material (see Fig. 5-1, *I* and *J*) (for further information about these issues, see Chapter 22).

What Does Root Canal Treatment Involve?

Treatment may require one to three appointments, depending on the diagnosis, the number of roots, and the complexity of

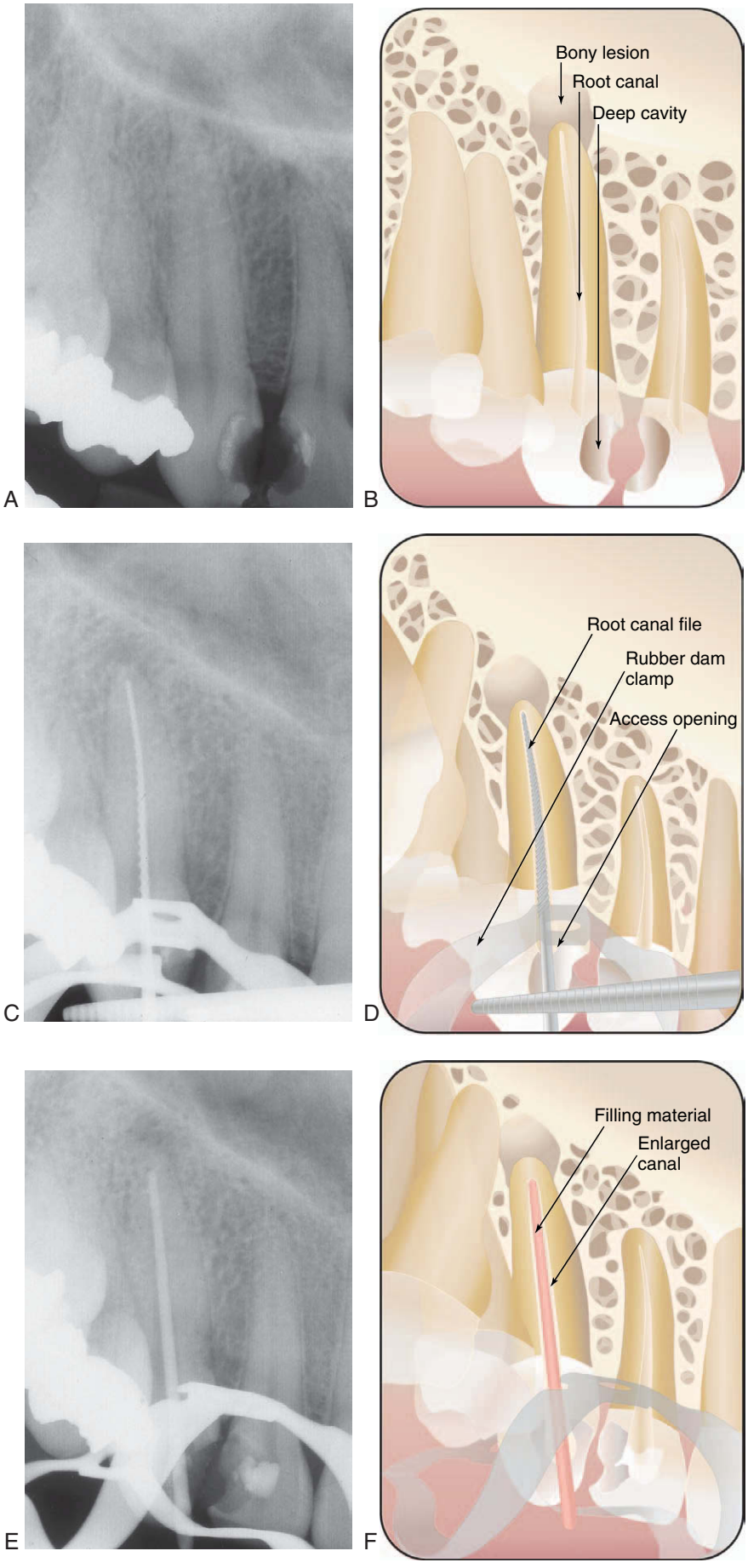


FIG. 5-1 Series of radiographs and illustrations demonstrating root canal treatment and restoration of a maxillary canine. **A-B,** Maxillary canine with periradicular lesion of endodontic origin. **C-D,** Endodontic file corresponding to length of canal; isolation with rubber dam throughout procedure. **E-F,** Endodontic filling material placed after cleaning and shaping of canal.

Continued

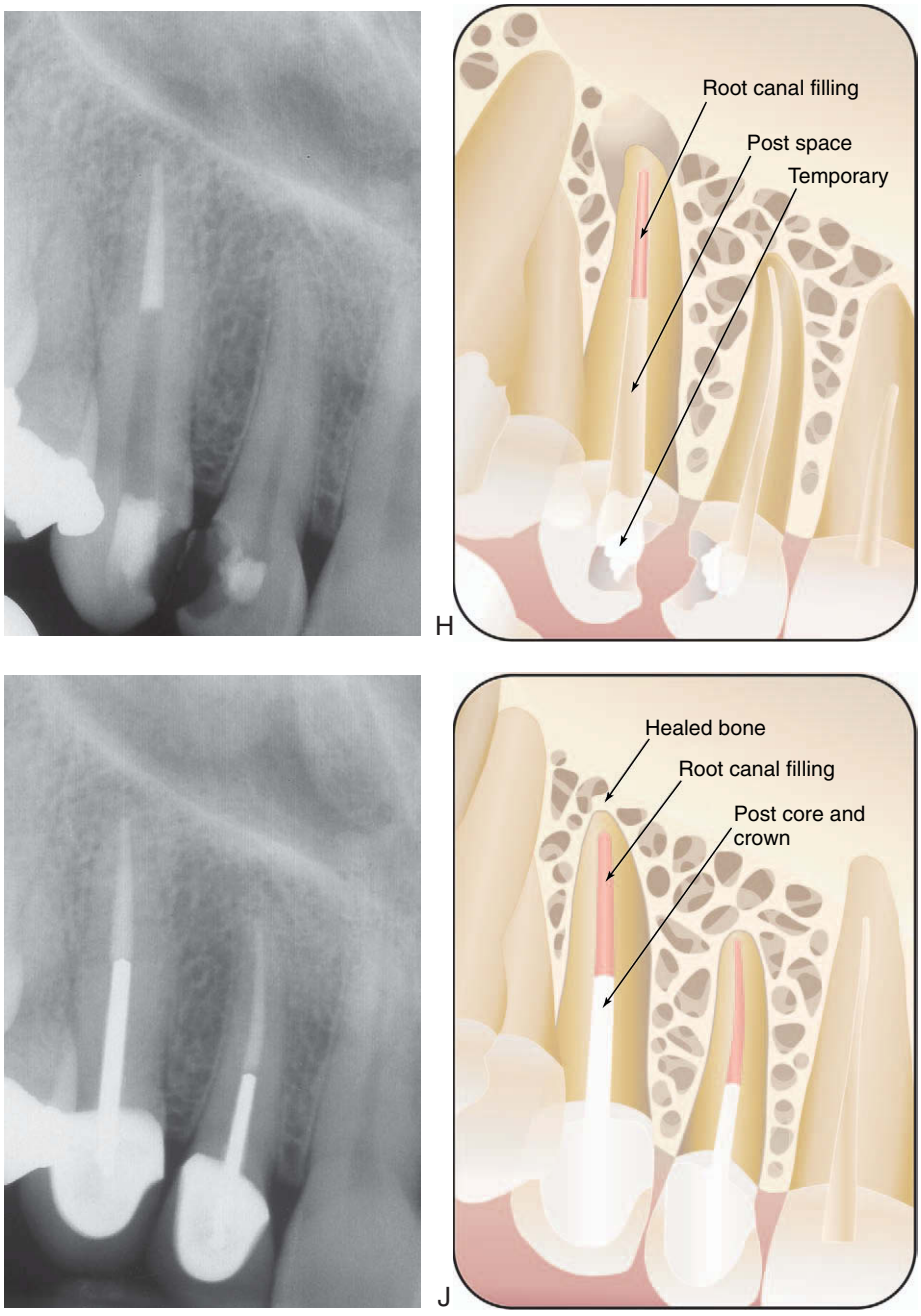


FIG. 5-1, cont'd G-H, Canal system filled and post space made. **I-J,** One-year follow-up shows completed restoration and healed periradicular bone.

the case. During these appointments the clinician removes the injured or diseased pulp tissue. The root canals are cleaned, enlarged, and sealed to prevent recontamination of the root canal system. The following steps (Fig. 5-1) describe the technical aspects of the treatment (illustrations, diagrams, radiographs, and digital images should be used as aids to the presentation):

1. Local anesthesia is usually administered.
2. The tooth is isolated with a rubber dam to prevent contamination from saliva and to protect the patient. This procedure is followed at each subsequent visit.
3. An opening is made through the top of the tooth to gain entrance to the root canal system.
4. The pulp tissue is painlessly removed with special instruments called *files*.
5. Periodic radiographs ("x-rays") or digital images must be taken to ensure that these instruments correspond to the exact length of the root so that the entire tissue can be removed. Electronic apex locators can be used as adjuncts to help determine or verify lengths.
6. The root canal is cleaned, enlarged, and shaped so that it can be filled or sealed properly.
7. Sometimes medications are placed in the opening to prevent infection between appointments.
8. If treatment cannot be completed in one visit, a temporary filling is placed in the crown opening between appointments.

9. At the final appointment the canal is sealed to safeguard it from further contamination.
10. Permanent restoration of the tooth is accomplished after completion of the root canal treatment.

Some additional points should be conveyed to the patient after treatment. The patient should not be given the impression that there will be no pain after the treatment.[65] In most cases, the mild discomfort the patient may experience is transitory and can usually be treated with an over-the-counter anti-inflammatory or analgesic agent, for example, an ibuprofen-containing compound. In fact, prophylactic administration of these drugs before the patient leaves the office will help reduce posttreatment discomfort by achieving therapeutic blood levels of analgesic before the local anesthesia wears off (see Chapter 20). In certain cases, simply handing the patient a written prescription for a stronger analgesic, "just in case," conveys a feeling of empathy and caring toward the patient and strengthens the doctor–patient relationship.

If the clinician wishes to refer the patient to an endodontist for treatment, skillful words of encouragement and explanation will convey the caring and concern behind this recommendation. Many patients already feel comfortable with their clinicians and are fearful of seeing someone new. In addition, they may not understand why a general clinician chooses not to do the root canal treatment. The referring clinician can help by carefully explaining the complex nature of the case and why it would be in the patient's best interests to visit the endodontist, who is specially trained to handle complex cases.[65]

Informed Consent

Much controversy surrounds the legal aspects of informed consent. The current thinking in the courts is that for consent to be valid, it must be freely given; that all terms must be presented in language that the patient understands; and that the consent must be "informed."[17,18,57] For consent to be informed, the following conditions must be included in the presentation to the patient:

- The procedure and prognosis must be described. (This includes prognosis in the absence of treatment.)
- Alternatives to the recommended treatment must be presented, along with their respective prognoses.
- Foreseeable risks and material risks must be described.
- Patients must have the opportunity to have questions answered.[57]

It is probably in the best interests of the clinician–patient relationship to have the patient sign a valid informed consent form. With the continuous rise in dental litigation, it is important to realize that "no amount of documentation is too much and no amount of detail is too little."[57] (For further information on this subject, see Chapters 11 and 27.)

Radiation Safety

A critical portion of the endodontic case presentation and informed consent involves educating the patient about the requirement for radiographs as part of the treatment. The clinician must communicate to the patient that the benefits of radiographs far outweigh the risks of receiving small doses of ionizing radiation, as long as techniques and necessary precautions are properly executed.[2] Although levels of radiation in endodontic radiography range from only 1/100 to 1/1000 of the levels needed to sustain injury,[53,64] it is still best to keep ionizing radiation to a minimum for the protection of both the patient and the dental delivery team.

Two simple analogies can be used to help the patient understand the small risk associated with dental radiographs. A patient would have to receive 25 complete full mouth series (i.e., 450 exposures) within a very short time to significantly increase the risk of skin cancer.[53] One full mouth survey (i.e., 20 Ektaspeed [E]–speed films with rectangular collimation) has been found to deliver less than one half the amount of radiation of a single chest film and less than 1% of the amount of a barium study of the intestines.[70] Nevertheless, the principles of ALARA (as low as reasonably achievable), which are techniques used to reduce radiation exposure, should be followed as closely as possible to minimize the amount of radiation that both patient and treatment team receive. ALARA also implies the possibility that no matter how small the radiation dose, there still may be some deleterious effects.[53,70]

Principles of ALARA

In endodontic radiography, fast (i.e., sensitive) speed film should be selected.[53] Although E-speed film allows for a reduction of approximately 50% of the radiation exposure required for D speed,[26] findings in observer preference studies have been mixed as to the quality, clarity, and diagnostic capability of E film compared with D film. Processing of E-speed film is also more sensitive.[26,27,39] Specialized radiographic systems[56,70] (using direct or indirect digital intraoral radiography) involve the digitization of ionizing radiation and use considerably smaller amounts of radiation to produce an image that is available immediately after exposure (see the later section, Digital Radiographic Techniques).

Meticulous radiographic technique helps reduce the number of retakes and obviates further exposure. Film-holding devices (discussed later in the chapter) along with correct film and tube head positioning are essential to maintain film stability and produce radiographs of diagnostic quality.[53,70] A quality assurance program for film processing should also be set up to ensure that films are properly processed.[53,70]

Dental units should be operated using at least 70 kVp (peak kilovoltage). The lower the kilovoltage, the higher the patient's skin dose. Optimally, 90 kVp should be used. Units operating at 70 kVp or higher must have a filtration equivalent of 2.5 mm of aluminum to remove the extraneous low-energy x-rays before the patient absorbs them.[53,70]

Collimation also reduces exposure level. Collimation is, essentially, the restriction of the x-ray beam size by means of a lead diaphragm so that the beam does not exceed 2.75 inches (7 cm) at the patient's skin surface. Open-ended, circular, or rectangular lead-lined cylinders, known as *position-indicating devices (PIDs)*, help direct the beam to the target. However, the universal rectangular cylinder also collimates the x-ray beam by decreasing beam size even more, reducing the area of skin surface exposed to x-radiation and reducing radiation burden by approximately 50% (Fig. 5-2). These PIDs, or cones, should be at least 12 to 16 inches long, because the shorter (i.e., 8-in.) cones that provide shorter source-to-film distances permit more divergence of the beam and more exposure to the patient.[53,70] Pointed cones, illegal in some states, should not be used because of the increased amount of scatter radiation they produce.



FIG. 5-2 Universal collimator snaps on the aiming ring to extend the extra protection of a rectangular collimator to round, open-ended cones. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*

On the basis of the December 2003 National Council on Radiation Protection and Measurements (NCRP) Report No. 145 on new dental x-ray guidelines (http://www.ncrponline.org/Publications/145press.html), leaded aprons are no longer required as long as other recommendations stated in the report are strictly followed; the report also states that thyroid collars are mandatory for children and should be provided for adults.[52] When exposing films, the clinician should stand behind a barrier. Plaster, cinderblock, and at least 2.5 inches of drywall provide the necessary protection from the radiation produced by dental units. If there is no barrier the clinician should stand in an area of minimal scatter radiation: at least 6 feet away from the patient and in an area that lies between 90 and 135 degrees to the beam.[53,70] All dental personnel who might be exposed to occupational x-radiation should wear film badges for recording exposure. If the concept of ALARA is strictly adhered to, no member of the dental team should receive doses close to the maximal permissible dose (MPD) (i.e., 50 millisieverts [mSv]/yr/whole body).[70]

For "declared" pregnant workers, the Nuclear Regulatory Commission limits the radiation dose to the fetus to 0.5 mSv during the gestation period. It is important to note that the MPD is specified as occupational exposure and should not be confused with exposure that patients receive as a result of radiographic procedures. Although no state-recommended maximal patient exposures exist, anyone who administers ionizing radiation is responsible for consulting the respective state's bureau of radiation control to obtain information about current laws. Nonetheless, every effort should be made to keep the radiation dose to all individuals as low as possible and to avoid any unnecessary radiation exposure.

Premedication With Antibiotics

Prophylactic coverage with antibiotics or antiinfectives is indicated only for the highest risk patients who are susceptible to infective endocarditis after bacteremia. The use of prophylactic antibiotics in these patients prevents blood-borne microorganisms from lodging on shunts and prostheses or from multiplying within a depressed system.[44,55,59,71] In 2007 there was a major revision of the 1997 guidelines.[71] The current recommendations are based on a collective body of evidence published in numerous studies over the past two decades. Studies now indicate that infective endocarditis is more likely to result from frequent random bacteremias of routine daily activities than from an isolated dental procedure. The 2007 guidelines recommend that only patients at highest risk are to receive antibiotic prophylaxis. Those cardiac conditions associated with the highest risk of an adverse outcome from endocarditis include patients with prosthetic cardiac valves, a previous history of infective endocarditis, certain congenital heart diseases, and cardiac transplantation patients who develop cardiac valvulopathies. It is also thought that antibiotic prophylaxis prevents only a small number of cases of infective endocarditis and that the risk of antibiotic-associated adverse events may actually exceed the benefit of prophylaxis.[5,66,67,71] The current recommendations of the American Heart Association (AHA, Dallas, TX) place increased emphasis on maintaining good oral hygiene in an effort to decrease the overall frequency of bacteremias that are associated with daily activities.

Conditions in the moderate-risk category include most other congenital cardiac malformations, rheumatic heart disease, hypertrophic cardiomyopathy, and mitral valve prolapse with valvular regurgitation or thickened leaflets or both. Patients with cardiac conditions in the moderate-risk category will no longer receive antibiotic prophylaxis according to the 2007 guidelines. Conditions in the negligible-risk category (i.e., no greater risk than for the general population) and for which prophylaxis is not recommended include previous coronary artery bypass graft surgery, mitral valve prolapse without valvular regurgitation, previous rheumatic fever without valvular dysfunction, and cardiac pacemakers (both intravascular and epicardial). On the basis of the recommendations in the 2007 guidelines, fewer patients will need to be covered with a prophylactic antibiotic regimen. This may raise concerns with patients who in the past have taken antibiotics for cardiac conditions and are now told that prophylaxis is no longer necessary. Therefore the clinician should be well versed in the new AHA guidelines and be prepared to discuss the revisions with inquiring patients.

The AHA has developed a standard prophylactic antibiotic regimen for patients at risk and a set of alternative regimens for those unable to take oral medications, for those who are allergic to the standard antibiotics, and for those who are not candidates for the standard regimen. The recommended standard prophylactic regimen for all dental, oral, and upper respiratory tract procedures is currently amoxicillin. This is because amoxicillin is better absorbed by the gastrointestinal tract and provides higher and more sustained serum levels than penicillin.

The major modification of the 1997 regimen was that the posttreatment dose had been eliminated; the rationale for this was that amoxicillin has a sufficiently high plasma level for an adequate time to prevent endocarditis. Erythromycin was also eliminated as a recommended drug in the penicillin-allergic patient because of the high incidence of gastrointestinal upset and the variability of the pharmokinetics of the various erythromycin preparations. The 2007 guidelines have placed less of an emphasis on invasive dental treatments as a causal factor for infective endocarditis and greater emphasis on factors associated with hygiene and oral health. The official AHA recommendations for prophylactic antibiotic regimens do not specify all clinical situations for which patients may be at risk. Thus

it is the responsibility of the clinician to exercise judgment or consult the patient's physician before giving treatment. With respect to premedication for dental patients with total joint replacements, there has been considerable controversy as to whether such patients require routine prophylaxis. In 1997 the ADA and American Academy of Orthopaedic Surgeons [AAOS, Rosemont, IL]) drafted an advisory statement[4] on antimicrobial premedication for dental patients with total joint replacements. This advisory statement was followed in 2003 by another advisory statement, which essentially is in agreement with the 1997 guidelines.[5] The joint organizations recognized that there was no agreed-on scientific evidence to support the contention that antibiotic prophylaxis is necessary to prevent metastatic infection in patients with total joint prostheses. They also agreed that the analogy between late prosthetic joint infections and infective endocarditis was invalid, because the anatomy, blood supply, types of microorganisms involved, and mechanisms of infection all differ. The ADA and AAOS concluded that antibiotic prophylaxis is not indicated for dental patients with pins, plates, and screws, nor is it routinely indicated for most dental patients with total joint replacements.

However, because limited evidence exists that some dental procedures are high-risk procedures (e.g., extractions, intraligamentary local anesthesia, endodontic surgery, and endodontic instrumentation "beyond the apex") and that some medically compromised patients with total joint replacements (e.g., those with insulin-dependent diabetes; inflammatory arthropathies, such as rheumatoid arthritis; immunosuppression; hemophilia; and previous prosthetic joint infections) may be at higher risk for hematogenous infections, an antibiotic regimen should be considered. Prophylaxis should also be recommended during the first 2 years after joint replacement. The antibiotic regimen consists of cephalexin, Cefazolin, or amoxicillin (2 g *per os,* 1 hour before procedure). For those allergic to penicillin or cephalosporin, the recommended antibiotic is clindamycin (600 mg *per os,* 1 hour before procedure). It is recommended that patients who are not allergic to penicillin but who are unable to take oral medications should receive cefazolin (1 g) or ampicillin (2 g), administered intramuscularly or intravenously, 1 hour before the dental procedure. For patients allergic to penicillin and unable to take oral medications, the recommendation is clindamycin (600 mg administered intramuscularly or intravenously, 1 hour before the dental procedure). Similar to the AHA guidelines, follow-up doses are no longer recommended. The advisory statement represents only recommended guidelines and is not intended as a standard of care, because it is impossible to make recommendations for all clinical situations in which late infection might occur in total joint prostheses. Clinicians must exercise their own clinical judgment in determining whether to premedicate a patient.

Bisphosphonate-Associated Osteonecrosis of the Jaws

Bisphosphonates are an important class of drugs that are commonly used to reduce the morbidity associated with osteoclast-mediated bone diseases. Bisphosphonates inhibit bone resorption, improving bone densities by a reduction in bone turnover. They are extremely useful in the treatment of osteoporosis, Paget's disease, hypercalcemia associated with certain malignancies such as multiple myeloma, and breast and prostate cancers. Bisphosphonates have a high affinity for bone and once incorporated into the skeleton can persist for many years; the estimated half-life for alendronate is known to be approximately 12 years.[43] Nine bisphosphonate drugs are currently approved for clinical use by the FDA. Seven of these formulations are oral bisphosphonates, with only pamidronate and zoledronate being administered intravenously. The intravenously administered forms have higher bioavailability and potencies than the oral forms.

In 2003 case reports describing necrosis of the bones of the jaw in long-term bisphosphonate patients began appearing in the literature.[10,48,50] At present, an exposed area of necrotic bone of the jaw that has been present for at least 8 weeks in a patient undergoing long-term bisphosphonate therapy, without a prior history of radiation treatment, has been defined as bisphosphonate-associated osteonecrosis of the jaw (ONJ). Most cases of ONJ have been diagnosed after a traumatic dental procedure such as a tooth extraction; however, there are also reports of spontaneous cases.[51] Most cases of ONJ have been associated with the intravenously administered bisphosphonates, but there have been reports of its occurrence with oral bisphosphonates.[61] Clinically, ONJ presents as a painful, soft tissue swelling and infection of the jaw, mobility of the teeth, and exposure of bone but at times the disease can be asymptomatic. Most intraoral sites affected are those areas of bone that are covered by thin and friable mucosa; especially vulnerable are the torus mandibularis and torus palatinus.[72] Treatment of ONJ is problematic and at present there are no evidence-based guidelines. There have been reports of limited responses to surgical debridement, segmental resections, antibiotic therapy, and hyperbaric oxygen therapy, and therefore the emphasis must be placed on prevention. Patients taking bisphosphonates must be informed of the possibility of potential oral complications and the clinician must be vigilant in assessing the known risk factors in these patients. Preventive procedures including caries control and appropriate endodontic treatment should be considered for high-risk patients to reduce the risk of developing ONJ, as its treatment is unpredictable. A multidisciplinary approach to managing patients with ONJ is advised, with the clinician working in close association with the oncologist, oral surgeon, and other specialists. Although the overall incidence appears to be low despite the vast number of patients who take these drugs, it is important to recognize that ONJ can be a life-changing, debilitating condition for those afflicted. As our present knowledge regarding bisphosphonate-associated osteonecrosis continues to evolve, the clinician is encouraged to stay informed and up to date about this relatively new pathologic entity (Table 5-1).

Antianxiety Regimens

Because patients often have been misinformed about root canal treatment, some may understandably experience increased anxiety about undergoing the procedure. Fortunately, however, the vast majority of patients are able to tolerate their anxiety, control their behavior, and allow treatment to proceed with few problems. Appropriate behavioral approaches can be used to manage most anxious dental patients. Retrospective studies[20] concerning dental anxiety have clearly demonstrated that explaining each procedure before beginning root canal treatment can effectively reduce a patient's anxiety. The clinician can also reduce patient anxiety by giving specific information

TABLE 5-1

U.S. Food and Drug Administration–Approved Bisphosphonates

Generic Name	Brand Name	Route of Administration	Manufacturer	FDA Approval Date	Common Side Effects
Alendronate sodium	Fosamax	Oral	Merck	1995	Gastrointestinal intolerance, headache, hypocalcemia, hypophosphatemia
Aldrendronate sodium plus vitamin D	Fosamax plus D	Oral	Merck	2005	Gastrointestinal intolerance, headache, bone pain, dizziness
Etidronate disodium	Didronel	Oral	Procter & Gamble	1977	Gastrointestinal intolerance, headache, bone pain, fever, increased serum creatinine
Ibandronate sodium	Boniva	Oral/intravenous	Roche and GlaxoSmithKline	2003	Gastrointestinal intolerance, headache, bone pain, hypercholesterolemia, dizziness
Pamidronate disodium	Aredia	Intravenous	Novartis	1991	Fever, fatigue, nausea, hypophosphatemia, hypokalemia, infusion site reaction
Risedronate sodium	Actonel	Oral	Procter & Gamble	1998	Gastrointestinal intolerance, headache, pain, rash, urinary tract infection, arthralgia, back pain, hypertension
Risedronate sodium plus calcium carbonate	Actonel with calcium	Oral	Sanofi-Aventis	1997	Gastrointestinal intolerance, dizziness, paresthesia, chest pain, edema, rhinitis, sinusitis
Zoledronic acid	Zometa	Intravenous	Novartis	2001	Gastrointestinal intolerance, bone pain, fever, fatigue

during treatment, by advising the patient about possible minor discomfort, and by explaining how that discomfort can be controlled. Verbal support, reassurance, and personal warmth also help to ease patient anxiety during root canal treatment. Many of these measures can be taken during the case presentation.

Although the clinician's hope and desire may not cure a patient's fear of root canal treatment, each clinician should realize that all anxious patients are not alike; therefore each patient should be managed individually. If behavioral solutions are not feasible or effective in a particular case, pharmacologic approaches to managing patient anxiety may be exercised. Selection of such pharmacotherapeutic techniques must involve a careful assessment of the relative risks and benefits of the alternative approaches. All pharmacologic treatment regimens include the need for good local anesthetic technique. For the management of mild to moderate anxiety states, these regimens range from nitrous oxide plus oxygen sedation to oral sedation to intravenous or conscious sedation (for further information on these issues, see Chapter 19 and online Chapter 26).

Pain Control by Pretreatment Administration of NSAIDs

During root canal cleaning and shaping, extrusion of small amounts of pulp tissue remnants and dentin filings is likely to occur. Often this extrusion results in additional inflammation and some posttreatment discomfort. Prophylactic administration of a nonsteroidal antiinflammatory drug (NSAID), such as 200 to 400 mg of ibuprofen 30 to 60 minutes before the procedure, has been shown to reduce or prevent posttreatment dental pain[36] (see Chapter 19 for further information).

Pain Control by Local Anesthesia

It is paramount to obtain a high level of pain control when performing root canal treatment; in no other specialty is this task as challenging or as demanding. The clinician must strive for a "painless" local anesthetic injection technique, with relatively rapid onset of analgesia (see Chapter 20).

PREPARATION OF RADIOGRAPHS

Radiographs are essential to all phases of endodontic therapy. They contribute information important for the diagnosis and the various treatment phases and help evaluate the success or failure of treatment. Because root canal treatment relies on accurate radiographs, it is necessary to master radiographic techniques to achieve films of maximal diagnostic quality. Such mastery minimizes retaking of films and avoids additional exposure of patients. Expertise in radiographic interpretation is essential for recognizing deviations from the norm and for understanding the limitations associated with endodontic radiography.

Functions, Requirements, and Limitations of the Radiograph in Endodontics

The primary radiograph used in endodontics is the periapical radiograph. In diagnosis this film is used to identify abnormal conditions in the pulp and periradicular tissues. It is also used

to determine the number of roots and canals, location of canals, and root curvatures. Because the radiograph is a two-dimensional image (a major limitation), it is often advantageous to expose additional radiographs at different horizontal or vertical angulations when treating multicanal and multirooted teeth. Exposing additional radiographs is also helpful when treating teeth with severe root curvature. These supplemental radiographs enhance visualization and evaluation of the three-dimensional structure of the tooth.

Technically, for endodontic purposes, a radiograph should depict the tooth in the center of the films. Consistent film placement in this manner will minimize interpretation errors, because the center of the films contains the least amount of distortion. In addition, at least 3 mm of bone must be visible beyond the apex of the tooth. Failure to capture this bony area may result in misdiagnosis, improper interpretation of the apical extent of a root, or incorrect determination of file lengths for canal cleaning and shaping. Finally, the image on the film must be as anatomically correct as possible. Image shape distortion caused by elongation or foreshortening may lead to interpretive errors during diagnosis and treatment.[30,70]

The bite-wing radiograph may be useful as a supplemental film. This film normally has less image distortion because of its parallel placement, and it provides critical information about the anatomic crown of the tooth. This information includes the anatomic extent of the pulp chamber, the existence of pulp stones or calcifications, recurrent caries, the depth of existing restorations, and any evidence of previous pulp therapy.[65] The bite-wing also indicates the relationship of remaining tooth structure relative to the crestal height of bone. Thus it can aid in determining the restorability of the tooth.

In addition to their diagnostic value, high-quality radiographs are mandatory during the treatment phase. Technique is even more critical, however, because working radiographs must be exposed while the rubber dam system is in place. Visibility is reduced, and the bows of the clamp often restrict precise film positioning. During treatment, periradicular radiographs are used to determine canal working lengths; the location of superimposed objects, canals, and anatomic landmarks (by altering cone angulations); biomechanical instrumentation; and master cone adaptation (see Fig. 5-1, *C* to *F*). After completion of the root canal procedure, a radiograph should be exposed to determine the quality of the root canal filling or obturation. Follow-up radiographs exposed at similar angulations enhance assessment of the success or failure of treatment (see Fig. 5-1, *I* and *J*).

The astute clinician can perceive that precise radiographic interpretation is undoubtedly one of the most valuable sources of information for endodontic diagnosis and treatment, but the radiograph is only an adjunctive tool and can be misleading. Information gleaned from proper inspection of the radiograph is not always absolute and must always be integrated with information gathered from a thorough medical and dental history, clinical examination, and various pulp-testing procedures (see Chapters 1 and 4).

Use of the radiograph depends on an understanding of its limitations *and* its advantages. The advantages are obvious: the radiograph allows a privileged look inside the jaw. The information it furnishes is essential and cannot be obtained from any other source, and its value is not diminished by a critical appraisal of its limitations.

One major limitation of radiographs is their inability to detect bone destruction or pathosis when it is limited to the cancellous bone. Studies[62] have proved that radiolucencies usually do not appear unless there is external or internal erosion of the cortical plate. This factor must be considered in evaluating teeth that become symptomatic but show no radiographic changes. In most cases, root structure anatomically approaches cortical bone and, if the plate is especially thin, radiolucent lesions may be visible before there is significant destruction of the cortical plate. Nevertheless, inflammation and resorption affecting the cortical plates must still be sufficiently extensive before a lesion can be seen on a radiograph.

Principles of Endodontic Radiography

Film Placement and Cone Angulation

For endodontic purposes, the paralleling technique produces the most accurate periradicular radiograph. Also known as the *long-cone* or *right-angle* technique, it produces improved images. The film is placed parallel to the long axis of the teeth, and the central beam is directed at right angles to the film and aligned through the root apex (Fig. 5-3, *A* and *B*). To achieve this parallel orientation it is often necessary to position the film away from the tooth, toward the middle of the oral cavity, especially when the rubber dam clamp is in position.[70] The long-cone (i.e., 16 to 20 in) aiming device is used in the paralleling technique to increase the focal spot-to-object distance. This has the effect of directing only the most central and parallel rays of the beam to the film and teeth, reducing size distortion.[49,53,70] This technique permits a more accurate reproduction of the tooth's dimensions, thus enhancing a determination of the tooth's length and relationship to surrounding anatomic structures.[30] In addition, the paralleling technique reduces the possibility of superimposing the zygomatic processes over the apices of maxillary molars, which often occurs with more angulated films, such as those produced by means of the bisecting-angle technique (Fig. 5-3, *C* and *D*). If properly used, the paralleling technique will provide the clinician with films with the least distortion, minimal superimposition, and utmost clarity.

Variations in size and shape of the oral structures (e.g., shallow palatal vault, tori, or extremely long roots) or gagging by the patient can make true parallel placement of the film impossible. To compensate for difficult placement, the film can be positioned so that it diverges as much as 20 degrees from the long axis of the tooth, with minimal longitudinal distortion. With maxillary molars, any increase in vertical angulation increases the chances of superimposing the zygomatic process over the buccal roots. A vertical angle of not more than 15 degrees should usually project the zygomatic process superiorly and away from the molar roots. To help achieve this, a modified paralleling technique[19] that increases vertical angulation by 10 to 20 degrees can be used. Although this orientation introduces a small degree of foreshortening, it increases periradicular definition in this troublesome maxillary posterior region. The Snapex system (DENTSPLY Rinn, Elgin, IL), a film holder and aiming device originally designed for the bisecting-angle technique, has been altered for the modified paralleling technique.[19] In conjunction with this technique, a distal angulated radiograph (i.e., a 10- to 20-degree horizontal shift of the cone from the distal, with the beam directed toward the mesial)

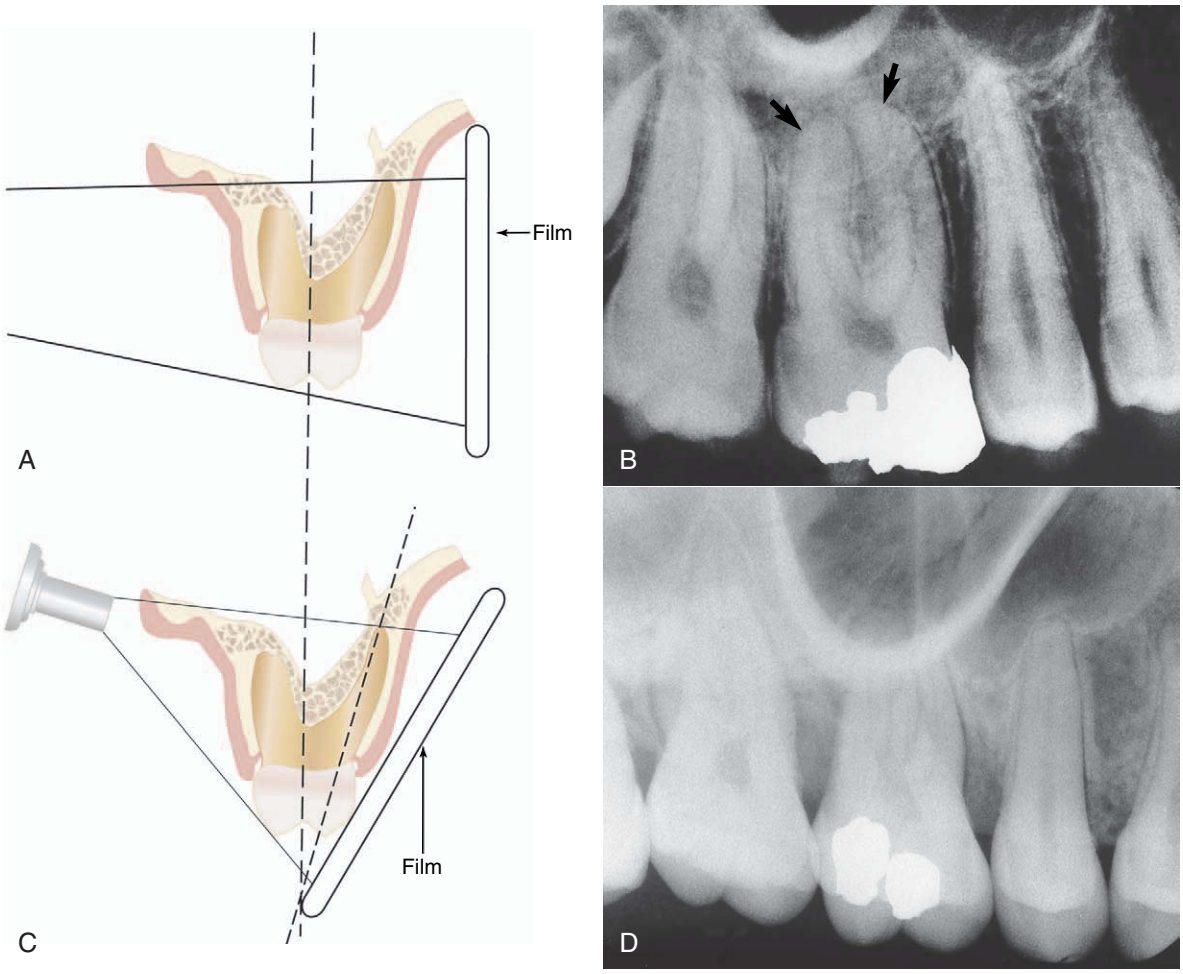


FIG. 5-3 A, Paralleling, or right-angle, technique. **B,** Projection of the zygomatic process above the buccal root apices by the right-angle technique, allowing visualization of the apices *(arrows)*. **C,** Bisecting-angle technique. **D,** Superimposition of the zygomatic process over the buccal root apices of the maxillary first molar by the bisecting-angle technique.

tends to project buccal roots and the zygomatic process to the mesial, thus enhancing anatomic clarity.[19]

The bisecting-angle technique is not preferred for endodontic radiography. However, when a modified paralleling technique cannot be used, there may be no choice because of difficult anatomic configurations or patient management problems.[19,49,53,70] The basis of this technique is to place the film directly against the teeth without deforming the film (see Fig. 5-3, *C* and *D*). The structure of the teeth, however, is such that with the film in this position, an obvious angle exists between the plane of the film and the long axis of the teeth. This causes distortion, because the tooth is not parallel to the film. If the x-ray beam is directed at a right angle to the film, the image on the film will be shorter than the actual tooth (i.e., foreshortened). If the beam is directed perpendicularly to the long axis of the teeth, the image will be much longer than the tooth (i.e., elongated). Thus, by directing the central beam perpendicular to an imaginary line that bisects the angle between tooth and film, the length of the tooth's image on the film should be the same as the actual length of the tooth.

Although the projected length of the tooth is correct, the image will show distortion because the film and object are not parallel and the x-ray beam is not directed at right angles to both. This distortion increases along the image toward its apical extent. The technique produces additional error potential, because the clinician must imagine the line bisecting the angle (an angle that, in itself, is difficult to assess). In addition to producing more frequent superimposition of the zygomatic arch over apices of maxillary molars, the bisecting-angle technique causes greater image distortion than the paralleling technique and makes it difficult for the clinician to reproduce radiographs at similar angulations to assess healing after root canal treatment[28] (see Fig. 5-3, *C* and *D*).

Film Holders and Aiming Devices

Film holders and aiming devices are required for the paralleling technique because they reduce geometric distortion caused by misorientation of the film, central beam, and tooth.[19,49,53,65,70] They also minimize cone cutting, improve diagnostic quality, and allow similarly angulated radiographs to be exposed during treatment and at recall. By eliminating the patient's finger from the x-ray field and thus the potential for displacing the film, these devices help to minimize retakes and make it easier for the patient and clinician to properly position the film.

A number of commercial devices are available that position the film parallel and at various distances from the teeth, but

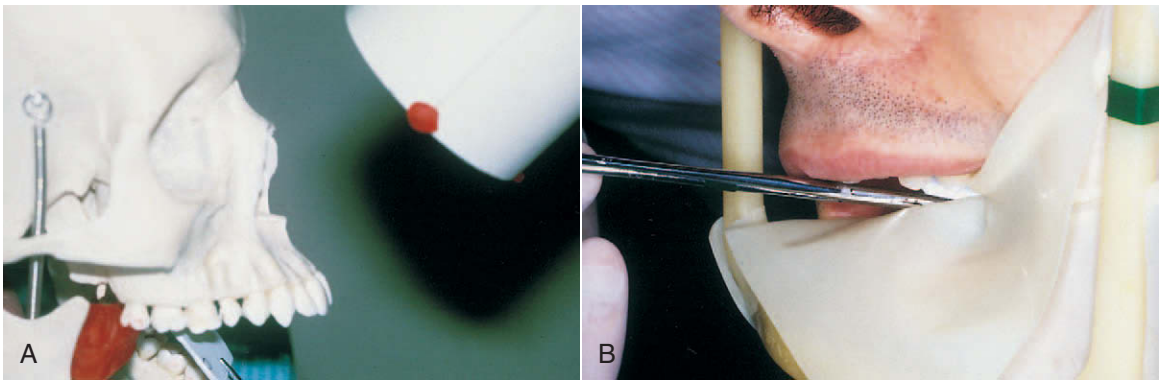


FIG. 5-4 A, With the paralleling technique, the tube head is positioned at a 90-degree angle to the film. The hemostat aids in film placement and in cone alignment. Note that the hemostat is resting on the mandibular anterior teeth so that the film is parallel with the long axis of the maxillary central incisors. **B,** Releasing a corner of the rubber dam aids in hemostat placement so that the film can be properly aligned. (***A,*** *Courtesy Dr. Eddy Tidwell;* ***B,*** *courtesy Dr. Michelle Speier.)*

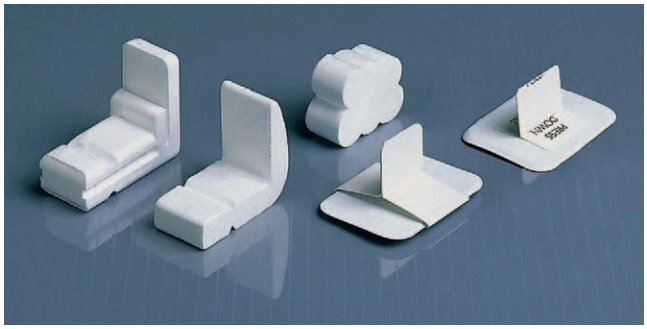


FIG. 5-5 Examples of XCP film-holding devices. *Left to right,* XCP Bite-Block, Stabe Bite-Block, EZ-Prop mouth prop, bite-wing loops, and adhesive bite-wing tabs. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*



FIG. 5-6 XCP instruments hold the radiograph film packets and aid in cone alignment. Cone cutting is prevented, and consistent angulation can be achieved. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*

one of the most versatile film-holding devices is the hemostat. The clinician positions a hemostat-held film, and the handle is used to align the cone vertically and horizontally. The patient then holds the hemostat in the same position, and the cone is positioned at a 90-degree angle to the film (Fig. 5-4, *A*). When taking working radiographs, a radiolucent, plastic, rubber dam frame, such as an Ostby or Young frame, should be used and not removed. To position the hemostat or other film-holding device, a corner of the rubber dam is released for visibility and to allow the subsequent placement of the device-held film (Fig. 5-4, *B*). The Stabe disposable film holder (DENTSPLY Rinn, Elgin, IL) (Fig. 5-5) is another film-holding device that is ideal for taking pretreatment and posttreatment films.

Besides the Dunvale Snapex system mentioned earlier, the major commercial film-holding and aiming devices include the XCP (extension cone paralleling) instruments, the EndoRay II endodontic film holder, the Uni-Bite film holder, the Snap-A-Ray film holder, the Snapex system film holder with aiming device, and the Crawford film holder system (Figs. 5-6 to 5-10).

Variations in the use of the XCP system, for example, can prevent displacement of the rubber dam clamp and increase periradicular coverage during endodontic procedures. The film is placed off center in the bite block, and the cone is placed off center with respect to the aiming ring. This allows for placement of the bite block adjacent to the rubber dam clamp without altering the parallel relation of the cone to the film. A customized hemostat (with rubber bite block attached) can

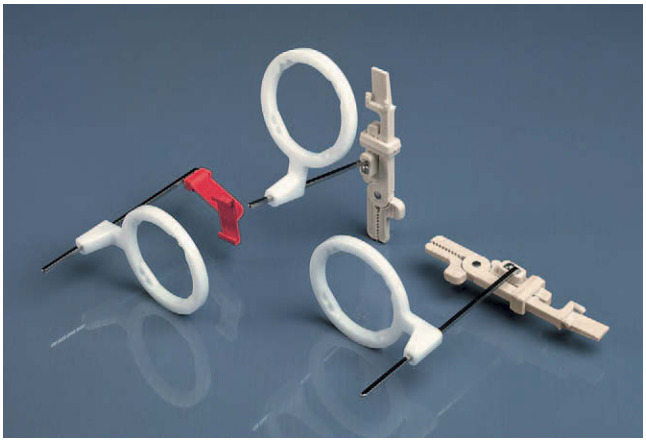


FIG. 5-7 Snapex film holder and aiming ring. The biting portion of the instrument is reduced to make it easier to place the instrument around the rubber dam. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*



FIG. 5-8 Snap-A-Ray film holder. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*

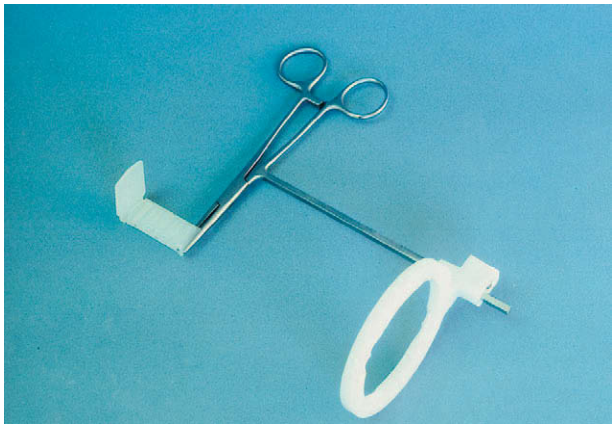


FIG. 5-9 Crawford film holder system. Components include a Kelly hemostat with aiming rod (attached), aiming ring, and bite block. *(Courtesy Dr. Frank Crawford.)*

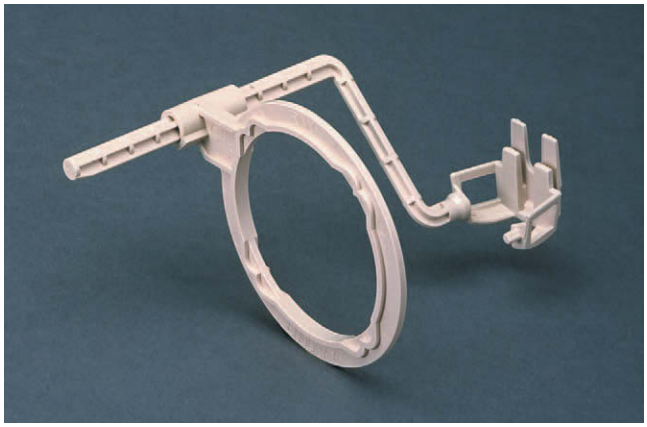


FIG. 5-10 EndoRay II: a film-holding device designed specifically for endodontic radiography. It fits over files, clamps, and dental dams without touching the subject tooth. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*

also be made to assist film placement during the taking of working radiographs. Other specialized film holders, such as the EndoRay and the Crawford film holder system, have been designed to help the clinician secure parallel working films with the rubber dam clamp in place. In general, these holders all have an x-ray beam–guiding device for a proper beam–film relationship and a modified bite block and film holder for proper positioning over or around the rubber dam clamp (see Fig. 5-10).

Exposure and Film Qualities

The intricacies of proper kilovoltage, milliamperage, and time selection serve as examples of how the diagnostic quality of a film may be altered by changes in the film's density and contrast.[53,70] Density is the degree of darkening of the film, whereas contrast is the difference between densities. The amount of darkening depends on the quantity and quality of radiation delivered to the film, the subject thickness, and the developing or processing conditions. Milliamperage controls the electron flow from cathode to anode; the greater the electron flow per unit of time, the greater will be the quantity of radiation produced. Proper density is primarily a function of milliamperage and time. Kilovoltage also affects film density by controlling the quality and penetrability of the rays. Higher kilovoltage settings produce shorter wavelengths that are more penetrating than the longer wavelengths produced at lower settings.[53,70] The ability to control the penetrability of the rays by alterations in kilovoltage affects the amount of radiation reaching the film and the degree of darkening or density. Altering exposure time or milliamperage or both for each respective unit can control variations in density.[53,70]

Contrast is defined as the difference between shades of gray or as the difference between densities. Most variation observed in endodontic radiography occurs because of subject contrast, which depends on the thickness and density of the subject and the kilovoltage used. Thus kilovoltage is really the only exposure parameter under the clinician's control that directly affects subject contrast.[30,53,70] Exposure time and milliamperage control only the number of x-rays; therefore they influence mainly the density of the film image. A radiographic film may exhibit a long-scale, or low, contrast (i.e., more shades of gray or more useful densities); high-kilovoltage techniques (e.g., 90 kVp) produce this long scale of contrast as a result of the increased penetrating power of the rays. This results in images with many more shades of gray and less distinct differences. Films exposed at low kilovoltage settings (e.g., 60 kVp) exhibit short-scale, or high, contrast, with sharp differences between a few shades of gray, black, and white.[53,70] Although they are perhaps more difficult to read, films exposed at higher kilovoltage settings (e.g., 90 kVp) make it possible to discriminate between images, often enhancing diagnostic quality; films exposed at a lower kilovoltage (e.g., 70 kVp) have better clarity and contrast between radiopaque and radiolucent structures, such as endodontic instruments near the root apex. Nevertheless, the optimal kilovoltage and exposure time should be individualized for each radiograph unit and exposure requirement.

Processing

Proper darkroom organization, film handling, and adherence to the time and temperature method of film processing play important roles in producing films of high quality.[53] For the sake of expediency in the production of working films in endodontics, rapid processing methods are used to produce relatively good films in less than 1 to 2 minutes (Fig. 5-11).[53,70] Although the contrast when using rapid-processing chemicals is lower than that achieved by conventional techniques, the radiographs have sufficient diagnostic quality to be used for treatment films and are obtained in less time and with less patient discomfort. Rapid-processing solutions are available



FIG. 5-11 Chairside darkroom allows rapid processing of endodontic working films. *(Courtesy DENTSPLY Rinn, Elgin, IL.)*

commercially, but they tend to vary in shelflife, in tank life, and in the production of films of permanent quality.

To maintain the radiographic image for documentation, it is recommended that after an image has been evaluated it be returned to the fixer for 10 minutes more and then washed for 20 minutes and dried. An alternative is to reprocess the film by means of the conventional technique. Double film packets can also be used for working films: one can be processed rapidly and the other conventionally. Regardless of what method is used for working films, a controlled time and temperature method should be used for the diagnostic qualities desired in pretreatment, posttreatment, and recall radiographs. All radiographs taken during the course of endodontic treatment should be preserved as a part of the patient's permanent record.

Examination and Differential Interpretation

Radiographic interpretation is not strictly the identification of a problem and the establishment of a diagnosis. The clinician must read the film carefully, with an eye toward diagnosis and treatment. Frequently overlooked are the small areas of resorption, invaginated enamel, separated files, minute fracture lines, extra canals or roots, curved and calcified canals, and, in turn, the potential problems they may create during treatment (Fig. 5-12). If a thorough radiographic examination is conducted, problems during treatment, additional time, and extra expense can be avoided or at least anticipated. As mentioned earlier, additional exposures at various angulations may be necessary to gain better insight into the three-dimensional structure of a tooth.

Many anatomic structures and osteolytic lesions can be mistaken for periradicular pathoses. Among the more commonly misinterpreted anatomic structures are the mental foramen and the incisive foramen. These radiolucencies can be differentiated from pathologic conditions by exposures at different angulations and by pulp-testing procedures. Radiolucencies not associated with the root apex will move or be projected away from the apex by varying the angulation. Radiolucent areas resulting from sparse trabeculation can also simulate radiolucent lesions. In such cases these areas must be differentiated from the lamina dura and periodontal ligament space.

A commonly misinterpreted osteolytic lesion is periapical cemental dysplasia ("cementoma") (Fig. 5-13). The use of pulp-testing procedures and follow-up radiographic examinations will prevent the mistake of diagnosing this as a periradicular pathosis. The development of this lesion can be monitored radiographically from its early, more radiolucent stage through its mature or more radiopaque stage.

Other anatomic radiolucencies that must be differentiated from periradicular pathoses are the maxillary sinus, nutrient canals, nasal fossa, and lateral or submandibular fossa. Many systemic conditions can mimic or affect the radiographic appearance of the alveolar process. A discussion of these conditions is beyond the scope of this chapter, but the reader is encouraged to read further in any oral pathology textbook.

Lamina Dura: A Question of Integrity

One key challenge in endodontic radiographic interpretation is to understand the integrity, or lack of integrity, of the lamina dura, especially in its relationship to the health of the pulp. Anatomically, the lamina dura[70] is a layer of compact bone (i.e., cribriform plate or alveolar bone proper) that lines the tooth socket. Noxious products emanating from the root canal system can effect a change in this structure that is visible radiographically. X-ray beams passing tangentially through the socket must pass through many times the width of the adjacent alveolus, and they are attenuated by this greater thickness of bone, producing the characteristic "white line." If, for example, the beam is directed more obliquely so that it is not as attenuated, the lamina dura appears more diffuse, or it may not be discernible at all. Therefore the presence or absence and integrity of the lamina dura are determined largely by the shape and position of the root and, in turn, by its bony crypt, in relation to the x-ray beam. This explanation is consistent with the radiographic and clinical findings of teeth with normal pulps and no distinct lamina dura.[62]

Changes in the integrity of the periodontal ligament space, the lamina dura, and the surrounding periradicular bone certainly have diagnostic value, especially when recent radiographs are compared with previous ones. However, the significance of such changes must be tempered by a thorough understanding of the features that give rise to these images.

Buccal Object Rule (Cone Shift)

In endodontic therapy it is imperative that the clinician know the spatial or buccolingual relation of an object within the tooth or alveolus. The technique used to identify the spatial relation of an object is called the *cone-* or *tube-shift technique*. Other names for this procedure are the *buccal object rule, Clark's rule,* and the *SLOB* (same lingual, opposite buccal) rule.[31,53,60,70] Proper application of the technique allows the clinician to locate additional canals or roots, to distinguish between objects that have been superimposed, and to distinguish between various types of resorption. It also helps the clinician to determine the buccolingual position of fractures and perforative defects, to locate foreign bodies, and to locate anatomic landmarks in relation to the root apex, such as the mandibular canal.[65]

The buccal object rule relates to the manner in which the relative position of radiographic images of two separate objects changes when the projection angle at which the images were made is changed. The principle states that the object closest to

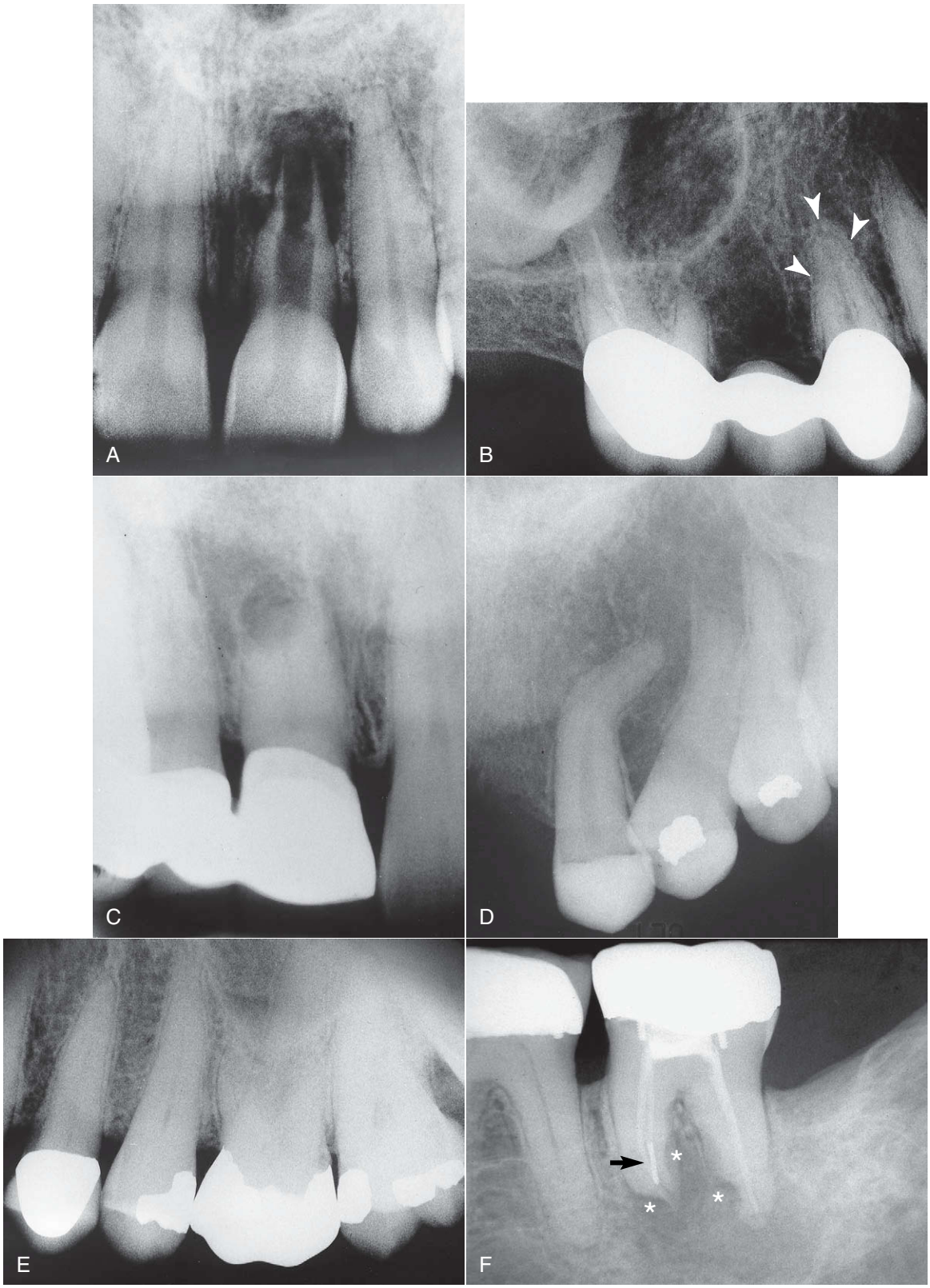


FIG. 5-12 A, Maxillary left central incisor with a history of trauma and thin walls in the apical one third of the tooth. Once an apical barrier is formed, pressures exerted during obturation could cause fracture. **B,** Maxillary right first premolar with three separate roots *(arrows)*. **C,** Maxillary right central incisor with a history of trauma. Apical resorption and calcification of the canal system complicate treatment. **D,** Dilacerated root system on maxillary left canine. **E,** Maxillary left first molar with calcification of the chamber and root canal system. **F,** Endodontically treated mandibular second molar with apical root resorption (*star* on mesial root) and external root resorption (*star* on distal root); separated file in mesial root *(arrow)*.

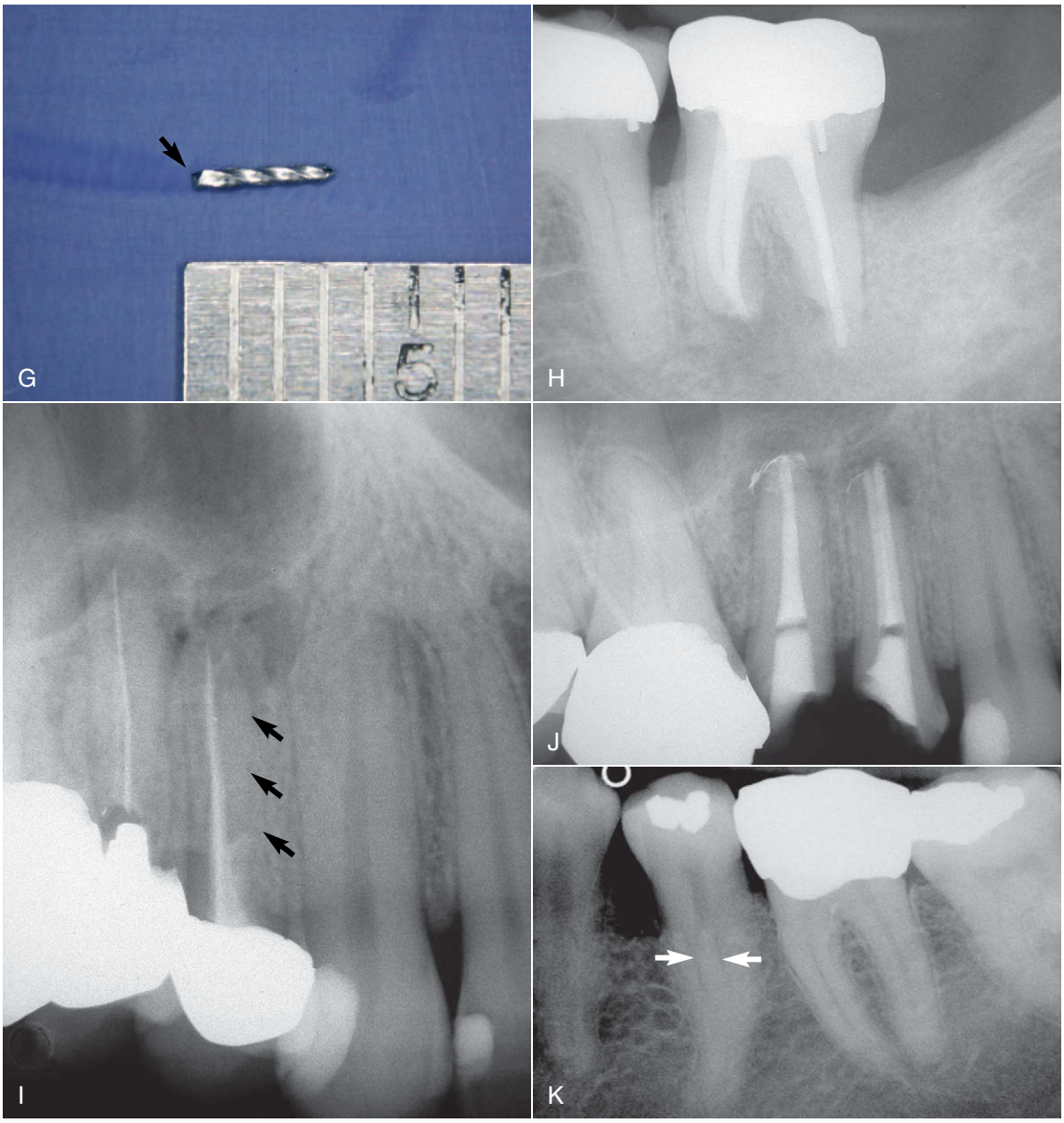


FIG. 5-12, cont'd G, Retrieved file. **H,** Completed retreatment. **I,** Angled radiograph showing evidence of another root *(apical arrows)* in an endodontically treated maxillary first premolar; *coronal arrow* indicates sealer in unprepared canal. **J,** Completed root canal treatment of two separate canals. **K,** Bifurcation *(arrows)* of the root canal system in a mandibular second premolar. *(**H, J,** and **K,** courtesy Dr. Francisco A. Banchs.)*

the buccal surface appears to move in the direction opposite the movement of the cone or tube head, when compared with a second film. Objects closest to the lingual surface appear to move (on a film) in the same direction that the cone moved; thus the "same lingual, opposite buccal" rule. Figure 5-14 shows three simulated radiographs of a buccal object (yellow circle) and a lingual object (red triangle) exposed at different horizontal angles. The positions of the objects on each radiograph are compared with the reference structure (i.e., the mesial root apex of the mandibular first molar). The first radiograph (see Fig. 5-14, *A* and *B*) shows superimposition of the two objects; in this case the tube head was positioned for a straight-on view. In the second radiograph (see Fig. 5-14, *C* and *D*), the tube head was shifted mesially, and the beam was directed at the reference object from a more mesial angulation. In this case the lingual object (red triangle) moved mesially with respect to the reference object, and the buccal object (yellow circle) moved distally with respect to the reference object. In the third radiograph (see Fig. 5-14, *E* and *F*), the tube head was shifted distally and the beam was directed at the reference object from a more distal angulation; here the triangle moved distally with respect to the mesial root of the mandibular first molar, and the circle moved mesially. These radiographic relations confirm that the lingual object (red triangle) moves in the same direction with respect to reference structures as the radiograph tube and that the buccal object (yellow circle) moves in the opposite direction of the radiograph tube. Thus, according to the rule, the object farthest



FIG. 5-13 Variations in stages of periapical cemental dysplasia (i.e., cementoma) on the four mandibular incisors. All teeth are vital. *(Courtesy Dr. Francisco A. Banchs.)*

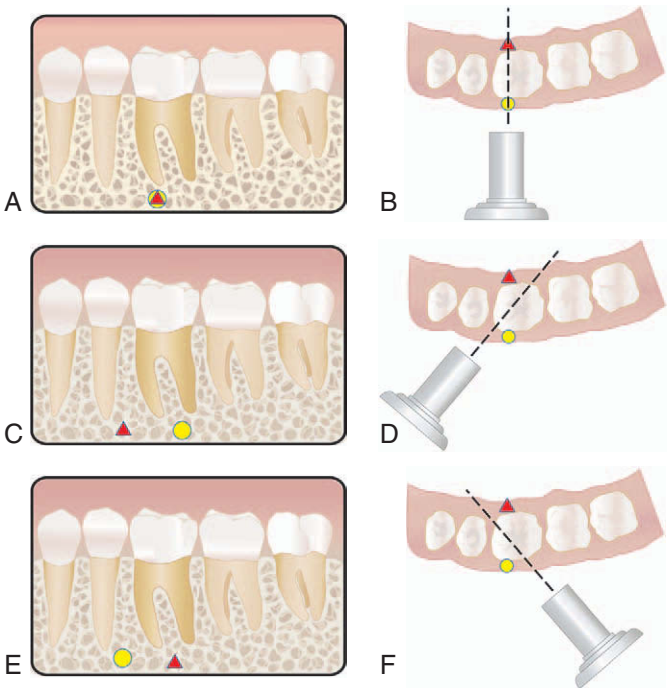


FIG. 5-14 Objects may be localized with respect to reference structures by using the buccal object rule (i.e., tube-shift technique). **A-B,** A straight-on view will cause superimposition of the buccal object *(yellow circle)* with the lingual object *(red triangle)*. **C-D,** Using the tube-shift technique, the lingual object *(red triangle)* will appear more mesial with respect to the mesial root of the mandibular first molar, and the buccal object *(yellow circle)* will appear more distal on a second view projected from the mesial. **E-F,** The object *(red triangle)* on the lingual surface will appear more distal with respect to the mesial root of the mandibular first molar, and the object *(yellow circle)* on the buccal surface will appear more mesial on a view projected from the distal aspect.

(i.e., most buccal) from the film moves farthest on the film with respect to a change in horizontal angulation of the radiograph cone. In an endodontically treated mandibular molar with four canals (Fig. 5-15), a straight-on view results in superimposition of the root-filled canals on the radiograph. If the cone is angled from mesial to distal, the mesiolingual and distolingual canals will move mesially and the mesiobuccal and distobuccal canals will move distally on the radiograph, when compared with the straight-on view.

The examples cited previously involve application of the buccal object rule, using changes in horizontal angulation. The clinician should be aware that this rule also applies to changes in vertical angulation (Fig. 5-16). To locate the position of the mandibular canal relative to mandibular molar root apices, radiographs must be taken at different vertical angulations. If the canal moves with or in the same direction as the cone head, the canal is lingual to the root apices; if the mandibular canal moves opposite the direction of the cone head, the canal is buccal to the root apices. The clinician should recognize the wide range of applicability of the buccal object rule in determining the buccolingual relationship of structures not visible in a two-dimensional image.

Digital Radiographic Techniques

The replacement of traditional radiographic film with digital sensors offers many advantages to radiography. The evolution of computer technology for radiography has allowed for nearly instantaneous image acquisition, image enhancement, storage, retrieval, and even transmission of images to remote sites in a digital format. The major advantages of using digital radiography in endodontics are that radiographic images are obtained immediately and radiation exposure is reduced from 50% to 90% compared with conventional film-based radiography.[56,70] The primary disadvantages of digital imaging systems are their high initial cost and potential for reduction in image quality when compared with conventional radiography.

Digital imaging systems require an electronic sensor or detector, an analog-to-digital converter, a computer, and a monitor or printer for image display.[70] (See Chapters 28 and

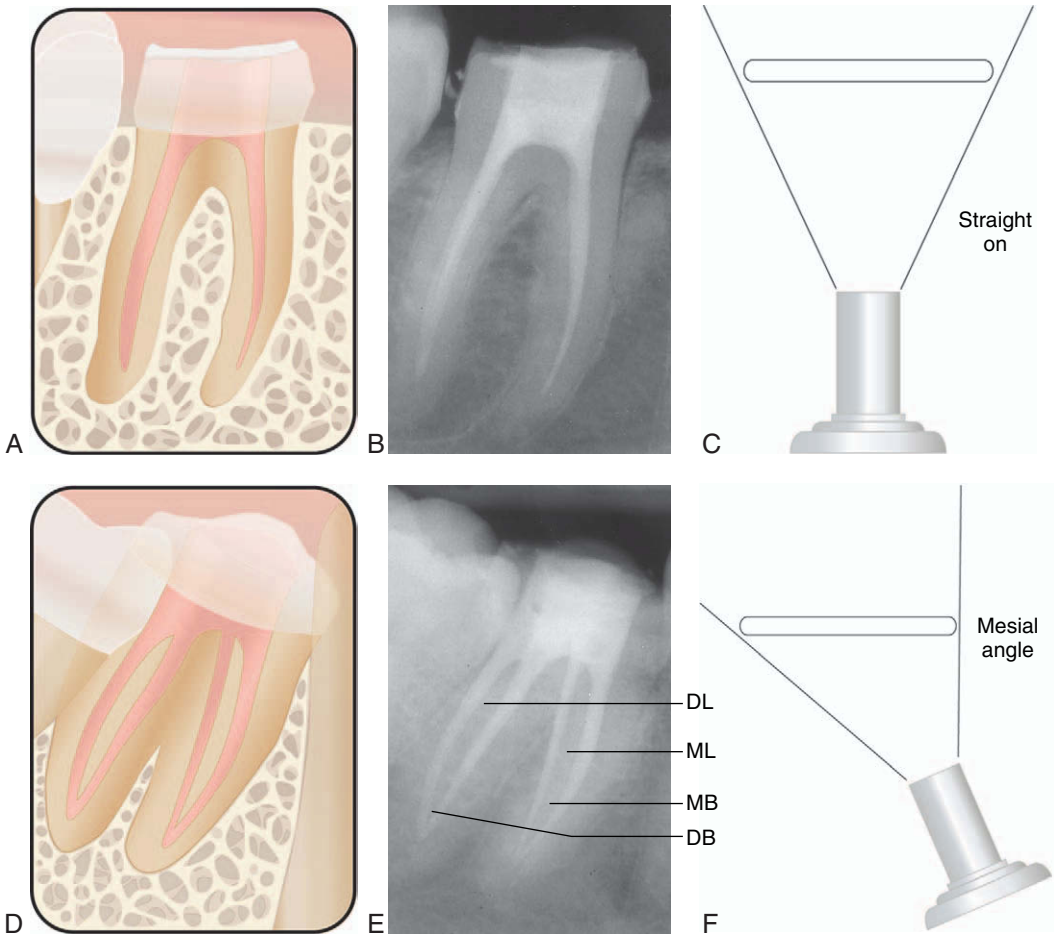


FIG. 5-15 Comparison of straight-on and mesial-angled views of an endodontically treated mandibular molar with four canals. **A-C,** Straight-on view of the mandibular molar shows superimposition of the root canal fillings. **D-F,** Mesiodistal angulation produces separation of the canals. The mesiolingual *(ML)* and distolingual *(DL)* root-filled canals move mesially (i.e., toward the cone), and the mesiobuccal *(MB)* and distobuccal *(DB)* root-filled canals move distally (i.e., away from the cone) on the radiograph.

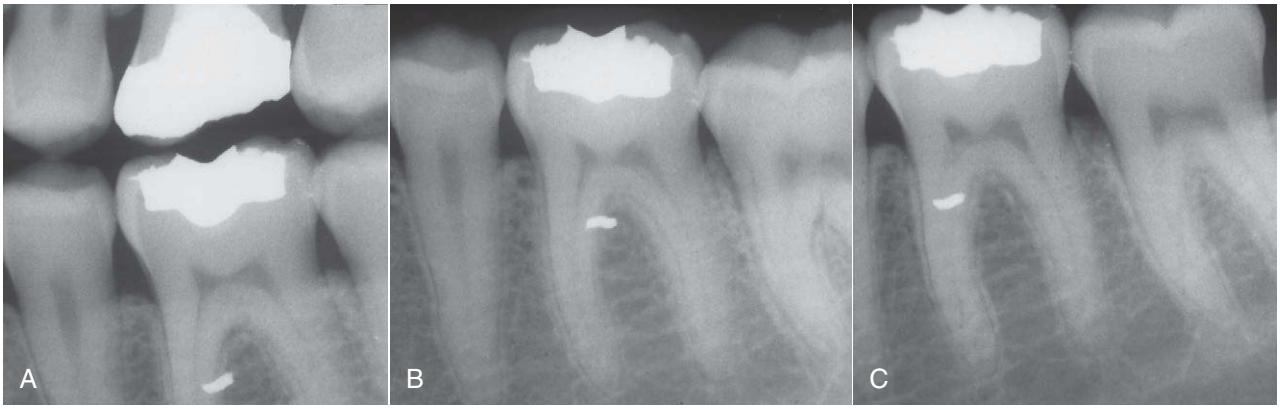


FIG. 5-16 Examples of the buccal object rule, using shifts in vertical and horizontal angulations. **A,** Bite-wing radiograph (straight-on view with minimal horizontal and vertical angulation) depicts amalgam particle superimposed over the mesial root of the mandibular first molar. To determine the buccolingual location of the object, the tube-shift technique (buccal object rule) must be applied. **B,** The periapical radiograph was exposed by shifting the vertical angulation of the cone (i.e., the radiograph beam was projected more steeply upward). Because the amalgam particle moved in the opposite direction to that of the cone (compared with the bite-wing radiograph), the amalgam particle lies on the buccal aspect of the tooth. **C,** The periapical radiograph was exposed by shifting the horizontal angulation of the cone. (The radiograph was exposed from a distal angle.) Compared with both **A** and **B,** each taken straight-on with minimal horizontal angulation, the amalgam particle moved opposite the direction of movement of the cone or tube head, confirming that the amalgam particle lies on the buccal aspect of the tooth.



FIG. 5-17 Digital imaging systems. **A,** Dexis digital x-ray system. **B,** Special shape of Dexis sensor. **C,** Schick desktop digital system with wireless sensor. **D,** Clinical placement of wireless sensor. **E,** Schick wired sensor covered by plastic sheath for infection control. *(**A-B,** Courtesy Dexis LLC, Des Plaines, IL; **C-E,** Courtesy Schick Technologies, Long Island City, NY.)*

29 online for a further discussion of digital imaging systems and how they function.)

Digitization of ionizing radiation first became a reality in the late 1980s with the development of the original RadioVisioGraphy (RVG) system by Dr. Francis Mouyen.[56] This system later was marketed as the RVGui (Kodak Dental Systems/Carestream Health website, http://www.kodakdental.com/en/digital-imaging/intraoral-radiography/kodak-rvg-6100.aspx). Other available systems include Dexis digital x-ray (Dexis, Des Plaines, IL) and computed dental radiography (CDR) (Schick Technologies, Long Island City, NY) (Fig. 5-17, *A* and *C*). The FDA has approved all these systems.

Direct digital systems have three components: (1) the "radio" component, (2) the "visio" component, and (3) the "graphy" component. The "radio" component consists of a high-resolution sensor with an active area that is similar in size to conventional film. However, length, width, and thickness vary slightly depending on the respective system (Fig. 5-17, *B* and *E*). The sensor is protected from x-ray degradation by a fiberoptic shield, and it can be disinfected. Specially designed multiple types of sensor holders are available; for infection control, disposable plastic sheaths are used to cover the sensor when it is in use (see Fig. 5-17, *E*). Wireless CDR sensors have become available through Schick Technologies. This technology provides cable-free sensors to allow enhanced mobility at chairside (see Fig. 5-17, *C* and *D*). CDR Wireless is the first wireless direct digital radiography system. Wireless sensors provide greater mobility at chairside while reportedly providing the same level of image quality acquired with conventional CDR systems. Sensors instantly transmit images directly from the mouth. The image is automatically transmitted to the computer via radio waves. Images do not need to be processed as with traditional film and storage phosphor plates. Chemical processing as with traditional film is not needed. Also, sensors do not need to be downloaded, erased, or reset between shots.

The second component of a direct digital system, the "visio" portion, consists of a video monitor and display-processing unit (see Fig. 5-17, *A* and *C*). As the image is

transmitted to the processing unit, it is digitized and stored by the computer. The unit magnifies the image for immediate display on the video monitor; it also can produce colored images and display multiple images simultaneously, including a full mouth series on one screen. Because the image is digitized, further manipulation of the image is possible; this includes enhancement, contrast stretching, and reversing. A zoom feature is also available to enlarge a portion of the image up to full-screen size.

The third component of a direct digital system is the "graphy," a high-resolution video printer that provides a hard copy of the screen image, using the same video signal. In addition, a digital intraoral camera can be integrated with most systems. Indirect digital imaging or cordless systems, such as Digora (Soredex-Finndent, Conroe, Texas) and the DenOptix digital imaging system (Gendex, Des Plaines, IL), involve the use of a reusable filmlike plate without wires. The image to be scanned by a laser (to digitize it before viewing on the computer) is recorded on this plate. Although indirect digital imaging still incorporates reduced radiation exposure and image manipulation, it usually takes slightly longer before the image can be viewed.

The advantages of both direct and indirect digital radiography seem numerous, but the primary ones include the elimination of standard radiograph film and processing chemicals, a significant reduction in exposure time (i.e., 80% to 90% reduction, when compared with D-speed film), and rapid image display. Virtually all systems can be linked with electronic record systems so that patient data can be stored, accessed, and transmitted easily. An exposure time in the range of hundredths of a second is all that is needed to generate an image.[70] One study showed that digital radiographic resolution was slightly lower than that produced with silver halide film emulsions, but the radiographic information may be increased with the electronic image treatment capabilities of the system.[56] These systems appear to be promising for endodontics and for the clinician.

Digital subtraction radiography[58] is a sensitive method for detecting changes in radiographic density over time. In endodontics, digital subtraction radiography may be especially useful for evaluating osseous healing after treatment and as an aid in diagnosis. By definition, subtraction radiography requires that two images have nearly identical image geometry; specialized positioning devices and bite registrations aid in matching the images. The subtracted image is a composite of the images, representing their variations in density. By subtracting all anatomic structures that have not changed between radiographic examinations, changes in diagnostic information become easier to interpret. Any change is displayed on the resultant image against a neutral, gray background. Advances[58] in computer technology have incorporated built-in algorithms to correct for variations in exposure and projection geometry. These advances have also enabled colorization of density changes so that hard tissue gain is represented by one color and hard tissue loss is represented by another color.

Orascopy and Endoscopy

Orascopy[7] (Fig. 5-18, *A*), and related endoscopic instruments, have been adapted for enhanced visualization in endodontics and consist of either flexible or rigid fiberoptic endoscopes. These fiberoptic probes are available in various diameters; the probes provide a large depth of field, and refocusing is not needed after the initial focus. Once the probe is applied, the clinician views the conventional or surgical site from the magnified image displayed on the monitor. Endoscopic endodontics allows the clinician to have a nonfixed field of vision, and probes can be manipulated at various angles and distances from an object without loss of focus or image clarity. With orascopy, finite fracture lines, accessory canals, missed canals and isthmuses, and apical tissues can be viewed (Fig. 5-18, *B-E*). Evolving technology will likely enhance the precision and accuracy of the fiberoptic probes.

PREPARATION FOR ACCESS: TOOTH ISOLATION

The use of the rubber dam is mandatory in root canal treatment.[16,18] Developed in the nineteenth century by S.C. Barnum, the rubber dam has evolved from a system that was designed to isolate teeth for placement of gold foil to one of sophistication for the ultimate protection of both patient and clinician.[65] The advantages[8,12,16,29,32] and absolute necessity of the rubber dam must always take precedence over convenience and expediency (a rationale often cited by clinicians who condemn its use). When properly placed, the rubber dam facilitates treatment by isolating the tooth from obstacles (e.g., saliva and tongue) that can disrupt any procedure. Proper rubber dam placement can be done quickly and will enhance the entire procedure.

The rubber dam is used in endodontics because it ensures the following[6,16,29,37,65]:

1. The patient is protected from aspiration or swallowing of instruments, tooth debris, medicaments, and irrigating solutions.
2. The clinician is protected from litigation because of patient aspiration or swallowing of an endodontic file. *Routine placement of the rubber dam is considered the standard of care.*[17,18]
3. A surgically clean operating field is isolated from saliva, blood, and other tissue fluids. The dam reduces the risk of cross-contamination of the root canal system, and it provides an excellent barrier to the potential spread of infectious agents.[16,29] *It is a required component of any infection control program.**
4. Soft tissues are retracted and protected.
5. Visibility is improved. The rubber dam provides a dry field and reduces mirror fogging.
6. Efficiency is increased. The rubber dam minimizes patient conversation during treatment and the need for frequent rinsing.

The clinician should be aware that in some situations, especially in teeth with crowns, access into the pulp system may be difficult without first orienting root structure to the adjacent teeth and periodontal tissues. Radiographically, the coronal pulp system is often obscured by the restoration, and, as a result, the clinician may misdirect the bur during access. In these cases it may be necessary to locate the canal system before placing the dam. In doing so the clinician can visualize root topography, making it easier to orient the bur toward the

*References 2, 9-12, 14, 19, 20, 23, 37, 46, 59.

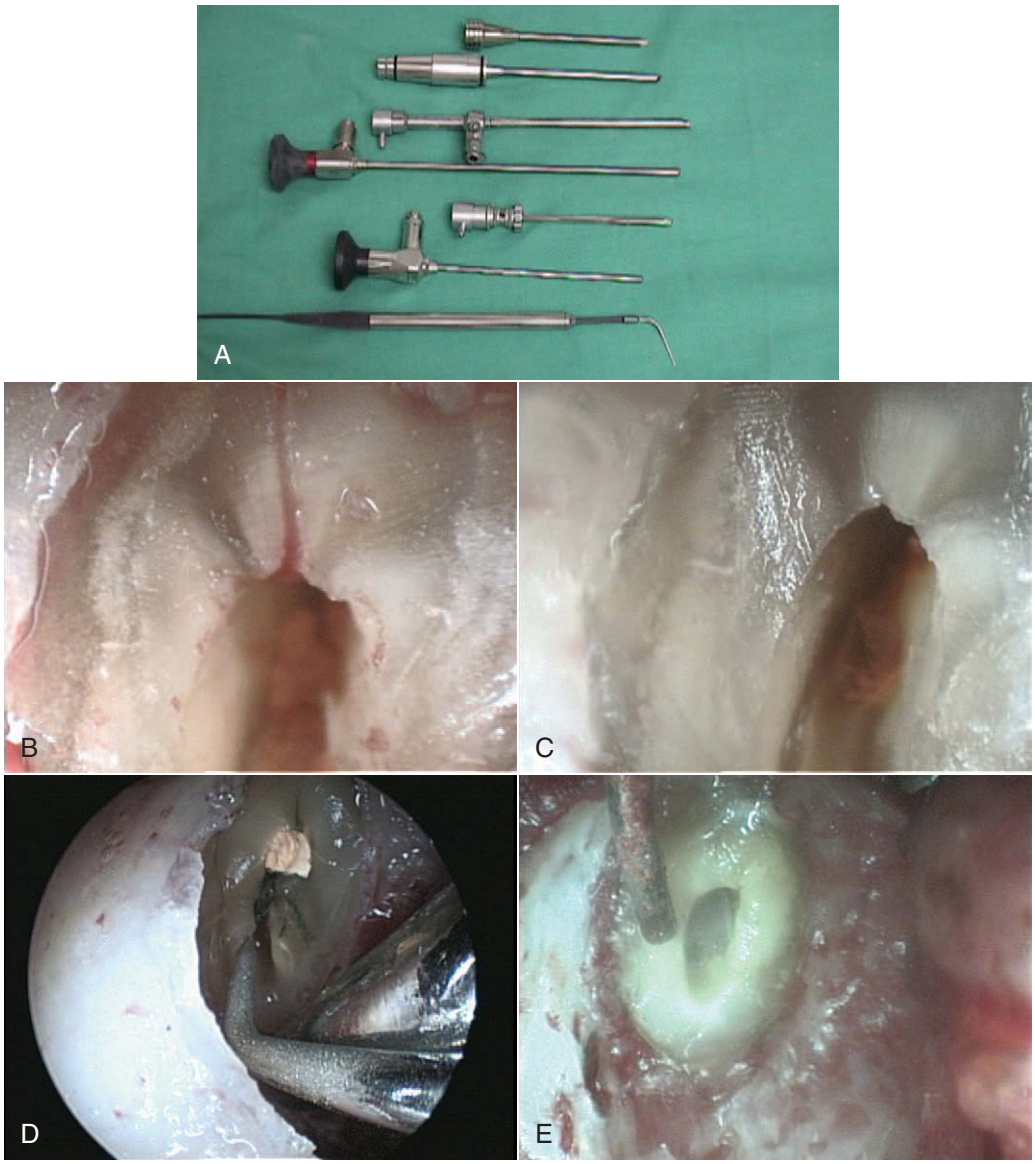


FIG. 5-18 A, Orascope instruments include various diameter probes. **B,** Endoscopic view of lateral canal at resected root end. **C,** Endoscopic view of resected root end after removal of lateral canal. **D,** Endoscopic view of ultrasonic preparation of isthmus and lingual canal at resected root end. **E,** Endoscopic view of Stropko syringe at prepared root end. *(Courtesy Dr. Barnet B. Shulman.)*

long axis of the roots and prevent perforation. Once the root canal system is located, however, the rubber dam should be immediately placed.

Armamentarium

The mainstay of the rubber dam system is the dam itself. These autoclavable sheets of thin, flat latex come in various thicknesses (e.g., thin, medium, heavy, extraheavy, and special heavy) and in two different sizes (5 × 5 and 6 × 6 in). For endodontic purposes, the medium thickness is probably best because it tends to tear less easily, retracts soft tissues better than the thin type, and is easier to place than the heavier types. However, a thinner gauge may be desirable to decrease tension if retainer placement is questionable or if the retainer is resting on a band. The dam is also manufactured in various colors, ranging from light yellow to blue to green to gray. The darker colored dams may afford better visual contrast, thus reducing eye strain. However, the lighter colored dams, because of their translucency, have the advantage of naturally illuminating the operating field and allowing easier film placement underneath the dam. Depending on individual preference and specific conditions associated with a tooth, the clinician may find it necessary to vary the color and thickness of the rubber dam used. Glare and eyestrain can be reduced and contrast enhanced by routinely placing the dull side of the dam toward the clinician.

For patients with latex allergies, a nonlatex rubber dam is available from Coltène/Whaledent (Cuyahoga Falls, OH) (Fig. 5-19). This powder-free, synthetic dam comes in one size (6 × 6 in.) and in one thickness (medium gauge). It has a shelflife of 3 years but only one third the tensile strength of a latex dam. Other companies provide nitrile rubber dams.



FIG. 5-19 Nonlatex dental dam is ideal for patients with known latex allergies. *(Courtesy Coltene/Whaledent, Inc., Cuyahoga Falls, Ohio.)*

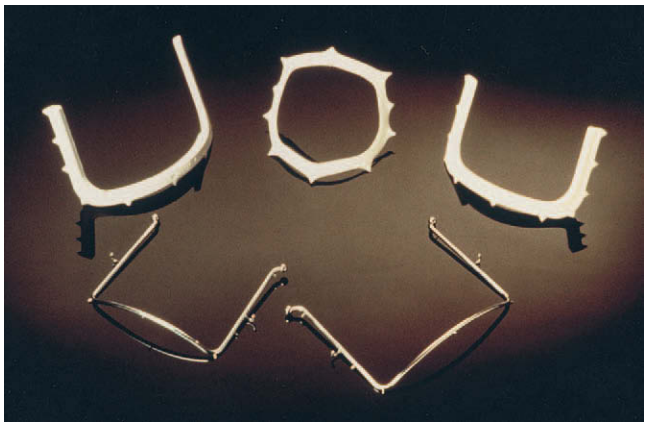


FIG. 5-20 Plastic radiolucent and metal rubber dam frames. *Top left,* plastic frame from Young Dental; *top center,* plastic Ostby frame from Coltène/Whaledent.

Another component of the rubber dam system is the rubber dam frame, which is designed to retract and stabilize the dam. Both metal and plastic frames are available, but plastic frames are recommended for endodontic procedures. They appear radiolucent, do not mask key areas on working films, and do not have to be removed before film placement. The Young Dental (Earth City, MO) rubber dam frame (plastic type), and the Nygaard-Ostby (N-O) frame (Hygenic brand; Coltène/Whaledent) are examples of radiolucent frames used in endodontics (Fig. 5-20). New to endodontics is a specially designed foldable plastic frame (Fig. 5-21), with a hinge to facilitate film or sensor placement without disengaging the entire frame. The disposable Insti-Dam (Zirc, Buffalo, MN) (Fig. 5-22) and HandiDam (Aseptico, Woodinville, WA) rubber dam systems also provide a radiolucent plastic frame. The OptraDam



FIG. 5-21 Foldable plastic rubber dam frame (Plast-Frame) with hinge to allow for easy film/sensor placement. *(Courtesy Hager Worldwide, Odessa, FL.)*

(Ivoclar Vivadent, Amherst, NY) is another disposable single-isolation device with a flexible outer ring, eliminating the need for an additional frame. Although metal frames (see Fig. 5-20) can be used, their radiopacity tends to block out the radiograph. If removed, this may result in destabilization of the dam and salivary contamination of the canal system, negating the disinfected environment that was previously attained.

Rubber dam clamps or retainers anchor the dam to the tooth requiring treatment or, in cases of multiple tooth isolation, to the most posterior tooth. They also aid in soft tissue retraction. These clamps are made of stainless steel, and each consists of a bow and two jaws. Regardless of the type of jaw configuration, the prongs of the jaws should engage at least four points on the tooth. This four-point clamp-to-tooth relationship stabilizes the retainer and prevents any rocking, which in itself can be injurious to both hard and soft tissues.[38,47]

Clamps are available from a variety of manufacturers and are specifically designed for all classes of teeth with a variety of anatomic configurations (Fig. 5-23). For most uncomplicated endodontic isolations, the clinician's basic armamentarium should consist of winged clamps, a butterfly-type clamp for anterior teeth, a universal premolar clamp, a mandibular molar clamp, and a maxillary molar clamp. The wings, which are extensions of the jaws, not only provide for additional soft tissue retraction but also facilitate placement of the rubber dam, frame, and retainer as a single unit (see the next section, Methods of Rubber Dam Placement).

Other retainers are designed for specific clinical situations in which clamp placement may be difficult. For example, when minimal coronal tooth structure remains, a clamp with apically inclined jaws may be used to engage tooth structure at or below the level of the free gingival margin. Retainers with serrated jaws, known as tiger clamps, also may increase stabilization of broken-down teeth. Another type of retainer, the S-G (Silker-Glickman) clamp, should also be included in the clinician's armamentarium (Fig. 5-24). Its anterior extension allows for retraction of a dam around a severely broken-down tooth, and the clamp itself is placed on a tooth proximal to the one being treated.

The remaining components of the rubber dam system include the rubber dam punch and the rubber dam forceps.

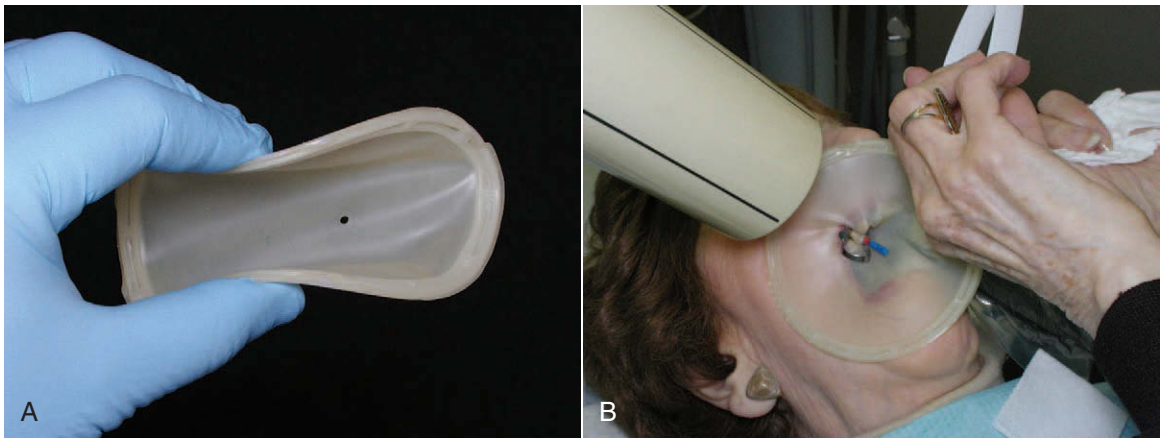


FIG. 5-22 Insti-Dam is a disposable dam system available in both latex and nonlatex. **A,** Bendable flexible frame allows for easy placement. **B,** Use of Insti-Dam during endodontic treatment. *(Courtesy Zirc, Buffalo, MN.)*



FIG. 5-23 Rubber dam clamps are color coded for ease in identification. *(Courtesy Coltène/Whaledent, Cuyahoga Falls, Ohio.)*



FIG. 5-24 The Silker-Glickman (S-G) clamp for isolation of severely broken-down teeth. *(Courtesy The Smile Center, Deerwood, MN.)*

The punch has a series of holes on a rotating disk from which the clinician can select according to the size of the tooth or teeth to be isolated. The forceps holds and carries the retainer during placement and removal.

Methods of Rubber Dam Placement

As mentioned earlier, an expedient method of dam placement is to position the bow of the clamp through the hole in the dam and place the rubber over the wings of the clamp (a winged clamp is required). The forceps stretch the clamp to maintain the position of the clamp in the dam, and the dam is attached to the plastic frame, allowing for the placement of the dam, clamp, and frame in one motion (Fig. 5-25). Once the clamp is secured on the tooth, the dam is teased under the wings of the clamp with a plastic instrument.

Another method is to place the clamp, usually wingless, on the tooth and then stretch the dam over the clamped tooth (Fig. 5-26).[29,56] This method offers the advantage of enabling the clinician to see exactly where the jaws of the clamp engage the tooth, thus avoiding possible impingement on the gingival tissues. Gentle finger pressure on the buccal and lingual apron of the clamp before the dam is placed can be used to test how securely the clamp fits. Variations of this method include placing the clamp and dam first, followed by the frame, or placing the rubber dam first, followed by the clamp and then the frame.[65]

A third method, the split-dam technique, may be used to isolate anterior teeth without using a rubber dam clamp. Not only is this technique useful when there is insufficient crown structure, as in the case of horizontal fractures, but also it prevents the possibility of the jaws of the clamp chipping the margins of teeth restored with porcelain crowns or laminates. Studies[38,47] on the effects of retainers on porcelain-fused-to-metal restorations and tooth structure itself have demonstrated that there can be significant damage to cervical porcelain, as well as to dentin and cementum, even when the clamp is properly stabilized. Thus for teeth with porcelain restorations, ligation with dental floss is recommended as an alternative method to retract the dam and tissues, or the adjacent tooth can be clamped.

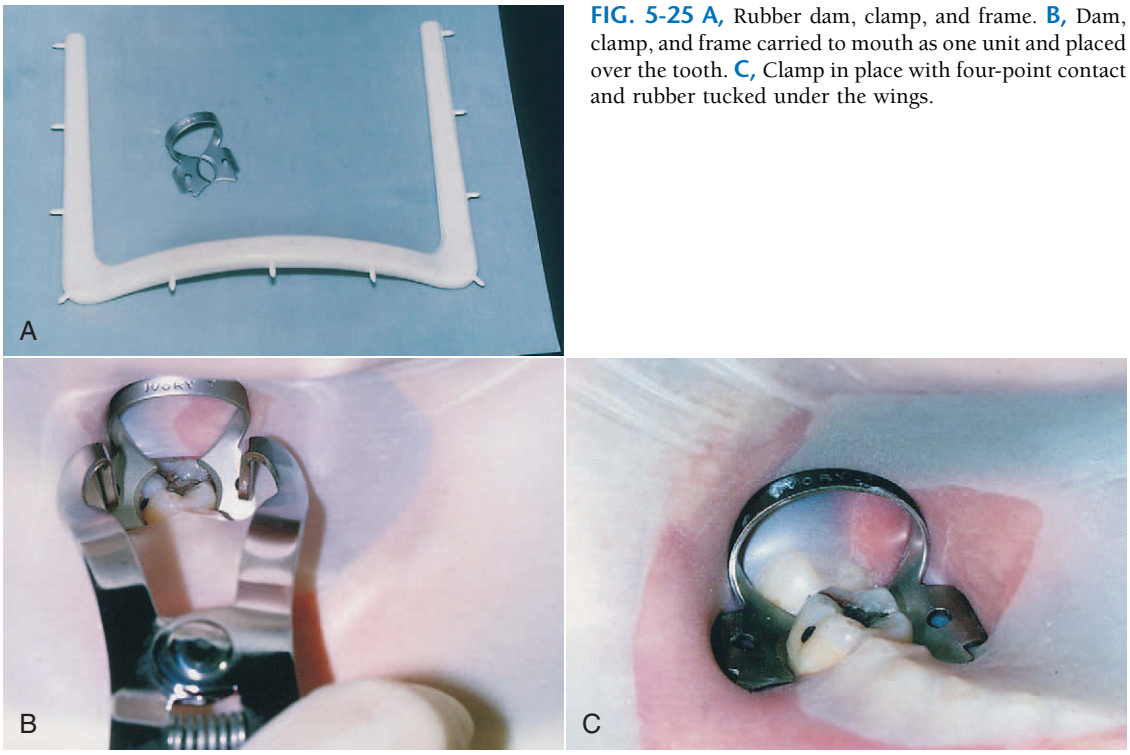


FIG. 5-25 A, Rubber dam, clamp, and frame. **B,** Dam, clamp, and frame carried to mouth as one unit and placed over the tooth. **C,** Clamp in place with four-point contact and rubber tucked under the wings.

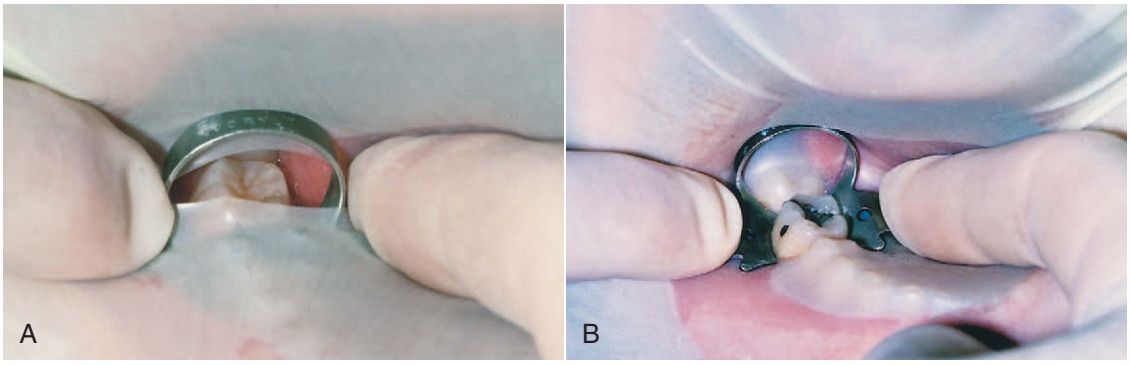


FIG. 5-26 A, After the clamp is placed, the dam is attached to the frame and gently stretched over the clamped tooth with the index finger of each hand. **B,** The clamp is tested for a secure fit with gentle finger pressure (alternately) on the buccal and lingual aspects of the clamp apron.

In the split-dam method (Fig. 5-27), two overlapping holes are punched in the dam. A cotton roll is placed under the lip in the mucobuccal fold over the tooth to be treated. The rubber dam is stretched over the tooth to be treated and over one adjacent tooth on each side. The edge of the dam is carefully teased through the contacts on the distal sides of the two adjacent teeth. Dental floss helps carry the dam down around the gingiva. The tension produced by the stretched dam, aided by the rubber dam frame, secures the dam in place. The tight fit and the cotton roll help produce a relatively dry field. If the dam has a tendency to slip, a premolar clamp may be used on a tooth distal to the three isolated teeth or even on an adjacent tooth (see Fig. 5-27, *A*). The clamp is placed over the rubber dam, which then acts as a cushion against the jaws of the clamp.

Aids in Rubber Dam Placement

Punching and Positioning of Holes

The rubber dam may be divided into four equal quadrants, and the proper place for the hole is estimated according to which tooth is undergoing treatment. The more distal the tooth, the closer to the center of the dam the hole is placed. This method becomes easier as the clinician gains experience. The hole must be punched cleanly, without tags or tears. If the dam is torn,

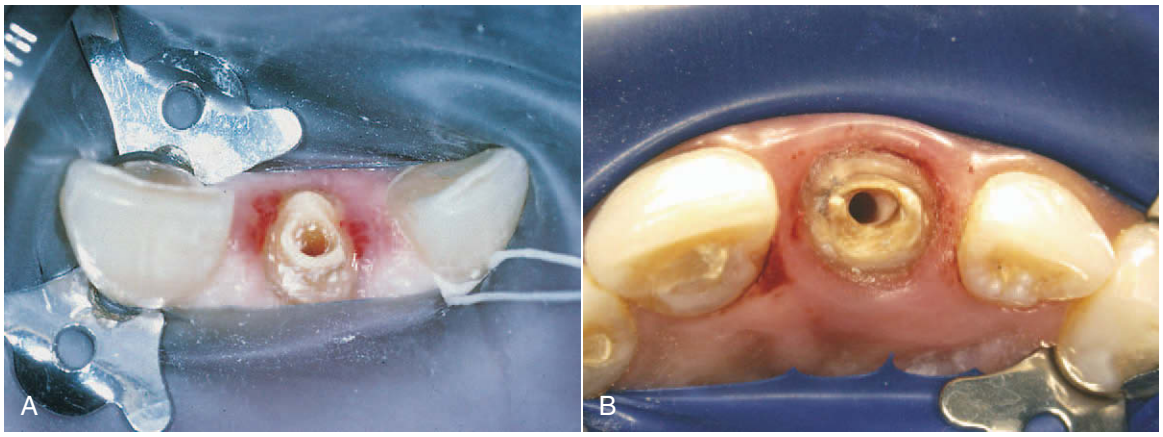


FIG. 5-27 Split-dam technique. **A,** Premolar clamp on maxillary central incisor along with ligation on the maxillary canine prevents dam slippage and aids in dam retraction during endodontic treatment on broken-down maxillary lateral incisor. **B,** Split dam used during post removal and retreatment of a maxillary central incisor. (***A,*** *Courtesy Dr. James L. Gutmann;* ***B,*** *courtesy Dr. Francisco A. Banchs.)*

it may leak or permit continued tearing when stretched over the clamp and tooth.

Orientation of the Dam and Bunching

The rubber dam must be attached to the frame with enough tension to retract soft tissues and prevent bunching, without tearing the dam or displacing the clamp. The rubber dam should completely cover the patient's mouth without infringing on the patient's nose or eyes. To prevent bunching of the dam in the occlusal embrasure, only the edge of the interseptal portion of the dam is teased between the teeth. Dental floss is then used to carry the dam through the contacts. These contacts should always be tested with dental floss before the dam is placed. A plastic instrument is used to invert the edge of the dam around the tooth to provide a seal.

Problem Solving in Tooth Isolation

Leakage

The best way to prevent seepage through the rubber dam is meticulous placement of the entire system. Proper selection and placement of the clamp; sharply punched, correctly positioned holes; use of a dam of adequate thickness; and inversion of the dam around the tooth all help reduce leakage through the dam and into the root canal system.[6,37,46,65] Nevertheless, there may be clinical situations in which small tears, holes, or continuous minor leaks may occur. These often can be patched or blocked with Cavit (3M), OraSeal caulking (UltraDent Products, South Jordan, UT), rubber base adhesive,[9] "liquid" rubber dam, or periodontal packing. If leakage continues, the dam should be replaced with a new one.

Because salivary secretions can seep through even a well-placed rubber dam, persons who salivate excessively may require premedication to reduce saliva flow to a manageable level. Failure to control salivation may result in salivary contamination of the canal system and pooling of saliva beneath the dam, as well as drooling and possible choking. Such occurrences can disrupt treatment and should be prevented. Excessive saliva flow can be reduced with an anticholinergic drug, such as atropine sulfate, propantheline bromide (Pro-Banthine), methantheline (Banthine), or glycopyrrolate (Robinul).[34] Therapeutic doses of atropine sulfate for adults range from 0.3 to 1 mg *per os,* 1 to 2 hours before the procedure. The synthetic anticholinergic drug propantheline bromide reportedly has fewer side effects than methantheline.[34] The usual adult dose of propantheline bromide for an adult is 7.5 to 15 mg, taken orally 30 to 45 minutes before the appointment. Because anticholinergics can cause undesirable autonomic effects, especially through various drug interactions, they should be used only in specific cases and only as a last resort.

Unusual Tooth Shapes or Positions That Cause Inadequate Clamp Placement

Some teeth do not conform to the variety of clamps available. These include partially erupted teeth, teeth prepared for crowns, and teeth fractured or broken down to the extent that their margins are subgingival. To handle these cases, rubber dam retainers may be customized by modifying the jaws to adapt to a particular tooth (Fig. 5-28).[69] In partially erupted teeth or cone-shaped teeth, such as those prepared for full coverage, one technique[68] is to place spots of self-curing resin on the cervical surface of the tooth. These resin beads act as a scaffold for the retainer during treatment. Another method[32] is to place small acid-etched composite lips on the teeth; these resin lips serve as artificial undercuts and remain on the teeth between appointments. When the root canal treatment is complete, the resin beads are easily removed. In multiple-treatment cases involving misshapen teeth, a customized acrylic retainer[63] can be used in conjunction with a dam to isolate the operating field.

Loss of Tooth Structure

If insufficient tooth structure prevents the placement of a clamp, the clinician must first determine whether the tooth is periodontally sound and restorable. Meticulous and thorough treatment planning often can prevent embarrassing situations for both the doctor and patient. One example is the case in which the endodontic treatment is completed before restorability is determined; it is then discovered that the tooth cannot be restored.

Once a tooth is deemed restorable but the margin of sound tooth structure is subgingival, a number of methods should be

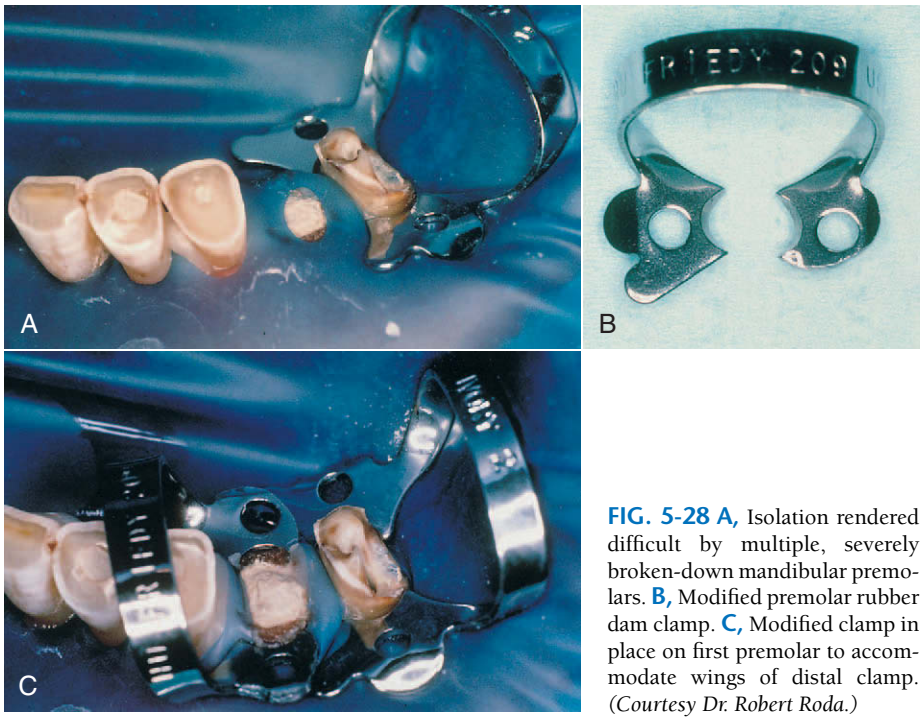


FIG. 5-28 A, Isolation rendered difficult by multiple, severely broken-down mandibular premolars. **B,** Modified premolar rubber dam clamp. **C,** Modified clamp in place on first premolar to accommodate wings of distal clamp. *(Courtesy Dr. Robert Roda.)*

considered. As mentioned earlier, less invasive methods, such as using a clamp with prongs inclined apically or using an S-G clamp, should be attempted first (see Fig. 5-24). If neither of these techniques effectively isolates the tooth, the clinician may consider clamping of the attached gingiva and alveolar process. In this situation it is imperative that profound soft tissue anesthesia exists before clamp placement. Although the procedure may cause some minor posttreatment discomfort, the periodontal tissues recover quickly with minimal posttreatment care.

Restorative Procedures

If none of the techniques mentioned previously is desirable, a variety of restorative methods may be considered to build up the tooth so that a retainer can be placed properly.[45,46,65] A preformed copper band, a temporary crown, or an orthodontic band (Figs. 5-29 and 5-30) may be cemented over the remaining natural crown. This band or crown not only enables the clamp to be retained successfully but also serves as a seal for the retention of intracanal medicaments and the temporary filling between appointments, if more than one appointment is necessary. These temporary bands or crowns have several disadvantages. One of their main problems is their inability to provide a superior seal. Another concern is that particles of these soft metals or cement can block canal systems during access opening and instrumentation. Third, these temporary crowns and bands, if they become displaced or are not properly contoured, can cause periodontal inflammation.

On occasion, so little tooth structure remains that even band or crown placement is not possible. In these cases it becomes necessary to replace the missing tooth structure to facilitate placement of the rubber dam clamp and prevent leakage into the pulp cavity during the course of treatment.[45,46,65] Replacement of missing tooth structure can be accomplished with composite resins; glass ionomer cements, such as Ketac-Silver (3M, St. Paul, MN), Fuji II (GC America, Alsip, IL) (Fig. 5-31), or Photac-Fil (3M); or dentin-bonding systems, such as Adper Scotchbond (3M), the Tenure bonding system (Den-Mat, Santa Maria, CA), Gluma (Heraeus Kulzer, South Bend, IN), OptiBond (Kerr, Orange, CA), PermaQuik (UltraDent Products, South Jordan, UT), or C&B-Metabond (Parkell, Edgewood, NY).[65] Although these newer dentin-bonding systems form a strong, immediate bond and are generally simple to use, any restorative method for building up a broken-down tooth is time-consuming, can impede endodontic procedures, and may duplicate restorative treatment. Many restorations that have been hollowed out by access cavities are weakened and require redoing.

Canal Projection

Canal projection is a technique that facilitates preendodontic buildup of broken-down coronal and radicular structure while preserving individualized access to the canal.[42] Although any of a number of syringeable materials (glass ionomer cements, temporary cements, permanent cements, etc.) and even "packable" composites may be used to project the canals, bonded injectable autopolymerizing composites have proven to be the most versatile and reliable buildup material for this technique (Fig. 5-32). The advantages of the technique are numerous:

Isolation: When deep coronal–radicular defects prevent clamp retention, a tooth is often considered to be endodontically untreatable. Canal projection essentially replaces missing structures to facilitate clamp retention, rendering many structurally debilitated teeth endodontically treatable.

Seal of chamber floor: The accessory canals that exit the chamber floor into the furcation are well documented and can conduct inflammatory agents from the deteriorating pulpal system into the furcation. In addition, when caries or

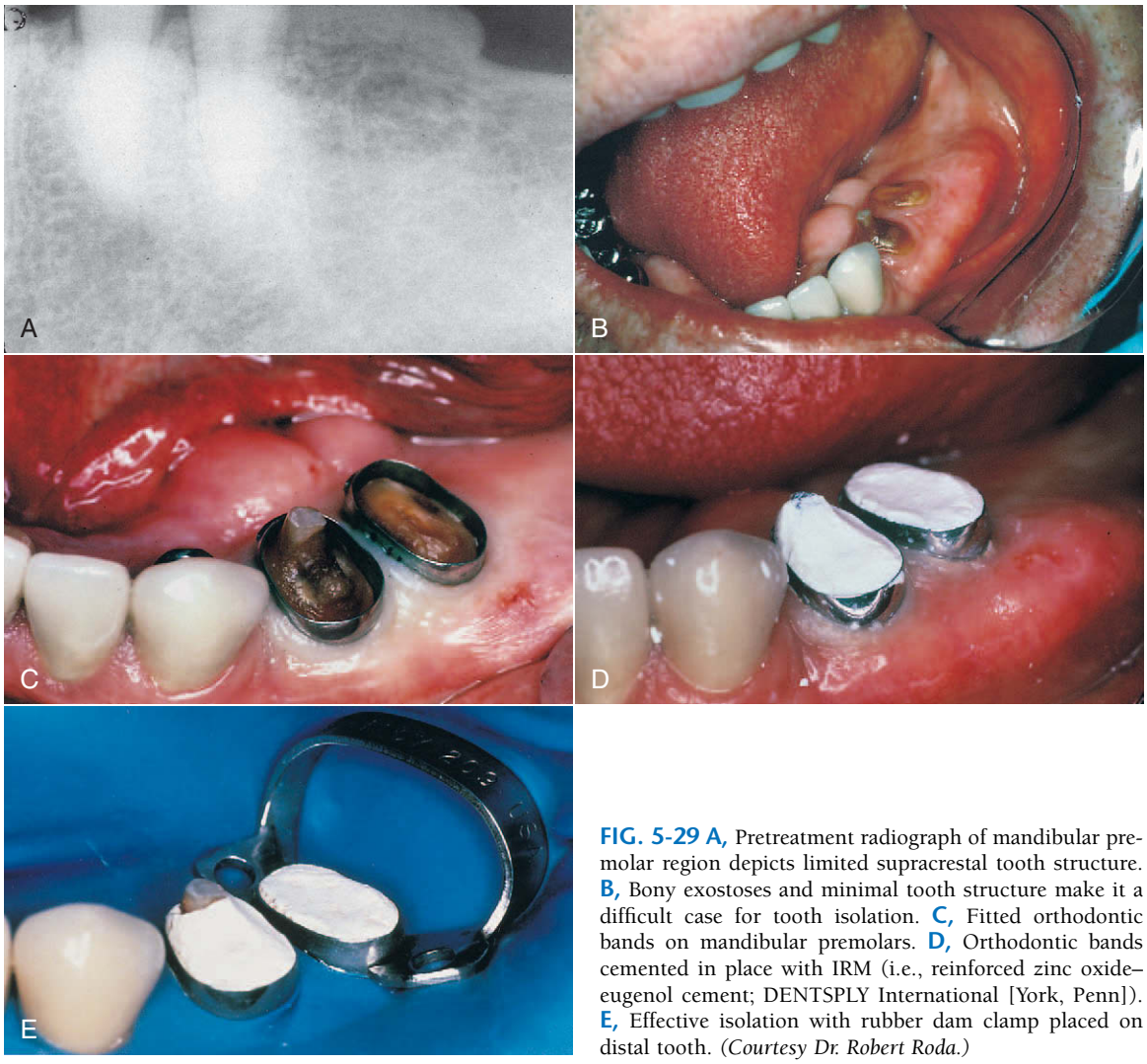


FIG. 5-29 A, Pretreatment radiograph of mandibular premolar region depicts limited supracrestal tooth structure. **B,** Bony exostoses and minimal tooth structure make it a difficult case for tooth isolation. **C,** Fitted orthodontic bands on mandibular premolars. **D,** Orthodontic bands cemented in place with IRM (i.e., reinforced zinc oxide–eugenol cement; DENTSPLY International [York, Penn]). **E,** Effective isolation with rubber dam clamp placed on distal tooth. *(Courtesy Dr. Robert Roda.)*

exploratory procedures lead to a paper-thin chamber floor, the risk of contaminants extending into the furcal apparatus increases significantly. Often such complicated cases require multiple visits, and the interappointment interval with customary temporary coronal seal provides opportunities for serious furcation degradation, particularly if a patient does not return in a timely fashion for completion of treatment. Sealing the chamber floor early in treatment greatly reduces this risk. As an initial step in the endodontic process, canal projection using bonded composite achieves this early objective.

Elongation of canals: The canal projection process elongates the "hydraulic chamber" of each canal from the access cavity floor to the occlusal surface, offering advantages during the hydraulic condensation of obturation materials. Essentially all currently accepted warm vertical compaction techniques (conventional warm vertical compaction, warm sectional compaction, and continuous wave compaction) rely on the rheology that takes place between the chamber floor and the primary exit of the canal. These methods focus their hydraulic effects on the apical region while only incidentally addressing those accessory and lateral canals that exit from the more coronal aspects of the system. In contrast, when the canal is projected, the hydraulic chamber is elongated as much as 5 to 10 mm such that the rheology occurs not just between the chamber floor and the primary canal exit but also between the occlusal surface and the primary exit. This allows for additional warm vertical compaction "strokes," additional warm "sections" of gutta-percha, or additional milliseconds spent in the continuous wave of compaction. Regardless of which hydraulic condensation method is used, canal projection provides greater opportunity to address coronally positioned exits.

Overlay of perforation repair: Early repair of perforation defects can have a significant effect on success of perforation treatment. As such, performing perforation repair before completion of endodontic therapy is often preferable. The delicate nature of these types of repairs, however, leaves them vulnerable to reaggravation during subsequent cleaning, shaping, and obturation procedures. It can be most disheartening to perform a complicated perforation repair procedure only to see it herniate into adjacent periodontal ligament and bone under obturation hydraulics. Delaying the repair until endodontic therapy is completed may put

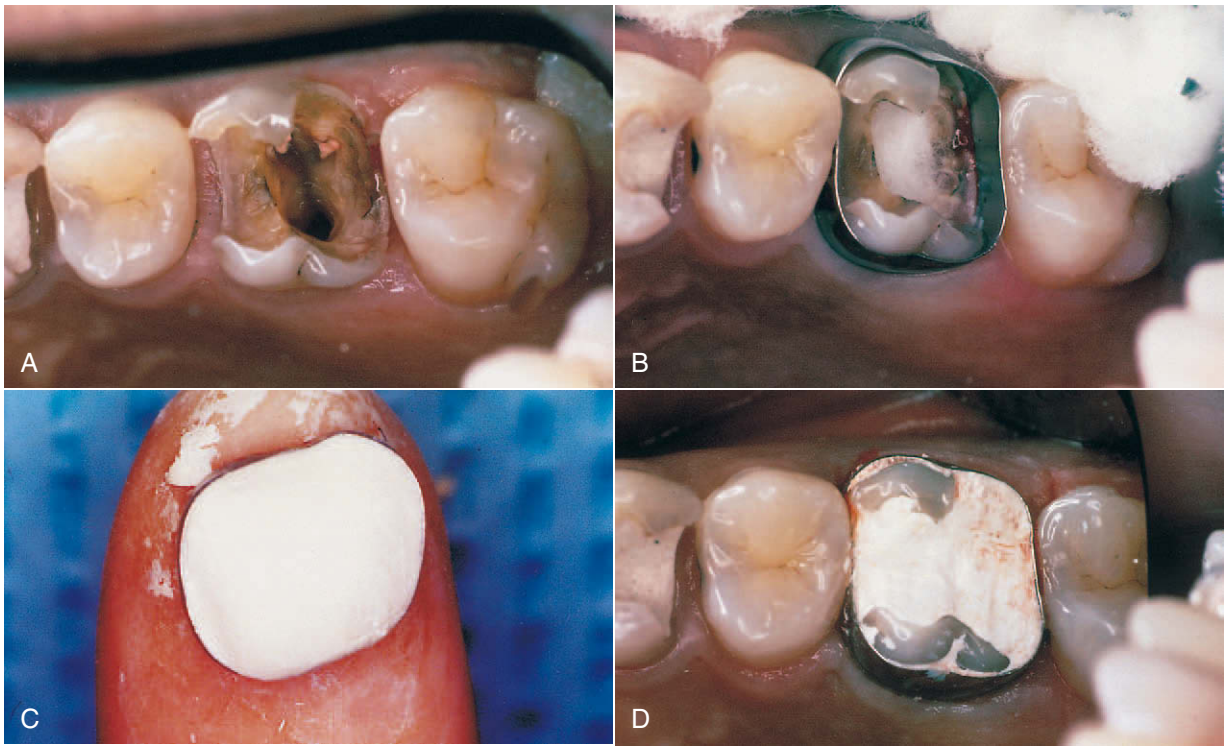


FIG. 5-30 A, Broken-down maxillary molar after removal of restoration, post, and caries. **B,** Fitted orthodontic band; cotton in access opening to protect orifices. **C,** IRM loaded into band before cementation. **D,** Completed temporary restoration before rubber dam placement. *(Courtesy Dr. Robert Roda.)*

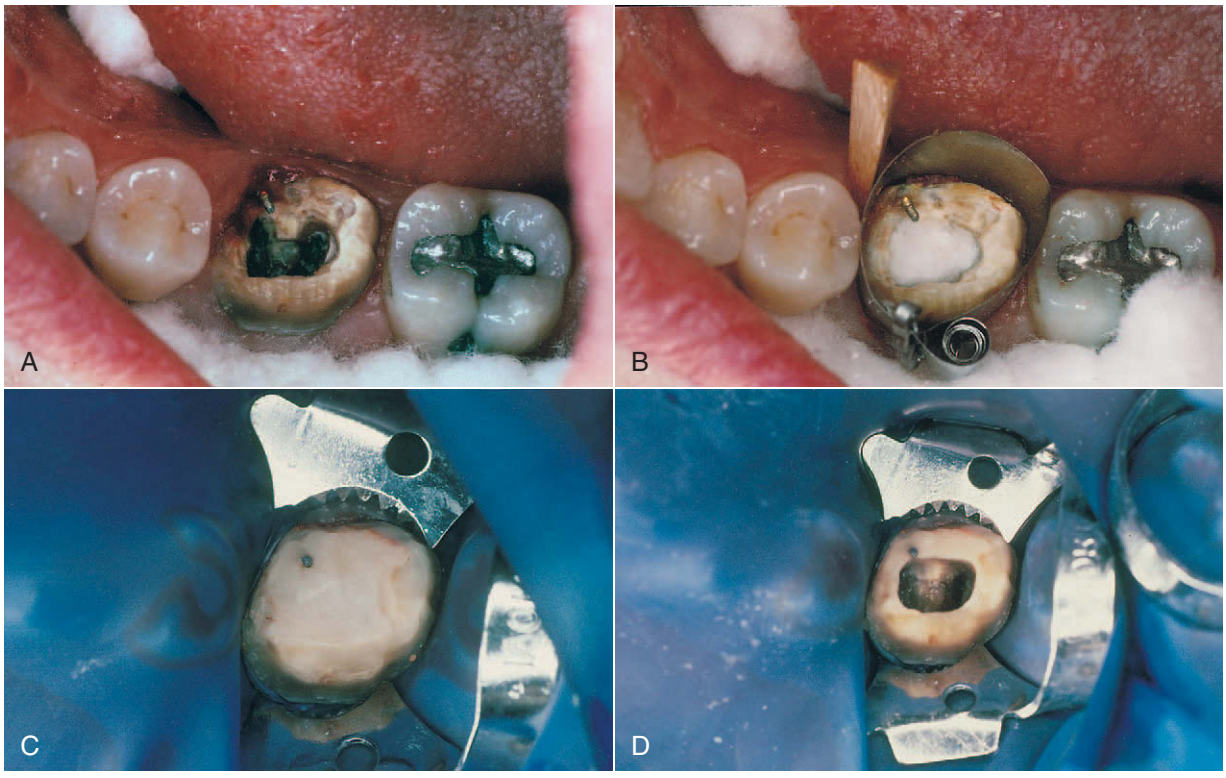


FIG. 5-31 A, Broken-down mandibular molar after crown and caries removal; preexisting pin will aid retention of restorative material. **B,** Isolation with wedged Automatrix. **C,** Completed temporary restoration using glass ionomer cement (Fuji II). **D,** Access through completed restoration after rubber dam placement. *(Courtesy Dr. Robert Roda.)*

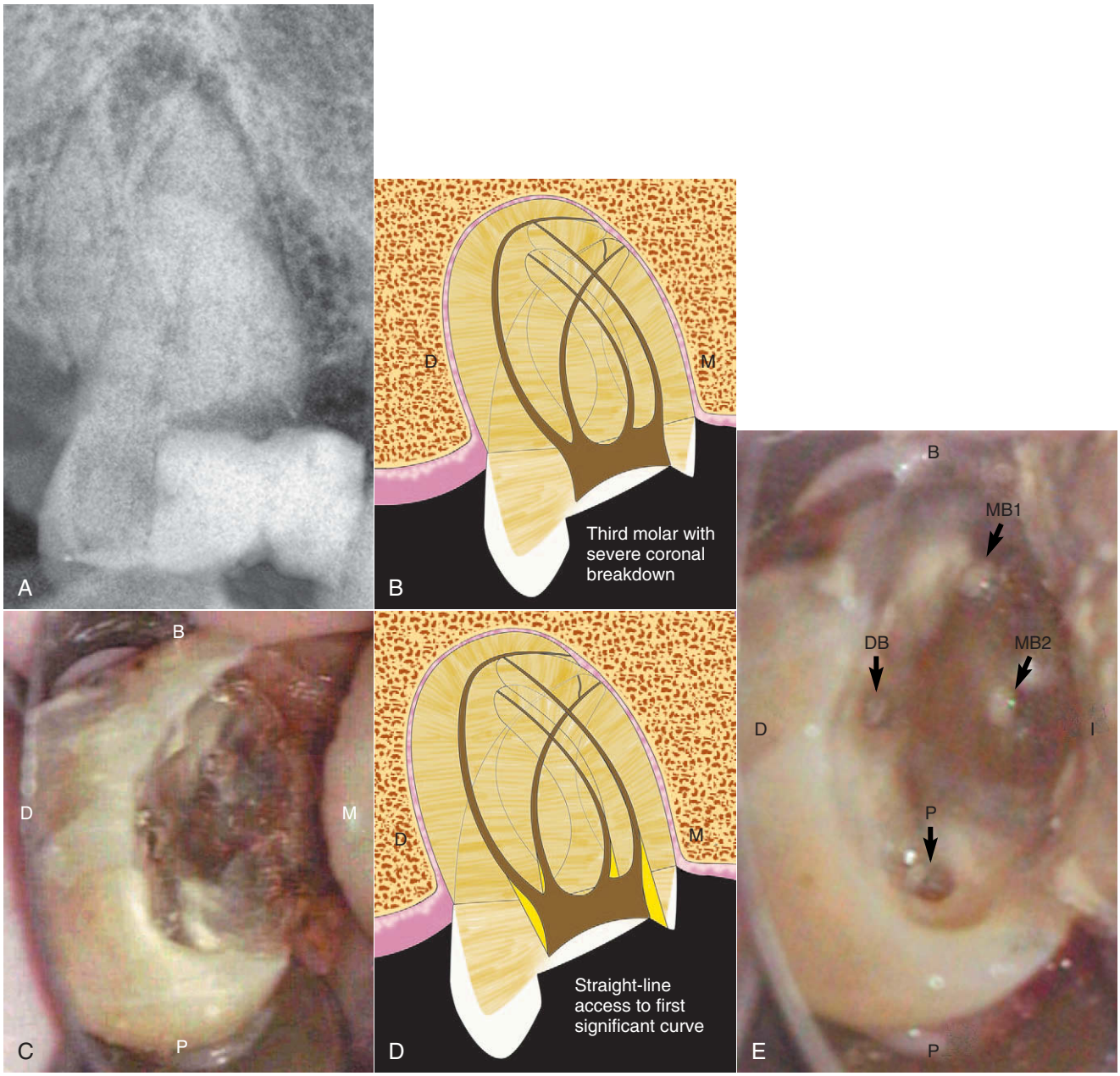


FIG. 5-32 Canal projection technique. **A-C,** In a tooth with severe coronal breakdown, after gaining access and locating all canals, it is essential to perform a thorough high-magnification examination of the floor and walls and to examine, as deeply as possible, all canals to ensure that all branchings and other possible irregularities have been identified before proceeding. **D,** In curved canals, straight-line access is created to the first significant curvature by removing dentin "triangles" with small, slow-speed round burs, working into the bulk of each root (i.e., working away from the furcal aspect of the root). **E,** If there is no significant curvature in a particular canal, then the orifice should be dimpled to the depth of a #2 slow-speed round bur. An appropriate matrix barrier is applied, and all bondable surfaces are etched and primed. Files are selected that will fit well into the body of each canal, and projectors (CJM Engineering, Santa Barbara, CA), tapered plastic sleeves available in various dimensions, are placed onto the files. The files are then inserted into the canals, and the projectors are slid apically until they are drawn into the seat of the previously prepared straight-line access or dimples.

the tooth at risk if the treatment cannot be completed in a single visit. In addition, performing endodontic therapy in the presence of an unrepaired perforation may needlessly expose the perforation site to irritating solvents and irrigants and to accidental penetration by instruments. In many cases, perforation repair with mineral trioxide aggregate (MTA) can be performed before completion of endodontic therapy.[28] After confirmation that the MTA has set, canal projection offers a means of fortifying the repair by overlaying it with a bonded resin or glass ionomer before proceeding with endodontic therapy.

Prevention of perforation: In those circumstances that lead to a paper-thin chamber floor, the risk of inadvertent perforation with a file, explorer, or bur can be reduced or

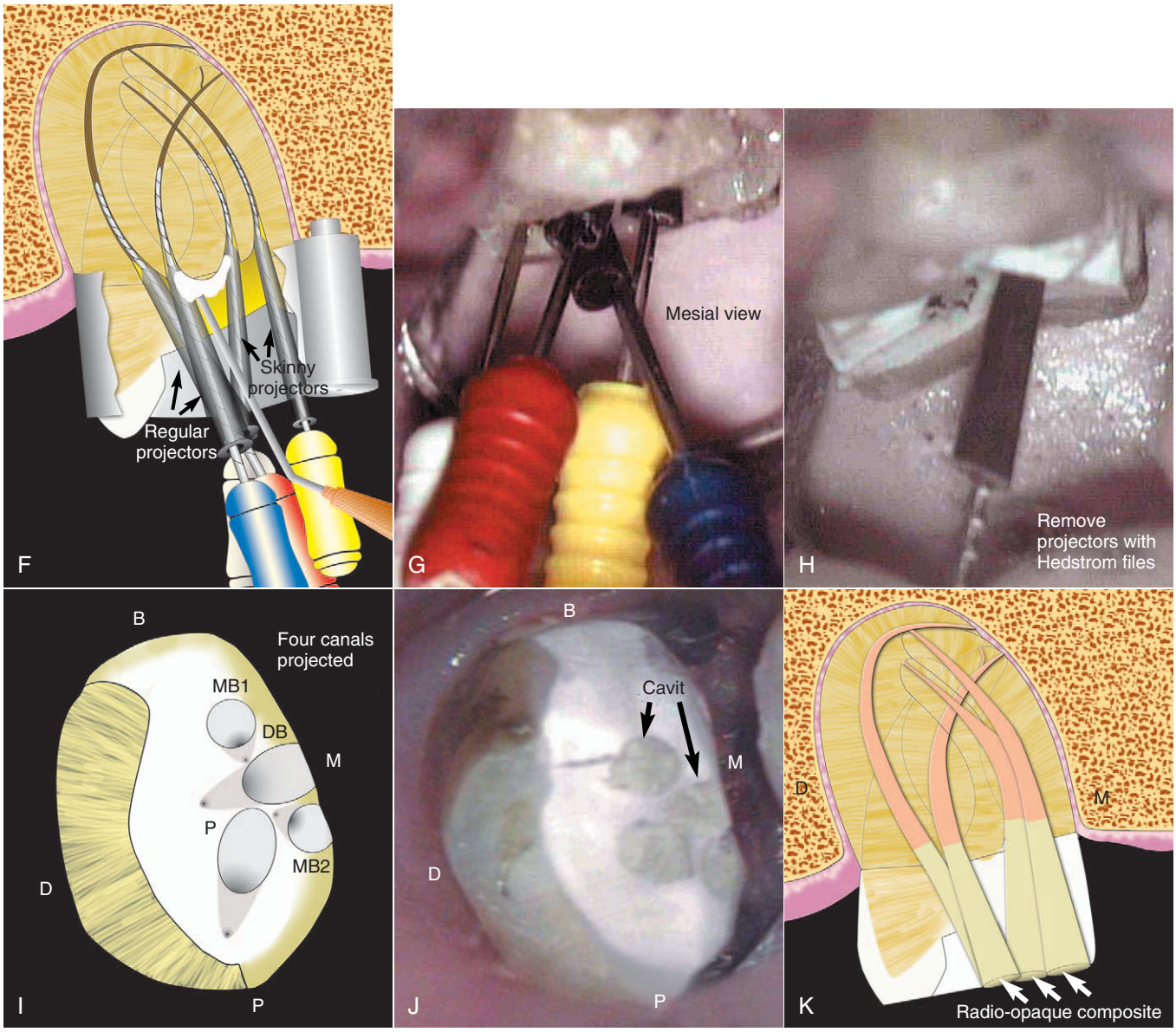


FIG. 5-32, cont'd F-G, In this particular example, regular projectors were used in the D and P canals and skinny projectors were used in the MB1 and MB2 canals to accommodate their proximity to each other. With the canals occupied by the files and projectors, autopolymerizing bonding agent is applied and distributed over all primed surfaces with a blast of air. Autopolymerizing composite buildup material is then injected from the chamber floor to the cavosurface of the access cavity. On early signs of polymerization, the files are removed, leaving the projectors embedded in the buildup material. A bull-nosed diamond is used to contour the composite and to flatten the occlusal surface along with the embedded projectors. Leaving the projectors embedded at this stage prevents composite debris from collecting in the projected canals. **H,** The projectors are removed by engaging a #60 Hedström file in the lumen of each projector and withdrawing. The external surfaces of projectors are treated with a releasing agent that provides for reliable removal from all types of buildup material. **I,** Depending on how the projectors are arranged relative to each other as they traverse from the chamber floor through the access cavity to the occlusal surface, the projected orifices will lie in various configurations on the occlusal surface. This procedure creates durable endodontic reference points. **J,** If a second treatment visit is required, the projected canals may be conveniently sealed by snipping 2 mm from the large-diameter end of the projectors, reinserting them into their respective canals, and sealing with Cavit. On the patient's return, the Cavit seals are removed with a #6 slow-speed round bur, a #60 Hedström file is again engaged in the lumen of each projector, and the projectors are withdrawn, leaving the canals free of temporary filling debris. **K,** After cleaning, shaping, and obturation, the projected portions of the canals are "dressed" with a high-speed football diamond to remove residual sealer and to freshen the composite, making it receptive to the final composite that is injected directly over gutta-percha from the subchamber level to the cavosurface of each projected canal.

Continued

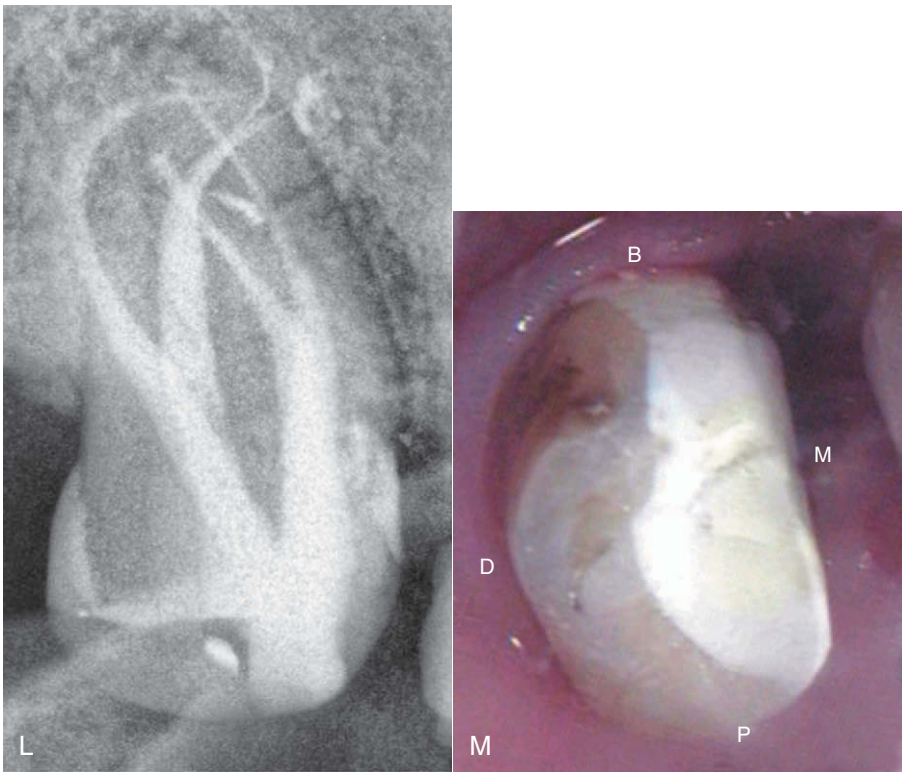


FIG. 5-32, cont'd L, Selecting a composite for this final step that is more radiopaque than the buildup composite creates a unique radiographic appearance showing the canal continuously flowing from the root end to the occlusal surface. **M,** Crown preparation can proceed immediately. A post can be easily placed through the projection buildup at the completion of endodontic therapy. Using either a high-speed football diamond or a slow-speed #8 round bur, the projected portion of the canal to receive the post is enlarged to the level of the chamber floor. According to the particular type of post to be placed, the radicular aspect of the canal is prepared in the typical fashion, and the post is cemented or bonded into the canal. The coronal portion of the post should be surrounded by syringeable composite, effectively anchoring the buildup to the natural tooth structure. *D,* Distal; *M,* mesial; *B,* buccal; *P,* palatal; *DB,* distobuccal; *MB1,* main mesiobuccal canal; *MB2,* second mesiobuccal canal. *(Courtesy Dr. C. John Munce.)*

eliminated by placing a preendodontic buildup via canal projection.

Recontour of irregularities on chamber floor and walls: Irregularities on the access cavity floor and walls can lead to complications during treatment. Misdirected access cavities, divots in walls, misdirected post preparations and ledges, and so on can be "corrected" by essentially reconstructing the walls and floor around "internal matrix barriers" via canal projection.

Reduced potential for crack initiation or propagation: On posterior teeth in particular, the period between initiation of endodontic therapy and placement of a restoration that provides cuspal protection can introduce the risk of coronal–radicular fracture—the longer the time period, the greater the risk. Bonded coronal buildup has been shown to reduce this risk.[24,35] Bonded canal projection performed at the first endodontic treatment visit imparts a measure of protection against the development of a fracture from the time treatment is initiated until the tooth is permanently restored. In teeth that present with incipient fractures, early placement of a bonded buildup can reduce the likelihood of propagation of these fractures as well.

Prevention of ingrowth of tissues: When coronal or radicular defects extend to axial walls at or beyond the gingival level, tissue ingrowth leads to contamination during endodontic therapy and subsequent restorative procedures. Preendodontic buildup, as with canal projection, eliminates this complexity.

Individualization of canals: In multicanal teeth, as the projectors pass through the buildup material to the occlusal surface, they can be oriented such that they are slightly separated from each other before the buildup material polymerizes. This can simplify management of canals that lie in close proximity to each other on the chamber floor. It also provides for individualization of the canals so that specific solvents, irrigants, medicaments, lubricants, and other agents can be placed into specific canals.

Elimination of "blind exploration" on the chamber floor and improvement of mechanics related to handpiece-driven files: Once all canals have been located, straight-line access created, and the canals projected to the occlusal surface of the buildup, the resultant projected orifices will lie clearly visible on the surface of the projection buildup, no longer obscured by prominent marginal ridges and other visual obstructions. This also creates a significant advantage for entry of handpiece-driven files—particularly nickel–titanium files—into mesial canals of molars. By virtue of their increased long-axis dimension, the tips of handpiece-driven files can be difficult to introduce into mesial canals, and nickel–titanium files are even more difficult because they will not retain a bend. When the canals are projected onto the occlusal surface of the preendodontic buildup, however, it becomes a simple matter to place the file tip within the rim of the projected canal on the occlusal surface, and then by merely advancing the file, it is driven smoothly into the canal.

Periodontal Procedures

As a result of excessive crown destruction or incomplete eruption, the presence of gingival tissue may preclude the use of a clamp without severe gingival impingement. Various techniques of gingivectomy (Fig. 5-33) or electrosurgery have been suggested for cases in which the remaining tooth structure still

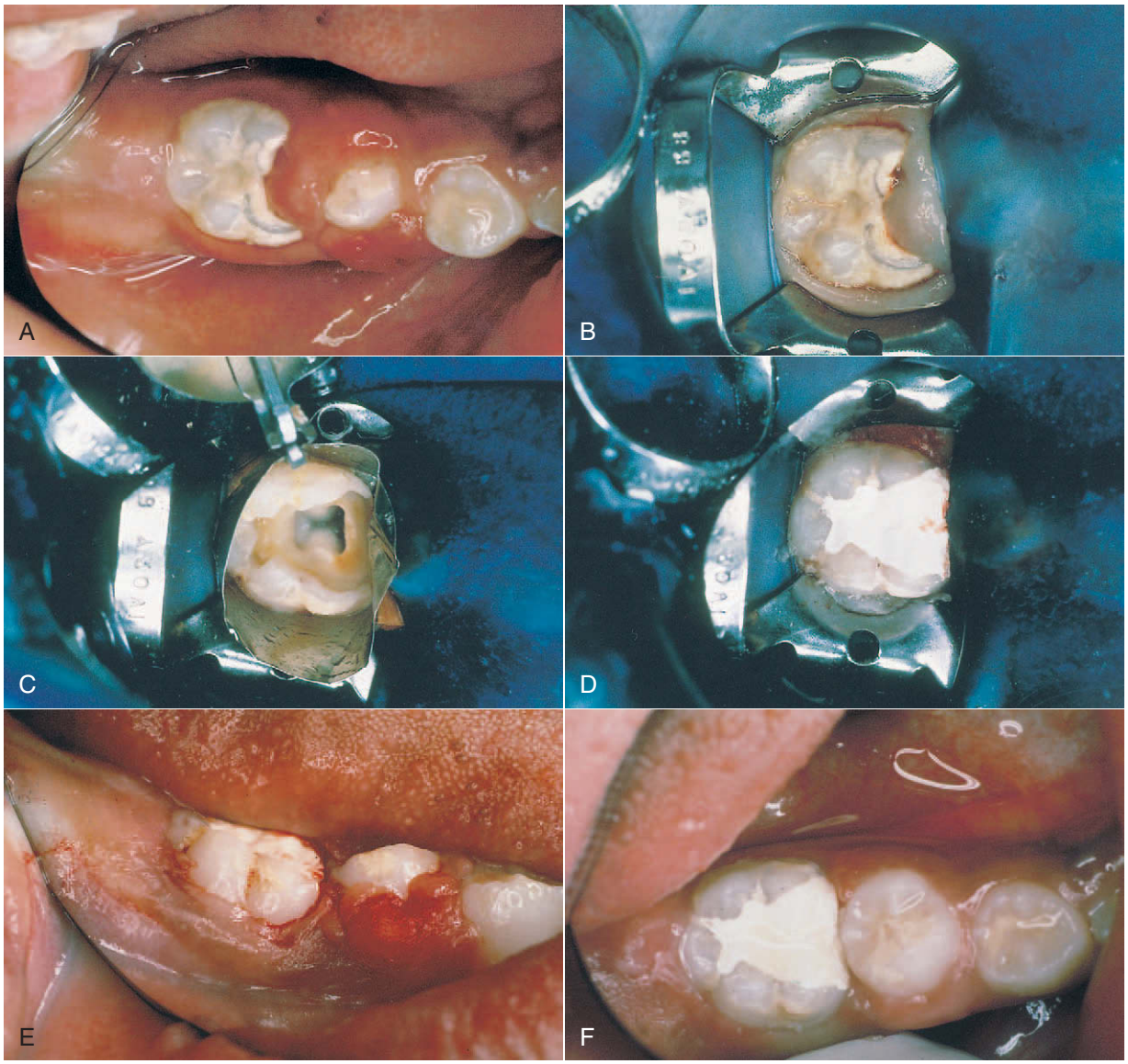


FIG. 5-33 A, Gingival hypertrophy on mandibular molar and erupting premolar of a young patient; mandibular molar requires root canal treatment. **B,** Rubber dam clamp impinging on gingival tissues; tissue removed with scalpel. **C,** Automatrix placed immediately after tissue removal; bleeding was minimal. **D,** Placement of IRM temporary restoration after pulpectomy. **E,** Posttreatment facial view immediately after gingivectomy; note hemostasis. **F,** Six-week posttreatment occlusal view exhibits fully exposed mandibular molar and recently erupted premolar. (*Courtesy Dr. Robert Roda.*)

lies above the crestal bone. With an inadequate zone of attached gingiva, osseous defects, or a poor anatomic form, an apically positioned flap with a reverse bevel incision is the technique of choice to "lengthen" the crown.[45,46]

Electrosurgery and the conventional gingivectomy are crown-lengthening procedures for teeth that have sufficient attached gingiva and no infrabony involvement.[45,46] The electrosurgery method offers the advantage of leaving a virtually bloodless site for immediate rubber dam placement. Electrosurgery units have become highly sophisticated and are capable of providing both cutting and coagulating currents that, when used properly, will not cause cellular coagulation. The wide variety of sizes and shapes of surgical electrodes enables the clinician to reach areas inaccessible to the scalpel. Further, electrosurgery facilitates the removal of unwanted tissue in such a manner as to recreate normal gingival architecture. This feature, combined with controlled hemostasis, makes the instrument extremely useful in the preparation of some teeth for placement of the rubber dam clamp.

The main drawback of electrosurgery is the potential for damage to the adjacent tissues; if the electrode contacts bone, significant destruction of bone can occur. As a result this technique is not recommended when the distance between the crestal level of bone and the remaining tooth structure is minimal. Compared with electrosurgery, conventional gingivectomy presents the major problem of hemorrhage after the procedure; this forces delay of endodontic treatment until tissues have healed.

The apically positioned flap[45,46] is a crown-lengthening technique for teeth with inadequate attached gingiva, infrabony pockets, or remaining tooth structure below the level of crestal bone. With this technique as well, endodontic treatment should be delayed until sufficient healing has occurred.

Orthodontic Procedures

The most common indication for orthodontic extrusion is a fracture of the anterior tooth margin below the crestal bone.[45,46] The clinician should be aware that, because bone and soft tissue attachments follow the tooth during extrusion, crown-lengthening procedures after extrusion are often necessary to achieve the desired clinical crown length and restore the biologic and aesthetic tissue relationships. Ultimately, the purpose of orthodontic extrusion is to erupt the tooth to provide 2 to 3 mm of root length above crestal bone level.

SUMMARY

Success in endodontic therapy is predicated on a host of factors, many of which are controllable before the clinician ever initiates treatment. Proper and thorough preparation of both patient and tooth for endodontic treatment should lay the groundwork for a relatively trouble-free experience that will increase the chances for the ultimate success of the entire treatment.

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CHAPTER

6

Armamentarium and Sterilization

PAUL D. ELEAZER

CHAPTER OUTLINE

SELECTION OF THE ARMAMENTARIUM
PRETREATMENT STERILIZATION AND DISINFECTION
TREATMENT
GENERAL CONSIDERATIONS FOR THE OFFICE
FUTURE CONSIDERATIONS

Preparation for endodontic treatment requires a somewhat different approach compared with many other types of dental therapy. Endodontic treatment breaks through the body's primary protective barrier, the integumentary system. Teeth are a specialized part of this system that also includes skin and mucous membranes; collectively, the integumentary system serves to protect humans from invasion by microbes, among other functions. This barrier is breached when caries, trauma, or other insults provide entry into the pulpal space of a tooth. Sterile instruments and proper technique are needed to avoid contamination by microorganisms or noxious chemicals during endodontic therapy.

The well-prepared clinician realizes that a posttreatment infection could be caused by a break in sterile technique. The astute clinician realizes that time spent before treatment in the organization and sterilization of instruments and supplies will lead to fewer posttreatment patient problems and result in a more efficient practice.

In addition to the need for impeccable sterility, endodontics differs from many other aspects of dentistry. Access and visibility are severely limited by the nature of the irregular and branching canal spaces within the roots. Individual variation means that clinicians cannot know exactly what condition is present throughout the entire root canal system (see Chapter 7 for more details). Enhanced lighting and magnification have resolved visual access problems to some extent (Figs. 6-1 to 6-4). Nevertheless, limitations remain and there continues to be a need for special instruments and techniques.

SELECTION OF THE ARMAMENTARIUM

Preparation for treatment begins with selection of the armamentarium for routine endodontic patient care. Instruments used for every patient should be assembled and sterilized before use. Box 6-1 lists a typical armamentarium that can be customized for the individual clinician (Figs. 6-5 to 6-7).

Root canal instruments of appropriate length and type, including files, reamers, broaches, and specialized burs, may be sterilized separately because variables (e.g., tooth length, canal size, and clinical diagnosis) may affect this part of the armamentarium. Additional items that are needed occasionally (e.g., chlorhexidine and an intraosseous injection kit) should be in sterile containers and readily available to the dental assistant (Figs. 6-8 and 6-9). This approach provides a basic endodontic setup tray with optional items sterilized and ready for use in individual cases. The overarching philosophical approach is to develop a standard setup for all patients and to have readily available specialized kits for specific procedures or clinical situations.

PRETREATMENT STERILIZATION AND DISINFECTION

The next phase after selection of the armamentarium is to develop a pretreatment sterilization plan. *Sterile* is defined as the absence of life forms. *Disinfection* means killing most life forms, especially pathogens. As commonly used, the term *disinfection* does not include killing spores. Manufacturers' claims for disinfectants are based on performance against *Mycobacterium*, which is the genus that includes the species that causes tuberculosis (TB). For assessing virucidal efficacy, the astute clinician should be aware that agents that kill the hardy hepatitis B virus will likely be effective at destroying all viruses.

The Centers for Disease Control and Prevention (CDC, Atlanta, GA) released new guidelines for sterilization in 2003, and these are available on the Internet (http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5217a1.htm).[12] Box 6-2 lists the elements of a typical sterilization plan. These guidelines clarify the need for staff training programs to ensure that sterilization procedures are effective. Specific items include weekly monitoring of the effectiveness of the process with biologic monitors, for example, packets of spores are placed in the sterilizer



FIG. 6-1 Surgical telescopes (loupes). *(Courtesy Orascoptic Research, Middleton, WI.)*



FIG. 6-2 Light for loupes. *(Courtesy High Q Dental, Scottsdale, AZ.)*



FIG. 6-3 Microscope. *(Courtesy Global Surgical, St. Louis, MO.)*



FIG. 6-4 Orascope unit. *(Courtesy Dr. James Bahcall.)*

BOX 6-1

Armamentarium for Routine Endodontic Patient Care

- Mirror
- Periodontal probe and standard dental explorer
- Anesthetic syringe
- Rubber dam instruments
  - Rubber dam punch
  - Rubber dam clamp holder
  - Rubber dam frame
- High-speed handpiece and burs
- Low-speed handpiece and burs
- Long shank excavator (31L)
- Canal-irrigating syringe(s), with container(s) for solution(s)
- Mixing spatula and mixing surface
- Obturation instruments
- Plastic instrument for temporary filling

for one cycle. The spores are then placed in growth medium to ensure that all have been killed. Overpacking the sterilizer is a common reason for inadequate sterilization because steam cannot penetrate as well. Thus assessment of the effectiveness of the sterilization plan is an essential feature of pretreatment planning (Fig. 6-10). The results of weekly monitoring should be kept in a log.

Monitoring the sterilizer by watching the gauges or relying on printouts of temperature, pressure, and time helps to ensure patient safety, but weekly biologic monitoring guards against sensor failure. Heat-sensitive indicators, such as special tapes that change color when exposed to sterilizer temperatures, are

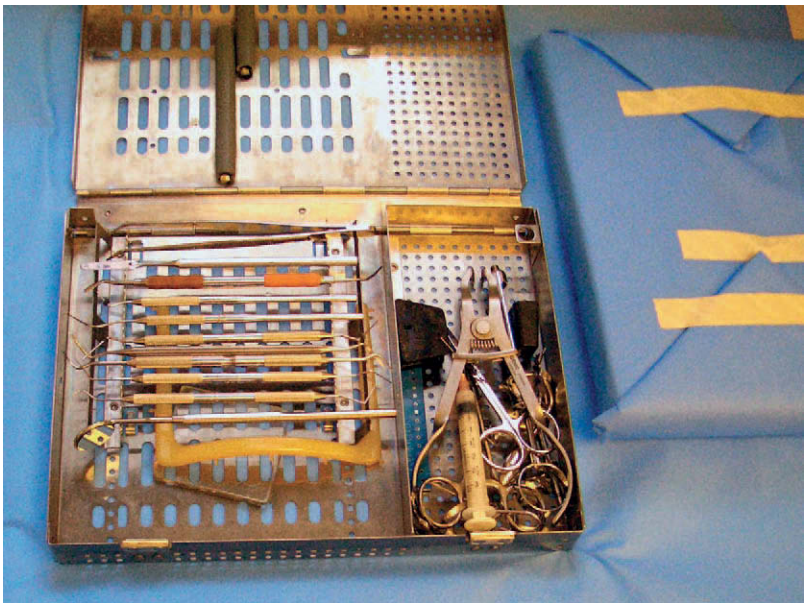


FIG. 6-5 IMS (instrument management system) cassette. *(Courtesy Hu-Friedy, Chicago, IL.)*



FIG. 6-6 Cassette wrapped in porous autoclave paper in preparation for sterilization. *(Courtesy Hu-Friedy, Chicago, IL.)*

also ineffective to ensure proper sterilization because these heat-sensing indicators merely show that the desired temperature has been reached, not that sufficient time has passed for proper killing. Accordingly, they are best used to simply record that the package was autoclaved; they should not be interpreted to indicate sterility.

Further guidelines about sterilizers include drying of instruments before removing them from the sterilizer. Wet wraps may tear easily or allow microbes to reach the sterile contents by wicking through the wrap. In addition, cooling avoids thermal injury to personnel. Some of the new sterilizers work faster because they vacuum air from the chamber before sterilization; these models also cool faster because of a second vacuum cycle after sterilization.

Another guideline reinforces the previous policy that the pathway from the sterilizer to the point of use should not cross the path of contaminated instruments as they are brought from the treatment area, cleaned, and packaged for resterilization. Thus separate areas are required for cleaning/sorting/packaging and for sterilization and cool down.

BOX 6-2

Elements of a Sterilization Plan

1. Transporting contaminated items from the operatory
   a. Items must be stored in a container to prevent perforation
2. Instrument-processing area
   a. Instruments must be sorted, cleaned (e.g., removal of all visible debris by scrubbing, ultrasonic cleaner), and rinsed with water in an area separate from sterilization
   b. Instruments should be inspected, assembled into trays, and wrapped for sterilization in packages containing sterilization indicator
   c. Instrument packages should be sterilized according to approved methods
   d. Instruments should be stored for event-related use
3. Environmental infection control
   a. Clinical contact surfaces (barrier protection, spray disinfectants)
   b. Dental unit water lines
   c. Housekeeping surfaces
4. Nonregulated and regulated medical waste
5. Monitoring plan
6. Training plan

Modified from Kohn WG, Collins AS, Cleveland JL, et al: Centers for Disease Control and Prevention (CDC): Guidelines for infection control in dental health-care settings—2003. MMWR Recomm Rep 52(RR-17):1, 2003. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5217a1.htm.

Approved methods of sterilization include pressurized steam (autoclaving), pressurized heated chemicals, dry heat, and cold chemicals (Figs. 6-11 to 6-13). Cold chemicals are not favored because the time cycle is difficult to monitor; they are recommended only for those items that cannot be heat sterilized. Room temperature chemical sterilants are not time efficient enough to be popular in dentistry. Cold sterilants

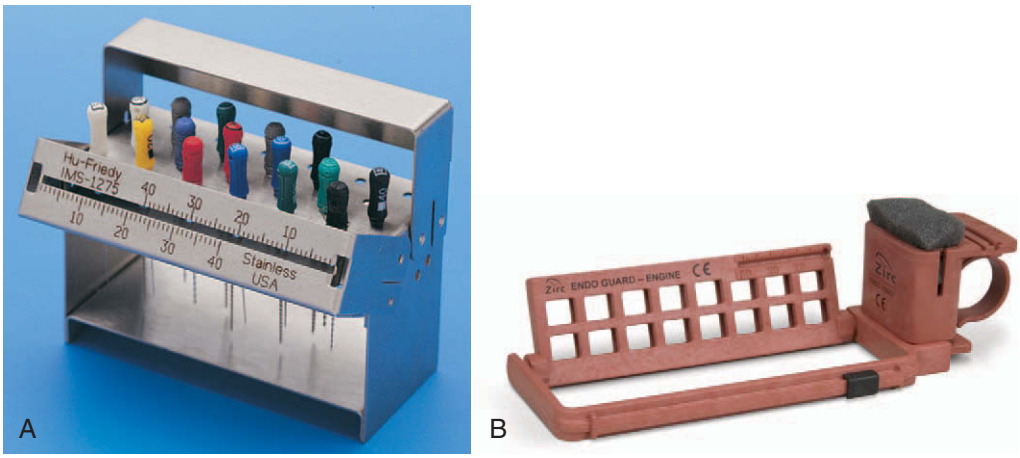


FIG. 6-7 A, File stand. **B,** File stand for rotary instruments. (***A,*** *Courtesy Hu-Friedy, Chicago, IL.* ***B,*** *Courtesy Zirc, Buffalo, MN.)*



FIG. 6-8 Ruddle post removal system. *(Courtesy SybronEndo, Orange, CA.)*



FIG. 6-9 Micro-apical placement device for insertion of MTA filling. *(Courtesy Roydent Dental Products, Johnson City, TN.)*

generally require extended soak times. Their proper use necessitates rinsing in sterile water to remove traces of disinfectant and handling with sterile tongs or sterile gloves to place them in a sterile storage container after treatment. Cold sterilants are poisonous and can be harmful to patients, even in small amounts.

Do not pack instrument sets tightly. Boxes with plenty of holes to admit steam through the overwrap are ideal. Closed boxes should be autoclaved with the lids ajar. Autoclaving is the typical method of sterilization for most health care facilities. The usual cycle is 30 minutes at 250° F (121° C) at 15 psi. Many offices use two autoclaves. This allows for rapid sterilization of handpieces or other expensive equipment to reduce inventory requirement, as well as serving as a backup when one machine is broken. Flash sterilization at higher temperatures of 273° F (134° C) for 10 minutes and 30 psi is also approved. The Statim brand autoclave (SciCan, Toronto, ON, Canada) uses the higher temperatures to reduce cycle time.

A Chemiclave chemical vapor sterilizer uses a solution of 72% ethanol and 0.23% formaldehyde in place of water in its "autoclave." This avoids the instrument corrosion typical of steam autoclaves. It uses 270° F (132° C) at 20 psi for 20 minutes, including drying time. It could be argued that a longer cycle time may be needed to meet guidelines for safety margins.[18] Ventilation or filtration is required to handle

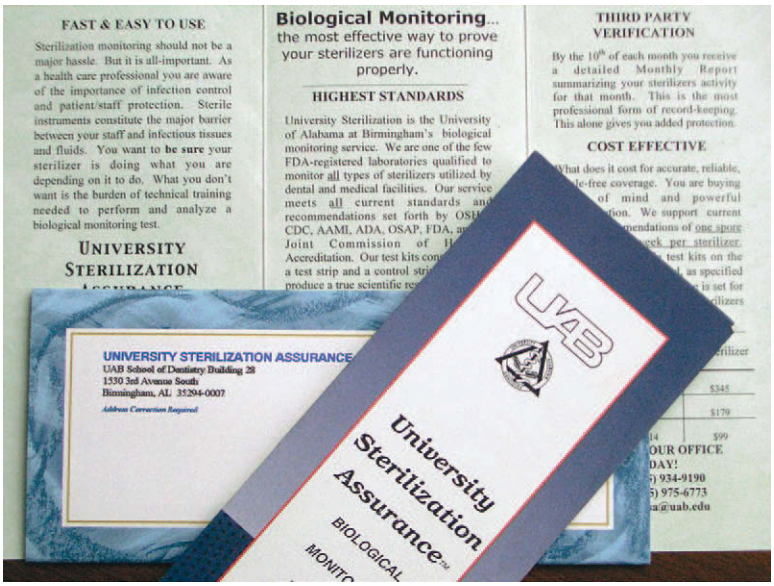


FIG. 6-10 Sterilizer monitoring service. Each week an envelope containing test spores is included in a sterilizer cycle and then mailed to the test center. Sterilizer performance is confirmed when no spores grow in culture. This lab is U.S. Food and Drug Administration (FDA) approved to monitor all types of sterilizers. *(Courtesy Dr. John Ruby, University of Alabama at Birmingham.)*

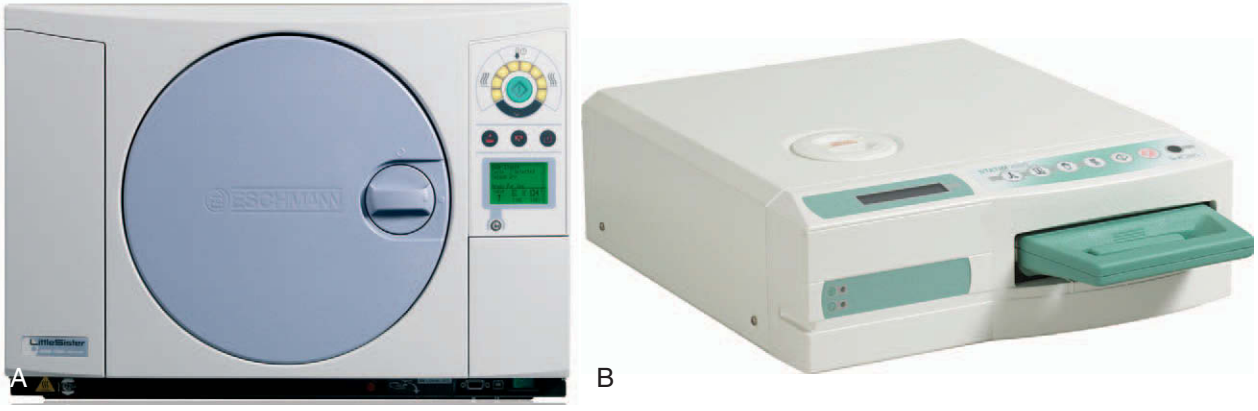


FIG. 6-11 Rapid-cycle autoclaves. **A,** Automatic, programmable autoclave. **B,** Statim cassette autoclave. *(**A,** Courtesy Eschmann Equipment, Lancing, West Sussex, England. **B,** Courtesy SciCan, Toronto, ON, Canada.)*



FIG. 6-12 Chemiclave chemical vapor sterilizer. *(From Bird DL, Robinson DS: Torres & Ehrlich Modern Dental Assisting, St. Louis, 2009, Saunders/Elsevier.)*



FIG. 6-13 Cox rapid sterilizer, using forced hot air. *(Courtesy Alfa Medical, Hempstead, NY.)*

formaldehyde fumes. Chemiclave chemicals can dissolve into liquids, so liquids should be steam autoclaved.

Dry heat sterilizers, either still air in an oven or forced air, also avoid corrosion of instruments. After preheating the instruments to sterilizing temperature, still air models provide sterility in 1 hour at 375° F (191° C). The Cox sterilizer (Alfa Medical, Hempstead, NY) uses forced hot air of the same temperature and sterilizes in 6 minutes. Wrapped packs increase the time needed for processing. Hot air sterilizers may require special wraps.

In-operatory sterilizers were once popular among clinicians for decontaminating instruments during treatment. Such sterilizers achieved 450° F (218° C), and typical cycles consisted of a few seconds for metal instruments.[31] Glass beads or salt were used to transfer dry heat to endodontic canal instruments. These sterilizers are no longer approved for use because of the possibility that clinicians relied on them for sterilization between patients. One study found that they were ineffective at killing spores on cotton and paper products.[31] Endodontic instruments used for recapitulation in a canal as it is progressively decontaminated may be disinfected chemically.[16]

After patient treatment, barriers should be removed and surfaces disinfected (Fig. 6-14). Aerosols generated by dental care can land anywhere, and therefore all surfaces that may be touched during treatment of the next patient should be disinfected. Special attention should be given to instrument holders and hoses. Door and drawer pulls should be included in the disinfection routine or covered with barriers. Pens, pencils, and even patient charts should be considered contaminated.

Disinfection of the treatment room includes wiping all surfaces to be used with an appropriate disinfectant. Governmental agencies catalog such disinfectants as low-level and intermediate-level disinfectants, and Table 6-1 provides

TABLE 6-1

Chemicals for Disinfection

Level	Spectrum	Use	Examples
Low level	All bacteria except mycobacteria and spores Some fungi and some viruses	Surfaces without blood	Quaternary ammoniums, some phenolics, some iodoforms
Intermediate level	Mycobacteria, not spores Most fungi and most viruses	Surfaces with blood	Quaternary ammoniums with alcohol, chlorines, phenolics, iodoforms
High level	All microbes except spores	Immersion	Glutaraldehyde, strong peroxides, *o*-phthalaldehyde

Note that high-level disinfectants are used as room-temperature sterilizing solutions with extended contact times.

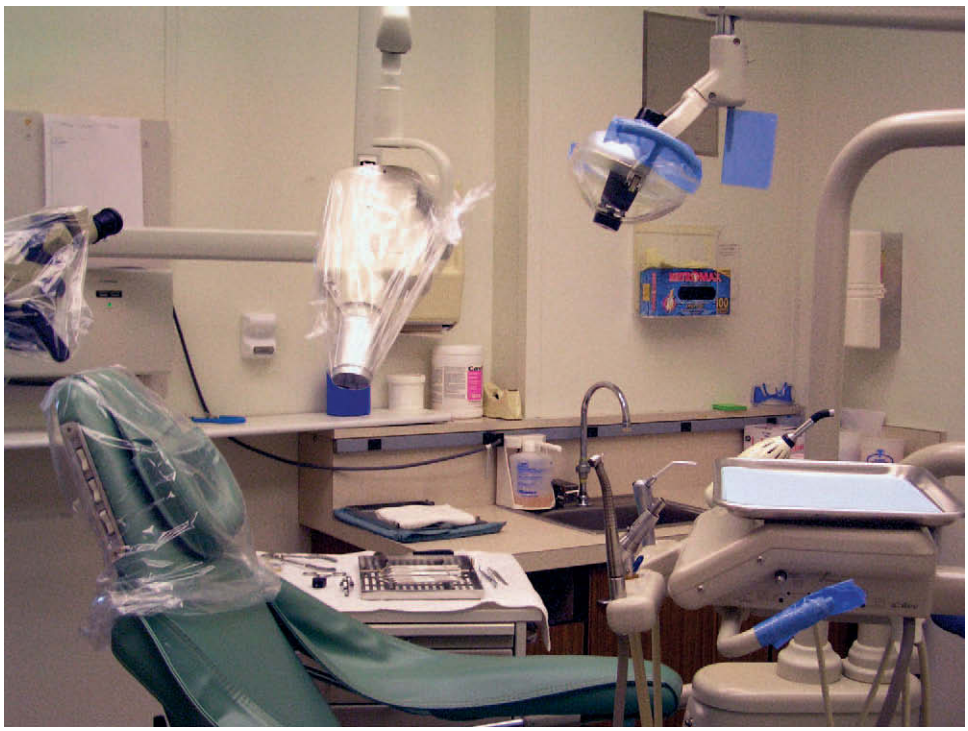


FIG. 6-14 Operatory plastic film barriers in place.

examples. High-level disinfectants are cold sterilizers. Low-level disinfectants are for surfaces that are not contaminated by blood. In dentistry, blood contamination is likely, and therefore intermediate-level disinfectants seem appropriate. Many low-level disinfectants are the same chemicals found in higher-level disinfectants. Greater concentrations often allow a given chemical to be classed higher.

After disinfection of the treatment surfaces, dental assistants should wash their hands and reglove before proceeding with barrier placement and opening of sterile packs. Fresh barriers should be placed on light handles, dental unit switches, chair controls, and so on. X-ray generators and microscopes can be effectively isolated by using large plastic bags, such as dental chair covers or laundry bags. Other items for barrier consideration include electric pulp testers and other thermal pulp testers. Recycled anesthetic cartridges used for ice testing should be autoclaved before adding water and freezing.

Some people express concern that resistance to disinfectants may occur, as has happened with antibiotics. This is not likely because disinfectants (in appropriate concentration) kill rapidly, and thus bacteria are not able to adapt to them.

The current report of the CDC indicates that sterile packs will remain sterile if they remain dry in closed cabinets and if the sterile wrapping remains intact.[12,21] This philosophy means that routine resterilization is not needed. Resterilization is indicated if the wrap has been exposed to fluids that may wick into the sterile side of the package or if an instrument has broken through the wrap. Packages should be dated for identification in case the biologic monitor shows incomplete spore kill.

An additional concern involves microbes in dental water lines. Chlorination or other municipal disinfection has brought a substantial improvement in public health in the last century. However, these steps do not ensure sterile water. Just as dental plaque bacteria colonize on teeth and cause problems, plastic water lines within the dental unit are a haven for bacteria to colonize. The sticky, dental plaque–like water-line biofilm grows unhindered by the slow water movement in dental water lines. Bacteria from patients can travel upstream into dental water lines. The relatively small numbers of bacteria allowable in municipal water supplies flourish within the dental unit, even in independent water bottles (Fig. 6-15). It is now known that biofilms establish themselves in these systems quickly. One approach to cleaning independent dental water lines is weekly use of strong chemicals, although staff compliance is a potential problem.[27] Another approach is continuous use of low-concentration chemicals.[10] Flushing water lines will not eliminate the biofilm, yet a 20- to 30-second flush between patients remains recommended to avoid microbes from one patient being transmitted to the next.[12] Regular culturing of dental unit water lines remains the only method to ensure that a system meets the CDC and U.S. Environmental Protection Agency (EPA) no-more-than-minimum standard of 500 colony-forming units (CFU)/ml of water (Fig. 6-16).

The American Dental Association goal is less than 200 CFU/ml, and the European Union strives for less than 100 CFU/ml.

One can readily see that organization and quality control monitoring are essential to avoid contamination of the endodontic treatment field. Organization should be a key item in assembling the armamentarium, sterilization of instruments before treatment, and preparation of the treatment area with disinfectants and barriers. Attention to every detail is needed to avoid microbial contamination.



FIG. 6-15 Independent water source. Water is pressurized by compressed air. *(Courtesy DentalEZ, Bay Minette, AL.)*

TREATMENT

Collection of a proper medical history is mandatory for proper treatment planning. Many examples illustrate the need for thorough consideration of medical aspects unique to each patient. For example, mutations of the tubercle bacillus, *Mycobacterium tuberculosis,* now endow it with resistance to multiple antibiotics. Infectious microscopic droplets hang in the air for hours after dental treatment, and this is a particular concern when treating patients with TB. These droplets pose a serious risk for staff and other patients. For public health reasons, patients with active TB must be treated in a special negative–air pressure room. Treatment room air must be vented outside through high-efficiency filters that are then properly treated. The use of pretreatment oral irrigants containing chlorhexidine gluconate has been recommended for reduction of microflora in aerosols.[12]

Patients with heart defects may also need special treatment.[9] Oral bacteria that escape from the oral cavity or from an endodontic infection can cause serious complications or even initiate disease in healthy hearts.

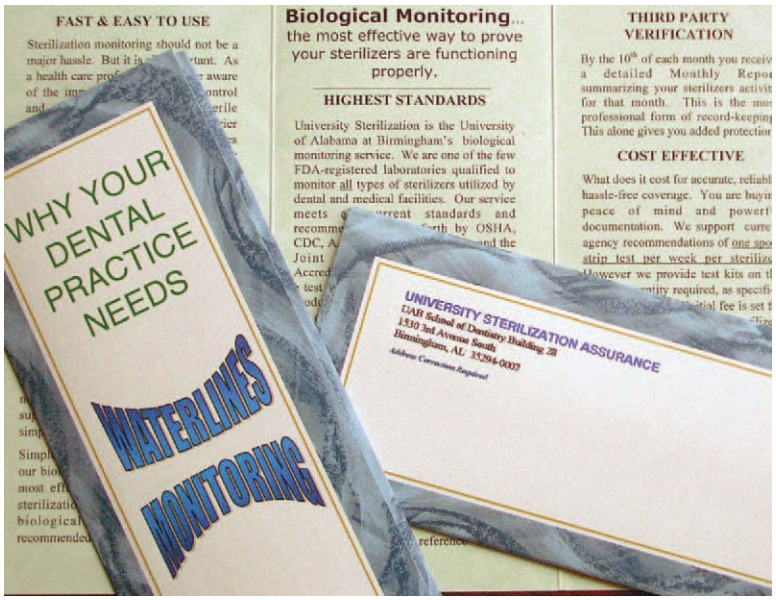


FIG. 6-16 Waterline monitoring service. This laboratory uses U.S. Environmental Protection Agency (EPA) methods to determine heterotrophic bacterial counts for drinking water. *(Courtesy Dr. John Ruby, University of Alabama at Birmingham.)*

Clothing worn by office personnel is a concern for staff safety. The purpose of protective fabric is to avoid contamination of skin and street clothes. Regulations mandate coverage of forearms. The garb should be changed if visibly soiled or wet by potentially infectious matter. *The protective clothing should not be worn outside the office.* Laundering at home is a potential source of contamination of family members and is not allowed.

Eye protection and masks should be donned before washing and gloving. Glasses and masks protect thin mucous membranes from possible contamination. Documentation of virus transmission via the conjunctiva of the eye has resulted in the recommendation for protection of these membranes.[26] Risk of human immunodeficiency virus (HIV) transfer to a health care worker exposed by mucous membrane is estimated to be about 0.1%.[15] Eyeglasses should have side shields.

Microscope controls should be covered with a barrier, such as a large plastic bag used for laundry or a dental chair cover. The plastic can be torn and stretched over the objective lens. The eyepieces should be adjusted for use with glasses (Fig. 6-17). Those who wear magnifying loupes should not adjust them during treatment unless followed by regloving. An assistant not involved with treatment may do so, or a barrier that can be immediately discarded may be used to adjust glasses or replace wet masks.

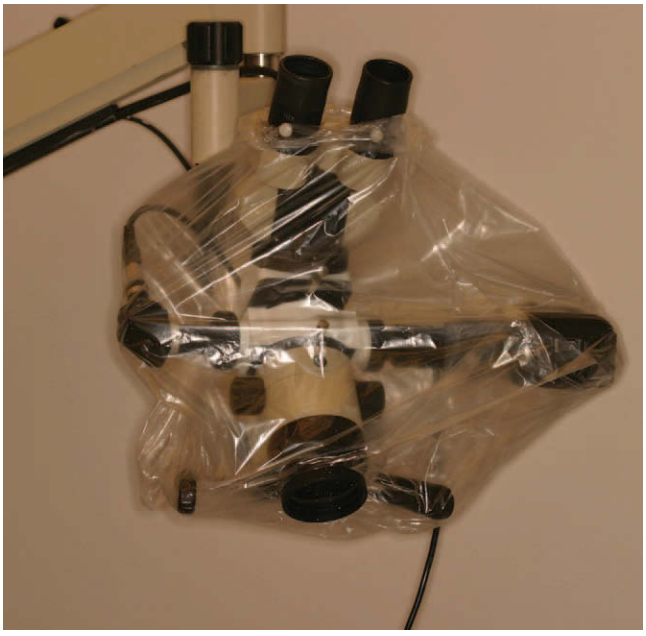


FIG. 6-17 Microscope controls covered with a barrier. *(Courtesy Dr. S. Craig Rhodes.)*

Masks commonly available for use in dentistry protect the wearer only partially. Small droplets containing bacteria can pass through most clinical masks. Wet masks are even less efficient and should be changed immediately. Masks that do not seal around the face fail to give maximal protection. The ideal mask has not yet been developed.

Dental treatment produces large amounts of potentially contaminated droplets during treatment. Rubber dam use reduces the aerosol, but infected pulp tissue can serve as a source for aerosol-borne biocontaminants. Most droplets from dental treatment quickly fall out of the air because of their relatively large size, but small particles can remain suspended in the air for long periods. Microbes are carried via these small droplets deep into lungs, where they cause infection. Airborne transmission definitely accounts for the spread of Legionnaires' disease, TB, severe acute respiratory syndrome (SARS), and influenza. Other diseases may spread by droplets. Use of a preprocedural mouth rinse reduces the aerosol along with proper high-velocity evacuation common in large-bore dental evacuators.[14,19]

Hand washing should precede gloving. Long fingernails and jewelry that could puncture glove material or shelter bacteria from the washing process should be avoided. Recently, a new latex glove material has been tested. Apparently, guayule (pronounced *why-YOU-lee;* Yulex, Maricopa, AZ) rubber

proteins have low allergenic potential. It is derived from a plant in the desert of southwestern United States.[1]

Sterile gloves should be worn for surgical procedures. Examination-type gloves may harbor microbes from the point of manufacture or from contamination at the point of use. Hand lotions may degrade the rubber of gloves and should not be used until treatment has concluded for the day. Sharp jewelry and artificial nails can penetrate the gloves, even if such penetrations are too small to be seen; thus they should not be worn with gloves. The glove material weakens during use, especially when using hand files. Gloves should be changed between patients and more often if harshly used. Interruption of treatment to answer the telephone or to develop a radiograph presents an opportune time for fresh gloves.

Evaluation of sterile technique often discloses contamination early in the treatment process. Procedures such as diagnosis or application of the rubber dam contaminate gloves. Gloves should be considered only a barrier for staff protection. Patient protection comes from careful attention to sterile technique, including not allowing nonsterile gloves to touch anything that will go into the root canal.

Because of the lack of total protection with gloves, it is recommended that a disinfectant hand wash be used. Chlorhexidine has a property called *substantivity,* meaning that it bonds to skin and maintains antibacterial action longer than other surgical scrubs. Alcohol-based hand rubs have recently become popular for hospital workers. Presently available waterless techniques appear less effective than a surgical soap-and-water method.[7]

Reducing bacteria within the oral cavity before treatment should be of concern to every clinician for every procedure. Degerming the oral cavity with an appropriate mouthwash before treatment has been recommended by Bender and Montgomery[2] as a means of reducing bacteremia.[11] Sterile technique should be used. Application of the rubber dam will typically contaminate gloves, and one approach is to change gloves after dam application. Another possibility is to have the assistant place the rubber dam and then reglove.[20]

Sharps should be handled with caution. Needles should be recapped with one hand to avoid possible injury to the hand holding the needle cover. Needles are available that recap automatically. Scalpel blades should be placed and removed from the handle by means of a hemostat or needle holder, with consideration to where hands are positioned, to avoid an injury in case something slips.

Disinfection of the canal continues to be a heavily researched topic. At present the only agent that dissolves pulp tissue is sodium hypochlorite. This explains its continued popularity among clinicians. Studies have demonstrated that 7 minutes of tissue contact time with sodium hypochlorite dissolves 75% of a tissue plug in vitro.[6,13,17] Dilute concentrations of sodium hypochlorite are less effective for dissolving remaining tissue, and therefore the full-strength concentration is popular. However, it should be remembered that even dilute solutions can cause untoward reactions if injected periapically or exposed to oral tissues. Another clinical use for sodium hypochlorite is for disinfection of gutta-percha cones. A 1-minute exposure to 1% or a 5-minute exposure to 0.5% has been shown to be an effective gutta-percha disinfectant.[3]

Small instruments such as canal files may be dropped into a chairside container of disinfectant when no longer needed. This approach will provide some protection to staff members if they are accidentally stuck by an instrument in the cleaning and repackaging process. Such an approach will not positively ensure protection from infectious agents but will certainly reduce risk. Some clinicians have adopted the approach of using files as disposable instruments. Files require sterilization before treatment, except those few sterilized by the manufacturer.[25] After single use, files can be discarded in the chairside sharps container. This philosophy of single use has the added advantage of reducing the possibility of fracture of instruments caused by repeated use (see also Chapters 8 and 9).

An often overlooked clinical problem concerns decontamination of radiographs. The use of digital radiography has improved patient protection with single-use sleeves covering the digital sensor. Proper technique dictates placing the barrier over the digital sensor when other barriers are placed before treatment commences. The barrier should completely cover the holder, or a sterilized holder or disposable holder should be used for each patient.

Conventional radiographic films or gloves worn from the treatment area into the developing area can contaminate this area. Staff should be required to disinfect conventional films and deglove before leaving the treatment room. Hand washing and regloving should occur before treatment continues.

Reusable instruments should be taken to a dedicated area for removal of gross debris by scrubbing or preferably by ultrasonic bath. Heavy gloves along with eye protection and a mask should be worn during instrument processing for sterilization. The ultrasonic bath should be a germicide to minimize infection spread to office workers. The ultrasonic bath cover should be used to reduce aerosols. Automatic instrument washers are also available for added cleaning efficiency and worker protection. Dedicated washers use copious amounts of high-temperature water to rapidly prepare instrument packs for autoclaving. The difficulty of thoroughly cleaning endodontic instruments was reported by researchers, who found that 76% of endodontic files from dental offices were contaminated with proteinaceous material.[28]

If time is not available to clean instruments immediately, they should be placed in a disinfectant bath to prevent drying of foreign material on the instruments because dried material is more difficult to remove.

If scrubbing is the only feasible means of cleaning, heavy puncture-resistant gloves should be worn and brushes should be long handled to minimize the risk of percutaneous injury. After the ultrasonic bath the instruments should be rinsed in tap water and wrapped for sterilization. Many offices favor cassette systems to lessen the risk of a stick injury. Staff should be cautioned about the risk of a sharp instrument protruding through an opening in a cassette, even one that is wrapped.

As instruments move along a path from dirty to clean and then sterile, the path should not cross itself in order to avoid cross-contamination.

GENERAL CONSIDERATIONS FOR THE OFFICE

Government guidelines spell out requirements for staff education.[12] Staff meetings are strongly recommended because of the many advantages of well-educated personnel in the dental office. Proof of staff meetings should be kept in a log. Box 6-3 includes items for consideration.

BOX 6-3

Written Records for Consideration by Clinicians

1. Manufacturer safety data sheets (MSDSs)
2. Posting of Department of Labor policies, for example, workers' compensation; may vary by state
3. Written infection control plan, including the following:
   a. Infection control coordinator (perhaps the clinician)
   b. Written plan to minimize exposure to infection by staff
   c. Annual record of employee training, with record of immediate training for new hires and those changing to jobs with patient contact
   d. Written plan for postexposure actions following potential infection transmission. Records of worker injury will necessitate a record that will remain in employee's confidential record. The written plan should prearrange for the following:
      (1) Reporting criteria and procedures, to include date, time, and details of injury, and infectious status of patient (perhaps on a previously prepared reporting form)
      (2) Evaluation by a physician (establish a relationship with a physician before injury occurs)
      (3) Counseling (establish a relationship with a counselor before injury occurs)
      (4) Treatment (probably with the physician listed in [2])
      (5) Medical follow-up (probably with the physician listed in [2])
4. Vaccinations, with log of appropriate updates (boosters)
5. Log of skin test for tuberculosis, especially for baseline for new hires
6. Log of sterilizer monitoring
   a. Log of pressure, temperature, and time of each load
   b. Weekly log of results of biologic monitor for each sterilizer
7. Log of hand-washing education and critique of employees' performance on a regular basis
8. Log of education about barrier placement need and technique and critique of employees' performance on a regular basis
9. Log of sharps hygiene education and critique of employees' performance on a regular basis
10. Log of annual review of exposure plan
11. Log of results of dental water monitoring
12. Log of medical and other waste education and critique of employees' performance on a regular basis
13. Log of patients who make unscheduled return visits for possible infection control breach
14. Plan for health care workers infected with various agents, including possible restriction of patient contact and conditions for return to full duty (e.g., workers with hepatitis A are restricted from patient contact until after the seventh day of onset of jaundice) (see Table 1 of reference 11, p. 8).

Modified from Table 5 of Kohn WG, Collins AS, Cleveland JL, et al: Centers for Disease Control and Prevention (CDC): Guidelines for infection control in dental health-care settings—2003. MMWR Recomm Rep 52(RR-17):1, 2003.

Vaccinations for all employees working in the treatment room must include the hepatitis B series of three injections. Other vaccinations are recommended as a means of preventing disease. Such expense is minimal considering lost time and possible sequelae to valuable workers. Box 6-4 includes the 2005 vaccination recommendations of the CDC.[12]

BOX 6-4

Vaccinations for Diseases Potentially Spread to Dental Office Workers

- Hepatitis B
- Influenza, needed yearly
- Measles
- Mumps
- Rubella (German measles)
- Varicella (chickenpox)

It is now evident that antibody levels to hepatitis B wane over time. CDC findings suggest that 60% of those vaccinated will have no detectable antibodies after 12 years.[4] Hepatitis B is a serious threat to unprotected individuals. The risk of clinical hepatitis B to an unvaccinated health care worker stuck with a needle from a patient positive for both hepatitis B surface antigen and hepatitis B e antigen is 22% to 31%.[5]

Janitors and those who dispose of dental office wastes must be protected from harm. They should be protected from sharps such as canal instruments that are misplaced in an area they will clean. Janitors need education in use of personal protective equipment, such as heavy gloves and appropriate disinfectants.

Dental offices do not generate large volumes of medical wastes.[23] Dental offices are estimated to generate only 1% to 2% of their waste as medical waste. The majority of waste is not subject to regulation. Used gloves, masks, and protective garb and barriers are not regulated waste.

Sharps are part of regulated medical waste. Sharps include needles, scalpel blades, endodontic instruments, and other items capable of breaking the skin. Used anesthetic cartridges are classified as sharps because they could break and cause an injury if placed in regular waste. Sharps should be discarded at the point of use in a rigid container with a biohazard label. After sealing this puncture-resistant container with a tamperproof seal, the container can be discarded in normal fashion.

Regulated medical waste includes items soaked in blood or saliva, such as 2 × 2 inch gauze or cotton rolls. This type of medical waste must be sealed in a leak-resistant biohazard bag that does not contain sharps. This type of waste could be placed in a sharps container. State regulations should be consulted before discarding biohazard bags in regular waste.

Liquid medical wastes, such as contents of a vacuum system separator, can be disposed of in a sanitary sewer, although care should be taken to avoid splatter when pouring into a toilet or sink. Normal protective gear including office garb, eyeglasses, and mask is needed for pouring liquid medical waste into a drain.

Some municipalities mandate mercury separators (endodontic offices may be exempt). These separators remove the heavy metal before it enters the city waste system and help the local government comply with regulations regarding mercury in solid waste and effluent from sewage treatment.

Nitrous oxide scavengers remove some of the gas exhaled by patients. These devices reduce but do not eliminate the gas from the office. Scavengers are connected to the central dental vacuum system and require a high flow rate. The vacuum system exhaust should be piped outside rather than into the mechanical room, where it may reenter the office air, because

nitrous oxide interferes with deoxyribonucleic acid (DNA) synthesis. Further, nitrous oxide increases the rate of congenital abnormalities in children of both males and females exposed to the gas in the dental office environment.[8]

FUTURE CONSIDERATIONS

Patient concerns will likely surface as more knowledge about existing pathogens becomes apparent and new pathogens arise. Clinicians should be ready to modify their practices and procedures to allay fears. Increased use of disposable products is likely. Many clinicians are already using new canal files for each patient.

A potential threat on the horizon are prions *(PREE-onz)*. Prions are glycoproteins found on cell surfaces. Modified versions of normal prions cause various slow-moving, often fatal diseases of the nervous system. Prions are almost impossible to sterilize in a dental office and they have an affinity for stainless steel. They can be killed by certain caustic chemical treatments, such as sodium hydroxide. Current World Health Organization (WHO, Geneva, Switzerland) recommendations for reprocessing heat-resistant instruments call for keeping instruments moist until decontamination begins, because drying strongly stabilizes the bond of prions to the instruments. Prions are also resistant to formalin or glutaraldehyde fixatives, with their bond strengthened as well. The WHO describes several methods to rid dirty instruments of prions before repackaging and routine sterilization.[33] The following methods, performed prior to routine sterilization, yield the best protection for office worker as well as patients.

Method 1: Immerse contaminated instruments in a pan of 1 N sodium hydroxide and heat in a gravity displacement autoclave for the normal cycle of 121° C for 30 min.

Method 2: Immerse in either 1 N sodium hydroxide or full-strength bleach (20,000 ppm available chlorine) for 1 hour; rinse; transfer to a dry, open pan; and heat in a gravity displacement autoclave at 121° C for 1 hour.

Method 3: Immerse in either 1 N sodium hydroxide or full-strength bleach (20,000 ppm available chlorine) for 1 hour; rinse; transfer to a dry, open pan; and autoclave in either a gravity displacement autoclave at 121° C or a conventional autoclave at 134° C for 1 hour.

Method 4: Immerse and boil in 1 N sodium hydroxide for 10 minutes.

Method 5: Immerse in 1 N sodium hydroxide or full-strength bleach for 1 hour, rinse and subject to routine sterilization.

Method 6: Autoclave at 134° C for 18 minutes.

Note that 1 N sodium hydroxide is made by adding 40 g of the crystal to 1 liter of distilled water.

Note that freshly opened 5.25% bleach (sodium hypochlorite) contains 25,000 ppm available chlorine. The WHO recommendations call for 20,000 ppm available chlorine. Today many commercial concentrations are 6%.

Research based on new technology will surely define more expeditious means of destroying prions. Investigations have explored the use of an industrial fabrication method, that is, low-pressure oxygen–argon plasma made by radiofrequency energy, to decontaminate and sterilize in one quick process. One study[32] found organic matter, about 1 µm thick, on endodontic files that had been decontaminated and sterilized by a local dental practice. One third of the files had contaminant thicknesses exceeding 50 µm. After plasma exposure for unspecified times, no nitrogen was present, suggesting the destruction of proteinaceous matter. Calcium remaining was interpreted as lingering dentin.[32] Although prions have high resistance to proteases, endopeptidases, such as keratinase, have been suggested as potential aids.[22] Disposal of contaminated files is best accomplished by incineration.[33]

Because of fears of the public about disease transmission in the dental office, many endodontists consider all canal instruments as single-use items. This overcomes the difficulty of staff injury during cleaning and repackaging. Apparently, complete cleaning of endodontic files is quite difficult with current procedures. Another study reports that ultrasonic baths are better than hand brushing, and that disinfectant washers are also a step in the right direction, albeit not completely effective.[30] Investigators found that even after cleaning by hand and ultrasonication, plus aggressive 24-hour immersion in disinfectants concentrated enough to corrode nickel–titanium files, residual debris remained.[29]

At present no vaccination exists against hepatitis C. Fortunately, the rate of disease transmission to health care workers is low. The estimates of disease transmission from a needle stick range up to 7%.[24]

Other hazards may also arise. Clinicians must keep abreast of research findings and changes in guidelines from regulatory agencies. New inventions and techniques will help clinicians deal with the current threats, as immunizations and innovations become available to make the workplace safer for staff and patients.

Air in the treatment room may contain spores. Although this has not been found to be problematic at present, it presents a possible concern for the future.

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CHAPTER 7

Tooth Morphology and Access Cavity Preparation

FRANK J. VERTUCCI and JAMES E. HADDIX

CHAPTER OUTLINE

COMPONENTS OF THE ROOT CANAL SYSTEM
ROOT CANAL ANATOMY
ANATOMY OF THE APICAL ROOT
OBJECTIVES AND GUIDELINES FOR ACCESS CAVITY PREPARATION
- Objectives
- Guidelines

MECHANICAL PHASES OF ACCESS CAVITY PREPARATION
- Armamentaria
- Access Cavity Preparations

CHALLENGING ACCESS PREPARATIONS
- Teeth With Minimal or No Clinical Crown
- Heavily Restored Teeth (Including Those With Full Veneer Crowns)
- Teeth With Calcified Canals
- Crowded Teeth
- Rotated Teeth

ERRORS IN ACCESS CAVITY PREPARATION
MORPHOLOGY AND ACCESS CAVITY PREPARATIONS FOR INDIVIDUAL TEETH

The hard tissue surrounding the dental pulp can take a variety of configurations and shapes. A thorough knowledge of tooth morphology, careful interpretation of angled radiographs, and adequate access to and exploration of the tooth's interior are prerequisites for treatment. Magnification and illumination are indispensable aids. This chapter describes and illustrates tooth morphology and explains the techniques crucial to achieving unobstructed direct access to the root canal and apical foramina. This chapter is divided into eight major sections: (1) components of the root canal system, (2) root canal anatomy, (3) anatomy of the apical root, (4) objectives and guidelines for access cavity preparation, (5) mechanical phases of access cavity preparation, (6) challenging access preparations, (7) errors in access cavity preparation, and (8) morphology and access cavity preparations for individual teeth.

Only after correct completion of this phase of therapy can the clinician perform thorough shaping and cleaning and three-dimensional (3-D) obturation. The optimal endodontic result is difficult to achieve if the access is not properly prepared. The clinician must have an understanding of the complexity of the root canal system to understand the principles and problems of shaping and cleaning, to determine the apical limits and dimensions of preparations, to perform microsurgical procedures successfully and to correct procedural errors.

Practitioners must have a thorough understanding of the internal anatomic relationships of teeth and must be able to visualize these relationships before undertaking endodontic therapy. Careful evaluation of two or more periapical radiographs, exposed at different horizontal angulations of the x-ray cone, is mandatory. These radiographs provide important information about root canal morphology. The clinician must keep in mind that the inclination of the x-ray tube significantly influences the ability to detect root canal systems present in premolar teeth. For example, 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.[131] However, in the mandibular second premolar, only the 40-degree horizontal angle correctly identifies the root canal morphology. The critical importance of a careful reading of each radiograph before and during root canal therapy is well recognized. As shown in a case report of five canals in a mandibular first molar, the radiographic appearance significantly aided recognition of the complex canal morphology.[69] This study concluded that "any attempt to develop techniques that require fewer radiographs runs the risk of missing information which may be significant for the success of therapy."

However, conventional radiographs may not always determine the correct morphology, 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.[144] When the *fast break* guideline was used (i.e., interpreting a sudden disappearance or narrowing of a canal as a sign of canal division [e.g., bifurcation]; Fig. 7-1), the result was failure to diagnose one third of

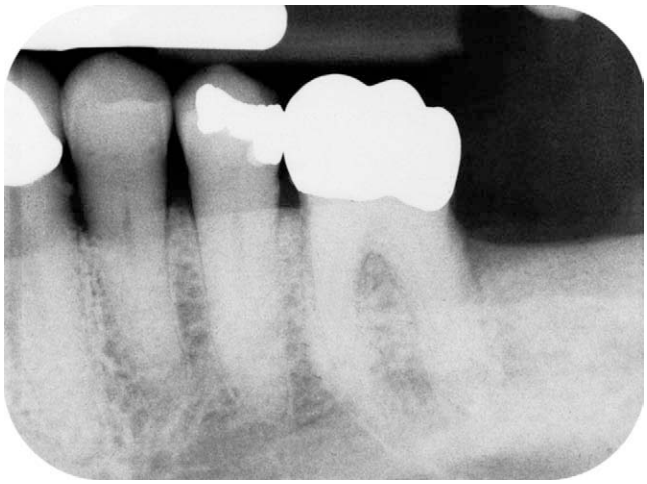


FIG. 7-1 Periapical radiographs can reveal clues to root canal morphology. Abrupt disappearance of the large canal in the mandibular premolars usually signifies a canal bifurcation.

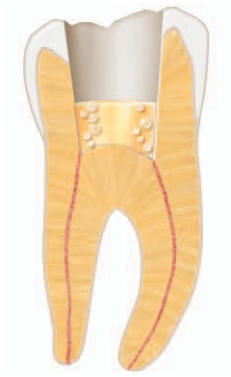


FIG. 7-2 Allowing sodium hypochlorite (NaOCl) to remain in the pulp chamber may help locate a calcified root canal orifice. Tiny bubbles may appear in the solution, indicating the position of the orifice.

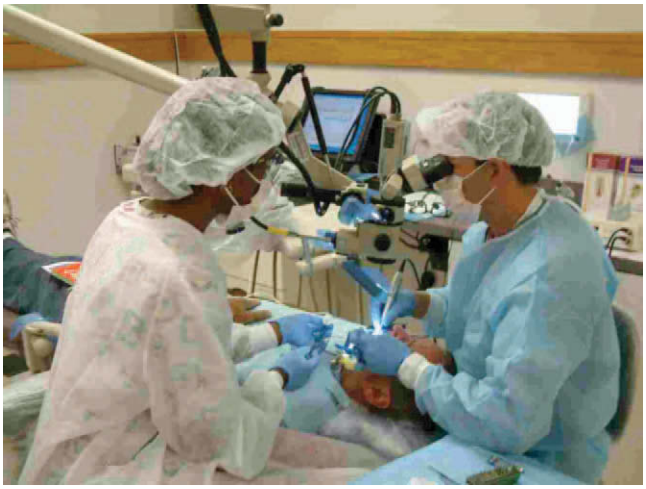


FIG. 7-3 The dental operating microscope (DOM) has vastly improved locating and negotiating of canal anatomy.

these divisions from a single radiographic view. Thus, evaluation of the root canal system is most accurate when the clinician uses information from several radiographic views together with a thorough clinical exploration of the interior and exterior of the tooth. Alternatively, the recent development of micro-CT has greatly increased our understanding of the complexities and three-dimensional relationships of the root canal systems (see also Chapter 9 and online Chapter 29).

The main objectives of root canal therapy are thorough shaping and cleaning of all pulp spaces and complete obturation of these spaces with an inert filling material. The presence of an untreated canal may be a reason for failure. A canal may go untreated because the clinician fails to detect it. It is extremely important that clinicians use all the armamentaria at their disposal to locate and treat the entire root canal system. The complexity of the spaces that must be accessed, shaped, cleaned, and filled is remarkable. However, even under the most difficult circumstances, current endodontic techniques have an exceptionally high rate of success.

Diagnostic measures are important aids in the location of root canal orifices. These measures include obtaining multiple pretreatment radiographs or cone beam CT (CBCT, see online Chapter 26), examining the pulp chamber floor with a sharp explorer, troughing grooves with ultrasonic tips, staining the chamber floor with 1% methylene blue dye, performing the sodium hypochlorite "champagne bubble" test (Fig. 7-2), and visualizing pulp chamber anatomy[108] and root canal bleeding points. Sequential application of 17% aqueous ethylenediaminetetraacetic acid (EDTA) and 95% ethanol (using the Stropko irrigator fitted with a 27-gauge notched irrigating needle) has been recommended for effective cleaning and drying of the pulp chamber floor before visual inspection of the canal system.[199]

An important aid for locating root canals is the dental operating microscope (DOM), which was introduced into endodontics to provide enhanced lighting and visibility (Fig. 7-3). The DOM enhances the clinician's ability to remove dentin with great precision, thereby minimizing procedural errors. Numerous studies have shown that it also significantly improves the clinician's ability to locate and negotiate canals. For example, the number of second mesiobuccal (MB-2) canals identified in maxillary molars increased from 51% with the naked eye to 82% with the microscope.[13] In another study, 41% of MB-2 canals were identified when magnifying loupes were used, and 94% were identified when the DOM was used.[181] Other clinicians have noted that use of the DOM improves the detection of MB-2 canals to more than 90% in maxillary first molars and 60% in maxillary second molars.[109,199] All these studies demonstrate that magnification and illumination greatly enhance root canal therapy. In contrast, one group of investigators determined that dental loupes and the DOM were equally effective for locating MB-2 canals in maxillary molars.[29] Other investigators determined that the DOM did not significantly enhance the ability to locate canals but did improve the ability to negotiate them.[79] Nevertheless, most clinicians would agree that the DOM makes canals easier to locate by magnifying and illuminating the grooves in the pulpal floor and by distinguishing the color differences of the dentin of the floor and walls.[43,108]

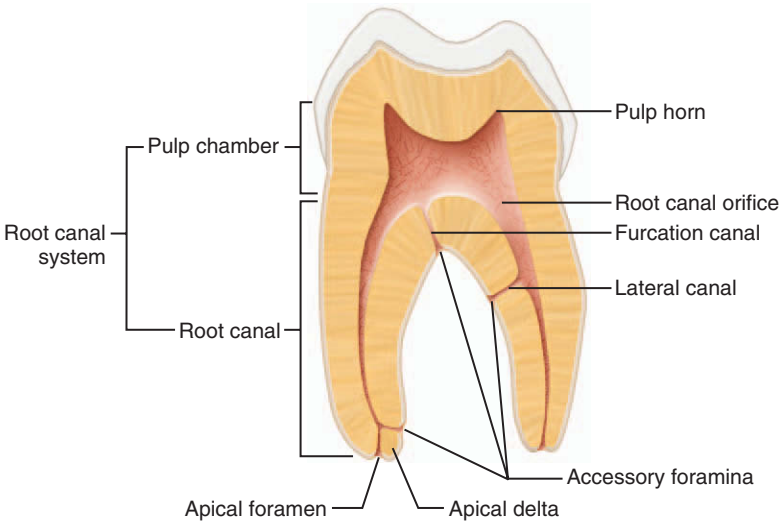


FIG. 7-4 Major anatomic components of the root canal system.

COMPONENTS OF THE ROOT CANAL SYSTEM

The entire space in the dentin where the pulp is housed is called the *root canal system* (Fig. 7-4). The outline of this system corresponds to the external contour of the tooth. However, factors such as physiologic aging, pathosis, and occlusion all potentially modify its dimensions through the production of secondary and tertiary dentin and cementum. 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(s), found in the anatomic root. Other features are the pulp horns; accessory, lateral, and furcation canals; canal orifices; apical deltas; and apical foramina. A 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 from the center of the root apex.[30,80,81,163,215,220] Nearly all root canals are curved, particularly in a faciolingual direction. These curvatures may pose problems during shaping and cleaning procedures because they are not evident on a standard facial radiograph. Angled views are 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 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.[215] 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.[49] Pathologically, they are significant because they serve as avenues for the passage of irritants, primarily from the pulp to the periodontium (see also Chapter 18).

Accessory canals may also occur in the bifurcation or trifurcation of multirooted teeth.[215] One study[219] called these *furcation canals* (Fig. 7-5). Furcation canals form as a result of the entrapment of periodontal vessels during the fusion of the diaphragm, which becomes the pulp chamber floor.[49] In mandibular molars these canals occur in three distinct patterns (Fig. 7-6). Tables 7-1 and 7-2 present the incidence of furcation canals for each tooth.

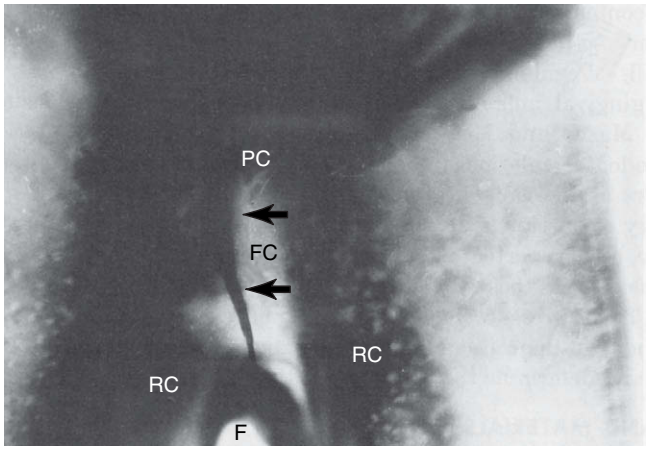


FIG. 7-5 Mandibular first molar showing a furcation canal (*FC, arrows*). *F,* Furcation; *PC,* pulp chamber floor; *RC,* root canal.

According to the findings of scanning electron microscopy (SEM), the diameter of furcation openings in mandibular molars varies from 4 to 720 µm.[216] 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. 7-7). 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 failed to demonstrate the presence of furcation and lateral canals in the coronal portion of these roots. In one study, the pulp chamber floor of 200 permanent molars was

TABLE 7-1

Morphology of the Maxillary Permanent Teeth*

Tooth	Root	No. 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.

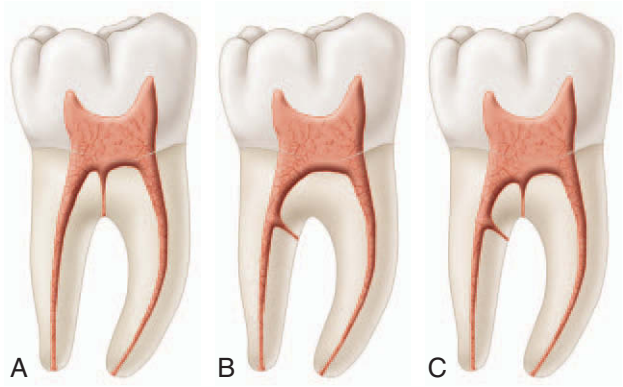


FIG. 7-6 Accessory canals occur in three distinct patterns in the mandibular first molars. **A,** In 13% a single furcation canal extends from the pulp chamber to the intraradicular region. **B,** In 23% a lateral canal extends from the coronal third of a major root canal to the furcation region (80% extend from the distal root canal). **C,** About 10% have both lateral and furcation canals.

stained with 0.5% basic fuscin dye.[85] Patent furcation canals were detected in 24% of maxillary and mandibular first molars, 20% of mandibular second molars, and 16% of maxillary second molars. These canals may be the cause of primary endodontic lesions in the furcations of multirooted teeth (Fig. 7-8) and provide a rationale for placing adhesive restorations on the floor of the pulp chamber to prevent furcal breakdown.

ROOT CANAL ANATOMY

Together with diagnosis and treatment planning, the knowledge of common root canal morphology and its frequent variations is a basic requirement for endodontic success. The significance of canal anatomy has been underscored by studies demonstrating that variations in canal geometry before shaping and cleaning had a greater effect on the changes that occurred during preparation than did the instrumentation techniques.[160-162]

From the early work of Hess and Zurcher[88] to more recent studies[33,82,101,184] demonstrating the anatomic complexities of the root canal system, it has long been established that a root with a 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.[50,54,150,163,190,215] Consequently, in treating each tooth the clinician must assume that complex anatomy occurs often enough to be considered normal. The first premolar in Fig. 7-9, *A* is a good example of complex anatomy. The extra root is not obvious in a pretreatment radiograph (see Fig. 7-9, *B*). Figure 7-10 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 shaping, cleaning, and obturation.

The clinician must be familiar with the various pathways root canals take to the apex. The pulp canal system is complex, and canals may branch, divide, and rejoin. Weine[232] categorized

TABLE 7-2

Morphology of the Mandibular Permanent Teeth*

Tooth	Root	No. 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.

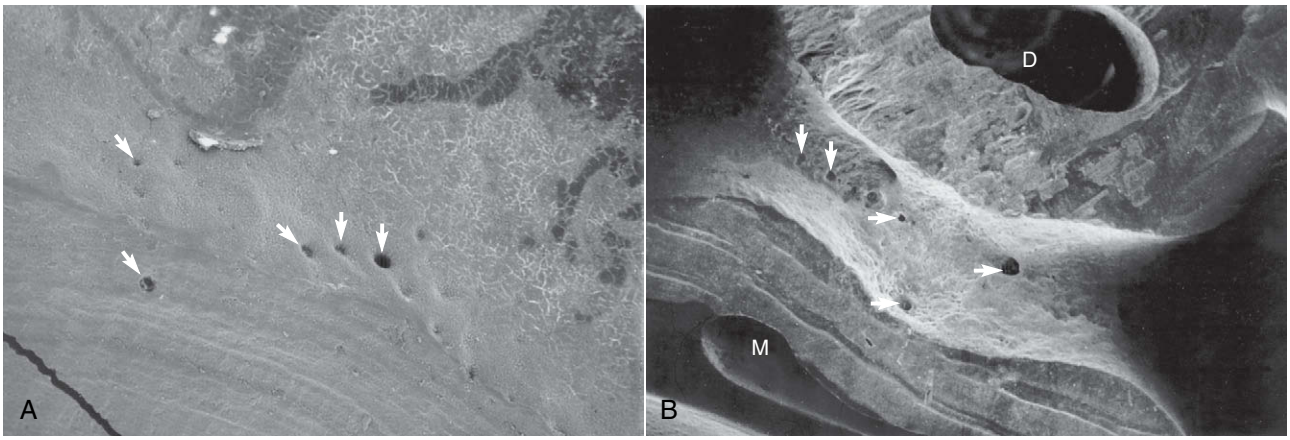


FIG. 7-7 A, Electron photomicrograph of the pulp chamber floor of a mandibular first molar. Multiple accessory foramina can be seen *(arrows)*, ranging from 20 to 140 µm (×20). **B,** Electron photomicrograph of the furcation surface of a mandibular first molar. Multiple accessory foramina can be seen on the furcation surface (×30). *D,* Distal canal; *M,* mesial canals.

the root canal systems in any root into four basic types. Others,[218] using cleared teeth in which the root canal systems had been stained with hematoxylin dye, found a much more complex canal system; they identified eight pulp space configurations, which can be briefly described as follows (Fig. 7-11):

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 redivide 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 redivides into two distinct canals short of the apex (1-2-1-2).

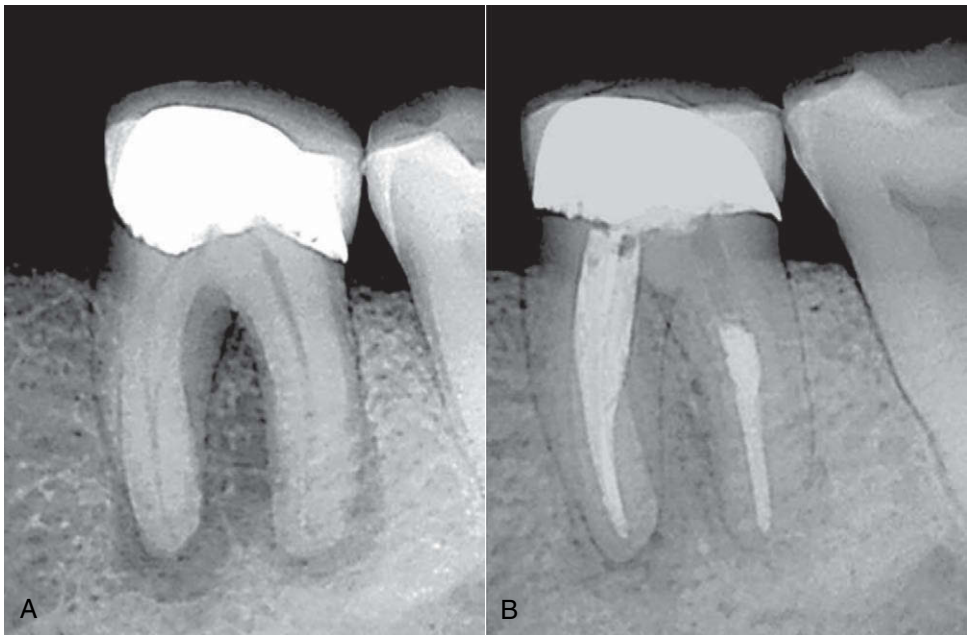


FIG. 7-8 A, Pretreatment radiograph of a mandibular first molar showing furcation and periradicular radiolucencies. **B,** One-year follow-up radiograph showing significant healing. *(Courtesy Dr. Raed S. Kasem, Clearwater, FL.)*

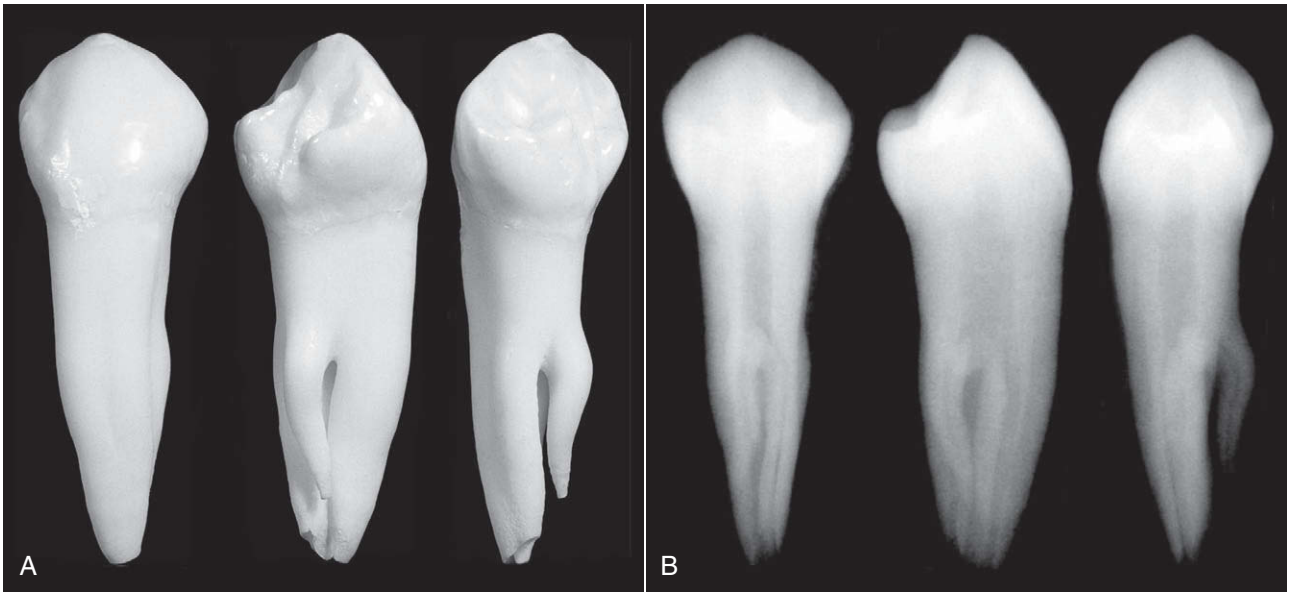


FIG. 7-9 A, Mandibular first premolar with three separate roots trifurcating at midroot. **B,** Radiograph of the three views. Small canals diverging from the main canal create a configuration that is difficult to prepare and obturate biomechanically.

Type VIII: Three separate, distinct canals extend from the pulp chamber to the apex (3).

The percentages of human permanent teeth with these canal configurations are presented in Tables 7-3 and 7-4. The anatomic variations present in these teeth are listed in Tables 7-1 and 7-2. The only tooth that showed all eight possible configurations was the maxillary second premolar.

Other studies using more than 1000 teeth have described similar morphologic results.[33] Another study used more than 1000 teeth and described similar morphologic results 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] The differences are most likely the result of variations of populations in the two studies.[10] Another group studied 100 mandibular anterior teeth and found two new root canal types.[101] In one configuration, two separate canals extended from the pulp chamber to midroot, where the lingual canal divided into two; all three canals joined in the apical third of the root and exited as one canal (Fig. 7-12, *A*). In the other configuration, one canal left the pulp chamber, divided into two in the middle third of the root, then rejoined to form one canal, which again split and exited as three separate canals with separate foramina (Fig. 7-12, *B*). Another set of variants was first observed in the

mandibular molars of Burmese; this study revealed seven additional canal configurations (Fig. 7-13).[82] These include three canals joining into one or two canals; two canals separating into three canals; two canals joining, redividing into two, and terminating as one canal; four canals joining into two; four canals extending from orifice to apex; and five canals joining into four at the apex. Another study evaluated gender-specific root canal configurations in 2800 teeth.[184] Of these specimens 99% were identical to those in the Vertucci classification. The remaining 1% (36 teeth) represented 14 additional canal morphologies, which occurred twice as often in mandibular teeth. These authors concluded that gender plays a role in determining canal morphology and that both gender and ethnic origin should be considered in the pretreatment evaluation for root canal therapy.

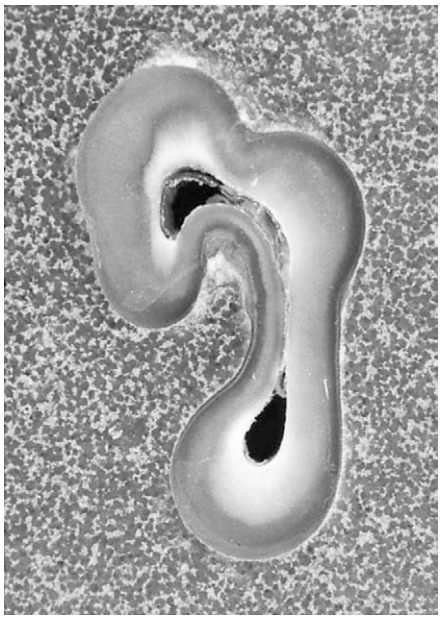


FIG. 7-10 Root section of a premolar showing a ribbon-shaped canal system.

In addition to in vitro studies, a large number of case reports have described a variety of complex canal configurations (see tables 7-8 to 7-27). Some authors have been critical of case studies reporting "freak" cases thought to be rare.[231] However, reports of complex anatomy from both in vitro and in vivo investigations seem to be increasing. This emphasizes the adage that it is easier to recognize an anatomic feature if one is already prepared to see it.

Specific types of canal morphology appear to occur in different racial groups. For example, compared with Caucasian patients, those of African descent have a higher number of extra canals in both the mandibular first premolar (33% versus 14%) and the mandibular second premolar (8% versus 3%).[211] In addition, patients of Asian descent have different percentages of canal configurations than those reported in studies dominated by white and black populations.[223-225,229] One well-recognized ethnic variant is the higher incidence of single-rooted and C-shaped mandibular second molars in Asians compared with other populations.[127] However, this is not always the case; the occurrence of two canals in the

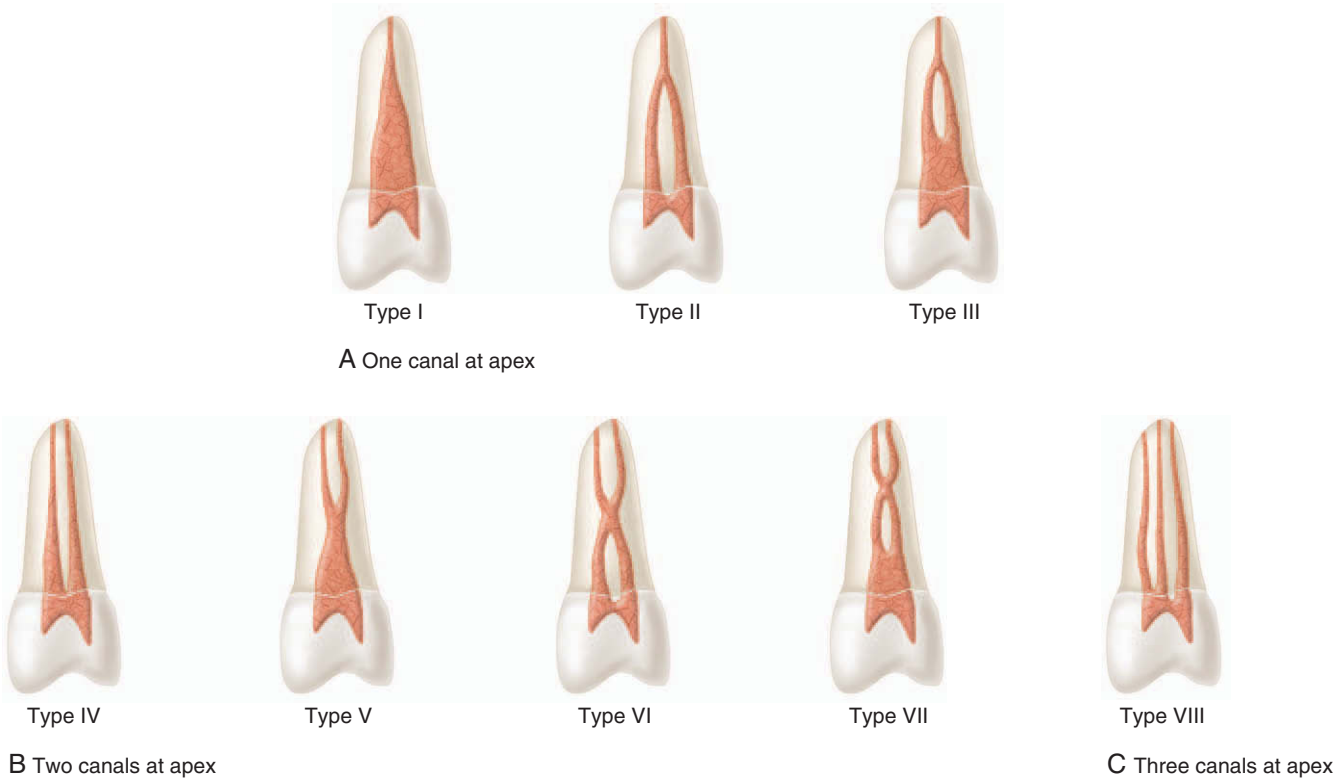


FIG. 7-11 Diagrammatic representation of Vertucci's canal configurations.

TABLE 7-3

Classification and Percentage of Root Canals in the Maxillary Teeth

Tooth	No. 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 7-4

Classification and Percentage of Root Canals in the Mandibular Teeth

Tooth	No. 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.

mesiobuccal root of maxillary first molars in Japanese patients is similar to that described for other ethnic groups.[233] All this information makes it clear that the clinician is confronted daily with highly complex and variable root canal systems. All available armamentaria must be used to achieve a successful outcome.

Examination of the pulp chamber floor can reveal clues to the location of orifices and to the type of canal system present. Importantly, if only one canal is present, it usually is located in the center of the access preparation. All such orifices, particularly if oval shaped, must be explored thoroughly with apically precurved small K-files (Fig. 7-14). 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. 7-15). The relationship of the two orifices to each other is also significant. The closer they are, the greater the chance the two canals join at some point in the body of

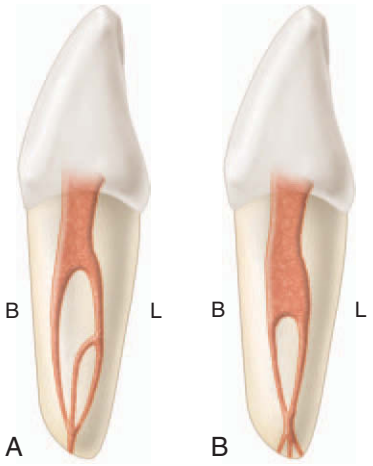


FIG. 7-12 Diagrammatic representation of Kartal and Yanikoglu's canal configurations. *B,* Buccal; *L,* labial.

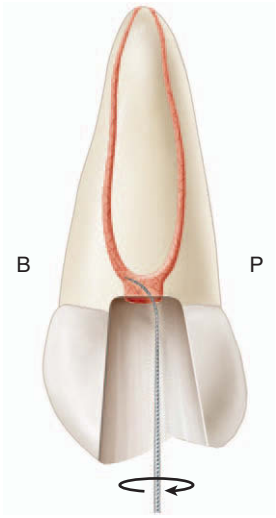


FIG. 7-14 An oval orifice must be explored with apically curved small instruments. The clinician should place the file tip in the orifice with the tip curved to the buccal side when trying to locate the buccal canal. A curved file tip is placed toward the palate to explore for the palatal canal. *B,* Buccal; *P,* palatal.

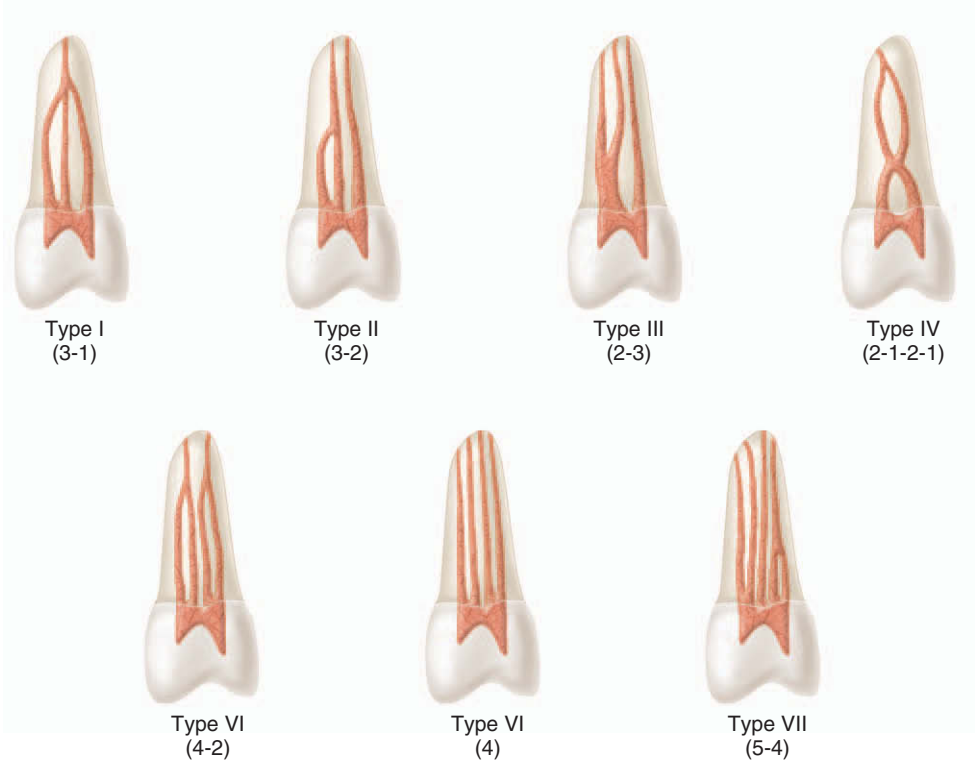


FIG. 7-13 Diagrammatic representation of Gulabivala and co-workers' supplemental canal configurations.

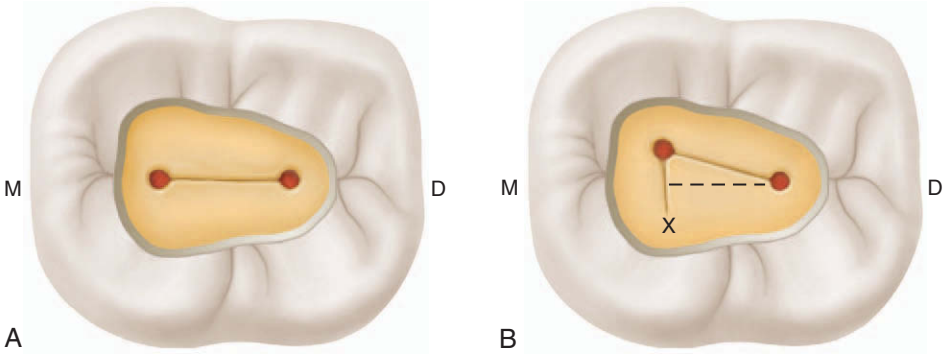


FIG. 7-15 A, In a mandibular second molar with two canals, both orifices are in the mesiodistal midline. **B,** If two orifices are not directly in the mesiodistal midline, a search should be made for another canal on the opposite side, using Krasner and Rankow's laws of anatomy in the area of "X." *D,* Distal; *M,* mesial.

the root. As the distance between orifices in a root increases, the greater the chance is that the canals will remain separate. The more separation between orifices the less the degree of canal curvature.[42] 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 to the buccal or to the lingual aspect, the clinician should suspect a second canal. If two canals are present, they will be smaller than a single canal.

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. This anatomy is best treated by preparing and obturating this canal to the apex and the buccal canal to the point of juncture. If both canals are enlarged to the apex, an hourglass preparation results; the point where the two canals join is more constricted than the preparation at the apex. Filling such a configuration leaves voids in the apical third and invites treatment failure, particularly if microorganisms or their by-products remain in the canal. Rotary nickel–titanium files must be used cautiously with this type of anatomy because instrument separation can occur as the file traverses the sharp curvature into the common part of the canal. 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. 7-16). One investigator[191] recommends 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. This requires modification of the access to achieve unobstructed passage of instruments into the lingual canal.

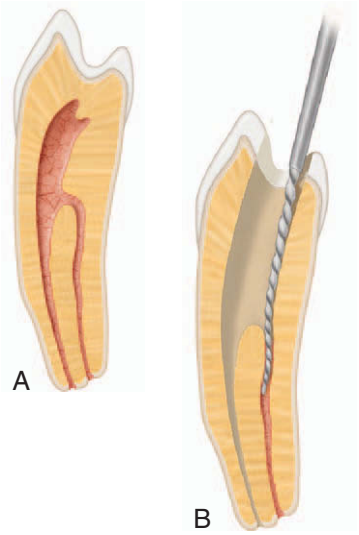


FIG. 7-16 A, Mesial view of a mandibular premolar with a Vertucci type V canal configuration. The lingual canal separates from the main canal at nearly a right angle. **B,** This anatomy requires widening of access in a lingual direction to achieve straight-line access to the lingual canal. This should be done with the dental operating microscope.

ANATOMY OF THE APICAL ROOT

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. 7-17).[110] 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 shaping, cleaning, and obturation. Pulp blood vessels are narrow at the AC, which makes successful treatment of inflammation in the canal difficult. Posttreatment discomfort generally is greater when this area is violated by instruments or filling materials, and the healing process may be compromised.

The CDJ is the point in the canal where cementum meets dentin; it is 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.[178,193]

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.[110] 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."[110] The

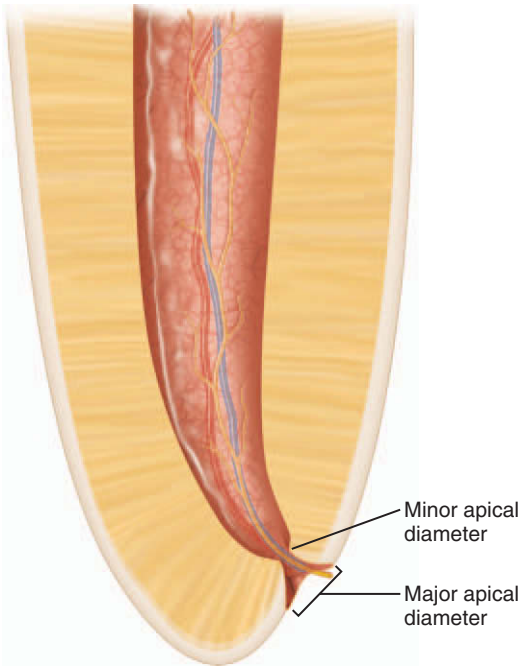


FIG. 7-17 Morphology of the root apex. From its orifice the canal tapers to the apical constriction, or minor apical diameter, which generally is considered the narrowest part of the canal. From this point the canal widens as it exits the root at the apical foramen, or major apical diameter. The space between the minor and major apical diameters is funnel shaped.

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.[110] By comparison, these sizes are larger than the cross-sectional diameter of #50 and #60 endodontic 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.[167] The 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. 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 endodontic files.

In maxillary anterior teeth, the root apex and main AF coincided in 17% of examined central incisors and canines and in 7% of lateral incisors.[136] 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 7-5. The labiolingual diameter in all maxillary anterior teeth is approximately 50 µm more than the mesiodistal diameter. This has definite implications for shaping and cleaning 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. In one study, more than one main foramen was found in all teeth except the palatal root of maxillary molars and the distal root of mandibular molars.[142] 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 7-6. Sizes ranged from 210 µm

*References 30, 80, 81, 163, 176, 200, 215, and 220.

TABLE 7-5

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.

TABLE 7-6

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, Kernani M, Prountzos F: 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.

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 why root canal therapy may fail in premolar teeth.

The morphology of the apical root varies tremendously; it includes numerous accessory canals; areas of resorption and repaired resorption; attached, embedded, and free pulp stones; and varying amounts of irregular secondary dentin.[137,138] 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. 7-18). This variable structure in the apical region presents challenges for root canal therapy. Obturation techniques that rely on the penetration of adhesives into dentinal tubules may not provide successful sealing in the apical region. Therefore the formation of a hybrid layer may become an important part of adhesive systems used in the apical root canal. Conversely, the reduction in dentinal tubules in this apical region leads to reduced chances of bacterial invasion into the dentinal walls of the root.

Considerable controversy exists over the exact termination point for root canal therapy.[187] Clinical determination of apical canal morphology is difficult at best. 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.[47,57,186,232] The apical root canal often is tapered, or walls are parallel to each other, or the canal has multiple constrictions. Some authors therefore have 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 have occurred.[232]

Because locating the AC and AF is difficult clinically, some researchers contend that the radiographic apex is a more reliable reference point.[246] These authors recommend that root canal procedures terminate at or within 3 mm from 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.[104,189] This leaves an apical pulp stump, which prevents extrusion of irritating filling materials into the periradicular tissues. With pulp necrosis, bacteria and their by-products may be present in the apical root canal, which could jeopardize healing. 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.[103,104,189] When therapy ended short of the 2-mm point or extended past the radiographic apex, the success rate declined by 20%. For retreatment cases, therapy 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.

Other investigators who evaluated apical and periradicular tissues after root canal therapy 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.[112-115,172] Procedures terminated more than 2 mm from the AC had the second worst prognosis. These findings occurred with vital and necrotic tissue and when bacteria were present beyond the AF. Sealer or guttapercha (or both) in the periradicular tissues, lateral canals, and apical ramifications may cause a severe inflammatory reaction. However, the authors of the studies acknowledge the difficulty of locating the AC clinically. Some researchers recommend that clinicians terminate all therapy at or beyond the radiographic apex and fill all apical ramifications and lateral canals.[180]

The apical limit of instrumentation and obturation continues to be the subject of major controversy in root canal therapy. However, modern electronic apex locators are reliable instruments that can help the clinician determine the working length of the root canal.

Two hallmarks of the apical region are its variability and unpredictability. The tremendous variation in canal shapes and diameters complicates cleaning and shaping procedures in all dimensions. Successful treatment depends on the anatomy of the root canal system, the dimensions of the canal walls, and the final size of enlarging instruments.

Sometimes the initial file chosen for exploring the canal anatomy and for binding in the canal is used as a measure of the diameter of the apical root canal. However, this technique does not accurately gauge the size of oval-shaped apical root canals. In one study, file binding occurred in 75% of such cases when the file contacted only one side of the apical canal wall, and it occurred without any apical wall contact in the remaining 25%. In 90% of the canals, the diameter of the initial instrument was smaller than the short diameter of the canal.[243] Consequently, using the first file to bind for gauging the diameter of the apical canal and as guidance for apical enlargement is not reliable. More recent studies have suggested that this problem could be remedied by removing the interferences in the coronal and middle thirds of the canal.[45,116] For example, radicular flaring before canal exploration removes interferences and increases the initial file size that is snug at the apex (almost two file sizes greater).[45] Early flaring gives the clinician a better sense of the size of the apical canal, allowing better decisions about the final diameter needed for apical shaping

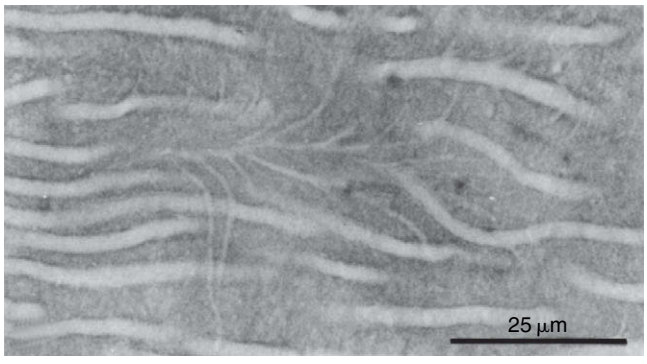


FIG. 7-18 Fine tubules and microbranches can be seen in the apical part of the root. *(From Mjör IA, Nordahl I:* Arch Oral Biol *41:401, 1996.)*

and cleaning. This is another advantage of the crown-down instrumentation technique.

The correlations between maximal canal diameters and instrument diameters varied considerably in a study that compared the shape of the apical root canal of maxillary first molars with the D_0 diameter of endodontic instruments.[71] Evaluation of the root canal diameter showed that a circular shape (the two diameters were equal) predominated in the palatal and MB-2 canals; a flat shape (the larger diameter exceeded the smaller by more than the radius) occurred most often in the main mesiobuccal (MB-1) canal; and both circular and flat shapes were found in the distobuccal canal. Flat and ribbon-shaped canals persisted near the apex even in elderly patients and mainly in the MB-1 canal. This finding was believed to arise from concentric narrowing of ribbon-shaped canals, primarily along the smaller diameter. Oval canals narrowed mainly along the larger diameter and tended to become more circular. The authors of this study concluded that the maxillary first molar has a very complicated canal shape at the apical limit and that this anatomy makes shaping, cleaning, and obturation difficult, particularly in the MB-1 and distobuccal canals. Because of this variability, guidelines for instrument calibers that would guarantee adequate canal preparation are virtually impossible to establish.

One comprehensive study reviewed the apical root canal diameters and tapers of each tooth group and demonstrated that root canals often are long and oval or ribbon shaped in the apical 5 mm (Table 7-7).[244] A long oval canal was defined as having a ratio of the diameters of the long-to-short canal axes greater than 2. This type of canal morphology was found to occur in 25% of the cross-sections studied. In these roots the buccal/lingual diameter is greater than the mesial/distal diameter. This finding held true for all canals except the palatal canal of maxillary molars. These canal measurements suggest

TABLE 7-7

Median Canal Diameter 1, 2, and 5 mm From Apexa

	Buccal/Lingual			Mesial/Distal		
Tooth (Canal) Position	**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.

that apical preparations should be taken to larger sizes than previously recommended.

The authors of another study concluded that "because of long oval canals, larger canal tapers in the buccal–lingual direction, wider ranges in the apical diameters of canals, and the lack of technology to measure these diameters, it is very difficult if not impossible to adequately debride all canals by instrumentation alone."[210] This fact was further emphasized by research that showed that neither the balanced force instrumentation method nor rotary nickel–titanium files allowed controlled preparation of the buccal and lingual extensions of oval canals.[174,245] The instruments created a round bulge in the canal, leaving the extensions unprepared and filled with smear layer and debris. Another series of studies evaluated the effectiveness of five nickel–titanium rotary instrumentation systems on canal debridement and found that all techniques left 35% or more of the canal surface area unchanged.[160,162]

The results of all these studies are fairly predictable, considering the highly variable and irregular morphology of root canal systems and the endodontic intracanal instruments currently in use, which cannot contact all the recesses present along canal walls. These instruments do a good job of shaping the canal but a poor job of accomplishing total canal debridement.

In an attempt to find better ways to clean and disinfect root canals, one group of investigators determined that enlarging canals to greater than the traditionally recommended apical sizes was the only way to remove culturable bacteria from the canal effectively.[35] The larger apical sizes optimized irrigation and disinfection and facilitated mechanical elimination of microbes. A similar study concluded that an increase in the size of canal instrumentation at working length produced an increase in canal cleanliness.[214] Irrigant volume, the number of instrument changes, and the depth of penetration of irrigant needles were less important factors contributing to canal debridement. A potential adverse effect of enlarging the apical canal diameter may be an increased risk of procedural errors or root fractures or both. Further research in this area is required.[1]

As a supplement to these procedures, acoustic streaming by sonic and ultrasonic irrigation appears to enhance the cleanliness of oval canals (Fig. 7-19).[98,121] However, further research must be done to determine the best combination of treatment techniques to effectively debride these highly variable root canal systems.

One study used high-resolution computerized tomography to create a detailed, 3-D model of the root canal system.[161] This method permits precise measurement of canal volume, area, and dimensions. The mean canal diameters in the apical 0.5 mm of maxillary molars were measured for the MB-1 canal (188 ± 5 μm), the distobuccal canal (174 ± 12 μm), and the palatal canal (318 ± 23 μm). Such information goes a long way toward the establishment of final instrumentation sizes for cleaning and shaping.

The apical anatomy studies also indicate that the root apex should be resected 2 to 3 mm during surgical procedures; this removes most of the unprepared and unfilled accessory canals, eliminating a potential reservoir of pathogens.[90,142] In one study, researchers used a bevel perpendicular to the long axis of the root, and apical ramifications and accessory canals increasingly were eliminated by 1 mm of root resection (52% and 40%, respectively), 2 mm of root resection (78% and 40%), and 3 mm of root resection (98% and 93%).[106] This showed that root-end resections of 3 mm are most effective for eliminating most of these structures.

In root-end resections a bevel perpendicular to the long axis of a root exposes a small number of microtubules.[137,138] However, a root resection with a 45-degree bevel exposes a significantly greater number of tubules, increasing the chance of leakage into and out of the root canal.[208,217] To prevent this, root-end cavity preparations should extend coronally to the height of the bevel (Fig. 7-20).

The root apex contains a variety of anatomic structures and tissue remnants. Intercanal connections can become exposed, and a single foramen may become multiple foramina. Treatment results are poor if this altered anatomy is not recognized, prepared, and obturated. One study evaluated the root apex of teeth with refractory apical periodontitis that did not respond to root canal therapy and found that 70% had significant apical ramifications.[222] This incidence strongly suggests a close relationship between the anatomic complexity of the root canal system and the persistence of periradicular pathosis.

An *isthmus* is a narrow, ribbon-shaped communication between two root canals that contains pulp or pulpally derived

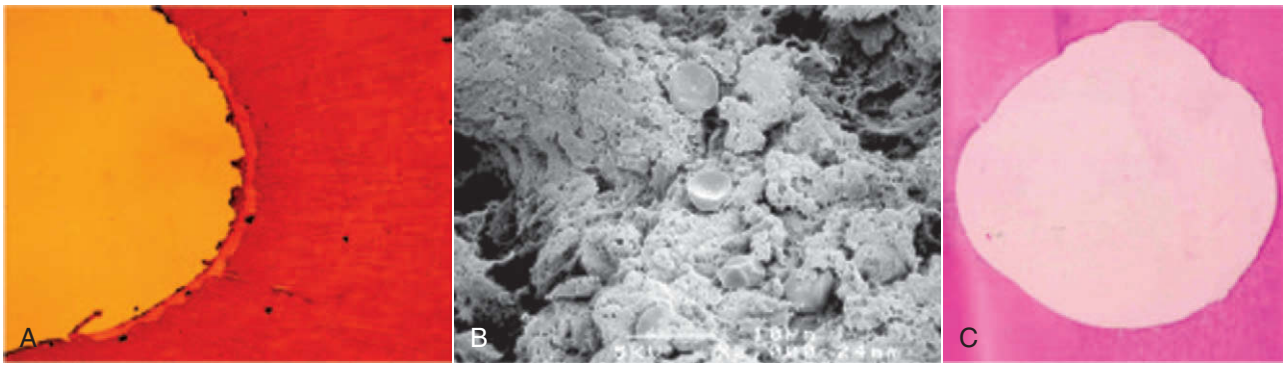


FIG. 7-19 A, Cross-section of a mandibular anterior tooth showing pulp remnants in the recesses of root canal walls in the apical 1 mm after preparation with a rotary nickel–titanium bur (size 40, #.08 taper) and manual irrigation (6% NaOCl and RC-Prep). **B,** Electron photomicrograph of the canal wall in **A,** showing canal wall debris (×3000). **C,** Cross-section of the mandibular anterior tooth showing clean canal walls in the apical 1 mm after preparation with a rotary nickel–titanium bur (size 40, #.08 taper) and sonic acoustic irrigation with 6% NaOCl.

tissue. All isthmi must be found, prepared, and filled during surgery, because they can function as bacterial reservoirs. Any root with two or more canals may have an isthmus. Therefore the presence of an isthmus should be suspected whenever multiple canals are seen on a resected root surface. In one study, the authors recommended that methylene blue dye be used to aid visualization of the outline of the resected root surface and thus detection of an isthmus.[34]

In one study, isthmi in the mesiobuccal root of maxillary first molars were found most often 3 to 5 mm from the root apex.[239] A complete or partial isthmus was found at the 4-mm level 100% of the time. In another study, partial isthmi were found more often than complete isthmi.[204]

Identification and treatment of isthmi are vital to the success of surgical procedures.[34] Investigators [106] identified five types of isthmi that can be found on a beveled root surface (Fig. 7-21).

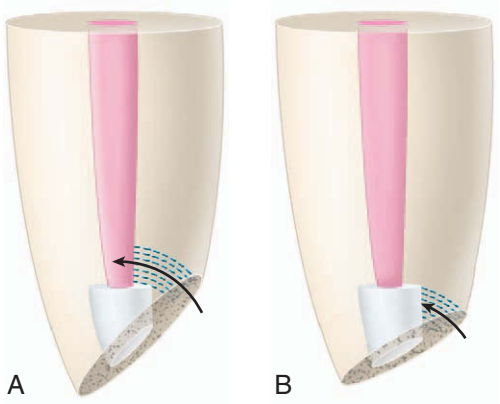


FIG. 7-20 Leakage through dentinal tubules originating at the beveled root surface. **A,** Reverse filling does not extend coronally to the height of the bevel. *Arrows* indicate a possible pathway for fluid penetration. **B,** Reverse filling extends coronally to the height of the bevel, blocking fluid penetration *(arrows)* into the root canal space. *(Redrawn from Vertucci FJ, Beatty RG:* J Endod *12:335, 1986.)*

Isthmi are found in 15% of anterior teeth; in maxillary premolar teeth, they are found in 16% at the 1-mm resection level and in 52% at the 6-mm resection level. The prevalence of isthmi increases in the mesiobuccal root of the maxillary first molar, from 30% to 50% as the root is resected from the 2- to 4-mm level. Eighty percent of the mesial roots of mandibular first molars have isthmi at the 3- to 4-mm resection level, whereas 15% of distal roots have isthmi at the 3-mm level. Microsurgical endodontic techniques have enabled clinicians to visualize the resected root surface and identify the isthmus, prepare it with ultrasonic tips, and fill the root-end preparation with acceptable materials. The recognition and microendodontic treatment of canal isthmi have significantly reduced the failure rate of endodontic surgery.[174,175]

OBJECTIVES AND GUIDELINES FOR ACCESS CAVITY PREPARATION

Objectives

Access is the first and arguably most important phase of nonsurgical root canal treatment. A well-designed access preparation is essential for a good endodontic result.[203] Without adequate access, instruments and materials become difficult to handle properly in the highly complex and variable root canal system. The objectives of access cavity preparation are (1) to remove all caries, (2) to conserve sound tooth structure, (3) to completely unroof the pulp chamber, (4) to remove all coronal pulp tissue (vital or necrotic), (5) to locate all root canal orifices, (6) to achieve straight- or direct-line access to the apical foramen or to the initial curvature of the canal, and (7) to establish restorative margins to minimize marginal leakage of the restored tooth.

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. 7-22). Straight line access provides the best chance of debridement of the entire canal space and reduces the risk of file breakage.[140] When prepared correctly the access cavity allows complete irrigation, shaping and cleaning, and quality obturation. Ideal access results in straight entry into the canal orifice, with the line angles forming

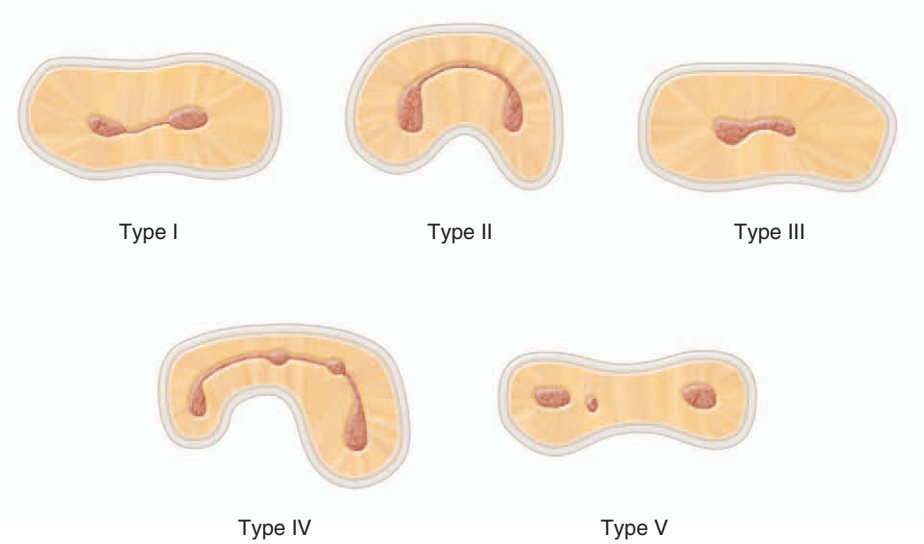


FIG. 7-21 Schematic representation of isthmus classifications described by Kim and colleagues.[106] Type I is an incomplete isthmus; it is a faint communication between two canals. Type II is characterized by two canals with a definite connection between them (complete isthmus). Type III is a very short, 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.



FIG. 7-22 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.

a funnel that drops smoothly into the canal(s). Projection of the canal center line to the occlusal surface of the tooth indicates the location of the line angles. Connection of the line angles creates the outline form. Modifications of the outline form may be needed to facilitate location of canals and to create a convenience form. The clinician must find a balance between creating adequate access and removing too much dentin, which could compromise the final restoration or promote crown fracture.

Guidelines

The guidelines described in the following sections are essential for the completion of an ideal access preparation.

Visualization of the Likely Internal Anatomy

Because internal anatomy dictates access shape, 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 tooth anatomy at the coronal, cervical, and root levels. 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 aids the determination of root location and direction. The clinician uses the information from these assessments to choose the direction of initial bur penetration.

Evaluation of the Cementoenamel Junction and Occlusal Anatomies

Traditionally, access cavities have been prepared in relation to the occlusal anatomy. However, complete reliance on the occlusal anatomy is dangerous because this morphology can change as the crown is destroyed by caries and reconstructed with various restorative materials. Complete dependence on the occlusal anatomy may explain the occurrence of some procedural errors. In one a study involving 500 pulp chambers, Krasner and Rankow[108] found that the cementoenamel junction (CEJ) was the most important anatomic landmark for determining the location of pulp chambers and root canal orifices. The study demonstrated the existence of a specific and consistent anatomy of the pulp chamber floor. These authors proposed nine guidelines, or laws, of pulp chamber anatomy to help clinicians determine the number and location of orifices on the chamber floor (Fig. 7-23).

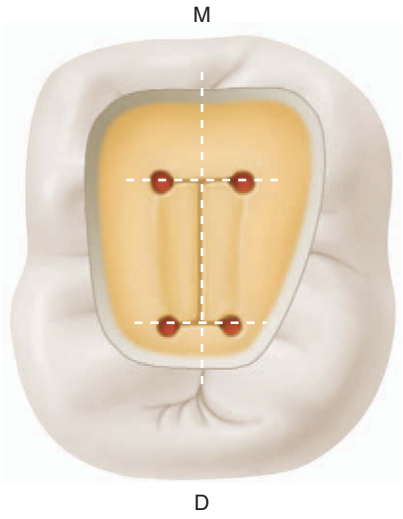


FIG. 7-23 Diagrammatic representation of Krasner and Rankow's first and second laws of symmetry and first through third laws of orifice location. *D,* Distal; *M,* mesial.

Law of centrality: The floor of the pulp chamber is always located in the center of the tooth at the level of the CEJ.

Law of 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.

Law 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 is the most consistent repeatable landmark for locating the position of the pulp chamber.

First law of 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.

Second law of symmetry: 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.

Law of color change: The pulp chamber floor is always darker in color than the walls.

First law of orifice location: The orifices of the root canals are always located at the junction of the walls and the floor.

Second law of orifice location: The orifices of the root canals are always located at the angles in the floor–wall junction.

Third law of orifice location: 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 laws.[108] Slightly fewer 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 means of achieving straight-line access and diminishing 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.[132] This allows better access to the lingual canal and improves canal debridement (Fig. 7-24).

Removal of All Defective Restorations and Caries Before Entry Into the Pulp Chamber

The clinician must remove all defective restorations before entering the root canal system. With an open preparation, canals are much easier to locate, and shaping, cleaning, and obturation are much easier to perform. In one study,[1] it was determined that clinicians were about 40% more likely to miss fractures, caries, and marginal breakdown if restorations were not completely removed. Working through restorations also allows restorative debris to become more easily lodged in the canal system (see Fig. 7-74, D).

All carious dentin must be removed during access preparation. This removal prevents irrigating solutions from leaking past the rubber dam into the mouth and prevents carious dentin and its bacteria from entering the root canal system. If a chamber wall is perforated during removal of carious dentin, allowing leakage of saliva into the pulp spaces, the wall must be repaired immediately with a temporary filling material, preferably from inside the cavity preparation. Sometimes removal of extensive defective restorations and carious dentin may not leave enough tooth structure for placement of a rubber dam clamp to seal against salivary contamination. A crown-lengthening procedure should be performed to correct this situation before the root canal procedure is begun.



FIG. 7-24 An incisal access cavity on mandibular anterior teeth may allow for improved straight-line access and canal debridement.

Removal of Unsupported Tooth Structure

Preparation of an access cavity results in the removal of part of the central portion of the tooth thereby reducing the tooth's resistance to occlusal stresses. After completing the preparation, the clinician should remove all unsupported tooth structure to assess restorability and to prevent tooth fracture. Unnecessary removal of sound tooth structure should be avoided.

Creation of Access Cavity Walls That Do Not Restrict Straight- or Direct-line Passage of Instruments to the Apical Foramen or Initial Canal Curvature

Complete clinician control over all enlarging and filling instruments is vital. 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 is dependent 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 down 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). Following this guideline minimizes the occurrence of procedural errors and maximizes the effectiveness of shaping, cleaning, and obturation instruments.

Delay of Dental Dam Placement Until Difficult Canals Have Been Located and Confirmed

Difficulty can arise in gaining access into teeth that are crowded and rotated, fractured to the free gingival margin, heavily restored and calcified, or part of a fixed prosthesis. In these situations the clinician's best course of action may be to prepare the initial part of the access cavity before placing the dental dam so that the inclination of root eminences can be visualized; this information can be used as an indicator of the direction of the long axis of the treated tooth. Micro-Openers (DENTSPLY Maillefer, Tulsa, OK) (Fig. 7-25) are excellent instruments for locating canal orifices when a dental dam has not been placed. These flexible, stainless steel hand instruments have #.04 and #.06 tapered tips. They also have offset handles that provide enhanced visualization of the pulp chamber.

The dental dam must be placed once the roof of the pulp chamber has been penetrated and the canals identified.

Location, Flaring, and Exploration of All Root Canal Orifices

A sharp endodontic explorer is used to locate canal orifices and to determine their angle of departure from the pulp

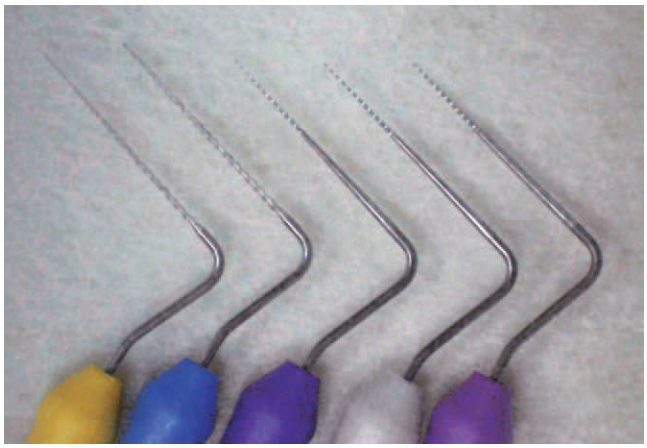


FIG. 7-25 Set of Micro-Openers (DENTSPLY Maillefer, Tulsa, OK) for canal identification and enlargement.

chamber. Next, all canal orifices and the coronal portion of the canals are flared to make instrument placement easier. The canals are then explored with small, precurved K-files (#6, #8, or #10). The clinician must take care to keep these instruments within the confines of the canal system until the working length has been accurately determined. A lubricating agent (e.g., RC-Prep [Premier Dental Products, Plymouth Meeting, PA], a water-based preparation that will not congeal vital pulp tissue), may be used on instruments and introduced into the canal. Congealed pulp tissue may form a collagen plug that blocks the apex, preventing complete shaping and cleaning.

Inspection of the Pulp Chamber, Using Magnification and Adequate Illumination

Magnification and illumination are particularly important in root canal therapy, especially for determining the location of canals; negotiating constricted, curved, and calcified canals; and débriding and removing tissue and calcifications from the pulp chamber. Enhanced vision allows the clinician to see internal dentin color changes and subtle landmarks that may not be visible to the unaided eye. Surgical loupes, endodontic endoscopes,[12] and the DOM are some of the commercially available instruments that can help the clinician accomplish these goals. A clinician trained in microscopic techniques has a better chance of locating and negotiating intricate root canal systems.

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. At least 3.5 mm of temporary filling material (e.g., Cavit [3M, St. Paul, MN]) is needed to provide an adequate coronal seal for a short time.[230] Recently, canal orifice plugs of composite, glass ionomer, and mineral trioxide aggregate (ProRoot MTA; DENTSPLY Tulsa Dental Specialties, Tulsa, OK) 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

Armamentaria

The preparation of an access cavity requires the following equipment:

- Magnification and illumination
- Handpieces
- Burs
- Endodontic explorer (DG-16, DE-17)
- Endodontic spoon
- #17 operative explorer
- Ultrasonic unit and tips

Magnification and Illumination

The access cavity cannot be prepared adequately without the use of magnification and an appropriate light source. At the least, the clinician needs surgical loupes with an auxiliary light source (see Chapter 6). The DOM is the preferred means of magnification and illumination.

Handpieces

An experienced clinician with good tactile awareness is likely to perform most phases of access preparation with a high-speed handpiece. After penetration of the dentin, a less experienced clinician may benefit from the increased tactile awareness offered by a slow-speed handpiece. 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 used with the DOM.

Burs

Numerous burs have been developed to assist the clinician with 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. 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. 7-26) are used extensively in the preparation of access cavities. They are used to remove 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. 7-27) or a diamond bur with a rounded cutting end (Fig. 7-28) 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 (see Fig. 7-75, *A*).

Fissure carbide and diamond burs with safety tips (i.e., noncutting ends) (Fig. 7-29) 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



FIG. 7-26 Access burs: #2, #4, and #6 round carbide burs.



FIG. 7-27 Access bur: #57 fissure carbide bur.

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, which are used as reference points for the working length determination.

Round diamond burs (sizes #2 and #4) (Fig. 7-30) are needed when endodontic access must be made through porcelain or ceramometal restorations. Diamond burs are less traumatic to porcelain than carbide burs and are more likely to



FIG. 7-28 Access bur: round-end cutting tapered diamond bur.



FIG. 7-29 Access burs: Safety-tip tapered diamond bur *(left)*; safety-tip tapered carbide bur *(right)*.

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 the clinician should switch to a carbide bur for metal or dentin penetration because of this bur's greater cutting efficiency.



FIG. 7-30 Access burs: #2 and #4 round diamond burs.



FIG. 7-31 Access bur: Transmetal bur.

A 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–manufacturing companies are aware of these issues and they are currently introducing medium- and fine-grit diamond burs that efficiently cut zirconia. These diamond burs should be used with copious water spray to minimize heat buildup in the zirconia crowns during access preparations. Also, some diamond burs 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. A transmetal bur (Fig. 7-31) is excellent for this purpose because of its exceptional cutting efficiency. To penetrate a metal restoration, the clinician should always use a new transmetal bur with water spray for maximal cutting effect.

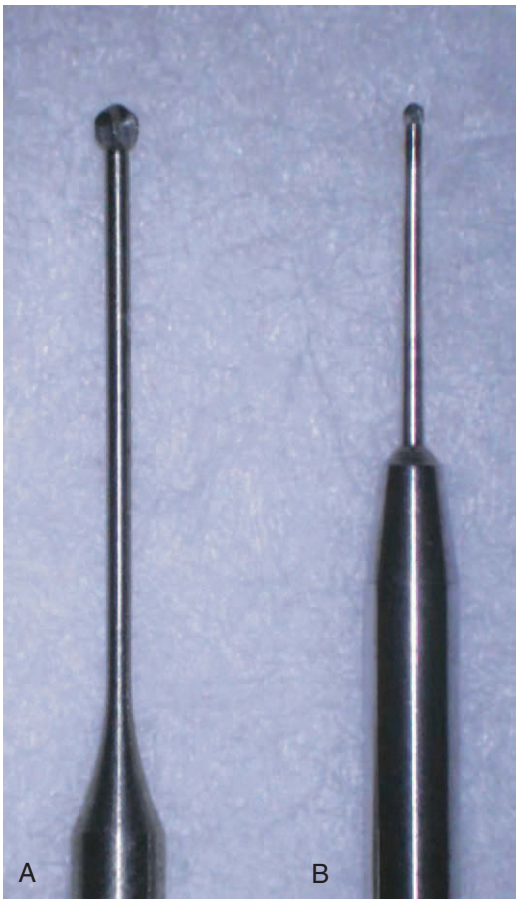


FIG. 7-32 Access burs. **A,** Mueller bur. **B,** LN bur.

If a tooth has a receded pulp chamber and calcified orifices, the clinician often must cut into the root to locate and identify the canal orifices. Extended-shank round burs, such as the Mueller bur (Brasseler USA, Savannah, GA) (Fig. 7-32, *A*) and the LN bur (DENTSPLY Maillefer, Tulsa, OK) (Fig. 7-32, *B*), are useful for this purpose. The Munce Discovery bur (CJM Engineering, Santa Barbara, CA) is similar to the Mueller 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 shaping and cleaning to enter the canal(s) easily and effortlessly. Gates-Glidden burs can be used for this purpose, starting with smaller sizes and progressing to the larger sizes (Fig. 7-33). More recently, #.12 tapered rotary endodontic files (Fig. 7-34) have been used for the flaring and blending procedure. When using either of these two techniques for canal flaring, the clinician must guard against removing excessive dentin on the furcation side of a root canal, which could create a "strip" perforation.

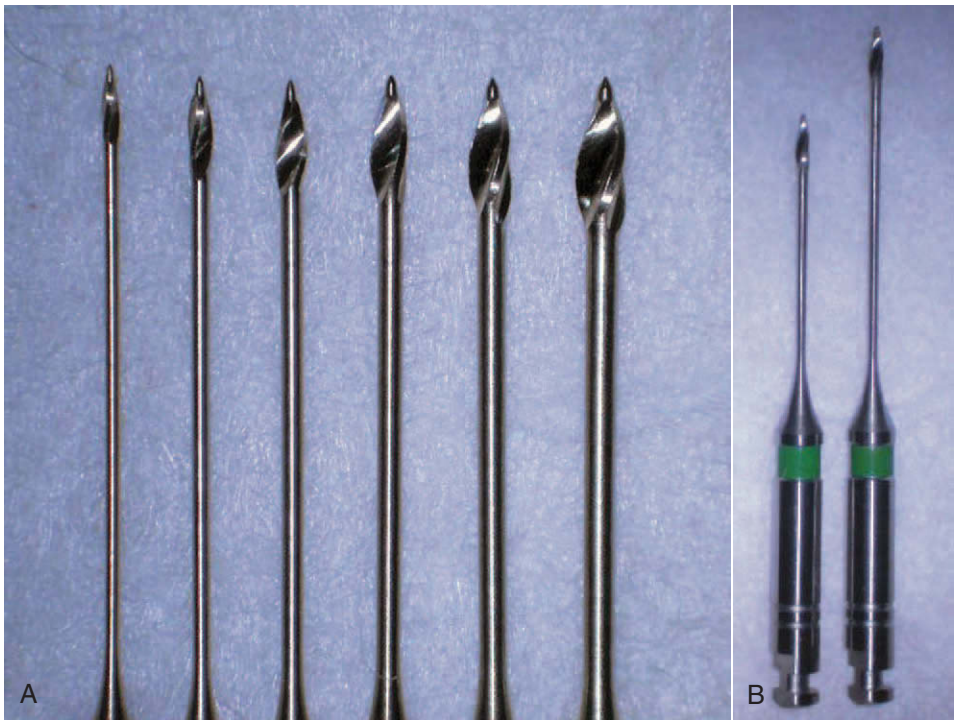


FIG. 7-33 Access burs. **A,** Gates-Glidden burs, 1 through 6. **B,** Gates-Glidden burs, short shank *(left)* and regular shank *(right)*.



FIG. 7-34 Access bur: #.12 taper nickel–titanium orifice opener.

Endodontic Explorer, Endodontic Spoon, #17 Operative Explorer

Various hand instruments are useful for preparing access cavities. The DG-16 endodontic explorer (Fig. 7-35) is used to identify canal orifices and to determine canal angulation. The JW-17 endodontic explorer (Fig. 7-35) (CK Dental Industries, Orange, CA) serves the same purpose, but its thinner, stiffer tip can be useful for identifying calcified canals. The endodontic spoon (Fig. 7-36) can be used to remove coronal pulp and carious dentin. A #17 operative explorer is useful for detecting any remaining pulp chamber roof, particularly in the area of a pulp horn (Fig. 7-37).

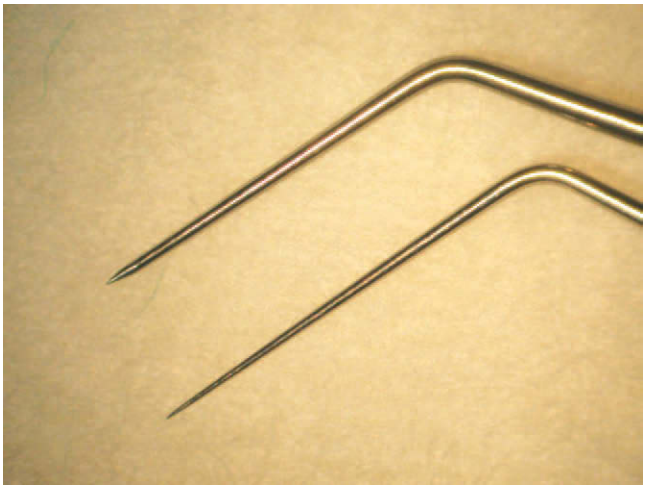


FIG. 7-35 Access instruments: DG-16 endodontic explorer *(top)*; JW-17 endodontic explorer *(bottom)*.

Ultrasonic Unit and Tips

An ultrasonic unit (Fig. 7-38) and tips specifically designed for endodontic procedures can be valuable aids in the preparation of access cavities. Ultrasonic tips (Fig. 7-39) can be used to trough and deepen developmental grooves to remove tissue



FIG. 7-36 Access instrument: Endodontic spoon.



FIG. 7-37 Removal of the pulp horn is evaluated with a #17 operative explorer.



FIG. 7-38 Endodontic ultrasonic unit. (*Courtesy SybronEndo, Orange, CA.*)

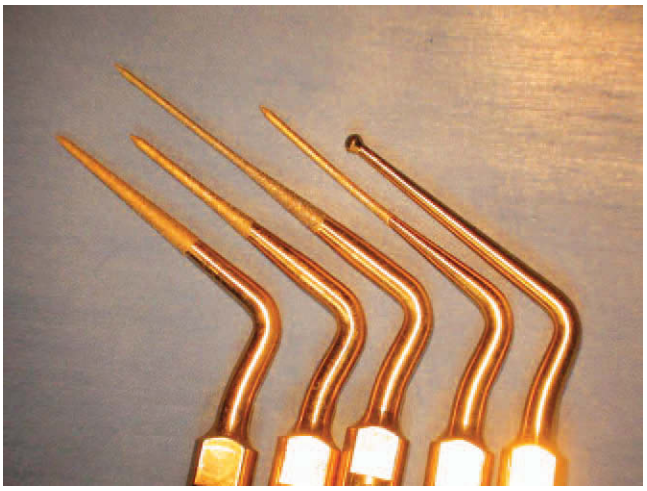


FIG. 7-39 Endodontic ultrasonic tips.

and explore for canals. Ultrasonic systems provide outstanding visibility compared with conventional handpiece heads, which typically obstruct vision. Fine ultrasonic tips are smaller than conventional round burs, and their abrasive coatings allow clinicians to sand away dentin and calcifications conservatively when exploring for canal orifices.

Access Cavity Preparations

Anterior Access Cavity Preparations

Many of the same steps are used in similar tooth types to prepare an access cavity. The following discussion outlines the steps for maxillary and mandibular anterior teeth. Tooth-specific access concerns are illustrated and discussed in the section Morphology and Access Cavity Preparations for Individual Teeth, later in the chapter.

REMOVAL OF CARIES AND PERMANENT RESTORATIONS

Caries typically is removed early, before the pulp chamber is entered. This minimizes the risk of contamination of the pulp chamber or root canal(s) with bacteria. Defective permanent restorations, whether amalgams, composite resins, or crowns, must be removed entirely to prevent coronal leakage from contaminating the pulp chamber, the root canal(s), or both after the endodontic appointment. Removal of defective permanent restorations also permits straight-line access and prevents restorative fragments from becoming lodged in the root canal system (see Fig. 7-74, *D*). If recurrent decay is detected or suspected, the permanent restoration must be removed entirely to prevent coronal contamination of the pulp chamber.

The management of intact permanent restorations when recurrent caries is not present requires some judgment. Amalgam and composite restorations typically are removed entirely to improve visibility during the search for root canal orifices. As stated in an earlier study,[1] clinicians were about 40% more likely to miss fractures, caries, and marginal breakdown if restorations were not completely removed. However, the clinician may want to retain the proximal portion of a class II restoration that extends subgingivally to aid in rubber dam isolation. When parts of existing permanent restorations are

not removed, the clinician usually can widen the access opening larger than is ideal at the expense of removing restorative material, not sound tooth structure. The remaining permanent restoration material is removed at the end of the appointment before the temporary restoration is placed.

Often clinicians decide to perform endodontic therapy through intact crowns rather than removing or replacing them. These access cavities are repaired after completion of the root canal procedure. The patient and the clinician must realize that visibility can be compromised in these circumstances, particularly if a DOM is not used. The clinician can enhance visibility by beveling the crown's cavosurface margins and by making sure all axial walls are glassy smooth.

INITIAL EXTERNAL OUTLINE FORM

Once caries and restorations have been addressed, the clinician creates an initial external outline opening on the lingual surface of the anterior tooth. This step often is performed during the removal of caries and restorations. For an intact tooth, the clinician should begin in the center of the lingual surface of the anatomic crown (Fig. 7-40, *A*). A #2 or #4 round bur or a tapered fissure bur is used to penetrate through the enamel and slightly into the dentin (approximately 1 mm). An outline form is created, similar in geometry to an ideal access shape for the particular anterior tooth (see Fig. 7-40, *B*); it is one 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 (see Fig. 7-40, *C*).

PENETRATION OF THE PULP CHAMBER ROOF

Experienced clinicians can comfortably penetrate the pulp chamber roof with a high-speed handpiece, but less experienced clinicians may find the increased tactile sensation of a slow-speed handpiece a safer option. Continuing with the

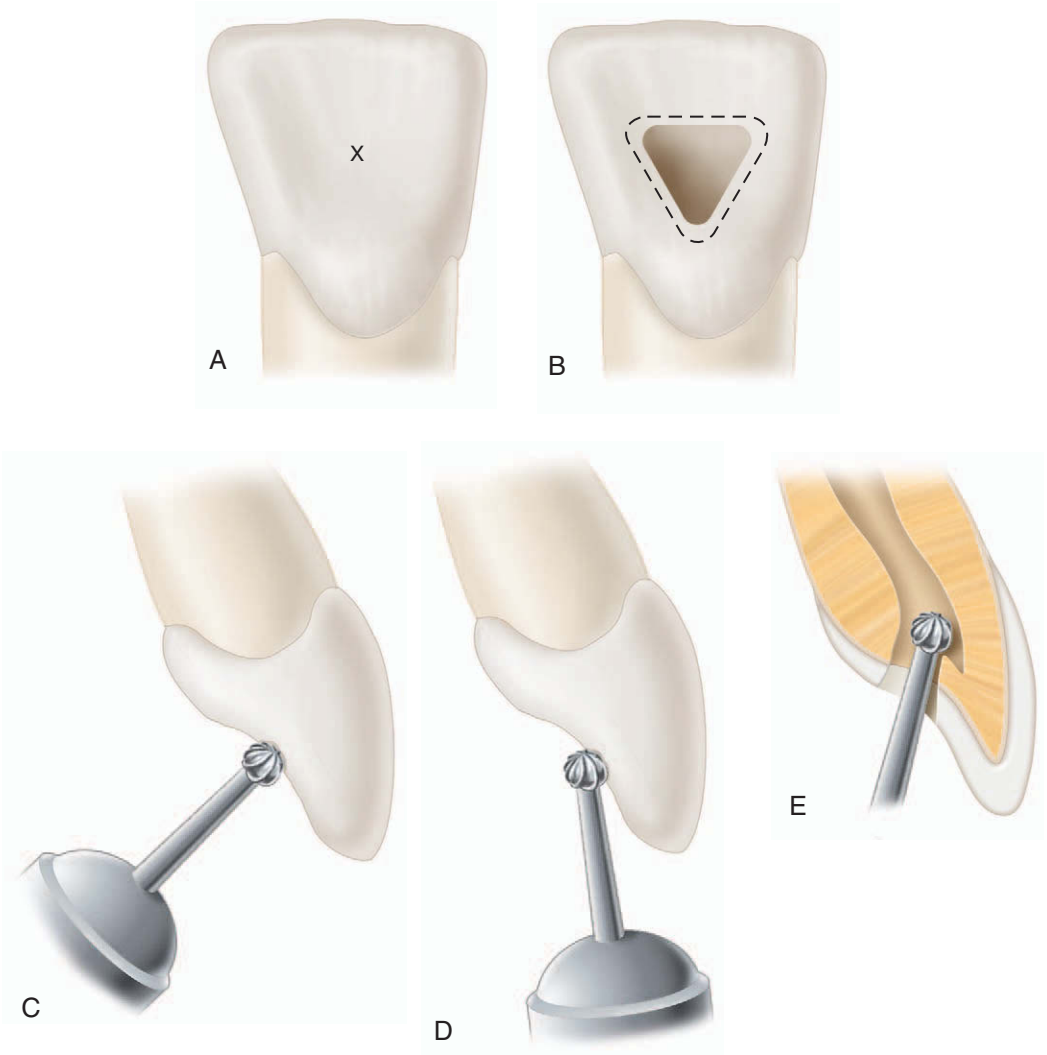


FIG. 7-40 A, In anterior teeth the starting location for the access cavity is the center of the anatomic crown on the lingual surface (*X*). **B,** Preliminary outline form for anterior teeth. The shape should mimic the expected final outline form, and the size should be one half to three fourths the 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.

same round or tapered fissure bur, the clinician changes the angle of the bur from perpendicular to the lingual surface to parallel to the long axis of the root (see Fig. 7-40, *D*). Penetration into the tooth is accomplished along this root's long axis until the roof of the pulp chamber is penetrated; frequently a drop-in effect is felt when this occurs. The clinician should measure the distance from the incisal edge to the roof of the pulp chamber on a dimensionally accurate pretreatment radiograph and limit penetration to this distance. If the drop-in effect is not felt at this depth, the clinician should evaluate the situation carefully to prevent a gouge or perforation. 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. If all looks good, the clinician should probe the access opening with an endodontic explorer, using magnification and illumination. Often the sharp explorer tip penetrates through the pulp chamber roof with firm pressure. Angled radiographs should be taken to assess progress if any confusion or doubt exists. A little caution and concern at this stage can prevent an iatrogenic mishap (see Fig. 7-75, *C*).

COMPLETE ROOF REMOVAL

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. 7-40, *E*). Because each tooth has a unique pulp chamber anatomy, working in this manner allows the internal pulp anatomy to dictate the external outline form of the access opening. In vital cases, pulp tissue hemorrhage can impair the clinician's ability to see the internal anatomy. In such cases, as soon as enough roof has been removed to allow instrument access, the coronal pulp should be amputated at the orifice level with an endodontic 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 with a chelating agent then can be introduced into the canal and rotated, thereby amputating the radicular pulp at a more apical level. This procedure is followed by irrigation with sodium hypochlorite. After hemorrhage has been controlled, allowing visibility, all of the pulp chamber roof, including the pulp horns, must be removed and all internal walls must be flared to the lingual surface of the tooth. Complete roof removal is confirmed with a #17 operative explorer if no "catches" are discovered as the explorer tip is withdrawn from the pulp chamber along the mesial, distal, and facial walls.

IDENTIFICATION OF ALL CANAL ORIFICES

After the pulp chamber has been unroofed, the canal orifices are located with an endodontic explorer (Fig. 7-41). This instrument is to the endodontist what a periodontal probe is to the periodontist. Used for reaching, feeling, and often digging at the hard tissue, it is the tactile extension of the clinician's fingers. Natural anatomy indicates the usual places for orifices, but restorations, dentinal protrusions, and dystrophic calcifications can dictate the actual configuration the clinician encounters. While probing the chamber floor, the explorer often penetrates or dislodges calcific deposits blocking an orifice. It also can be used to evaluate straight-line access. Positioning the explorer in an orifice allows the clinician to check the shaft for clearance from the axial walls and to determine the angle at which a canal departs the main chamber (Fig. 7-42).



FIG. 7-41 An endodontic explorer is used to search for canal orifices.

The endodontic explorer is preferred over the rotating bur as the instrument for locating canal orifices. Not only is the explorer a safer option, its double-ended design also offers two angles of approach. The clinician should keep in mind the probability of finding additional canals in the tooth and the most likely anatomic location of these canals.

In a study[94] involving maxillary molars, the authors looked at several pretreatment variables and their influence on the clinician's likelihood of detecting root canals. The results of this study indicated that the number of root canals detected varied according to age and caries. Age was a significant factor in detecting fewer canals, most likely due to the calcification and morphologic changes that occurs in root canals as a patient ages. The presence of caries at the time of treatment resulted in the detection of greater number of root canals. However, because caries was associated with younger individuals in this study, age seems to play the dominant role in the clinician's likelihood of detecting root canals.

REMOVAL OF THE LINGUAL SHOULDER AND ORIFICE AND CORONAL FLARING

Once the orifice(s) has been identified and confirmed, the lingual shoulder is removed. This structure is the lingual shelf of dentin that extends from the cingulum to a point approximately 2 mm apical to the orifice (Fig. 7-43). Its removal aids straight-line access and allows for more intimate contact of files with the canal's walls for effective shaping and cleaning.

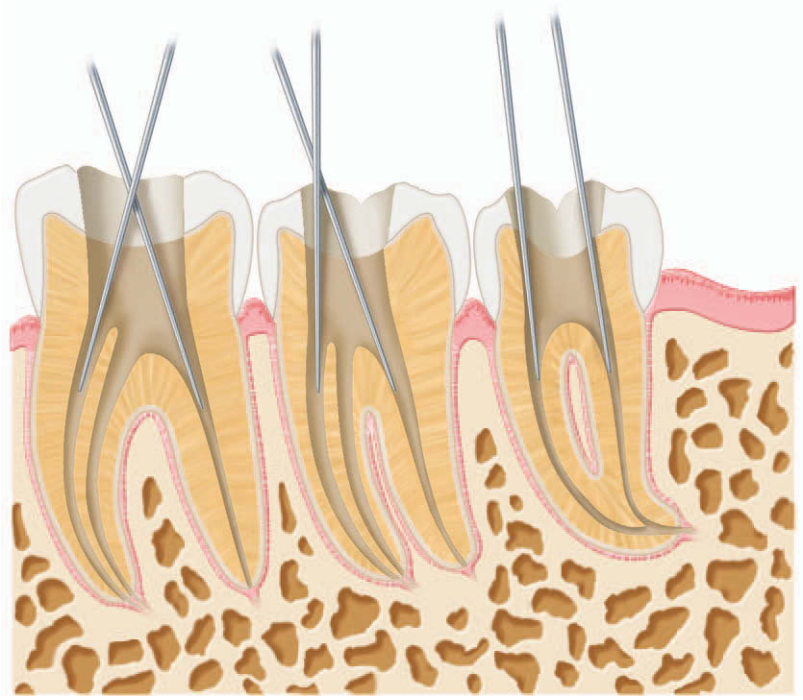


FIG. 7-42 Root canal orifices are explored to assess straight-line access and to determine the path of insertion for endodontic instruments.

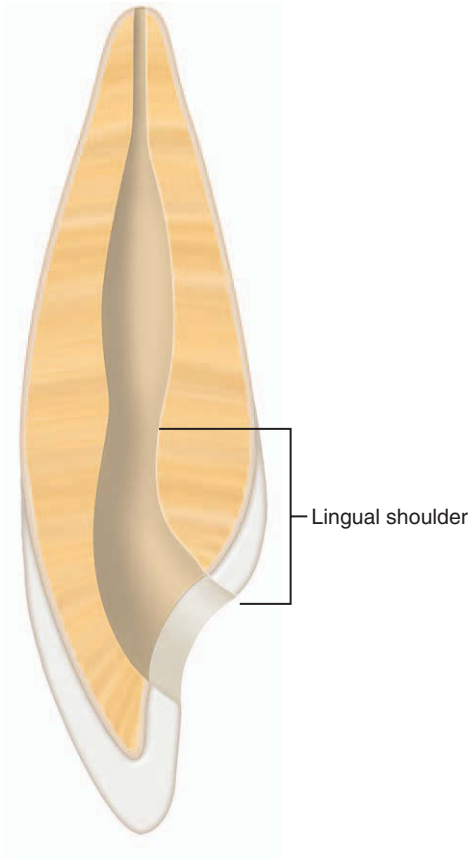


FIG. 7-43 Lingual shoulder of the anterior tooth, extending from the cingulum to 2 mm apical to the orifice.

The lingual shoulder can be removed with a tapered safety-tip diamond or carbide bur or with Gates-Glidden burs. The tip of a fine safety-tip diamond bur is placed approximately 2 mm apical to the canal orifice and inclined to the lingual during rotation to slope the lingual shoulder. The clinician must be careful when using this bur to avoid placing a bevel on the incisal edge of the access preparation (Fig. 7-44). When Gates-Glidden burs are used, the largest that can passively be placed 2 mm apical to the orifice is used first. During rotation, the bur is leaned against the lingual shoulder and withdrawn. The clinician can increase the size of these burs sequentially, depending on the size of the canal, and repeat the shaping of the lingual wall until the lingual shoulder of dentin has been eliminated.

During this process the orifice should also be flared so that it is contiguous with all walls of the access preparation. This can be done with small to large Gates-Glidden burs. These burs are used in a circumferential filling motion, flaring each wall of the canal in sequence. To prevent iatrogenic mishaps on thin walls facing a root concavity, these burs should be placed passively into the canal and rotated as they are gently leaned against a canal wall and withdrawn.

Another approach to flaring the orifice involves the use of rotary nickel–titanium orifice openers; they should be used at slow speeds and low torque.

STRAIGHT-LINE ACCESS DETERMINATION

After the lingual shoulder has been removed and the orifice(s) flared, the clinician must determine whether straight-line access has been achieved. Ideally, an endodontic file can approach the apical foramen or the first point of canal curvature undeflected. Unnecessary deflection of the file can result in numerous consequences related to loss of instrument control. Deflected instruments function under more stress than



FIG. 7-44 Placing an incisal bevel on the lingual surface of a maxillary anterior tooth can lead to fracture of the permanent restoration during occlusal function.

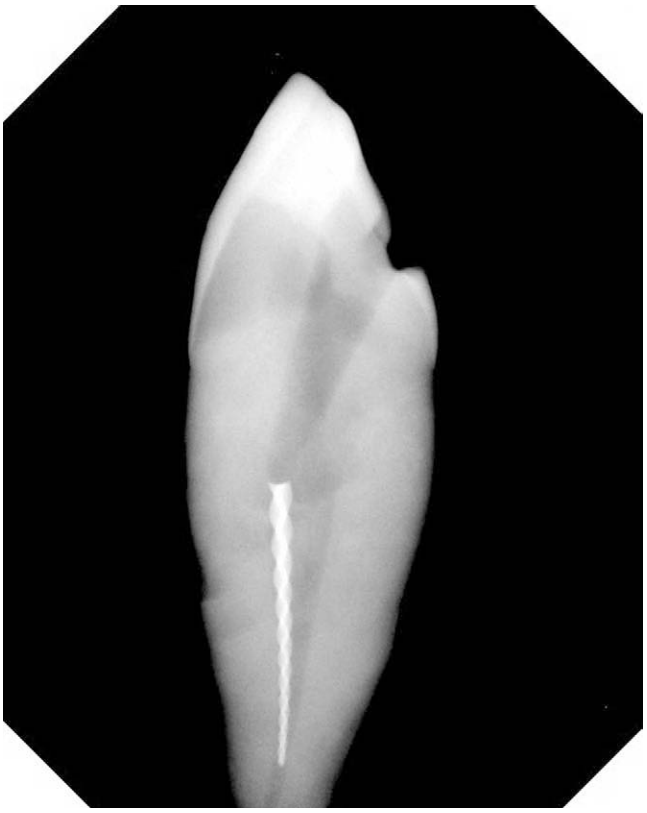


FIG. 7-45 Separation of a rotary endodontic instrument as a result of underextended access preparation rather than canal binding.

undeflected instruments and are more susceptible to separation during the shaping and cleaning process (Fig. 7-45). Deflected instruments also lack access to critical areas of the canal and therefore do not shape and clean effectively. Attempts to shape and clean without straight-line access often lead to procedural errors such as ledging, transportation, and zipping (Fig. 7-46). A ledge is an iatrogenically created root canal wall irregularity that may impede placement of an intracanal instrument to the apex. Transportation occurs in the portion of the canal apical to a curvature when canal wall structure opposite the curve is removed, tending to straighten the canal curvature. Zipping, or elliptication of the apical foramen, occurs when an overextended file transports the outer wall of the apical foramen. Conversely, undeflected instruments provide better tactile sensation, which is necessary for "feeling" the canal anatomy and "feeling" how the file is performing in the root canal system.

Straight-line access is evaluated by inserting into the canal the largest file that fits passively to the apical foramen or the point of the first canal curvature. This internal length can be determined by measuring a diagnostic pretreatment periapical radiograph. The file is inserted gently and withdrawn as the clinician "feels" for canal binding or deflection. If deflection is detected, the clinician must reevaluate the adequacy of lingual shoulder removal before changing the incisal edge position of the access preparation. Inadequate removal of the lingual shoulder causes the file to deflect in a facial direction, and an



FIG. 7-46 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.



FIG. 7-47 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.

inexperienced clinician may overextend the incisal edge of the access preparation in an attempt to achieve straight-line access (Fig. 7-47). If the lingual shoulder has been adequately removed and the file still binds on the incisal edge, the access cavity should be extended farther incisally until the file is not deflected. 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

The clinician should inspect and evaluate the access cavity, using appropriate magnification and illumination. Although this 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 canal must be evaluated for a bifurcation (see Fig. 7-14).

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. Definite, smooth cavosurface margins allow the clinician to place and finish a composite resin final restoration with the precision necessary to minimize coronal leakage. Such leakage could jeopardize the success of the root canal procedure.

Another factor the clinician must consider when finalizing the access margins 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 movement. Therefore the restorative margins of 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 excursive functional loads and ultimately result in coronal leakage. Obviously, if the anterior tooth requires a crown as the final restoration, the access cavosurface margin becomes a less critical factor.

INDIVIDUAL ANTERIOR TEETH

Please see the figures in the section Morphology and Access Cavity Preparations for Individual Teeth, later in the chapter.

Posterior Access Cavity Preparations

The process of preparing access cavities on posterior teeth is similar to that for anterior teeth, but enough differences exist to warrant a separate discussion.

REMOVAL OF CARIES AND PERMANENT RESTORATIONS

The discussion of caries and permanent restoration removal presented in the previous section, Anterior Access Cavity Preparations, applies equally to posterior teeth. Posterior teeth requiring root canal therapy typically have been heavily restored or the carious process is extensive. Such conditions, along with the complex pulp anatomy of posterior teeth, can make the access process challenging.

INITIAL EXTERNAL OUTLINE FORM

The removal of caries and existing restorations often accomplishes the creation of an initial external outline form. As with anterior teeth, the pulp chamber of posterior teeth is positioned in the center of the tooth at the level of the CEJ. An access starting location must be determined for an intact tooth. In maxillary premolars this point is on the central groove between the cusp tips (Fig. 7-48). Crowns of mandibular premolars are tilted lingually relative to their roots (Fig. 7-49), and the starting location must be adjusted to compensate for this tilt (Fig. 7-50). 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 clinician must establish the mesial and distal boundary limitations (Fig. 7-51). Evaluation of bite-wing radiographs is an accurate method of assessing the mesiodistal extensions of the pulp chamber. The mesial boundary for both the maxillary and mandibular molars is a line connecting the mesial cusp tips. Rarely are pulp chambers 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

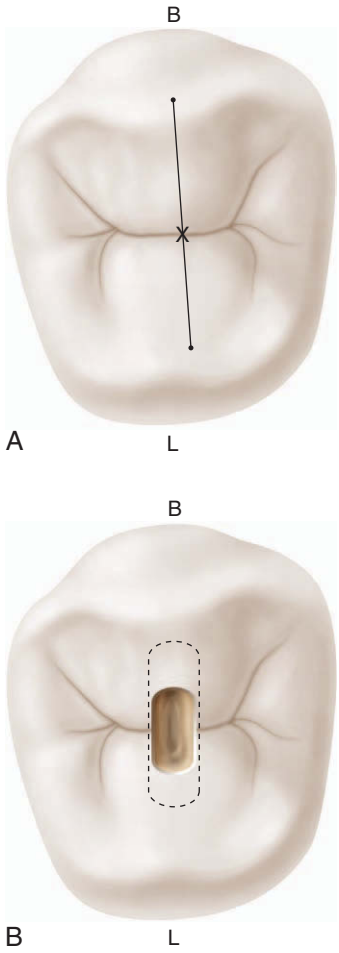


FIG. 7-48 A, Starting location for access to the maxillary premolar (*X*). **B,** Initial outline form (*dark area*) and projected final outline form (*dashed line*). *B,* Buccal; *L,* lingual.

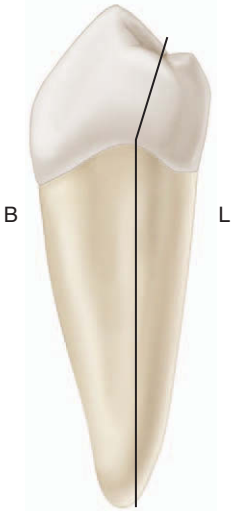


FIG. 7-49 The crown of a mandibular premolar is tilted lingually relative to the root. *B,* Buccal; *L,* lingual.

on the central groove halfway between the mesial and distal boundaries.

Penetration through the enamel into the dentin (approximately 1 mm) is performed with a #2 round bur for premolars and a #4 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 one 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 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 an oval.

PENETRATION OF THE PULP CHAMBER ROOF

Continuing with the same round or tapered fissure bur, the clinician changes the angle of penetration 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(s) 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. 7-52).

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, the clinician should carefully evaluate the angle of penetration before drilling deeper. In multirooted posterior teeth, the clinician must guard against lateral and furcation perforations. Aggressive probing with an endodontic explorer often can help locate the pulp chamber.

COMPLETE ROOF REMOVAL

A round bur, a tapered fissure bur, or a safety-tip diamond or carbide bur is used to remove the roof of the pulp chamber completely, including all pulp horns (Fig. 7-53, *A* and *B*). Visibility problems caused by vital pulp hemorrhage should be handled as described in the previous section for anterior teeth (see Anterior Access Cavity Preparations). The goal is to funnel the corners of the access cavity directly into the orifices, and a safety-tip diamond or carbide bur performs this task nicely; it can be set on the pulp floor and the entire axial wall shaped at one time with little chance of gouging (Fig. 7-54). The safety-tip diamond or carbide bur is 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

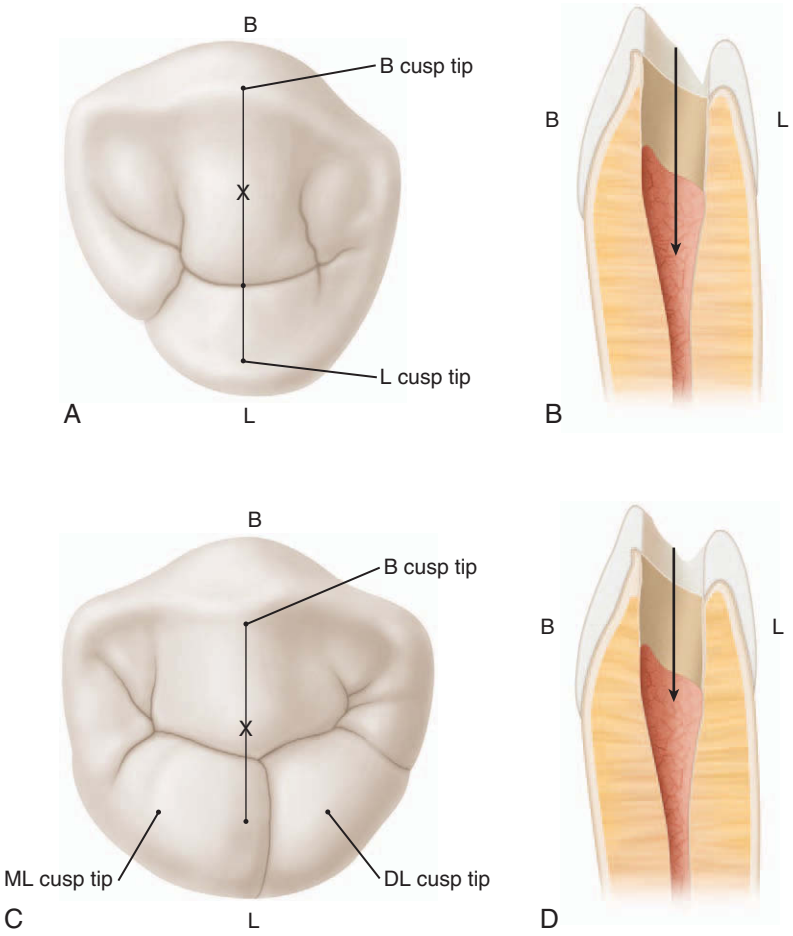


FIG. 7-50 A, Mandibular first premolar and access starting location (*X*) (occlusal view). **B,** Mandibular first premolar and starting location (proximal view). **C,** Mandibular second premolar and access starting location (*X*) (occlusal view). **D,** Mandibular second premolar and starting location (proximal view). *B*, Buccal; *DL*, distolingual; *L*, lingual; *ML*, mesiolingual.

are located at the corners of the final preparation to facilitate the shaping and cleaning process. 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. 7-55), 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 anterior teeth the lingual shoulder is the internal anatomic structure that must be removed as an impediment to straight-line access. In posterior teeth the internal impediments are the cervical dentin bulges and the natural coronal canal constriction. The cervical bulges are shelves of dentin that frequently overhang orifices in posterior teeth, restricting access into root canals and accentuating existing canal curvatures. These bulges can be removed with safety-tip diamond or carbide burs or Gates-Glidden burs. The instruments should be placed at the orifice level and leaned toward the dentin bulge to remove the overhanging shelf (see Fig. 7-53, *C* and *D*).

After the shelf has been removed, the orifice and constricted coronal portion of the canal can be flared with Gates-Glidden burs, which are used in a sweeping upward motion with lateral pressure away from the furcation. An alternative method is to use a #.10 or #.12 tapered engine-driven nickel–titanium file to establish the upper canal shape. As the orifice is enlarged, it should be tapered and blended into the axial wall so that the clinician can slide from the corner of the external outline form down the axial wall into the orifice without encountering any obstructions (Fig. 7-56).

STRAIGHT-LINE ACCESS DETERMINATION

As with anterior teeth, straight-line access is paramount to successful shaping. Files must have unimpeded access to the apical foramen or the first point of canal curvature to perform properly during shaping and cleaning. The clinician must assess each canal for straight-line access and make all adjustments necessary to achieve this goal (see Fig. 7-56, *O*).

VISUAL INSPECTION OF THE PULP CHAMBER FLOOR

Please refer to the earlier section under Anterior Access Cavity Preparations; and Fig. 7-57.

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 root canal therapy is a crown or onlay.

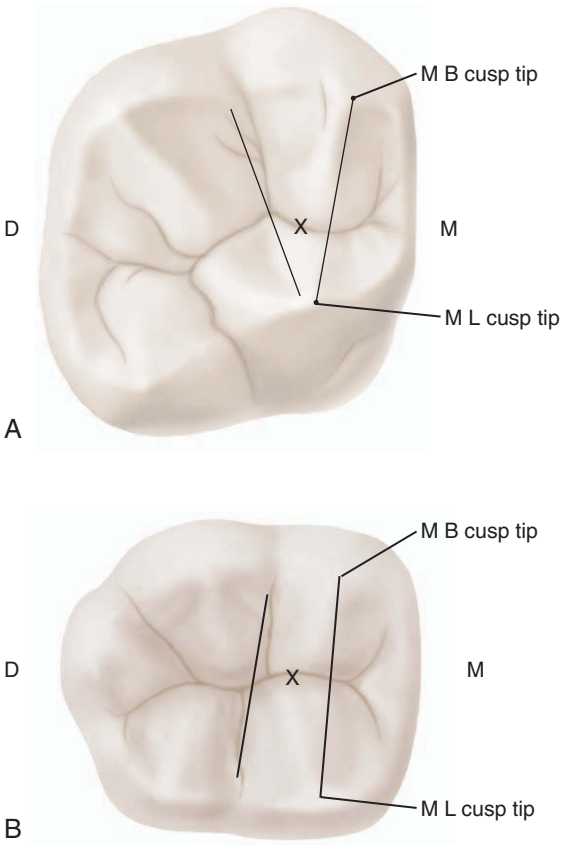


FIG. 7-51 A, Mesial and distal boundary of a maxillary molar showing the access starting location (*X*). **B,** Mesial and distal boundary of a mandibular molar showing the access starting location (*X*). *D*, Distal; *M*, mesial; *MB*, mesiobuccal; *ML*, mesiolingual.

INDIVIDUAL POSTERIOR TEETH

Please see the figures in the section Morphology and Access Cavity Preparations for Individual Teeth, later in the chapter.

CHALLENGING ACCESS PREPARATIONS

Teeth With Minimal or No Clinical Crown

Several factors can cause the loss of a significant portion of a tooth's clinical crown. Caries left untreated can cause loss of coronal tooth structure. Badly decayed teeth typically can fracture under occlusal function because of the undermined and unsupported remaining tooth structure. Similarly, teeth that

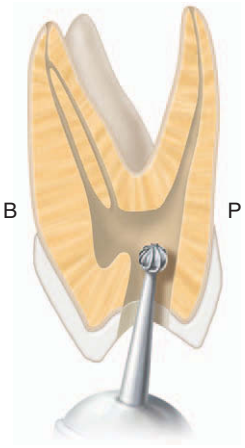


FIG. 7-52 Angle of penetration toward the largest canal (palatal) in a maxillary molar. *B*, Buccal; *P*, palatal.

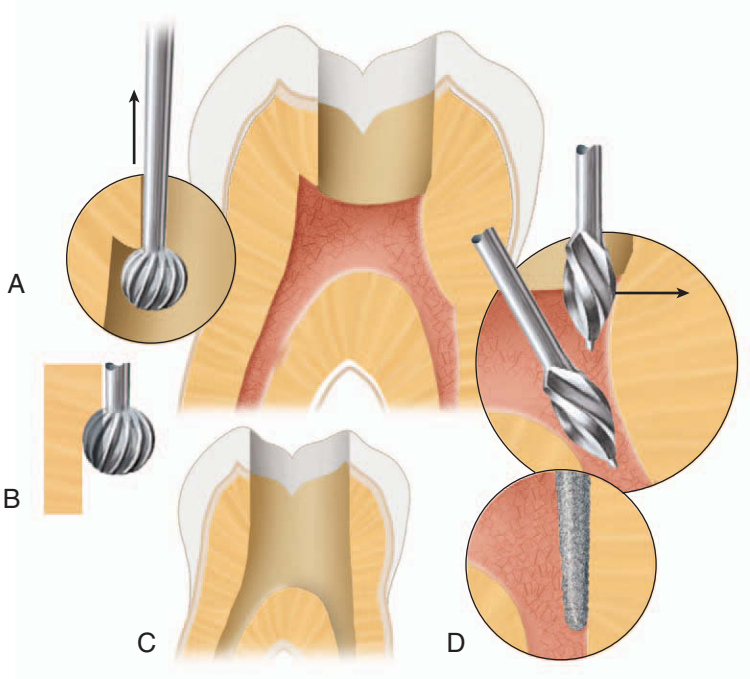


FIG. 7-53 A, Pulp roof/pulp horn removal. The round bur hooks under the lip of the pulp horn. **B,** The bur is rotated and withdrawn in an occlusal direction to remove the lip. **C,** Removal of a cervical dentin bulge. A Gates-Glidden bur is placed just apical to the orifice and withdrawn in a distoocclusal direction. **D,** A safety-tip tapered diamond bur is used to blend and funnel the axial wall from the cavosurface margin to the orifice.

have been heavily restored with amalgam, composite resin, or glass ionomer restorative materials can have minimal coronal tooth structure. These restorative materials provide no extracoronal support for the tooth, and the fillings can fall out during occlusal function, leaving little or no clinical crown remaining. External trauma can cause the clinical crown to fracture, sometimes shearing off to the free gingival margin.

Creating an access cavity on a tooth with little or no clinical crown might seem to be a simple procedure. In young teeth, traumatic fractures often expose the pulp chamber, making preparation easy. However, in older teeth that have had caries or large restorations, the pulp chambers typically have receded or calcified. Loss of significant coronal anatomy to guide penetration angles can make access quite difficult.

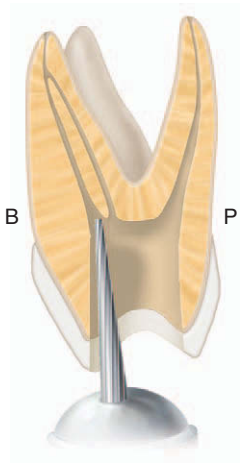


FIG. 7-54 Safety-tip carbide bur is used to shape the axial wall in one plane from the orifice to the cavosurface margin. *B,* Buccal; *P,* palatal.

Before beginning an access cavity on these teeth, the clinician should study their root angulation on pretreatment radiographs and examine the cervical crown anatomy with an explorer (Fig. 7-58). 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 is attempted (Fig. 7-59). Because the external root anatomy is formed by odontoblasts in the pulp, by visualizing the root anatomy both radiographically and clinically, the clinician should have a good idea of access penetration angles. Every effort is made to stay centered within the root for the best chance of locating the pulp canal.[25] The depth of penetration needed to reach the pulp canal is measured on a pretreatment radiograph. If the clinician reaches this depth 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 clinician can redirect the penetration angle if necessary and move the preparation apically. As soon as the pulp canal is identified, the dental dam must be placed and the access preparation finalized, using the guidelines discussed earlier in this chapter.

Heavily Restored Teeth (Including Those With Full Veneer Crowns)

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.

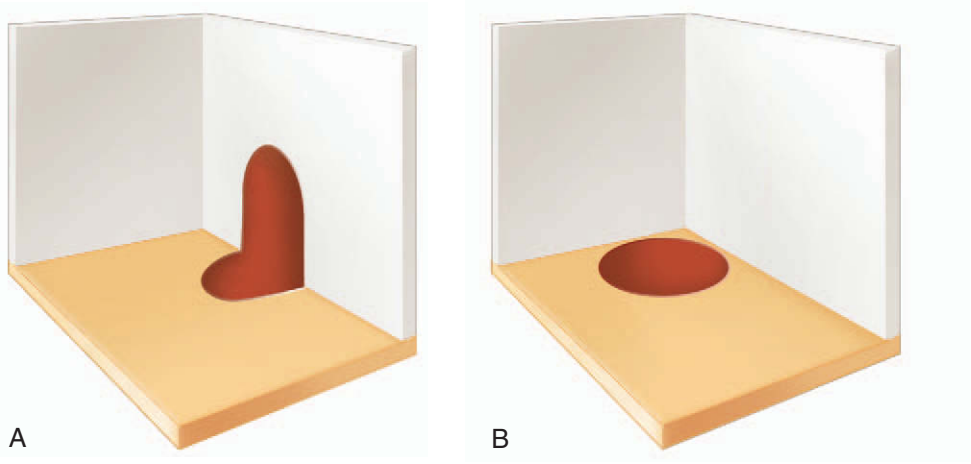
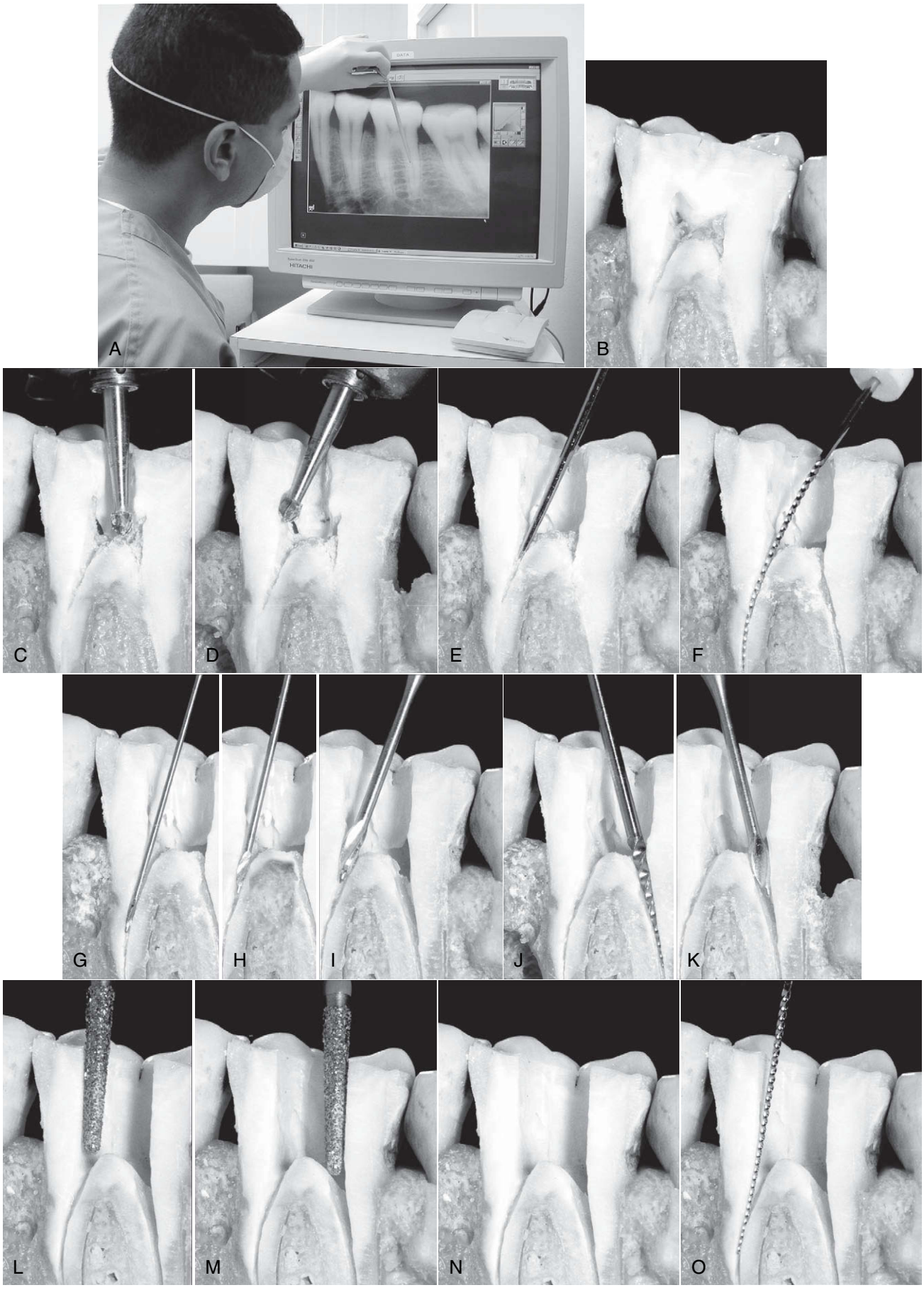


FIG. 7-55 A, Mouse hole effect caused by extension of the orifice into the axial wall. **B,** Orifice that lies completely on the pulp floor.

FIG. 7-56 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-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.


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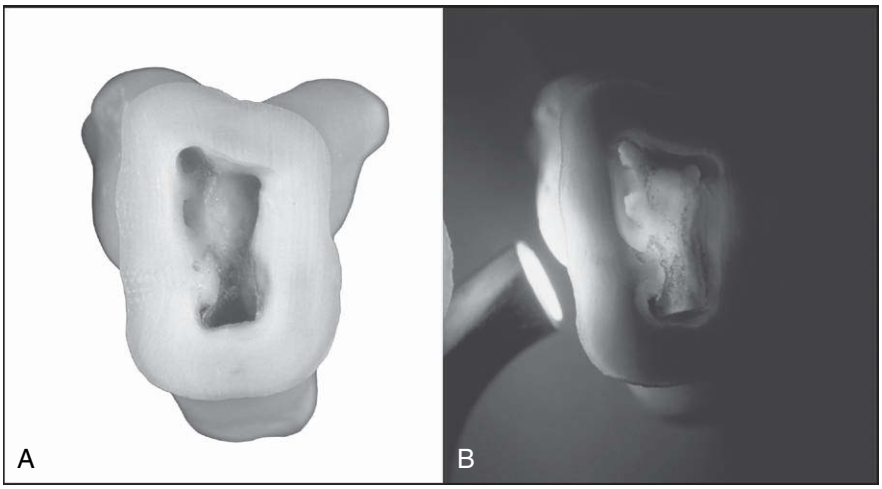


FIG. 7-57 A fiberoptic light can be applied to the cervical aspect of the crown to help obtain maximal visibility with magnification. Transillumination often reveals landmarks otherwise invisible to the unaided eye.



FIG. 7-58 The cervical area of the tooth is explored before access is started.

The crown-to-root angulation often is altered when large restorations or crowns correct occlusal discrepancies (Fig. 7-60). 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.[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. 7-57).

In most cases, complete removal of large restorations is the wisest course (Fig. 7-61). These restorations often have leaky, defective margins or recurrent caries or both. Removing the restoration allows the clinician better visibility of the internal anatomic structures through direct visualization and increased light penetration. With increased visibility the clinician can check for recurrent caries and fracture lines on the pulp chamber walls or floor. As stated earlier in a study,[1] clinicians are 40% more likely not to detect these anomalies when restorations are not removed completely. 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. Another reason to remove these restoration remnants completely is to prevent pieces of the restorative material from falling into the root canal. Instruments can rub against restoration fragments during shaping and cleaning, creating filings that can be carried into the canal system (see Fig. 7-74, D). Complete 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(s). For example, class V restorations often cause calcification of the coronal canal, making location of the canal through the occlusal approach quite difficult. Removal of the class V restoration allows more direct access to the calcified canal, which makes location and treatment much easier. Any remaining canals can be treated through the conventional occlusal access cavity (Fig. 7-62).

When an extensive restoration is a full or partial veneer crown, the restoration must be evaluated thoroughly. If any concerns arise about recurrent decay or leaky margins, the crown should be removed before the access cavity is prepared. Removal of the crown allows elimination of all recurrent caries and improves the visibility of the pulp spaces.

Creation of an access through an intact full or partial veneer crown should be done with caution. When such restorations are placed, they often change the crown-to-root angulation to correct preexisting occlusal discrepancies. Full veneer crowns also can alter 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 often masks the underlying pulp chamber. In these situations the clinician's 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 aides in this process.

Metal veneer 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 ceramometal restorations must be handled delicately to minimize the potential for fracture. The clinician should use a round diamond bur and copious water spray to penetrate the porcelain. After porcelain penetration, a

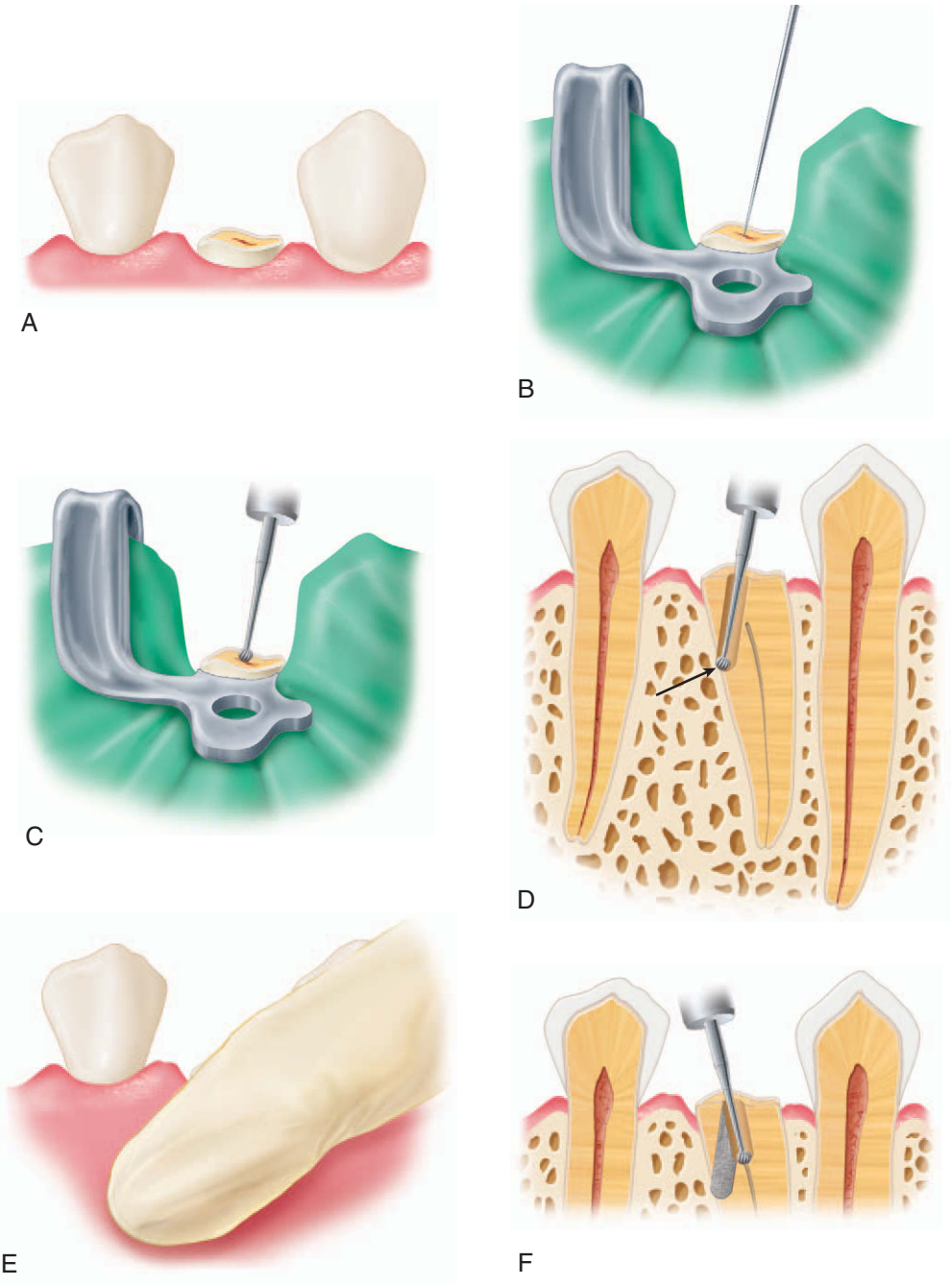


FIG. 7-59 Access cavity preparation when the anatomic crown is missing. **A,** A mandibular first premolar with the crown missing. **B,** An endodontic explorer fails to penetrate the calcified pulp chamber. **C,** A long-shank round bur is directed in the assumed long axis of the root. **D,** Perforation of the root wall (*arrow*), resulting from the clinician's 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; DENTSPLY Tulsa Dental Specialties, Tulsa, OK). The dental dam is placed as soon as the canal is identified.

transmetal bur and copious water spray should be used to penetrate the metal coping; the water spray minimizes heat buildup, which could fracture the porcelain (Fig. 7-63).

Many clinicians tend to be too conservative when preparing an access cavity through a veneer crown. An attempt 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 clinician should search the margins and internal spaces for caries, leakage, and fractures. If no problems are discovered, the clinician may proceed with shaping and cleaning, leaving the full veneer crown in place.

Teeth With Calcified Canals

A pretreatment radiograph (Figs. 7-64 and 7-65) often appears to reveal total or nearly total calcification of the pulp chamber

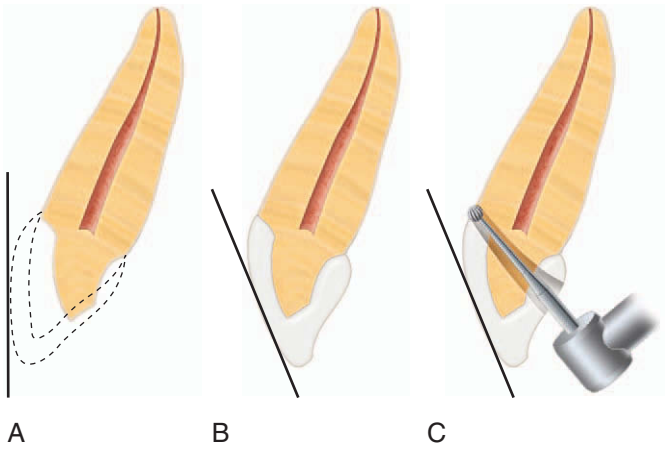


FIG. 7-60 Access cavity error resulting from alteration of the original tooth contours by a full veneer crown. **A,** Original crown contour of the tooth. **B,** A full veneer crown is used to change the original crown contour for esthetic purposes. **C,** Access perforation resulting from reliance on the full veneer crown contour rather than the long axis of the root.

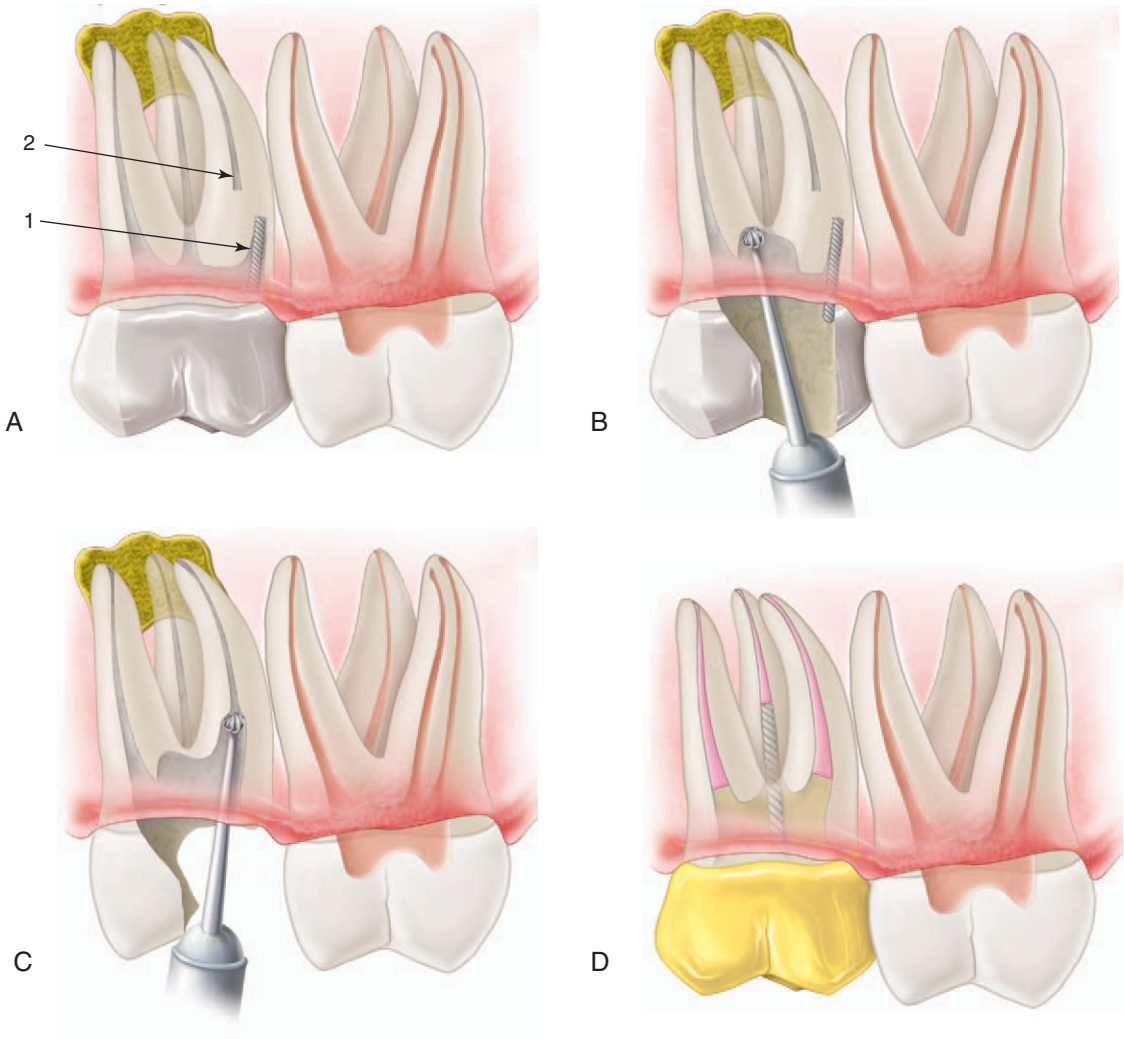


FIG. 7-61 A, In a heavily restored maxillary second molar that requires root canal therapy, the clinician may attempt access to the canals. Pretreatment radiographs demonstrate three important factors: (1) a reinforcing pin is in place *(arrow)*; (2) at least two thirds of the coronal portion is restorative 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.

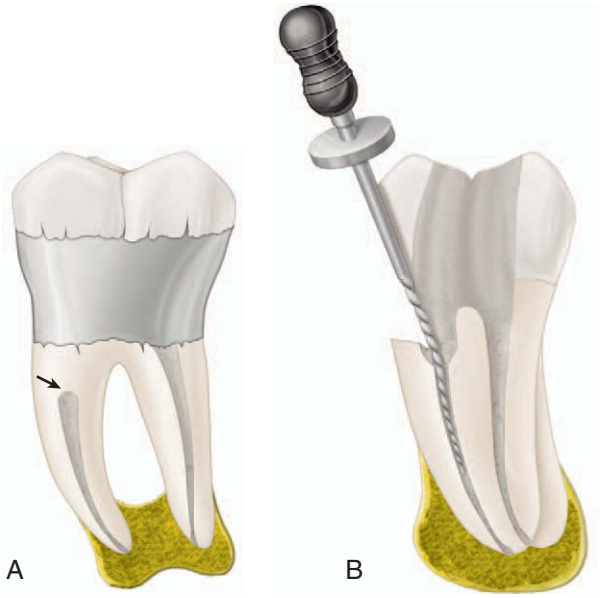


FIG. 7-62 A, Extensive class V restoration necessitated by root caries and periodontal disease that led to canal calcification *(arrow)*. **B,** Access to the canal is occluded by calcification. Removal of the facial restoration may be required to obtain access from the buccal surface.

and radicular canal spaces. 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 narrowing of the root canal system. Canals become less calcified as they approach the root apex. Despite severe coronal calcifications, the clinician must assume that all canals exist and must be shaped, cleaned, and obturated to the canal terminus.

Teeth with severe pulp calcification may present problems with locating and negotiating root canals. The use of magnification and transillumination, as well as careful examination of color changes and pulp chamber shapes, can help the clinician safely locate canals. The clinician should search for root canal orifices only after completely preparing the pulp chamber and cleaning and drying its floor (95% denatured ethanol is 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. The clinician must be aware of these color differences when searching for calcified orifices and must keep in mind that root canal orifices are located at the angles formed by the floor and walls and at the end points of developmental grooves. Additional aids for locating calcified root canals

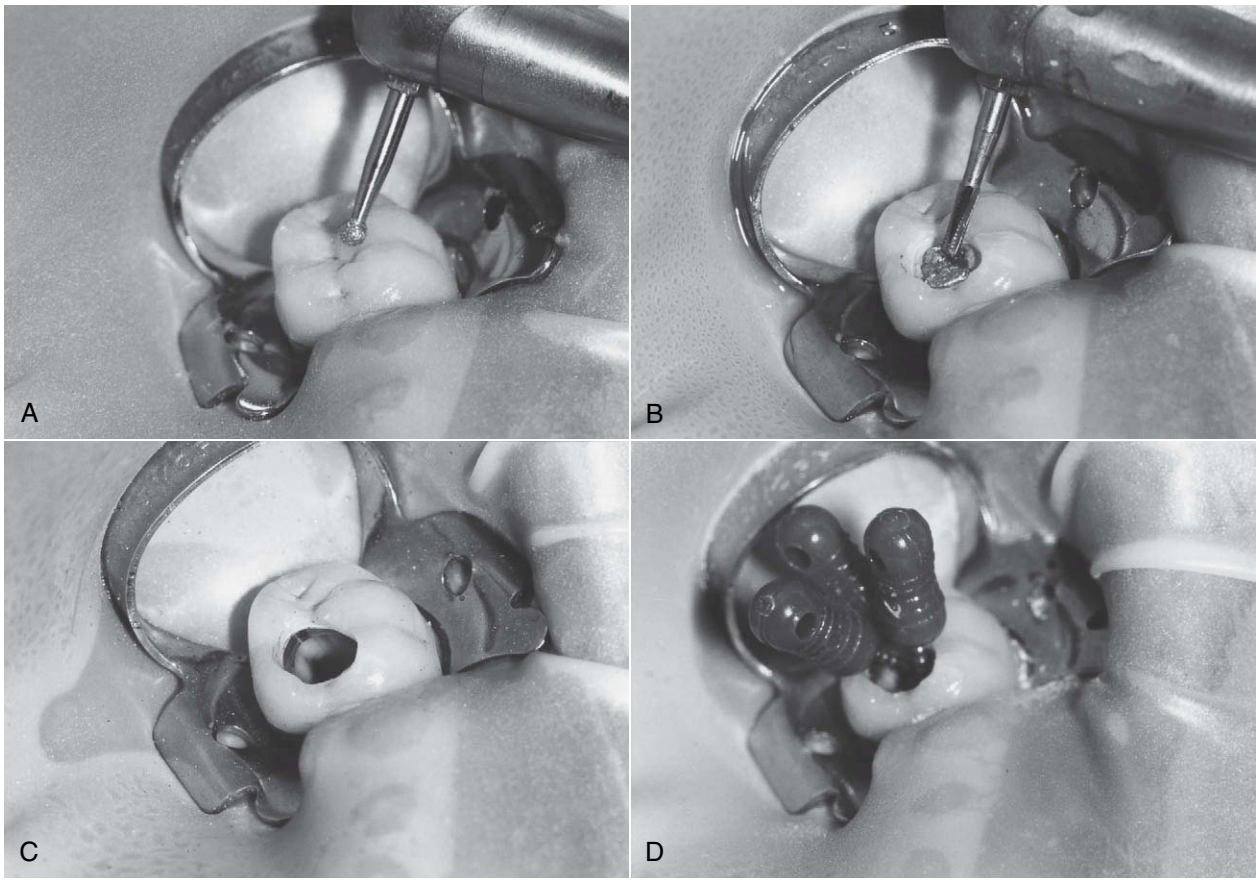


FIG. 7-63 Access cavity preparation through a ceramometal crown. **A,** A round diamond bur is used to penetrate the porcelain. **B,** After the access outline has been made with the round diamond bur, a transmetal bur is used to cut through the metal. **C,** Prepared access cavity, which allows a direct approach to the canals. **D,** Test files can be placed on the access cavity walls without impingement.



FIG. 7-64 Radiograph taken when a patient's symptoms (i.e., nonlingering cold sensitivity and sensitivity to sweets) first appeared. The mandibular first molar was not treated endodontically because tests showed it to be vital. Caries was removed from under the mesial amalgam, and calcium hydroxide was placed over the cavity proximal to the pulp space.



FIG. 7-65 Radiograph of the mandibular first molar in Fig. 7-64 taken 13 years later shows severe calcification of the pulp chamber and periradicular radiolucencies.

include staining the pulp chamber floor with 1% methylene blue dye, performing the sodium hypochlorite "champagne bubble" test (see Fig. 7-2) and searching for canal bleeding points.

In severely calcified teeth, calcified dentin must be removed slowly down the root. The clinician can use long, thin ultrasonic tips under the high magnification of a DOM to avoid removing too much tooth structure. As the search moves apically, two radiographs must be taken, 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 calcified canals is a challenge. When the canal is located, a small K-file (#6, #8, or #10) coated with a chelating agent should be introduced into the canal to determine patency. This file should not be removed until some canal enlargement has occurred. It 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

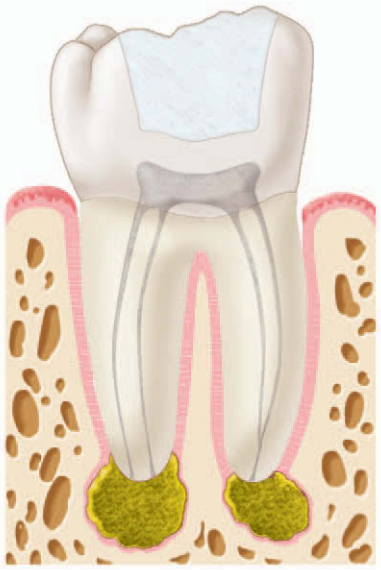


FIG. 7-66 Mandibular first molar with a class I restoration, calcified canals, and periradicular radiolucencies. Presumably pulp exposure has occurred, resulting in calcification and ultimately necrosis of the pulp tissue.

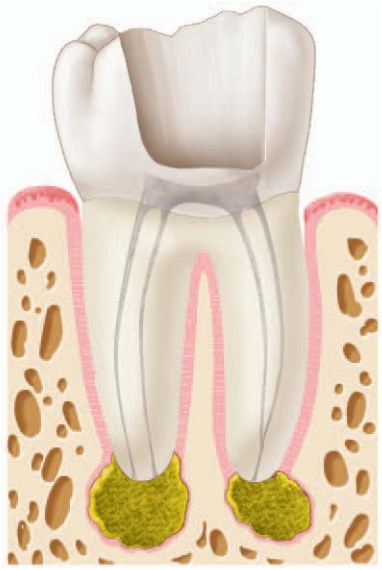


FIG. 7-67 Excavation of a restoration and base material. The clinician should extend the cavity preparation toward the assumed location of the pulp chamber, keeping in mind that pulp chambers are located in the center of the tooth at the level of the cementoenamel junction (*CEJ*).

the furcation. This safely enlarges the coronal canal and moves it laterally to avoid the furcation. It also creates a path of insertion for larger files and for preflaring burs. Figures 7-64 through 7-71 illustrate several methods that can be used to locate calcified spaces. For the most successful results, the sequence should be followed as shown.

The wise clinician stops excavating dentin if a canal orifice cannot be found to avoid weakening the tooth structure.

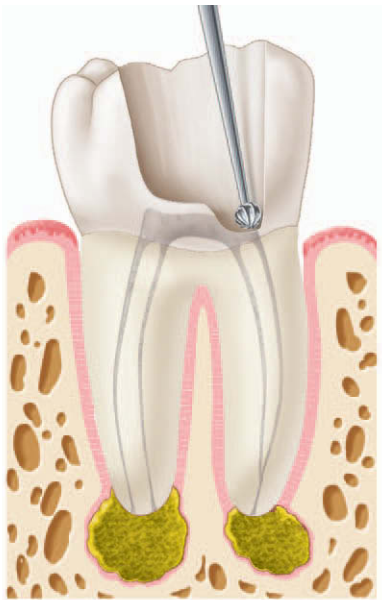


FIG. 7-68 The clinician uses a long-shank #2 or #4 round bur to remove dentin, attempting to locate calcified canals by following Krasner and Rankow's anatomic laws.

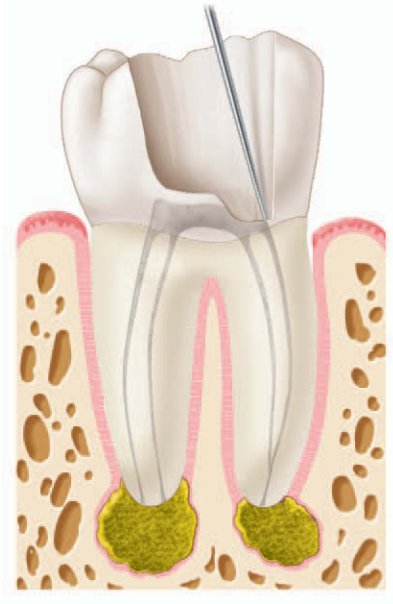


FIG. 7-69 An endodontic explorer is used to probe the pulp floor. A straight ultrasonic tip may be used to remove dentin. Angled radiographs must be taken to monitor progress.

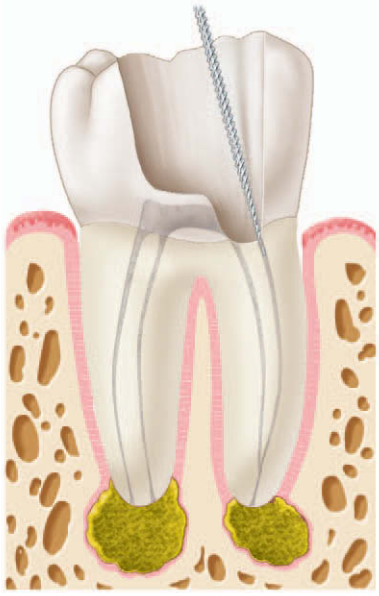


FIG. 7-70 At the first indication of a canal space, the smallest instrument (i.e., a #.06 or #.08 file) should be introduced into the canal. Gentle passive movement, both apical and rotational, often produces some penetration. A slight pull, signaling resistance, usually is an indication that the canal has been located. This should be confirmed by radiographs.

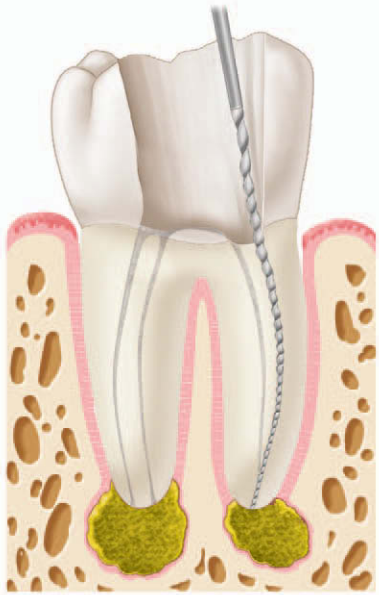


FIG. 7-71 A small hand K-file negotiates the canal to its terminus. An apex locator or radiograph is used to confirm the file's position.

Serious errors can arise from overzealous or inappropriate attempts to locate canals. Root wall or furcation perforations can occur even with the most careful search. In such cases the communication with the periodontal tissues must be repaired immediately (Fig. 7-72). Retrograde surgical procedures may be a more conservative approach to treating the tooth when compared with perforations or root fractures. No rapid technique exists for dealing with calcified root canals. Painstaking removal of small amounts of dentin with the aid of the DOM and radiographic confirmation has proved to be the safest approach.

Crowded Teeth

Conventional access preparations may not be possible in patients with crowded teeth. The decision regarding an alternative approach must be based on straight-line access

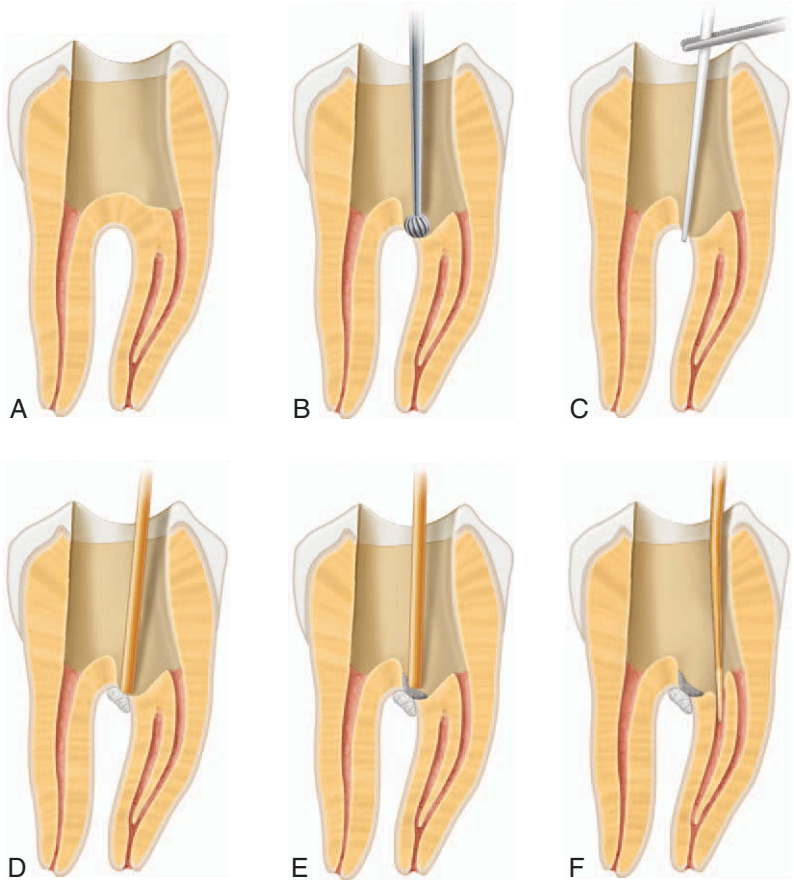


FIG. 7-72 Perforation repair. **A,** Access is gained into two canals, but the calcified canal orifice has not been located. **B,** Minute furcal perforation noted during the search for the canal. **C,** Absorbent points are used to control hemorrhage. **D,** CollaCote (Zimmer Dental, Carlsbad, CA) is introduced to provide a base for repair material. **E,** MTA (DENTSPLY Tulsa Dental, Tulsa, OK) is introduced. **F,** Location of the calcified canal; enhanced vision, including the dental operating microscope, is recommended.

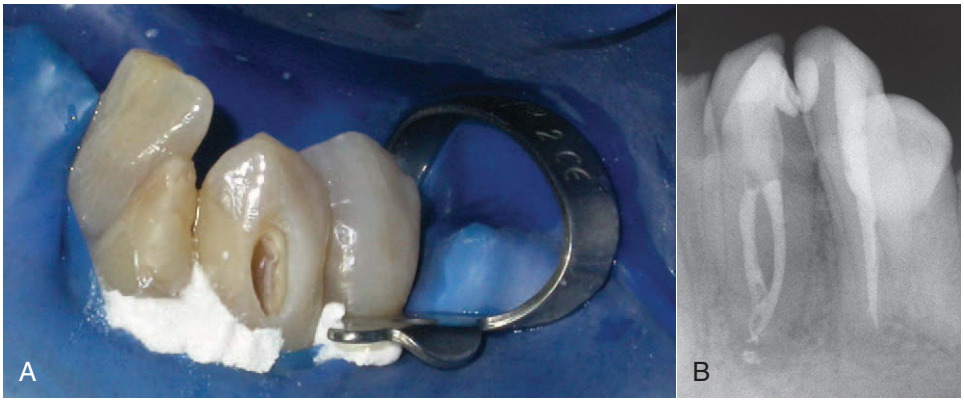


FIG. 7-73 A, Access cavity on crowded mandibular teeth. The access preparation is performed through the buccal surface on tooth #22. Tooth #23 also has been accessed through the buccal surface; root canal therapy was performed, and the access cavity was permanently restored with composite. **B,** Obturation.

principles and conservation of tooth structure. In certain circumstances a buccal access preparation may be the treatment of choice (Fig. 7-73). Modern restorative materials have made repair of this access esthetically acceptable.

Rotated Teeth

Rotated teeth can present problems for the clinician during access cavity preparation because of the altered crown-to-root relationships. According to one investigator,[141] diagnostic periapical radiographs, although only two dimensional, 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, the clinician should visualize likely access variations before entering the tooth. 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 also can occur when tooth angulations are not considered during preparation of an access cavity. Such problems include the following:

- 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 debride all pulp tissue from the chamber

The best way to handle these problems is to prevent them from occurring. A thorough radiographic examination is crucial. The initial outline form occasionally can be created without the dental dam; this 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. 7-74 through 7-76.

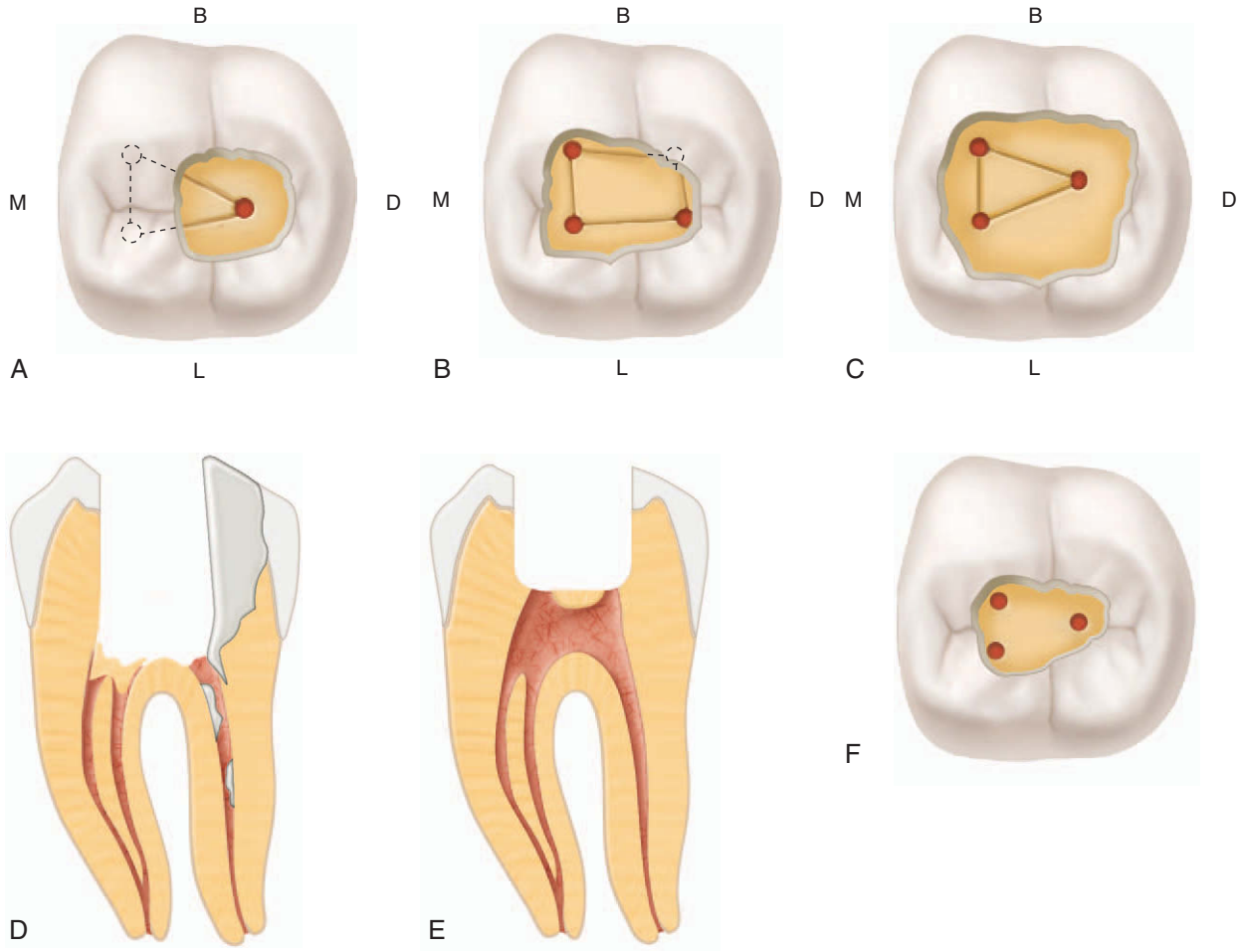


FIG. 7-74 A, Poor access placement and inadequate mesial extension leave both mesial orifices uncovered. Information about the position and location of pulp chambers can be obtained through evaluation of pretreatment radiographs, especially bite-wing radiographs, and assessment of the tooth anatomy at 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.

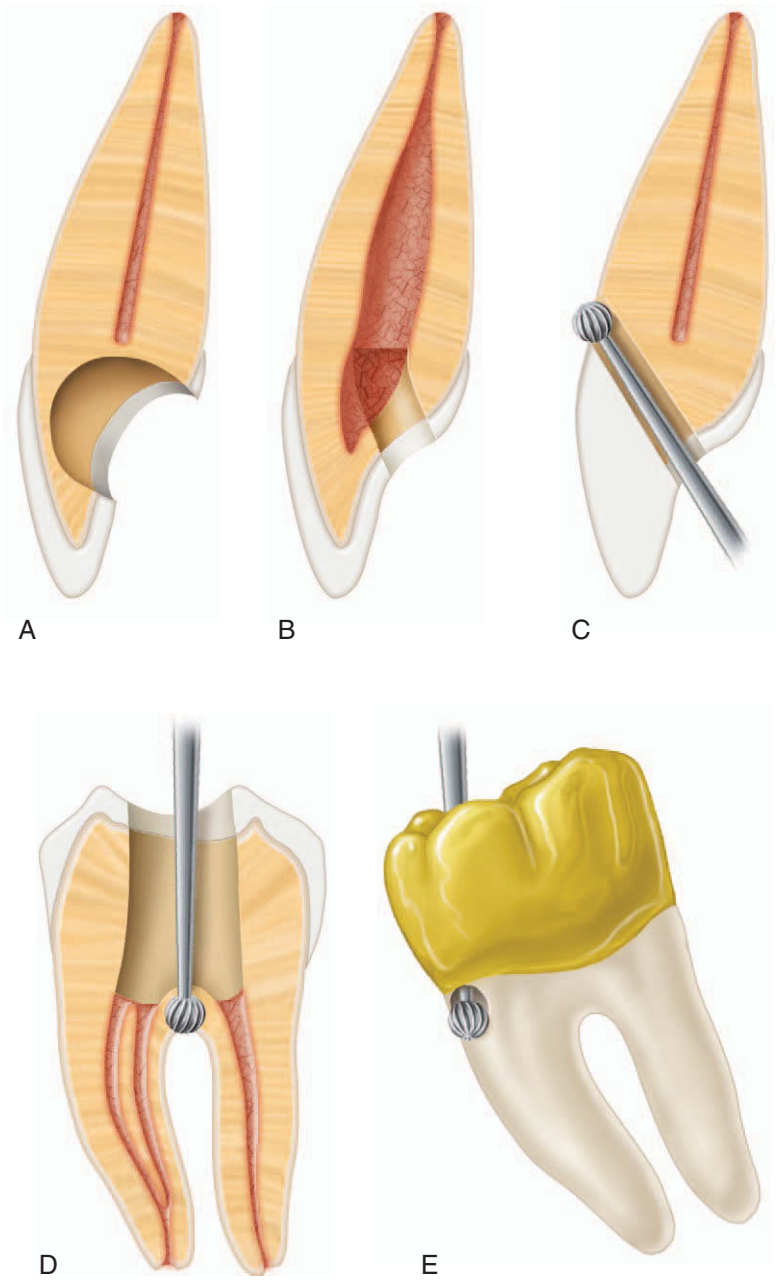


FIG. 7-75 A, Overzealous tooth removal caused by improper bur angulation and failure to recognize the lingual inclination of the tooth. This results in weakening and mutilation of the coronal tooth structure, which often leads to coronal fractures. **B,** Inadequate opening; the access cavity is positioned 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. They must be repaired (see Fig. 7-72) as soon as they are made for a satisfactory result. **E,** Perforation of the mesial tooth surface caused by failure to recognize that the tooth is 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.

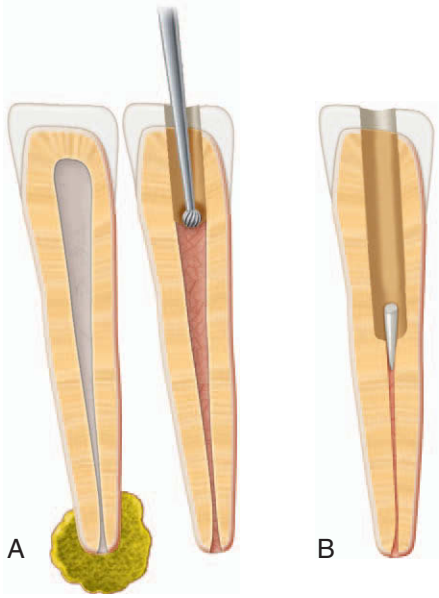


FIG. 7-76 A, The most embarrassing error, with the greatest potential for medical and legal damage, is entering the wrong tooth because of incorrect dental dam placement. When the crowns of teeth appear identical, the clinician should mark the tooth with a felt-tip marker before the dental dam is placed. **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.

MORPHOLOGY 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 3-D 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 3-D reconstructions and visualization. The following individuals made this project possible:

Tomographic scan: Courtesy Michael J. Flynn, PhD, Director, X-Ray Imaging Research Laboratory, Henry Ford Health Sciences, Detroit, MI; also professor (adjunct) of nuclear engineering and radiological science, University of Michigan, Ann Arbor, MI

3-D reconstructions and visualizations: Courtesy Kevin Montgomery, PhD, technical director, Stanford-NASA National Biocomputation Center, Palo Alto, CA

Facilitator: Dr. Paul Brown

Radiographs: Courtesy Dr. L. Stephen Buchanan, Santa Barbara, CA; Dr. John Khademi, Durango, CO; Dr. Raed S. Kasem, Clearwater, FL; Dr. Gary Manasse, Jacksonville, FL; Dr. Michael DeGrood, DeBary, FL; and Dr. Kevin Melker, Clearwater, FL

Access cavity illustrations: Designed and formatted by Dr. Richard Burns, San Mateo, CA; and Dr. Eric Herbranson, San Leandro, CA

Maxillary Central Incisor

The root canal system outline of the maxillary central incisor reflects the external surface outline. 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. Multiple canals are rare (Table 7-8), but lateral canals are common.

The external access outline form for the maxillary central incisor is a rounded triangle with its base toward the incisal aspect (Figs. 7-77 through 7-81). 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.

Continued

Maxillary Central Incisor—cont'd

TABLE 7-8

Studies of Apical Canal Configurations for the Maxillary Central Incisor

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)	Four Canals (%)
Vertucci[215]	Clearing	USA	100	—	—
Pineda and Kuttler[163]	Radiographic	Mexico	100	—	—
Caliskan et al[33]	Clearing	Turkey	100	—	—
Kasahara et al[102]	Clearing	Japan	—	—	—
Mangani and Ruddle[126]	Clinical	USA	—	—	Case report
Todd[209]	Clinical	USA	—	Case report	—
Genovese and Marsico[73]	Clinical	Italy	—	Case report	—
Sinai and Lustbader[188]	Clinical	USA	—	Case report	—
Von der Vyver and Traub[221]	Clinical	South Africa	—	Case report	—
Cabo-Valle and Gonzalez-Gonzalez[31]	Clinical	Spain	—	Case report	—
Mader and Konzelman[124]	Clinical	USA	—	Case report	—
Lambruschini and Camps[111]	Clinical	France	—	Case report	—
Gonzalez-Plata and Gonzalez-Plata[77]	Clinical	Mexico	—	Case report	—
Patterson[152]	Clinical	USA	—	Case report	—
Heling[86]	Clinical	Israel	—	Case report	—
Al-Nazhan[6]	Clinical	Saudi Arabia	—	Case report	—
Lin et al[117]	Clinical	China	—	Case report	—
Sponchiado et al[196]	Clinical	Brazil	—	Case report	—
Sert and Bayirli[184]	Clearing (men)	Turkey	98 (98)	2 (2)	—
	Clearing (women)	Turkey	100 (98)	2 (2)	—

*Number in parentheses represents the percentage of cases with two canals joining into one canal.

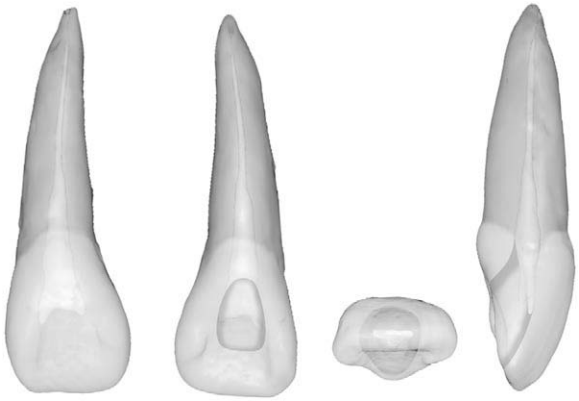


FIG. 7-77 Maxillary central incisor. Development and anatomic data: average time of eruption, 7 to 8 years; average age of calcification, 10 years; average length, 22.5 mm. Root curvature (most common to least common): straight, labial, distal.

Maxillary Central Incisor—cont'd

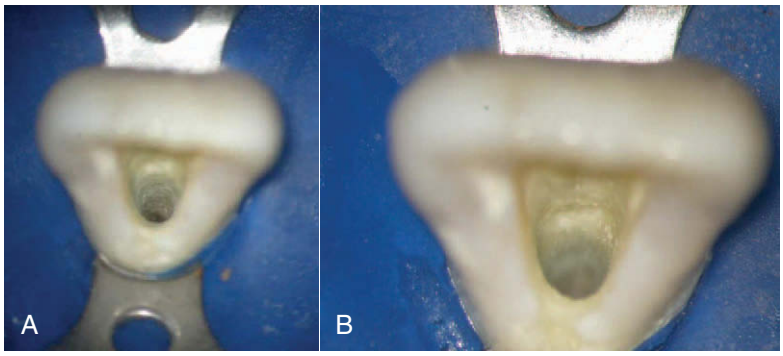


FIG. 7-78 Access cavity for a maxillary central incisor as viewed through the dental operating microscope. **A,** ×3.4 magnification. **B,** ×8.4 magnification.



FIG. 7-79 Curved accessory canal with an intersecting straight lateral canal.

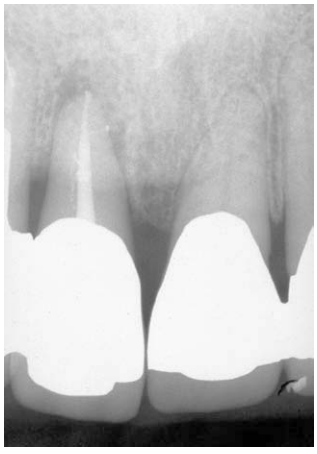


FIG. 7-80 Parallel accessory canal to main canal with a simple lateral canal.

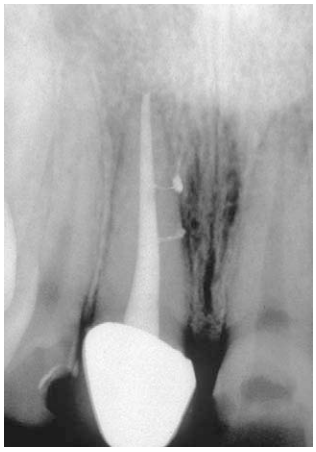


FIG. 7-81 Double lateral canals.

Maxillary Lateral Incisor

The pulp chamber outline of the maxillary lateral incisor is similar to that of the maxillary central incisor; however, it is smaller, and two or no pulp horns may be present. 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. The clinician must know the anatomy of the pulp chamber before starting the access cavity. 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. Normally only one root canal is present, but two and three canals have been reported (Table 7-9).

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. 7-82 through 7-85). 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 lingual radicular or developmental groove.[119,149-151,154-157] Although this groove may be present on the roots of all anterior teeth, it is more common in the maxillary lateral. The groove can lose its periodontal attachment as a result of both periodontal and endodontic etiologies. There is generally direct communication between the groove and the pulp cavity and this occurs primarily through dentinal tubules.

Dens invaginatus is another anomaly and this has been classified into three types based on severity,[188] from simple to more complex. Type 1 is an invagination that is confined to the crown. Type 2 is an invagination that extends past the cementoenamel junction but does not involve the periradicular tissues. Type 3 is an invagination that extends beyond the cementoenamel junction and can have a second apical foramen. Often surgical and orthograde root canal therapy is necessary to treat this condition.*

TABLE 7-9

Studies of Apical Canal Configurations for the Maxillary Lateral Incisor

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)
Vertucci[215]	Clearing	USA	100	—	—
Pineda and Kuttler[163]	Radiographic	Mexico	100	—	—
Caliskan et al[33]	Clearing	Turkey	95.1	4.9	—
Pecora et al[158]	Clinical	Brazil	—	Case report	—
Thompson et al[207]	Clinical	USA	—	Case report (1)	—
Fabra-Campos[62]	Clinical	Spain	—	Case report	—
Christie et al[40]	Clinical	Canada	—	Case report	—
Walvekar and Behbehani[227]	Clinical	Kuwait	—	—	Case report
Collins[44]	Clinical	Australia	—	Case report	—
Sert and Bayirli[184]	Clearing (men)	Turkey	97 (6)	(3)	—
	Clearing (women)	Turkey	100 (10)	—	—

*Numbers in parentheses represent 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.

Maxillary Lateral Incisor—cont'd

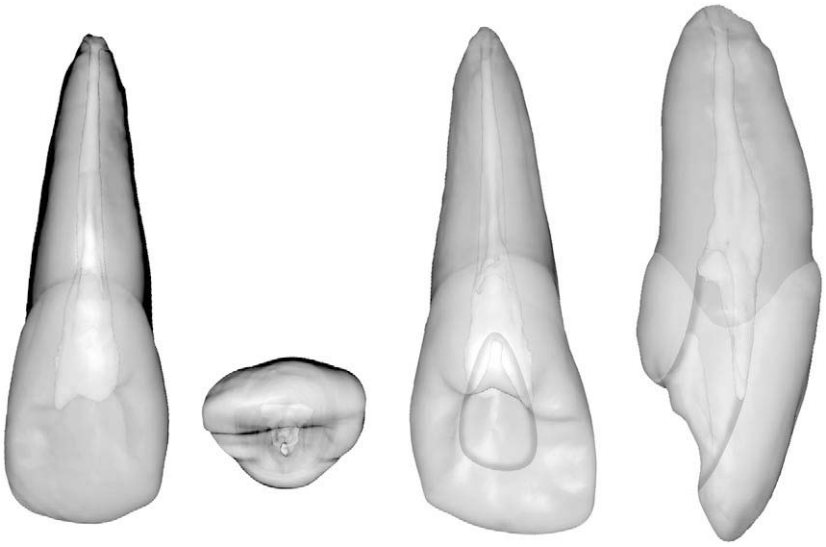


FIG. 7-82 Maxillary lateral incisor. Development and anatomic data: average time of eruption, 8 to 9 years; average age of calcification, 11 years; average length, 22 mm. Root curvature (most common to least common): distal, straight.

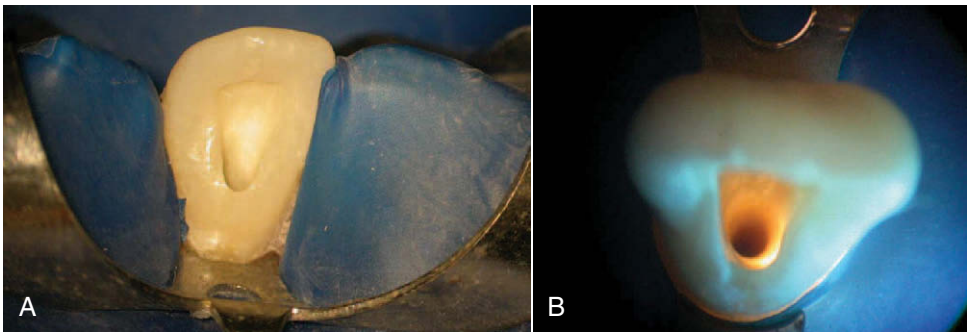


FIG. 7-83 Access cavity for a maxillary lateral incisor as viewed through the dental operating microscope. **A,** ×3.4 magnification. **B,** ×5.1 magnification with cervical fiberoptic transillumination.



FIG. 7-84 Lateral incisor with a canal loop and multiple lateral canals with associated lesions.



FIG. 7-85 Multiple accessory foramina.

*References 151, 160, 163, 170, 171, 173, 176, 182, 189, 200.

Maxillary Canine

The root canal system of the maxillary canine is similar in many ways to that of the maxillary incisors. 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. Usually one root canal is present, although two canals have been reported (Table 7-10). The thin buccal bone over the canine eminence often disintegrates, and fenestration is an occasional finding. Accurate determination of the length is critical. Another effect of this fenestration is a slight, permanent apical pressure sensitivity that occasionally occurs after root canal therapy. This sensitivity can best be corrected by apical root surgery.

The external access outline form is oval or slot shaped because no mesial or distal pulp horns are present (Figs. 7-86 through 7-89). 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.

TABLE 7-10

Studies of Apical Canal Configurations for the Maxillary Canine

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)
Vertucci[215]	Clearing	USA	100	—
Pineda and Kuttler[163]	Radiographic	Mexico	100	—
Caliskan et al[33]	Clearing	Turkey	97.8	2.2
Sert and Bayirli[184]	Clearing (men)	Turkey	98 (7)	2
	Clearing (women)	Turkey	96	4
Weisman[237]	Clinical	Australia	—	Case report
Alapati et al[3]	Clinical	Kuwait	—	Case report

*Number in parentheses represents the percentage of cases with two canals joining into one canal.

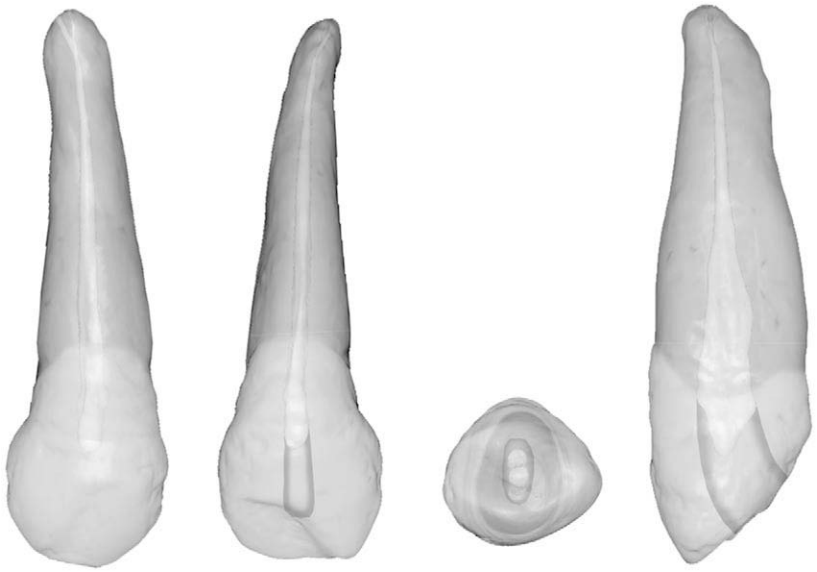


FIG. 7-86 Maxillary canine. Development and anatomic data: average time of eruption, 10 to 12 years; average age of calcification, 13 to 15 years; average length, 26.5 mm. Root curvature (most common to least common): distal, straight, labial.

Maxillary Canine—cont'd

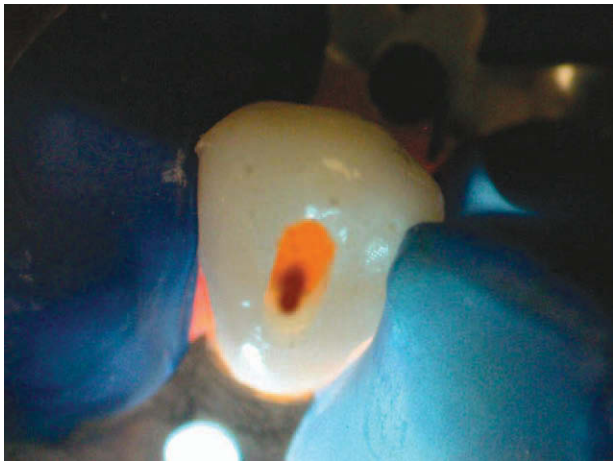


FIG. 7-87 Access cavity for a maxillary canine as viewed through the dental operating microscope. (×5.1 magnification with cervical fiber-optic transillumination.)

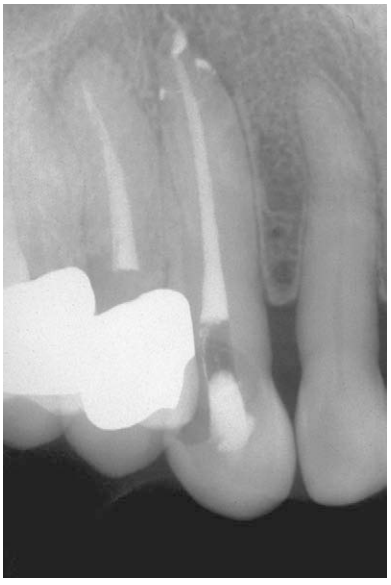


FIG. 7-88 Canine with multiple accessory foramina.

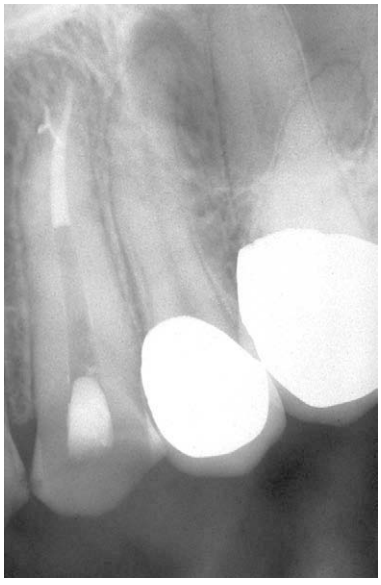


FIG. 7-89 Canine with lateral canal dividing into two canals.

Maxillary First Premolar

The majority of maxillary first premolars have two root canals regardless of the number of roots. Ethnicity plays a factor in that Asian people have a higher incidence of one canal than do other ethnic groups.[118]

A furcation groove or developmental depression on the palatal aspect of the buccal root is another anatomic feature. Its prevalence has been reported as between 62% and 100%.[97,202] This groove may pose a risk to endodontic and prosthodontic treatment of this tooth. 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 palatal pulp horn. The buccal pulp horn usually is larger. From the occlusal level the chamber maintains a similar width to 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 (Table 7-11). If two canals are present, they are labeled buccal and palatal; three root canals are designated mesiobuccal, distobuccal, and palatal. Directional positioning of endodontic pathfinder 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 can fenestrate through the bone, leading to the same problems that arise with canines (i.e., inaccurate apex location, chronic posttreatment sensitivity to palpation over the apex, and an increased risk of an irrigation accident).

The maxillary first premolar is prone to mesiodistal root fractures and fractures at the base of the cusps, particularly the buccal cusp. If a fracture is suspected, all restorations should be removed and the coronal anatomy inspected with fiberoptic light and magnification. Full occlusal coverage is required after root canal therapy to prevent cuspal and/or crown/root fracture.

The access preparation for the maxillary first premolar is oval or slot shaped (Figs. 7-90 through 7-94). 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 not to overextend the preparation in that direction, as 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 (see Fig. 7-91, *D*).

TABLE 7-11

Studies of Apical Canal Configurations for the Maxillary First Premolar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[215]	Clearing	USA	26	69	5
Pineda and Kuttler[163]	Radiographic	Mexico	50.1	49.4	0.5
Caliskan et al[33]	Clearing	Turkey	9.8	90.2	—
Carns and Skidmore[36]	Plastic cast resin	USA	22	72	6
Walker[223]	Radiographic	Southern China	36	64	—
Kartal et al[100]	Clearing	Turkey	9.7	88.6	1.7
Pecora et al[157]	Clearing	Brazil	17.1	80.4	2.5
Soares and Leonardo[194]	Clinical	Brazil	—	—	Case report
Kerekes and Tronstad[105]	Ground sections	Norway	10	80	2
Green[80]	Ground sections	USA	3 (26)	70 (4)	—
Sert and Bayirli[184]	Clearing (men)	Turkey	31 (19)	68 (5)	4 (2)
	Clearing (women)	Turkey	29 (17)	67 (4)	4
Zaatar et al[252]	Clearing	Kuwait	21.5 (10.1)	74.7	3.8

*Numbers in parentheses represent the percentage of cases with two canals joining into one canal.

†Numbers in parentheses represent the percentage of cases with one canal dividing into two canals.

‡Number in parentheses represents the percentage of cases with one canal dividing into three canals.

Maxillary First Premolar—cont'd

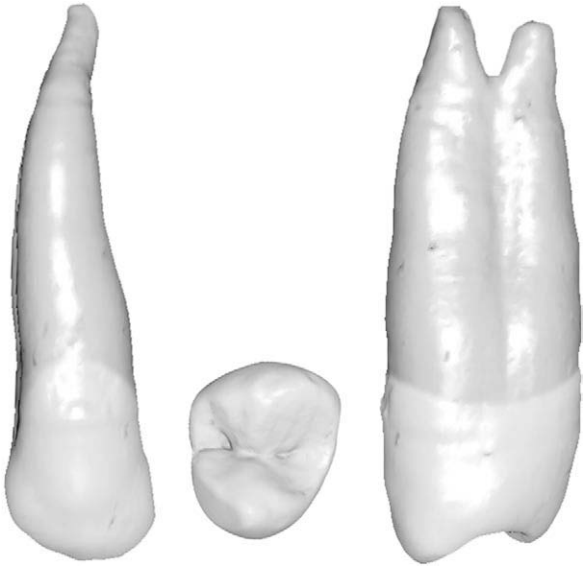


FIG. 7-90 Maxillary first premolar. Development and anatomic data: average time of eruption, 10 to 11 years; average age of calcification, 12 to 13 years; average length, 20.6 mm. Root curvature (most common to least common): buccal root—lingual, straight, buccal; palatal root—straight, buccal, distal; single root—straight, distal, buccal.

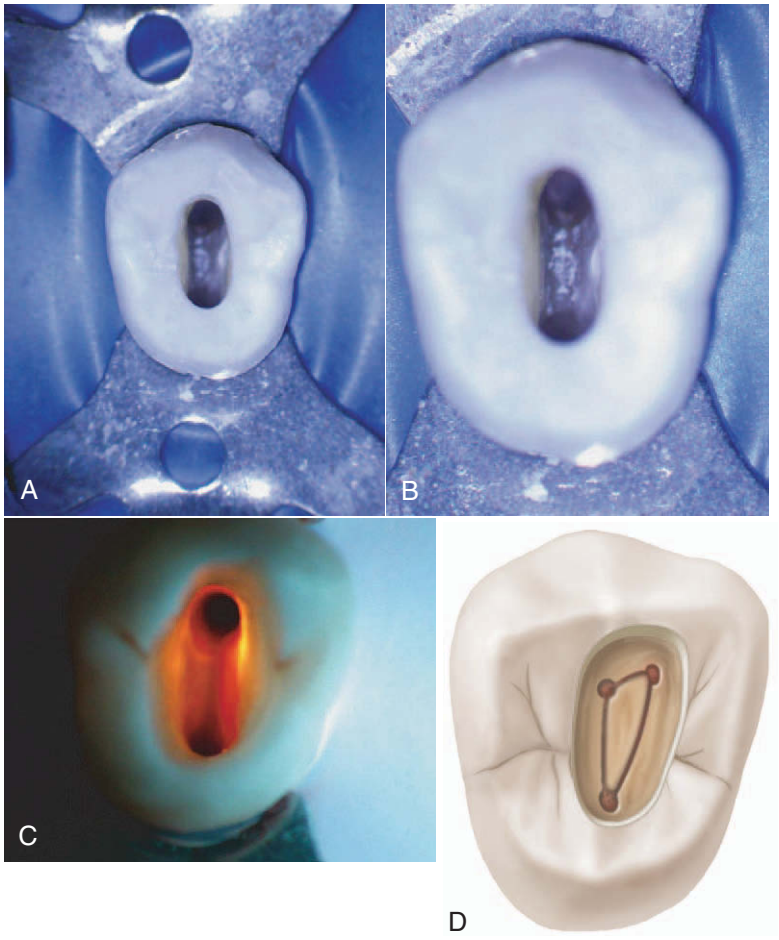


FIG. 7-91 Access cavity for a maxillary first premolar as viewed through the dental operating microscope. **A,** ×3.4 magnification. **B,** ×5.1 magnification. **C,** ×8.4 magnification with cervical fiberoptic transillumination. **D,** Schematic representation of a three-canal access preparation.



FIG. 7-92 Lateral bony lesion associated with a filled lateral canal.

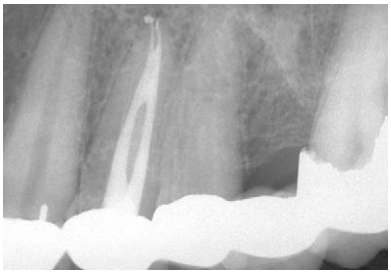


FIG. 7-93 Two canals that have fused and then redivided.



FIG. 7-94 Three canals.

Maxillary Second Premolar

The root canal system of the maxillary second premolar is wider buccolingually than mesiodistally. This tooth may have one, two, or three roots and canals (Table 7-12). Two or three canals can occur in a single root. Directional positioning of the endodontic pathfinder or a small file 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(s) 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. The proximity of this tooth to the sinus can lead to drainage of a periradicular abscess into the sinus and exposure of the sinus during apical root surgery.

Like the maxillary first premolar, the second premolar is prone to mesiodistal root fractures and fractures at the base of the cusps, usually the buccal cusp. If a fracture is suspected, all restorations in the tooth should be removed and the coronal anatomy inspected with a fiberoptic light and magnification. Full occlusal coverage is required after root canal therapy to prevent cusp and/or crown/root fracture.

When two canals are present, 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 and corresponds to the width between the buccal and palatal pulp horns (Figs. 7-95 through 7-99). If three canals are present, the external access outline form is the same triangular shape illustrated for the maxillary first premolar (see Fig. 7-91, D).

TABLE 7-12

Studies of Apical Canal Configurations for the Maxillary Second Premolar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[215]	Clearing	USA	75	24	1
Pineda and Kuttler[163]	Radiographic	Mexico	81.8	18.2	—
Caliskan et al[33]	Clearing	Turkey	72	28	—
Kartal et al[100]	Clearing	Turkey	55	44.3	0.7
Pecora et al[156]	Clearing	Brazil	67.3	32.4	0.3
Soares and Leonardo[194]	Clinical	Brazil	—	—	Case report
Ferreira et al[66]	Clinical	Brazil	—	—	Case report
Barkhordar and Sapone[17]	Clinical	USA	—	—	Case report
Low[119]	Clinical	Hong Kong	—	—	Case report
Green[80]	Ground sections	USA	96 (24)	4 (4)	—
Sert and Bayirli[184]	Clearing (men)	Turkey	49 (23)	46 (12)	5 (2)
	Clearing (women)	Turkey	75 (37)	21 (4)	4 (1)
Zaatar et al[252]	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 represent the percentage of cases with two canals joining into one canal.

†Numbers in parentheses represent the percentage of cases with one canal dividing into two canals.

‡Numbers in parentheses represent the percentage of cases with one canal dividing into three canals.

Maxillary Second Premolar—cont'd

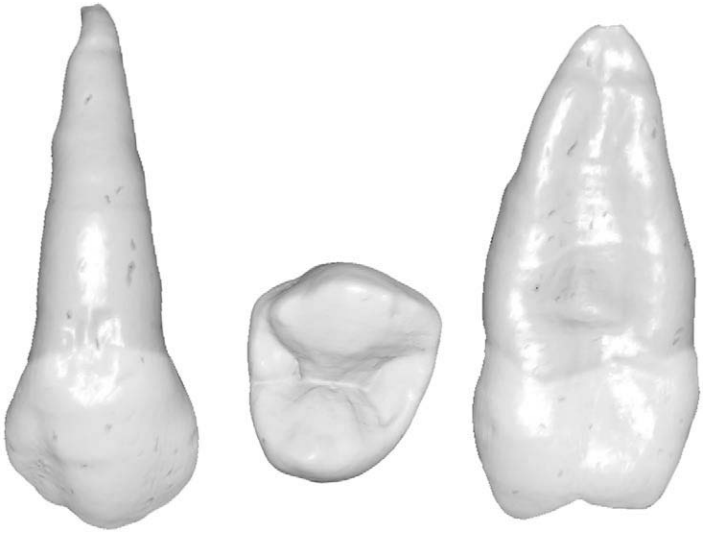


FIG. 7-95 Maxillary second premolar. Average time of eruption, 10 to 12 years; average age of calcification, 12 to 14 years; average length: 21.5 mm. Root curvature (most common to least common): distal, bayonet, buccal, straight.

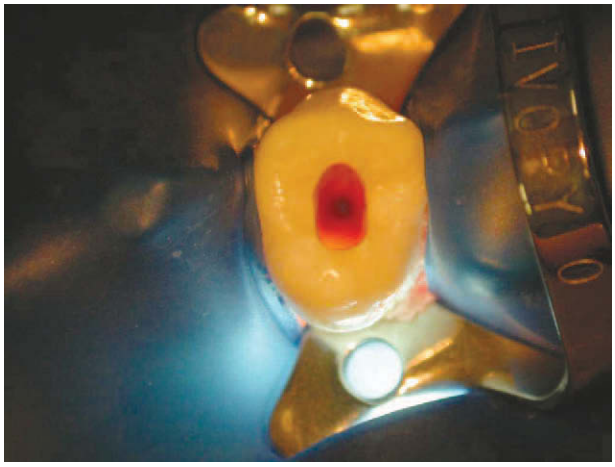


FIG. 7-96 Access cavity for a maxillary second premolar as viewed through the dental operating microscope. (×5.1 magnification with cervical fiberoptic transillumination.)



FIG. 7-97 Second premolar with three canals and a large lateral canal.



FIG. 7-98 Single canal that has divided into two canals.

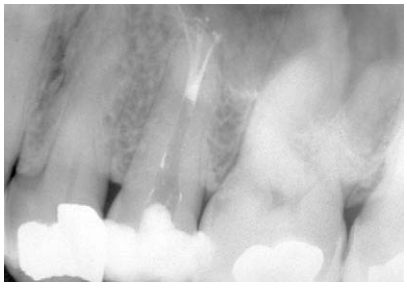


FIG. 7-99 Single canal that has split into three canals.

Maxillary First Molar

The maxillary first molar is the largest tooth in volume and one of the most complex in root and canal anatomy. 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 (MB-1) is buccal and mesial to the distobuccal orifice and is positioned within the acute angle of the pulp chamber. The second mesiobuccal canal orifice (MB-2) is located palatal and mesial to the MB-1. A line drawn to connect the three main canal orifices—the mesiobuccal (MB) orifice, distobuccal (DB) orifice, and palatal (P) 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, or three root canals (Table 7-13). The palatal root often curves buccally at the apical one third, which may not

TABLE 7-13

Studies of Apical Canal Configurations for the Palatal Root of the Maxillary First Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[215]	Clearing	USA	100	—	—
Pineda and Kuttler[163]	Radiographic	Mexico	100	—	—
Caliskan et al[33]	Clearing	Turkey	96.7	33.3	—
Acosta et al[2]	Ground section and magnification	Chili	100	—	—
Thomas et al[206]	Radiographic	USA	99.1 (0.9)†	0.9	—
Pecora et al[156]	Clearing	Brazil	100	—	—
Wasti et al[229]	Clearing	Pakistan	66.7	33.3	—
al Shalabi et al[9]	Clearing	Ireland	98.8	1.2	—
Martinez-Berná and Ruiz-Badanelli[129]	Clinical	Spain	Three case reports	—	—
Bond et al[27]	Clinical	Pakistan	Case report (100)	—	—
Beatty[20]	Clinical	USA	Case report	—	—
Maggiore et al[125]	Clinical	USA	—	—	Case report
Cecic et al[37]	Clinical	USA	—	Case report	—
Baratto-Filho et al[15]	Clinical	Brazil	—	Case report	—
Wong[242]	Clinical	USA	—	—	Case report
Thews et al[205]	Clinical	USA	—	Two case reports	—
Christie et al[41]	Clinical	Canada	—	Two case reports	—
Alavi et al[4]	Clearing	Thailand	100	—	—
Sert and Bayirli[184]	Clearing (men)	Turkey	97 (3)	3	—
	Clearing (women)	Turkey	95	2 (2)	3 (3)
Ng et al[147]	Clearing	Burma	100	—	—
Zaatar et al[252]	Clearing	Kuwait	100	—	—
Barbizam et al[16]	Clinical	Brazil	—	Case report	—
Gopikrishna et al[78]	Clinical (spiral computed tomography)	India	—	Case report	—

*Numbers in parentheses represent the percentage of cases with two canals joining into one canal.

†Numbers in parentheses represent the percentage of cases with one canal dividing into two canals.

‡Numbers in parentheses represent the percentage of cases with one canal dividing into three canals.

Maxillary First Molar—cont'd

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 (Table 7-14). From its orifice, the canal(s) 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. It may have one, two, or three root canals (Table 7-15). 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 clinician must take care not to instrument the wall excessively because a strip perforation may result.

The DOM has been used to study the location and pathway of the MB-2 canal in maxillary first and second molars.[79] The clinician must always keep in mind that the location of the MB-2 canal varies greatly; this canal generally is located mesial to or directly on a line between the MB-1 and palatal orifices, within 3.5 mm palatally and 2 mm mesially of the MB-1 orifice (Figs. 7-100 and 7-101). These authors[79] found that not all MB-2 orifices lead to a true canal. A true MB-2 orifice was present in only 84% of molars in which a second orifice was identified (Fig. 7-102).[197]

Negotiation of the MB-2 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. 7-103 through 7-107). This procedure causes the canal, when present, to shift mesially, meaning that the access wall must be moved farther mesially. Troughing may need to be 0.5 to 3 mm deep. Care must be taken to avoid furcal wall perforation of this root. Apical to the troughing level the canal may

TABLE 7-14

Studies of Apical Canal Configurations for the Distobuccal Root of the Maxillary First Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†
Vertucci[215]	Clearing	USA	100	—
Pineda and Kuttler[163]	Radiographic	Mexico	96.4	3.6
Caliskan et al[33]	Clearing	Turkey	98.4	1.6
Acosta et al[2]	Ground section and magnification	Chili	100	—
Thomas et al[206]	Radiographic	USA	96.7 (0.5)	3.3
Pecora et al[156]	Clearing	Brazil	100	—
Wasti et al[229]	Clearing	Pakistan	83.3	16.7
al Shalabi et al[9]	Clearing	Ireland	97.5	2.5
Martinez-Berná and Ruiz-Badanelli[129]	Clinical	Spain	—	Case reports (3)
Hulsmann[92]	Clinical	Germany	—	Case report
Bond et al[27]	Clinical	Pakistan	—	Case report
Beatty[20]	Clinical	USA	Case report	—
Maggiore et al[125]	Clinical	USA	Case report	—
Cecic et al[37]	Clinical	USA	Case report	—
Baratto-Filho et al[15]	Clinical	Brazil	Case report	—
Wu et al[243]	Clinical	USA	Case report	—
Alavi et al[4]	Clearing	Thailand	100	—
Sert and Bayirli[184]	Clearing (men)	Turkey	97 (6)	2 (1)
	Clearing (women)	Turkey	96 (7)	4
Ng et al[147]	Clearing	Burma	97.8	1.1
Zaatar et al[252]	Clearing	Kuwait	100	—

*Numbers in parentheses represent the percentage of cases with two canals joining into one canal.

†Numbers in parentheses represent the percentage of cases with one canal dividing into two canals.

Continued

Maxillary First Molar—cont'd

TABLE 7-15

Studies of Apical Canal Configurations for the Mesiobuccal Root of the Maxillary First Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)
Vertucci[215]	Clearing	USA	82 (17)	18	—
Pineda and Kuttler[163]	Radiographic	Mexico	51.5 (12.2)	48.5 (12.8)	—
Caliskan et al[33]	Clearing	Turkey	75.4 (41)	24.6 (1.6)	—
Acousta Vigouraux and Trugeda Bosaans[2]	Ground section and magnification	Chili	28.4	69.3	2.3
Seidberg et al[182]	Sections	USA	75 (37)	25	—
Weine et al[235]	Sectioning	USA	86 (37.5)	14	—
Stropko[199]	Clinical (microscope)	USA	45.1 (38.1)	54.9	—
Fogel et al[68]	Clinical (microscope)	Canada	68.3 (39.4)	31.7	—
Pomeranz and Fishelberg[166]	Clearing	USA	52 (21)	48	—
Kulild and Peters[109]	In vitro (microscope)	USA	41 (38.6)	59 (2.4)	—
Thomas et al[206]	Radiographic	USA	73.6 (27.3)	26.4 (12)	—
Neaverth et al[145]	Clinical		37.7 (16.7)	61.9 (1.8)	0.4
Weine et al[234]	Radiographs with files	Japan	66.2 (24.2)	33.8 (3.4)	—
Gilles and Reader[75]	SEM and microscope	USA	61.9 (52.4)	38.1 (4.8)	—
Pecora et al[158]	Clearing	Brazil	92.5 (17.5)	7.5	—
Hartwell and Bellizzi[84]	Clinical (mesiobuccal root of maxillary first molar)	USA	81.4	18.6	—
Weller and Hartwell[238]	Radiograph of root canal teeth	USA	61	39	—
Wasti et al[229]	Clearing	Pakistan	56.6 (23.3)	43.4 (13.3)	—
al Shalabi et al[9]	Clearing	Ireland	36.4 (15.9)	62.6 (6.1)	1
Bond et al[27]	Clinical	Pakistan	—	Case report	—
Beatty[20]	Clinical	USA	—	—	Case report
Maggiore et al[125]	Clinical	USA	—	Case report	—
Cecic et al[37]	Clinical	USA	—	Case report	—
Baratto-Filho et al[15]	Clinical	Brazil	Case report	—	—
Wong[242]	Clinical	USA	Case report	—	—
Alavi et al[4]	Clearing	Thailand	53.8	46.2	—
Sert and Bayirli[184]	Clearing (men)	Turkey	65 (62)	34 (6)	—
	Clearing (women)	Turkey	57 (7)	43 (16)	—
Ng et al[147]	Clearing	Burma	57.8	41.1	—
Zaatar et al[252]	Radiographs of root canal teeth	Kuwait	85	15	—
Sykaras and Economou[201]	Radiographs and microscope	Greece	91 (23)	9	—
Imura et al[93]	Clearing	Japan	11.8	88.2	—
Nosonowitz and Brenner[149]	Clinical	USA	84.9	15.1	—
Pattanshetti et al[153]	Clinical	Kuwait	100 (41.8)	—	—
Smadi and Khraisat[192]	Clearing	Jordan	53 (30)	47 (11)	—
Rwenyonyi et al[177]	Clearing (mesiobuccal root)	Uganda	80.1 (5)	19.4 (9.1)	0.5
	Clearing (distal buccal root)	Uganda	98.2 (0.5)	(1.8)	—

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.

Maxillary First Molar—cont'd

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 (MB-1, MB-2, DB, and P) (see Fig. 7-103). One study demonstrated that the access cavity should not extend into the mesial marginal ridge.[241] 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 MB-1 and DB orifices and not to the buccal surface of the tooth.

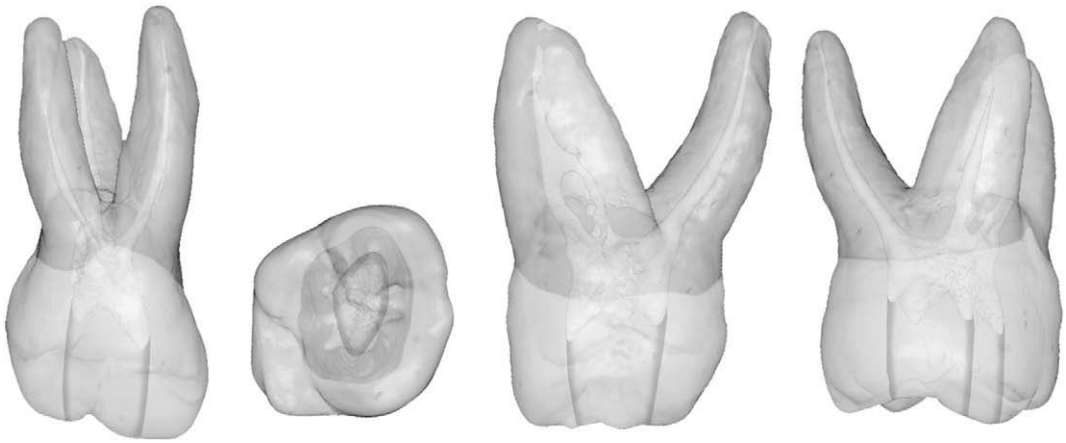


FIG. 7-100 Maxillary first molar. Average time of eruption, 6 to 7 years; average age of calcification, 9 to 10 years; average length, 20.8 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—buccal, straight.

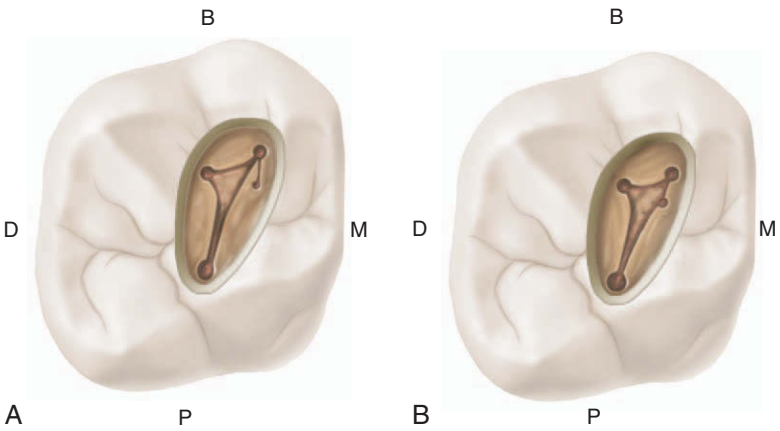


FIG. 7-101 The two locations of the second mesiobuccal *(MB-2)* canal orifices in a maxillary first molar. *B,* Buccal; *D,* distal; *M,* mesial; *P,* palatal.

Continued

Maxillary First Molar—cont'd

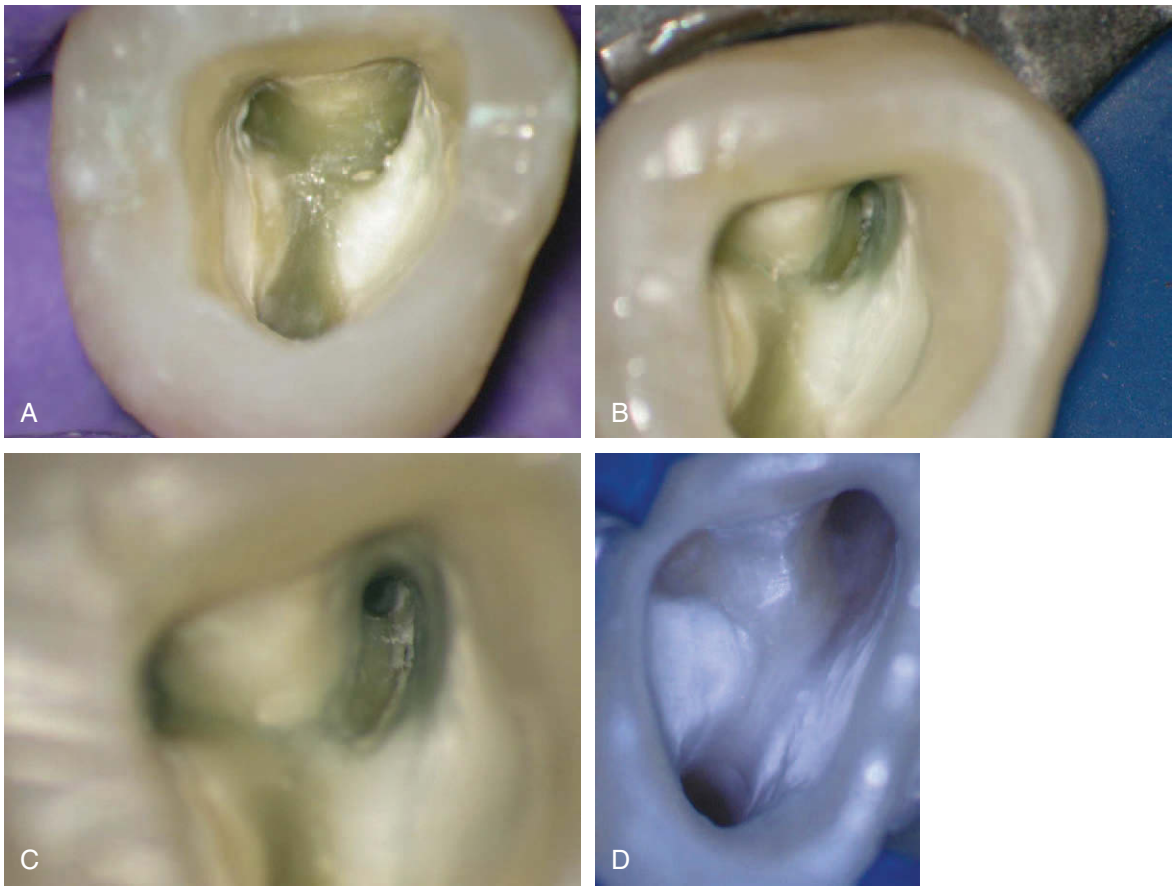


FIG. 7-102 Access cavity for a maxillary first molar as viewed through the dental operating microscope. **A,** Four apparent orifices, with a projection of dentin covering the mesial groove. (×3.4.) **B,** Removal of the mesial projection and troughing of the mesial groove to locate the second mesiobuccal *(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.)

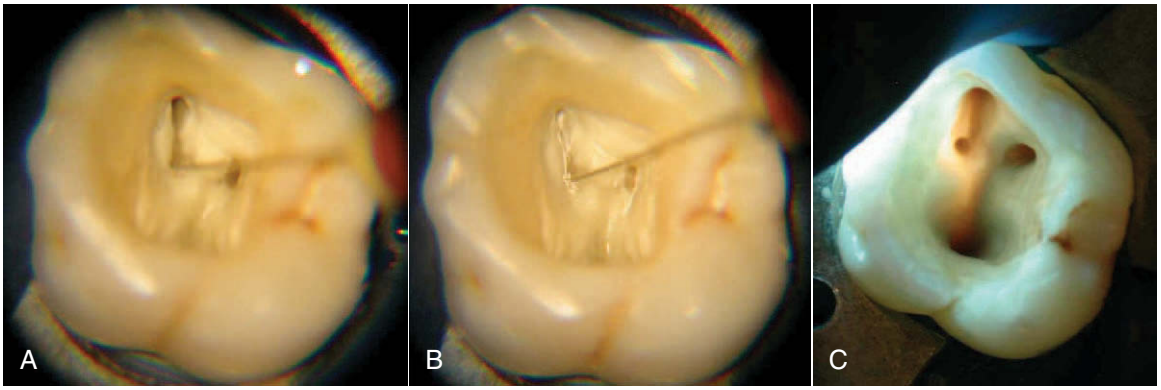


FIG. 7-103 Access preparation for a maxillary first molar as viewed through the dental operating microscope. **A,** Four apparent orifices located under ×3.4 magnification. **B,** The second mesiobuccal canal is located by deepening the mesial groove. (×5.1.) **C,** Four distinct canal orifices can be seen. (×5.1 magnification with cervical fiberoptic transillumination.)

Maxillary First Molar—cont'd



FIG. 7-104 Four canals with loops and accessory canals.



FIG. 7-105 Two canals in both buccal roots with a common foramen in each root.

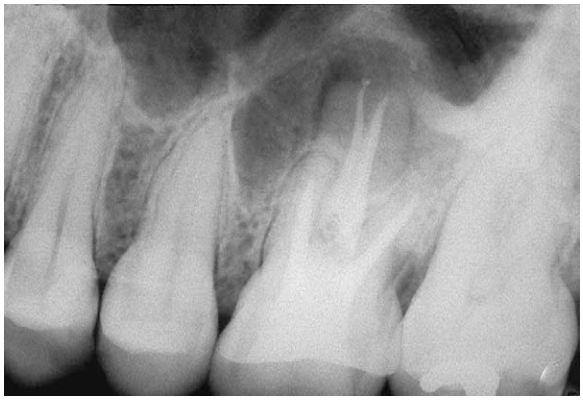


FIG. 7-106 Two separate canals in palatal root. *(Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)*

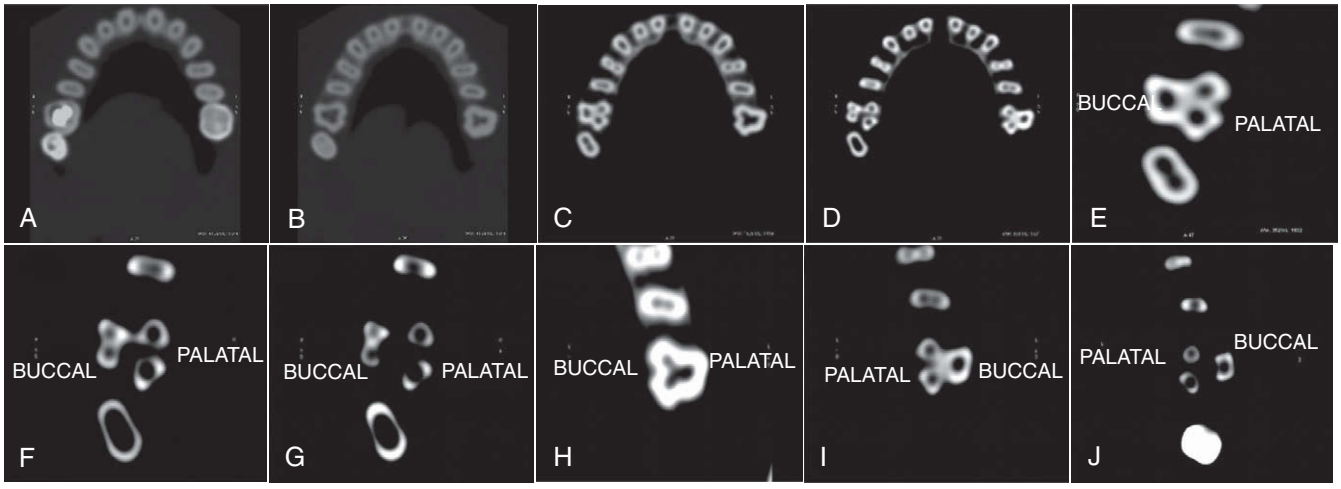


FIG. 7-107 A-D, Axial section spiral computerized tomography (SCT) image of the maxillary arch. **E-G,** Enlarged axial section SCT image of tooth #3 showing two palatal roots and two canals in the fused buccal root. **H-J,** Enlarged axial section SCT image of tooth #14, showing the two palatal roots and a single canal in the fused buccal root. *(From Gopikrishna V, Reuben J, Kandaswamy D:* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *105:e74, 2008.)*

Maxillary Second Molar

Coronally, the maxillary second molar closely resembles the maxillary first molar. The root and canal anatomy are similar to those of the first molar, although differences occur. The distinguishing morphologic feature of the 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 (Tables 7-16 to 7-18). Four canals are less likely to be present in the second molar than in the first molar. The three main orifices (MB, DB, and P) usually form a flat triangle and sometimes almost a straight line (Figs. 7-108 through 7-113). 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; and the palatal orifice usually is 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 fused roots occasionally have only two canals; in rare cases, they have only one. Teeth with two canals usually have a buccal and a palatal canal of equal length and diameter (see Fig. 7-109, 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

TABLE 7-16

Studies of Apical Canal Configurations for the Mesiobuccal Root of the Maxillary Second Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Four Canals (%)
Vertucci[215]	Clearing	USA	88 (17)	12	—	—
Pineda and Kuttler[163]	Radiographic	Mexico	72.8 (8.2)	27.2 (14.4)	—	—
Caliskan et al[33]	Clearing	Turkey	68.7 (23.6)	27.1 (4.2)	4.2	—
Pecora et al[158]	Clearing	Brazil	80 (22)	20	—	—
Gilles and Reader[75]	SEM and light microscopy	USA	62 (32.4)	38 (2.7)	—	—
Stropko[199]	Clinical (microscope)	USA	78 (45.6)	22	—	—
Eskoz and Weine[60]	Radiographic		80.6 (20.9)	19.4 (3)	—	—
Pomeranz and Fishelberg[166]	Clinical	USA	75.9 (13.8)	24.1	—	—
Nosonowitz and Brenner[149]	Clinical	USA	94.4 (25.5)	5.6	—	—
al Shalabi et al[9]	Clearing	Ireland	50 (5.6)	50 (16.7)	—	—
Benenati[24]	Clinical	USA	Case report	—	—	—
Fahid and Taintor[63]	Clinical	USA	Case report	—	—	—
Sert and Bayerl[184]	Clearing (men)	Turkey	71 (45)	28	1	—
	Clearing (women)	Turkey	81 (25)	18	—	1
Kulild and Peters[109]	In vitro (microscope)	USA	54.2	45.8	—	—
Alavi et al[4]	Clearing	Thailand	53.8	44.6	—	1
Zaatar et al[252]	Radiographs of root canal teeth	Kuwait	93.5 (17.4)	6.5	—	—
Imura et al[93]	Clearing	Japan	30	70	—	—
Ng et al[147]	Clearing	Burma	74	26	—	—
Rwenyonyi et al[177]	Clinical (mesial root)	Uganda	88.7 (1.8)	11.7 (5)	—	—
	Clinical (distal buccal root)	Uganda	99.5	(0.5)	—	—
	Clinical (palatal root)	Uganda	99.5 (0.5)	(0.5)	—	—

*Numbers in parentheses represent the percentage of cases in which two canals joined into one.

†Numbers in parentheses represent the percentage of cases in which one canal divided to form two.

Maxillary Second Molar—cont'd

TABLE 7-17

Studies of Apical Canal Configurations for the Distobuccal Root of the Maxillary Second Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)
Vertucci[215]	Clearing	USA	100	—
Pineda and Kuttler[163]	Radiographic	Mexico	100	—
Caliskan et al[33]	Clearing	Turkey	100	—
Pecora et al[158]	Clearing	Brazil	100	—
al Shalabi et al[9]	Clearing	Ireland	100	—
Benenati[24]	Clinical	USA	Case report	—
Fahid and Taintor[63]	Clinical	USA	—	Case report
Sert and Bayirli[184]	Clearing (men)	Turkey	100 (2)	—
	Clearing (women)	Turkey	100 (2)	—
Zaatar et al[252]	Radiographs of root canal teeth	Kuwait	100	—

*Numbers in parentheses represent the percentage of cases with two canals joining into one canal.

TABLE 7-18

Studies of Apical Canal Configurations for the Palatal Root of the Maxillary Second Molar

Author(s)	Type of Study	Country	One Canal (%)	Two Canals (%)	Three Canals (%)
Vertucci[215]	Clearing	USA	100	—	—
Pineda and Kuttler[163]	Radiographic	Mexico	100	—	—
Caliskan et al[33]	Clearing	Turkey	97.9	2.1*	—
Pecora et al[158]	Clearing	Brazil	100	—	—
al Shalabi et al[9]	Clearing	Ireland	100	—	—
Benenati[24]	Clinical	USA	—	Case report	—
Sert and Bayirli[184]	Clearing (men)	Turkey	100	—	—
	Clearing (women)	Turkey	100	—	—
Zaatar et al[252]	Radiographs of root canal teeth	Kuwait	100	—	—
Ulusoy and Görgul[213]	Clinical	Turkey	—	Case report	—
Shin et al[185]	Clinical (microscope)	South Korea	—	Case report	—

*Number represents the percentage of cases in which one canal separates into two.

visibility, especially when interference arises from the malar process, a more perpendicular and 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.[159]

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. 7-110). 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 MB and P 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.

Continued

Maxillary Second Molar—cont'd

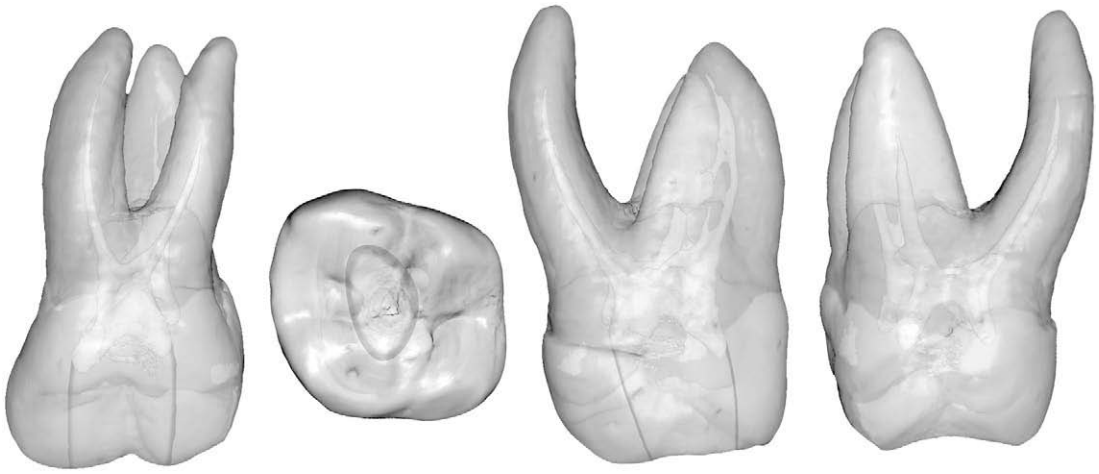


FIG. 7-108 Maxillary second molar. Average time of eruption, 11 to 13 years; average age of calcification, 14 to 16 years; average length, 20 mm. Root curvature (most common to least common): mesiobuccal root—distal, straight; distobuccal root—straight, mesial, distal; palatal root—straight, buccal.

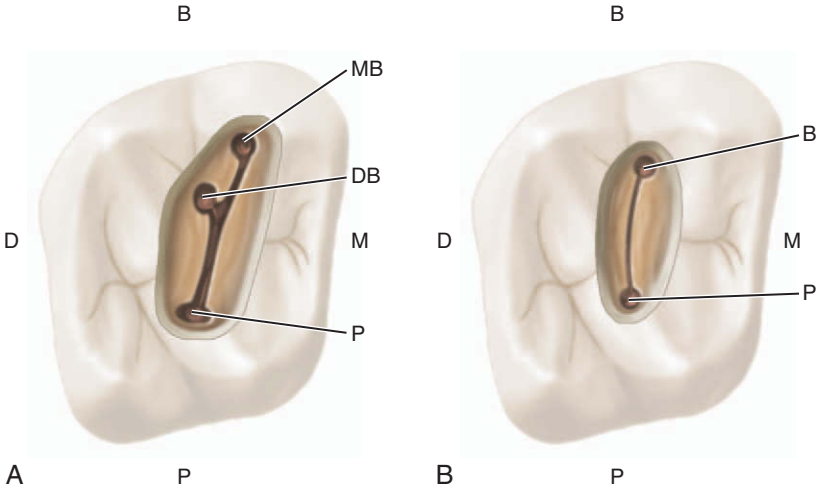


FIG. 7-109 A, Three canal orifices in a maxillary second molar. **B,** Two canal orifices in a maxillary second molar. *B,* Buccal; *P,* palatal; *D,* distal, *DB,* distobuccal; *M,* medial; *MB,* mesiobuccal.

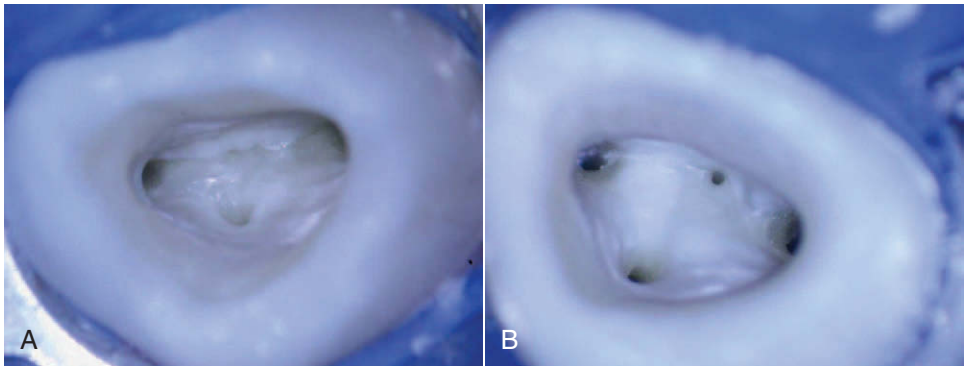


FIG. 7-110 Access cavity for a maxillary second molar as viewed through the dental operating microscope. **A,** Dentin projection covering the mesial aspect of the floor of the pulp chamber. (×8.4.) **B,** Fourth canal orifice (second mesiobuccal, MB-2), which was identified after removal of the dentin projection and troughing of the groove connecting the MB-1 orifice and the palatal (P) canal orifice. (×8.4.)

Maxillary Second Molar—cont'd

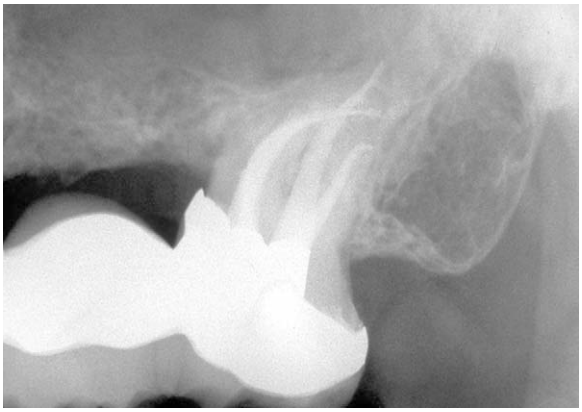


FIG. 7-111 Severely curved mesiobuccal root with a right-angle curve in the distobuccal (DB) root.

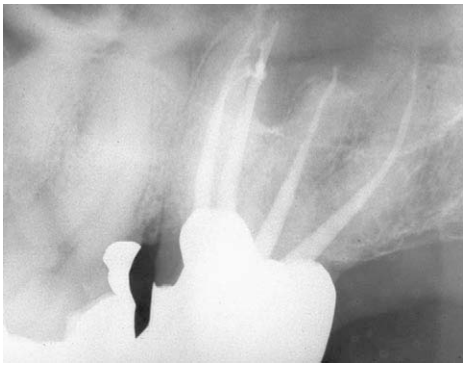


FIG. 7-112 Four canals (MB-1, MB-2, DB, and P) can be seen.



FIG. 7-113 Four canals with two distinct palatal roots and canals.

Maxillary Third Molar

Loss of the maxillary first and second molars often is the reason the third molar must be considered as a strategic abutment. Another indication for root canal therapy and full coverage is a fully functioning mandibular third molar in an arch that has sufficient room for full eruption and oral hygiene.

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 (Table 7-19; Figs. 7-114 through 7-116). 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 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. The MB, DB, and P orifices often lie nearly in a straight line as the DB orifice moves even closer to the line connecting the MB and P orifices. The resultant access cavity is an oval or highly obtuse triangle.

TABLE 7-19

Studies of Apical Canal Configurations for the Maxillary Third Molar

				Number of Canals (%)					
Authors	**Type of Study**	**Country**	**Number of Roots**	**1**	**2**	**3**	**4**	**5**	**6**
Sidow et al[186]	Clearing	USA	1	2.7	2.7	6	2	1.3	0.7
	Clearing	USA	2 (4.7%*)	—	—	22	4.7	0.7	—
	Clearing	USA	3	—	0.7	29.3	14.7	1.7	—
	Clearing	USA	4	—	—	—	6	2.6	—
Pineda and Kuttler[163]	Radiographic	Mexico	Mesiobuccal	42 (20.6*)	31.1	21	5.9	—	—
Pecora et al[158]	Clearing	Brazil	Mesiobuccal	86 (18*)	14	—	—	—	—

*Percentage of cases in which two canals joined to form one.

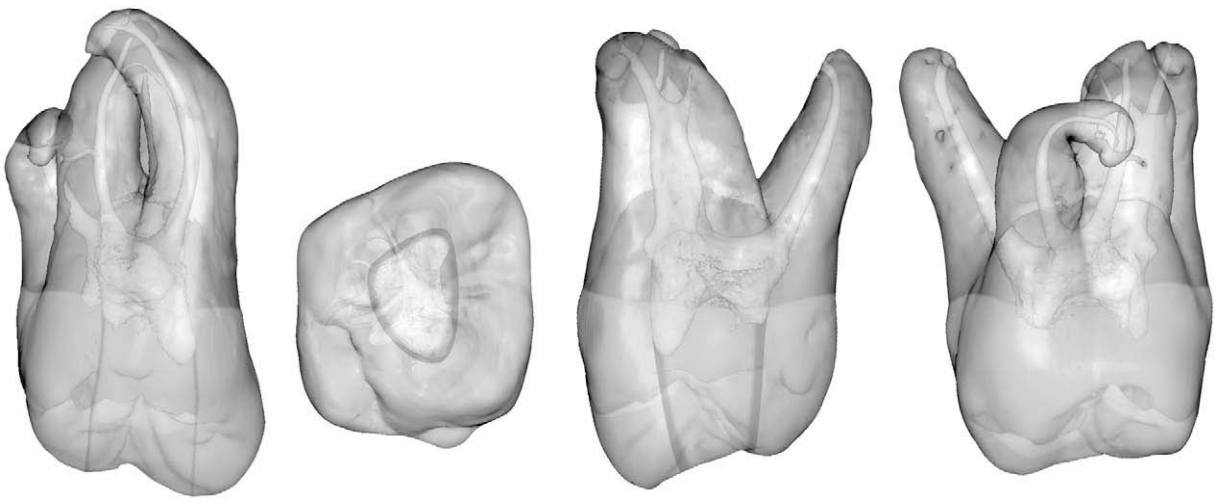


FIG. 7-114 Maxillary third molar. Average time of eruption, 17 to 22 years; average age of calcification, 18 to 25 years; average length: 17 mm.

Maxillary Third Molar—cont'd

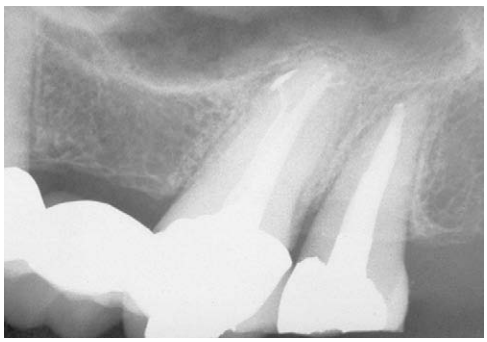


FIG. 7-115 Canals that have fused into a single canal. Multiple accessory canals can be seen in the second molar.

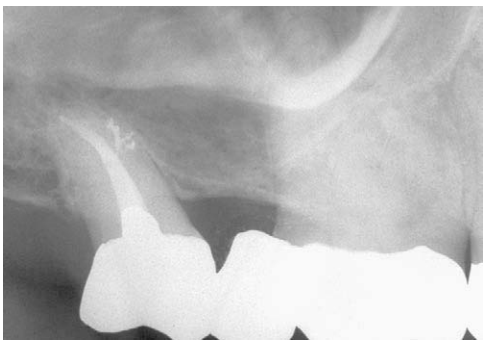


FIG. 7-116 Distal bridge abutment with major accessory canal.

Mandibular Central and Lateral Incisors

The root canal systems and access cavities for the two mandibular incisors are so similar they are discussed together. 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, 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 (Table 7-20). One study determined that a relationship existed between crown size (expressed as the M-D/F-L index) and the incidence of bifid root canals in these teeth.[228] 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. 7-117 through 7-120). 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[148] determined that by age 40 years the mandibular incisor pulp chamber has decreased in size sufficiently to routinely justify an oval access cavity. 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 clinician should extend the access preparation well into the cingulum gingivally. Because the lingual surface of this tooth is not involved with occlusal function, butt joint junctions between the internal walls and the lingual surface are not required.

Continued

Mandibular Central and Lateral Incisors—cont'd

TABLE 7-20

Studies of Apical Canal Configurations for the Mandibular Incisors

Author(s)	Type of Study	Country	Tooth	One Canal (%)*	Two Canals (%)†	Three Canals (%)‡
Vertucci[215]	Clearing	USA	Central	97 (5)	3	—
Pineda and Kuttler[163]	Radiographic	Mexico	Central	97.9 (2)	2.1	—
Caliskan et al[33]	Clearing	Turkey	Central	96 (13.7)	2†	2
Madeira and Hetem[123]	Clearing	Brazil	Central	99.7 (11)	0.3	—
Funato et al[70]	Clinical	Japan	Central	—	Case report	—
Sert and Bayirli[184]	Clearing (men)	Turkey	Central	89 (54)	10 (2)	1 (1)
	Clearing (women)	Turkey	Central	85 (55)	13 (3)	2
Walker[226]	Radiographic	Southern China	Central	99	1	—
Vertucci[215]	Clearing	USA	Lateral	98 (5)	2	—
Pineda and Kuttler[163]	Radiographic	Mexico	Lateral	98.7 (3.2)	1.3	—
Caliskan et al[33]	Clearing	Turkey	Lateral	98 (13.7)	2†	—
Madeira and Hetem[123]	Clearing	Brazil	Lateral	99.2 (11.1)	0.8	—
Sert and Bayirli[184]	Clearing (men)	Turkey	Lateral	90 (52)	10	—
	Clearing (women)	Turkey	Lateral	90 (54)	10 (2)	—
Walker[226]	Radiographic	Southern China	Lateral	99	1	—
Karagöz-Kucukay[99]	Clearing	Turkey	Incisors	85	15†	—
Kartal and Yanikoglu[101]	Clearing	Turkey	Incisors	92 (17)	9 (3†)	—
Benjamin and Dowson[23]	Radiographic with files	USA	Incisors	98.7 (40.1)	1.3	—
Miyashita et al[135]	Clearing	Japan	Incisors	96.9 (9.3)	3.1 (1.7†)	—
Rankine-Wilson and Henry[168]	Radiographic	Australia	Incisors	94.6	5.4	—
Kartal and Yanikoglu[101]	Clearing	Turkey	Incisors	92	7	1
Green[80]	Ground sections and microscope	USA	Incisors (both)	95 (17)	5 (1)	—
Al-Qudah and Awawdeh[8]	Clearing	Jordan	Incisors (both)	91.3 (17.6)	8.7 (3.6)	—

*Numbers in parentheses represent the percentage of cases with two canals joining into one canal.

†Numbers in parentheses represent the percentage of cases with one canal dividing into two canals.

‡Number in parentheses represents the percentage of cases with one canal dividing into three canals.

Mandibular Central and Lateral Incisors—cont'd

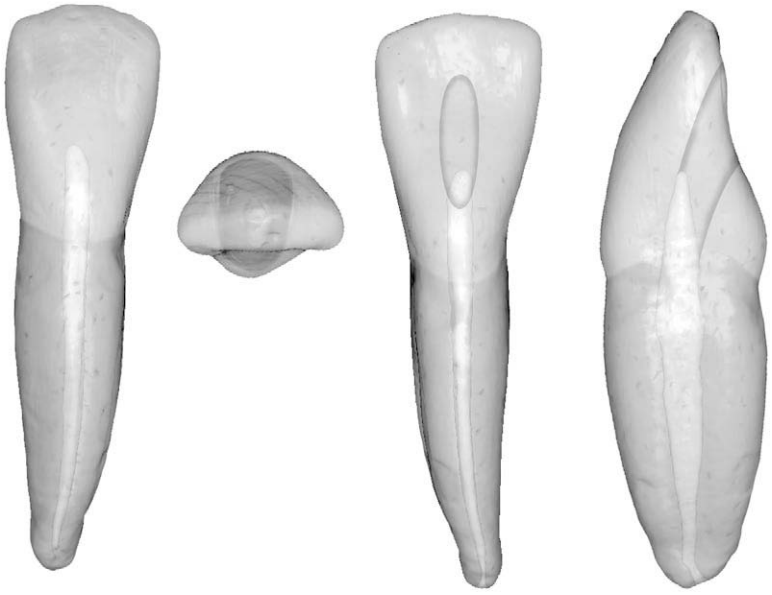


FIG. 7-117 Mandibular central/lateral incisors. Average time of eruption, 6 to 8 years; average age of calcification, 9 to 10 years; average length: 20.7 mm. Root curvature (most common to least common): straight, distal, labial.

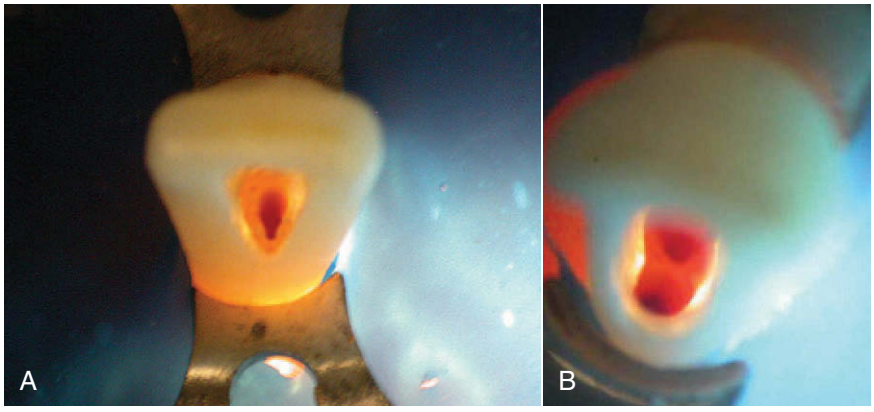


FIG. 7-118 Access cavity for the mandibular incisors as viewed through the dental operating microscope. **A,** One canal orifice. (×8.5 magnification with cervical fiberoptic transillumination.) **B,** Two canal orifices. (×8.5 magnification with cervical fiberoptic transillumination.)

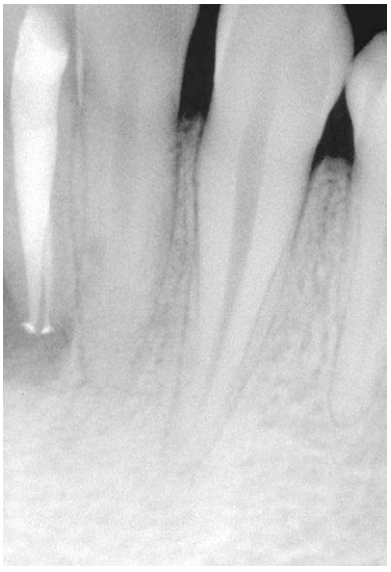


FIG. 7-119 Double-rooted mandibular lateral incisor.

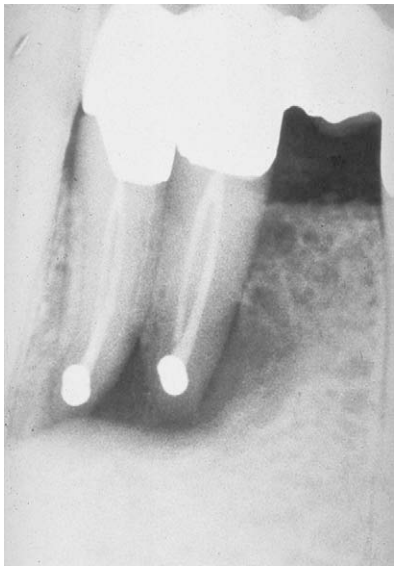


FIG. 7-120 Two canals in the mandibular lateral and central incisors.

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 (Table 7-21). 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 (Figs. 7-121 through 7-126). 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. As with the mandibular incisors, butt joint relationships between internal walls and the lingual surface are not necessary.

TABLE 7-21

Studies of Apical Canal Configurations for the Mandibular Canine

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)	Three Canals (%)
Vertucci[215]	Clearing	USA	94 (14)	6	—
Pineda and Kuttler[163]	Radiographic	Mexico	95 (13.5)	5	—
Caliskan et al[33]	Clearing	Turkey	98 (3.9)	2†	—
Pecora et al[157]	Clearing	Brazil	97.1 (4.9)	2.9	—
D'Arcangelo et al[51]	Clinical	Italy	—	Case report	—
Orguneser and Kartal[151]	Clinical	Turkey	—	Case report (3 → 2)‡	—
Heling et al[87]	Clinical	Israel	—	—	Case report
Green[80]	Ground sections and microscope	USA	97 (10)	3 (2)	—
Sert and Bayirli[184]	Clearing (men)	Turkey	100 (10)	—	—
	Clearing (women)	Turkey	97 (35)	3	—

*Numbers in parentheses represent 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.

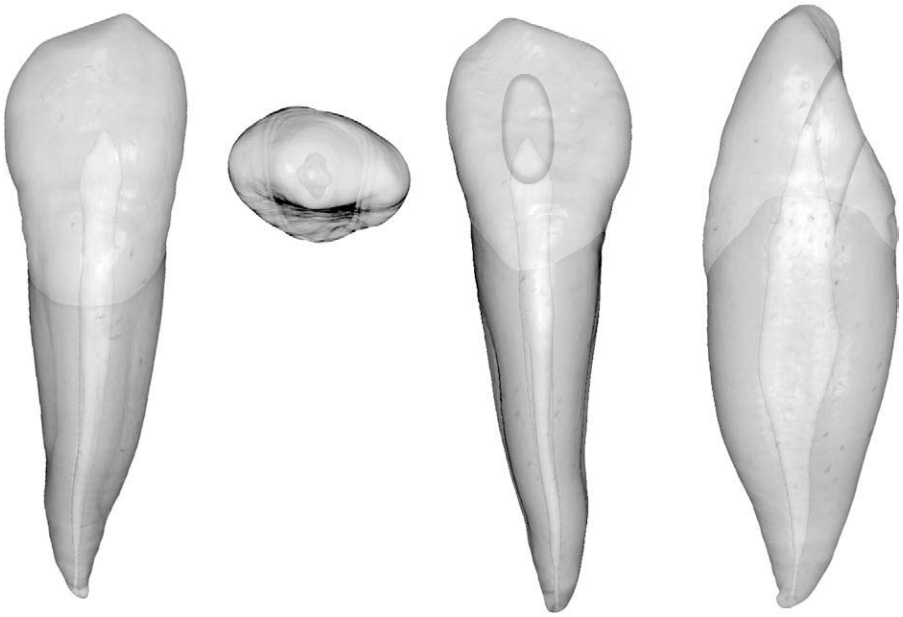


FIG. 7-121 Mandibular canine. Average time of eruption, 9 to 10 years; average age of calcification, 13 years; average length, 25.6 mm. Root curvature (most common to least common): straight, distal, labial.

Mandibular Canine—cont'd

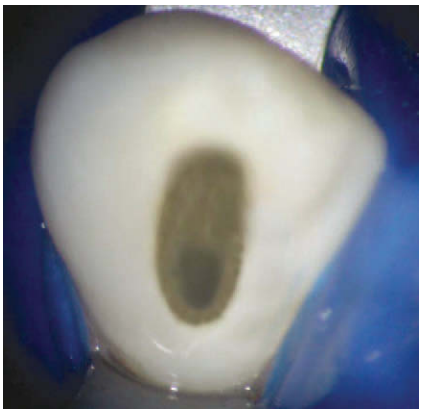


FIG. 7-122 Access preparation for a mandibular canine as viewed through the dental operating microscope. (×5.1.)



FIG. 7-123 One canal with a sharp mesial curvature at the apex.



FIG. 7-124 Two canals with the lateral canal above the crest of bone; the lateral canal is probably responsible for the pocket depth.



FIG. 7-125 Two separate root canals.



FIG. 7-126 Mandibular canine and lateral incisor with two canals.

Mandibular First Premolar

As a group, the mandibular premolars are difficult to treat. They have a high flare-up and failure rate. A possible explanation may be the extreme variations in root canal morphology in these teeth. The root canal system of the mandibular first premolar is wider buccolingually than mesiodistally. 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 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 (Table 7-22). One study reported a C-shaped canal anatomy in this tooth.[14]

The oval external outline form of the mandibular first premolar typically is wider mesiodistally than its maxillary counterpart, making it more oval and less slot shaped (Figs. 7-127 through 7-131). 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 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.

TABLE 7-22

Studies of Apical Canal Configurations for the Mandibular First Premolar

Author(s)	Type of Study	Country	One Canal (%)	Two Canals (%)*	Three Canals (%)	Four Canals (%)
Vertucci[215]	Clearing	USA	74	25.5 (24)	0.5	—
Pineda and Kuttler[163]	Radiographic	Mexico	74.2	24.9 (23.4)	0.9	—
Caliskan et al[33]	Clearing	Turkey	75.5 (7.6†)	18.8 (9.4)	5.7	—
Zillich and Dowson[254]	Radiographic	USA	80.7	18.9	0.4	—
Baisden et al[14]	Serial sections	USA	76	24 (24)	—	—
Yoshioka et al[251]	Clearing	Japan	80.6	15.1‡	4.3	—
Trope et al[211]	Radiographic	USA				
Black patients			67.3	—	32.8	—
White patients			86.3	—	13.7	—
Green[80]	Ground sections and microscope	USA	94 (4)	6 (2)	—	—
Sert and Bayirli[184]	Clearing (men)	Turkey	85 (20)	15 (5)	0	—
	Clearing (women)	Turkey	94 (38)	4	2	—
Zaatar et al[252]	Radiographs of root canal teeth	Kuwait	60	40	—	—
Walker[225]	Radiographic	S. China	65	35	—	—
Nallapati[143]	Clinical	USA	—	—	Case report	—
Moayedi and Lata[139]	Clinical	—	—	—	Case report	—
Yang[249]	Clinical	Australia	—	—	Case report	—
Chan et al[39]	Clinical	Taiwan, China	—	—	Case report	—
England et al[59]	Clinical	USA	—	Case report	—	—
Hülsman[91]	Clinical	Germany	—	—	Case report	—
Lu et al[120]	Radiography and sectioning	China	60	40	—	—
Al-Qudah and Awawdeh[8]	Clearing	Jordan	64.8 (6.6)	33 (18.6)	2.2	—

*Numbers in parentheses represent 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).

Mandibular First Premolar—cont'd

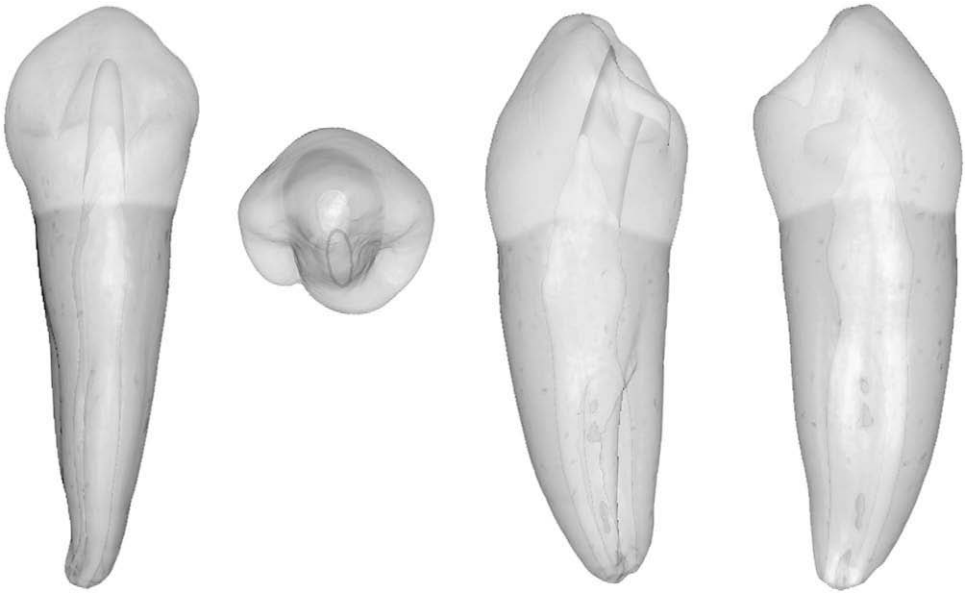


FIG. 7-127 Mandibular first premolar. Average time of eruption, 10 to 12 years; average age of calcification, 12 to 13 years; average length: 21.6 mm. Root curvature (most common to least common): straight, distal, buccal.

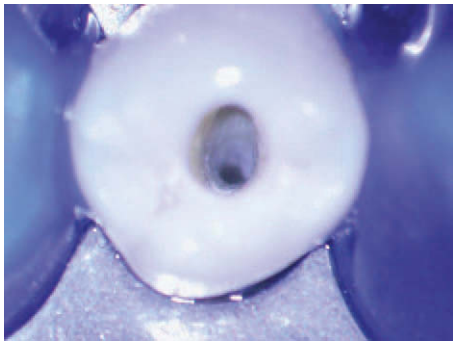


FIG. 7-128 Access cavity for a mandibular first premolar as viewed through the dental operating microscope: one orifice. (×5.1.)



FIG. 7-129 Two canals. *(Courtesy Dr. Raed Kasem, Clearwater, FL.)*

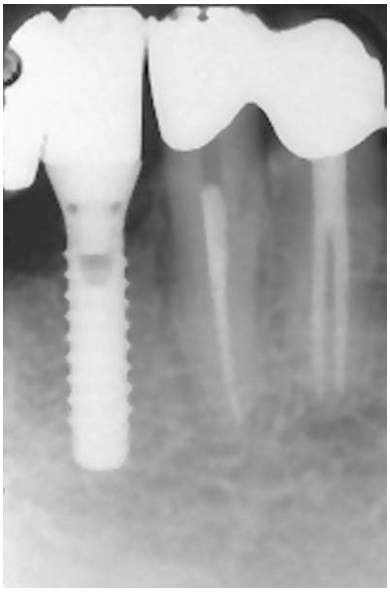


FIG. 7-130 Single canal that has divided into two.



FIG. 7-131 Three canals.

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. 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 (Table 7-23).

The access cavity form for the mandibular second premolar varies 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, and therefore the lingual access extension typically is halfway up the lingual cusp incline.[56] 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. 7-132 through 7-138).

TABLE 7-23

Studies of Apical Canal Configurations for the Mandibular Second Premolar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Four Canals (%)
Vertucci[215]	Clearing	USA	97.5	2.5	—	—
Pineda and Kuttler[163]	Radiographic	Mexico	98.8	1.2	—	—
Caliskan et al[33]	Clearing	Turkey	93.6	6.4	—	—
Kerekes and Tronstad[104]	Sections	Norway	85	15	—	—
Okumura[150]	Clearing	Japan	98.2	1.8	—	—
Zillich and Dowson[254]	Radiographic and file	USA	88.4 (1)	11.2	0.4	—
Trope et al[211]	Radiographic	USA				
Black patients			92.2	—	7.8	—
White patients			97.2	—	2.8	—
Holtzman[89]	Clinical	Israel	—	—	—	Case report
El Deeb[58]	Clinical	USA	—	—	Case report	—
Rödig and Hülsmann[173]	Clinical	Germany	—	—	Case report	—
Bram and Fleisher[28]	Clinical	USA	—	—	—	Case report
Rhodes[170]	Clinical	England	—	—	—	Case report
Macri and Zmener[122]	Clinical	Argentina	—	—	—	Case report (5 → 4)‡
Green[80]	Ground sections and microscope	USA	94 (4)	6 (2)	—	—
Sert and Bayirli[184]	Clearing (men)	Turkey	71 (14)	29 (11)	—	—
	Clearing (women)	Turkey	92 (7)	8 (8)	—	—
Nallapati[143]	Clinical	USA	—	—	Case report	—
Fischer and Evans[67]	Clinical	USA	—	—	Case report	—
Sachdeva et al[179]	Clinical (spiral computed tomography)	India	—	—	—	Case report
Al-Qudah and Awawdeh[8]	Clearing	Jordan	76.8 (4.8)	22.8 (15.3)	0.5	—
DeMoor and Calberson[56]	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.

Mandibular Second Premolar—cont'd

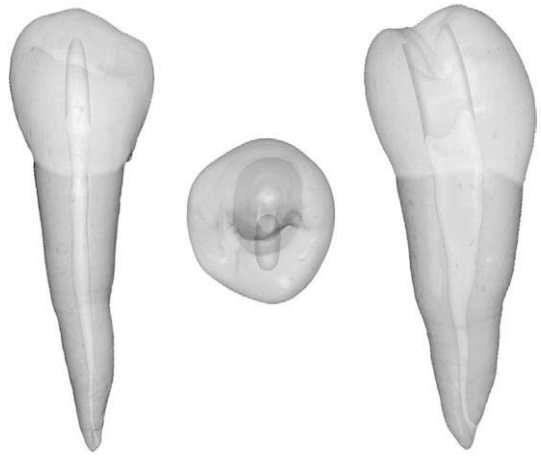


FIG. 7-132 Mandibular second premolar. Average time of eruption, 11 to 12 years; average age of calcification, 13 to 14 years; average length: 22.3 mm. Root curvature (most common to least common): straight, distal, buccal.

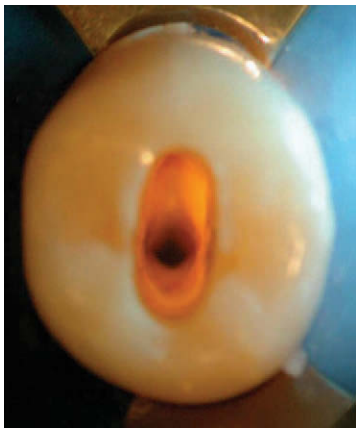


FIG. 7-133 Access cavity for a mandibular second premolar as viewed through the dental operating microscope: one canal orifice. (×5.1 magnification with cervical fiberoptic transillumination.)



FIG. 7-134 Two canals. *(Courtesy Dr. Haider AlZubaidi, Ocala, FL.)*



FIG. 7-135 Single canal that has divided at the apex. *(Courtesy Dr. Haider AlZubaidi, Ocala, FL.)*

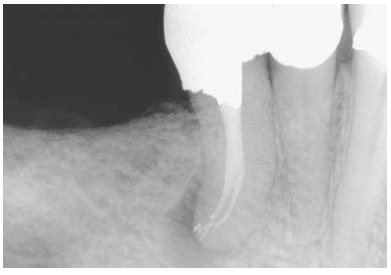


FIG. 7-136 Single canal that has divided and crossed over at the apex.



FIG. 7-137 Three separate canals. *(Courtesy Dr. Haider AlZubaidi, Ocala, FL.)*

Continued

Mandibular Second Premolar—cont'd

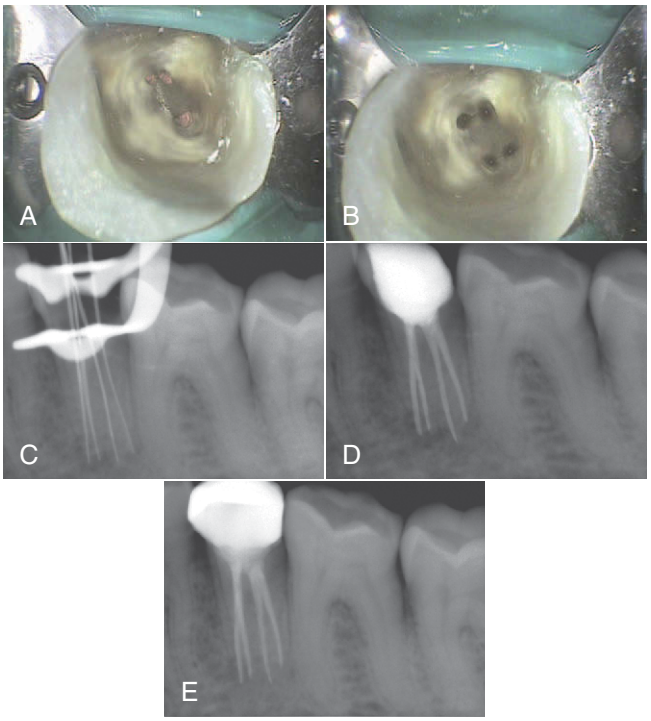


FIG. 7-138 A, Initial access opening. **B,** Access opening showing orifices of the four root canals. **C,** Working length determination. **D,** Immediate posttreatment radiograph. **E,** Recall radiograph 1 year posttreatment. *(From Sachdeva GS, Ballal S, Gopikrishna V, Kandaswamy D:* J Endod *34:104, 2008.)*

Mandibular First Molar

The earliest permanent posterior tooth to erupt, the mandibular first molar seems to be the tooth that most often requires root canal treatment. It often is extensively restored, and it is subjected to heavy occlusal stress. Therefore 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 (Tables 7-24 and 7-25). The canals in the mesial root are the MB and ML canals; a middle mesial (MM) canal sometimes is present in the developmental groove between the MB and ML canals. The incidence of an MM canal ranges from 1%[215] to 15%.[76] The canals in the distal root include the distal canal (if only one canal is present) and the DB, DL, and middle distal (MD) canals (if more than one is present).[74] 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 (MB, ML, DB, and DL) are present.

The presence of two separate distal roots is rare but does occur. In such cases the DL root is smaller than the DB root and usually more curved. Also, the DL root often has a sharp apical hook toward the buccal side that is not obvious on radiographs. 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. Care must be taken to minimize instrumentation against these walls, because overzealous cutting of the concavity can lead to a strip perforation of the root.

The mesial canal orifices usually are well separated within the main pulp chamber and connected by a developmental groove. The MB orifice commonly is under the mesiobuccal cusp, whereas the ML orifice generally is found just lingual to the central groove. On occasion an MM canal orifice is present in the groove between the MB and ML orifices (Figs. 7-139 through 7-147). 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

Mandibular First Molar—cont'd

TABLE 7-24

Studies of Apical Canal Configurations for the Mesial Root of the Mandibular First Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Four Canals (%)
Vertucci[215]	Clearing	USA	40 (28)	59 (8)	1	—
Pineda and Kuttler[163]	Radiographic	Mexico	43 (30)	57 (7)	—	—
Caliskan et al[33]	Clearing	Turkey	40 (37)	56.6 (7)	3.4	—
Skidmore and Bjorndal[190]	Cast resin	USA	44.5 (38)	55.5	—	—
Pomeranz et al[165]	Clinical	USA	—	1.6	1.6	—
Wasti et al[229]	Clearing	Pakistan	(23)	73.7	3.3	—
Zaatar et al[253]	Clearing	Kuwait	63.3 (57)	34.7	—	—
Sperber and Moreau[195]	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[130]	Clinical	Spain	—	—	Case report	—
Fabra-Campos[61]	Clinical	Spain	—	—	Case report	—
Baugh and Wallace[19]	Clinical	USA	—	Case report (3 → 2)§	—	—
Ricucci[171]	Clinical	Italy	—	Case report (3 → 2)§	—	—
DeGrood and Cunningham[53]	Clinical	USA	—	Case report (3 → 2)§	—	—
Jacobsen et al[95]	Clinical	USA	—	Case report (3 → 2)§	Case report	Case report
Reeh[169]	Clinical	USA	—	—	—	Case report
Sert and Bayirli[184]	Clearing (men)	Turkey	52 (52)	44 (3)	3	—
	Clearing (women)	Turkey	49 (46)	51 (6)	—	—
Walker[226]	Radiographic	Southern China	24	75	1	—
da Costa et al[52]	Clearing	Brazil	54.6	45.4	—	—
Pattanshetti et al[153]	Clinical	Kuwait	(100)	—	—	—

*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.

‡Studied incidence in middle mesial canals only.

§Three canals reconfigured to form two; 2% represents three canals that join into two.

developmental groove between the MB and ML orifices. The clinician should use magnification to explore this developmental groove carefully 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 canal. This is best accomplished with the DOM (see Fig. 7-141).

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 an MD canal orifice is present.

If three root canals (MB, ML, and D) 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 (DB and DL) 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 MB 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 are 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

Continued

TABLE 7-25

Studies of Apical Canal Configurations for the Distal Root for the Mandibular First Molar

Author(s)	Type of Study	Country	One Canal (%)*	Two Canals (%)†	Three Canals (%)
Vertucci[215]	Clearing	USA	85 (15)	15 (8)	—
Pineda and Kuttler[163]	Radiographic	Mexico	85.7 (12.7)	14.3 (8.6)	—
Caliskan et al[33]	Clearing	Turkey	81.4 (33)	16.9 (7)	1.7
Zaatar et al[253]	Clearing	Kuwait	83.7 (8)	16.3	—
Sperber and Moreau[195]	Ground sections	Senegal	78	(22)	0.2
Gulabivala et al[82]	Clearing	Pakistan	83.3 (4)	16 (3)	0.7
Wasti et al[229]	Clearing	Pakistan	56.7 (27)	43.3 (20)	—
Skidmore and Bjorndal[190]	Cast resin	USA	88.8 (18)	11.2	—
Ricucci[171]	Clinical	Italy	Case report	—	—
DeGrood and Cunningham[53]	Clinical	USA	—	Case report (3 → 2)‡	—
Martinez-Berná and Badanelli[130]	Clinical	Spain	Case report (3 → 1)§	Case report (3 → 2)‡	—
Beatty and Krell[22]	Clinical	USA	—	Case report	—
Reeh[169]	Clinical	USA	—	—	Case report
Beatty and Interian[21]	Clinical	USA	—	—	Case report
Friedman et al[69]	Clinical	Israel	—	—	Case report
Stroner et al[198]	Clinical	USA	—	—	Case report
Sert and Bayirli[184]	Clearing (men)	Turkey	89 (36)	9 (3)	2
	Clearing (women)	Turkey	85 (31)	15 (2)	—
Walker[226]	Radiographic	Southern China	72	28	—
da Costa et al[52]	Clearing	Brazil	90.5	9.5	—
Pattanshetti et al[153]	Clinical	Kuwait	95.5 (40)	4.5	—

*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.

‡Three canals reconfigured to form two.

§Three canals reconfigured to form one.

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 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. 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. Distal extension must allow straight-line access to the distal canal(s). The buccal wall forms a straight connection between the MB and DB orifices, and the lingual wall connects the ML and DL orifices without bowing.

A variation in root morphology is the presence of an extra distolingual root.[195] Usually this root has a type I canal configuration. It was found that two thirds of the first mandibular molars of a Chinese population possessed this variation.[250] Similarly, this distolingual root occurred in 4% of mandibular first molars of a Kuwait population.[154] These results confirm the observation that East Asian populations have more three-rooted mandibular first molars than other racial groups.[7]

Mandibular molars, mainly first molars, may also have an additional root located lingually or buccally. Although this is a rare occurrence in white populations it is more common in Asian populations.[212] The radix entomolaris (RE) is a supernumerary root located distolingually in mandibular molars, 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 disto- to mesiolingually from the main canal or canals of the distal root; the orifice of the RP is located mesio- 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 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, have a coronal curvature, or can have separate coronal and apical curvatures.[55]

Mandibular First Molar—cont'd

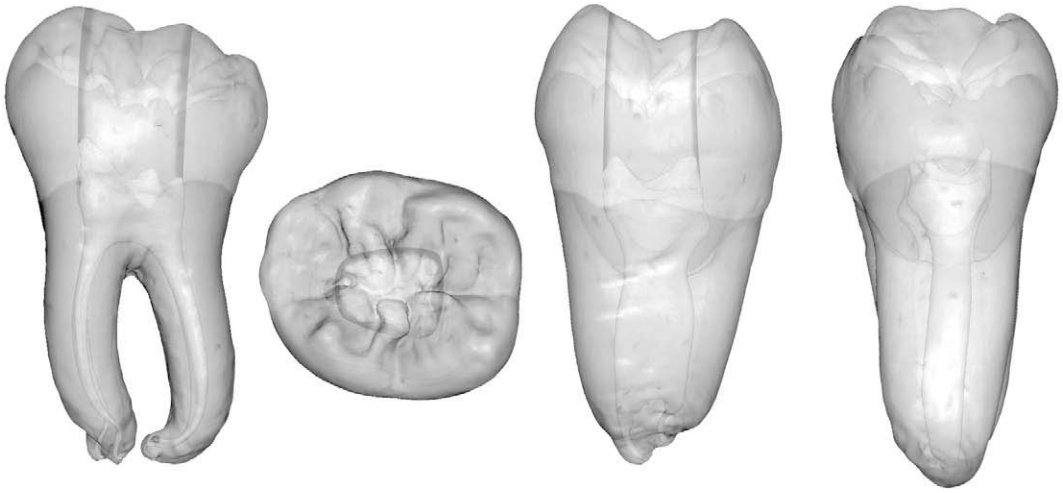


FIG. 7-139 Mandibular first molar. Average time of eruption, 6 years; average age of calcification, 9 to 10 years; average length, 21 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal.

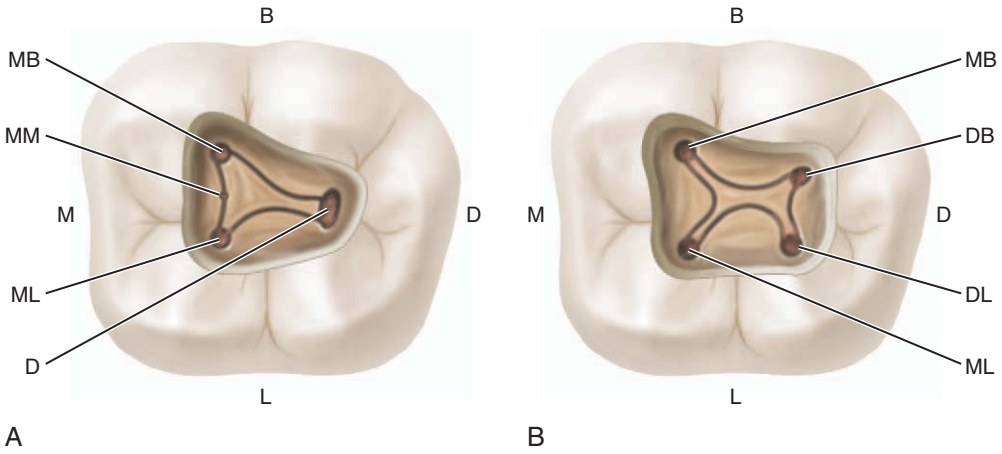


FIG. 7-140 Access cavities for the mandibular first molar. **A,** Three mesial canal orifices and one distal canal orifice. **B,** Two mesial and two distal canal orifices. *B,* Buccal; *D,* distal, distal orifice; *DB,* distobuccal orifice; *DL,* distolingual orifice; *L,* labial; *M,* mesial; *MB,* mesiobuccal orifice; *ML,* mesiolingual orifice; *MM,* middle mesial orifice.

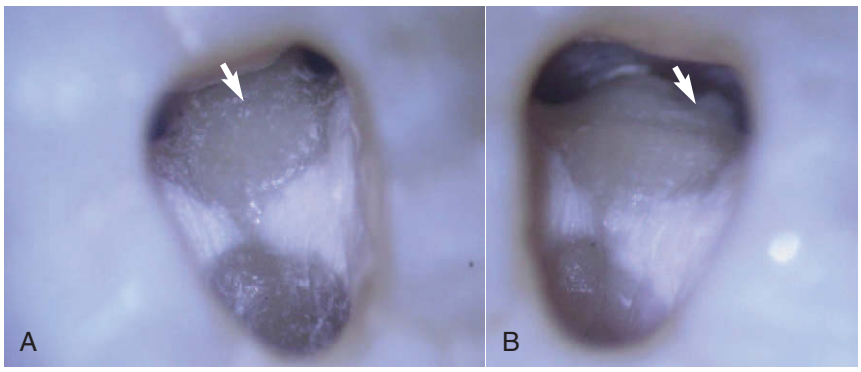


FIG. 7-141 Access cavity for a mandibular first molar as viewed through the dental operating microscope. **A,** Three canal orifices (*MB, ML,* and *D*) and a dentinal projection (*arrow*) can be seen. (×5.1.) A possible middle mesial (*MM*) orifice may be present along the mesial groove. **B,** Troughing of the mesial groove allows identification of the MM canal orifice (*arrow*). (×5.1.)

Continued

Mandibular First Molar—cont'd



FIG. 7-142 Two mesial and two distal canals.

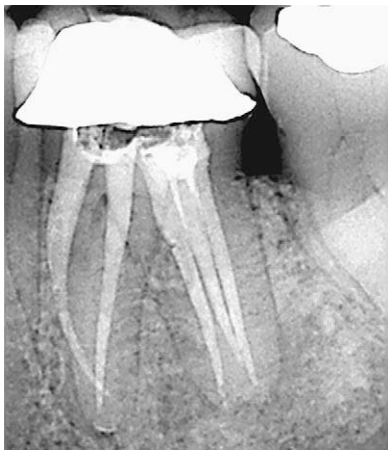


FIG. 7-143 Three canals in distal root. *(Courtesy Dr. Raed Kasem, Clearwater, FL.)*



FIG. 7-144 Three mesial canals.



FIG. 7-145 Three orifices in the mesial root and three orifices in the distal root. *(Courtesy Dr. Haider AlZubaidi, Ocala, FL.)*

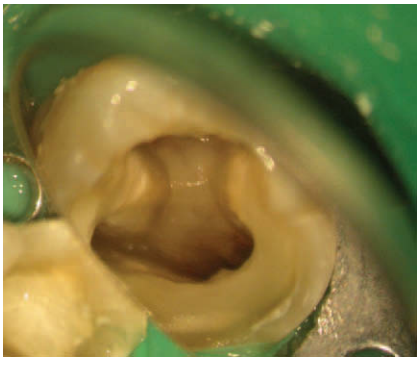


FIG. 7-146 Radix entomolaris. Notice lingual position of its orifice in relation to the two canals in the distal root. *(Courtesy Dr. William J. Aippersbach, Venice, FL.)*

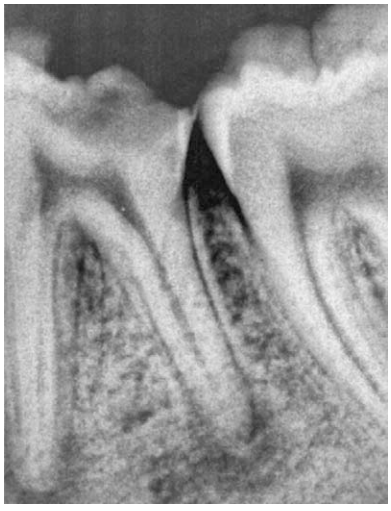


FIG. 7-147 Pretreatment radiograph. Notice position and appearance of the distolingual root. *(Courtesy Dr. William J. Aippersbach, Venice, FL.)*

Mandibular Second Molar

The mandibular second molar is somewhat smaller coronally than the first molar and tends to be more symmetrical. 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.[48]

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 (Table 7-26; Figs. 7-148 through 7-153). 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[134] (see Fig. 7-144, A) that often occurs in this tooth. The distal aspect of the mesial root and the mesial aspect of the distal root have concavities, which must be evaluated during cleaning and shaping procedures. The apices of this tooth often are close to the mandibular canal; therefore the clinician must take care not to allow instruments or filling material to invade this space, because paresthesia may result.

TABLE 7-26

Studies of Apical Canal Configurations of the Mandibular Second Molar

Author(s)	Type of Study	Country	Root	One Canal (%)*	Two Canals (%)†	Three Canals (%)	Other (%)
Vertucci[215]	Clearing	USA	Mesial	65 (38)	35 (9)	—	—
Pineda and Kuttler[163]	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[236]	Radiographic with file	USA	Mesial	56 (52)	40	—	1.3 (one canal); 2.7 (C shaped)[128]
Manning[127]	Clearing	Australia	Mesial	73.5 (32)	24.5 (10)	2	—
Wells and Bernier[240]	Clinical	USA	Mesial	Case report	—	—	—
Beatty and Krell[22]	Clinical	USA	Mesial	—	—	Case report	—
Pomeranz et al[165]	Clinical	USA	Mesial	—	2.6 (3 → 2)§	2.6	—
Sert and Bayirli[184]	Clearing (men)	Turkey	Mesial	70 (56)	30 (7)	—	
	Clearing (women)	Turkey	Mesial	61 (50)	39 (6)	—	
Vertucci[215]	Clearing	USA	Distal	95 (3)	4 (1)	—	
Pineda and Kuttler[163]	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[236]	Radiographic with file	USA	Distal	94.6 (9)	1.3	1.3 (one canal); 2.7 C shaped	
Manning[127]	Clearing	Australia	Distal	98.3	(1.7)	—	
Beatty and Krell[22]	Clinical	USA	Distal	—	Case report	—	
Wells and Bernier[240]	Clinical	USA	Distal	Case report (M and D join)	—	—	
Sert and Bayirli[184]	Clearing (men)	Turkey	Distal	96 (18)	4 (2)	—	
	Clearing (women)	Turkey	Distal	93 (19)	7 (5)	—	

M, Medial; *D,* distal.

*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.

‡Studied incidence in middle mesial canals only.

§Three canals reconfigured to form two.

Continued

Mandibular Second Molar—cont'd

Mandibular second molars may have one to six canals, although the most prevalent configurations are two, three, and four canals (see Fig. 7-149). 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.

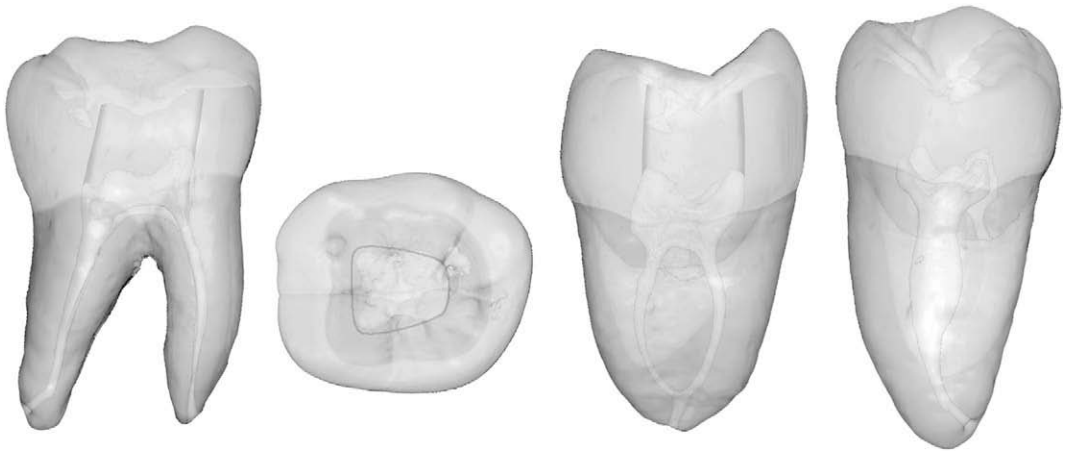


FIG. 7-148 Mandibular second molar. Average time of eruption, 11 to 13 years; average age of calcification, 14 to 15 years; average length, 19.8 mm. Root curvature (most common to least common): mesial root—distal, straight; distal root—straight, distal, mesial, buccal; single root—straight, distal, bayonet, lingual.

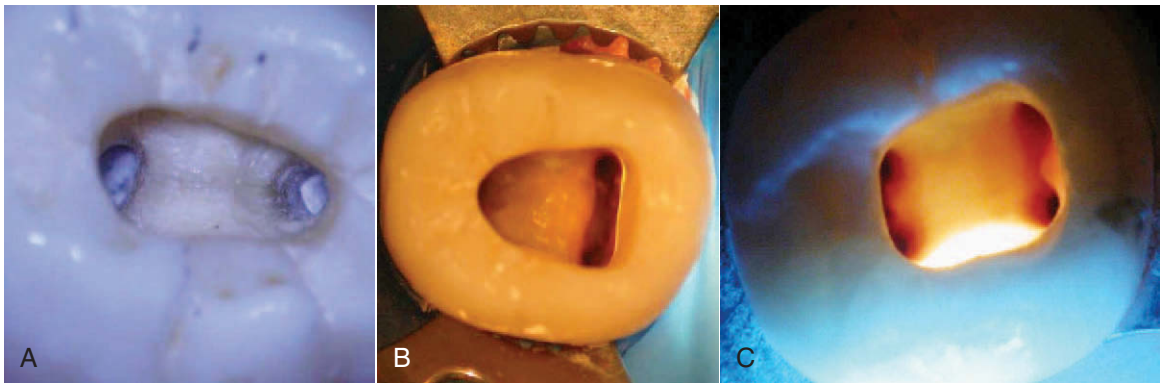


FIG. 7-149 Access cavity for a mandibular second molar as viewed through the dental operating microscope. **A,** Two canal orifices (M and D). (×5.1.) **B,** Three canal orifices (MB, ML, and D). (×3.4 magnification with cervical fiberoptic transillumination.) **C,** Four canal orifices identified (MB, ML, DB, and DL). (×5.1 magnification with cervical fiberoptic transillumination.)

Mandibular Second Molar—cont'd

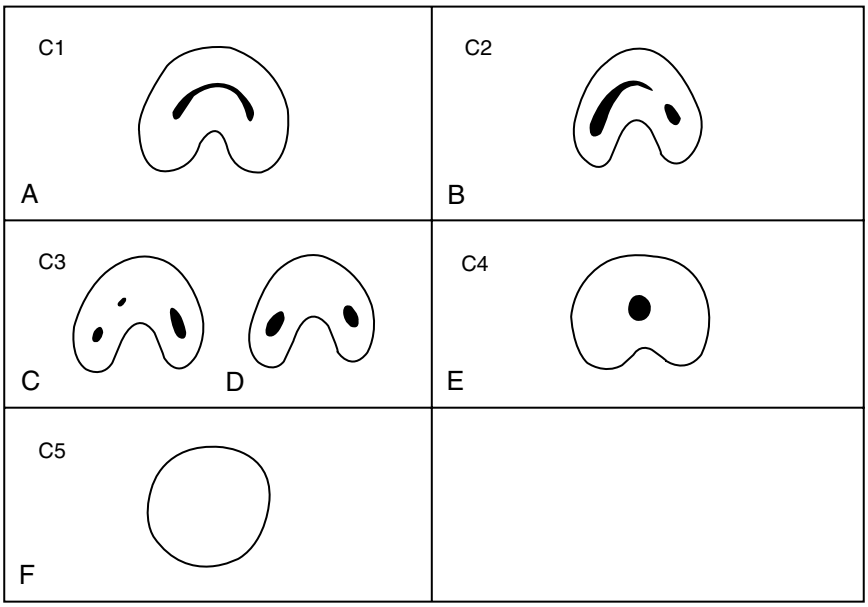


FIG. 7-150 Classification of C-shaped canal configuration. *(From Fan B, Cheung G, Fan M, Gutmann J:* J Endod *30:899, 2004.)*

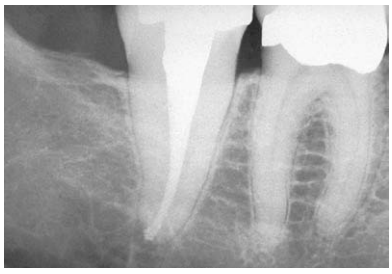


FIG. 7-151 Anastomosis of all canals into one.



FIG. 7-152 Two canals with an accessory canal at the distal root apex.



FIG. 7-153 Fusion of mesial canals at the apex.

Mandibular Third Molar

The mandibular third molar is anatomically unpredictable and must be evaluated on the basis of its root formation. 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 (Table 7-27; Figs. 7-154 through 7-157). C-shaped canals also can occur. Most of these teeth can be successfully treated endodontically 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.

Continued

Mandibular Third Molar—cont'd

TABLE 7-27

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[186]	Clearing	USA	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[163]	Radiographic	Mexico	Mesial		83.5 (18*)	16.5 (3†)	—	—	—	—
			Distal		95.7 (4*)	4.3(2†)	—	—	—	—
Plotino[164]	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.

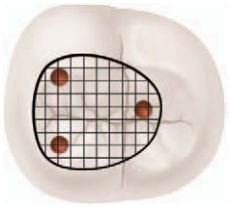


FIG. 7-154 Mandibular third molar. Average time of eruption, 17 to 21 years; average age of calcification, 18 to 25 years; average length: 18.5 mm.



FIG. 7-156 Complex root anatomy. *(Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)*



FIG. 7-155 Five canals. *(Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)*

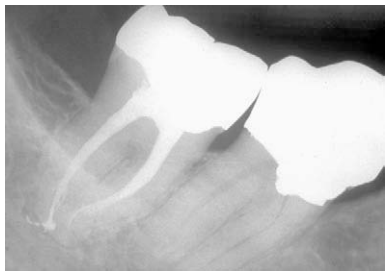


FIG. 7-157 Complex apical anatomy.

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. The C-shaped canal system can assume many variations in its morphology. The original classification[133] has been modified and has produced a more detailed description of C-shaped root and canal morphology. The "C" shape 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.[64]

Category I (C1): The shape is an uninterrupted "C" with no separation or division (see Fig. 7-150, *A*).

Category II (C2): The canal shape resembles a semicolon resulting from a discontinuation of the "C" outline (see Fig. 7-150, *B*), but either angle α or β should be no less than 60 degrees.

Category III (C3): Two or three separate canals (see Fig. 7-150, *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. 7-150, *E*).

Category V (C5): No canal lumen can be observed (is usually seen near the apex only) (see Fig. 7-150, *F*).

The C-shaped root canal system was first reported in 1979.[46] Most C-shaped canals occur in the mandibular second molar (Figs. 7-158 through 7-166), but they also have been reported in the mandibular first molar, the maxillary first and second molars, and the mandibular first premolar. Two investigators reported the first case of a C-shaped canal in a maxillary molar.[146] 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. 7-159, *B*).[34] Investigators who examined 309 Chinese maxillary second molars found C-shaped root canals in 4.9%.[248]

C-shaped mandibular molars are so named because of the cross-sectional morphology of their fused roots and their root 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. 7-159, *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, ribbonlike, 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.[46] Other investigators determined that C-shaped canals in mandibular second molars can vary in shape and number along the root length.[133] This makes cleaning, shaping, obturation, and restoration of these teeth difficult.



FIG. 7-158 C-shaped canal anatomy: one continuous canal from pulp chamber floor to apex.

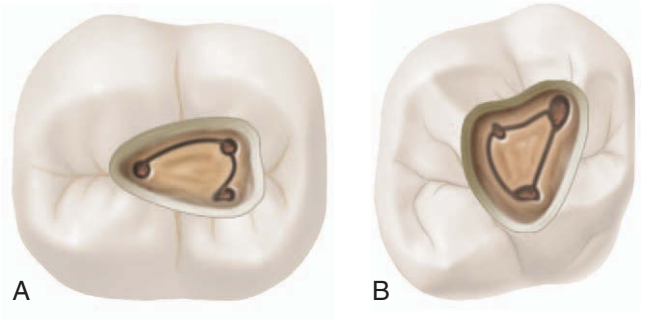


FIG. 7-159 C-shaped canal anatomy. **A,** Mandibular second molar. **B,** Maxillary first molar.

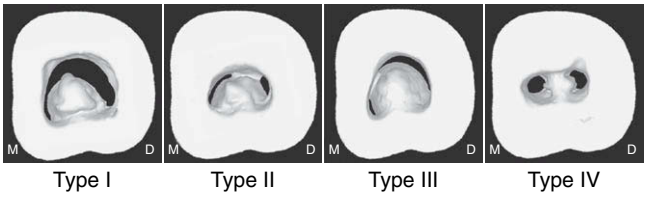


FIG. 7-160 Types of pulpal floor. *M,* Mesial side; *D,* distal side. *(From Min Y, Fan B, Cheung G, Gutmann J, Fan M:* J Endod *32, 1155, 2006.)*

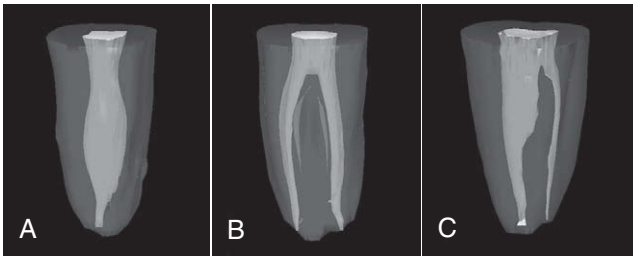


FIG. 7-161 Three-dimensional classification of C-shaped canal configuration. **A,** Merging type; **B,** symmetrical type; **C,** asymmetrical type. *(From Gao Y, Fan B, Cheung G, Gutmann J, Fan M:* J Endod *32, 1062, 2006.)*

Continued

Teeth With C-Shaped Root Canal Systems—cont'd

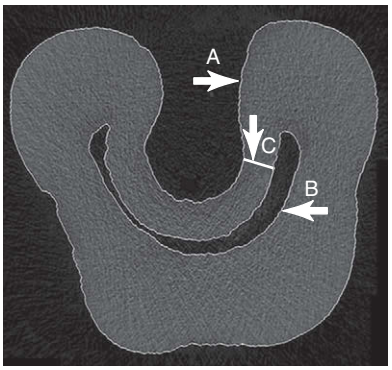


FIG. 7-162 Microcomputed tomographic image of the minimal wall thickness measurement. **A,** The outer root surface; **B,** the inner canal wall; **C,** the thinnest wall thickness. *(From Gao Y, Fan B, Cheung G, Gutmann J, Fan M:* J Endod *32, 1062, 2006.)*

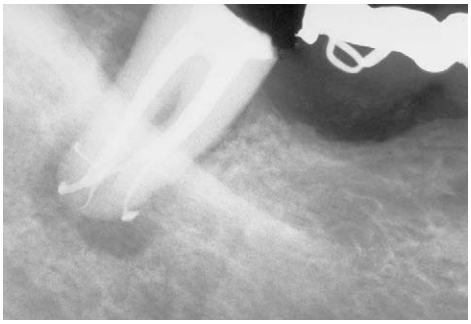


FIG. 7-163 Mandibular second molar with multiple foramina.

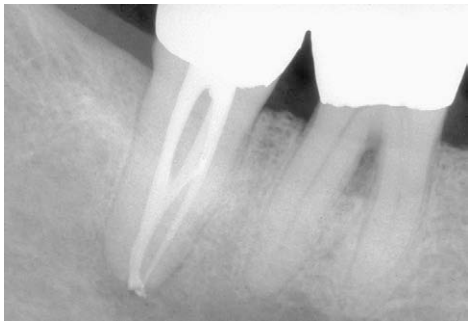


FIG. 7-164 Mandibular second molar with interconnecting canal anatomy.

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



FIG. 7-165 Pretreatment appearance of a mandibular first molar with a C-shaped canal.



FIG. 7-166 Root canal obturation showing the ribbonlike canal space. *(Courtesy Dr. Paulo Nogueira, São Paulo, Brazil.)*

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.[233] Several variants of the C-shaped canal morphology were noted, the most common being two or three canals that merged and exited as one canal.[233]

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 white population. Investigators in Japan[107] and China[247] 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.[247] Another study found a 19% rate in Lebanese subjects[83] and 11% of mandibular second molars from a Saudi Arabian population.[5] 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.[183]

Four types of pulpal floors were found in mandibular second molars:

Teeth With C-Shaped Root Canal Systems—cont'd

Type I: A peninsula-like floor with continuous C-shaped orifice (see Fig. 7-160)

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. 7-160)

Type III: Only one mesial, striplike dentin connection exists 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. 7-160)

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. 7-160)

Not all C-shaped mandibular second molars with C-shaped canal systems have a C-shaped pulpal floor. This makes diagnosis difficult. However, it was determined radiographically that a tooth with a C-shaped canal system always has a fused root with a longitudinal groove in the middle of the root.[65] It was also found that there are three types of C-shaped canal systems in mandibular second molars (see Fig. 7-161).[72]

Type I (merging type): Canals merge to one main canal before exiting at the apical foramen

Type II (symmetrical type): Separated mesial and distal canals in each root, which exit as separate canals

Type III (asymmetrical 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. 7-162).[72]

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.[38]

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; however, use of the DOM, sonic and ultrasonic instrumentation, and plasticized obturation techniques have made treatment successful.

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CHAPTER

8

Instruments, Materials, and Devices

ZVI METZGER, BETTINA BASRANI, and HAROLD E. GOODIS

CHAPTER OUTLINE

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- Assessment of Cracks and Fractures
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- Sealers and Cements
- Standards and Properties

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- Injection Techniques
- Rotary Techniques

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- Erbium-Doped Yttrium Aluminum Garnet Lasers
- Diode Lasers
- Erbium/Chromium-Doped Yttrium Scandium Gallium Garnet Lasers

The past decades have seen many changes in the practice of endodontics, such as in materials, techniques, equipment, instrument design, and the types of metals used to manufacture endodontic instruments. However, the goals of nonsurgical endodontic therapy has not changed: "Root canal systems must be cleaned and shaped: cleaned of their organic remnants and shaped to receive a three-dimensional hermetic (fluid-tight seal) filling of the entire root canal space."[332]

DIAGNOSTIC MATERIALS AND DEVICES

Assessment of Pulp Sensibility

One of the greatest diagnostic challenges in clinical practice is the accurate assessment of pulp status. A variety of pulp testing approaches exist, and there may be confusion as to their validity or appropriateness in different clinical situations. When tests are evaluated, there are some terms that may lead to more definitive and possibly quantifiable endodontic diagnoses. These terms are *sensitivity,* the ability of a test to identify teeth with disease; *specificity,* the ability of a test to identify teeth without disease; and *predictive value,* the ability of a test to foretell what the diagnosis actually is.

Pulp stimulation with cold or heat is the oldest method of evaluating the pulp's health and its ability to respond to external stimulation, but evaluation of pulpal response must not be confused with vitality testing, which requires assessment of pulpal circulation.[140]

Cold Tests

Cold thermal testing causes contraction of the dentinal fluid within the dentinal tubules, resulting in a rapid outward flow of fluid within the patent tubules. This rapid movement of dentinal fluid results in hydrodynamic forces acting on the Aδ nerve fibers within the pulp-dentin complex, leading to a sharp

The authors acknowledge the excellent scholarship of Drs. Van Himel and John McSpadden in authoring previous editions of this chapter.

sensation lasting for the duration of the thermal test. A variety of cold tests may be employed; the major difference between them is the degree of cold applied to the tooth.[140]

A simple means of applying a cold stimulus to a tooth is to wrap a sliver of ice in wet gauze and place it against the buccal surface, comparing the reaction between the test tooth and a control tooth. Pencils of ice can be made by filling a plastic straw with water and freezing it in an upright position in a freezer.[352]

Ethyl chloride (−41° C) may be sprayed onto a cotton pledget, resulting in the formation of ice crystals (Fig. 8-1) prior to application to the tooth. Dichlorodifluoromethane (DDM) (−50° C) is a compressed refrigerant spray that can also be sprayed onto a cotton pledget for cold testing (Fig. 8-2). More recently, ozone-friendly non-chlorofluorocarbon sprays have been introduced in certain countries.

Another effective cold stimulus is frozen carbon dioxide (CO_2), also known as *dry ice* or *carbon dioxide snow* (−78.5° C). For testing purposes, a solid stick of CO_2 is prepared by delivering CO_2 into a custom-made plastic cylinder, and the stick is applied to the buccal surface of the tooth. This method is particularly effective when trying to assess teeth that have been restored with full-coverage metal restorations.[397] Rickoff et al.[314] reported that CO_2 snow applied to a tooth for 5 minutes did not jeopardize the health of the pulp, nor does it damage the surface of the enamel.[300] On the other hand, CO_2 may cause pitting of the surface of porcelain restorations when applied for as little as 5 seconds.[203] When testing with a cold stimulus, the advantage of refrigerant sprays and frozen carbon dioxide is that the cold stimulus does not go into a liquid phase but rather a gas phase. This allows testing to be specific to one tooth at a time, without cold water from melted ice stimulating adjacent teeth.



FIG. 8-1 Ethyl chloride sprayed onto a cotton pledget, resulting in the formation of ice crystals. *(Courtesy Dr. R. Portigliatti.)*



FIG. 8-2 Hygenic Endo-Ice. *(Courtesy Coltène/Whaledent, Cuyahoga Falls, OH.)*

Ice-cold water is another useful and inexpensive test. The tooth being tested should be isolated with a rubber dam and then bathed with water from a syringe. Cold tests should be applied until the patient definitely responds or the stimulus has been applied for a maximum of 15 seconds.[121] One of the significant benefits of ice-water testing is that it can be used on any tooth regardless of the type of restoration that may be present. Overall, cold tests appear to be more reliable than heat tests,[93,341] and there is a general consensus that the colder the stimulus, the more effective the assessment of tooth innervation status.

Heat Test

Warm thermal testing is not employed as often as cold testing because most patients are more sensitive to cold stimuli, and warm thermal tests have been traditionally more difficult to perform. Heat testing can be undertaken using a stick of heated gutta-percha or hot water. A gutta-percha stick, preferably base-plate gutta-percha, is heated with a naked flame or an electric heater until it becomes soft and glistens.[149] It is then applied to the Vaseline-coated surface of the test tooth. It is purported that a tooth surface temperature as high as 150° C can be achieved with this technique[319]: gutta-percha softens at 65° C and may be heated in delivery devices up to 200° C. This test may be difficult to use on posterior teeth because of limited access. A further disadvantage is that excessive heating may result in pulp damage.[280] Prolonged heat application will result in biphasic stimulation of Aδ fibers initially, followed by the pulpal C fibers. Activation of C fibers may result in a lingering pain, therefore heat tests should be applied for no more than 5 seconds. However, inadequate heating of the gutta-percha stick could result in the stimulus being too weak to elicit a response from the pulp.[140]

The use of hot water, administered through an irrigating syringe under rubber dam isolation, has also been described as a means of thermal testing. Frictional heat may be generated by using a rubber cup intended for prophylaxis (without paste) against the buccal aspect of a tooth. The normal use of thermal tests on teeth has been shown not to be harmful to healthy pulp tissue.[170] The System B (SybronEndo, Orange, CA) allows dentists to set specific temperatures for warm thermal testing. After the surface of the tooth has been lubricated, a test tip can be attached to the handle of the System B and the temperature set at 150° F. The preheated tip is placed on the surface of the tooth, and the patient's response is evaluated.

One of the problems of testing with heat is that often a nonvital tooth increases in pain secondary to heat stimuli. In

the clinical situation whereby a patient reports pain to heat and relief with cold stimuli, the application of cold to individually selected teeth may eliminate the patient's tooth pain. Therefore, in these situations, the application of cold with relief of pain can be diagnostic for a nonvital pulp.

Electric Pulp Test

The objective of the electric pulp test (EPT) is to stimulate intact Aδ nerves in the pulpal-dentin complex by applying an electric current to the tooth surface. A positive result stems from an ionic shift in the dentinal fluid within the tubules, causing local depolarization and subsequent generation of an action potential from intact Aδ nerves.[292]

The electric pulp tester is a battery-operated instrument connected to a probe that is applied to the tooth under investigation. It functions by producing a pulsating electrical stimulus, the initial intensity of which should be set at a very low value to prevent excessive stimulation and discomfort. The intensity of the electric stimulus is then increased steadily at a preselected rate, and a note is made of the readout on the digital display when the patient acknowledges a warm or tingling sensation. The readout is not a quantitative measurement of pulp health; it simply provides evidence that the Aδ fibers are sufficiently healthy to function.[140]

Bipolar and monopolar are the two stimulating modes available. The bipolar mode is presumably more accurate because the current is confined to the coronal pulp, but most EPTs are still monopolar.[352]

The requirements of an EPT are an adequate stimulus, an appropriate application method, and careful interpretation of results (Fig. 8-3). Tooth isolation during EPT is essential. Drying the enamel, placement of an interproximal plastic strip, and use of a rubber dam can prevent the spread of electrical impulses to adjacent teeth or gingival tissue. Electric current can also be transferred between adjacent teeth through contacting metallic restorations.[64] A conducting medium should also be used to ensure that maximum current passes from the electrode to the tooth surface.[261] A laboratory study[247] concluded that the interface medium made no appreciable difference to either the voltage or the electric current transmitted. However, a more recent study did demonstrate that different media influence the responses gained from electric testing.[262]



FIG. 8-3 Electronic pulp tester. *(Courtesy SybronEndo, Orange, CA.)*

There are several considerations regarding optimal placement of the tester electrode. The response threshold is reached when an adequate number of nerve terminals are activated to attain what is termed a *summation effect*.[281] An area of high neural density should have a relatively fast and strong response and requires the least electric current. Therefore, the most desirable area of assessment in incisor teeth is at the incisal edge.[32] The tester should be applied on the tooth surface adjacent to a pulp horn, the area of highest nerve density within the pulp. This position equates to the incisal third region of anterior teeth and the mid-third region of posterior teeth. The threshold for response may be influenced by the thickness of the enamel and dentin overlying the pulp. Thus, the response threshold for healthy teeth may be lowest in incisors, slightly greater in premolars, and greatest in molar teeth. A recent study has revealed that the optimum site for tester electrode placement on molars is on the tip of the mesiobuccal cusp.[225]

Assessment of Pulp Vascularity

Vitality testing requires the measurement of pulpal blood flow. Several devices are used to evaluate circulatory changes rather than the neural integrity of the pulp, and a number of these have been used experimentally to evaluate pulpal health.

Crown Surface Temperature

An investigator[103] performed extensive research in the field of crown surface temperature. This pulp vitality technique is based on the hypothesis that vital teeth are warmer and will rewarm quicker after cooling than nonvital teeth. His experiments concluded that time/temperature curves comparing the warming of vital versus nonvital teeth were diagnostically informative.

Attempts to use color change of cholesteric liquid crystals as a diagnostic tool to measure crown temperature change[173] have also met with some success. And a more recent study using an infrared thermographic camera showed crown temperature patterns of nonvital teeth to be slower to rewarm than those of vital teeth.[320]

Transmitted Light Photoplethysmography

Transmitted light photoplethysmography (TLP) is a noninvasive technique used to monitor pulpal blood flow and has been successfully applied in animal and human studies. It has been suggested that TLP incurs less signal contamination from the periodontal blood flow than is the case for laser Doppler flowmetry (LDF).[227,268]

Laser Doppler Flowmetry

LDF was developed to assess blood flow in microvasculature systems such as the retina, mesentery, renal cortex, and skin.[171] It has recently been used in intact teeth in animals[137,139] and in man.[253,421] LDF utilizes a light beam (helium neon 632.8 nm) which is scattered by moving red blood cells.[421] Newer machines have varied the wavelength between 600 and 700 nm, depending on the light absorption properties of the tissue tested. This light beam undergoes a frequency shift according to the Doppler principle. The backscattered light is picked up by photodetectors and produces a signal that is proportional to the red cell flux (number of cells × average velocity). This can be used as a measure of pulpal blood flow, expressed as a percentage of full-scale deflection at a given gain. The

technique appears objective, noninvasive, and accurate. A growing number of reports indicate it is a method that can easily be adapted to the testing of a tooth and is promising as a pulp vitality tester. It is especially effective in young traumatized teeth with large pulps that do not respond well to other forms of sensitivity testing.[129,130,253,421]

Several studies have reported successful use of LDF to study human pulpal blood flow. The value of this method has been well documented, but its high cost and difficulty of use in clinical situations have prevented widespread use.

Pulse Oximetry

Pulse oximetry has been used to detect vascular integrity in the tooth.[335] The process measures oxygen saturation in external soft tissue. It has been suggested that it is capable of passing its signal through enamel and dentin. It may also be used on natural tooth structure. The device, as LDF, utilizes a probe that transmits red (640 mm) and infrared light through the tissue (received by a photodetector). Since oxygenated and deoxygenated hemoglobin absorb different amounts of each light, pulsating changes in blood volume cause periodic changes in the amount of light absorbed by the vascular bed before reaching the photodetector. To date, it has not been considered capable of judging pulp vitality in a manner that would allow the proper degree of sensitivity and specificity. It must again be stressed that LDF and pulse oximetry can only be used on natural tooth structure, never on restorations. When used clinically and having a possible response, a rubber dam should isolate the test teeth, and the gingival tissue below the dam should be blocked with an opaque substance (tin foil).

Xenon-133 Radioisotopes

Radioactive materials for measurement of pulpal blood circulation were previously used in the radiolabeled microsphere injection method.[191,192,396] A method utilizing a radiation probe with xenon-133 (^{133}Xe) radioisotope to differentiate between vital and pulpless teeth on the basis of blood supply has been found effective.[396] However, the use of radioactive materials is expensive, restricted on humans, and requires special licensing requirements. To this point, the most promising of these experimental methods are those using the measurement of light passing through or deflected from the blood in the pulp.

Dual Wavelength Spectrophotometry

Oximetry by spectrophotometry uses a dual wavelength light source (760 and 850 nm) to determine the oxygen saturation level of the pulpal blood supply. Preliminary tests on teeth were promising, since the blood oxygenation and volume could be detected.[284] The instrument might be useful not only in determining pulp necrosis but also the inflammatory status of the pulp. It shows promise as a pulp tester because it is noninvasive, objective, small, and portable.

Doppler techniques, pulse oximetry, and photoplethysmography are all used in medicine and in dental research. However, they have been less successfully applied to routine endodontic care, because the circulatory system of the pulp is encased in a rigid structure and therefore is difficult to study without the removal of hard tissue. Consequently, the need for an absolute rigid observation point in the Doppler technique and the interference of extrapulpal circulatory systems in pulse oximetry and photoplethysmography have limited the introduction of these methods to endodontic practice.

Assessment of Cracks and Fractures

Magnification

The introduction of the operative microscope marked outstanding progress in dentistry. Magnification improves the performance of clinical procedures, and the operating microscope has dramatically improved endodontic nonsurgical treatment and periradicular surgery. The use of the operating microscope and microsurgical instrumentation have resulted in greater precision in surgical procedures, less operative trauma, improved healing, better esthetic results, and more predictable treatments. Speed, control, comfort, and adjustment are other distinct advantages.

LOUPES

Magnification loupes attached to eyeglasses are relatively inexpensive, and the learning curve for their use is much shorter than for the microscope. However, there are several shortcomings to loupes compared to the operating microscope. Each loupe has a fixed magnification power (typically less than 4×) that cannot be increased or decreased. Most loupes have no integrated light source, although often they are employed with an accompanying headlamp system. There is no way to digitally document viewed images. The focus is adjusted through the movements of the clinician's head, creating postural positions that may not be ergonomic.

OPERATING MICROSCOPES

All microscope lenses are designed and installed in a telescopic system (Fig. 8-4). This means they are already focused to infinity. They send parallel beams of light to each eye, allowing the viewing of objects three dimensionally with an excellent depth impression.

Some characteristics of microscopes are: (1) wide range of magnification, (2) coaxial illumination that allows light to



FIG. 8-4 Zeiss Operating Microscope. (*Courtesy OperatingMicroscopes.com.*)

enter even the deepest areas of examination, (3) easy photographic and video capture documentation, (4) the capacity to make focus adjustments by moving the microscope or its parts, maintaining an ergonomic work position.

The major advantage of using light and magnification is the increased diagnostic potential. The microscope is an excellent tool for detecting cracks, additional canals, perforations, and the like.[252] It is important to clarify the difference between magnification and differentiation. *Magnification* is defined as viewing an object at a greater size; *differentiation* refers to making an object distinct or specialized. The microscope alone is very useful, but sometimes additional adjuncts must be employed for better differentiation. Methylene blue used in conjunction with magnification can add important information, especially for fracture detection. Additionally, some operating microscopes employ colored light sources which help in the differentiation between bone and root surfaces during apicoectomies.

Periodontal Probing

Periodontal probing is an essential part of the endodontic diagnostic process. The tooth should be evaluated interproximally and at every millimeter on both the buccal and lingual surfaces, with emphasis on the convex part of the root. Each furcation should also be explored with a periodontal or furcation probe. Periodontal probing is especially useful in helping to detect vertical root fractures (VRF). If the probe sinks abruptly into an isolated deep, narrow periodontal defect, the level of suspicion for a VRF increases. This is true especially when there is no evidence of periodontal disease associated with other areas of the tooth or adjacent teeth. If a VRF is suspected but cannot be directly visualized, magnification, illumination, and staining can be very helpful.

Elastic, plastic, periodontal probes (Premier, Plymouth Meeting, PA) (Fig. 8-5) have been advocated for better detection of defects associated with VRFs. A periodontal pocket associated with a VRF—in an otherwise periodontally intact tooth—differs from a common periodontal pocket by having a deep, narrow pocket with a small occlusal entrance. The flexibility of the plastic probes allows them to be introduced into pockets that may not permit the insertion of rigid metal probes (Fig. 8-6).

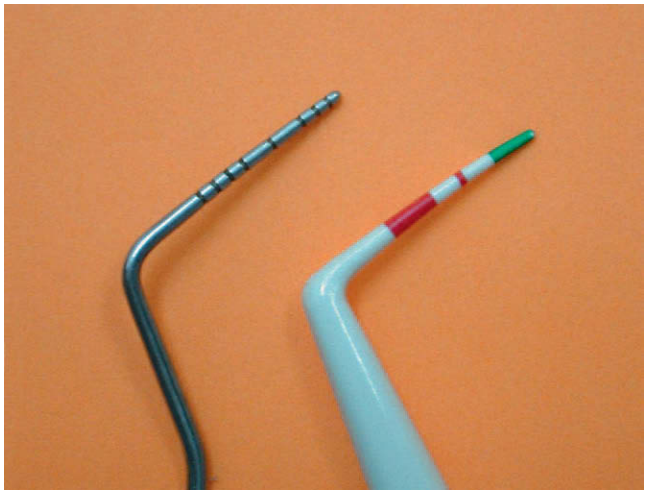


FIG. 8-5 Flexible periodontal probe (Premier, Plymouth Meeting, PA) and a conventional rigid metal probe.

Biting Tests

The Tooth Slooth (SybronEndo) (Fig. 8-7) has proved very useful for the differential diagnosis of various stages of incomplete crown fractures. The design of the device permits a chewing force to be applied selectively to one cusp at a time. When this selective force is applied to a cracked cusp of a vital tooth, the coronal dentin distorts, which is allowed by the crack and results in a painful response. This sensation is not present in other intact cusps of the same tooth. This device can be effective when cotton rolls or wooden sticks are not helpful.

Staining

Dyes can aid greatly in the diagnosis of either caries or fractures. Caries detectors have been used effectively to identify caries not otherwise evident with either visual inspection or traditional hand instruments. Methylene blue dye, India ink or iodine tincture have been very effective dyes used to diagnose the presence of crown or root fractures.[206]

Transillumination

Transillumination is the transmission of light through tissues of the body. A common example is the transmission of light



FIG. 8-6 A Premier periodontal probe in a narrow and deep pocket associated with VRF. Note the blanching that indicates the narrow nature of the pocket.



FIG. 8-7 Tooth Slooth. (*Courtesy Profession Results, Inc., www.toothslooth.com.*)

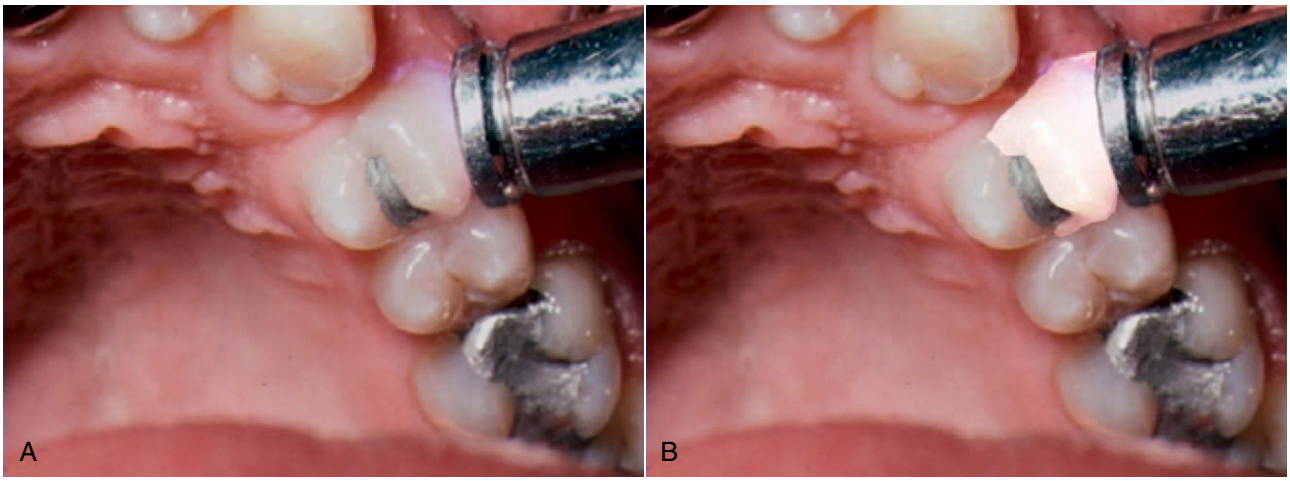


FIG. 8-8 A crack in the crown of a tooth, located by using a transilluminator.

through fingers, producing a red glow due to red blood cells absorbing all other colors of the light. Transillumination is an excellent technique for viewing caries, fractures, narrow root canal orifices, and other clinical entities. It is used by passing an intense light through the side of the tooth and can substantially enhance the clinician's diagnostic and treatment abilities. Healthy tooth structure has an index of light transmission greater than that of caries or calculus. A fracture line or a calcified root canal orifice also have a reduced ability to transmit light. Therefore, if an intense light is applied in direct proximity to a tooth, and other extraneous light is reduced, these clinical entities will appear as distinct dark areas in the otherwise bright structure (Fig. 8-8). The transilluminator tip is placed on either the facial or lingual surface of the tooth or the root, and the area can be viewed from the occlusal surface or the surface opposite the transilluminator. Direct vision or a dental mirror may be used for viewing, depending upon the area under examination. The exact placement of the transilluminator differs for caries, endodontics, fractures, and so forth.

The acceptance of transillumination as a diagnostic technique has been greatly enhanced by the availability of modern devices made expressly for this purpose.[118] These transilluminators use white light–emitting diode (LED) lamps to generate the necessary intense white light. The light is then transmitted to the working area, without heat, using a narrow fiberoptic rod or fiberoptic cables. An important feature is that a fiberoptic rod can be easily removed and autoclaved to avoid cross-contamination between patients. The small size and portability of these new devices enhances the convenient use of transillumination.

Assessment of Images

Radiographs

Radiography is an essential part of endodontic diagnosis. Modern technology is rapidly shifting toward digital filmless imaging and other new image-enhancing techniques (see online Chapter 29). Therefore the clinician must be well versed in this diagnostic field.

The radiographic examination should always be evaluated together with other diagnostic tests and clinical examination. Initial sensitivity tests can suggest which type of radiograph will be most advantageous. If a vital tooth is evaluated, a bitewing radiograph would be advantageous to detect caries or other potential causes of pulpal inflammation. If periapical disease is suspected by the previous tests, a periapical radiograph is indicated. All radiographs should be taken using holders that allow parallelism and standardization. If comparative radiographs will be necessary on a subsequent patient visit, it is useful to fabricate an individual impression material bite-block so that the angulation of follow-up radiographs will be as similar as possible. Extra oral radiographs, like occlusal or PAs, may also be useful in certain cases.

Radiography cannot directly detect pulpal inflammation, but caries or defective restorations seen on the radiograph may suggest pulpal inflammation. Condensing apical periodontitis is a near-pathognomonic sign of longstanding pulpitis. Also, the presence of an apical radiolucency of endodontic origin may be a good indication that necrosis or a necrotic zone is present in the pulp space.

The radiograph is one of the best means at our disposal for determining the existence of a chronic periapical condition. It is characterized by a rather well-defined radiolucent area, or dark shadow, which may or may not be exactly at the apex of the tooth.

Technology has advanced in the area of radiology to include F-speed or Insight conventional film, xeroradiography, digital radiography, subtraction radiography, phosphor images, ultrasound, and recently also cone-beam computed tomography (CBCT). But improvement in our ability to interpret periradicular/periapical pathosis is still debatable with some of these methodologies. Comparing xeroradiography and E-speed conventional film, investigators[22] reported no difference in the detection of periapical bone pathosis.

Another study[399] found that digitally subtracted images were more sensitive for identifying cortical and cancellous bone changes than conventional films. In an in vitro model, other investigators[90] found that subtraction radiography was capable of discriminating between health and disease. Still others[265] found that the progress of chronic apical periodontitis may be followed predictably by their subtraction methodology. Digital subtraction radiography may also be useful in the

evaluation of healing following endodontic therapy.[433] Another investigation[207] concluded that conventional film radiography performed slightly better for the detection of periapical bone lesions than direct digital radiography, and that image processing did not improve the observer performance.

Researchers[296] reported that cortical bone lesions were identified with greater accuracy than trabecular bone lesions, regardless of whether digital radiography or conventional films were used.

Another study[109] demonstrated that there was no significant difference in the accuracy of detecting artificially prepared periapical lesions between Schick CDR and Trophy RVGui DDR systems Their results using digital radiography were in agreement with Seltzer et al's.[341] finding using conventional radiography: artificially produced lesions were not radiographically observed unless the cancellous lesion extended to the junction of the cancellous and cortical bone.

Ultrasound

Coinvestigators[66] have introduced the potential of ultrasound real-time imaging in endodontics and found it able to distinguish between granulomas and cysts. This promising potential awaits further long-term investigations.

Cone-Beam Computed Tomography and Other Scans

CBCT (Fig. 8-9), ultrasound, and other emerging technologies seem to be very promising tools for determining a more accurate diagnosis of periradicular pathoses.[67,153,246,275] The reader is referred to Chapter 29 for further information about CBCT.

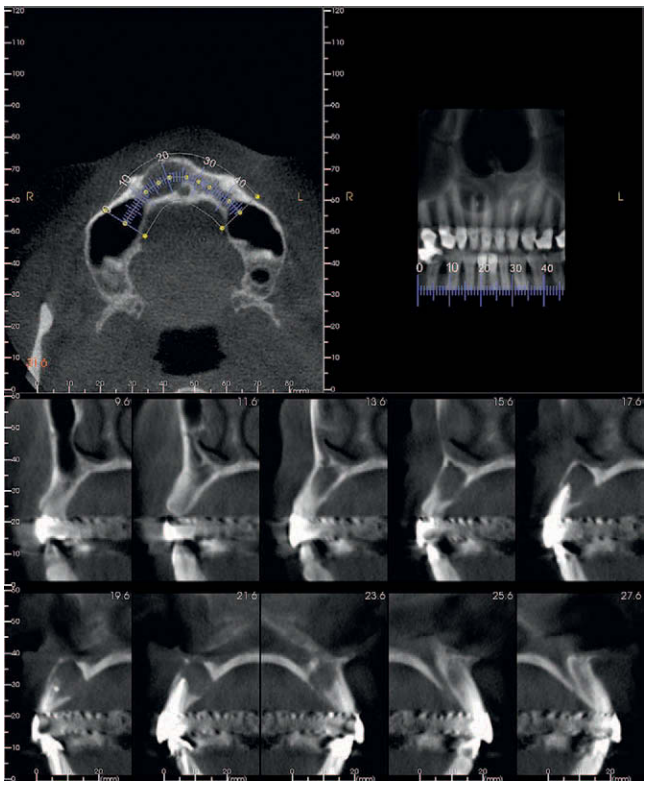


FIG. 8-9 CBCT example: These series of images show an ovoid radiolucent area in the anterior right maxilla. Both teeth 1.1 and 1.2 were endodontically treated; however, given the epicenter of the entity relative to the teeth, the radiolucent area was interpreted to be a keratocystic odontogenic tumor. *(Courtesy Dr. E. Lam.)*

ENDODONTIC INSTRUMENTS

Although most instruments used in general dentistry also can be used for endodontic therapy, some hand instruments are designed specifically for endodontic procedures. In addition, many different types of instruments have been designed for procedures performed inside the pulp space. These include manually operated instruments for root canal preparation, rotary and other engine-driven and energized instruments for root canal preparation, and instruments for root canal obturation.

Standardized specifications have been established to improve instrument quality. For example, the International Standards Organization (ISO) has worked with the Fédération Dentaire Internationale (FDI) through the Technical Committee 106 Joint Working Group (TC-106 JWG-l) 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, pluggers, and spreaders (ANSI No. 71).

Hand Instruments

Traditional dental hand instruments have been modified for endodontic uses. A typical set of endodontic instruments might include a front-surface mouth mirror, a D-5 explorer, a D-16 endodontic explorer, cotton pliers, a spoon excavator, a series of pluggers, a plastic instrument, a hemostat, a periodontal probe, and a ruler. The endodontic explorer has two straight, very sharp ends that are angled in two different directions from the long axis of the instrument.

The mouth mirror should be a front-surface mirror, especially when magnifying loupes or an operating microscope are used. Several types of endodontic spoons are available. These spoons have a much longer offset from the long axis of the instrument (for better reach inside constricted pulp chambers) than regular dental spoons. The spoons are used to remove carious material and to excise pulp tissue; therefore, they should be kept well sharpened (Fig. 8-10). The cotton pliers should preferably be of the locking type that can securely hold gutta-percha cones when transferred from the dental assistant to the dentist's hand. The periodontal probe should be a flexible one (see Fig. 8-5). The exact type and number of instruments usually depend on the techniques used and clinician's preference.

Microinstruments

With the enhanced use of microscopes in endodontics, especially during apical surgery, many specially designed miniaturized instruments have been introduced. These include miniature mirrors, probes and pluggers (for details see Chapter 21).

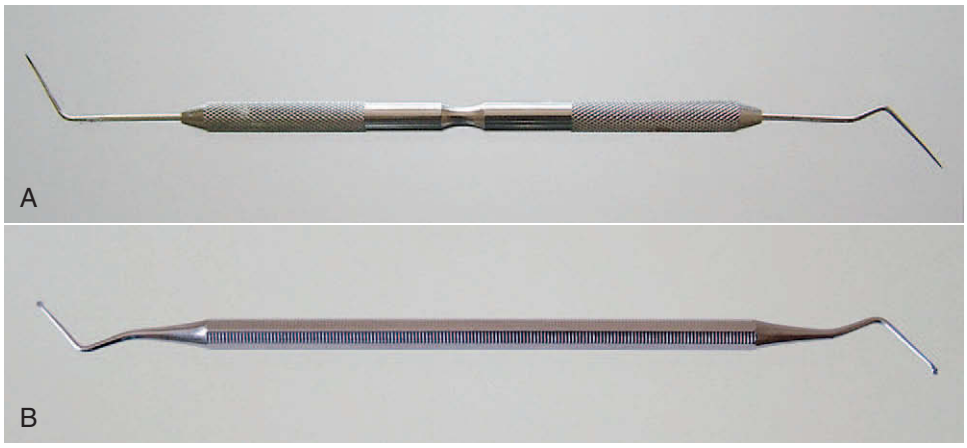


FIG. 8-10 Endodontic explorer **(A)** and endodontic spoon **(B).** *(From Boyd LR:* Dental instruments: a pocket guide, *ed 3, Saunders Elsevier, St Louis, 2009.)*

Instruments for Cleaning and Shaping the Root Canal Space

The purposes of this section is to provide the principles necessary for the clinician to better understand the ideal design for current and future instruments. Most instructional materials mistakenly attempt to teach step-by-step techniques rather than explain the physics of the instruments. However, an increasing number of new products and their advocates have created confusion in the selection process. For this reason, the clinician must understand the basic mechanical, physical, and scientific principles of instrumentation (Box 8-1).

The two primary goals of root canal instrumentation are (1) to provide a biologic environment (infection control) conducive to healing and (2) to develop a canal shape receptive to obturation. Historically, most instruments used to shape the canal were designed to be used by hand. Recently, rotary instrumentation has gained considerable popularity and may be used in conjunction with hand instruments (Box 8-2). The information in the following sections should facilitate the most efficient use of rotary instruments, minimizing the chance of instrument or procedural mishaps and allowing the clinician to achieve ideal treatment results.

Group I: Manually Operated Instruments

Manually operated instruments are all instruments that are generically called *files*. Defining endodontic instruments by function, files are instruments that enlarge canals with reciprocal insertion and withdrawal motions. Reamers cut and enlarge canals with rotational motions. Before using either instrument, the clinician must make sure the canal is patent.

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 originally were manufactured by the same process. 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 (Fig. 8-11). 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 is used for reaming; a three- or four-sided configuration with more spirals is used for filing.

Historically, root canal instruments were manufactured from carbon steel. Subsequently, the use of stainless steel greatly improved the quality of instruments. More recently, the introduction of the nickel-titanium (NiTi) alloy in the manufacture of endodontic instruments has resulted in significant improvements in canal shaping because of its increased flexibility compared to stainless steel (this metal is described later in the chapter).

BOX 8-1

Terminology for the Physical Properties of Instruments

Successful use of an instrument depends on the ways the material, design, and technique relate to the forces exerted on the instrument. The following terms quantify the actions and reactions to these forces.

Stress: The deforming force measured across a given area.

Stress concentration point: An abrupt change in the geometric shape of a file, such as a notch, which results in a higher stress level at that point than along the surface of the file where the shape is more continuous.

Strain: The amount of deformation a file undergoes.

Elastic limit: A set value representing the maximal strain that, when applied to a file, allows the file to return to its original dimensions. After the strain is removed, the residual internal forces return to zero.

Elastic deformation: The reversible deformation that does not exceed the elastic limit.

Shape memory: A condition that exists when the elastic limit is substantially higher than is typical for conventional metals. It allows an instrument to regain its original form after being deformed.

Plastic deformation: Permanent bond displacement, which occurs when the elastic limit is exceeded. The file does not return to its original dimensions after strain is removed.

Plastic limit: The point at which a plastic-deformed file breaks.

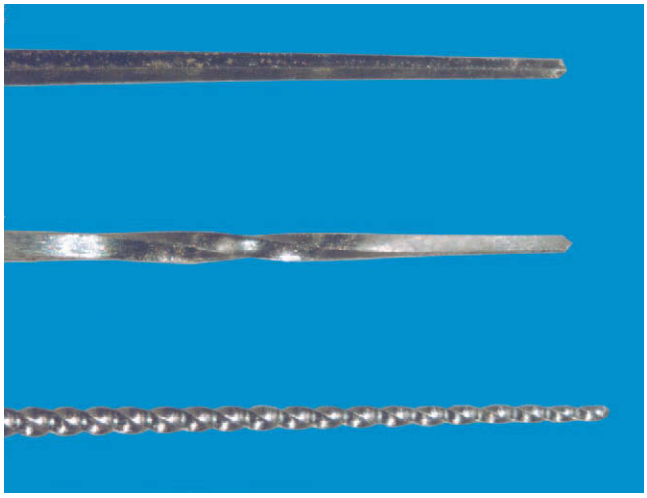


FIG. 8-11 The two ends of a blank, tapered pyramidal wire are stabilized *(top)* and then one end is rotated to create a spiral shape on the file's working surface *(middle)*. Multiple rotations produce the final spiral shape *(bottom)*. *(Courtesy John T. McSpadden, Lookout Mountain, GA.)*

BOX 8-2

Classification of Instruments Used for Cleaning and Shaping the Root Canal Space

Endodontic instruments for root canal preparation can be divided into six groups:

- **Group I:** Manually-operated instruments, such as barbed broaches and K-type and H-type instruments.
- **Group II:** Low-speed instruments with a latch-type attachment. Typical instruments in this group are Gates-Glidden (GG) burs and Peeso reamers. They are typically used in the coronal part of the canal and never used in a canal curvature.
- **Group III:** Engine-driven nickel-titanium rotary instruments. They consist of a rotating blade that can safely be operated in, and adapt itself to, curved root canals. Most engine-driven instruments available today belong to this group.
- **Group IV:** Engine-driven instruments that adapt themselves three-dimensionally to the shape of the root canal. Like other nickel-titanium instruments, they adapt to the shape of the root canal longitudinally but additionally they adapt also to the cross-section of the root canal. There is currently only one instrument in this group: the self-adjusting file (SAF; ReDent-Nova, Raanana, Israel).
- **Group V:** Engine-driven reciprocating instruments.
- **Group VI:** Ultrasonic instruments.

BARBED BROACHES AND RASPS

Dating from the early to mid-19th century, broaches and rasps were the earliest endodontic instruments used to extirpate the pulp and enlarge the canal (Fig. 8-12). Still used today, these instruments are manufactured by hacking a round, tapered wire with a blade to form sharp, projecting barbs that cut or snag tissue. Specifications have been set for both the barbed broach and the rasp (ANSI/ADA standard No. 63, ISO/FDI standard No. 3630/1).

A barbed broach does not cut or machine dentin; this instrument is mostly used to engage and remove soft tissue from the canal. It is also an excellent tool for removing cotton or paper points that have accidentally become lodged in the root canal.

K-TYPE INSTRUMENTS

The K-file and K-reamer are the oldest useful instruments for cutting and machining dentin (Fig. 8-13). They are made from a stainless steel wire that is ground to a tapered square or triangular cross-section and then twisted to create either a file or a reamer. A file has more flutes (see Components of a File) per length unit than a reamer.

K-type instruments are useful for penetrating and enlarging root canals. The instrument works primarily by compression-and-release destruction of the dentin surrounding the canal. Generally, a reaming motion (i.e., constant file rotation) causes less transportation than a filing motion (reciprocating or "watch-winding" file rotation).[406] (*Transportation* is the excessive loss of dentin from the outer wall of a curved canal in the apical segment. This procedural error can lead to perforation of the root canal system or an inability to negotiate the canal space apical to the canal transportation.) A stainless steel K-file can be precurved to a desired form to facilitate insertion and minimize transportation. Permanent deformation occurs when the flutes become wound more tightly or opened more widely (Fig. 8-14). When such deformation occurs, the instrument should no longer be used. Instruments fracture during clockwise motion after plastic deformation.[158,345] This occurs when the instrument becomes bound while the force of rotation continues. Interestingly, although the force required for failure is the same in both directions of rotation,[205,213] 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.

H-TYPE INSTRUMENTS

An H-type instrument has spiral edges arranged to allow cutting only during a pulling stroke (Fig. 8-15). An example is a Hedström file. An H-type instrument is better for cutting than a K-type instrument, because it has a more positive rake angle (see Components of a File) and a blade with a cutting rather than a scraping angle. Bending a Hedström file results in points of greater stress concentration than occurs with K-type instruments. These concentration points can lead to the propagation of cracks and fatigue failure.[158] Clinically, fatigue happens without any external physical signs of stress, such as the flute changes seen in K-type instruments (see Fig. 8-14).

Currently all H-type instruments are ground from a tapered blank. Hedström files are formed by grinding a single continuous flute. Computer-assisted machining technology has allowed the development of H-type instruments with very complex forms. This process, called *multiaxis grinding,* allows adjustment of the rake angle, helix angle, multiple flutes, and tapers. H-files cut the canal wall when pulled or rotated clockwise; the file is relatively ineffective when pushed or rotated counterclockwise. Because the H-file generally has sharper edges than the K-file, it has a tendency to screw into the canal during rotation, particularly if the instrument's blades are nearly parallel. Awareness of screwing-in forces is important for avoiding instrument failure.

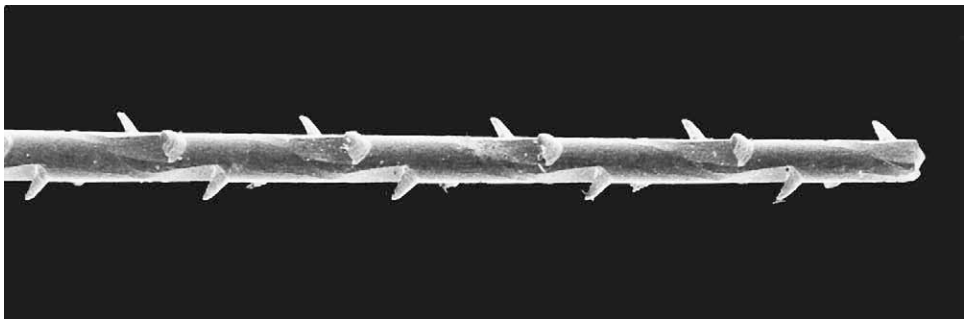


FIG. 8-12 Barbed broach (Union Broach, York, PA).

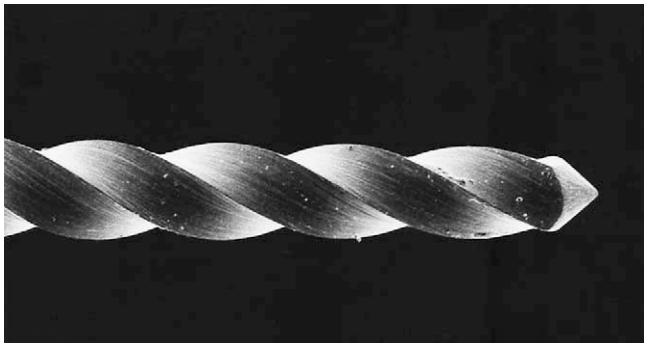


FIG. 8-13 K-file #40 (DENTSPLY Maillefer, Ballaigues, Switzerland).

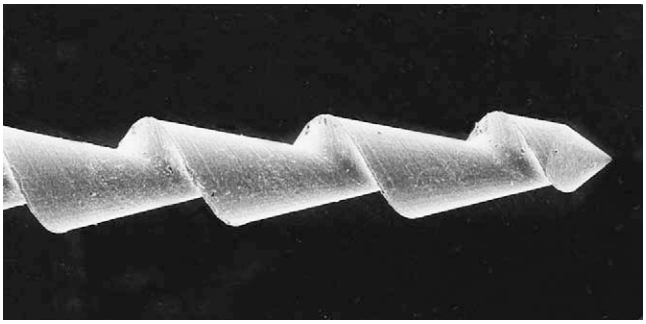


FIG. 8-15 Hedström file #50 (DENTSPLY Maillefer, Ballaigues, Switzerland).

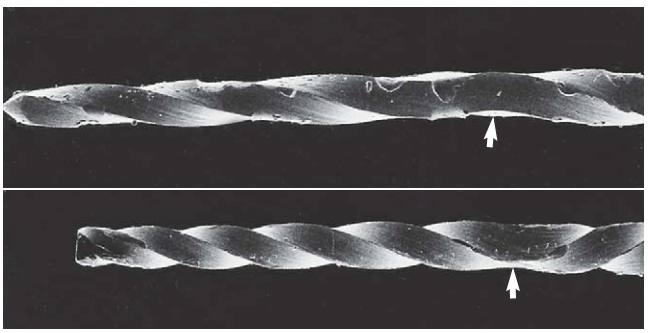


FIG. 8-14 K-files stressed to deformation by clockwise and counterclockwise twisting (*arrow indicates deformed areas*). These instruments are close to fracture and should be discarded.

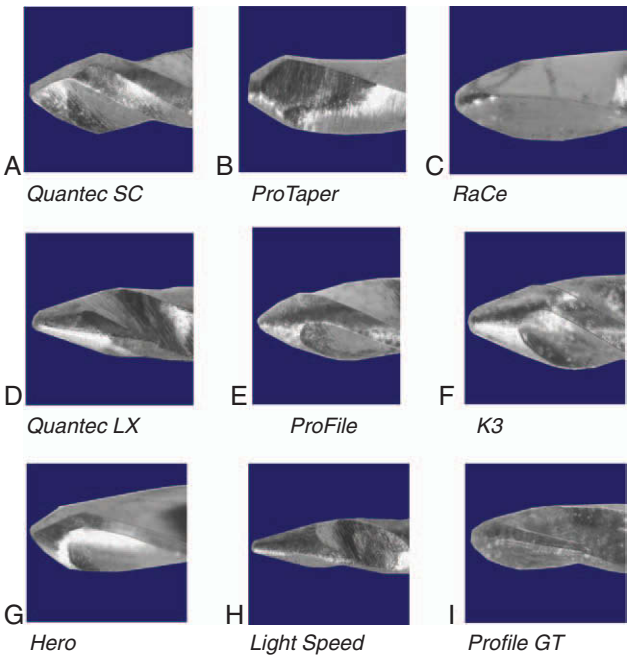


FIG. 8-16 Instrument tips arranged in order from cutting to noncutting. *(Courtesy John T. McSpadden, Lookout Mountain, GA.)*

INSTRUMENT DESIGN MODIFICATIONS

K-files and H-files can be modified into numerous designs. Often the instruments can be improved for more effective instrumentation by changing the geometric dimensions, using computerized multiaxis grinding machines. For example, changing the cross-sectional geometry of a K-type instrument from square to rhomboid enhances the instrument's flexibility and rake angle. However, the possible geometries can complicate adherence to ISO and ANSI standards.

TIP DESIGN

Studies have shown that tip design can affect file control, efficiency, and outcome in the shaping of root canal systems.[266,267] The tip of the original K-file resembled a pyramid (see Fig. 8-13). Instrument tips have been described as *cutting, noncutting,* and *partially cutting,* although no clear distinction exists among the three types (Fig. 8-16).

The instrument tip has two functions: to guide the file through the canal and to enlarge the canal. A clinician who is unfamiliar with the tip design of a particular instrument is apt

to do either of the following: (1) transport the canal (if the tip is capable of enlarging the canal and remains too long in one position) 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). Transportation of the original axis of the canal can occur by remaining too long in a curved canal with a tip that has efficient cutting ability.

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 the file is engaged 360 degrees, canal transportation is unlikely to occur. Only with overuse does the file begin to cut on one side, resulting in transportation. Most instrumentation errors occur when the file tip is loose in the canal, which gives it a propensity to transport the canal.

A good beginner's rule is this: If the canal is smaller than the file, the prudent use of a cutting tip is more efficient. If the canal is larger than the tip, using a less-effective cutting tip can help prevent transportation (see Fig. 8-16). Much has been written about the importance of various sophisticated tip modifications to prevent such ledging, but little scientific evidence exists that any one design is better than another.[190,308,309,316,321]

METAL ALLOYS

The development of nitinol, an equiatomic alloy composed of nickel and titanium,[412] has proved a significant advancement in the manufacture of endodontic instruments. NiTi is called an *exotic metal* because it does not conform to the typical rules of metallurgy. Because it is a superelastic metal, the application of stress does not result in the usual proportional strain seen in other metals such as stainless steel. When stress is initially applied to NiTi, the result is proportional strain, but the strain remains essentially the same as the application of additional stress reaches a specific level, forming what is called a *loading plateau*. Eventually, of course, application of more stress results in more strain, which increases until the file breaks. This unusual property is the result of a molecular crystalline phase transformation. 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 of its unique crystalline structure, a NiTi file has *superelasticity,* or the ability to return to its original shape after being deformed. Simply stated, NiTi alloys currently are the only readily available affordable materials with the flexibility and toughness for routine use as effective rotary endodontic files in curved canals.

Attempts to improve the NiTi alloy continue, and recent reports indicate that new NiTi alloys may be five times more flexible than currently used alloys.[186] Microscopic surface defects are considered a contributing factor for crack propagation and instrument fracture. Therefore, attempts to improve surface characteristics by electropolishing, surface coatings, and surface implantation have been used for this purpose.[216,310]

Group II: Low-Speed Rotary Instruments

Many types of rotary instruments are used during endodontic procedures. In addition to regular burs adapted for endodontics, various types of root canal reamers are used to shape the canal space, to place or remove root canal filling materials, or to prepare a post space.



FIG. 8-17 Gates-Glidden (GG) burs made of stainless steel. *(From Johnson WT:* Color atlas of endodontics, *St Louis, 2002, Saunders.)*

BURS

In addition to conventional burs, burs with extended shanks for low-speed contra-angle handpieces are useful for providing good visibility during deep preparation of the pulp chamber. This is particularly important when using an operating microscope when performing such procedures after access to the pulp chamber has been achieved. Straight-line access to the initial point of curvature traditionally has been accomplished using rotary instruments such as Gates-Glidden burs and Peeso instruments. These reamers are available in a 32-mm length and a 28-mm length for posterior teeth (Figs. 8-17, 8-18). Use of these instruments should be limited to the straight portion of the canal preparation. The risk of perforation with these instruments becomes a real possibility with attempts to instrument beyond the point of curvature or if the instruments are used to cut laterally. The risk of lateral cutting resulting in perforation is lower with Gates-Glidden burs than with the other instruments mentioned. This risk is especially pronounced on the furcation sides of mesial roots of molars. Gates-Glidden instruments are also available in nickel-titanium. The Peeso reamer is used mostly for post space preparation (Fig. 8-19).

Group III: Rotary Instruments for Canal Preparation

COMPONENTS OF A FILE

To make the best use of files, the clinician should be familiar with the parts of each file and understand how variations in design affect instrumentation (Fig. 8-20). 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, and a 0.31 mm diameter 3 mm from the tip. Some manufacturers express the taper in terms of percentage (e.g., a #.02 taper is a 2% taper). Historically, as an

ISO standard, a file was fluted and tapered at 2% for 16 mm, but now files incorporate a wide variation of lengths and tapers of working surfaces. 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.

The *flute* of the file is the groove in the working surface used to collect soft tissue and dentin chips removed from the wall of the canal. The effectiveness of the 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 from the wall of the canal and severs or snags soft tissue. Its effectiveness depends on its angle of incidence and sharpness. If a surface projects axially from the central axis as far as the cutting edge between flutes, this surface is called the *land* (or sometimes the *marginal width*) (Figs. 8-21 and 8-22). The land touches the canal walls at the periphery of the file and reduces the tendency of the file to screw into the canal, reduces transportation of the canal, reduces the propagation of microcracks on its circumference, supports the cutting edge, and limits the depth of cut. Its position relative to the opposing cutting edge and its width determine its effectiveness. 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* (Fig. 8-23). The angle the cutting edge forms with the long axis of the file, called the *helix angle,* augers debris collected in the flute from the canal. This angle is important for determining which file technique to use (see Fig. 8-23; Fig. 8-24).

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. If the angle formed by the leading edge and the surface to be cut (its tangent) is obtuse, the rake angle is said to be *positive* or *cutting*. If the angle formed by the leading edge and the surface to be cut is acute, the rake angle is said to be *negative* or *scraping* (Fig. 8-25). However, the rake angle may not be the same as the *cutting angle*. The cutting angle, or the *effective rake angle,* is a better indication of a file's cutting ability and is determined by measuring the angle formed by the cutting (leading) edge and the radius when the file is sectioned perpendicular to the cutting edge. If the flutes of the file are symmetric, the rake angle and the cutting angle are essentially the same.

The *pitch* of the file is the distance between a point on the leading edge and the corresponding point on the adjacent leading edge, or it may be the distance between corresponding

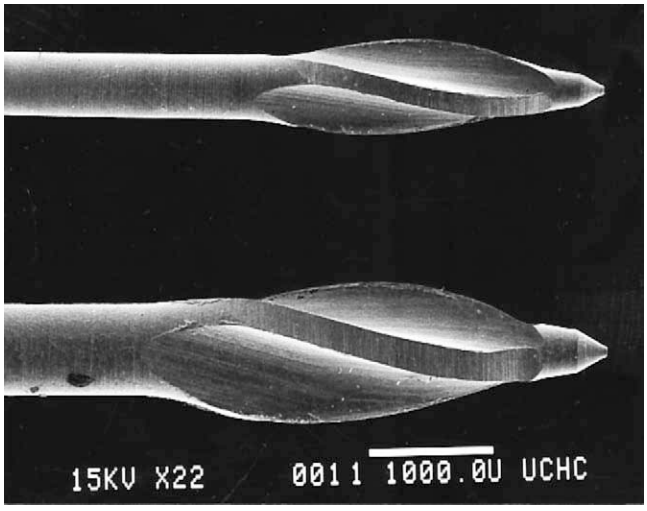


FIG. 8-18 Working part of a Gates-Glidden bur made of stainless steel. Note the rounded safety tip and the lack of sharp cutting edges. The instrument has a marginal land for centering the drill in the canal and to allow safer machining of the canal walls.

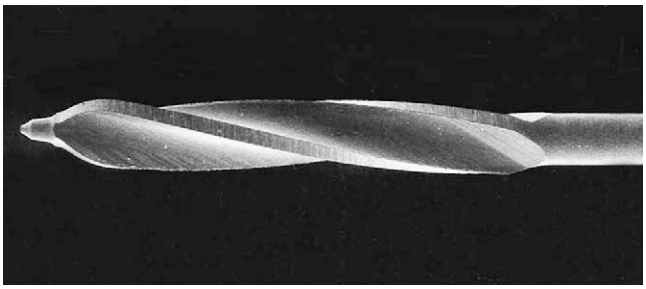


FIG. 8-19 Peeso Reamer (Union Broach, York, PA). Note the safety tip and marginal lands on the machining surfaces.

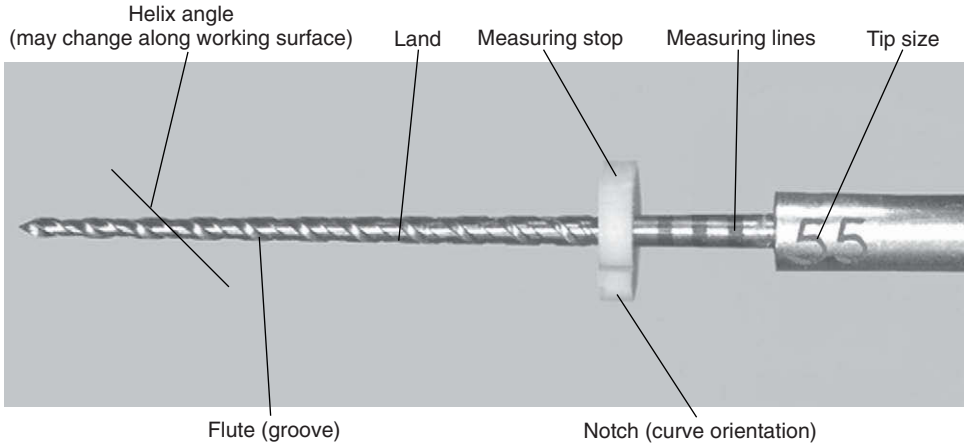


FIG. 8-20 Components of an endodontic rotary instrument. *(Courtesy John T. McSpadden, Lookout Mountain, GA.)*

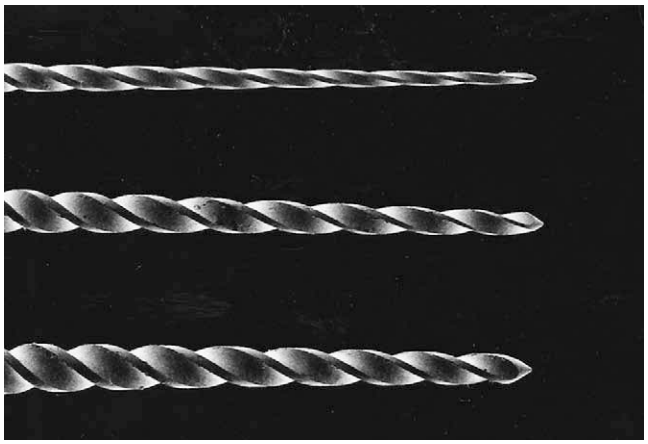


FIG. 8-21 ProFile rotary NiTi instruments, #3, #5, and #6 (DENTSPLY Tulsa Dental, Tulsa, OK). The instruments have marginal lands that guide the instrument in the center of the canals and around curvatures.

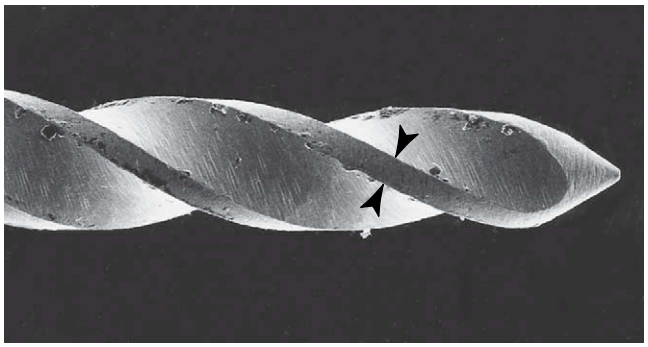


FIG. 8-22 Rotary ProFile NiTi file, #5 (DENTSPLY Tulsa Dental, Tulsa, OK). Note the instrument's wide marginal land (*arrows*).

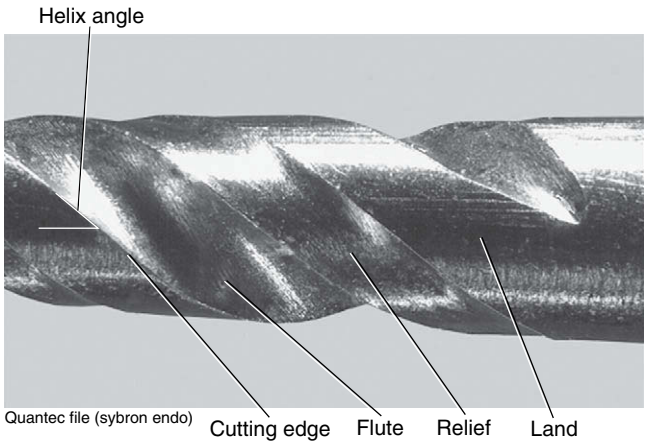


FIG. 8-23 Components of the Quantec NiTi rotary instrument (SybronEndo, Orange, CA). (*Courtesy John T. McSpadden, Lookout Mountain, GA.*)

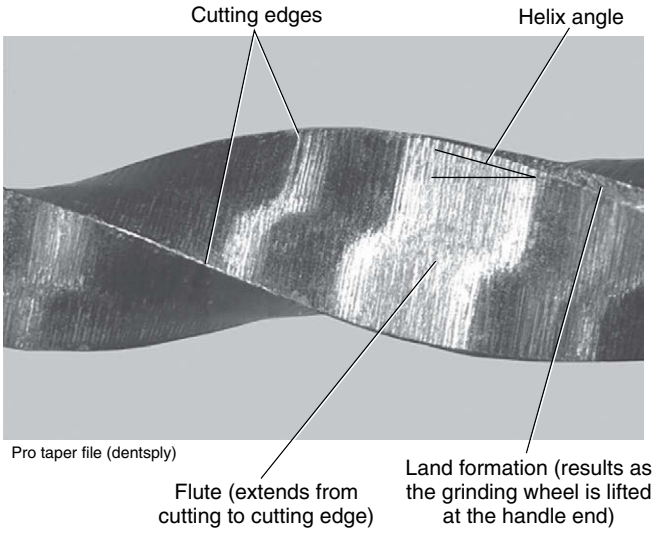


FIG. 8-24 Components of the ProTaper NiTi rotary instrument (DENTSPLY Tulsa Dental, Tulsa, OK). (*Courtesy John T. McSpadden, Lookout Mountain, GA.*)

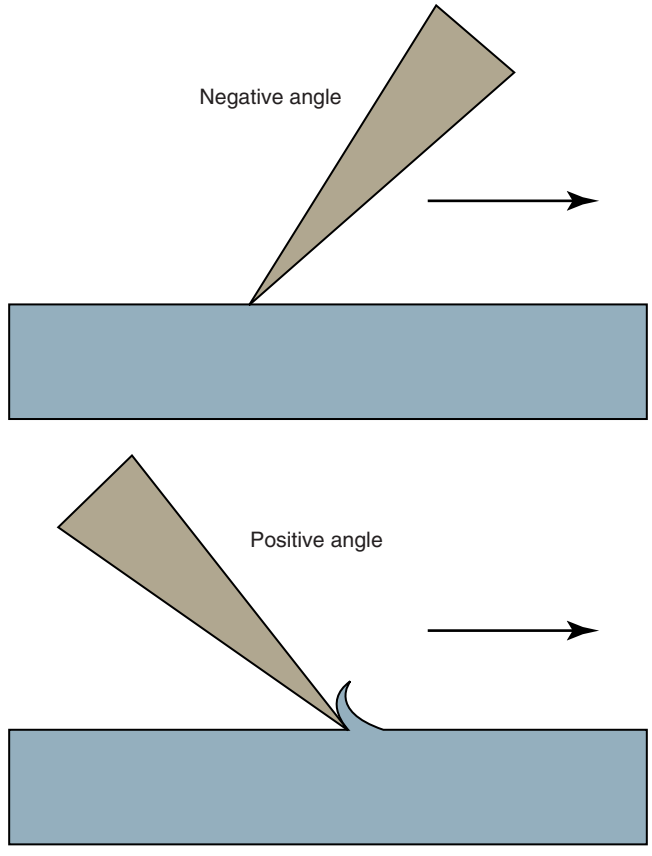


FIG. 8-25 Positive and negative rack angles. Negative angle results in scraping action; positive angle results in cutting action.

points within which the pattern is not repeated. The smaller the pitch or the shorter the distance between corresponding points, the more spirals the file has and the greater the helix angle. Most files have a variable pitch, one that changes along the working surface. Because the diameter increases from the file tip toward the handle, the flute becomes proportionately deeper, resulting in a *core taper* that is 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



FIG. 8-26 Torque- and speed-controlled electric micromotor and handpiece (X-Smart Endodontic Motor). *(All rights owned by and used with permission from DENTSPLY International, Inc.)*

ratios of these quantities can vary between instruments of the same series. A change in any of these features can influence the file's effectiveness or its propensity for breakage as it progresses into the canal space; this can explain why some files act uncharacteristically compared with other files in the same series. In one study, investigators using electric and air-driven handpieces with rotary NiTi instruments found no significant difference in file distortion or breakage between the two handpieces at 150 revolutions per minute (rpm).[40] Other researchers have shown that the ability to select precise rpm[71] and torque[122] settings affects the efficiency and durability of instruments (Fig. 8-26). Determining and maintaining a file's proper rotational speed is more difficult with an air handpiece than with an electric handpiece. For this reason, the clinician would be wise to use an electric handpiece when instrumenting with rotary files. The popularity of electric handpieces among clinicians appears to support the conclusion that regardless of the design used for rotary NiTi instruments, an electric handpiece—rather than an air-driven handpiece—should be used because it allows precise speed and torque control (see Fig. 8-26).

INSTRUMENT DESIGNS

Design changes are made in endodontic instruments to help prevent procedural errors, increase efficiency, and improve the quality of canal shaping. Examples of instruments commonly used today are the ProFile and ProFile GT, the ProTaper, the Lightspeed LSX, the Quantec, the Twisted File (TF),[123,212] the RaCe, the EndoSequence, and the EZ-Fill SafeSider. For more information about these instruments and others, see Chapter 9. In many patients, the apical canal is larger than the largest file used at working length, so many design changes have been directed toward enabling the clinician to increase the size of the largest file used at working length. The following design components can be used to prevent excess stress on instruments.

1. The difference between the file's minimum and maximum diameters can be reduced so that the torque required for rotating the larger diameter does not exceed the plastic limit of the smaller diameter.
2. The space between the tip and the maximum diameter can be reduced so that the required torque does not exceed the ultimate strength of any part of the file.
3. 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.
4. The continuity of the blade engagement can be interrupted.
5. The number of flute spirals can be eliminated or reduced to the smallest number necessary to prevent excessive torque, which results from the accumulation of debris.
6. A means can be provided to complete the file function before the flutes fill with debris.
7. Any land width can be minimized to reduce abrasion on the canal surface.
8. The file can be given an asymmetric cross-section to help maintain the central axis of the canal.
9. The number of flutes with similar helix angles can be reduced. When helix angles are dissimilar, screwing-in forces are reduced; when flutes have no helix angles, screwing-in forces are eliminated.
10. Positive cutting angles can be incorporated to enhance the efficiency of canal enlargement.
11. Blades can be made appendages or projections from the file shaft rather than ground into the shaft.
12. Channels can be cut along the long axis of the file to facilitate its removal if it breaks.

Group IV: Engine-Driven Three-Dimensionally Adjusting Files

SELF-ADJUSTING FILE

The self-adjusting file (SAF; ReDent-Nova, Raanana, Israel) represents a new approach in file design and mode of operation.[260] The file is a hollow device, designed as a cylinder of thin-walled, delicate NiTi lattice with a lightly abrasive surface (Figs. 8-27 and 8-28). An initial glidepath is established with 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 (Fig. 8-29). It is operated with a modified KaVo handpiece (GENTLEpower 20LP with a 3LDSY head, KaVo Dental GmbH, Biberach/Riss, Germany) that generates in-and-out vibrations with 5000 vibrations per minute and 0.4 mm amplitude (Fig. 8-30). The overall concept is that the compressed file will adapts itself to the root canal walls, applying a uniform cutting action (see Fig. 8-28) gradually removing a uniform dentin layer from the canal walls.

As noted, the file is hollow, which allows for continuous irrigation through the file while operated in the root canal. The irrigant is delivered through a free-rotating hub to which a silicone tube is attached (see Fig. 8-30). Either a special irrigation unit (Fig. 8-31) (VATEA, ReDent-Nova, Raanana, Israel) or any appropriate dispenser-type unit may be used to deliver a constant flow of irrigant at 5 ml/min. This maintains a continuous flow of fresh, fully active irrigant that carries with its outflow tissue debris and the dentin powder generated by the file.[256]

One file is used throughout the procedure. It is initially compressed into the root canal (see Fig. 8-29) and gradually enlarges while cleaning and shaping the canal. The unique feature of this file is that it adapts to the shape of the canal not



FIG. 8-27 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. *(Courtesy ReDent-Nova, Raanana, Israel.)*

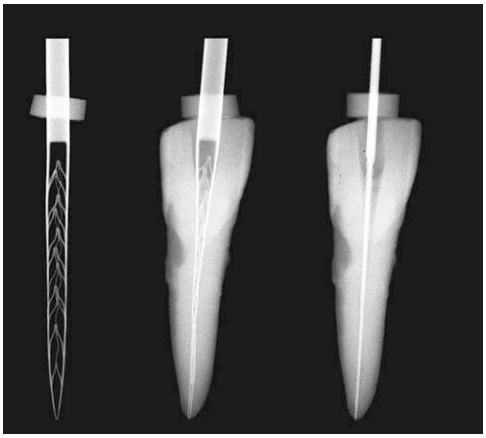


FIG. 8-29 The SAF instrument adapted into a root canal that was initially prepared with #20 K-file. *Right:* A #20 K-file in the canal. *Left:* The SAF file in its relaxed form. *Center:* The SAF file inserted into the same narrow canal. It will apply delicate pressure on the canal wall, attempting to resume its original shape. *(Courtesy ReDent-Nova, Raanana, Israel.)*

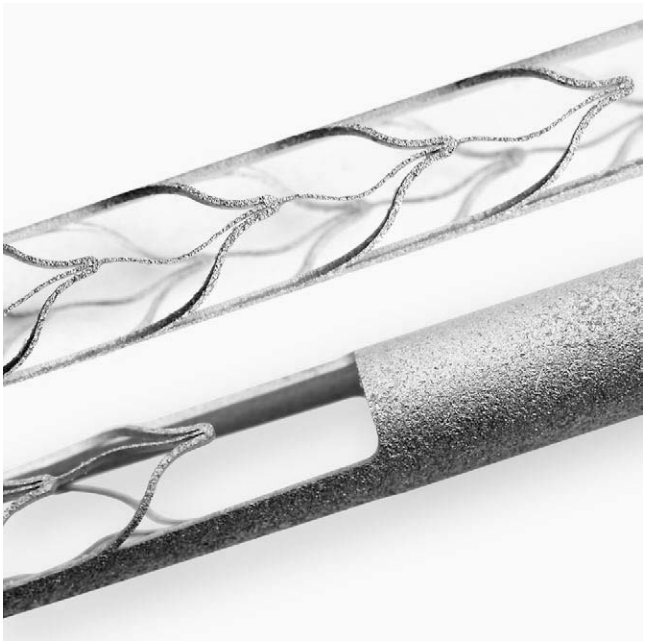


FIG. 8-28 The abrasive surface and details of the lattice of the SAF instrument. The extreme elasticity is the total of the elasticity of each of the delicate NiTi segments. *(Courtesy ReDent-Nova, Raanana, Israel.)*



FIG. 8-30 The VATEA continuous irrigation unit used with the SAF instrument. The unit has two containers and provides a continuous flow (low pressure, 5 ml/min) of either irrigant (i.e., sodium hypochlorite and EDTA) through double silicon tubes that are connected to the hubs on the front of the device. It is controlled by finger-operated switches located on the handpiece. *(Courtesy ReDent-Nova, Raanana, Israel.)*

only longitudinally, as every NiTi file does, but also to the cross-section of the canal. Consequently, the basic shape of the root canal is preserved: a root canal with a round cross-section is enlarged to one with a similar but larger cross-section, whereas a flat root canal will be enlarged to a flat root canal of bigger dimensions (see Fig. 8-31). This unique feature of the SAF file makes it different from any other currently available file system.

Because it has no rigid metal core, the file is also extremely adaptable longitudinally. The inherent tendency to straighten curved root canals and transport their apical part to the outer side of the curvature, which is typical of most other file systems, is greatly avoided. The absence of a metal core also makes the SAF extremely resistant to fracture. File separation—a major problem with other NiTi files—has not been reported to occur, and mechanical failure (a rare occurrence if it happens) is

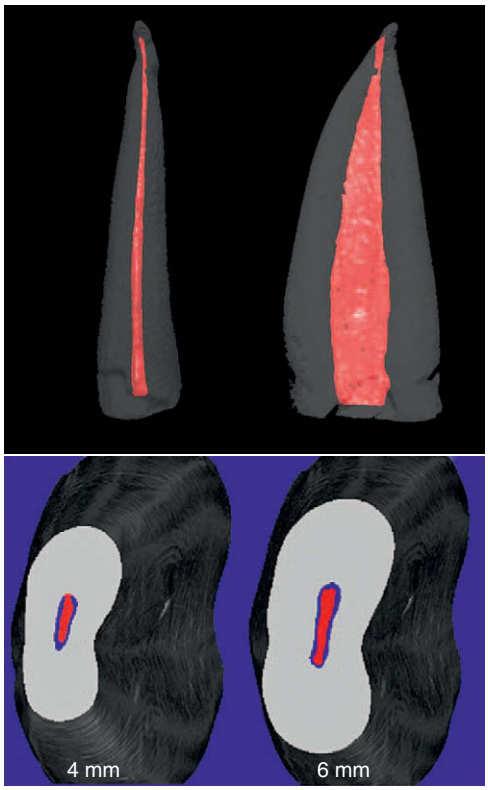


FIG. 8-31 A micro–computed tomography (microCT) analysis of the operation of the SAF instrument in a flat canal with extremely oval cross-section. *Upper:* Buccolingual and mesiodistal views of the root canal reconstructed from microCT. *Lower:* Cross-sections at 4 and 6 mm from the apex. *Red:* before, *blue:* after treatment. *(Courtesy ReDent-Nova, Raanana, Israel.)*



FIG. 8-32 P5 Neutron piezoelectric ultrasonic unit. *(Courtesy Satelec, Merignac Cedex, France.)*



FIG. 8-33 ENAC piezoelectric ultrasonic device. *(Courtesy Osada USA, Los Angeles, CA.)*

limited to local tears in the delicate NiTi lattice.[260] It is clear that this interesting conceptual device will be subjected to extensive independent study over the next few years.

Group V: Engine-Driven Reciprocating Instruments

ENDO-EZE RECIPROCATING FILES

The Giromatic handpiece, a rotary instrument in use since 1969, delivers 3000 quarter-turn reciprocating movements per minute. Rasps and barbed broaches are most often used in Giromatic handpieces, but K-type and H-type instruments also can be used. The Endo-Eze file system (Ultradent, South Jordan, UT) is a recently introduced addition for Giromatic handpieces. The set has four instruments that are designed to clean the middle third of the canal. The sizes and tapers are 0.10 #0.025 taper, 0.13 #0.35 taper, 0.13 #0.45 taper and 0.13 #0.06 taper. The use of stainless steel hand instruments is suggested for the apical third of the canal.

Group VI: Sonic and Ultrasonic Instruments

A radically different 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 (Fig. 8-32). 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 primarily available. Ultrasonic devices, which operate at 25 to 30 kHz, include the magnetostrictive Cavi-Endo (DENTSPLY Caulk, Milford, DE), the piezoelectric ENAC (Osada, Tokyo) (Fig. 8-33), the EMS Piezon Master 400 (Electro Medical Systems [EMS] Vallée de Joux, Switzerland), and the P5 Neutron (see Fig. 8-32) (Satelec, Merignac Cedex, France). Sonic devices, which operate at 2 to 3 kHz, include the Sonic Air MM 1500 (Micro Mega, Prodonta, Geneva, Switzerland), the Megasonic 1400 (Megasonic Corp, House Springs, MO), and the Endostar (Syntex Dental Products, Valley Forge, PA). Ultrasonic devices use regular types of instruments (e.g., K-files), whereas sonic devices use special instruments 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 electric handpiece. The magnetostrictive system generates considerable heat, and a special cooling system is needed in addition to the irrigation system for the root canal. The piezoelectric transducer transfers more energy to the file

than does the magnetostrictive system, making it more powerful. Working with no water cooling becomes essential when using an operating microscope, because the water spray may obstruct visualization.

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.[5]

Ultrasonic devices have proved very efficient for irrigating root canal systems. During free ultrasonic vibration in a fluid, two significant physical effects are observed: cavitation and acoustic streaming. During oscillation in a fluid, a positive pressure is followed by a negative pressure. If the file's tensile strength is exceeded during this oscillation of pressure gradients, a cavity is formed in the fluid in the negative phase. During the next positive-pressure phase, the cavity implodes with great force; this is *cavitation*. Under normal clinical conditions, the power of dental ultrasonic units is too low to create significant cavitation effects on the dentin walls. *Acoustic streaming* creates small, intense, circular fluid movement (i.e., eddy flow) around the instruments. The eddying occurs closer to the tip than in the coronal end of the file, with an apically directed flow at the tip. Acoustic streaming increases the cleaning effect of the irrigant in the pulp space through hydrodynamic shear stress. The increased amplitude that occurs with the smaller file sizes enhances the acoustic streaming.[6] This has proved valuable in the cleaning of root canals because conventional irrigation solutions do not penetrate small spaces well.

Acoustic streaming has little direct antimicrobial effect. Both cavitation and acoustic streaming are dependent on the free vibration of the file. The limits of the space in a root canal significantly inhibit the practical utility of ultrasonic devices for root canal cleaning. Depending on size and power, the file tip may have amplitudes that require a canal size of at least a #30 file through a #40 file for free oscillation. Any contact with the root canal walls dampens oscillation. As the contact with the canal wall increases, the oscillation is dampened and becomes too weak to maintain acoustic streaming. Using a small file size with minimal contact to the root canal wall provides optimal cleaning conditions.[7]

Ultrasonic devices have proved disappointing as instruments for improving the removal of dentin from the root canal walls. They do improve the ability to clean the pulp space (Fig. 8-34) and difficult-to-débride areas through acoustic streaming.[12,68,402,403] However, it is unclear whether this can be achieved during regular preparation when the file is actively dampened and little acoustic streaming takes place. Cleaning is further enhanced by the excellent irrigation systems some of the devices provide. Application of a freely oscillating file with sodium hypochlorite (NaOCl) irrigation for several minutes to aid pulp space disinfection is believed to be useful after complete biomechanical instrumentation of the pulp space.[361]



FIG. 8-34 Start-X ultrasonic tip #2; designed for working on the pulp chamber floor and walls when looking for 2MB canals. Note the surface texture of the instrument that allows long-lasting active surface. *(All rights owned by and used with permission from DENTSPLY International, Inc.)*

Sonic devices are more useful for true hard-tissue removal during root canal preparation. Because the files operate like a conventional handpiece, the file vibrations are less likely to be dampened by contact with the root canal walls. Therefore the special files used in these systems are true bulk dentin removers. New tip designs for piezoelectric ultrasonic units have also been found extremely effective in dentin removal in the pulp chamber and orifice of the canals (see Fig. 8-34).

National and International Standards for Instruments

As a result of concerns that arose nearly 45 years ago, efforts were made to standardize endodontic files and root filling materials. As mentioned previously, this resulted in an international standard for endodontic files, known in the United States as *ANSI standard No. 58* for Hedström files and *ANSI standard No. 28* for K-files (Table 8-1). The standards have several similarities, but some important differences exist. Fig. 8-35 shows the measurements dictated by the standards. The size designation is derived from the projected diameter at the tip of the instrument. This is an imaginary measurement and is not reflected in the real size of the working part of the instrument. The taper of the instruments is designed to be a 0.02 taper, namely a diameter-increase by .02 mm for each mm of length, starting at the tip. The working diameter is the product of the taper and the length of the tip. Three standard lengths are available at 21 mm, 25 mm, and 31 mm. The working part of the instrument must be at least 16 mm. As stated previously, tapers other than .02 and working parts of instruments less than 16 mm are now available and outside the standard.

This system of numbering files with at least 15 different sizes replaced the old, somewhat imperfect system that numbered the sizes from #1 through #6. Although the new standard

TABLE 8-1

Dimensions of Standardized K-File, H-File, and Gutta-Percha Cones (ANSI No. 28, 58, and 78*)

Size	D_0	D_{16}	Color
006	*0.06*	*0.38*	*No color assigned*
008	*0.08*	*0.40*	*No color assigned*
010	0.10	0.42	Purple
015	0.15	0.47	White
020	0.20	0.52	Yellow
025	0.25	0.57	Red
030	0.30	0.62	Blue
035	0.35	0.67	Green
040	0.40	0.72	Black
045	0.45	0.77	White
050	0.50	0.82	Yellow
055	0.55	0.87	Red
060	0.60	0.92	Blue
070	0.70	1.02	Green
080	0.80	1.12	Black
090	0.90	1.22	White
100	1.00	1.32	Yellow
110	1.10	1.42	Red
120	1.20	1.52	Blue
130	1.30	1.62	Green
140	1.40	1.72	Black

*Sizes in italics are for files that are commercially available but not covered by American National Standards Institute (ANSI) regulation No. 28 or No. 58. Colors are not required for instrument handles or gutta-percha cones, but the size must be printed on the handle. Tolerances are ± 0.02 ≥30 mm ± 2 mm.

includes many sizes, astute clinicians may include fewer instrument sizes for their special work habits.

In recent years, suggestions to change 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. Considering the fact that most manufacturers already are unable to size their instruments within the accepted range, the introduction of half sizes seems unnecessary. However, if standards are strictly adhered to, the use of half sizes seems more reasonable for instrument systems such as the LightSpeed, in which the strength of the instrument is such that full-size increments may generate stresses beyond the tolerance of the instrument. The standards are overdue for reevaluation in light of recent technologic changes.

Effectiveness and Wear of Instruments

Although the advertising literature is rich in claims of superiority of various file designs, few of these claims can be verified by well-designed studies in the peer-reviewed endodontic literature. No standards exist for either the cutting or 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. These two parameters are radically different. Methods exist for measuring machining, but currently no good method is available for measuring cutting. Some studies have attempted to evaluate cutting, but the methodologies have involved the use of a drilling motion with K-type instruments and at a speed higher than that used for clinical procedures.

Some studies of machining have evaluated the effectiveness of an instrument when used with a linear movement. These studies showed that instruments can differ significantly, not

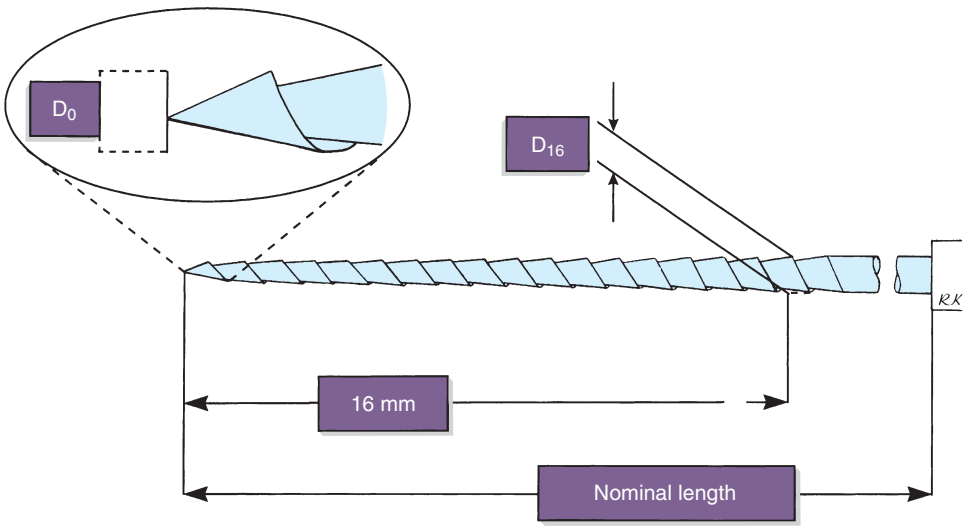


FIG. 8-35 Measuring points for American National Standards Institute (ANSI) and American Dental Association (ADA) regulations #28 and #58, which cover K-type and H-type instruments. The measuring point for the diameter of the instrument (size) is imaginary (D_0) and projects the taper of the instrument at the tip. Therefore an instrument with a short tip is more true to its size than an instrument with a long tip. D_{16} represents the diameter at the end of the working part, which must be at least 16 mm long.

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. The variation for Hedström files is greater, ranging from 2.5 to more than 50 times.[379] 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. The Hedström file, therefore, is approximately 10 times more effective at removing dentin than the K-file.

In the machining process, the 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, which is a modified K-file, shows variables similar to those of K-files. The Flex-R file (SybronEndo), which is a ground instrument, more closely resembles the H-files in its variation in effectiveness. It also is much more effective at substrate removal than the K-files but cannot measure up to the H-files' ability to machine.

Modern endodontic stainless steel instruments are fabricated from excellent metal alloys and have considerable resistance to fracture. The clinician who practices careful application of force and a strict program of discarding instruments after use should have few instrument fractures. Stainless steel files are so inexpensive 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.

Devices for Periapical Débridement Through a Root Canal Access

The Apexum device (Apexum, Or-Yehuda, Israel) is currently the single representative of this group[258,259] (Fig. 8-36). It represents a totally different concept than that applied by most root canal cleaning and shaping systems. The goals of most current concepts are to maintain the original apical constriction by all means and to avoid any substantial overinstrumentation past the apical foramen. The Apexum concept requires widening the apical foramen to create an opening 330 µm in diameter by passing a #30 file 1 mm beyond the apex. This may represent some enlargement of the apical foramen in certain cases, but in other cases, the natural apical foramen diameter may be larger than this.[43] Further instrumentation with larger instruments (#40 or #45) to a length 1 mm short of the apical foramen are then used to recreate an artificial apical stop and constriction.

Two miniature devices are then used to débride the periapical tissues through the root canal access. The first is the Coarse Apexum Ablator, which is made from a prebent memory-shape NiTi wire encased in a NiTi tube that serves as its sheath (see Fig. 8-36). The instrument, still encased in its sheath, is inserted to the artificial apical stop. Then the inner wire is gently pushed through the apical foramen and into the periapical lesion.

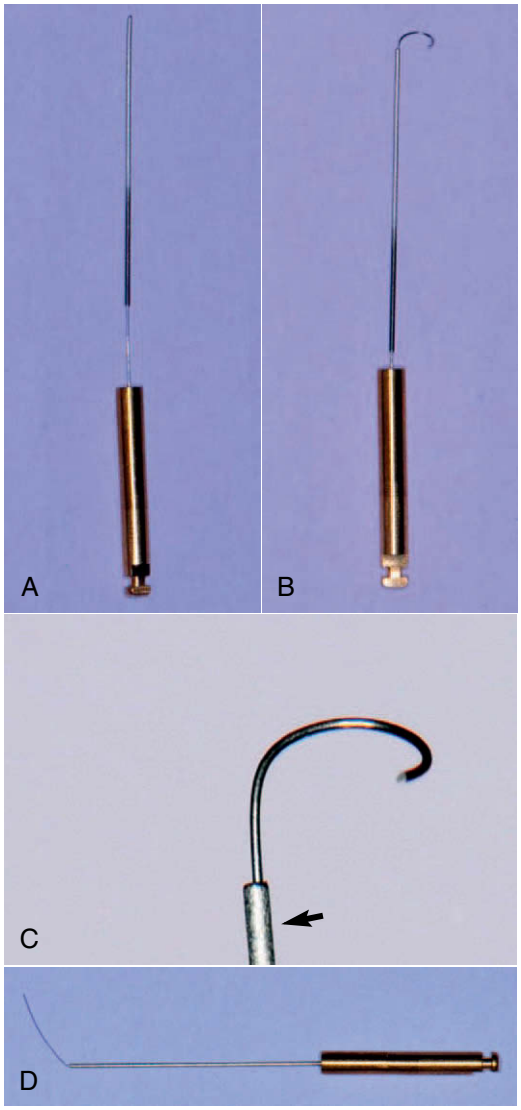


FIG. 8-36 Apexum device. **A,** The Coarse Apexum Ablator in its NiTi sheath (*arrow in C*). **B-C,** The active part was extruded and regained its preformed shape that allows it to rotate in the periapical lesion (250 rpm, 30 sec), mincing the soft tissue without engaging the walls of the bony crypt. **D,** The Fine Apexum Ablator, inserted into the lesion after the Coarse Apexum Ablator is removed; rotates at 7000 rpm for 30 seconds, turning the minced tissue into a thin suspension that can be washed out.[258,259] *(Courtesy Apexum, Or-Yehuda, Israel.)*

While in the periapical area, it regains its prebent shape (Figs. 8-37 and 8-38). It is then attached to a low-speed handpiece and rotated at 250 rpm for 30 seconds, thus coarsely mincing the periapical tissues. The first instrument is then removed, and the second instrument, the Fine Apexum Ablator, is inserted, passed through the apical foramen, and rotated at 7000 rpm for 30 seconds, turning the tissue into a thin suspension. The resulting suspension is then washed out coronally through the canal space using sterile saline solution delivered into the periapical area using a syringe and a thin, 30-gauge blunt needle.

The method is used as a supplementary treatment for enhancement the healing kinetics of periapical lesions for

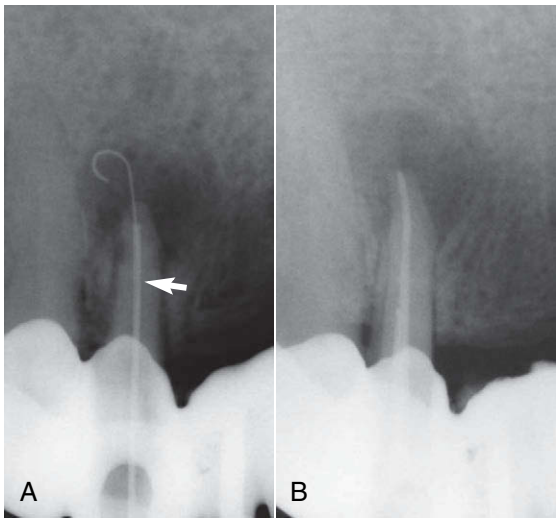


FIG. 8-37 Apexum in clinical use. **A,** The Coarse Apexum Ablator in a periapical lesion of an upper single rooted tooth. Note the sheath in the root canal *(arrow)*. **B,** Root canal obturation (lateral condensation) immediately after the Apexum procedure has been completed.[258] *(Courtesy Apexum, Or-Yehuda, Israel.)*



FIG. 8-38 Apexum in clinical use. A lower molar with two Coarse Apexum Ablator devices in the mesial and distal periapical lesions. Note the distance from the inferior alveolar nerve canal. Both instruments were inserted at the same time for purposes of illustration. *(Courtesy Apexum, Or-Yehuda, Israel.)*

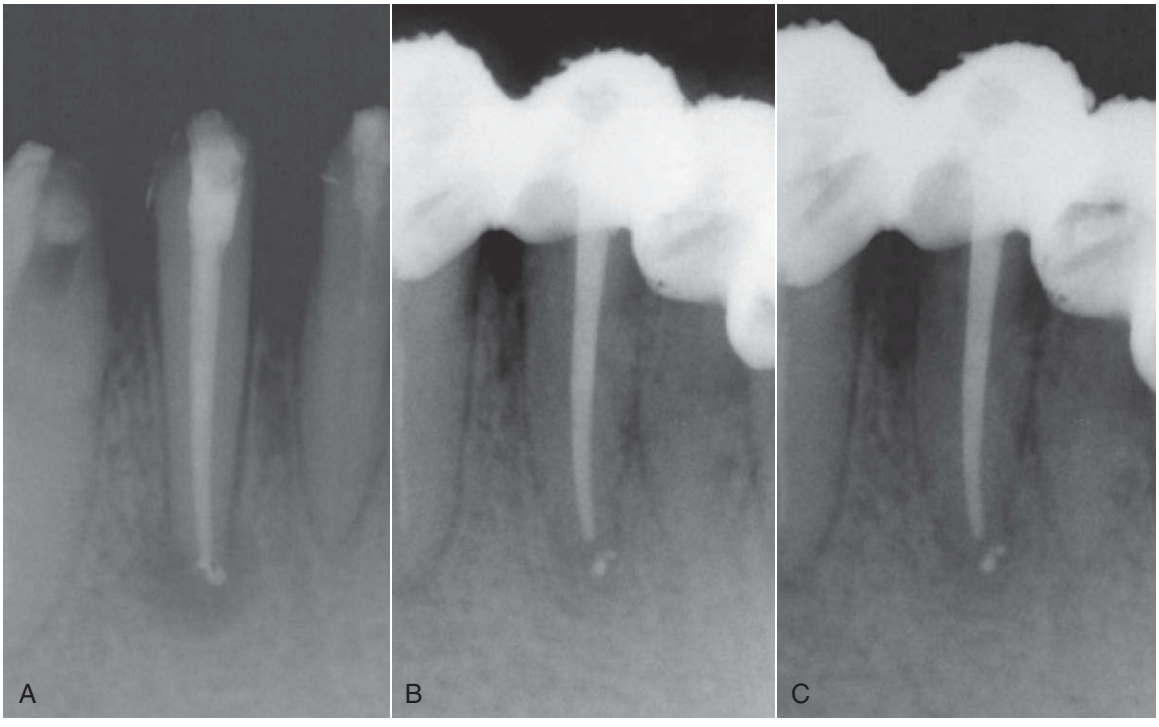


FIG. 8-39 Periapical healing after the Apexum procedure. **A,** Immediate postoperative radiograph. Note the periapical lesion. **B,** Three-month follow up. **C,** Six-month follow-up.[258] *(Courtesy Apexum, Or-Yehuda, Israel.)*

promoting faster healing (Fig. 8-39).[258,259] The Apexum procedure is applied only after completion of cleaning, shaping, and disinfection of the root canal and before root canal obturation. This interesting device will clearly be subjected to additional research over the next few years.

Devices for Measuring Root Canal Length

Radiographs, tactile sensation, the presence of bleeding on paper points, and knowledge of root morphology have been used to determine the length of root canal systems. Sunada[381]

developed the original electronic apex locator when he suggested 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.[114,115] 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.[115] However, these devices must not be considered flawless, because several variables are known to affect their accuracy. For example, immature roots can present problems.[180] Once the roots mature (i.e., formed a narrow apical foramen) and the instruments are able to contact the canal walls, the electronic apex locator's accuracy greatly improves. Some investigators have found no statistical difference between roots with vital and necrotic tissue.[135,250] Because apical root resorption is prevalent in necrotic cases with long-standing apical lesions, these researchers also concluded that apical resorption does not have a significant effect on the accuracy of electronic apex locators.

Recently 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.[95] Furthermore, radiographs may also add essential anatomic information that may 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.[197] 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.[197] This study found that electrolytes did not have a significant effect on the accuracy of the unit. Some third-generation apex locators are the Endex Plus, or Apit, (Osada, Los Angeles, CA),[141] the Root ZX (Fig. 8-40) (J. Morita, Kyoto, Japan),[114,211] and the Neosono Ultima EZ (Satelec, Mount Laurel, NJ). 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.

A fourth-generation apex locator was introduced with the Elements Diagnostic Unit (see Fig. 8-44), the Apex Locator (SybronEndo), and the Bingo 1020/Ray-X4 (Forum Engineering Technologies, Rishon Lezion, Israel). The Bingo uses only one of its two frequencies at a time (8 Hz or 400 Hz). According to the manufacturer, the Elements unit (which operates at frequencies of 0.5 and 4 kHz) compares the resistance and capacitance information to a database to determine the distance between the file and the apex. When the file tip reaches the area of the apical foramen, the apex locator emits a signal.

Recently, miniaturized electronic apex locators were introduced by SybronEndo (Fig. 8-41) and MedicNRG, Kibutz Afikim, Israel (the Apex NRG[277,278]) (Fig. 8-42).

An apex locator typically has four parts: (1) the lip clip, (2) the file clip, (3) the instrument itself, and (4) a cord connecting the other three parts. A display indicates the advancement of the file toward the apex (see Fig. 8-40).



FIG. 8-40 Root ZX apex locator with lip clip and file holder. *(Courtesy J. Morita USA, Irvine, CA.)*



FIG. 8-41 SybronEndo mini apex locator. *(Courtesy SybronEndo, Orange, CA.)*

These electrical instruments are generally safe, but manufacturers' instructions state that they should not be used on patients with pacemakers without consulting the patient's cardiologist.[30,113] When connected directly to cardiac pacemakers in vitro, four of five electronic apex locators did not interfere with the function of the pacemaker.[126]

Instruments for Root Canal Obturation

After the root canal has been properly cleaned and enlarged, the space is obturated with a manufactured material. A number of obturation methods are practiced, but lateral and vertical compactions are the two most common. Many specialized



FIG. 8-42 NRG miniature electronic apex locator. *(Courtesy Medic-NRG, Kibutz Afikim, Israel.)*



FIG. 8-43 System B heating device for vertical compaction and removal of gutta-percha. *(Courtesy SybronEndo, Orange, CA.)*

TABLE 8-2

Size Designations for Auxiliary Gutta-Percha Cones*

Designation	D_3	D_{16}	Taper
XF	0.20	0.45	0.019
FF	0.24	0.56	0.025
MF	0.27	0.68	0.032
F	0.31	0.80	0.038
FM	0.35	0.88	0.041
M	0.40	1.10	0.054
ML	0.43	1.25	0.063
L	0.49	1.55	0.082
XL	0.52	1.60	0.083

*The cones are pointed. The diameters 3 mm (D_3) and 16 mm (D_{16}) from the tip are prescribed. Tolerance is ± 0.05 mm, and length is ≥30 mm ± 2 mm. *XF*, Extra-fine; *FF*, fine-fine; *MF*, medium-fine; *FM*, fine-medium; *M*, medium; *ML*, medium-large; *L*, large; *XL*, extra-large.

instruments are available for every type of method. Spreaders and pluggers are the significant instruments for obturation. The spreader is a tapered, pointed instrument intended to displace gutta-percha laterally for insertion of additional accessory gutta-percha cones. The plugger is similar but has a blunt end. In smaller sizes, the spreader and plugger are often used interchangeably. These instruments are available with handles or as finger-held instruments. The instruments with handles are potentially dangerous because the tips of the working ends are offset from the long axes of the handles. This results in strong lateral wedging forces on the working ends if the instruments are not operated carefully. The risk of vertical damage to the root is greatly reduced with finger spreaders and pluggers. Each clinician must choose the appropriate spreader and plugger according to personal working preferences. Standardized instruments are available with the same taper as the files (e.g., #.02). Considering the greater taper of standardized accessory gutta-percha cones (Table 8-2), nonstandardized spreaders with a larger taper sometimes may be used to better accommodate the gutta-percha.

When using spreaders and accessory gutta-percha cones, the accessory cone selected must reach the depth of the penetration of the spreader. To accomplish that, the accessory cone must be *thinner* and/or with a *smaller taper* than the spreader used. If accessory cones which do not reach that depth are used, the procedure will result in voids at the apical end of each spreader penetration, leading the operator to an illusion that the root canal is properly obturated.

In recent years, spreaders and pluggers have become available in nickel-titanium. NiTi spreaders have been shown to reach deeper into canals than the stainless steel type when #.02 tapered gutta-percha is used in canals with a curvature of more than 20 degrees. When #.04 taper gutta-percha was used, the NiTi spreaders were more effective regardless of the degree of curvature.[424]

Heat carriers are used for vertical-compaction obturation techniques. Traditionally, heat carriers are handled similarly to pluggers. They are used to transfer heat to the gutta-percha in the root canal, allowing apical and lateral displacement of the gutta-percha. Electrical heat carriers are more common today, either as independent units such as Touch 'n Heat and System B (SybronEndo, Orange, CA) (Fig. 8-43), or incorporated into multifunctional systems such as Elements (Fig. 8-44) (SybronEndo). Recently, battery-charged, handheld heat carriers were introduced, such as the vibrating heat carrier DownPack[67] (Hu-Friedy, Chicago, IL) (Fig. 8-45) or HotTip (Fig. 8-46) (Discus Dental, Culver City, CA). These devices can be heated to controlled levels with a self-contained cordless unit.



FIG. 8-44 Elements unit, combining a System B element with one delivering injectable gutta-percha. *(Courtesy SybronEndo, Orange, CA.)*



FIG. 8-45 DownPack: hand-held heating and vibrating unit for warm gutta-percha compaction. *(Courtesy Hu-Friedy, Chicago, IL.)*

A Lentulo spiral may be used for placement of the sealer, cement, and calcium hydroxide ($Ca[OH]_2$) dressings. The Lentulo spiral is a safe instrument if used correctly but if used carelessly, it may engage the wall of the root canal and break. It must be operated clockwise in the handpiece, inserted not rotating to working length, then retracted 1 to 2 mm to make



FIG. 8-46 "HotTip": hand-held heating unit for warm gutta-percha compaction. *(Courtesy Discus Dental LLC; Culver City, CA.)*

sure it is free to rotate without engaging the canal walls. It should be started and rotated at a slow speed while being gradually withdrawn from the root canal. This instrument effectively drives the paste into the root canal. However, for optimal effect, the spiral must be as large as possible so that the paste is forced forward as the material is squeezed between the canal walls and the spiral. Endodontic files, paper points, and syringes also are commonly used to apply sealer in the root canal system, but they are all less effective than a Lentulo spiral if filling a root canal entirely with sealer is desired.

Devices for Removing Root Canal Obstructions

There are many devices available for removing natural and manufactured obstructions from within the canal. Please see Chapter 25 for details on these devices.

MATERIALS FOR DISINFECTING THE PULP SPACE

Chemomechanical Preparation

To increase the efficacy of mechanical preparation and bacteria removal, instrumentation must be supplemented with active irrigating solutions. *Irrigation* is defined as washing out a body cavity or wound with water or a medicated fluid. *Aspiration* is defined as the process of removing fluids or gases from the body with a suction device.

The objectives of irrigation are both mechanical and biologic. The mechanical objective involves flushing out debris, lubricating the canal, and dissolving organic and inorganic tissue. The biologic function of the irrigants is related to their antimicrobial effect.

Irrigation Hydrodynamics

The effectiveness 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 (Box 8-3).[181]

The size and length of the irrigation needle relative to the canal space dimension is of utmost importance for the effectiveness of proper irrigation. If the external diameter of the needle is too large or rigid, it may inhibit introduction of the irrigant into the more apical extent of the root canal or into areas of curved canals. The internal diameter of the needle correlates to the necessary pressure for moving the syringe plunger and the velocity at which the irrigant is expressed. Narrow needles require more pressure of the plunger and will extrude the irrigant with higher velocity than large needle diameters. Although a needle with a larger internal diameter

BOX 8-3

Ideal Characteristics of an Endodontic Irrigant[58,149,390]

The ideal irrigant should:

1. Be an effective germicide and fungicide.
2. Be nonirritating to the periapical tissues.
3. Remain stable in solution.
4. Have a prolonged antimicrobial effect.
5. Be active in the presence of blood, serum, and protein derivates of tissue.
6. Have low surface tension.
7. Not interfere with repair of periapical tissues.
8. Not stain tooth structure.
9. Be capable of inactivation in a culture medium.
10. Not induce a cell-mediated immune response.
11. Be able to completely remove the smear layer, and be able to disinfect the underlying dentin and its tubules.
12. Be nonantigenic, nontoxic, and noncarcinogenic to tissue cells surrounding the tooth.
13. Have no adverse effects on the physical properties of exposed dentin.
14. Have no adverse effects on the sealing ability of filling materials.
15. Have convenient application.
16. Be relatively inexpensive.

will extrude the irrigant with a larger volume over time, the irrigant cannot be introduced as deeply. Moser and Heuer[276] measured the pressure necessary for activation of the plunger in different sizes and types of irrigation needles. They determined lower pressure was necessary for irrigation with larger needle diameter sizes (23 to 24 gauge) compared with smaller diameter sizes (24 to 30 gauge). However, larger needles placed at a greater distance from the apex were less efficient during irrigation than smaller diameter needles placed closer to the apex.[174] Common injection needles have an external diameter of 0.40 mm (27 gauge), but special irrigation tips with external diameters of 0.30 mm (30 gauge) are available as well (Table 8-3). To improve safety of irrigation and prevent apical extrusion of the irrigant, some of these needles release the solution through lateral openings and have a closed, safe-ended tip (Fig. 8-47). The Stropko Flexi-Tip (30 gauge) needle is fabricated out of NiTi to improve its flexibility and penetration into curved canals.

TABLE 8-3

Sizes and Manufacturers of Needles Used for Root Canal Irrigation

Manufacturer	Product	Gauge
DENTSPLY	Max-I-Probe	21–30
Ultradent, South Jordan, UT, USA	NaviTip	29, 30
	NaviTip FX Tip (brush-covered needle)	30
	Capillary Tip	25, 28
	Endo-Eze Tip/Deliver Eze	18, 19, 20, 22, 30, 31
	Endo-Eze Irrigator Tip/Deliver Eze Spülkanüle	27
KerrHawe, Bioggio, Switzerland	NaviTip	21–30
Hager & Werken, Duisburg, Germany	Miraject Endotec	21–25
Vista Dental Products	Stropko Flexi-Tip (NiTi)	30
KerrHawe	KerrHawe irrigation probe	
Transcoject, Neumünster, Germany	Spülkanülen Endo	23, 25, 27, 30

Adapted from Hülsmann M, Rödig T, Nordmeyer S: Complications during root canal irrigation. *Endod Topics* 16:27, 2007.

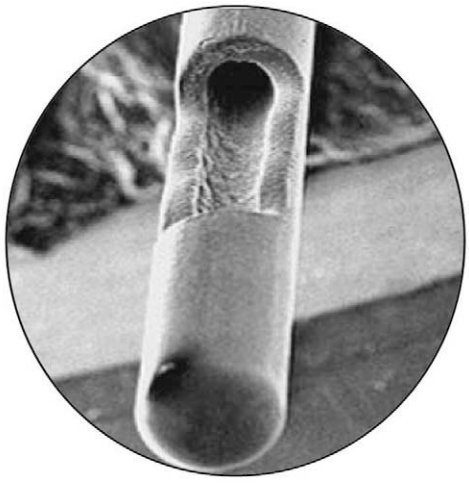


FIG. 8-47 Scanning electron microscope (SEM) image of 30-gauge safety needle. *(Courtesy Dr. F. Tay.)*

Sodium Hypochlorite

NaOCl is the most commonly used irrigating solution. It is an excellent antibacterial agent, capable of dissolving necrotic tissue, vital pulp tissue, and the organic components of dentin and biofilms.[343]

NaOCl solution, commonly known as *bleach,* 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.

History

Hypochlorite was first produced in 1789 in Javelle, France, by passing chlorine gas through a solution of sodium carbonate. The resulting liquid, known as "Eau de Javelle" or "Javelle water" was a weak solution of NaOCl. However, this process was not very efficient, and alternate production methods were sought. One such method involved the extraction of chlorinated lime (known as *bleaching powder*) with sodium carbonate to yield low levels of available chlorine. This method was

commonly used to produce hypochlorite solutions for use as a hospital antiseptic that was sold under the trade names "Eusol" and "Dakin's solution." NaOCl as a buffered 0.5% solution was recommended for the irrigation of wounds during World War I by Dakin.[74] Coolidge[69] later introduced NaOCl to endodontics.

Mode of Action

Please see Chapter 9 for further information on this subject.

Allergic Reactions to Sodium Hypochlorite

Although few reports have been published[101,156] on allergic-like reactions to NaOCl, real allergies to NaOCl are unlikely to occur, since both sodium and chlorine are essential elements in the physiology of the human body. Nevertheless, hypersensitivity and contact dermatitis may occur in rare cases. In cases of hypersensitivity to NaOCl, chlorhexidine should not be used either (because of its chlorine content). Use of an alternative irrigant with high antimicrobial efficacy, such as iodine potassium iodide, should be considered, assuming there is no known allergy to the irrigant. Irrigants such as alcohol or tap water are less effective against microorganisms and do not dissolve vital or necrotic tissue. $Ca(OH)_2$ could be used as a temporary medicament because it dissolves both vital and necrotic tissue.[10,161]

Increasing the Efficacy of Hypochlorite Preparations

One alternative approach to improving the effectiveness of hypochlorite irrigants in the root canal system[439] could be to increase the temperature of low-concentration NaOCl solutions, which improves their immediate tissue-dissolution capacity. Furthermore, heated hypochlorite solutions remove organic debris from dentin shavings more efficiently. The antimicrobial properties of heated NaOCl solutions have also been evaluated. The bactericidal rates for NaOCl solutions are more than doubled for each 5° C rise in temperature in the range of 5° C to 60° C. This was corroborated in a recent study[359] using steady-state planktonic *Enterococcus faecalis* cells. A temperature rise of 25° C increased NaOCl efficacy by a factor of 100. The capacity to dissolve human dental pulp using 1% NaOCl at 45° C was found to be equal to that of a 5.25% solution at 20° C. Additionally, the systemic toxicity of preheated low concentrations of NaOCl irrigants should be less than that of a more concentrated unheated solution. However, there are no clinical studies available at this point to support the use of heated NaOCl.

In this context, it should also be noted that time is a factor that has gained little attention in endodontic studies. Even fast-acting biocides such as NaOCl require an adequate working time to reach their potential. Chlorine, 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.[268] Continuous replenishment of the irrigant is essential, especially since rotary root canal preparation techniques have expedited the shaping process. The optimal time a hypochlorite irrigant needs to remain in the canal system is an issue yet to be resolved.

Chlorhexidine

History

Chlorhexidine (CHX) was developed more than 50 years ago at Imperial Chemical Industries in England and first marketed in the United Kingdom in 1953 as an antiseptic cream.[104] Since 1957 it has been used for general disinfection purposes and the treatment of skin, eye, and throat infections in both humans and animals.[104,231]

Molecular Structure

CHX belongs to the polybiguanide antibacterial family, consisting of two symmetric four-chlorophenyl rings and two bisguanide groups connected by a central hexamethylene chain. CHX is a strongly basic molecule and is stable as a salt. CHX digluconate salt is easily soluble in water.[147]

Mode of Action

CHX is a wide-spectrum antimicrobial agent, active against gram-positive and gram-negative bacteria as well as yeasts.[81] Owing to its cationic nature, CHX is capable of electrostatically binding to the negatively charged surfaces of bacteria,[72] damaging the outer layers of the cell wall and rendering it permeable.[165,178,179]

Depending on its concentration, CHX can have both bacteriostatic and bactericidal effects. At high concentrations CHX acts as a detergent; by damaging the cell membrane, it causes precipitation of the cytoplasm and thereby exerts a bactericidal effect. At low sublethal concentrations, CHX is bacteriostatic, causing low molecular-weight substances (i.e., potassium and phosphorous) to leak out without the cell being irreversibly damaged. It also can affect bacterial metabolism in several other ways, such as abolishing sugar phosphotransferase system (PTS) transport activity and inhibiting acid production in some bacteria.[25]

Substantivity

Due to the cationic nature of the CHX molecule, it can be absorbed by anionic substrates such as the oral mucosa.[237,240] CHX has the ability to bind to proteins such as albumin, present in serum or saliva, pellicle found on the tooth surface, salivary glycoproteins, and mucous membranes.[317,398] This reaction is reversible.[169] CHX can also be adsorbed onto hydroxyapatite and teeth. Studies have shown that the uptake of CHX onto teeth is also reversible. 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%, a stable monolayer of CHX is adsorbed and formed on the tooth surface, which might change the physical and chemical properties of the surface and may prevent or reduce bacterial colonization. At higher concentrations (>0.02%), a multilayer of CHX is formed on the surface, providing a reservoir of CHX which can rapidly release the excess into the environment as the concentration of CHX in the surrounding environment decreases.[97]

The antibacterial substantivity of three concentrations of CHX solution (4%, 2%, and 0.2%) after 5 minutes of application has shown a direct relationship between the concentration of CHX and its substantivity.[271] On the contrary, Lin et al.[226] attributed the substantivity of CHX to its ability to adsorb onto

the dentin during the first hour. They stated that it is only after the saturation point is reached after the first hour that the antimicrobial capability of CHX increases with time. Furthermore, Komorowski et al.[199] revealed that a 5-minute application of CHX did not induce substantivity, so dentin should be treated with CHX for 7 days. Taken together, it seems that residual antimicrobial activity of CHX in the root canal system remains for up to 12 weeks.

Cytotoxicity

In the medical field, CHX is normally used at concentrations between 0.12% and 2.0%. According to Löe,[231] at these concentrations, CHX has a low level of tissue toxicity, both locally and systemically. In another report, when 2% CHX was used as a subgingival irrigant, no apparent toxicity was noted on gingival tissues.[232,369] Moreover, CHX rinses have been reported to promote the healing of periodontal wounds.[14] Based on these reports, Jeansonne et al.[184] assumed that the periapical tissues would be as tolerant to CHX as gingival tissues. In two studies when CHX and NaOCl were injected into subcutaneous tissues of guinea pigs and rats, an inflammatory reaction developed; however, the toxic reaction from CHX was less than that of NaOCl.[289,432] Furthermore, when CHX was applied as a rinse in the extraction sites of the third molars on the day of surgery and several days after, it was reported to reduce the incidence of alveolar osteitis.[56] There are only a few reports in the literature of allergic and anaphylactic reactions to CHX.[127,286]

Conversely, some studies have reported unfavorable effects of CHX on the tissues. Hidalgo and Dominguez[168] demonstrated that CHX is cytotoxic to some lines of cultured human skin fibroblasts. Recently the behavior of osteoblastic human alveolar bone cells in the presence of CHX and povidone iodine (PI) has been investigated. It has been reported that CHX has a higher cytotoxicity profile than povidone iodine.[49] Faria et al.[105] also demonstrated that CHX injected in the hind paw of mice could induce severe toxic reactions. They reported that CHX induced apoptosis at lower concentrations and necrosis at higher concentrations when added to cultured L929 fibroblast cells.

Another interesting observation has recently been reported whereby the byproduct of combining CHX with NaOCl is the formation of toxic breakdown products such as parachloroaniline (PCA) that may have a negative impact on tissues.[26] The toxicity level of CHX on periapical tissues when applied in the root canals, especially with other irrigants, merits further investigation.

Chlorhexidine Application in Endodontics

CHX has been extensively studied as an endodontic irrigant and intracanal medication, both in vivo[20,228,239,293] and in vitro.* In vitro, CHX has at least as good or even better antimicrobial efficacy than $Ca(OH)_2$.[354] Notably, 2% CHX was very effective in eliminating a biofilm of *E. faecalis*.[223] In vivo, it inhibits experimentally induced inflammatory external root resorption when applied for 4 weeks.[228] In infected root canals, it reduces bacteria as effectively as $Ca(OH)_2$ when applied for 1 week.[20] Unlike $Ca(OH)_2$, CHX has substantive antimicrobial activity that, if imparted onto the root dentin, has the potential to prevent bacterial colonization of root canal walls for prolonged periods of time.[184,199] This effect depends on the concentration of CHX, but not on its mode of application, which may be as a liquid, gel, or controlled-release device.[27]

*References 24, 27, 28, 184, 199, and 217.

Chlorhexidine as an Endodontic Irrigant

CHX in liquid and gel form has been recommended as an irrigant solution, and its different properties have been tested in several studies, both in vitro[137] and in vivo.* Many investigations have conducted studies on the antibacterial effectiveness of CHX in different concentrations. It has been demonstrated that 2% CHX as an irrigant has a better antibacterial efficacy than 0.12% CHX in vitro, so the antibacterial efficacy of CHX evidently depends on its concentration level.[28] Since NaOCl is still the most commonly used irrigant, the antibacterial efficacy of CHX has been compared to that of NaOCl. The results from these studies are inconclusive, but in general, no significant difference between the two solutions has been reported. Unlike NaOCl, CHX lacks a tissue-dissolving property. Therefore, NaOCl is still considered the primary irrigating solution in endodontics.

The cleanliness of root canals by CHX in gel and liquid forms was evaluated using scanning electron microscopy in two separate experiments. In an in vitro study, the canals treated with 2% CHX gel were cleaner than those treated with 2% CHX liquid or 5.25% NaOCl, and it was suggested that the mechanical action of the gel might have facilitated the cleansing of the canals. Another in vitro study showed that the 2% CHX liquid was inferior to 2.5% NaOCl in cleaning the canals.[431]

The antibacterial effectiveness of CHX in infected root canals has been investigated in several studies. Investigators[315] 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.

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.[437]

In a recent study, the antibacterial efficacy of 2% CHX gel was tested against 2.5% NaOCl in teeth with apical periodontitis, with the bacterial load assessed using real-time quantitative polymerase chain reaction (RTQ-PCR) and colony forming units (CFU). The bacterial reduction in the NaOCl group was significantly greater than 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.[405] 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.[356,357]

Chlorhexidine as an Intracanal Medication

CHX in liquid, gel, or in a controlled-release device has been suggested as an alternative intracanal medication to replace $Ca(OH)_2$. This has been the focus of many in vitro[78,199,217,354]

*References 99, 208, 220, 356, 357, 386, 405, and 437.

and in vivo studies.* The results of in vitro experiments were mostly in favor of CHX regardless of its mode of application. The results have also demonstrated the potential antibacterial substantivity of CHX in the root canals.

Researchers[155] developed an experimental model using dentin powder particles to investigate the possible inactivation of some antibacterial medicaments when they come in contact with dentin. The medicaments tested were $Ca(OH)_2$, 1% NaOCl, 0.5% and 0.05% CHX acetate, and different concentrations of iodine potassium iodide (IKI). 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.

Contradicting results have been reported when evaluating the effect of different intracanal medications on sealing of the root canals. In an in vitro study using extracted human teeth, all root canals, after 10 days of intracanal medication, were obturated and then tested for microbial leakage. The root canals medicated with CHX gel demonstrated less resistance against bacterial leakage compared to the root canals medicated with $Ca(OH)_2$.[23] In contrast, Wuerch et al.[430] found no significant difference in leakage between the canals medicated with CHX and $Ca(OH)_2$ and reported that 2% CHX gel and $Ca(OH)_2$ paste did not adversely affect the apical seal of the root canal system.

In a human ex vivo model, the antibacterial efficacy of 0.2% CHX liquid applied for 24 hours was tested against a saline solution. After extraction, the infected teeth were endodontically treated using CHX or saline for irrigation and then further medicated with either of the solutions. Samples were obtained and evaluated using culture technique at each treatment step. In both groups, the numbers of bacteria were decreased after instrumentation and irrigation of the canals. However, after 24 hours of medication, it was reported that the numbers of bacteria were further decreased in the CHX group, in contrast to the saline group, where an increase was noted.[78] CHX may also have an effect on reduction of inflammatory external root resorption caused by infection. Lindskog et al.[228] assessed the therapeutic effect of a 4-week intracanal application of CHX gel on inflammatory root resorption in replanted infected teeth of monkeys. They reported that the extent of inflammatory resorption was significantly reduced compared to nonmedicated teeth, suggesting that CHX may be a useful adjunct in the management of inflammatory root resorption.

These in vitro and animal studies suggest that CHX has the potential to replace $Ca(OH)_2$ as an intracanal medicament, but the limitations of the in vitro experiments make the conclusions obtained difficult to extrapolate to actual clinical situations. In vivo human experiments are therefore required to test the efficacy of CHX as an intracanal medication.

Compared to the number of in vitro studies, very few in vivo studies have been conducted to assess the effectiveness of CHX as an intracanal medication. An in vivo investigation[20] 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. Using a culture method, it was reported that 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 $Ca(OH)_2$.[20]

Another in vivo study evaluated the antibacterial effectiveness of 2% CHX liquid as an intracanal medication in teeth with apical periodontitis.[293] The results showed 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 et al.[301] It was speculated that the CHX liquid may have partially escaped from the apical foramen, and that the higher-viscosity gel form might have been better suited as an intracanal medication.[293]

However, a different study[239] demonstrated that 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. The results were not significantly different among the medication groups. Similar results were found by other investigators,[238] where after a randomized controlled trial of 30 patients, they concluded that a final rinse with MTAD (*m*ixture of *t*etracycline, *a*cid, and *d*etergent) and intracanal application of 2% CHX gel did not reduce bacterial counts beyond levels achieved by chemomechanical preparation using NaOCl.

Chlorhexidine and Calcium Hydroxide

During the last few years, researchers have studied the combination of $Ca(OH)_2$ and CHX, with the concept that their antimicrobial properties interact in a synergistic fashion that enhances their efficacy. The high pH of $Ca(OH)_2$ was unaffected when combined with CHX.[26] However, the results have not been conclusive. Some in vitro studies have reported an improved antibacterial action when both agents were combined,[28,102,438] while other studies reported conflicting results.[157]

Recent animal studies have evaluated the tissue reactions to the mix of $Ca(OH)_2$/CHX, showing that the combination exerts good antimicrobial properties[364] and improves healing of the periapical tissues. In vivo studies have shown that the mix is at least as good as both agents applied separately in necrotic teeth with apical periodontitis,[239] as well as in previously treated cases with persistent apical periodontitis. A more recent study utilizing a CHX-based protocol of 0.12% CHX as an irrigant followed by a 7-day intracanal medication of $Ca(OH)_2$/0.12% CHX has shown promising results. The authors concluded that chemomechanical canal shaping using 0.12% CHX solution as an irrigant significantly reduced the number of intracanal bacteria, but it failed to render the canals bacteria free. Further intracanal medication with a $Ca(OH)_2$/CHX paste significantly improved the results by reducing the number of bacteria.[353] Taken together, it seems that the usefulness of mixing $Ca(OH)_2$ with CHX remains unclear and controversial.

Chlorhexidine and Coronal Penetration of Bacteria

Because of its antimicrobial substantivity, it seems that CHX preparations delay entry of bacteria through the coronal

*References 20, 70, 83, 98, 228, 239, 293, and 441.

portion of the tooth into the root canal system. A laboratory study[138] 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. The canals without a coronal restoration but medicated with CHX showed recontamination after an average time of 3.7 days, the group with $Ca(OH)_2$ after 1.8 days, and the group with CHX + $Ca(OH)_2$ after 2.6 days. The canals medicated with CHX and restored with intermediate restorative material (IRM) showed recontamination within 13.5 days, the group with $Ca(OH)_2$ + IRM after 17.2 days, and the group with CHX + $Ca(OH)_2$ + IRM after 11.9 days. The group with no medication but restored with IRM showed recontamination after an average time of 8.7 days. There were statistically significant differences between the groups ($P < 0.05$). All groups without a coronal restoration were recontaminated significantly more quickly than those restored with IRM, except those teeth that had a restoration but no medicament. The groups with intracanal medication and a coronal restoration were not significantly different from each other.

An ex vivo study[408] assessed coronal dye penetration of extracted human teeth after root canal treatment using 1% NaOCl, 1% NaOCl + 17% ethylenediamine tetra-acetic acid (EDTA), 2% CHX gel, 2% CHX gel + 1% NaOCl and distilled water. Results revealed that the least dye penetration occurred with 1% NaOCl + 17% EDTA and 2% CHX gel. NaOCl, distilled water, and 2% CHX gel + 1% NaOCl had more dye penetration with a significant difference compared with NaOCl + 17% EDTA and 2% CHX gel and compared with one another.

Lambrianidis and colleagues[210] have shown that viscous irrigants, including those containing CHX gluconate, were less soluble substances that can leave residue on root canal surfaces and potentially affect root canal obturation. The efficiency of removing $Ca(OH)_2$/CHX gel, $Ca(OH)_2$/CHX solution and $Ca(OH)_2$/saline pastes with the use of instrumentation and irrigation with NaOCl and EDTA solutions has been assessed. None of the techniques used in this study removed the interappointment root canal medicaments effectively. Overall, $Ca(OH)_2$/CHX (gel) paste was associated with significantly larger amount of residue, whereas the $Ca(OH)_2$/CHX mixture was associated with less residue than the other two medicaments. Overall, because of its substantivity, CHX as an intracanal medicament/irrigant may delay the coronal recontamination of the root canal system.

Interaction Between CHX, NaOCl, and EDTA

A suggested clinical protocol[439] for treating dentin prior to root canal obturation consists of irrigation with NaOCl to dissolve the organic components, irrigation with EDTA to eliminate the smear layer, and irrigation with CHX to increase the antimicrobial spectrum of activity and impart substantivity. Although such a combination of irrigants may enhance the overall antimicrobial effectiveness,[208] possible chemical interactions between the irrigants must be considered. Some studies have reported the occurrence of color change and precipitation when NaOCl and CHX are combined[26,439] (Fig. 8-48). 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. The formation of a precipitate could be explained by the acid-base reaction that occurs when NaOCl and CHX

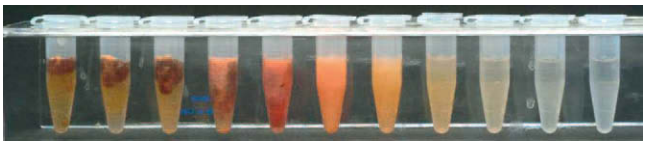


FIG. 8-48 Color change and precipitation occur when NaOCl and CHX are combined.

are mixed together. CHX, a dicationic acid, has the ability to donate protons. NaOCl is alkaline and can accept protons from the dicationic acid. This proton exchange results in the formation of a neutral and insoluble substance referred to as the *precipitate*.[26] Basrani et al.[26] evaluated the chemical nature of this precipitate and reported that there was an immediate reaction when 2% CHX was combined with NaOCl, even at the low concentration (0.023%). They found that the amount of PCA directly increased with the increasing concentration of NaOCl. PCA has been shown to be toxic in humans with short-term exposure, resulting in cyanosis, which is a manifestation of methemoglobin formation. In another study, the effect on root dentin and dentinal tubules was evaluated subsequent to irrigating root canals with a combination of NaOCl and CHX. They concluded that the NaOCl/CHX precipitate tends to occlude the dentinal tubules and suggested that until this precipitate is studied further, caution should be exercised when irrigating with both NaOCl and CHX. In summary, the combination of NaOCl and CHX causes color changes and formation of a neutral and 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.[439]

The combination of CHX and EDTA produces a white precipitate, so a group of investigators[311] 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.

Dentin Bonding of Chlorhexidine

During the last 2 decades, chemical and technical advances have contributed to increases in resin-dentin bond strength. However, the premature loss of bond strength is one of the problems that still affects adhesive restorations[270] and markedly reduces their durability.[54,80,117] Researchers[53] evaluated the effect of CHX on resin-dentin bond stability ex vivo. 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.[53] Because of its broad-spectrum matrix metalloproteinase (MMP)-inhibitory effect, CHX may significantly improve resin-dentin bond stability.

Allergic Reactions to Chlorhexidine

CHX, although reported to be a relatively safe solution, may induce allergic reactions. The sensitization rate has been reported in several studies to be approximately 2%.[202] One case of an anaphylactic shock was reported after the application of 0.6% CHX to intact skin that had signs of a rash following a minor accident.[15] Further allergic reactions such as anaphy-



FIG. 8-49 BioPure MTAD. *(Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

laxis, contact dermatitis, and urticaria have been reported following direct contact to mucosal tissue or open wounds.[92,303,340,363] There are no publications reporting allergic reactions following root canal irrigation with CHX.[181]

MTAD and Tetraclean

Recently, MTAD (Fig. 8-49) and Tetraclean, two new irrigants based on a mixture of antibiotics, citric acid, and a detergent, have been developed. MTAD is the first irrigating solution created which is capable of removing the both the smear layer and disinfecting the root canal system.[392] It is a mixture of 3% doxycycline hyclate, 4.25% citric acid, and 0.5% polysorbate-80 (Tween 80) detergent.[392] Commercially available as BioPure MTAD (DENTSPLY Tulsa Dental, Tulsa, OK), 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.*

Tetraclean (Ogna Laboratori Farmaceutici, Muggio, Italy) is another 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

There is no detailed information on the exact mechanism of action of MTAD in the removal of the smear layer and the killing of bacteria. In most studies, its effect on the smear layer is attributed to the existence of doxycycline and citric acid. These two components in solution have been separately reported as being effective in the removal of the smear layer.[163] Its antibacterial effect is mostly attributed to the doxycycline, an isomer of tetracycline. Tetracyclines—including tetracycline HCl, minocycline, and doxycycline—are broad-spectrum antibiotics that are effective against a wide range of microorganisms. Tetracycline is a bacteriostatic antibiotic which exerts its effect through the inhibition of protein synthesis. According to Torabinejad et al.,[393] this property may be advantageous because in the absence of bacterial cell lysis, antigenic byproducts (i.e., endotoxin) are not released. In high concentrations, tetracycline may also have a bactericidal effect. The role of citric acid in killing bacteria is not well known. Tween 80, the other component of MTAD, seems to have limited antibacterial activity, yet it may increase the antibacterial effect of some substances by directly affecting the bacterial cell membrane. It may also facilitate the penetration of MTAD into dentin. On the contrary, Tween 80 may also be a nutrient for some bacteria, and it may inactivate the antibacterial properties of some disinfecting agents such as CHX and povidone iodine. 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.

*References 29, 31, 346, 347, 392, and 393.

Surface Tension

According to Grossman and Meiman,[150] low surface tension is one of the ideal characteristics of an irrigant. Lower surface tension may help by allowing the irrigant to penetrate better into the dentinal tubules and inaccessible areas of the root canal system.[388]

To be more effective in debris removal and to penetrate more readily into the root canal system, irrigants must be in contact with the dentin walls. The closeness of this contact is directly related to its surface tension.[133] Irrigants with a low surface tension are more suitable as endodontic irrigants. To decrease the surface tension, Tween 80 has been added to the MTAD solution.

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.[389,390]

Antibacterial Efficacy

Reported results regarding the antibacterial properties of MTAD are conflicting. The studies measuring zones of inhibition on agar plates have shown consistently that MTAD was an effective antibacterial agent against *E. faecalis*.[73,201,393] Tay et al.[389,390] also found larger zones of bacterial inhibition using dentin cores irrigated with MTAD compared to NaOCl-irrigated dentin cores. When they applied MTAD to dentin that was already irrigated with 1.3% NaOCl, however, they noticed a contradictory result: the diameters of the zones of inhibition were significantly smaller than those of MTAD alone but comparable to those irrigated with 1.3% NaOCl alone. They concluded that the antimicrobial effect of MTAD was lost due to oxidation of the MTAD by NaOCl.

A study using extracted human teeth contaminated with saliva showed that MTAD was more effective than 5.25% NaOCl in disinfection of the teeth.[347] In contrast, Krause et al.,[201] using bovine tooth sections, showed that 5.25% NaOCl was more effective than MTAD in disinfection of dentin discs inoculated with *E. faecalis*.

In another study performed on extracted human teeth inoculated with *E. faecalis,* a protocol of 1.3% NaOCl followed by 5 minutes of MTAD was more effective in the disinfection of canals than a protocol of 5.25% NaOCl followed by 1 minute of 17% EDTA and then 5 minutes of 5.25% NaOCl as a final rinse.[347] In a series of studies, MTAD has failed to show superior antibacterial efficacy against bacterial biofilms. Bacteria collected from the teeth of patients diagnosed with apical periodontitis were grown as a biofilm on hemisections of root apices. MTAD was an effective antibacterial agent in this model, but it was unable to completely disrupt the bacterial biofilm, compared to 6% NaOCl. NaOCl (5.25%) was the most

effective irrigant against a biofilm of *E. faecalis* generated on cellulose nitrate membrane filters, whereas the bacterial load reduction using MTAD was not significant.[134] The results of testing antibacterial efficacy of medicaments obtained from in vitro studies should be analyzed with caution. They may be influenced by factors such as the test environment, bacterial susceptibility, and the different methodologies used to evaluate the results.[354]

In vivo Clinical Trial

With the exception of one study[394] that evaluated the effect of MTAD on postoperative discomfort, there have been no other in vivo studies to address the other characteristics of MTAD. Malkhassain et al.[238] 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 chemomechanical preparation using NaOCl alone.

Protocol for Use

The clinical protocol for the use of MTAD *was developed on the basis of a pilot project*.[346] The results of this project showed consistent disinfection of the infected root canals could occur after chemomechanical preparation using 1.3% NaOCl as a root canal irrigant followed by a 5-minute rinse of MTAD.

Ethylenediamine Tetra-Acetic Acid

EDTA is often suggested as an irrigation solution because it can chelate and remove the mineralized portion of the smear layer (Fig. 8-50). It is a polyaminocarboxylic acid with the formula $[CH_2N(CH_2CO_2H)_2]_2$. This colorless, water-soluble solid is produced on a large scale for many applications. 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.

History

The compound was first described in 1935 by Ferdinand Munz, who prepared the compound from ethylenediamine and chloroacetic acid. Today, EDTA is mainly synthesized from ethylenediamine (1, 2-diaminoethane), formaldehyde (methanal), and sodium cyanide.

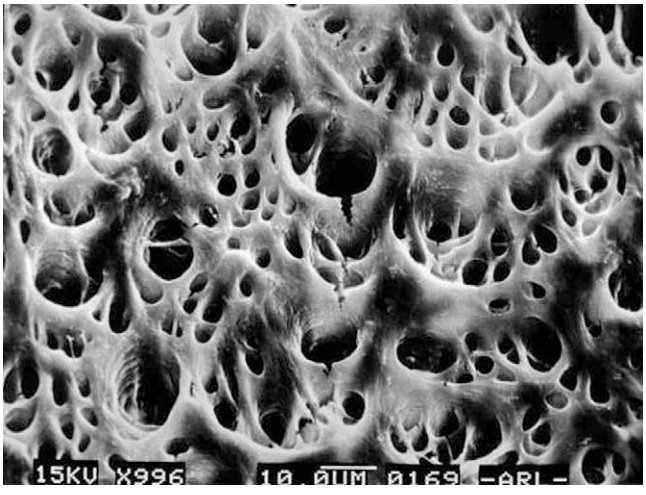


FIG. 8-50 Scanning electron microscope (SEM) image of root canal dentin exposed to 2 minutes of EDTA.

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.

Applications in Endodontics

EDTA alone normally cannot remove the smear layer effectively; a proteolytic component (e.g., NaOCl) must be added to remove the organic components of the smear layer.[136] Commercial products with such combinations are available. Endo-Dilator N-Ø (Union Broach, York, PA) is a combination of EDTA and a quaternary ammonium compound. Such an irrigation fluid has a slight detergent effect in addition to the chelating effect. Two newer irrigating solutions, MTAD (discussed earlier) and Smear Clear (SybronEndo), have recently been studied. Smear Clear is a clear, odorless, water-soluble solution containing water, 17% EDTA salts, a cationic surfactant (cetrimide), and anionic surfactants.

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. The decalcifying process is self-limiting. For root canal preparation, EDTA has limited value alone as an irrigation fluid. Since it is capable of decalcifying up to 50 µm, it can open up an occluded, very fine canal. This depth, added from two opposite canal walls, will result in a 100-µm opening, equivalent to the tip of a #010 file.

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.[151] 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.[151] Although never shown in a randomized clinical trial, an alternating irrigating regimen of NaOCl and EDTA may be more efficient in reducing bacterial loads in root canal systems than NaOCl alone. Antiseptics such as quaternary ammonium compounds (EDTAC) or tetracycline antibiotics (MTAD) 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.

Interaction of EDTA and NaOCl

Investigators[146] studied the interactions of EDTA with NaOCl. They concluded that EDTA retained its calcium-complexing 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.

Hydrogen Peroxide

Hydrogen peroxide (H_2O_2) has been used as an endodontic irrigant for years, mainly in concentrations ranging between 3% and 5%. It is active against bacteria, viruses, and yeasts.

Hydroxy free radicals (•OH) destroy proteins and DNA. The tissue-dissolving capacity of H_2O_2 is clearly lower than that of NaOCl.[330] When used in combination with NaOCl, bubbling will occur as a result of nascent oxygen being released through the chemical reaction between these two liquids. Generally this is no longer recommended as a routine irrigant.

Iodine Potassium Iodide

IKI, used as a solution of 2% iodine in 4% potassium iodide, has been proposed and is used as an endodontic disinfectant because of its excellent antibacterial properties and low cytotoxicity. However, it may act as a severe allergen[306] and also stains dentin.[330]

Choosing the Ideal Irrigant

Although iodine is less cytotoxic and irritating to vital tissues than NaOCl and CHX,[370,371] it has a higher risk of causing an allergic reaction.[306] The same is true for quaternary ammonium compounds. Sensitivities to hypochlorite and CHX are rare.[439]

Of all the currently used substances, NaOCl appears to be the most ideal because it covers more of the requirements for endodontic irrigant than any other known compound. Hypochlorite has the unique capacity to dissolve necrotic tissue and the organic components of the smear layer. It kills sessile endodontic pathogens organized in biofilms and in dentinal tubules as efficiently as CHX or iodine at comparable concentration.[290,373,401] Inactivation of endotoxin by hypochlorite has been reported, but the effect is minor compared to that of a $Ca(OH)_2$ dressing.

Intracanal Medication

When treatment cannot be completed in one appointment [see Chapters 4 and 15], the surviving intracanal bacteria often proliferate between appointments.[47,415] To curtail bacterial regrowth and possibly even improve bacterial suppression, an intracanal medication can be advantageous. Interappointment antimicrobial medication acts to inhibit proliferation and further eliminate surviving bacteria, as well as minimize ingress through a leaking restoration.

Phenolic Preparations

Phenol (C_6H_5OH), or carbolic acid, is one of the oldest antimicrobial agents used in medicine. 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. One survey noted a decrease in the use of classic phenolic intracanal medicaments with a corresponding increase in the use of $Ca(OH)_2$ or no medication.[128]

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%). At such a concentration, the antimicrobial effect in vivo is lower than optimal and of very short duration.[254]

Derivatives of phenol are stronger antiseptics and toxins than phenol. Phenolic compounds are often available as camphorated solutions. Camphoration results in a less toxic phenolic compound because it slows 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.[370,371] Phenols are ineffective antiseptics under clinical conditions.[46,96]

Formaldehyde

Formaldehyde, used as formocresol, has been used extensively in endodontic therapy despite its high toxicity and mutagenic and carcinogenic potential.[221] The formaldehyde component of formocresol may vary substantially between 19% and 37%. Tricresol formalin, another formaldehyde preparation, contains 10% tricresol and 90% formaldehyde. 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.[371,372]

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.

Halogens

Chlorinated solutions have been used for many years to irrigate root canals. They are also used as an intracanal dressing 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.[324] 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 disinfection of rubber dam and tooth surfaces during the preparation of an aseptic endodontic workfield.[272]

Calcium Hydroxide

Hermann introduced[166,167] the use of $Ca(OH)_2$ in endodontics in 1920. Although its use was well documented for its time, the clinical applications over the next 25 years were not well known. Its use in root canal treatment as an intracanal medication has been associated with periradicular healing and few adverse reactions. Its use in root canal treatment was promoted by a series of papers[46,48] documenting the antibacterial efficacy of $Ca(OH)_2$ in human root canals. Subsequent studies substantiated these reports,[290,360] and the routine use of $Ca(OH)_2$ as an interappointment intracanal medicament became widespread.[327]

$Ca(OH)_2$ cannot be categorized as a conventional antiseptic, but it kills bacteria in the root canal space. The value of $Ca(OH)_2$ in endodontic treatment of necrotic, infected teeth is now well documented. $Ca(OH)_2$ normally 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.

$Ca(OH)_2$ is a slow-acting antiseptic. Direct-contact experiments in vitro show that a 24-hour contact period is required for complete killing of enterococci.[324,360] 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.[350] 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 extraordinary ability to hydrolyze the lipid moiety of bacterial lipopolysaccharides (LPS), thereby inactivating the biologic activity of the lipopolysaccharide and reducing its effect.[322,323] This is a very 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.

$Ca(OH)_2$ may be mixed with sterile water or saline; this formula is also available commercially from a number of manufacturers in 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. 8-51). The mixture should be thick to carry as many $Ca(OH)_2$ particles as possible. This slurry is best applied with a Lentulo spiral. For maximum effectiveness, the root canal must be filled homogeneously to the working length. Saturated $Ca(OH)_2$ solution mixed with a detergent is an effective antimicrobial agent suitable for irrigation.

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.[233,358] Also, the removal of $Ca(OH)_2$ is frequently incomplete, resulting in a residue covering 20% to 45% of the canal wall surfaces, even after copious irrigation with saline, NaOCl, or EDTA.[211] Residual $Ca(OH)_2$ can shorten the setting time of zinc oxide eugenol–based endodontic sealers.[243] 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*.[416] Recently 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 $Ca(OH)_2$,[155,307] and one clinical study[301] has shown that the number of bacteria-positive canals actually increased after $Ca(OH)_2$ medication. 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.[301,415]

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.

Chlorhexidine

CHX is also used as an intracanal medication and has already been discussed extensively under its own heading.

Ledermix

Ledermix (Lederle Pharmaceuticals, Wolfratshausen, Germany) is a corticosteroid-antibiotic paste. Medicaments such as Ledermix paste have been recommended as routine intracanal medicaments. Ledermix paste has been advocated as an initial dressing, particularly if the patient presents with endodontic symptoms.

Schroeder developed the material that is now commercially marketed as Ledermix paste.[339] Ledermix paste contains triamcinolone acetonide as an antiinflammatory agent, at a concentration of 1%. When the Ledermix materials and other similar corticosteroid-containing formulations were commercially released, there was a considerable amount of opposition to their use.[195]

Ledermix paste is a nonsetting, water-soluble paste material for use as root canal medicament or as a direct or indirect



FIG. 8-51 Calcijet 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. *(Courtesy Centrix, Shelton, CT.)*

pulp capping agent. 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] 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. After the first 24 hours, 30% of the triamcinolone was released. By the end of 14 weeks, the remaining 70% had been released.

Immediate intracanal placement of Ledermix has been studied for replanted teeth, because it has been shown to inhibit root resorption after extended dry time. In a recent study done in dogs, 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).[57]

The Triple-Antibiotics Paste

The triple-antibiotics regimen, composed of metronidazole, ciprofloxacin, and minocycline, was first tested[328] 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.[391] The clinical effectiveness of the triple-antibiotic paste in the disinfection of immature teeth with apical periodontitis has been reported.[425] 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.

Bioactive Glass

Research is underway in the use of bioactive glass as an intracanal medicament. In one study,[440] 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. Some new obturating materials (e.g., Resilon [Pentron Clinical Technologies, Wallingford, CT]) contain bioactive glass.

New Irrigation and Disinfection Techniques and Devices

Other than conventional irrigation, additional techniques for endodontic disinfection have been proposed and tested, including laser systems and gaseous ozone. Recently several new devices for endodontic irrigation and/or disinfection have been introduced, among which are the EndoActivator System (DENTSPLY Tulsa Dental Specialties, Tulsa, OK), passive ultrasonic irrigation, EndoVac (Discus, Culver City, CA), the Safety-Irrigator (Vista Dental Products, Racine, WI), the self-adjusting file (ReDent, Raanana, Israel), photoactivation disinfection, and ozone. These new devices use pressure, vacuum, oscillation, and/or a combination with suction. For most of these devices and techniques, no data exists on the clinical efficacy, risk, frequency, and intensity of apical irrigant extrusion or the problems encountered with the use of negative pressure or oscillation.

EndoActivator

The EndoActivator System uses safe, noncutting polymer tips in an easy-to-use subsonic handpiece to quickly and vigorously agitate irrigant solutions during endodontic therapy (Fig. 8-52).

In a recent study,[84] the safety of various intracanal irrigation systems was analyzed by measuring the apical extrusion of irrigant. They concluded that EndoActivator had a minimal, although statistically insignificant, amount of irrigant extruded out of the apex when delivering the subsonic activation of the irrigant into the pulp chamber and canal. Manual, ultrasonic, and Rinsendo (Dürr Dental, Bietigheim-Bissingen, Germany) groups had significantly greater amount of extrusion compared with EndoVac and EndoActivator.

Passive Ultrasonic Activation

Ultrasonic devices were first introduced in endodontics by Richman.[312] Ultrasonically activated files have the potential to prepare and débride root canals mechanically. The files are driven to oscillate at ultrasonic frequencies of 25 to 30 kHz. They operate in a transverse vibration, setting up a characteristic pattern of nodes and antinodes along their length.[413,414] Unfortunately, it proved difficult to control the cutting of dentin during ultrasonic preparation, which resulted in apical perforations and irregular canal shapes.[235,380]

However, it has been shown that ultrasonically driven files are effective for the irrigation of root canals. Two types of ultrasonic irrigation have been described in the literature[6,420]: one where irrigation is combined with simultaneous ultrasonic instrumentation (UI) and another without simultaneous instrumentation, called *passive ultrasonic irrigation* (PUI). During UI, the file is intentionally brought into contact with the root canal wall. UI has been shown to be less effective in removing simulated pulp tissue from the root canal system or smear layer from the root canal wall than PUI. This can be explained by a reduction of acoustic streaming and cavitation.[6] Because the root canal anatomy is complex,[302] an instrument



FIG. 8-52 EndoActivator system. *(Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK.)*

will never contact the entire root canal wall.[429] Thus, UI could result in uncontrolled cutting of the root canal wall without effective cleaning.

Passive ultrasonic irrigation was first described by Weller et al.[420] The term *passive* does not adequately describe the process, since it is in fact active. When it was first introduced, the term *passive* related to the noncutting action of the ultrasonically activated file. PUI relies on the transmission of acoustic energy from an oscillating file or smooth wire to an irrigant in the root canal. The energy is transmitted by means of ultrasonic waves and can induce acoustic streaming and cavitation of the irrigant.[6] After the root canal has been shaped to the master apical file (irrespective of the preparation technique used), a small file or smooth wire (for example, size #15) is introduced in the center of the root canal, as far as the apical region. The root canal is then filled with an irrigant solution, and the ultrasonically oscillating file activates the irrigant. Since the root canal has already been shaped, the file or wire can move freely, and the irrigant can penetrate more easily into the apical part of the root canal system,[204] with the cleaning effect being more significant.[235] Using this noncutting methodology, the potential to create aberrant shapes within the root canal are reduced to a minimum. A file larger than a #15 or #20 will only oscillate freely in a wide root canal. A #25 file may in fact produce less acoustic streaming than a #15 and #20 file.[6] Consequently, using a file larger than a #20 may be considered fundamentally different from the basic principle of PUI. The cleaning efficacy of PUI implies the effective removal of dentin debris, microorganisms (planktonic or in biofilm), and organic tissue from the root canal. Because of the active streaming of the irrigant, its potential to contact a greater surface area of the canal wall will be enhanced.

If ultrasonic activation of the hypochlorite irrigant is to be used, it appears important to apply the ultrasonic instrument after canal preparation has been completed. A freely oscillating instrument will cause more ultrasound effects in the irrigating solution than a counterpart that binds to canal walls.[318] In addition, ultrasonic files can cause uncontrolled cutting of the canal walls, especially if used during preparation.[380] Therefore, it appears best to insert a slim, noncutting instrument in a controlled fashion after canal preparation.[402,403] As of recently, smooth wires fitting to an ultrasonic device have been commercially available. However, objective guidelines regarding their risks and benefits have not been ascertained.[439]

EndoVac

EndoVac is a combined irrigation/evacuation system. The irrigant is extruded from the system with pressure at the root canal orifice. The evacuator, a microcannula, extends to the apical region of the root canal; the dimensions of the needle are size #55 with a 2% taper. High-volume suction from the dental unit results in negative apical pressure and thus passively sucks the irrigant from the orifice to the apical part of the root canal, which is passively evacuated from the canal space. Apical extrusion of the irrigant will probably be reduced, since the canal is irrigated with negative (as opposed to positive) pressure.[283] There is little research to demonstrate this device is safe and effective when there is an open (immature) apex or in cases where there is a pathologic perforating root resorption.

A similar device has been presented by Fukumoto et al.[119,120] The irrigant is delivered by a needle (outer diameter 0.41 mm, inner diameter 0.19 mm) and a tubing pump (Masterflex, Cole Palmer Instruments, Vernon Hills, IL) to the coronal or middle part of the root canal and aspirated by a second needle (outer diameter 0.55 mm, inner diameter 0.30 mm), which is introduced into the apical part of the root canal. The aspiration pressure is −20 kPa. The irrigant is flushed from the coronal to the apical part of the root canal and there finally suctioned. This technique in an in vitro experiment on teeth with resected apices resulted in significantly less overextrusion of irrigant than a conventional irrigation technique with the needle tip placed 2 mm short of the apex. When the needle was placed 3 mm from the apex, the results did not significantly differ (Figs. 8-53 to 8-56).

Safety-Irrigator

The Safety-Irrigator 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. As of this printing, no information on its safety or efficacy is available (Fig. 8-57). Absent clear evidence of safety and efficacy through peer-reviewed literature, the reader is advised to use caution in choosing an irrigating device.



FIG. 8-53 EndoVac system for NaOCl delivery and vacuum evacuation. Open-ended microcannula. *(Courtesy Discus Dental LLC, Culver City, CA.)*

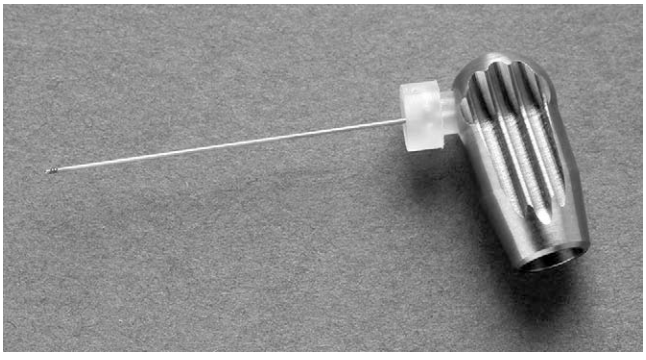


FIG. 8-54 EndoVac system for NaOCl delivery and vacuum evacuation. Closed-ended microcannula. *(Courtesy Discus Dental LLC, Culver City, CA.)*

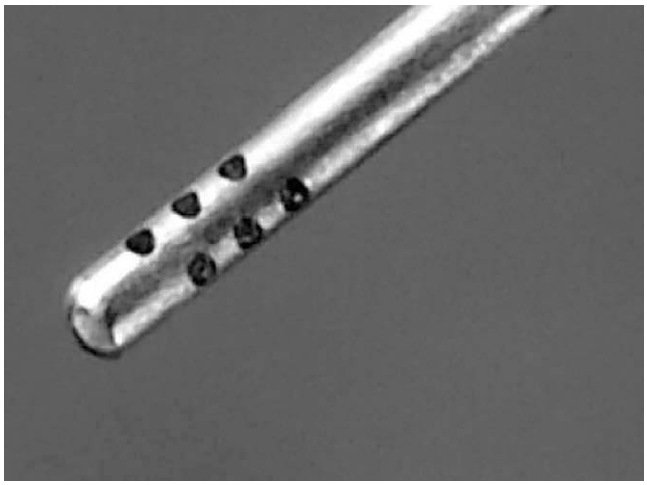


FIG. 8-55 EndoVac system: magnification of the closed-ended microcannula. *(Courtesy Discus Dental LLC, Culver City, CA.)*

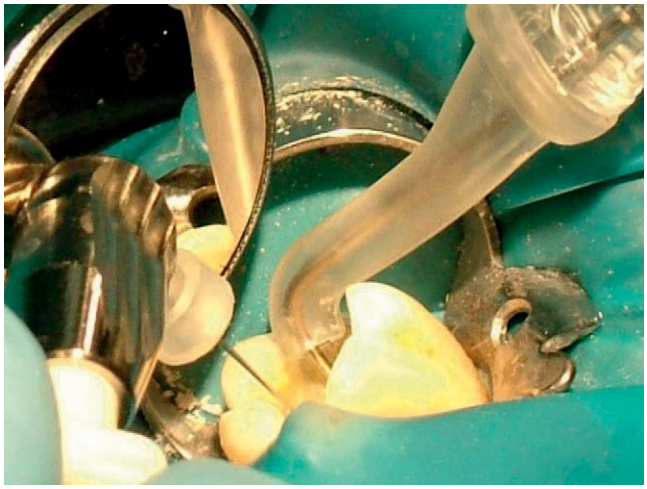


FIG. 8-56 Clinical use of the EndoVac system and Safety-Irrigator.



FIG. 8-57 The Safety-Irrigator. *(Courtesy Vista Dental Products, Racine, WI.)*

Self-Adjusting File

The SAF was recently introduced primarily as an endodontic file (discussed at length earlier under Instruments for Cleaning and Shaping the Root Canal Space). It should also be considered as an irrigation device since the file is hollow, which allows for continuous irrigation. The irrigant is delivered through a free-

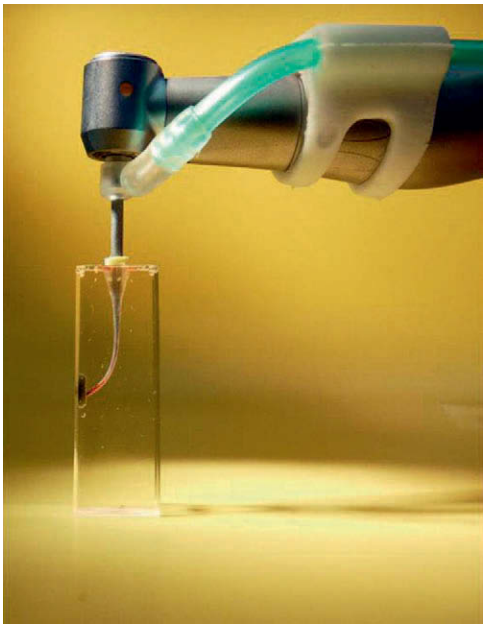


FIG. 8-58 The self-adjusting file (SAF; ReDent-Nova, Raanana, Israel). The hollow endodontic file is used also as a continuous irrigation device throughout the cleaning and shaping of the root canal. *(Courtesy ReDent-Nova, Raanana, Israel.)*

rotating hub to which a silicone tube is attached (Fig. 8-58). Either a special irrigation unit (see Fig. 8-30) (VATEA, ReDent, Raanana, Israel) or any physio-dispenser-type unit may be used to deliver a constant flow of irrigant at 5 ml/min. This maintains a continuous flow of fresh, fully active irrigant, facilitating an outflow of tissue debris and dentin powder that is generated by the file use. No positive pressure is thought to be created in the canal during this continuous irrigation procedure. The open metal lattice allows the irrigant to escape freely, minimizing the risk of irrigant transportation beyond the apical foramen.[260] The replacement of irrigation fluid in the apical part of curved canals is demonstrated in Fig. 8-59. Further research on this device is underway.

HealOzone

Ozone (also known as triatomic oxygen or trioxygen) is a naturally occurring compound consisting of three oxygen atoms. It is found in nature in the form of a gas in the stratosphere at a concentration of 1 to 10 ppm, being continually created from and destroyed into molecular O_2. The reliable microbiologic and metabolic properties of ozone, in either the gaseous or aqueous phases, make it a useful disinfectant with a wide range of activity. Ozone, in the gaseous or aqueous phase, has been shown to be a powerful and reliable antimicrobial agent against bacteria, fungi, protozoa, and viruses. It is generally accepted that the oxidant potential of ozone induces the destruction of cell walls and cytoplasmic membranes of bacteria and fungi. During this process, ozone attacks glycoproteins, glycolipids, and other amino acids and inhibits the enzymatic control system of the cell. This results in increased membrane permeability, the key element of cell viability, leading to immediate functional cessation. The ozone molecules then readily enter the cell and cause the microorgan-

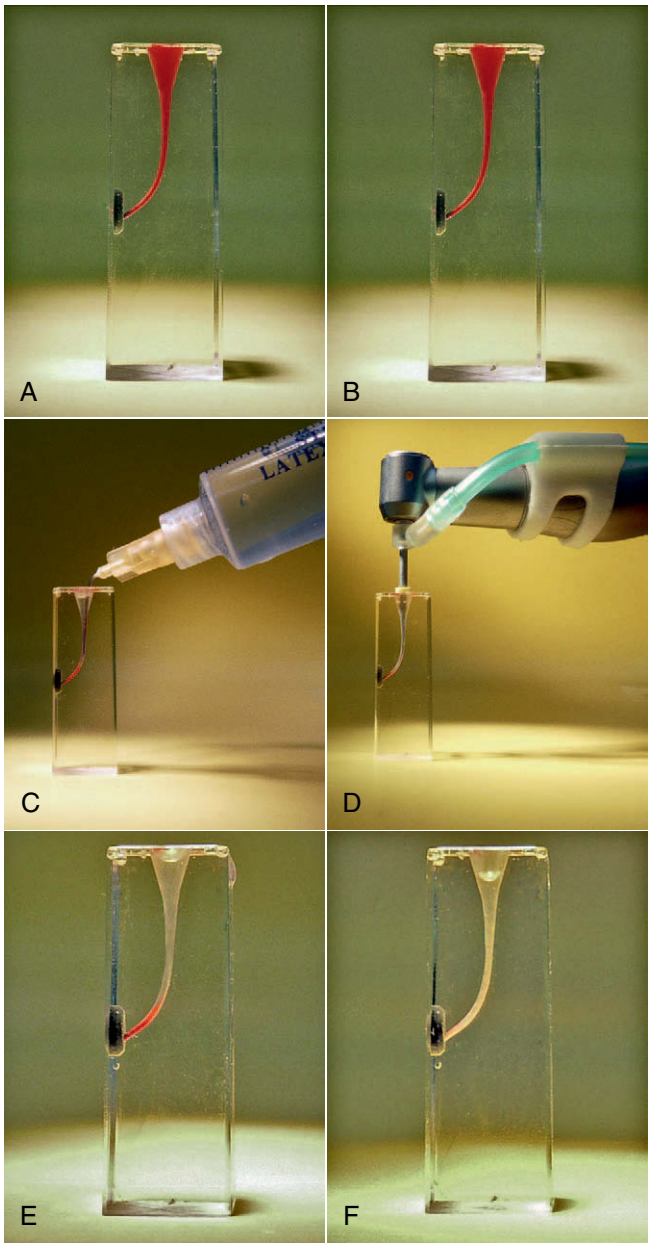


FIG. 8-59 Replacement of irrigation fluid in the apical part of curved canals. Transparent training blocks were filled with colored liquid **(A-B).** The canal was rinsed with either 20 ml of water using a syringe **(C)** or using the SAF instrument with a continuous flow of an equal amount of water **(D).** The short needle was unable to replace the liquid beyond the curve **(E),** but the SAF irrigation did it effectively **(F).**[260]

ism to die. Ozone can also attack many biomolecules, such as the cysteine, methionine, and histidine residues of proteins. By oxidizing the biomolecules featured in dental diseases, ozone has a severely disruptive effect on cariogenic bacteria, resulting in the elimination of acidogenic bacteria. The strongest naturally occurring acid—produced by acidogenic bacteria during cariogenesis—is pyruvic acid. Ozone can decarboxylate this acid to acetic acid. It has been shown that remineralization of incipient carious lesions can be encouraged when the production of acetic acid, or other high *p*Ka acids found in resting plaque, buffers plaque fluid.[245]

E.A. Fisch (1899–1966) was the first dentist to use ozonated water in his practice and introduced it to the German surgeon Erwin Payr (1871–1946) who used it from that time forward in surgery. He reported his results at the 59th Congress of the German Surgical Society in Berlin (1935). In dental surgery, ozonated water was used to promote hemostasis, enhance local oxygen supply, and inhibit bacterial proliferation. Theoretically, ozone can reduce the bacterial count in active carious lesions and therefore may temporarily arrest the progression of caries, resulting in preventing or delaying the need for tooth restorations.[313] For review on ozone use in dentistry see Azarpazhooh and Limeback.[17]

Ozone in Endodontics

Four in vitro studies[100,164,279,282] were identified investigating the bactericidal effect of ozone compared to 2.5% NaOCl, the standard irrigation solution in endodontics. The results of these studies[164,279,282] are controversial. The hazards of ozone when used in endodontics have not been investigated. Care should be taken with regard to the patient's and the dentist's exposure to the gas. Fig. 8-60 shows ozone equipment with a special tip for intracanal application.

Superoxidized Water

Superoxidized water,[152] also called electrochemically activated water[241,365] or oxidative potential water,[162,344] is saline that has been electrolyzed to form superoxidized water, hypochlorous acid, and free chlorine radicals. It is 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.[116] 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 upon 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 Japan (oxidative potential water).[240,241] Although the solutions bear different names, the principles in the manufacturing process are probably the same.

The use of superoxidized water is sparse in the endodontic literature but shows early promise. The solutions from both technologies have been tested for their ability to débride root canals,[162,241] remove smear layer,[344,365] and kill bacteria[172] and bacterial spores.[234] Results are favorable, while also showing biocompatibility with vital systems.[183]

Anolyte and catholyte solutions generated from one such technology (Radical Waters Halfway House 1685, S. Africa) have shown promise as antibacterial agents against laboratory-grown, single-species biofilm models.[132] Such solutions have been recommended as suitable for removing biofilms in dental unit water lines[240] 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.



FIG. 8-60 HealOzone equipment with a special tip for intracanal application. (*A, Courtesy KaVo Dental GmbH, Biberach/Riss, Germany.*)

Photoactivation Disinfection

Photodynamic therapy (PDT) or light-activated therapy (LAT) may have endodontic applications because of its antimicrobial effectiveness.[159] In principle, the strategy utilizes a selectively accumulated or endogenously produced photosensitizer (photoactive compound). The activation of photosensitizer molecules by a specific wavelength of light produces deleterious chemical entities that kill bacteria. The strong oxidizer generated during the process can act on multiple targets in a microbial cell, resulting in membrane damage, enzyme inactivation, and genomic and plasmid DNA damage, resulting in instantaneous death. In addition, the light-activated killing of bacteria has a wide spectrum of antimicrobial activity with a minimal chance of giving rise to a resistant microbial population, making it an ideal disinfectant. Fighting biofilm-mediated localized human infections is one of the potential medical applications of LAT.[410] The principal agent involved in bacterial killing is the oxygen-based free radical.[159] Unlike systemic therapeutic agents, LAT has the advantage of dual selectivity. Only those cells which have selectively accumulated the photosensitizer or possessing endogenously produced photosensitizers will be affected upon irradiation. Separately, the low-intensity light and the nontoxic photosensitizer generally used in LAT are harmless. In addition, the use of an optic fiber helps in directing the irradiation to the intended site of application. Studies have highlighted the possibility of killing antibiotic-resistant strains of pathogenic bacteria using LAT.[409,411] Because of the wide spectrum of antimicrobial activity, instant bacterial killing, and ability to target biofilm bacteria, the application of LAT-based regimens has great promise in root canal disinfection.[400] However, a thorough knowledge about the behavior of the photosensitizer and the physicochemical characteristics of the microenvironment prevailing in the root canal system is necessary for disinfection to be effectively achieved. In advanced stages of infection with mineralized biofilms, it may be necessary to do a minimal mechanical preparation of the root canal prior to LAT to achieve maximum disinfection.

IntraLight Ultraviolet Disinfection

Ultraviolet (UV) light is widely used for disinfection purposes. IntraLight (InterLight, Or-Yehuda, Israel) consists of an intracanal UV illuminator shaped with dimensions of an endodontic file, which allows uniform circumferential illumination of the root canal walls with 254 nm UV light. In a recent in vitro study,[255] root canals were infected with *E. faecalis* bacteria that

were allowed to grow and establish a biofilm on the root canal walls. Treatment with 5% NaOCl for 10 minutes failed to result in negative cultures in 53% of the canals. Similar results were reported by others.[255,257] An additional illumination of the canal walls with 254 nm UV light (300 mJ/cm^2) rendered 96% of the canals free of cultivable bacteria. A wide range of bacteria are sensitive to 254 nm UV light, including those that are resistant to $Ca(OH)_2$.[257] The device is used as a supplemental disinfection after NaOCl irrigation in the canal. Otherwise, bacteria in the inner layers of thick biofilms may be protected from the UV light, which is absorbed by bacteria in the outer layers.[257]

ROOT CANAL FILLING MATERIALS

Solid Materials

After the root canal system has been appropriately prepared, it must be obturated with a material capable of completely preventing communication between the oral cavity and the periapical tissue wound. Materials used for this purpose should be compatible with healing. This is achieved by attempting to seal the root canal system at both its coronal and apical ends, as well as throughout the canal system, to seal the openings of accessory canals. Apical and intracanal obturation blocks the exit to the periradicular tissues from organisms that have survived in the root canal after cleaning and shaping. Coronal obturation prevents reinfection of the pulp space from the oral environment. The required physical and biologic properties make the selection of obturation material critical. The materials commonly used for root canal fillings normally can be divided into a solid phase and a cementing medium (i.e., a sealer).

Gutta-Percha

Gutta-percha is the most commonly used root canal filling material. It is a linear crystalline polymer that melts at a set temperature, with a random but distinct change in structure resulting. (See Chapter 10 for further discussion of the physical properties of gutta-percha.) It occurs naturally as 1,4-polyisoprene and is harder, more brittle, and less elastic than natural rubber.

The crystalline phase has two forms, the alpha phase and the beta phase. The forms differ only in the molecular repeat distance and single-bond form. The alpha form is the material that comes from the natural tree product. The processed, or beta, form is used in gutta-percha for root fillings.[333] When heated, gutta-percha undergoes phase transitions. The transition from beta phase to alpha phase occurs at around 115° F (46° C). An amorphous phase develops at around 130° F to 140° F (54° C to 60° C). When cooled very slowly (i.e., 1° F per hour), gutta-percha crystallizes to the alpha phase. Normal cooling returns the gutta-percha to the beta phase. Gutta-percha cones soften at a temperature above 147° F (64° C).[139,333] These cones can easily be dissolved in chloroform and halothane and dissolve less in turpentine or xylene.

Modern gutta-percha cones that are used for root canal fillings contain only about 20% gutta-percha (Box 8-4). The major component is zinc oxide (60% to 75%). The remaining 5% to 10% consists of various resins, waxes, and metal sulfates. Antiseptic gutta-percha with various antimicrobial agents has been suggested, and several studies are available concerning the effect of these additives (see Medicated Gutta-Percha).

BOX 8-4

Composition of Gutta-Percha for Endodontic Use

Gutta-percha (19%–22%)
Zinc oxide (59%–79%)
Heavy metal salts (1%–17%)
Wax or resin (1%–4%)

Because gutta-percha cannot be heat sterilized, other decontamination methods must be used. The most practical method is to disinfect the gutta-percha in NaOCl before use. This can be done in 1 minute if the cone is submerged in a 5% solution of NaCOl.[342] However, after this disinfection and before its use for obturation, it is imperative that the gutta-percha be rinsed in ethyl alcohol to remove crystallized NaOCl; such crystals may impair the obturation seal.[349]

Obturation with gutta-percha normally requires some form of compaction pressure, but real compression of gutta-percha is practically impossible.[333] Pressure applied during root canal filling procedures does not compress gutta-percha, but rather compacts the gutta-percha cones to obtain a more three-dimensionally complete fill of the root canal system.

After heating, while cooling, there is a slight shrinkage of approximately 1% to 2% when the gutta-percha has solidified. Prevention of shrinkage is practically impossible in vertical warm compaction.[139,334] Careful control of the temperature during warm compaction is crucial to prevent focal areas of unnecessarily high temperatures. Better temperature control is available with electrically controlled heating devices as the Touch 'n Heat, System B, Elements units (SybronEndo, Orange, CA), or the recently introduced vibrating heat carrier, Down-Pack[67] (Hu-Friedy, Chicago, IL), or HotTip (Discus Dental, Culver City, CA), all of which are more commonly used for this purpose (see Figs. 8-41 through 8-44).

Gutta-percha oxidizes with exposure to air and light and eventually becomes brittle.[198,242] It should be stored in a cool, dry place (e.g., a refrigerator) for better shelf life. Methods of "rejuvenating" aged gutta-percha have been suggested.[189]

Gutta-percha cannot be used as the sole filling material; it lacks the adherent properties necessary to seal the root canal space. Therefore, a sealer (cement) is always needed for the final seal. (See the following sections on sealers.)

Manufacturers now supply gutta-perch cones in tapers matching the larger tapered rotary instruments (#.02, #.04, and #.06) (Fig. 8-61). An international standard has been accepted for gutta-percha cones, based on similar size and taper standards set forth for the endodontic files (ANSI No.78) (see Table 8-1). The tolerance is much less stringent for gutta-percha compared to files. An endodontic file must be manufactured with a tolerance of +0.05 mm. Consequently, with the same size instrument and gutta-percha cone, a difference in diameter of 0.07 mm (more than one file size) is possible (see Table 8-2 for gutta-percha cone sizes).

Gutta-percha has been extensively investigated as a root canal filling material in animals and has been proven biocompatible. Compared with sealers used for root canal obturation,

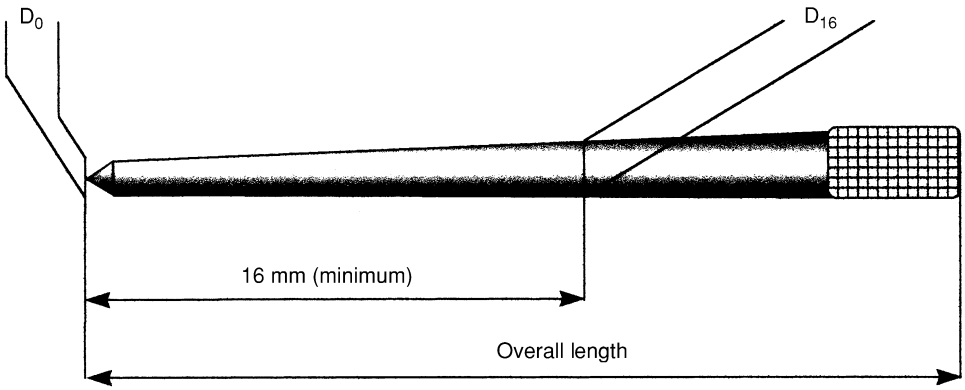


FIG. 8-61 Measurement points for gutta-percha cones as determined by ADA and ANSI standard #78.

it clearly has the lowest tissue toxicity. When compared to Resilon, both materials were equally biocompatible.[38] After subcutaneous implantation, gutta-percha normally becomes surrounded by a defined capsule rich in cells but without a significant number of inflammatory cells, although some macrophages are present.[37,288,362] Results from more sensitive assays in vitro support the in vivo results, suggesting that gutta-percha used for root canal fillings has a low toxicity.[295] Nevertheless, gutta-percha in the form of very small particles induces an intensive foreign body reaction, with massive accumulation of mononucleated and multinucleated macrophages.[362] This is not surprising, since materials that are normally considered inert (e.g., Teflon) cause similar reactions when presented to the tissues as an irregular surface or particle.[50]

Resilon

Resilon (Pentron Clinical Technologies, Wallingford, CT), a thermoplastic, synthetic, polymer-based root canal filling material, was developed in an attempt to create an adhesive bond between the solid-core material and the sealer. It is designed to be used with Epiphany (Pentron Clinical Technologies), a new resin sealer with a bonding capacity to dentin. Resilon can be supplied in the same ISO sizes and shapes (cones and pellets) as gutta-percha. The manufacturer has stated that it can be used with any current root canal obturation technique (lateral compaction, thermoplasticized, carrier, injection). When manufactured in cones, Resilon's flexibility is similar to that of gutta-percha. Based on polyester polymers, Resilon contains bioactive glass and radiopaque fillers (bismuth oxychloride and barium sulfate) with a filler content of approximately 65%. It can be softened with heat or dissolved with solvents such as chloroform. This characteristic allows the use of current retreatment techniques for nonhealing cases. Because it is a resin-based system, it is compatible with current restorative techniques in which cores and posts are placed with resin bonding agents (Figs. 8-62 to 8-64).[348]

Coated Gutta-Percha

Gutta-percha is now available that may achieve bonding between the solid core and a resin sealer. The uniform layer is placed on the gutta-percha cone by the manufacturer. When the material comes in contact with the resin sealer, a resin bond is formed. The manufacturer claims that this will inhibit leakage between the solid core and the sealer. The technique calls for use of EndoRez sealer (Ultradent, South Jordan, UT) with this new solid-core material (Fig. 8-65). The material with a resin sealer was compared with gutta-percha for microleakage. The results indicated a bonding between core and sealer resulting in far less microleakage than gutta-percha.[348] Another study tested the apical sealer of methacrylate-coated gutta-percha, and it caused the least amount of leakage.[445]

Medicated Gutta-Percha

The melding of an antibacterial substance to a gutta-percha cone or other solid-core obturation materials may have utility in preventing root canal therapy failures due to coronal or apical microleakage. In an early study, $Ca(OH)_2$ and CHX combined with zinc oxide and eugenol were found effective against the test organisms studied.[305] It is interesting to note that gutta-percha and Resilon appear to have no antibacterial or antifungal effects in and of themselves. When tested against five common organisms found in necrotic root canal systems, both materials proved not to be effective.

Several studies have attempted to examine gutta-percha cones which were formed with various antibacterial and antifungal preparations. In one study,[42] gutta-percha was impregnated with either CHX or $Ca(OH)_2$ and tested against eight organisms commonly found in infected root canal systems. CHX demonstrated inhibition against all organisms studied, while $Ca(OH)_2$ did not inhibit any of the same organisms. The antimicrobial effect of gutta-percha cones containing a mixture of both CHX and $Ca(OH)_2$ was found superior when compared the antimicrobial effectiveness of either substance when tested separately.[291] Another study testing CHX or $Ca(OH)_2$ with gutta-percha found CHX to be significantly more effective when tested in cultures of gingival fibroblasts.[383] When epithelial tumor cells and gingival fibroblasts were used to test gutta-percha cones containing either CHX or $Ca(OH)_2$, CHX samples showed significantly lower protein synthesis. Both tested materials induced cell growth–specific alterations.[422]

Early studies of medicated gutta-percha examined the use of iodoform in the solid-core material. While inhibitory against some pathogens, it was not inhibitory for all organisms.[351] A later study showed no differences between iodoform and regular gutta-percha samples in delaying microleakage of *E. faecalis*.[59] Gutta-percha discs impregnated with iodoform or other iodine combinations were tested against usual root canal system pathogens. Medicated gutta-percha inhibited all

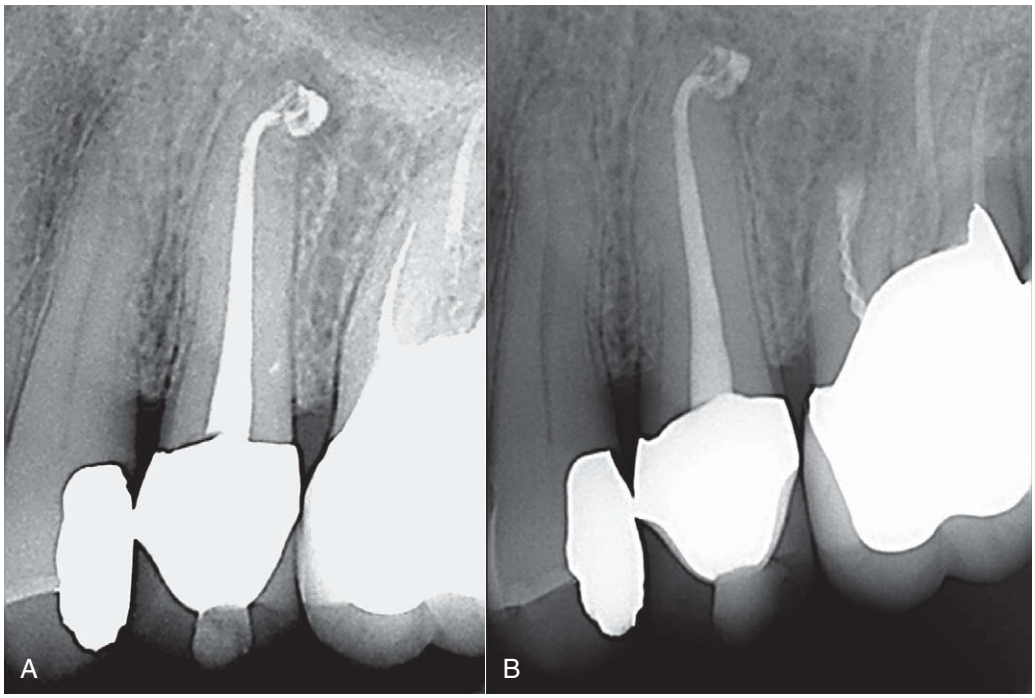


FIG. 8-62 A, Resilon/Epiphany cone root canal system obturation. **B,** Recall radiograph taken 8 months after initial treatment shows complete healing. *(Courtesy Dr. Richard Rubenstein, Farmington Hills, MI.)*

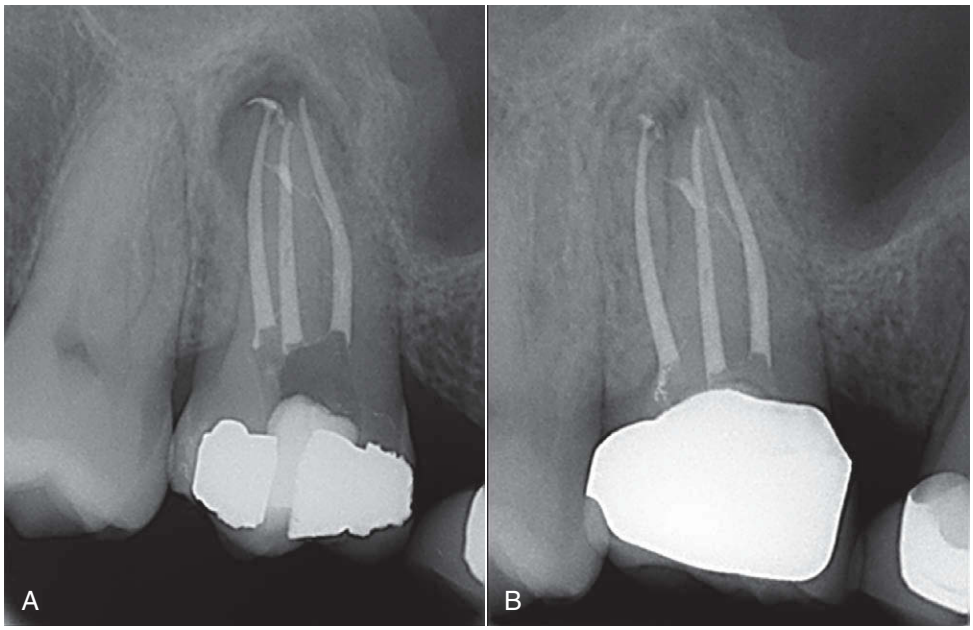


FIG. 8-63 A, Resilon/Epiphany root canal system obturation. **B,** Recall radiograph shows healing after 6 months. *(Courtesy Dr. Richard Rubenstein, Farmington, MI.)*

bacterial growth for 24 hours and was statistically more effective than regular gutta-percha.[35]

When tetracycline impregnation was compared to iodoform-medicated gutta-percha cones and Resilon and tetracycline discs, the impregnated cones inhibited growth for all species tested.[251] Tetracycline integrated with gutta-percha inhibited all test organisms when compared to conventional gutta-percha.[36]

Although effective in the cited studies, medicated gutta-percha cones are not used on a regular basis. More testing must be done to investigate their toxicity, antibacterial and antifungal potentials, and potential for allergic reactions.

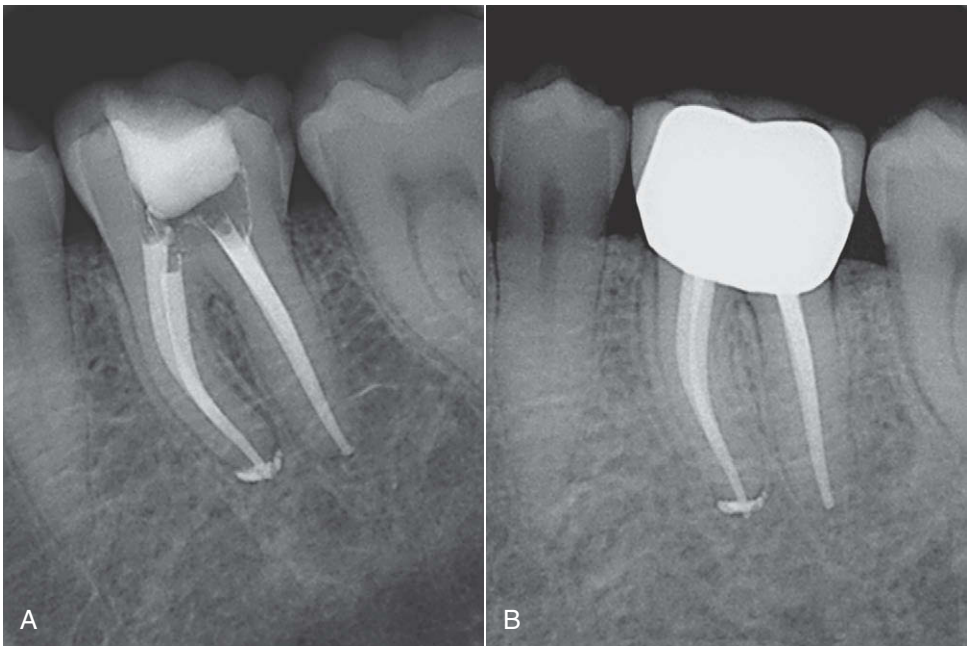


FIG. 8-64 A, Resilon/Epiphany root canal system obturation. **B,** Recall radiograph shows healing after 7 months. *(Courtesy Dr. Richard Rubenstein, Farmington, MI.)*

Sealers and Cements

Endodontic Sealers

The sealer plays an important role in the root canal filling. It fills all the space the solid-core material is unable to fill because of the solid core's physical limitations. A good sealer adheres strongly to the dentin and the core material. The sealer also must have cohesive strength to hold the obturation material together. Sealers ideally should be antimicrobial, which would be an important role in the success of root canal therapy. Sealers usually are made of a mixture that hardens through a chemical reaction. This reaction normally includes the release of toxic material, which makes the sealer less biocompatible. In general, the sealer is the critical component when the toxicity of material is assessed.

The sealer must have some degree of radiopacity to be visible on adequately exposed radiographs. Additives used to enhance radiopacity are silver, lead, iodine, barium, and bismuth. Compared with gutta-percha cones, most sealers have a slightly lower radiopacity and can be distinguished from gutta-percha alone or in combination with the sealer. A variety of sealers are available, and the clinician must be careful to evaluate all characteristics of a sealer before selecting one (Fig. 8-66, *A-F*).

Zinc Oxide Eugenol Cements

Many endodontic sealers are simply zinc oxide eugenol (ZnOE) cements that have been modified for endodontic use. The mixing vehicle for these materials is mostly eugenol. The powder contains zinc oxide that has been finely sifted to enhance the flow of the cement. The setting time is adjusted to allow for adequate working time. These cements easily lend themselves to the addition of chemicals; paraformaldehyde was sometimes added for antimicrobial and mummifying effects, germicides for antiseptic action, rosin or Canada balsam for greater dentin adhesion, and occasionally corticosteroids for suppression of inflammatory reactions. However, the use of formaldehyde is completely unacceptable, given the possibility of its circulation to other body tissues and organs. Corticosteroids are generally not included in sealers in the United States; it appears that their effect is limited by the amount contained in the preparation.[176]

Zinc oxide is a valuable component of the sealer. It is effective as an antimicrobial agent. The incorporation of rosins containing resin acids in sealers initially may have been for the adhesive properties.[148] Rosins (i.e., colophony), which are derived from a variety of conifers, are composed of approximately 90% resin acid. Resin acids are amphophilic, with the carbon group being lipophilic, affecting the lipids in cell membranes. Thus the resin acids have a strong antimicrobial effect that in mammalian cells is expressed as cytotoxicity. The resin acids work similarly to quaternary ammonium compounds by increasing the cell membrane permeability of affected cells. Although toxic, the combination of zinc oxide and resin acids overall may be beneficial. Under certain conditions, resin acids may react with zinc, forming a resin acid salt (i.e., resinate). This matrix-stabilized zinc resinate is only slightly soluble in water,[249] so ZnOE cements with resin components are less soluble than regular ZnOE cements.

The antimicrobial action of zinc oxide in both gutta-percha cones and many sealers create a low-level but long-lasting antimicrobial effect. In one study, a ZnOE sealer had a statistically significant larger mean zone of inhibition than three $Ca(OH)_2$ sealers.[264] When a new preparation of ZnOE sealer (Fill Canal) was tested, it showed large zones of inhibition against all microorganisms tested.[355]

The setting of ZnOE cements is a chemical process combined with physical embedding of zinc oxide in a matrix of zinc eugenolate. The particle size of the zinc oxide, pH, and the presence of water regulate the setting and other additives

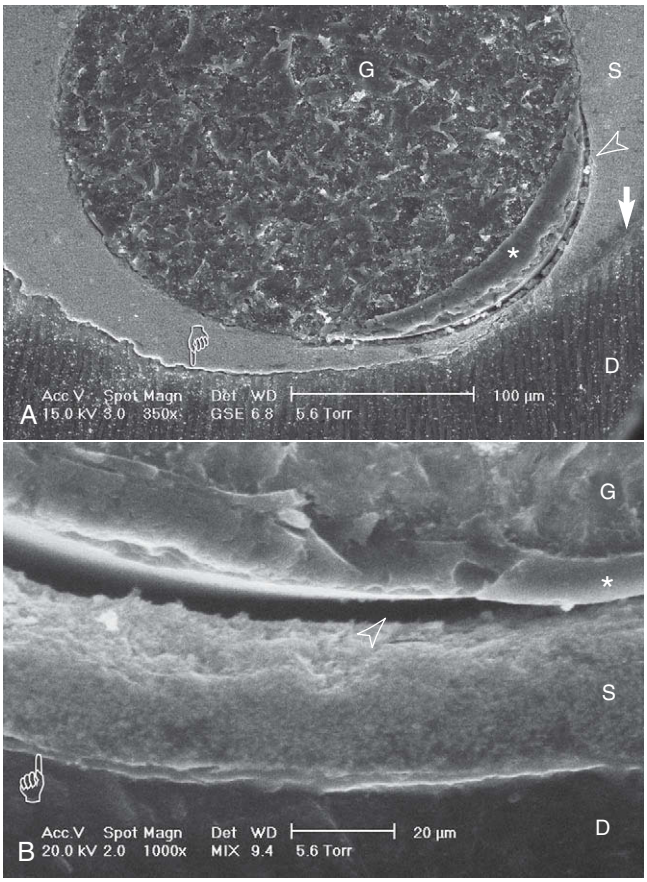


FIG. 8-65 A, EndoRez environmental scanning electron microscope (ESEM) image under hydrated conditions. When a gap appears, one can be certain that it is true, not an artifact. ESEM taken at relative humidity of 95%. *D,* Radicular dentin from coronal third of root canal wall; *G,* gutta-percha; *S,* EndoRez sealer; *asterisk,* proprietary coat in EndoRez GP cones; *open arrowhead,* true (not artifact) gap between root filling material and sealer; pointer: true gap along sealer-dentin interface; pointer: a gap-free region along sealer-dentin interface (×350). **B,** EndoRez ESEM, higher magnification of **(A),** labels are similar. *Pointer,* Hybrid layer probably caused by demineralization of radicular dentin with 0.5M EDTA; *open arrowhead,* between sealer and gutta-percha (×1000). *(Courtesy Dr. Franklin Tay, Medical College of Georgia, Augusta, GA.)*

that might be included in special formulas. The formation of eugenolate constitutes hardening of the cement, and $Ca(OH)_2$ accelerates this action, so canal systems containing $Ca(OH)_2$ must be well irrigated before obturation. Free eugenol always remains in the mass and acts as an irritant.

Several of the companies producing ZnOE root canal cements have been replaced by new companies, resulting in new names for older preparations such as Rickerts sealer, Procosol, and Wach's sealer. Fill Canal (Ligas Odontologicas, Sao Paulo, Brazil), Tubli-Seal (SybronEndo), and Pulp Canal Sealer (SybronEndo) are three successors to the original ZnOE cements and have been recently investigated. *E. faecalis* is a highly resistant organism found in necrotic root canal systems. That organism was exposed to two ZnOE sealers, Roth's 811 and Kerr EWT. Roth's exhibited the largest zone of inhibition.[263] Sultan, a ZnOE sealer, was one of the three most potent bacterial-growth inhibitors (*E. faecalis*).[61,304,353] When *Candida albicans* was used as the test organism, a ZnOE preparation demonstrated the largest zone of inhibition.[269] A direct contact test determined that the virulence of endodontic pathogens determine response to sealers.[298]

Historically, formaldehyde was commonly mixed into endodontic sealers, but formaldehyde is a dangerous additive to any sealer; it adds to the already toxic effect of eugenol and prevents or delays healing. Chapters 11 and 27 further discuss why potential major liabilities exist for any dentist who unwisely chooses to use formaldehyde or paraformaldehyde during endodontic therapy.

Chloropercha

Chloropercha was another type of sealer used for many years. It is made by mixing white gutta-percha (i.e., alba) with chloroform. This allowed gutta-percha root filling to fit better in the canal. However, chloropercha has no adhesive properties and is no longer used as an obturation material in root canal therapy. The use of chloroform has been sharply curtailed for many years because of its demonstrated toxicity.[436] In endodontics, the amounts normally used are insignificant and cause no detectable health hazard.[39,60,338] Nevertheless, the clinician must take prudent steps to reduce vaporization during use, because chloroform is highly volatile. When used to soften gutta-percha during revision of old root fillings, the chloroform should be dispensed through a syringe and hypodermic needle and only passively expressed into the area of the pulp chamber. For other uses, the exposure time, amount used, and chloroform surface exposed should be kept to a minimum.

Some chloroform substitutes, such as halothane and turpentine, are in use. Compared to chloroform, halothane is less effective at softening gutta-percha, is similarly hepatotoxic, and has a higher local toxicity (Table 8-4). For these reasons, halothane is not a good substitute. Turpentine is not carcinogenic but is reported to cause allergic reactions.[52,395] It has a high local toxicity and dissolves gutta-percha poorly. Therefore, no good substitute exists for the use of chloroform in endodontic procedures. With careful workplace hygiene, little risk is associated with the occasional use of minute amounts of chloroform in preparing customized master cones or when performing endodontic retreatments, provided no federal or state laws are violated.[21,244]

Calcium Hydroxide Sealers

Several $Ca(OH)_2$-based sealers are now commercially available, such as Sealapex (Sybron Endo), RealSeal (Sybron Endo) Apexit and Apexit Plus (Ivoclar Vivadent). These sealers are promoted as having therapeutic effects because of their $Ca(OH)_2$ content (Box 8-5). The antimicrobial effect of $Ca(OH)_2$ is thought to occur because of its ability to release hydroxyl ions and by having a high pH.[297] Short-term direct contact tests have shown Sealapex and Apexit to be mildly effective antimicrobial agents.

These sealers also have poor cohesive strength.[325] No evidence supports the contention that a $Ca(OH)_2$ sealer provides any advantage for root canal obturations or has any of the desirable biologic effects of $Ca(OH)_2$ paste. In a study of diffusion of hydroxyl ions into surrounding dentin after root filling with Sealapex and Apexit, no traces were found in teeth filled with Apexit. Some hydroxyl ions could be detected in the dentin close to the root filling with Sealapex.[378] In vivo

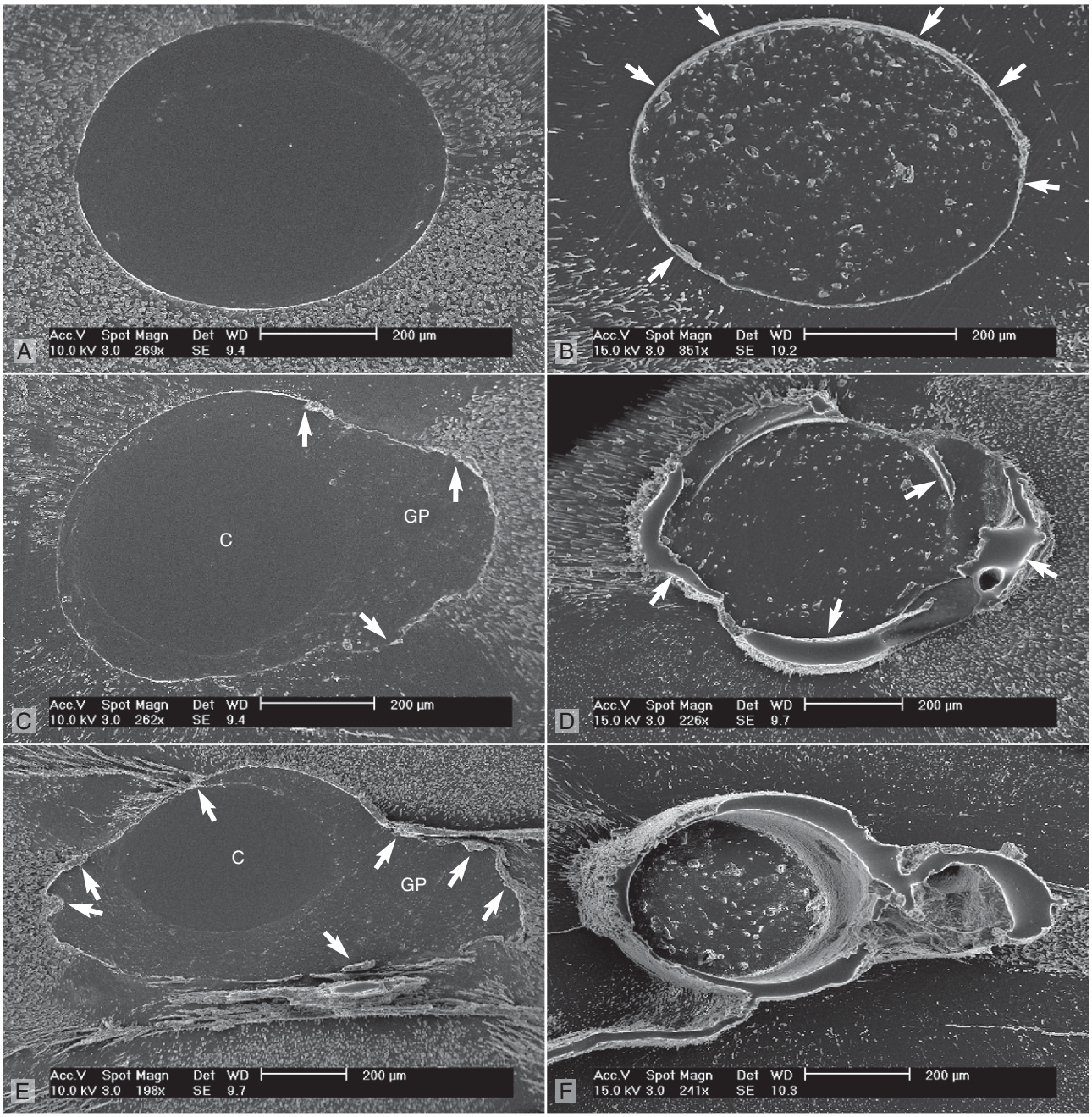


FIG. 8-66 Scanning electron microscope replicas comparing Resilon/Epiphany and Thermafil/AH Plus in variously shaped root canal systems. Impressions of the sectioned canals were taken with an ultraflow viscosity polyvinylsiloxane (PVS), and the replicas were directly examined. They are negative impressions of gaps, with PVS impression material flowing into the gaps. When the impression material was removed, a gap would appear as a flap of PVS material sticking out of the impression. All leaflets of PVS gaps appear blunt and stick out from the regions that represent root-filling material *(arrows)*. Two views of each canal morphology are shown. **A,** Thermafil/AH Plus (×269), round, narrow canal shape. **B,** Resilon/Epiphany (×351), round, narrow canal shape. **C,** Thermafil/AH Plus (×198), wider, irregular canal shape. **D,** Resilon/Epiphany (×241), wider, irregular canal shape. **E,** Thermafil/AH Plus (×198), narrow, ribbon-like (rotary instrument) shape. **F,** Resilon/Epiphany (×241), narrow, ribbon-like, (rotary instrument) shape. *(Courtesy Dr. Franklin Tay, Medical College of Georgia, Augusta, GA.)*

studies of Sealapex and CRCS (Coltene Whaledent, Altstätten, Switzerland) have demonstrated that these sealers easily disintegrate in the tissue, and both cause chronic inflammation.

Many studies have been conducted on various classes of root canal sealers, including $Ca(OH)_2$. The majority of these studies report that *all sealers leak*.[85] $Ca(OH)_2$ appears to be effective in formation of dentin bridges when used as a direct capping material,[4,107] which may indicate its ability to encourage formation of root-end hard tissue. It also appears to demonstrate acceptable antimicrobial activity. Success was seen at the same levels observed for mineral trioxide aggregate.[387]

TABLE 8-4

Toxic Effect of Gutta-Percha Solvents on L929 Cells In Vitro*

Time	Control	Chloroform	Halothane	Turpentine
AIR EVAPORATION				
Fresh Mix	7.2 ± 0.7	94.4 ± 3	102.5 ± 7.5	87.3 ± 2.2
1 day	10.1 ± 0.4	11.3 ± 1.8	12.6 ± 0.9	66.6 ± 5.3
7 days	9.5 ± 0.4	11.6 ± 0.9	14.8 ± 1	12.5 ± 2.8
LIQUID EVAPORATION				
Fresh Mix	7.2 ± 0.7	102.1 ± 6.4	98.4 ± 6.6	87 ± 2.1
1 day	10.1 ± 0.4	89.1 ± 6.7	103.9 ± 9	71.8 ± 4.7
7 days	9.5 ± 0.4	11.6 ± 5.8	10.1 ± 2.8	64.2 ± 3.9

*Gutta-percha (2.5 g) was dissolved in chloroform, halothane, or turpentine (5 ml). The cell injury was measured as mean percentage of release of ^{51}Cr (M ± SD) at various times after mixing. The solvents were allowed to evaporate in air or through a liquid layer. The higher the release, the higher the toxicity. (From Barbosa SV, Burkard DH, Spångberg LSW: Cytotoxic effect of gutta-percha solvents. *J Endod* 20:6, 1994.)

BOX 8-5

Composition of Calcium Hydroxide Sealers: Sealapex, CRCS, and Apexit*

Sealapex

Base
- Calcium hydroxide (25%)
- Zinc oxide (6.5%)

Catalyst
- Barium sulfate (18.6%)
- Titanium dioxide (5.1%)
- Zinc stearate (1%)

CRCS

Powder
- Calcium hydroxide
- Zinc oxide
- Bismuth dioxide
- Barium sulfate

Liquid
- Eugenol
- Eucalyptol

Apexit

Base
- Calcium hydroxide (31.9%)
- Zinc oxide (5.5%)
- Calcium oxide (5.6%)
- Silicon dioxide (8.1%)
- Zinc stearate (2.3%)
- Hydrogenized colophony (31.5%)
- Tricalcium phosphate (4.1%)
- Polydimethylsiloxane (2.5%)

Activator
- Trimethyl hexanedioldiasalicylate (25%)
- Bismuth carbonate basic (18.2%)
- Bismuth oxide (18.2%)
- Silicon dioxide (15%)
- 1,3-Butanedioldisalicylate (11.4%)
- Hydrogenized colophony (5.4%)
- Tricalcium phosphate (5%)
- Zinc stearate (1.4%)

*Proportions are unavailable for CRCS and incomplete for Sealapex.

Despite the favorable responses seen in the cited study, the results must be considered ambivalent and variable. Adhesion of the sealer to the prepared dentin wall and the solid-core obturation material also is a consideration. There is no creditable information that states that $Ca(OH)_2$ sealers in and of themselves provide adhesive forces. One study does state that use of an erbium-doped yttrium aluminum garnet (Er:YAG) laser does increase the adhesiveness of $Ca(OH)_2$ sealers.[27]

Polymers

Polymers are macromolecules composed of 10,000 or more atoms. Rubber has a polymeric structure based on a repeating isoprene (monomer). Polymeric molecules, discovered in nature (rubber), have led to synthetic analogs with a variety of properties and applications, such as adhesives (root canal sealers). While it is more convenient to discuss polymer sealers according to their classification or type, it may be more meaningful to describe them according to their ability to act in a particular manner based on what or how each group of sealers' characteristics are when used, such as their adhesiveness, sealing ability, and similar properties.

When considering the adhesiveness of a sealer, it must have the ability to adhere to both the prepared dentin wall and the solid-core obturation material used. As such, adhesiveness becomes very important in the material's ability to seal the prepared root canal system from coronally and apically, since the preparation procedures appear unable to sterilize the root canal space and at most may only disinfect the system.

Other significant sealer considerations include their biocompatibility, radiopacity, cytotoxicity, and bond strength.

Most of the newer sealers are polymers, including AH26 and AH Plus (DENTSPLY Caulk), Epiphany (Pentron Clinical Technologies), and EndoRez (Ultradent).

AH26

AH26 is an epoxy resin that was initially developed as a single obturation material. Because of its positive handling characteristics, it has been extensively used as a sealer. It has a good flow, seals well to dentin walls, and has sufficient working time.[224] Like most sealers, AH26 is very toxic when freshly prepared.[295] Earlier research indicated that AH26 toxicity was limited and principally caused by release of small traces of formaldehyde.[34,176] However, recent studies using more modern methods of analysis indicate that the sealer's mechanism for induced cytotoxicity may be due to activation of COX-2 in RNA gene expression,[175] leading to decreases in iNOS protein expression,[215] and the synergistic effects of root canal sealers on LPS induces COX-2 expression by macrophage cells.[214] Toxicity may decline rapidly during setting, and after 24 hours the cement may have one of the lowest toxicities of endodontic sealers.[187] A new formulation of AH26 is AH Plus. This is a two-paste mixing system that assures a better mixture and does not release formaldehyde upon setting.[63,218] It is more radiopaque and has a shorter setting time (approximately 8 hours), lower solubility, and a better flow compared with AH26. One study demonstrated that AH Plus had a lower short- and long-

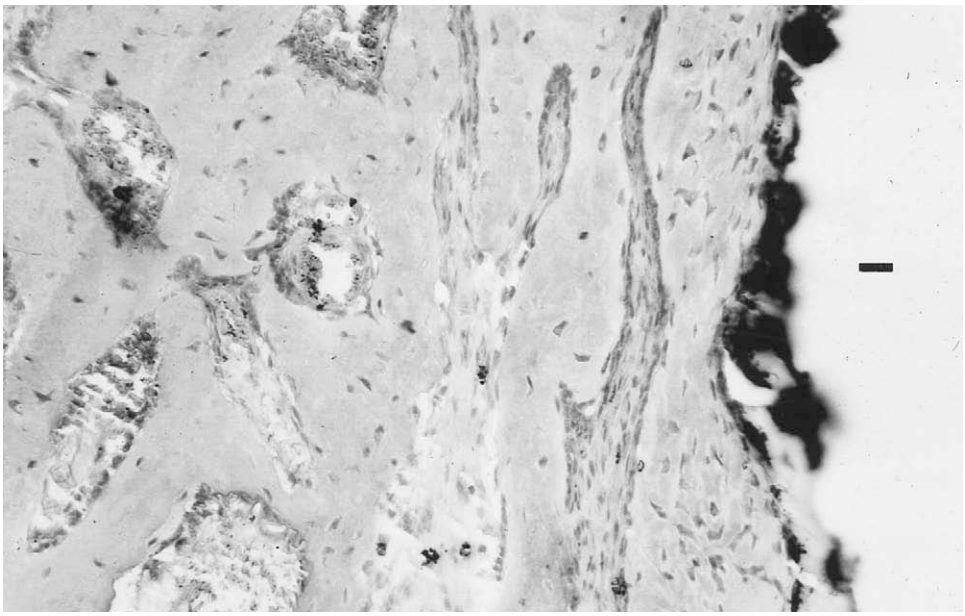


FIG. 8-67 AH26 implant in the mandible of a guinea pig 12 weeks after implantation. Bone has grown up and integrated with the implant. Remnants of the implant, which was lost during histologic preparation, can be seen as a black area at the top of the image. Complete healing was observed without signs of inflammation.

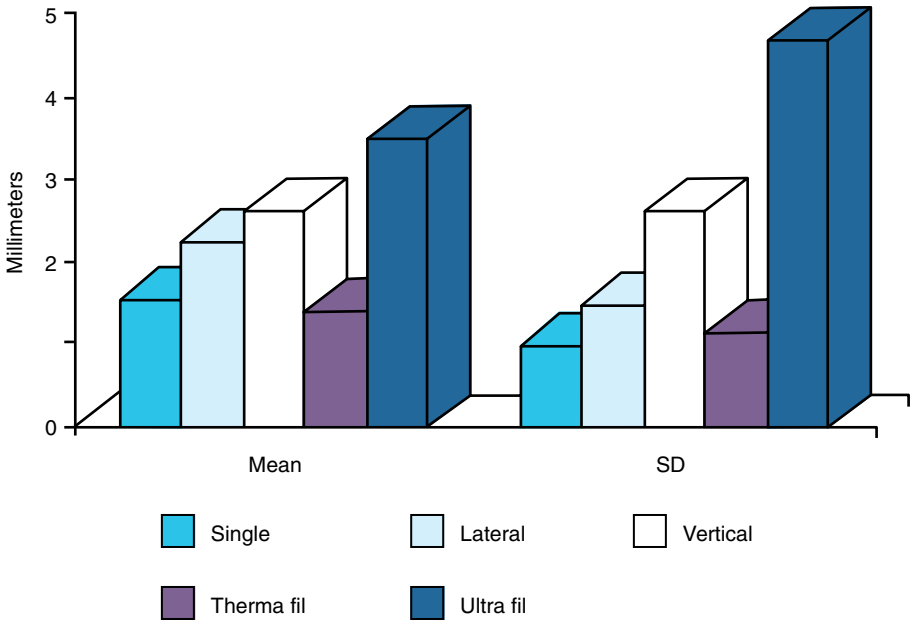


FIG. 8-68 Comparison of dye penetration after various obturation methods using AH26 sealer. The dye was delivered with a vacuum method, and penetration was measured as millimeters of leakage. The single cone technique and use of ThermaFil resulted in the least leakage, followed by lateral compaction, vertical compaction, and use of UltraFil. The standard deviation is high, as is common for leakage studies, reflecting the variability in the obturation method. The less complicated the obturation method, the lower the standard deviation.[69]

term toxicity level and was less genotoxic than AH26 (Figs. 8-67 and 8-68).[16,222]

Epiphany

Epiphany is a dual curable dental resin composite sealer composed of BisGMA, ethoxylated BisGMA, UDMA, and hydrophilic difunctional methacrylates with fillers of $Ca(OH)_2$, barium sulfate, barium glass, and silica. The total filler content of the sealer is approximately 70% by weight. Biocompatibility has been demonstrated both in vitro and in vivo, resulting in approval by the U.S. Food and Drug Administration. Epiphany was designed for use with Resilon instead of gutta-percha, although it can also be used with either core material. Unlike other resin sealers, this system's sealer requires a self-etch primer before placement of the resin sealer.[348] The newest iteration of the sealer utilizes a self-etching injectable paste that bonds to the prepared dentin walls and the solid-core material. Although not without controversy, used with Resilon cones, the subsequent obturated canal system may be fracture resistant.[182,423]

EndoRez

EndoRez is a UDMA resin-based root canal sealer with hydrophilic properties that allow good penetration into dentinal tubules. In addition to its biocompatibility,[442,445] it offers good radiopacity. EndoRez is as radiopaque as gutta-percha, which simplifies radiographic interpretation (Fig. 8-69).[444] The system includes a sealer, solid cores (resin-coated gutta-percha) and an accelerator and does not rely on heat to soften the material or pluggers to exert pressure to move the core apically. Much like Resilon/Epiphany, the bonding to both the dentin and solid core is thought to make the completed obturation resistant to fracture.[160]

Glass Ionomer Cement

Glass ionomer cements have been introduced as endodontic sealers (Ketac-Endo [ESOE]) and are known to cause little tissue irration,[443] with low toxicity in vitro. Questions remain about the quality of the seal achieved with Ketac-Endo, because dentin and sealer adhesive failures have been observed.[77] Some investigators have expressed concern about glass ionomers' solubility compared with other sealers.[331]

Silicon-Base Sealer

RoekoSeal (Roeko, Langenau, Germany) is a polydimethylsiloxane-based root canal sealer. One in vitro study comparing it with eugenol-based Sealapex or $Ca(OH)_2$-based Kerr's Pulp Canal Sealer concluded that RoekoSeal was less cytotoxic.[8] It has adequate solubility properties.[331] When its resistance to coronal and apical leakage was compared with that of AH26, no significant difference was observed.[61] With both sealers, removal of the smear layer improved the coronal and apical seal. Roeko recently introduced another version of its silicone sealer, GuttaFlow. It comes in a unidose capsule and is injected after mixing. The silicone is mixed with gutta-percha powder to form what the company calls a "two-in-one" cold filling system. GuttaFlow and RoekoSeal have recently been examined for spreadability on dentin and gutta-percha surfaces and compared to Roth 801 and AH26 sealers. Use of a sufficient load during lateral compaction is necessary for RoekoSeal and GuttaFlow to satisfactorily wet gutta-percha and dentin.[200] Microleakage and sealing ability also have been tested. When RoekoSeal and GuttaFlow were compared to other sealers, leakage patterns were found to be similar to other sealers.[75] GuttaFlow showed similar leakage results when used with lateral compaction or System B techniques, but it demonstrated expanding capacity over time compared to compacted gutta-percha and AH26 over a 12-month period.[200] When GuttaFlow was compared to two single-cone obturation systems, it was found that the apical seal was as effective as AH Plus used with vertical compaction.[274] However, in a dual-chamber leakage model with *Streptococcus mutans* as a biologic marker, thermoplasticized gutta-percha with AH Plus using vertical compaction was more effective in minimizing bacterial leakage.[273] Evaluating the long-term sealing ability of GuttaFlow compared to Epiphany, PCS, and AH Plus, GuttaFlow and Epiphany allowed less fluid movement.[41]

Sealers Containing Formaldehyde

A large group of endodontic sealers and cements had substantial paraformaldehyde additives. Some of the more common brands were Endomethasone, Riebler's paste (Amubarut; Wera Karl, Biensingen, Germany), and N2 (Indrag-Agsa, Bologne,

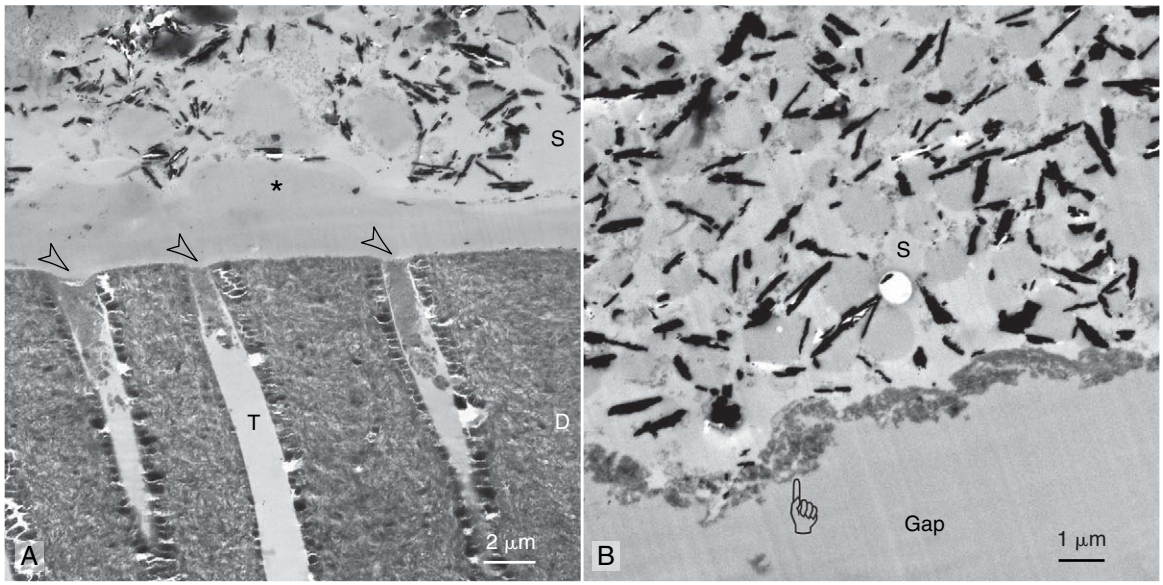


FIG. 8-69 Transmission electron microscope image illustrates the application of EndoRez sealer to smear layer–covered dentin (created by Profile nickel-titanium instruments) following use of 6.15% NaOCl initial rinse and sterile distilled water as final rinse. (Manufacturer recommends use of EDTA as final rinse.) **A,** A gap (asterisk) seen between the sealer (S) and radicular dentin (D). Smear plugs *(open arrowheads)* retained along orifices of dentin tubules (T). The smear layer was originally loosely attached to the canal wall dentin surface. EndoRez was applied to canal walls after NaOCl and distilled water irrigation, the resin portion of EndoRez (fillers) did not penetrate the loosely arranged smear layer particles. **B,** During polymerization of EndoRez, shrinkage stresses pulled the smear layer *(pointer)* away from the dentin, resulting in the smear layer being attached to sealer (S). Gap filled with epoxy resin used for embedding. *(Courtesy Dr. Franklin Tay, Medical College of Georgia, Augusta, GA.)*

Italy). All of these sealers have essentially the same toxicity. N2 is also known as *RC-2B* or the *Sargenti technique*. Historically, it has been heavily commercialized. There are no creditable studies available as to establish this material's use clinically. (Chapter 11 presents a more comprehensive discussion.) There are no clinical conditions to justify the use of these pastes.

N2 is basically a ZnOE sealer. Its composition has varied extensively over the years. The significant amount of lead oxide and smaller amount of organic mercury that were formerly major toxic components are often missing in more recent formulations. However, N2 still contains unacceptable amounts of formaldehyde.[44] Because it contains 6% to 8% paraformaldehyde (and sometimes hydrocortisone and prednisolone), it loses substantial volume when exposed to fluid. It also absorbs more than 2% of fluid during the first week in situ.

N2 is extremely toxic in experiments in vitro and in animal experiments. The tissue reaction normally observed is a coagulation necrosis within a very short time. The coagulated tissue is altered to such an extent that it cannot undergo any repair for months because it is impregnated with formaldehyde. With time, the formaldehyde may be washed out of the necrotic tissue, allowing either bacteria to be established in the necrosis or, if the blood supply is adequate, repair to take place.

The Interstate Commerce Commission of the U.S. Government has banned the transport of N2 across state lines, effectively halting the sale of these types of sealers. Please read Chapters 11 and 27 to gain a fuller understanding of the legal consequences of using these unacceptable pastes in unwary patients.

Standards and Properties

Physical Properties

Endodontic sealers are listed under ANSI/ADA specification number 57, Endodontic Sealing Material 2000 (reaffirmed 2006). This specification is for material used in endodontics within the tooth to seal the root canal space. This specification in an adoption of ISO6876:2001. ANSI/ADA Standard 57 outlines various test methods for evaluation of the physical properties of endodontic sealer-filling materials. Sealers are classified into two categories, depending on the intended use: type I materials are intended to be used with core materials; type II materials are intended for use with or without core material or sealer. Type I materials are divided into three classes. Class 1 includes materials in the form of powder and liquid that set through a nonpolymerizing process; class 2 includes materials in the form of two pastes that set through a nonpolymerizing process; class 3 includes polymer and resin systems that set through polymerization. The subclasses for type II materials are the same as for type 1 materials, except that metal amalgams are also included. ANSI/ADA No. 57 describes testing methods for working time, setting time, flow, film thickness, solubility, and disintegration; it also establishes a specific requirement for radiodensity. The tests for endodontic core materials include determinations of the material's dimensions, brittleness, and flow. A separate ANSI/ADA standard (No. 78) examines prefabricated metallic or polymeric-based cones used in obturation procedures. Despite the standard's often detailed requirements, significant disagreement exists on the ideal properties of endodontic sealers and fillers, so most of these expectations are guidelines that have not significantly affected the industry.

Biologic Properties

ANSI/ADA No. 41 recommends protocols for biologic evaluation of dental materials. This document covers recommended standard practices for biologic evaluation of the safety of materials used in dentistry and is not intended for use in the evaluation of pharmacologically active medicaments. This document outlines recommended test protocols for various dental materials, including certain guidelines for endodontic filling materials. These methods include general toxicity assessments (LD_{50}), cytotoxicity assessments in vitro, sensitization assays, mutagenicity assays, implantation tests, and usage tests. It was last updated in 2006. Several test methods are presented for each of these factors, depending on the type of material.

Root canal filling materials generally are toxic, and none fulfills the expectations set forth in ANSI/ADA No. 41. However, the methods described in this specification can be used to distinguish more toxic materials. Less toxic materials produce a less intense or shorter chemical insult to the remaining apical pulp or apical periodontium. If the wound area is free of bacteria when the initial chemical necrosis occurs, as results from sealers containing paraformaldehyde, tissue repair should occur as the initial irritant declines in intensity. Some tissue irritation may occur as a result of phagocytosis of particles of the material, but an expanding lesion would not develop.[362]

Endodontic sealers other than those that contain paraformaldehyde should not be implicated as the cause of a periradicular bone lesion. If the tissue in the apical root area is not sterile due to infection within the root canal system, presence of sealer should not cause a chemically induced periradicular necrosis. Thus, materials that cause extensive tissue necrosis inside the root canal may become vehicles for the development of failure of endodontic treatment. This supports the idea that treatment should focus on the proper application of asepsis and antisepsis, and that materials that are as biocompatible as possible should be used.

Sealers and cements are the very toxic component of gutta-percha root filling techniques. Therefore, the clinician must take great care in selecting materials and must have an understanding of what each material contributes to a disease process. ZnOE cements have a significant drawback in their release of free eugenol and loss of volume during the hydrolysis that takes place after setting. This is somewhat mitigated by its change into a relatively inert eugenolate substance within 24 to 48 hours. Several of the polymer materials have high toxicity during the polymerization phase but may become practically inert when polymerized. Sealers with inclusions of dissolvable components, such as $Ca(OH)_2$, lose these components in the tissue, resulting in compromise of the integrity of the obturation.

DELIVERY SYSTEMS FOR ROOT CANAL OBTURATION MATERIALS

The search for a more simplified and proficient obturation technique has led to the development of many new hybrid materials. Differences focus on alternative systems for introducing the solid-core material into the root canal system while

maintaining length control. The newer methods currently in use vary considerably. Most hybrid obturation methods require modification of the outline of the root canal preparation. One hybrid preparation technique involves the use of a rotating instrument that spins in the opposite direction of normal rotations and feeds the obturation material into the canal system instead of moving it out of the system. For optimal results, the clinician must consider technique variations before attempting a hybrid obturation method. A comparison of the more commonly practiced obturation methods, using an objective and sensitive vacuum dye penetration method, failed to show any major difference in the quality of the obturation.[69] However, dye penetration techniques have come into question in recent years. Other evaluation methods, such as dye extraction and fluid filtration techniques, seem to produce more consistent and credible data.[51]

The introduction of new materials, techniques, and delivery systems is challenging basic beliefs and concepts previously thought to be standard. For example, use of a single cone of gutta-percha with sealer historically has been considered unacceptable. However, with the introduction of new technology for cleaning and shaping and the development of innovative sealer delivery systems, a paradigm shift may be occurring toward improved canal preparation to receive more flowable sealers and a single insertion of a core material. To validate the long-term success of any of the obturation techniques, clinical studies to support evidence-based practice must be one of the goals of endodontic research.

Carrier-Based Systems

ThermaFil, ThermaFil Plus, ProTaper, and GT Obturators (DENTSPLY Tulsa Dental, Tulsa, OK) are obturation systems in which alpha-phase gutta-percha is preapplied to a flexible solid central carrier (Fig. 8-70). The gutta-percha obturator is heated in a proprietary heater (ThermaPrep Plus Oven, DENTSPLY Tulsa Dental) (Fig. 8-71) to the appropriate softness, and after the placement of canal sealer, the obturator is placed into the canal. These devices are available with a plastic, stainless steel, or titanium carrier core. In one study, ThermaFil demonstrated a greater percentage of canal obturation than either System B compaction or lateral compaction.[76] If necessary, the plastic core material can be softened with chloroform or heated using a System B[428] for easy removal. These obturators offer an alternative method of obturation with gutta-percha.

SimpliFill (Discus, Culver City, CA) (Fig. 8-72) is an obturation system designed to be used with the LightSpeed instrumentation system. The technique is based on a presized plug (5 mm long) of gutta-percha or Resilon which is attached to the end of a metal carrier. After sealer has been placed, the appropriate size SimpliFill is placed to working length, and the carrier is removed, leaving the apical segment obturated and the coronal segment open. Sealer is then injected with a syringe into the coronal segment, and a single cone (gutta-percha or Resilon) or post is inserted. In a study of coronal microbial leakage, an apical plug of SimpliFill in combination with FiberFill (Pentron Clinical Technologies, Wallingford, CT) in the coronal segment of canals was found to have significantly less leakage than lateral, vertical, and Obtura III (SybronEndo, Orange, CA) compaction techniques.[348] A later study found that SimpliFill samples demonstrated the most complete coverage in both coronal and apical sections when compared to



FIG. 8-70 ThermaFill obturators. Currently, obturators are provided to fit many of the individual types of files used in the canal. In this case: ProTaper instruments sizes F_3 and F_4 with their corresponding obturators. *(All rights owned by and used with permission from DENTSPLY International, Inc.)*



FIG. 8-71 ThermaPrep Plus oven. *(All rights owned by and used with permission from DENTSPLY International, Inc.)*



FIG. 8-72 SimpliFill system. Gutta-percha cone carried on a file-like metal handle. *(Courtesy Discus Dental LLC, Culver City, CA.)*



FIG. 8-73 Obtura gun for injectable gutta-percha. *(Courtesy Obtura Spartan Endodentics, Earth City, MO.)*

vertical compaction, Obtura III, System B and ThermaFil Plus.[177]

Injection Techniques

Several techniques have been described for introducing gutta-percha into the root canal system after the gutta-percha has been plasticized with heat. The Obtura system (Fig. 8-73) is currently the most commonly used injection delivery system for gutta-percha.[385] It dispenses a heavy form of gutta-percha heated to a high temperature. Although the temperature of the gutta-percha in the Obtura III injection gun is as high as 302° F to 338° F (150° C to 170° C), the temperature of the extruded material may range from 140° F (60° C) to 280° F (138° C).[89] When the Obtura III was compared to the System B heat source using different heat points, neither exceeded an increase of 10° C at any root level monitored.[382] Intracanal temperatures after delivery, measured with intracanal thermocouples, have ranged from 107.6° F to 192.2° F (42° C to 89° C). Periodontal injuries have been reported after excessive heat during endodontic treatment of teeth in the ferret and the dog.[329] Recent studies testing the Obtura III found a 10° C increase in the mandibular anterior teeth in vitro,[229] less temperature rise than when a gutta-percha carrier was combined with an injectable gutta-percha.[230] Shrinkage of gutta-percha in these injectable gutta-percha systems does not appear to be different from the shrinkage of normally heated gutta-percha. When plasticized by heat, gutta-percha has a volume loss of approximately 2% after cooling.[145]

Rotary Techniques

Frictional heat also can be used to soften gutta-percha. This was first suggested with the introduction of the McSpadden Compactor device (DENTSPLY Maillefer). This technique has undergone many variations, with the development of several compaction instruments. The heat generated may exceed safe levels compared to other heat compaction techniques.[329] One such device, Quickfil (J.S. Dental, Ridgefield, CT), has the ability to obturate root canals comparable to lateral compaction.[62] Volumetric changes in the gutta-percha after friction heat compaction are similar to other types of gutta-percha compactions using heat. The major observed problem was the inability of the heated gutta-percha to fill the entire canal system due to the presence of sealer in the apical third of the preparation. Rotary techniques, although still used by practitioners, have become less popular in many instances with the introduction of the new solid-core resin polymer sealers.

Heating Systems

Although not injectable systems, Touch 'n Heat and System B (SybronEndo, Orange, CA), are involved with the placement and removal of gutta-percha from root canal systems using heat. Touch 'n Heat delivers a heated, pointed probe into a mass of gutta-percha, removing the material in small increments and down packing with pluggers after each removal. The gutta-percha is warmed approximately 5 mm ahead of the heating probe, and at a distance of 5 mm from the measured end of the master cone, a final down packing is made to move the remaining mass to the apex. The System B device is an outgrowth of the Touch 'n Heat and attempts to remove the greater bulk of gutta-percha to within 5 mm of the apex in one heated mass. These techniques are often combined with an injectable technique of obturation.

TEMPORARY RESTORATIONS

If endodontic therapy cannot be completed in one visit, the pulp space must be closed with a temporary restoration. The same is true whenever a root canal procedure is completed. This restoration must provide a satisfactory seal to prevent bacteria and fluids in the oral cavity from contaminating the pulp space. The restoration must have enough structural strength to withstand the masticatory forces and retain the occlusal seal. A literature search identified 28 materials that have been tested between 2000 and 2009 (Box 8-6). The most common such materials are IRM (DENTSPLY Caulk), TERM (DENTSPLY Caulk), Cavit (3M ESPE St. Paul, MN.) and Coltosol (Coltene Whaledent). IRM is a reinforced zinc oxide cement available as a powder-liquid mix in single-dose mixing capsules. Cavit is a premixed material composed of zinc oxide, calcium sulfate, glycol, and polyvinyl acetate, polyvinyl chloride, and triethanolamine. It sets upon contact with water. TERM is a filled composite resin that is light activated. When Cavit, IRM, TERM and Fermit (Ivoclar Vivadent, Schaan, Lichtenstein) (the latter two being light-cured preparations) were compared over a 21-day period, Cavit was found to be more resistant to leakage at 7 days.[19,88] Coltosol and IRM were tested against two resins, and results showed they sealed statistically better than the resins. Coltosol is a non-eugenol preparation delivered by syringe.[435] In another study,[442] coronal microleakage was evaluated after thermocycling, comparing Cavit, IRM, and a polycarboxylate cement (Ultratemp Firm).

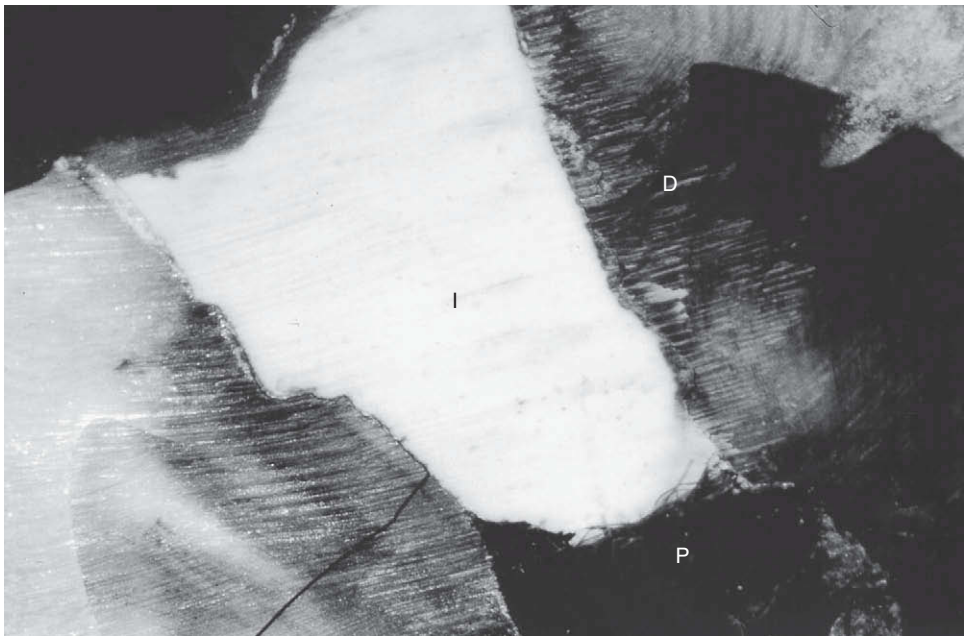


FIG. 8-74 Temporary filling of IRM (white I). The dye has penetrated the margins and dyed the content of the pulp chamber (P). The dentin (D) has been stained by the dye penetrating the margins.

BOX 8-6

Temporary Cements

Algenol
Alsip
Cavit, Cavit W,F
Canseal
Caviton
Clearfil
Coltosol
Core Restore
Dynest AP
Dyract AP
Fermit, Fermit N
Fermin
Fuji-IX-GP, Fuji II LC
GI, GI Resin Modified
IRM
Ketac Fil, Ketac Fil Plus
Ketac Silver
Polycarboxylate Cement, Eugenol Free
Scotch Bond
Temp Bond
TERM
Ultratemp Firm
Vidrion R
Zinc oxide eugenol
Zinc phosphate cement
Z100 (Composite resin)

There was no statistically significant marginal microleakage between the three; IRM also absorbed the dye into the bulk of the material. IRM has extensive marginal leakage of fluid, whereas Cavit seems to absorb fluid into the entire body of the restoration.[18,209] These findings are surprising; however, because of its eugenol content, IRM may provide a bacterial barrier but allow leakage of other liquid substances (Figs. 8-74 and 8-75). Therefore, if Cavit or other types of relatively soft temporary cements are used, they must be placed at a thickness of at least 4 to 5 mm.[419] If a more robust temporary restoration is required for longer than 1 week, the soft cement must be covered with a harder cement such as IRM, glass ionomer cement, or resin.

The temporary coronal seal of endodontically treated teeth will not prevent coronal leakage if left for long time periods.[19] The interval between sealing the canal system and placing the core buildup is possibly more important than the material used. In a Colleagues for Excellence newsletter, the American Association of Endodontists stated that "restoration … should commence as soon as possible after RCT."[65] Whenever possible, the bonded core buildup should be placed at the obturation appointment.

LASERS

Lasers are among the alternative methods used in endodontic treatment. Various laser wavelengths have properties that may be useful when applied to access cavity preparation, cleaning and shaping of root canal systems, and three-dimensional obturation procedures. The use of lasers for endodontics is becoming more predictable with the development of different wavelengths and radially emitting probes. One of the greatest strengths in using laser irradiation is its ability to cause a change in the hard tissue of the root canal space dentin. The change occurs in the morphology and physical aspects of dentin by melting and resolidifying dentin in a manner that closes the orifices of the dentinal tubules, which may harbor microorganisms and other organic pulpal remnants. However, this strength also turns out to be laser irradiation's greatest weakness. This occurs because of the dentin's location, con-

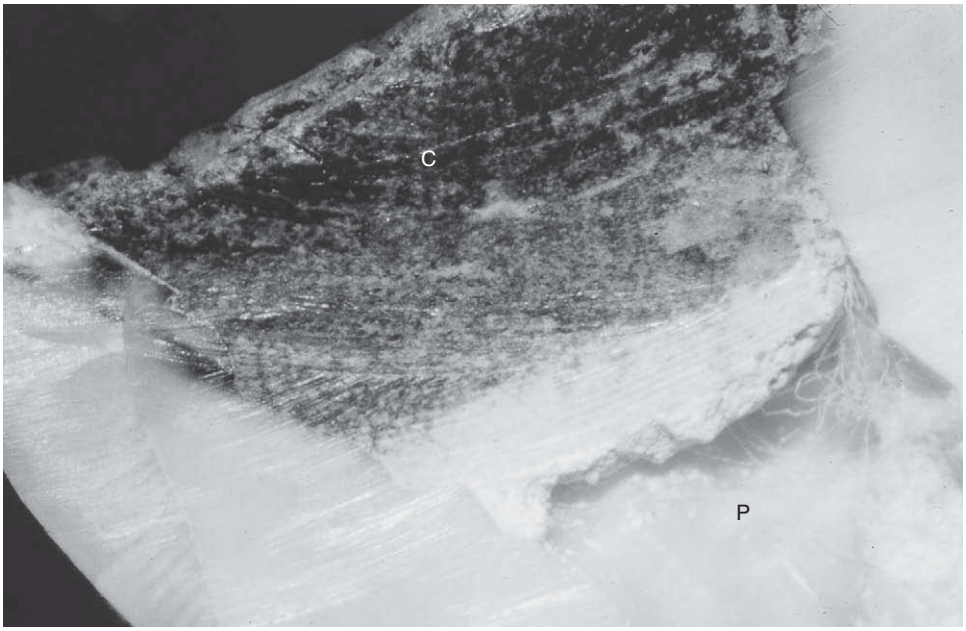


FIG. 8-75 Temporary filling with Cavit. Dye has been absorbed into the body of the restoration *(C)* but has not penetrated to the pulp chamber *(P)*, which is filled with white cotton. The inner part of the Cavit is unstained.

fined to a space that is curved and cylindrical that cannot be viewed in a manner where the changes can be identified or visualized, such as can occur in cavity preparation procedures. A further weakness is the amount of energy needed to effect change of internal morphology. Intertubular dentin surfaces are in the shape of calcospherites rather than the relatively flat surfaces seen in extratubular coronal dentin, which can be easily visualized and affected by the laser. The amount of energy required for intertubular procedures generates increased temperatures that may cause harm to the attachment apparatus (cementum, periodontal ligament, and alveolar bone).

Dental lasers currently in use operate in several areas of the electromagnetic spectrum, including the infrared, visible, and ultraviolet ranges.[374,375] Each laser wavelength in a particular range allows the device to target various tissues and carry out different procedures. Carbon dioxide (CO_2) is highly absorbed by tissues with high water content, and neodymium-doped yttrium aluminum garnet (Nd:YAG) can be absorbed by vascular tissues, having greater effect on gingiva and dental pulp tissue, respectively.

Early attempts using lasers to carry out cleaning and shaping functions were less than successful. The Nd:YAG laser used a contact probe that simulated a hand instrument, but it was composed of a fused silica (glass); it could not be precurved and could not be seen radiographically if it separated within the root canal system. In addition, the probe emitted energy at its end rather than radially over a 360-degree range. The result was a preparation that was neither fully clean nor shaped. Other lasers used noncontact probes that emitted the laser beams through a system of mirrors in an articulated arm (wave guide with CO_2). Their lack of contact with the dentin walls of the root canal system precluded a sense of touch available to the operator.[194,248]

Neodymium-Doped Yttrium Aluminum Garnet Lasers

Several laser systems are now available that appear to be useful in root canal system preparation. Necessary to the success of these procedures is an understanding of the effects of irradiation on the morphology of the inner dentin walls. Nd:YAG lasers have been extensively studied in the past several years. A study showed that at increased power, the system produced globular formations and melting and glazing typical of earlier studies with this wavelength.[326] The same group found that root canal systems treated with NaOCl and EDTA with Nd:YAG irradiation demonstrated partial obliteration of dentin tubules and removal of smear layer.[106] When an Nd:YAG laser was compared to a diode laser, scanning electron microscope (SEM) images showed fusion and resolidification of the dentin surface, more evident with the Nd:YAG group, but the diode group had more uniform changes.[79] The erbium/chromium-doped yttrium scandium gallium garnet (Er,Cr:YSGG) laser has also been extensively studied over the past several years since its inception, but to date, the studies have not demonstrated the same results seen with older laser systems. Improvement in the use of these systems will lead to a device that accomplishes all the requirements of a well-cleaned and shaped system and aid in three-dimensional obturation.[294]

More recent investigations have indicated that lasers are effective when used in infected root canal systems. When patients were treated using a step-back technique, half the teeth were treated with the Nd:YAG laser using NaOCl and H_2O_2 irrigants. Tenderness to percussion was less at 1 week and 3 months in the laser-treated groups compared to controls.[196]

Removal of filling materials and broken files in retreatment cases is often difficult but effective if temperatures are con-

trolled.[434] A recent study compared the removal of gutta-percha using two solvents with an Nd:YAG system, which raised root surface temperatures 4° C.[407] Laser energy alone was found to be effective, whereas solvents did not appreciably improve removal.

Dentin exposed to the Nd:YAG laser was evaluated using five different irrigation solutions. The surfaces of the teeth were examined using a SEM, while dispersive x-ray fluorescence spectrometry assessed mineral content. Calcium levels and calcium/phosphorus ratios decreased significantly with NaOCl solutions, suggesting changes at the molecular level. Surface examination showed that the lowest scores for root cleaning occurred with the use of EDTA and the Nd:YAG laser.[154] The use of the Nd:YAG laser has been suggested as a more efficient way of handling this procedure. A study using the Nd:YAG laser using a flexible quartz fiber probe which is able to be concentrated in a specific area was used to remove gutta-percha and Sealapex or AH26 and was compared to use of Gates-Glidden drills and K-files. Results showed that the use of laser ablation was significantly shorter than conventional methods.[11]

Erbium-Doped Yttrium Aluminum Garnet Lasers

Together with removal of obturation materials, lasers have been used in attempts to alter the dentin surfaces of root canal systems to create greater sealer adhesion. Single-rooted teeth were tested using an Nd:YAG laser. Black ink was used in one test group as an initiator, with two groups lased without ink. Controls were not exposed to laser energy. Results demonstrated leakage in all groups at both coronal and apical test areas.[82] Using an Er:YAG laser, extracted teeth were tested to determine whether increased adhesion occurred between ZnOE and resin sealers. The resin-based sealer (Sealer 26) adhered better to dentin prepared with and without laser exposure than ZnOE-based sealers.[367] Increasing the frequency and use of EDTA increased adhesion when a resin sealer was used with either an Nd:YAG laser or Er:YAG laser.[368] When a 980-nm diode laser was tested, bond strength again increased for the resin sealer (AH26) but did not affect adhesion of Epiphany sealer.[9]

When India ink was used to absorb energy to cause an effect, the Nd:YAG laser was found to remove smear layer and reduce apical leakage in extracted, obturated human teeth.[144] Others found the same results, showing that this laser wavelength was useful for reducing apical leakage in obturated root canal systems.[55,236] The Nd:YAG laser was also used to seal the dentin surface after apicoectomy ex vivo.[143,376,377]

The use of laser energy in endodontics receiving the most attention relates to cleaning and shaping (instrumentation) and laser-assisted endodontic procedures involving the use of laser irradiation within the confines of the root canal system. As noted earlier, this may cause increased temperatures extending to the outer root surface of the tooth and lead to damage to the attachment apparatus. An Er,Cr:YSGG laser used to instrument the root canal system walls for 7 seconds found average rises of temperature on the outer root surfaces were less than 8° C. An 810-nm diode laser recorded temperatures at less than 7° C when energy input was alternated with periods of no energy input. A 910-nm diode laser recorded differing temperatures depending on what area of the root canal system was being affected. Highest temperatures were found in the apical third but were judged not to be in the range harmful to the attachment apparatus. An 810-nm diode laser was able to melt dentin, which resolidified and closed tubular orifices. Temperature rise was reported to be in the range of 1.6° C to 8.6° C, below those temperatures that would adversely affect the PDL.[74]

The permeability of root dentin is an important consideration in endodontic therapy, and the laser has been suggested as a means of removing smear layer and melting and solidifying the walls of the preparation. Comparison of the Nd:YAG device to an Er:YAG laser showed that the former had lessened radicular dentin permeability with various irrigation regimens.[45] An in vitro study using extracted teeth that were inoculated with *E. coli* and *E. faecalis* found that Nd:YAG, Ho:YAG (holmium-doped yttrium aluminum garnet), and Er:YAG lasers eliminated 99% of the organisms without causing unfavorable temperature increases at the settings used.[110]

Temperature rise is an important consideration in the use of any laser energy. One study used 20 extracted human teeth and an Er:YAG laser at two irradiation intensities with water spray.[193] A temperature increase of less than 6° C was recorded at the apical third and less than 4° C in the midportion of the root, suggesting that the thermal effect on periodontal tissues may be minimal.

Although no widespread agreement exists on the need to remove the smear layer in root canal preparations, the Er:YAG laser has proved effective at accomplishing this task. An Er:YAG laser was compared with a CO_2 laser for ability to remove the smear layer.[384] A comparison was also made with NaOCl, H_2O_2, and EDTA irrigation regimens. The Er:YAG laser was found to be the most effective of these methods at removing the smear layer. A more recent study, using radially emitting probes with Er:YAG and Er,Cr:YSGG lasers, increased the action of EDTA in removing thick, artificially created smear layers.[131] An associated study found that the use of a radial-firing tip and Er,Cr:YSGG laser was a suitable tool for the elimination of microorganisms and removal of smear layer from root canal systems. A study in modification of root canal dentin found that by varying the electromagnetic wavelength, Er:YAG energy induced different modifications to the root canal surface, which may have some utility in preparation procedures.[91]

Sterilization of root canal systems has continued to be an aim of successful endodontic therapy. Chemical and other liquid medicaments have been utilized alone or in combination in an attempt to sterilize these systems, with no lasting effect. The idea of removing smear layer appears to be necessary in sterilization, and lasers have been suggested for that use and altering the intracanal dentin through melting and resolidification, causing closure of dentinal tubule orifices. An Er:YAG laser was used in posterior dogs' teeth that were necrotic with chronic periradicular lesions. Group 1 was treated by biochemical preparation, and group 2 was also bioprepared but with laser irradiation. Teeth were sampled for anaerobes, aerobes, and total colony-forming units (CFU) 7 days post treatment. Results showed increased CFU for all microorganisms and significant reduction of anaerobes in group 1 and anaerobes and total CFU in group 2.[219] Using an

Er:YAG laser, other investigators[404] found statistically similar results between laser-treated and conventionally treated groups. One study found that treatment with an Er:YAG laser showed effective antimicrobial action against *E. coli* and *Staphylococcus aureus*.[337] The results were significantly better than those achieved with NaOCl. More recently, lasers were found to be effective against *Candida albicans* and four other test organisms. However, 70% of specimens lased 3 mm short of the apex remained infected.[299]

Diode Lasers

New laser systems using diodes are now available. In the presence of methylene blue or other dyes, diode lasers activate the dyes and cause photochemical reactions that produce reactive oxygen species. The process is termed *photoactivated disinfection* (PAD) and has the potential to eradicate persistent endodontic infections in cases where conventional methods may be unsuccessful. When used against four common root canal microorganisms *(Fusobacterium nucleatum, Peptostreptococcus micros, P. intermedia, Streptococcus intermedius),* significantly higher numbers were killed. When pathogens in planktonic phase and *E. coli* biofilms were sensitized with methylene blue and treated with red light, all organisms were eliminated. When energy levels were raised, 97% of *E. faecalis* was also eliminated. Other studies resulted in the conclusion that the use of photoactivated disinfection resulted in maximum elimination of microorganisms, especially *E. faecalis*.*

However, when comparing teeth treated conventionally, using rotary instrument NaOCl and EDTA versus the added use of Er:YAG laser system, neither method eliminated infection.[185] An in vitro study using the Er:YAG laser system found that it was effective against six of the bacterial species examined. The numbers of viable cells in the biofilms were significantly decreased with atrophic changes in bacterial cells and biofilm density.[285] When an Nd:YAG laser was used to kill microorganisms seeded on dentin discs with three endodontic pathogens, bacterial eradication occurred. However, when microcolonies were found in biofilms, bacterial eradication was reduced.

Erbium/Chromium-Doped Yttrium Scandium Gallium Garnet Lasers

The Nd:YAG system was found to be more effective than the Er,Cr:YSGG laser, with the former demonstrating a reduction of 97% and 98% of organisms.[417] Researchers[94] found the Er,Cr:YSGG system to be less effective than a 3% solution of NaOCl, with the latter inhibiting growth of *E. faecalis* in all root canal systems tested. A later study found that the same system eliminated bacteria and smear layer debris in root canals, with only moderate elevation of temperature on the outer root surfaces.[336] Others have demonstrated the same results when comparing the Er,Cr:YSGG system to NaOCl, using a radically emitting laser probe for 2 minutes; 99.7% bacterial reduction occurred with the laser.[142] A high-powered 810-nm diode laser was used to examine the ability to achieve disinfection in deep layers of dentin. A group of 10 extracted teeth were treated with rotary files, 0.5% NaOCl, 17% EDTA, and irradiated, with a second group treated the same but without laser exposure. A third group was treated only with saline irrigation. Results showed 100% and 98.3% disinfection when compared with the control group.[86] However, few of the investigators cited were willing to call the effects *sterilization* as opposed to *disinfection*. There are also differences in results that appear to be dependent on wavelength. *Therefore, the use of lasers still remains somewhat controversial for use in sterilization of root canal systems.*

Laser irradiation has been used in the preparation of root-end restorations. The root-end areas prepared with burs expose dentinal tubules, which may allow leakage of microorganisms and their byproducts. When preparations using an Er:YAG laser were compared to an Nd:YAG laser and high-speed burs, the lasers demonstrated lower infiltration indices than high-speed burs.[287] In a similar study, results determined that a transversely excited atmospheric pressure (TEA) CO_2 laser and an Er:YAG laser showed clean, smooth root-end preparation surfaces with sealed tubules and a reduction of permeability to methylene blue dye.[143] An Er:YAG laser was compared to root-end ultrasonic preparation using three filling materials (MTA, Super EBA, IRM). The laser-treated areas demonstrated lower microleakage values for all three materials.[188] When comparing use of a low-speed bur and an Er,Cr:YSGG laser for reverse root-end preparations using cyanoacrylate and MTA as filling materials, MTA and laser samples presented significantly higher permeability rates.[426] Alternating Er:YAG and Nd:YAG irradiation preparations with high-speed hand piece preparation showed no differences between either group exposed to lasers and no differences between bur preparations in microleakage percentages.[13] Diode lasers are available at much lower costs than other systems and can also be used for root-end preparations. Groups treated with a 980-nm laser were compared to non-laser groups, demonstrating absence of smear layer and reduced leakage when compared with nonlased teeth.[418] However, *when used in combination with MTA for reverse filling, the laser radiation did not improve apical sealing.*[87]

Laser energy may prove useful for treating diseases of the root canal system and periradicular regions, but years of successful endodontic care have demonstrated the value of several already proven methods. Until independently supported, reproducible studies are published in peer-reviewed journals—wherein findings match present marketing claims—the reader is advised to exercise prudent caution before embracing these newer laser technologies.

SUMMARY

An explosion in knowledge and technology has created an exciting time in the specialty of endodontics. New instruments and materials seem to appear faster than clinicians can learn about the preceding versions. This has created an educational challenge for practitioners, universities, and manufacturers, requiring a greater degree of cooperation among these groups than ever before. Clinicians should only use those instruments and materials that have been shown safe and effective by independent studies.

*References 33, 108, 111, 112, 124, 125, and 366.

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CHAPTER 9

Cleaning and Shaping of the Root Canal System

OVE A. PETERS and CHRISTINE I. PETERS

CHAPTER OUTLINE

FRAMEWORK FOR ROOT CANAL TREATMENT
- Pathophysiology of Endodontic Disease
- Dental Anatomy
- Clinical Objectives

CLEANING AND SHAPING: TECHNICAL ISSUES
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- Motors and Devices
- Disinfectants, Dentin Surface Modifiers, and Lubricants

CLEANING AND SHAPING: CLINICAL ISSUES
- Biologic Objectives
- Mechanical Objectives
- Concepts and Strategies

CANAL PREPARATION TECHNIQUES
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- Rotary Instrumentation
- Other Systems

CANAL CLEANING TECHNIQUES
- Irrigation Modes and Devices
- Eradication of Microorganisms
- Smear Layer Management

FRAMEWORK FOR ROOT CANAL TREATMENT

Clinical endodontics encompasses a number of treatments, but perhaps the most important is treating pulps and root canal systems (with or without periradicular pathosis of pulpal origin) so that patients can retain their natural teeth in function and esthetics. The treatment of traumatic dental injuries and prophylactic treatment of vital pulps to maintain vitality are different from pulpectomies in which root canal instrumentation is required. However, endodontic therapy essentially is directed toward one specific set of aims: to cure or prevent periradicular periodontitis.[429]

Routine orthograde root canal treatment is a predictable and usually highly successful procedure, both in relatively straightforward (Fig. 9-1) and more difficult cases (Figs. 9-2 and 9-3). In recent studies and reviews, favorable outcome rates of up to 95% were reported for the treatment of teeth diagnosed with irreversible pulpitis[39,99,139]; favorable outcome rates of up to 85% were reported for necrotic teeth.[98,140,303,311]

To date, many treatment modalities, including nickel-titanium rotary instruments, have not been shown to have a statistically relevant impact on treatment outcomes.[303] This poses a real problem in the age of evidence-based therapy, because a new therapeutic technique should provide a better result than standard procedures in clinical tests. The small number of relevant prospective clinical studies is only partly offset by numerous in vitro experiments. This chapter includes pertinent information from such studies, as well as results from our own experiments, because rotary nickel-titanium instruments have become widely used adjuncts in root canal treatment.

Pathophysiology of Endodontic Disease

Many prospective and peritreatment factors have been suggested as links to favorable treatment outcomes in endodontic therapy. Such factors include the patient's age and gender, the position of the tooth in the arch, extension of the root canal filling, and the use of certain interappointment dressings, such as calcium hydroxide [$Ca(OH)_2$]. The presence of a periradicular osseous lesion (i.e., "apical periodontitis") appears to be a relevant prognostic factor that reduces the likelihood of a favorable outcome for root canal treatment; however, lesion size by itself is not an indication for endodontic surgery (see Chapter 22). Fig. 9-4 shows two cases in which large osseous lesions were treated by orthograde approaches. At recall appointments, the teeth were asymptomatic, and a reduction in lesion size was evident in both cases.

Some may question whether lesions such as the ones in Fig. 9-4 are in fact cysts. Several studies have demonstrated that lesion size shows little correlation with the incidence of radicular cysts.[55,222,275,279] Only histologic examination can prove whether a radiolucency is in fact a cyst. True cysts are believed to heal only after surgical enucleation,[278] whereas the noncystic majority of apical processes heal predictably by orthograde endodontic treatment without surgery. An orthograde approach, therefore, appears to be beneficial in clinically asymptomatic cases and should include recall appointments at appropriate intervals (see Chapter 14).

If clinical symptoms persist or begin after endodontic therapy, surgery may be performed in addition to orthograde root canal treatment. In the case shown in Fig. 9-5, a large

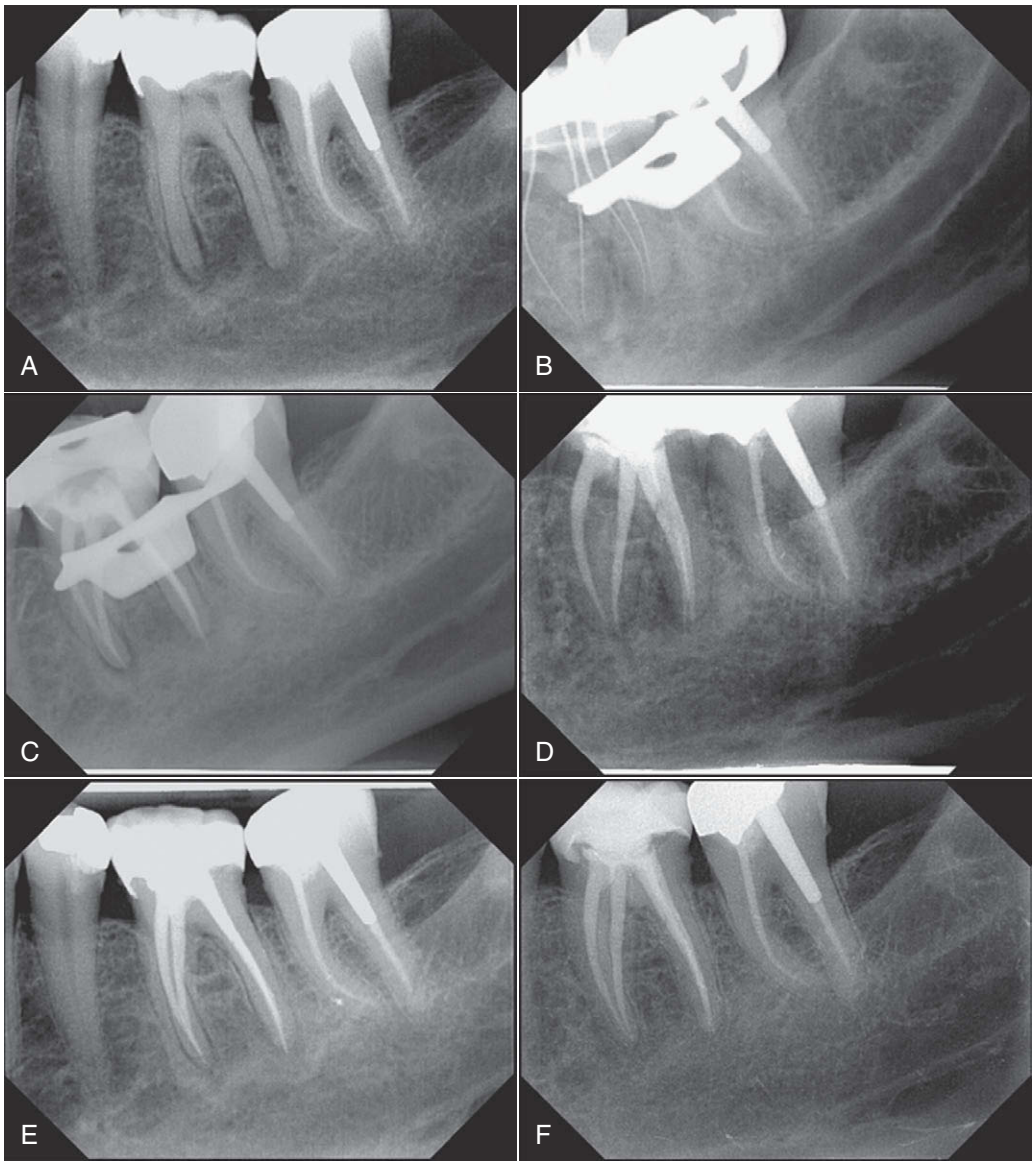


FIG. 9-1 Effect of routine root canal treatment of a mandibular molar. **A,** Pretreatment radiograph of tooth #19 shows radiolucent lesions adjacent to both mesial and distal root apices. **B,** Working length radiograph shows two separate root canals in the mesial root and two merging canals in the 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 shows evidence of additional periradicular healing. **F,** Five-year recall radiograph; tooth not only is periapically sound but also clinically asymptomatic and fully functional.

lesion that extended into the maxillary sinus and nasal cavity was treated surgically 1 week after orthograde therapy of teeth #7 and #8, which included removal of two instrument fragments. The lesion was completely enucleated during surgery, and a tissue biopsy specimen was submitted for histologic processing; the lesion was diagnosed as a radicular cyst. As expected in this case, the patient reported discomfort after surgery. This supports the preference for a nonsurgical approach whenever possible.

When root canal therapy is part of a comprehensive treatment plan, a favorable outcome for the root canal portion is a prime requirement. Extended bridgework and removable dentures depend on healthy periradicular tissues, just as they depend on healthy marginal and apical periodontal tissues. Fig. 9-6 presents a case in which a removable denture seemed unavoidable at the first examination. After extractions and root canal therapy were performed, small-unit, fixed partial dentures were placed. These reconstructions remain fully functional and allow this patient to benefit from the natural dentition.

In summary, orthograde root canal treatment has a high degree of predictability both in normal and complex cases. Some limitations exist, but the potential for a favorable outcome is significant. As indicated previously, the shaping and cleaning procedures performed as part of root canal treatment are directed against microbial challenges to the root canal system.[170]



FIG. 9-2 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.)*

Microorganisms can breach dental hard-tissue barriers through several avenues, the most common being dental caries (Fig. 9-7).[327]

Dental Anatomy

Pulpal reactions may be observed as soon as the *diffusion barrier* (the remaining dentin thickness) is sufficiently permeable for bacteria or their toxins to affect the pulp[56] (see Fig. 9-7). Under experimental conditions, pulpal inflammation can be detected only a few hours after topical application of bacterial components to exposed dentin.[46,286] In an established lesion, a bacterial ecosystem evolves, with synergisms and antagonisms among the microorganisms (see Chapter 15). These interactions play an important role in the course of the disease when intraradicular biofilms develop and bacteria invade dentinal tubules.[236] Two key factors initiate and modify inflammatory reactions such as the development of microabscesses in subodontoblastic regions: the penetration of bacterial components and the release and diffusion of inflammatory mediators.

The stereotypic pulpal defense reaction is hard-tissue deposition (Fig. 9-8 and see Fig. 9-7) by primary and secondary odontoblasts.[56] Hard tissue is laid down as a response to a stimulus (*reactionary* or *reparative dentinogenesis*) and thus takes place within a defined spatial relationship to that stimulus, occurring slightly apical to the lesion.

Hard-tissue deposition is a natural event with aging[451] (*secondary dentinogenesis*), which creates a higher degree of treatment difficulty in older patients.[452] Clinicians note a radiographically detectable decrease in the size of the pulp space that occurs most often in the coronal regions but also can be seen in the more apical areas. This condition is not a contraindication to orthograde endodontic therapy, but it does require additional attention to clinical procedures such as preenlargement and prebending of hand files (discussed later in this chapter).

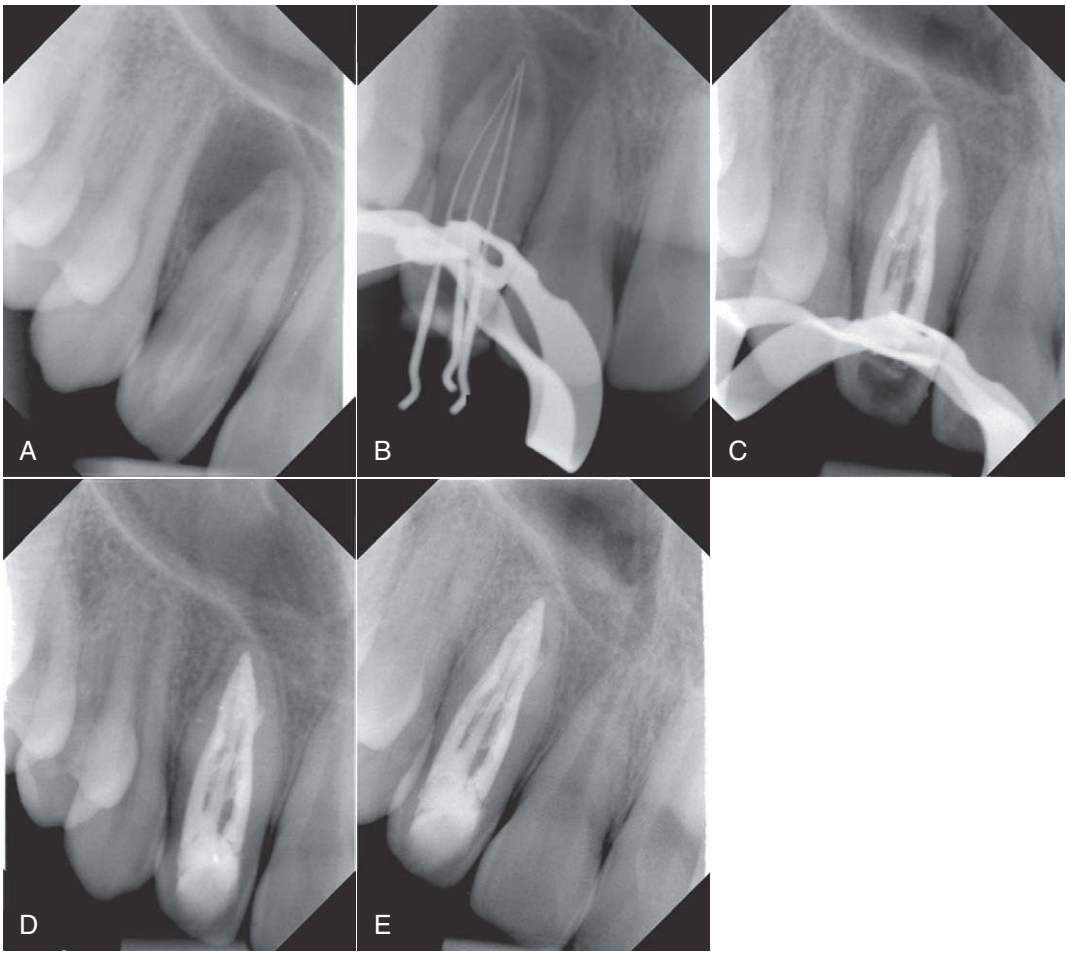


FIG. 9-3 Root canal treatment in a case with unusual and complicated anatomy. **A,** Pretreatment radiograph of tooth #7 in a 12-year-old boy shows a substantial periradicular lesion and evidence of additional radicular anatomy (i.e., a dens-in-dente type II, according to Oehlers' classification).[285] **B,** Working length radiograph shows three separate root canals. **C,** Posttreatment radiograph 2½ months after shaping of the root canal systems with a nickel-titanium rotary system aided by ultrasonically activated K-files and dressing with calcium hydroxide four times for about 2 weeks each. Note the substantial periradicular bone fill. **D,** One-year recall radiograph shows evidence of periradicular healing. **E,** Two-year recall radiograph shows sound periradicular tissues.

The process of *calcific metamorphosis* is a response to traumatic injury.[22] It is characterized by a reduction in the size of both the radicular and coronal pulp spaces. Conversely, teeth with signs of hard-tissue deposition caused by bacterial attack show an initial reduction of pulp space size coronally, which may involve the pulp chamber and canal orifices (see Fig. 9-7). This situation calls for meticulous preparation of an access cavity and preenlargement of canal orifices in a nondestructive manner. Depending on the timing of inoculation and the number of microbes, hard-tissue deposition also may occur more apically.[215]

Reparative dentin may form a diffusion barrier sufficient for the pulp to recover, depending on the severity of the bacterial challenge and the capability of the defense mechanisms.[242] Unfortunately, no consensus exists on the best therapy to allow this recovery to occur.[47] Further into the disease progress, and if the carious lesion persists, bacteria may be present in sufficient concentrations to induce pulpal inflammation. This is triggered by molecular signals (e.g., cytokines) released from cells such as macrophages and neutrophils well before microbes are actually present intrapulpally (see Chapter 15). At this stage, with a diagnosis of reversible pulpitis, endodontic treatment may be avoidable, provided the source of the irritants is removed.

To deliver adequate endodontic therapy, the clinician must understand that apical periodontitis is the endpoint of a disease flow that in most cases originates coronally, either with carious lesions or a traumatized pulp (see Fig. 9-7). As stated previously, opportunistic bacteria may invade dental hard tissue, and their byproducts eventually may reach the pulp space (see Chapter 15). Host response factors, such as the recruitment of neutrophil granulocytes and local development of neurogenic inflammation, act against microbial invasion, but this line of defense may succumb to the challenge if the carious defect is not repaired. Then, after microabscesses form, circulation changes occur; coronal and subsequently radicular pulp may become nonperfused and thus necrotic.

At various points in this process, bacterial factors such as lipopolysaccharides and peptidoglycans[201] can reach periapical tissues through apical and accessory foramina. Zones of bone resorption (appearing as radiolucencies) may develop, depending on the balance between microbial virulence factors and

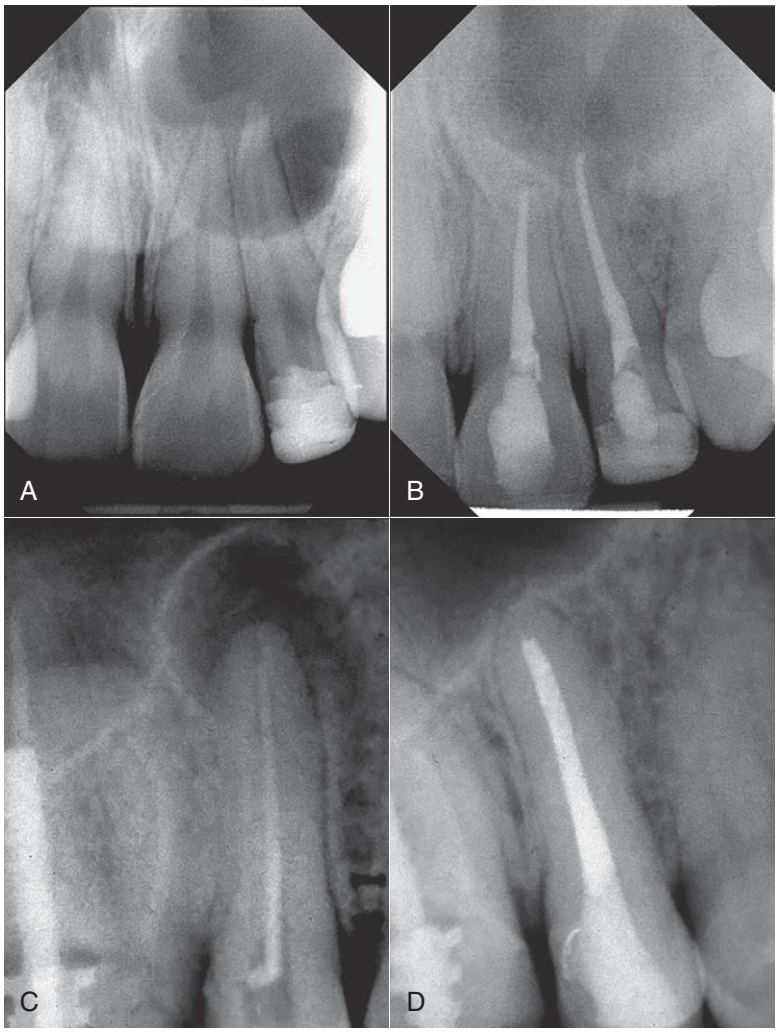


FIG. 9-4 Potential of root canal treatment in cases of substantial periradicular destruction. **A,** Pretreatment radiograph of teeth #8 and #9 shows a large lesion. Neither tooth responded to cold tests. **B,** Two-year follow-up radiograph shows bone fill. The canals were shaped with rotary and hand instruments, and obturation was performed using laterally compacted gutta-percha with AH Plus as the sealer. **C,** Pretreatment radiograph of tooth #4, which has a previously filled root canal; a large periradicular lesion and insufficient obturation can be seen. **D,** Two-year posttreatment radiograph shows evidence of bony healing after nonsurgical retreatment. (***A-B*** *courtesy Dr. M. Zehnder;* ***C-D*** *courtesy Dr. F. Paqué.)*

host defenses.[402] The development of apical periodontitis is associated with a significantly less encouraging prognosis after orthograde endodontic treatment.[99,387,389]

One school of thought emphasizes the importance to successful endodontic therapy of cleaning and filling lateral and accessory canals.[337,458] Clinical radiographs of beautifully performed cases support this position; the contribution of accessory canals to lesion development in certain cases seems highly likely (see Fig. 9-2). However, this pathogenesis depends on the volume of accessory canals and the amount of bacteria harbored in them. Another subject of controversy is the clinical importance and mechanisms of dentinal tubule infection[235,236,300] with bacteria and fungi (Fig. 9-9).

In most cases, lesions are associated with the main root canal systems (see Figs. 9-1 and 9-3 to 9-5) and form periapically around the main foramina. The main canal unquestionably has the highest bacterial load, and important studies link reduction of the viable intracanal bacterial load to favorable outcomes for endodontic therapy.[207,299,387] A primary aim of all endodontic procedures, and most notably of cleaning and shaping, is to remove canal contents, specifically infective microorganisms and necrotic tissues.[2]

Clinical Objectives

A wide spectrum of possible strategies exists for attaining the goal of removing the canal contents and eliminating infection. Investigators[244] introduced a minimally invasive approach to removing canal contents and accomplishing disinfection that did not involve the use of a file: the noninstrumentation technique.[137] This system consisted of a pump, a hose, and a special valve that was cemented into the access cavity (Fig. 9-10, *A*) to provide oscillation of irrigation solutions (1% to 3% sodium hypochlorite[3]) at a reduced pressure. Although several in vitro studies[243,245,246] demonstrated that canals can be cleaned and subsequently filled using this system (see Fig. 9-10, *B* and *C*), preliminary clinical results have not been as convincing (see Fig. 9-10, *D*).[27]

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. 9-10, *C*). 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

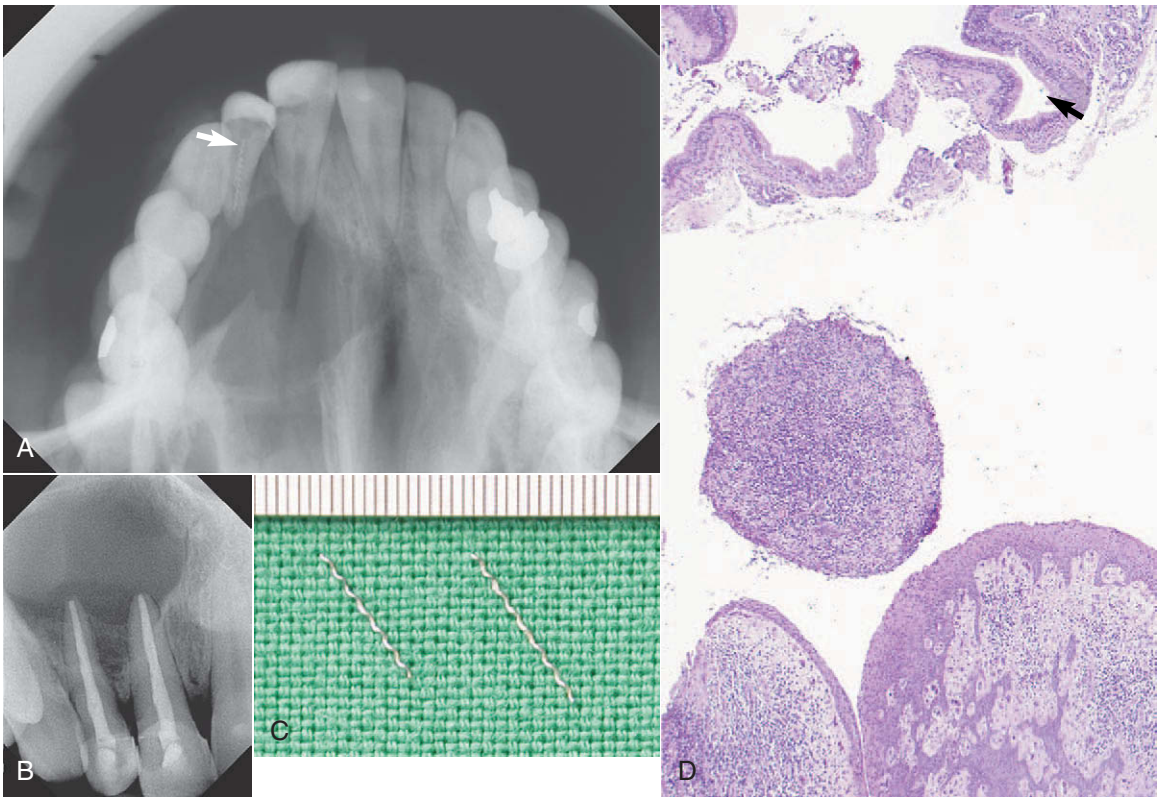


FIG. 9-5 Possibilities and limitations of orthograde endodontic therapy. In this case, a large lesion in the right maxilla was enucleated and histologically diagnosed as a radicular cyst. **A,** Pretreatment occlusal plane radiograph shows a large periradicular lesion in the right maxilla, as well as two separated instruments in tooth #7 *(arrow)*. **B,** Posttreatment periapical radiograph of tooth #7 and necrotic tooth #8, which were obturated after calcium hydroxide dressings had been placed for 2 weeks. Obturation was done with laterally compacted gutta-percha and Roth's 801 sealer. **C,** Two lentulo spiral fragments removed from tooth #7 (ruler gradation is 0.5 mm). **D,** Histologic slide shows both respiratory epithelium *(arrow)* and squamous epithelial lining and inflammatory cells, supporting the diagnosis. *(**C** Courtesy Dr. I. Hegyi.)*

process, such as how large the apical preparation should be and what are the correct diameter, length, and taper.[204]

The foundation of the endodontic treatment plan is an adequate diagnostic process (see Chapter 1), which includes obtaining diagnostic radiographs from various angles. Also, the restorability and periodontal status of teeth to be treated endodontically must be determined. In some cases, buildups or crown lengthening is required for preendodontic restoration to allow proper isolation, create pulp chambers that retain irrigants, and facilitate interappointment temporary restorations. In many cases, the existing restoration may have to be removed so an adequate diagnosis can be made and the immediate cause of endodontic treatment can be assessed.[1]

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. The intent of this chapter is to assist clinicians with that task and to provide a much-condensed background in radicular anatomy, pulpal pathophysiology, and nickel-titanium metallurgy.

Endodontic therapy has been compared to a chain, 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 débridement, the creation of space for the delivery of medicaments, and optimized canal geometries for adequate obturation.[301] These tasks are attempted within a complex anatomic framework, as recognized in the early 20th century by Walter Hess[183] (Fig. 9-11) (see Chapter 7).

Unfortunately, canal preparation results are adversely affected by the highly variable root canal anatomy.[12,13,188,274,308] This fact is especially true for conventional hand instruments and to a lesser degree for most nickel-titanium rotary instruments.[50,301] Therefore the radicular anatomy is briefly reviewed as it pertains to cleaning and shaping.

Root canal curvature can be assessed clinically from radiographs, preferably taken from various angles. However, it is well documented that curves in the mesiodistal plane often are greater than those in the more readily accessible buccal-lingual plane.[103,312] In vitro, a full account of three-dimensional canal anatomy can be seen with interactive micro–computed tomographic (μCT) reconstructions (Figs. 9-12 and 9-13).

The clinician must understand the five commonly encountered canal paths: canals that merge, curve, recurve, dilacerate, or divide.[337] All five situations are risk factors for file breakage and should be carefully evaluated, as is done for more basic considerations such as the estimated canal length, position of the primary curve, canal diameter, and apical topography.

Early anatomic studies[162,163,219] evaluated the position and topography of the apical foramina and the position of the apical constriction. These studies found that the *physiologic*

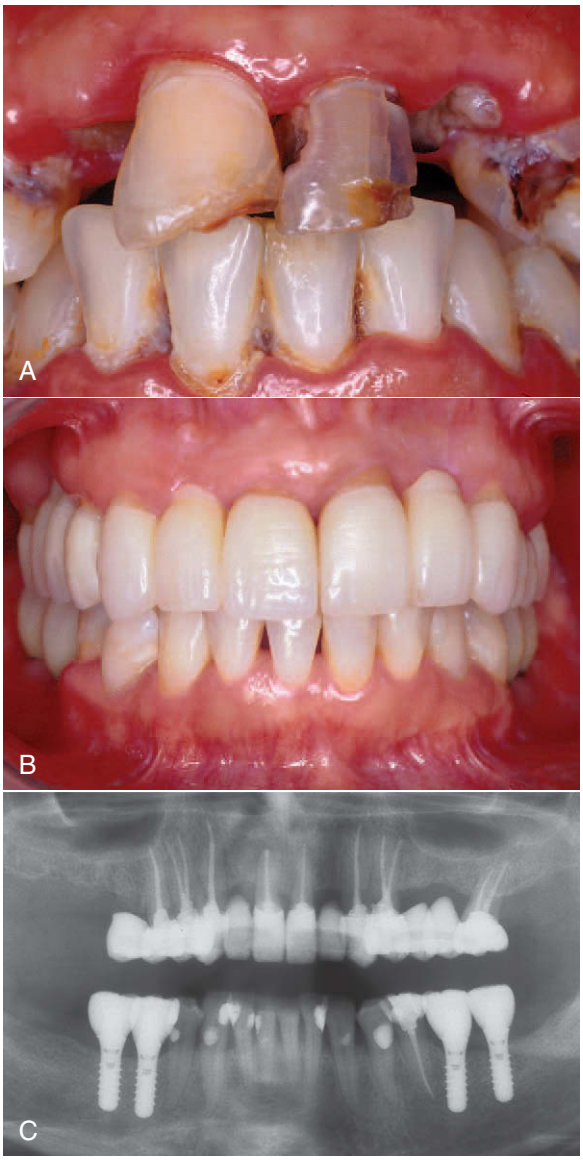


FIG. 9-6 Root canal therapy as part of a comprehensive treatment plan. The patient, who was recovering from intravenous drug addiction, requested restorative dental treatment. Because of extensive decay, several teeth had to be extracted, and nine teeth were treated endodontically. Root canal treatment 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.)*

foramen, or *canal terminus,* was located up to 1 mm coronal to the *anatomic apex,* or *root tip.* This observation has been confirmed by later studies.[123,267]

Clinically, the landmark detected from radiographs (the *radiographic apex*) does not necessarily coincide with the anatomic apex because of projection artifacts. Taken together, these observations suggest that shaping to the radiographic apex is likely to produce overinstrumentation past the apical foramen, with possible clinical sequelae of posttreatment pain and inoculation of microorganisms into periapical spaces.[45,47,126,168]

Foramen diameter was also an issue in both early[162,219] and more recent studies.[68,123,267,403] The smallest canal diameter, called the *apical constriction,* was located 0.5 to 0.7 mm coronal to the canal terminus.[162,219] A wide range of diameters has been reported in that region, from 0.2 to about 1 mm[68,210-212,219,267]; the concept of a single apical constriction has also been challenged.[123] Moreover, studies have shown that clinicians usually underestimate apical dimensions.[461] Clearly the apical anatomy presents the clinician with major challenges (Fig. 9-14), such as apically dividing canals, nonround cross sections, and delta-like configurations. In addition, canal cross sections that are wide buccolingually[458] are difficult to instrument with rotary techniques.

The clinician must choose the strategies, instruments, and devices to deal with these challenges and control the preparation shape, length, and width precisely. This allows the clinician to use endodontic therapy to address acute (Fig. 9-15) and chronic (Fig. 9-16) forms of the disease processes described previously. Recall radiographs taken at appropriate intervals will demonstrate longevity and favorable outcomes (see Figs. 9-1 to 9-4, 9-6, and 9-16) if clinical objectives are maintained (Box 9-1).

BOX 9-1

Basic Objectives in Cleaning and Shaping

The primary objectives in cleaning and shaping the root canal system are to:

- 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

CLEANING AND SHAPING: TECHNICAL ISSUES

Because several technical issues arise with the instruments and devices used for cleaning and shaping, a short review of these products is provided here (also see Chapter 8). A vast array of instruments, both handheld and engine-driven, are available for root canal preparation. Up to the last decade of the past century, endodontic instruments were manufactured from stainless steel. With the advent of nickel-titanium,[370] instrument designs began to vary in terms of taper, length of cutting blades, and tip design. Files traditionally have been produced according to empiric designs, and most instruments still are devised by individual clinicians 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 versions rapidly 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

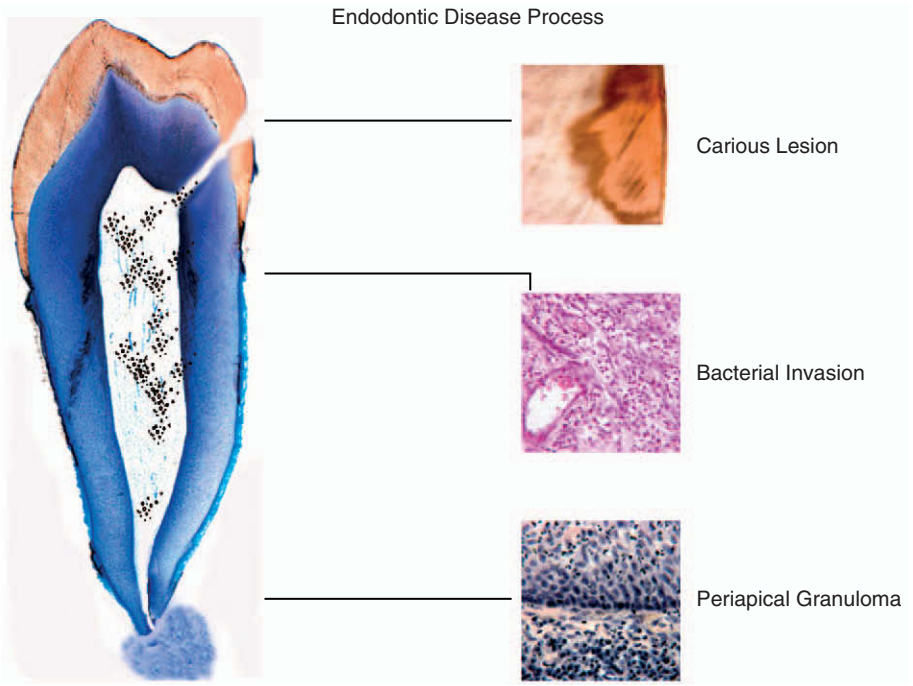


FIG. 9-7 Schematic representation of the progression of pulpal disease and the development of periradicular pathosis. A carious lesion leads to contact of toxins and microbes with the coronal pulp, resulting in inflammation and infection. The stereotypic defense reaction of dental pulp then occurs: hard-tissue deposition. This reaction may lead to repair or to additional hard-tissue deposition (e.g., as calcific metamorphosis). The next step may be formation of microabscesses, changes in circulation during inflammation, and ultimately progression of infection into the radicular pulp space. Finally, periradicular osseous lesions may develop if the bacterial challenge persists. *(Courtesy Professor H.-U. Luder and T. Häusler.)*

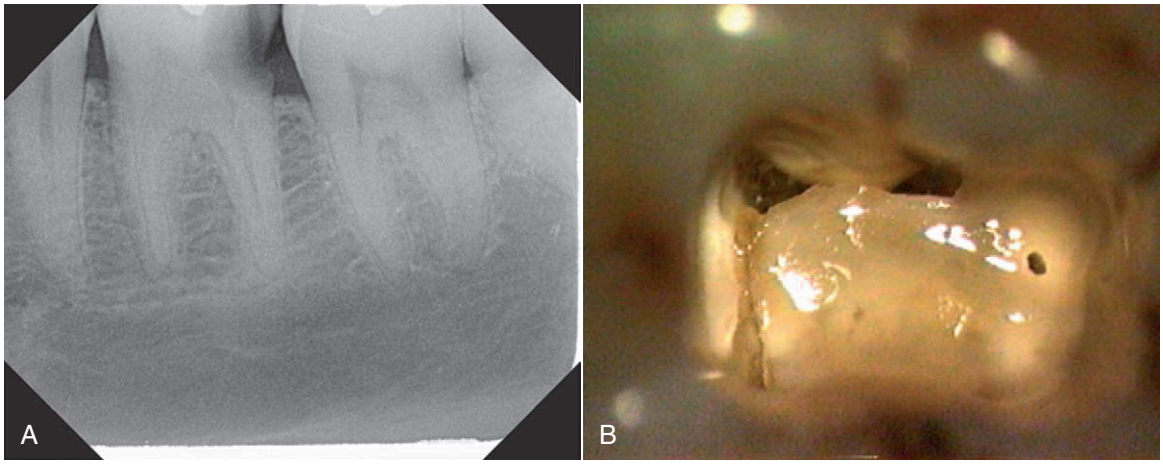


FIG. 9-8 Evidence of coronal hard-tissue deposition. **A,** Periapical radiograph of tooth #19 shows evidence of reduced coronal and radicular pulp space. **B,** Intraoral photograph, taken through an operating microscope (×25), of access cavity of the tooth shown in **A;** note the calcific metamorphosis.

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.

Hand and Engine-Driven Instruments

Hand instruments have been in clinical use for almost 100 years, and they still are an integral part of cleaning and shaping procedures. A norm established by the American Dental Association (ADA) and the International Standards Organization (ISO)[21,117] sets the standards for broaches, K-type files and reamers, Hedström files, and paste carries; however, the term *ISO-normed instruments* currently is used mainly for K-files (Fig. 9-17). One important feature of these instruments is a defined increase in diameter of 0.05 or 0.1 mm, depending on the instrument size (Fig. 9-18).

Broaches

Barbed broaches 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 nickel-titanium rotary shaping instruments, but broaching occasionally may be useful for expediting proce-

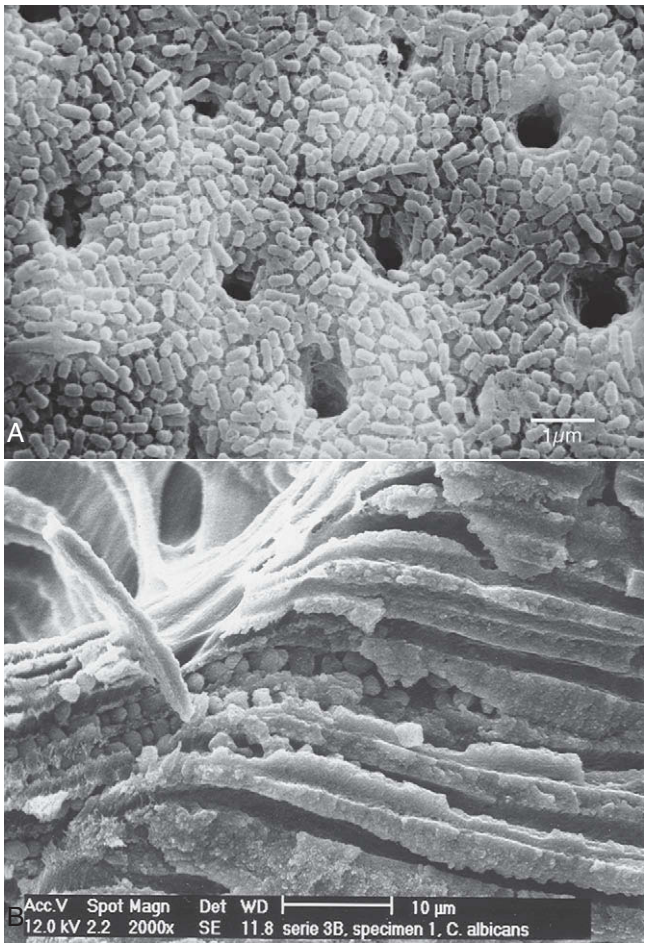


FIG. 9-9 Presence of microorganisms inside the main root canal and dentinal tubules. **A,** Scanning electron micrograph of a root canal surface shows a confluent layer of rod-shaped microbes (×3000). **B,** Scanning electron micrograph of a fractured root with a thick smear layer and fungi in the main root canal and dentinal tubules. (***A*** *courtesy Professor C. Koçkapan;* ***B*** *courtesy Professor T. Waltimo.)*

dures and removing materials (e.g., cotton pellets or absorbent points) from canals.

K-Files

K-files were manufactured by twisting square or triangular metal blanks along their long axis, producing partly horizontal cutting blades (Fig. 9-19). Noncutting tips, also called *Batt tips,* are created by grinding and smoothing the apical end of the instrument (see Fig. 9-19). Investigators introduced a modified shape, 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.[335] Similar techniques are required to manufacture NiTi K-files[413] such as the NiTi-Flex (DENTSPLY Maillefer, Ballaigues, Switzerland). NiTi K-files are extremely flexible and are especially useful for apical enlargement in severe apical curves. They can be precurved, but only with strong overbending; this subjects the file to excess strain and should be done carefully. Because of their flexibility, the smaller NiTi files (sizes up to #25) are of limited use.

Cross-sectional analysis of a K-file reveals why this design allows careful application of clockwise and counterclockwise rotational and translational working strokes. ISO-normed K- and Hedström files are available in different lengths (21, 25, and 31 mm), but all have a 16-mm-long section of cutting flutes (see Fig. 9-17). The cross-sectional diameter at the first rake angle of any file is labeled D_0. The point 1 mm coronal to D_0 is D_1, the point 2 mm coronal to D_0 is D_2, and so on up to D_{16}. The D_{16} point is the largest diameter of an ISO-normed instrument. Each file derives its numeric name from the diameter at D_0 and is assigned a specific color code (see Fig. 9-17).

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 length (#.02 taper) (see Fig. 9-17). Thus a size #10 instrument has a diameter of 0.1 mm at D_0 and a corresponding diameter of 0.42 mm at D_{16} [0.1 mm + (16 × 0.02 mm)]. For a size #50 instrument, the diameters are 0.5 mm at D_0 and 0.82 mm at D_{16}.

The tip size increases by 0.05 mm for file sizes #10 to #60; for sizes #60 to #140, the absolute increase is 0.1 mm (see Fig. 9-18). Recalculation of these diameter increments into relative steps (in percentages) reveals dramatic differences: the step from size #10 to #15 is 50%, whereas the increase from size #55 to #60 is less than one fifth of that change (see Fig. 9-18).

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 endpoint 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.[337]

The ISO specifications inadvertently complicated the cleaning and shaping of root canal systems. The ISO-normed design is a simplification that has specific disadvantages, and it may explain the clinical observation that enlarging from size #10 to #15 is more difficult than the step from size #55 to #60. The introduction of Golden Medium files (DENTSPLY Maillefer), which have tip sizes between the ISO-stipulated diameters, seemed to solve the problem. However, their use is not that important clinically, because the approved machining tolerance of ± 0.02 mm negates the intended advantage. Moreover, although ± 0.02 mm tolerance is stipulated by the ISO norm (see Fig. 9-17), most manufacturers do not adhere to it.[208,365,404,487]

A subsequent modification involved tips with a constant percentage of diameter increments, the Series 29. The first ProFile instruments (DENTSPLY Tulsa Dental, Tulsa, OK, USA) followed this design, with a nominal diameter increase of 29%. 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 actually greater than the percentage change found in the ISO file series.

Hedström Files

Hedström files are milled from round, stainless steel blanks. They are very efficient for translational strokes,[355] but rotational working movements are strongly discouraged because of the possibility of fracture. Hedström files up to size #25 can be efficiently used to relocate canal orifices and, with adequate

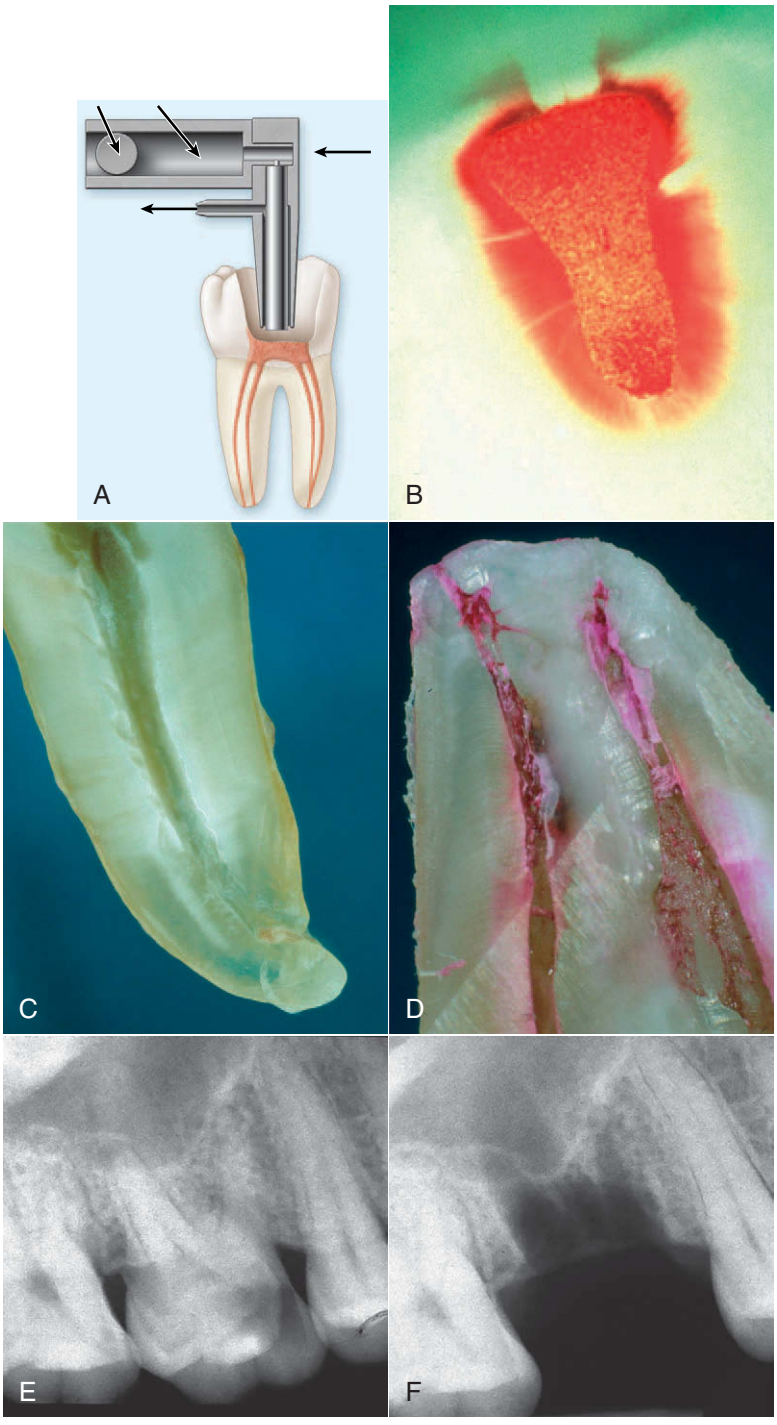


FIG. 9-10 Spectrum of strategies for accomplishing the primary aim of root canal treatment: elimination of infection. **A,** Schematic diagram of minimally invasive therapy using the noninstrumentation technique (NIT). **B,** Example of teeth cleaned in vitro using NIT. Note the clean intracanal surface, 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.)*

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. 9-20). As with stainless steel K-files, Hedström files should be single-use instruments.[397]

Gates-Glidden Drills

Gates-Glidden (GG) drills are important instruments that have been used for more than 100 years without noteworthy design changes. These instruments, especially the nickel-titanium FlexoGates model (DENTSPLY Maillefer),[151] usually work well for preenlargement of coronal canal areas.[114,261] However, when misused, GG drills can dramatically reduce radicular wall thickness.[149,197,239]

GG instruments are manufactured in a set and numbered 1 to 6 (with corresponding diameters of 0.5 to 1.5 mm); the number of rings on the shank identifies the specific drill size. GG instruments are available in various lengths and made by several manufacturers. Each instrument has a long, thin shaft

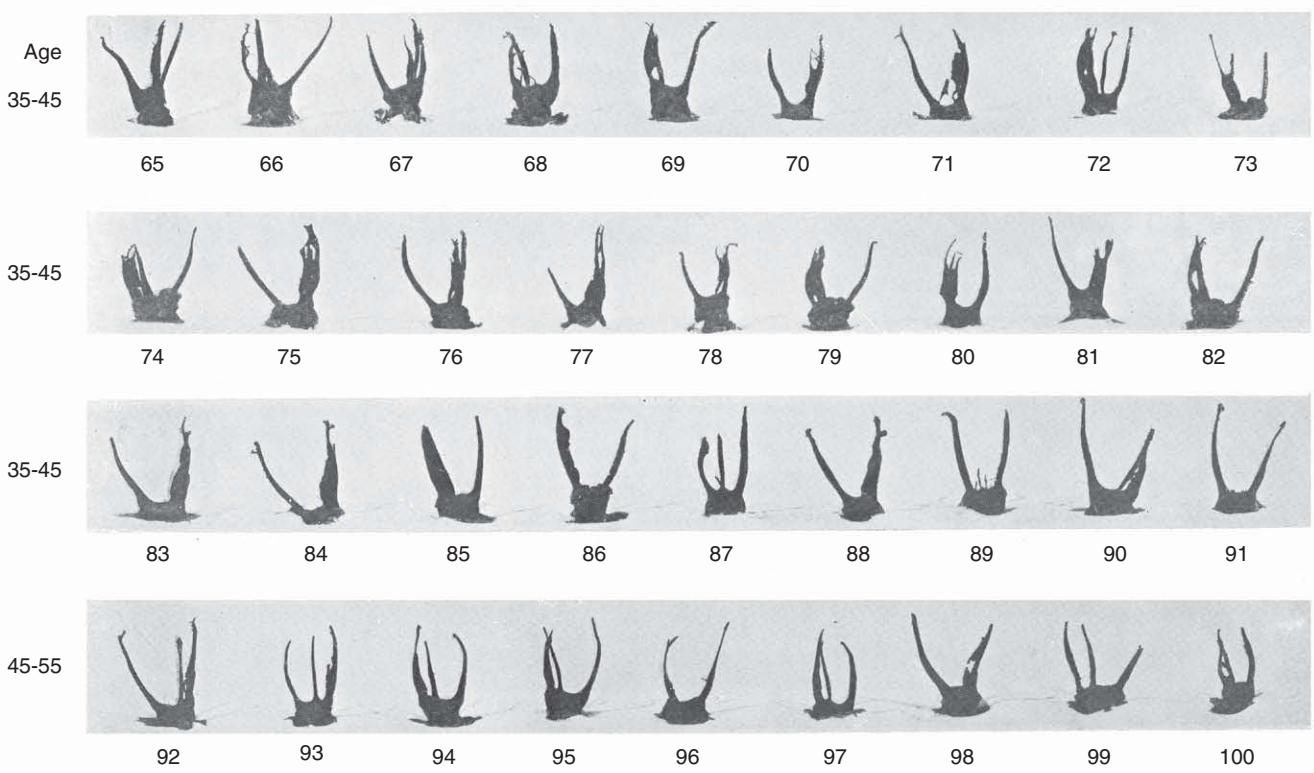


FIG. 9-11 Panel of 36 anatomic preparations of maxillary molars from the classic work by Professor Walter Hess of Zurich. Note the overall variability of root canal systems and the decrease of canal dimensions with age. *(From Hess W:* The anatomy of the root canals of teeth of the permanent dentition, *London, 1925, John Bale, Sons & Danielsson.)*

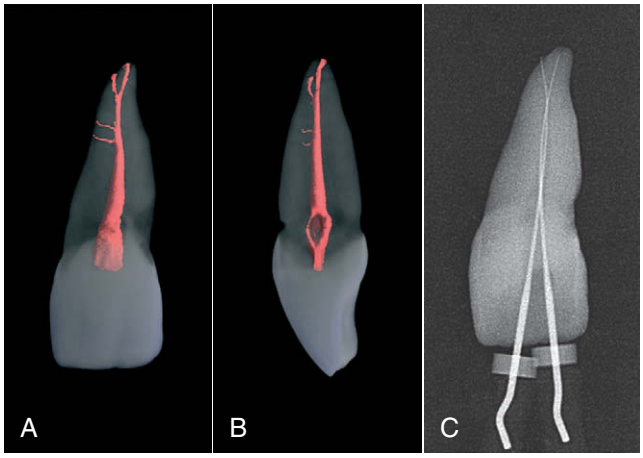


FIG. 9-12 Micro–computed tomographic scans of dental anatomy (36 µm resolution). **A,** Clinical view of tooth #9 shows two accessory canals and an apical bifurcation. **B,** Mesiodistal view of the tooth shown in **A**. **C,** Working length radiograph, with files placed in both apical canal aspects.

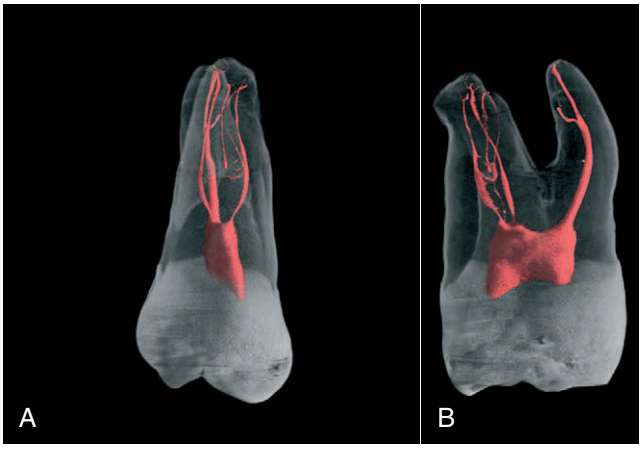


FIG. 9-13 Micro–computed tomographic scans of more complicated dental anatomy (36 µm resolution). **A,** Clinical view of tooth #3 shows a fine mesiobuccal and distobuccal canal system with additional anatomy in all three roots. **B,** Mesiodistal view of the tooth shown in **A.**

with parallel walls and a short cutting head. Because of their design and physical properties,[66] GG drills are side-cutting instruments with safety tips; they can be used to cut dentin as they are withdrawn from the canal (i.e., on the outstroke).[337] Used this way, their cutting action can deliberately be directed away from external root concavities in single-rooted and furcated teeth.[4] GG instruments should be used only in the straight portions of the canal, and they should be used serially and passively.[424]

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

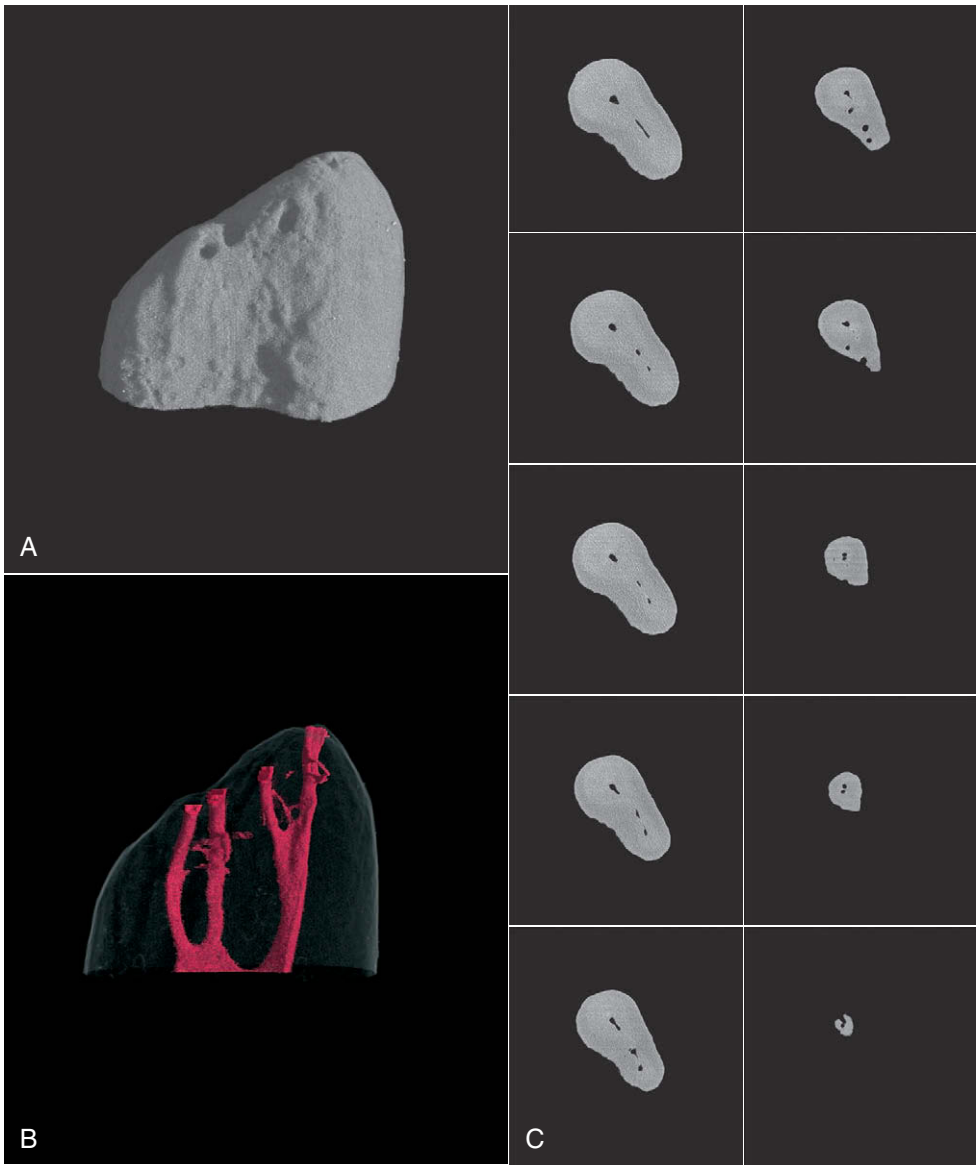


FIG. 9-14 Micro–computed tomographic scan of anatomy of the apical 5 mm of a mesiobuccal root (8 µm resolution). **A-B,** Three-dimensional reconstruction of outer contour and root canal systems. **C,** Cross sections 0.5 mm apart.

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.

As stated earlier, when used adequately, GG instruments are inexpensive, safe, and clinically beneficial tools. 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. 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. Gates-Glidden drills may be used safely and to their fullest potential at 750 to 1500 rpm. As with nickel-titanium rotary instruments, GG drills work best when used in electric gear reduction handpieces rather than with air motors.

Nickel-Titanium Rotary Instruments

Since the early 1990s, several instrument systems manufactured from nickel-titanium (NiTi) have been introduced into endodontic practice. The specific design characteristics vary,

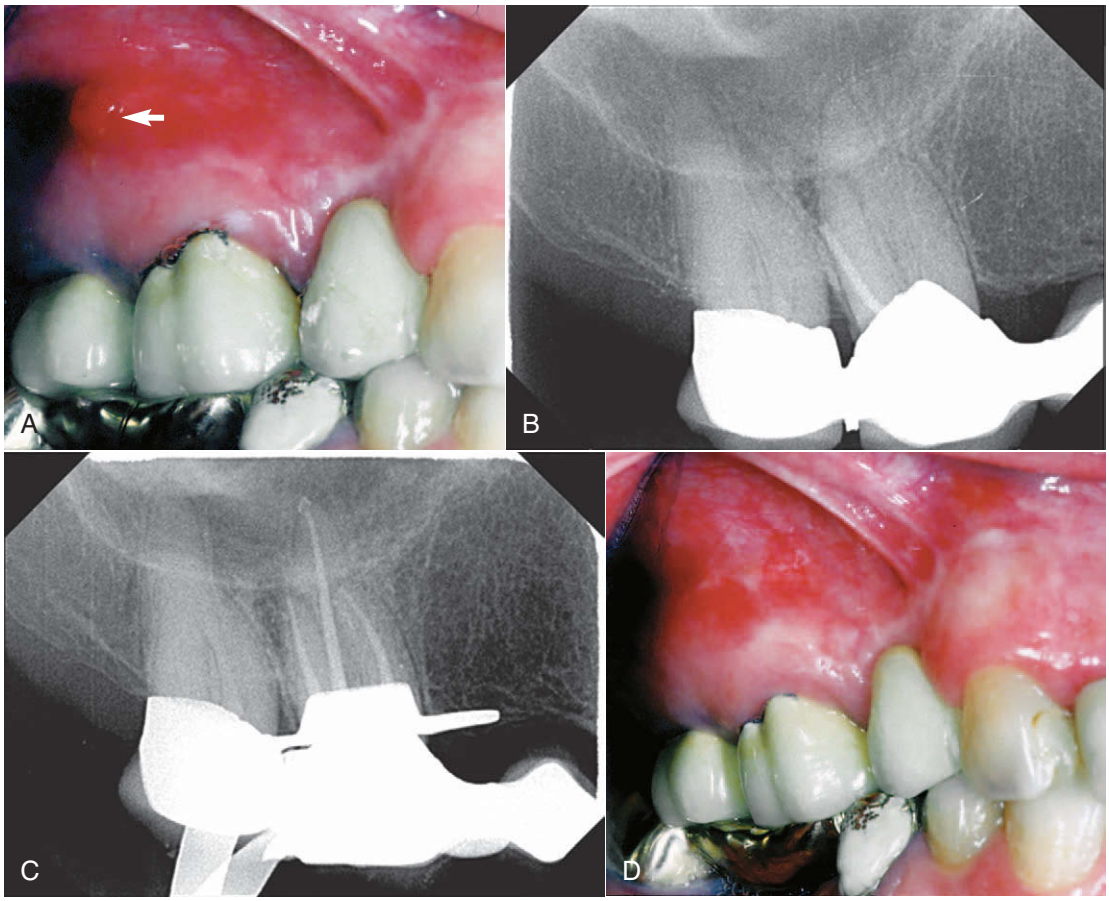


FIG. 9-15 Sinus tract as a sign of a chronic apical abscess and effect of routine root canal treatment. **A,** Intraoral photograph of left maxillary region with draining sinus tract *(arrow)* periapical to tooth #14. **B,** Pretreatment radiograph with gutta-percha point positioned in the sinus tract, 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.

such as tip sizing, taper, cross section, helix angle, and pitch (Fig. 9-21). Some of the early systems have been removed from the market or play only minor roles; others, such as ProFile (DENTSPLY Tulsa Dental, DENTSPLY Maillefer), are still widely used. New designs continually are produced, but the extent to which clinical outcomes (if any) will depend on design characteristics is difficult to forecast.[303]

Most of the instruments described in this section are manufactured by a grinding process, although some are produced by laser etching and others by plastic deformation under heating. Precision at the surface quality is not really at a high level, whereas the tolerances are. Surface quality also is an important detail (see Fig. 9-21), because cracks that arise from superficial defects play a role in instrument fracture.[16] Superficial defects such as metal flash and rollover are common in unused NiTi instruments.[125,251,489]

Attempts have been made to improve surface quality by electropolishing the surface and coating it with titanium nitride.[325,353] The latter process also seems to have a beneficial effect on cutting efficiency.[353]

In essence, two properties of the NiTi alloy are of particular interest in endodontics: superelasticity (Fig. 9-22) and high resistance to cyclic fatigue (discussed later). These two properties allow continuously rotating instruments to be used successfully in curved root canals. Many variables and physical properties influence the clinical performance of NiTi rotaries.[218,302,370,413]

Much of what is known about NiTi instruments, including reasons for instrument fracture[33] and instrument sequences, has been gleaned from clinical practice. In vitro research continues to clarify the relationship between NiTi metallurgy and instrument performance, but already NiTi rotary instruments have become an important adjunct in endodontics.[301]

NiTi rotary instruments have substantially reduced the incidence of several clinical problems (e.g., blocks, ledges, transportation, perforation), but they are also believed to fracture somewhat more easily than hand instruments. This does not by itself predispose a case to posttreatment disease; rather, a retained instrument fragment limits access of disinfecting irrigants to the root canal system, possibly impeding sufficient elimination of microorganisms.[172]

The following sections describe the instruments most widely used in the United States and Europe for root canal preparation. 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, the LightSpeed; group II, rotary instruments with #.04 and #.06 tapers, which includes the ProFile and many other

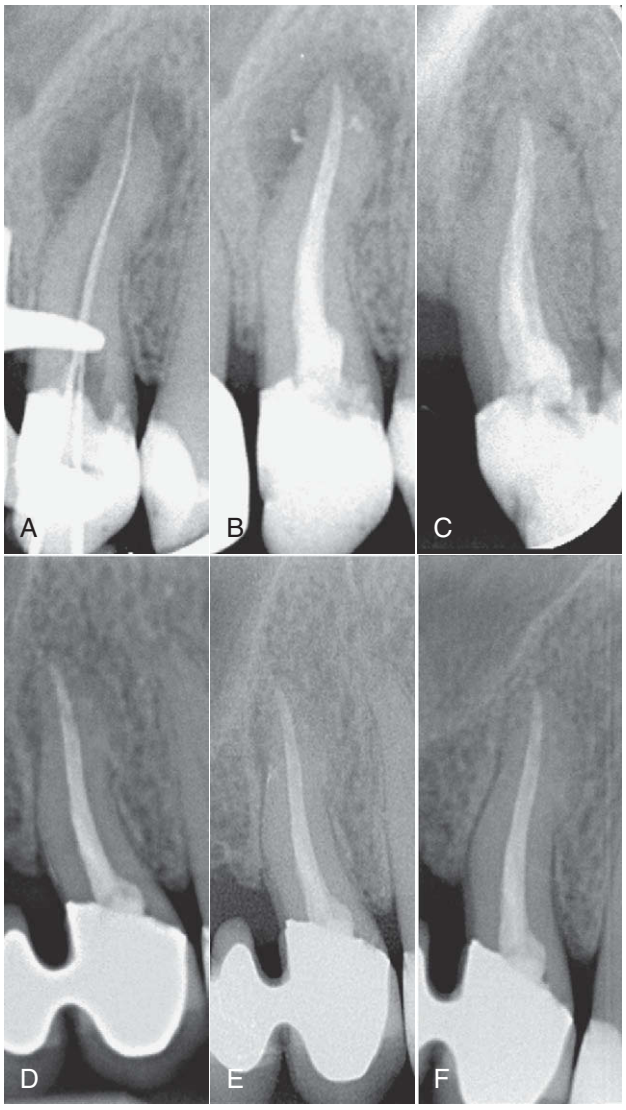


FIG. 9-16 Relationship of radicular anatomy and endodontic disease as shown by filled accessory canals. **A,** Working length radiograph of tooth #13 shows lesions mesially and distally but not apically. **B,** Posttreatment radiograph shows the accessory anatomy. **C,** Six-month recall radiograph before 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.

models; and group III, rotary instruments with specific design changes, such as the ProTaper (DENTSPLY Maillefer) and RaCe (FKG, La Chaux-de-Fonds, Switzerland).

LightSpeed and LightSpeed LSX Instruments

The LightSpeed file, developed by Dr. Steve Senia and Dr. 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 part. The same design principles apply to the recently developed LSX instrument (Discus Dental, Culver City, CA, USA) (Fig. 9-23) that is manufactured not by milling but by stamping. 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 recommended working speed for LS1 instruments is 1500 to 2000 rpm and for LSX, 2500 rpm. Both variants should be used with minimal torque,[32] owing to the thin shaft.[120]

Cross sections of LightSpeed LS1 cutting parts show three round excavations, the U-shape design common to many earlier NiTi instruments, whereas the LSX is shaped like a flat chisel in cross section (see Fig. 9-23). Because of the thin noncutting shaft, both types of LightSpeed instruments are considerably more flexible than any other instrument on the market. In addition, cyclic fatigue is lower than with all other instruments, allowing the use of higher rpm speeds. All LightSpeed instruments feature a noncutting tip.

Because of their design, LightSpeed LS1 and LSX require specific instrumentation sequences to produce canal shapes amenable to root canal filling. The current recommendation calls for an apical 4-mm zone to be prepared to a cylindrical, nontapered shape. This section may then be filled with the proprietary SimpliFill system (Discus Dental). Different sequences are required for lateral compaction or other filling techniques.

The original LightSpeed is a widely researched NiTi rotary instrument,[49,319,321,375,415,416] and most reports have found that the system has a low incidence of canal transportation and preparation errors. Loss of working length was also minimal in most of these studies. Data regarding the LSX are sparse. One report found similar shaping abilities for LSX and LightSpeed LS1 assessed with a double-exposure technique.[196]

ProFile

The ProFile system (DENTSPLY Tulsa Dental) was introduced by Dr. Ben Johnson in 1994. In contrast to the LightSpeed instrument with its thin, flexible shaft, 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 taper (Fig. 9-24). The tips of the ProFile Series 29 rotary instruments had a constant proportion of diameter increments (29%). Because of the nonstandardized diameters, obturation was performed with nonstandardized gutta-percha cones, using either lateral compaction or thermoplastic obturation of gutta-percha (see Chapter 10). Later, another ProFile series with ISO-sized tips (DENTSPLY Maillefer) was developed and marketed in Europe. This set was believed to better accommodate standardized gutta-percha cones, which are predominantly used in Europe. Subsequently, instruments with even greater tapers and 19-mm lengths were introduced, and a .02 variant was added.

Cross sections of a ProFile instrument show a U-shape design with radial lands and a parallel central core. Lateral views show a 20-degree helix angle, a constant pitch, and bullet-shaped noncutting tips. Together with a neutral or 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.

The recommended rotational speed for ProFile instruments is 150 to 300 rpm, and to ensure a constant rpm level, the preferred means is electrical motors with gear reduction rather than air-driven motors.

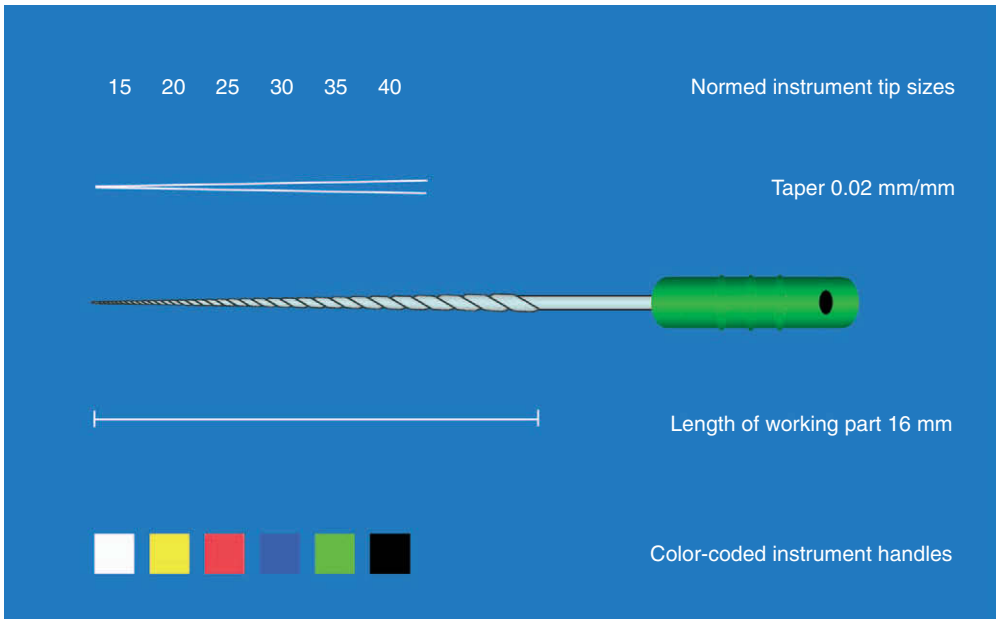


FIG. 9-17 Schematic drawing of an ISO-normed hand instrument size #35. Instrument tip sizing, taper, and handle colors are regulated by the ISO/ANSI/ADA norm.

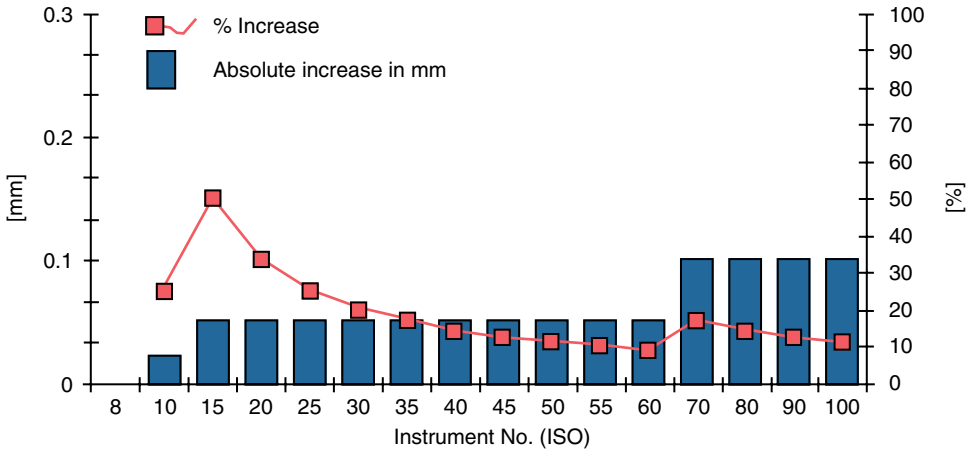


FIG. 9-18 Increase in tip diameter in absolute figures and in relation to the smaller file size. Note the particularly large increase from size #10 to size #15.

ProFile instruments shaped canals without major preparation errors in a number of in vitro investigations.[72,73,417,418] A slight improvement in canal shape was noted when size .04 and .06 tapered instruments were used in an alternating fashion.[71] Loss of working length did not exceed 0.5 mm[71-73,417,418] and was not affected by the use of .06 tapered instruments.[71] Comparative assessments in vitro suggested that ProFile prepared mesial canals in mandibular molars with less transportation than K3 and RaCe.[14]

A very recent addition to the ProFile family of instruments is the Vortex (DENTSPLY Tulsa Dental). The major change lies in the non-landed cross section, whereas tip sizes and tapers are similar to existing ProFiles. Manufactured using M-Wire, Profile Vortex also have varying helical angle to counteract the tendency of non-landed files to thread into the root canal. At this point, comparatively little clinical or experimental data are available for ProFile Vortex.

GT and GTX Files

The Greater Taper, or GT file, was introduced by Dr. Steve Buchanan in 1994. This instrument also incorporates the U-file design and was marketed as ProFile GT. 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. This decreased the length of the cutting flutes and increased the taper. The instruments had a variable pitch and an increasing number of flutes in progression to the tip; the apical instrument diameter was 0.2 mm. Instrument tips were noncutting and rounded (Fig. 9-25); these design principles are mostly still present in the current incarnation, the GTX instrument. The main differences are the NiTi alloy type used (M-Wire, manufactured by SportsWire, Langley, OK) and a different approach to instrument usage, emphasizing the use of the #20 .06 rotary.

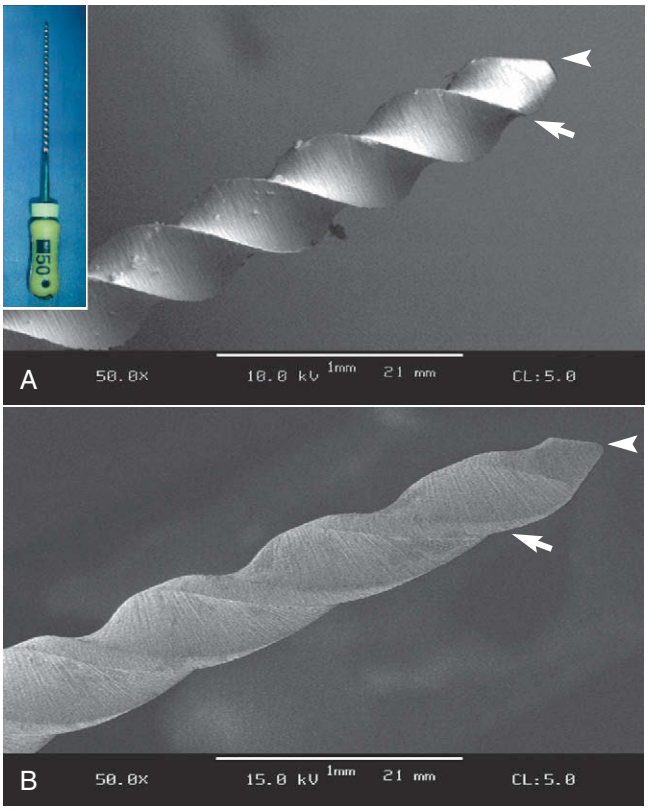


FIG. 9-19 Flute geometry and tip configuration of a hand file *(insert)* and a NiTi rotary instrument. **A,** K-file with sharp cutting edges *(arrow)* and Batt tip *(arrowhead)*. **B,** GT rotary file with rounded, noncutting tip *(arrowhead)*, smooth transition, and guiding radial lands *(arrow)*.

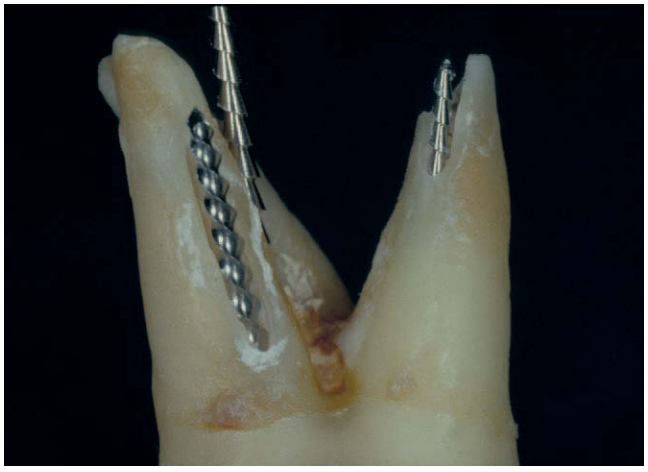


FIG. 9-20 Result of an overenthusiastic attempt at root canal treatment of a maxillary second molar with large stainless steel files. Multiple strip perforations occurred; consequently, the tooth had to be extracted.

The GTX set currently includes tip sizes 20, 30, and 40, in tapers ranging from .04 to .010 (see Fig. 9-25). The recommended rotational speed for GT and GTX files is 300 rpm, and the instrument should be used with minimal apical force to avoid fracture of the tip.

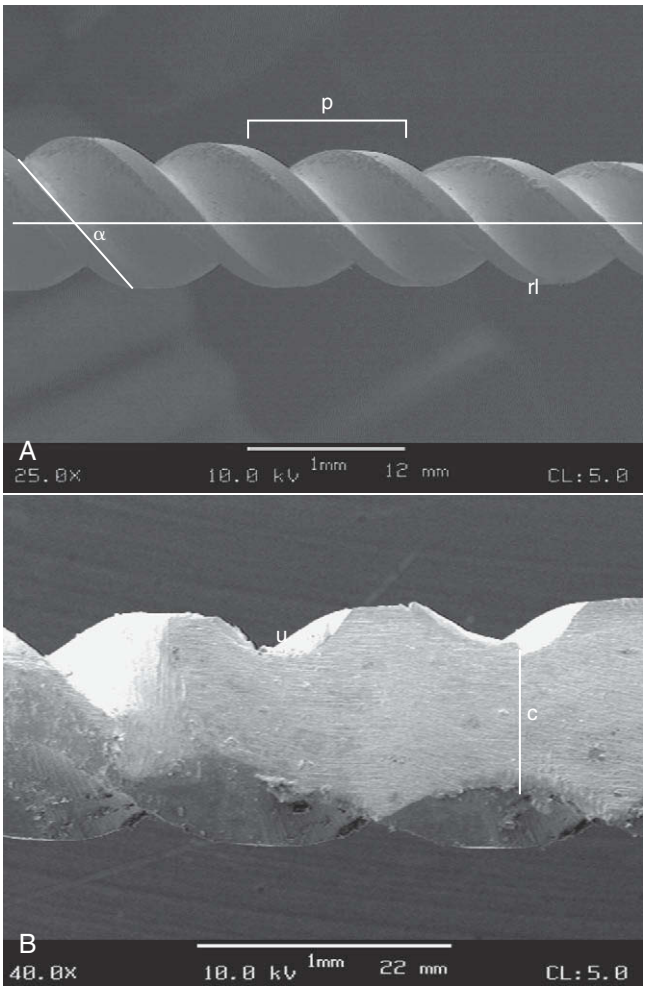


FIG. 9-21 Design characteristics of nickel-titanium rotary instruments. **A,** Lateral view showing the details of the helix angle, pitch *(p)*, and the presence of guiding areas, or radial lands *(rl)* (scanning electron micrograph [SEM], ×25). **B,** Ground working part of the instrument in **A,** showing U-shaped excavations and the dimension of the instrument core *(c)*.

Studies on GT files found that the prepared shape stayed centered and was achieved with few procedural errors.[149,178,310,316,479] A shaping assessment using µCT showed that GT files machined statistically similar canal wall areas compared with ProFile and LightSpeed preparations.[310] These walls were homogeneously machined and smooth.[294,479]

HERO 642, Hero Shaper

First-generation rotary systems had neutral or slightly negative rake angles. Several second-generation systems were designed with positive rake angles, which gave them greater cutting efficiency. HERO instruments (MicroMega, Besançon, France) are an example of a second-generation system; the original system known as *HERO 642* has now been replaced by HERO Shaper, with very little difference in the instrument design.

Cross sections of HERO instruments show geometries similar to those of an H-file without radial lands (Fig. 9-26). Tapers of .02, .04, and .06 are available in sizes ranging from #20 to #45. The instruments are relatively flexible (the acronym

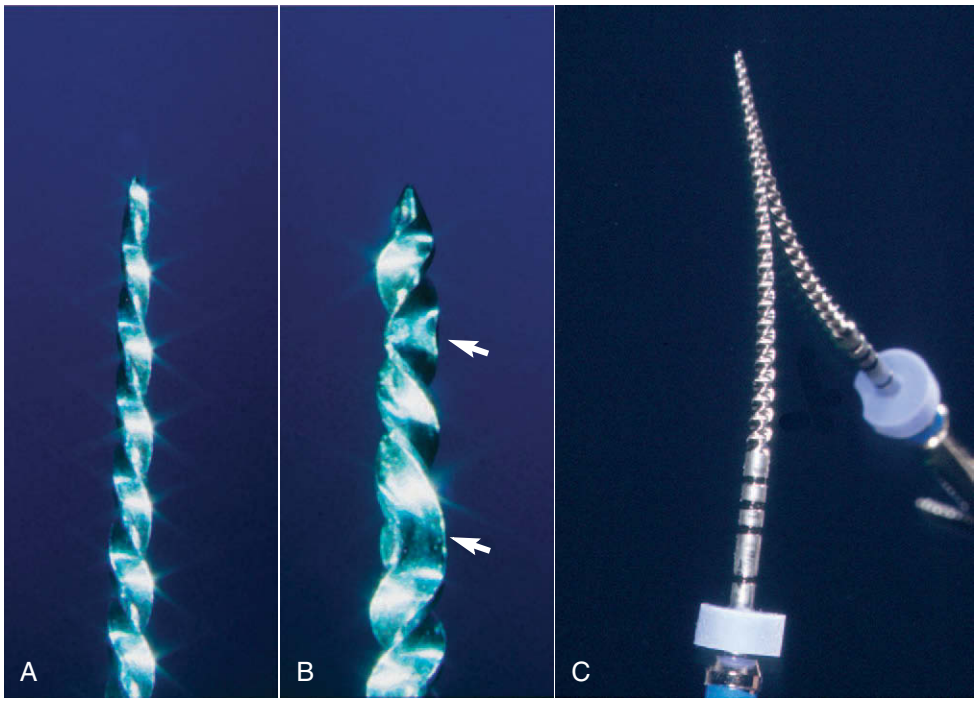
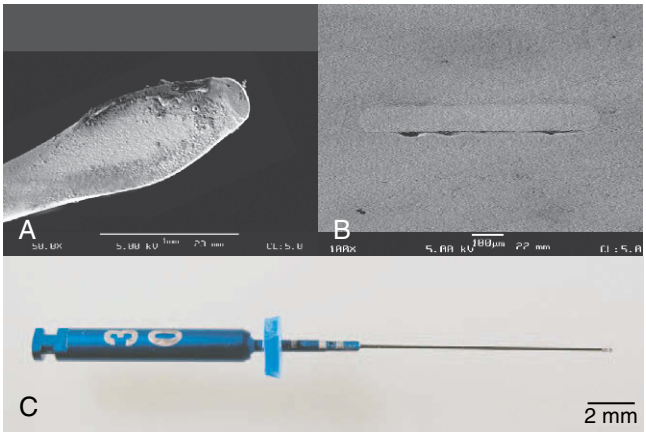


FIG. 9-22 Deformation of endodontic instruments manufactured from nickel-titanium alloy. **A** and **B,** Intact and plastically deformed ProFile instruments (*arrow* indicates areas of permanent deformation). **C,** ProFile instrument placed on a mirror to illustrate elastic behavior.



D

Number of instruments/ sets	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
12 (20-80)	20-130	5 (20-70) 10 (80-130)	2500 rpm, (minimal axial force)	21, 25, and 31 mm

FIG. 9-23 Design features of a LightSpeed instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

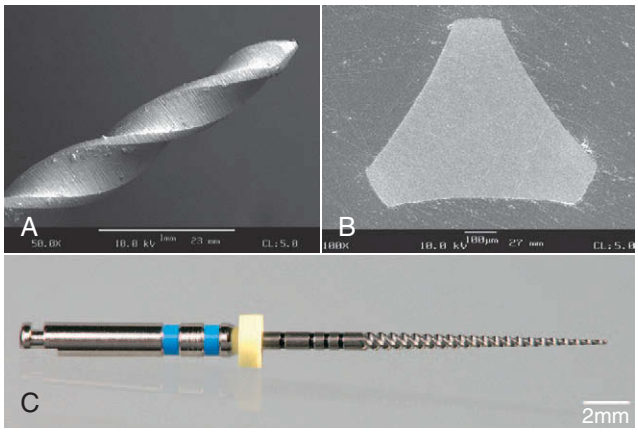
HERO stands for *h*igh *e*lasticity in *ro*tation) but maintain an even distribution of force into the cutting areas.[430,431] HERO instruments have a progressive flute pitch and a noncutting passive tip, similar to other NiTi rotary systems. The instruments are coded by handle color.

Research with HERO files indicates a shaping potential similar to that of the FlexMaster[189] (DENTSPLY VDW, Munich, Germany) and the ProFile,[147] although in one study the HERO induced more changes in cross-sectional anatomy.[157] HERO instruments also were found to cause some aberrations when used in simulated canals with acute curves[414] but were safer than Quantec SC instruments (SybronEndo, Orange, CA).[193] More recently, HERO Shapers were found to have a better centering ability compared to RaCe instruments in resin blocks.[28] Comparing earlier HERO 642 and current HERO Shaper rotaries, no differences were found assessing cross sections before and after shaping in a modified Bramante technique.[84]

ProTaper Universal

The ProTaper system is based on a unique concept and originally comprised just six instruments: three shaping files and three finishing files. This set is now complemented by two larger finishing files and a set designed for retreatment procedures. The instruments were designed by Dr. Cliff Ruddle, Dr. John West, and Dr. Pierre Machtou. In cross section, ProTaper shows a modified K-type file with sharp cutting edges and no radial lands (Fig. 9-27); this creates a stable core and sufficient flexibility for the smaller files. The cross section of finishing files F3, F4, and F5 is slightly relieved for increased flexibility. A unique design element is varying tapers along the instruments' long axes. The three shaping files have tapers that increase coronally, and the reverse pattern is seen in the five finishing files.

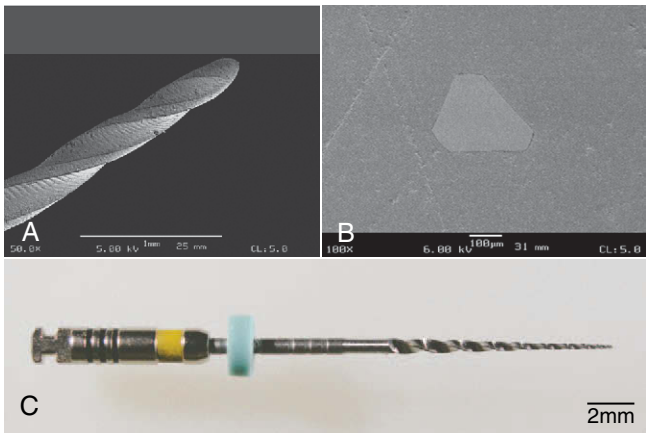
Shaping files #1 and #2 have tip diameters of 0.185 mm and 0.2 mm, respectively, 14-mm-long cutting blades, and partially active tips. The diameters of these files at D_{14} are 1.2 and 1.1 mm, respectively. The finishing files (F1-F5) have tip diameters of 0.2, 0.25, 0.3, 0.4, and 0.5 mm, respectively, between D_0 and D_3, and the apical tapers are .07, .08, .09, .05, and .04, respectively. The finishing files have rounded noncutting tips.



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

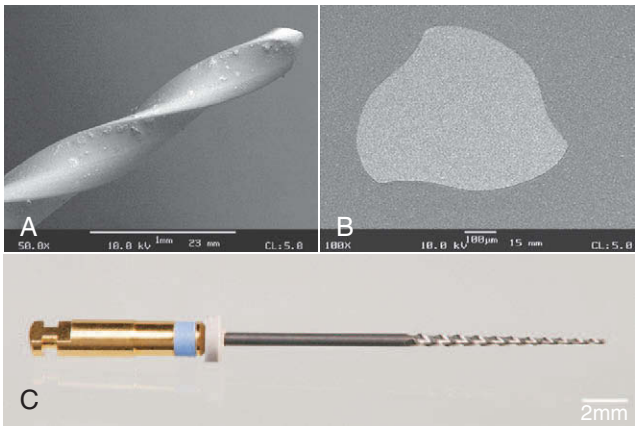
FIG. 9-24 Design features of a ProFile instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.



D

Number of instruments/ sets	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
Size 20 rotaries: 2 Size 30 rotaries: 3 Size 40 rotaries: 3	(.04, .06) (.04, .06, .08) (.04, .06, .08)	10	300 rpm	21, 25, and 31 mm

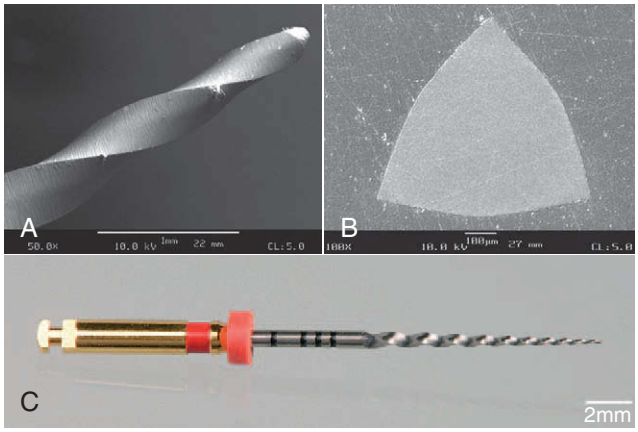
FIG. 9-25 Design features of a GT-file. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.



D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
12	20, 25, 30 with .02, .04, and .06 taper; 35 to 45 with .02 taper	5	300-600, with minimal axial force	21, 25 mm

FIG. 9-26 Design features of a HERO instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.



D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
6 (3 shaping files; SX, S1, S2; 3 finishing files; F1, F2, F3)	19-30	Vary along the working part of an individual instrument	150 to 350 minimal axial force, low to medium torque to fracture, varying working torque	19, 21, 25 mm

FIG. 9-27 Design features of a ProTaper instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

The convex triangular cross section of ProTaper instruments reduces the contact areas between the file and the dentin. The greater cutting efficiency inherent in this design has been safely improved by balancing the pitch and helix angle, preventing the instruments from inadvertently threading into the canal. The instruments are coded by colored rings on the handles. ProTaper instruments can be used in gear reduction electrical handpieces at 250 to 300 rpm, in accordance with universally recognized guidelines. Two usage characteristics have been recommended for ProTaper. The first is the preparation of a glide path, either manually[298] or with special rotary instruments.[52] An enlargement to a size approaching the subsequent rotaries' tips prevents breakage and allows assessment of the canal size.[298] The second specific recommendation is the use of a more lateral "brushing" working stroke. Such a stroke allows the clinician to direct larger files coronally away from danger zones and counteract any "threading-in" effect.[58] Both usage elements should be considered good practice for other instruments, particularly more actively cutting ones.[314]

In a study using plastic blocks, the ProTaper created acceptable shapes more quickly than GT rotary, ProFile, and Quantec instruments[479] but 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 (Brasseler Komet, Lemgo, Germany).[440] In a comparison of ProTaper and K3 instruments (SybronEndo, Orange, CA), Bergmans et al.[48] found few differences, with the exception of some transportation by the ProTaper into the furcation region. 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.[308] It has been recommended that ProTaper be combined with less tapered, more flexible rotaries to reduce apical transportation.[199]

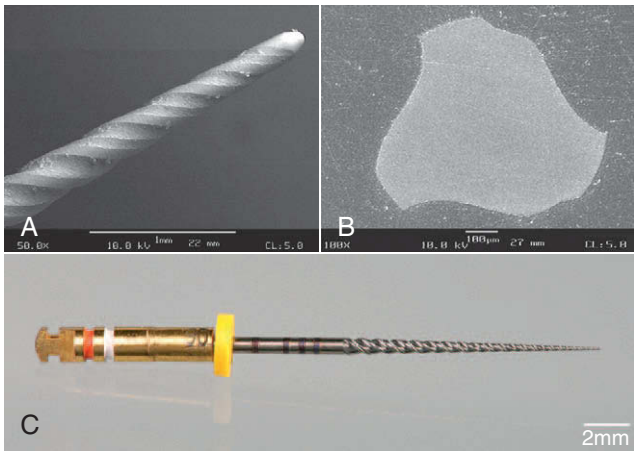
K3

In a sequence of constant development 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 and the HERO 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 (Fig. 9-28): 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 complicated design is relatively difficult to manufacture, resulting in some metal flash (see Fig. 9-28).

Like most other 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 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[48] and superior to that achieved with hand instruments.[357] More recently, when curved canals in lower molars were shaped to a size #30 .06,[14] K3 had less canal transportation in a modified Bramante model than RaCe but more than ProFile.



D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
27	15-45 with .02 taper; 15-60 with .04 and .06 taper	5	300 to 350, minimal axial force	21, 25, 30 mm

FIG. 9-28 Design features of a K3 instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

FlexMaster

The FlexMaster file system currently is unavailable in the United States. It also features .02, .04, and .06 tapers. The cross sections (Fig. 9-29) have a triangular shape, with sharp cutting edges and no radial lands. This makes for a relatively solid instrument core and excellent cutting ability. The overall manufacturing quality is high, with minimal metal flash and rollover.

FlexMaster files have rounded, passive tips; the tip diameters are 0.15 to 0.7 mm for size .02 instruments and 0.15 to 0.4 mm for size .04 and .06 files (see Fig. 9-29). In addition to the standard set, the Intro file, which has a .11 taper and a 9-mm cutting part, is available. 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[188] and that it performed on par with other systems.[189,455] Clinical studies confirmed that the FlexMaster showed superior shaping characteristics compared with K-files.[358] Also, novice dental students were able to shape plastic blocks successfully with the FlexMaster after a short training period.[392,393] Tested in a well-described model of simulated canals, FlexMaster instruments led to few aberrations but took longer than preparation with RaCe files.[263] 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.[362]

RaCe, Bio Race

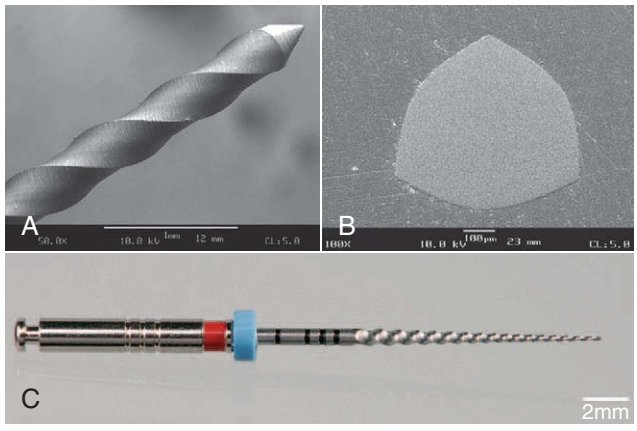
The RaCe has been manufactured since 1999 by FKG and was later distributed in the United States by Brasseler (Savannah, GA). The name, which stands for *reamer with alternating cutting edges,* describes just one design feature of this instrument (Fig. 9-30). Light microscopic imaging of the file shows flutes and reverse flutes alternating with straight areas; this design is aimed at reducing the tendency to thread the file into the 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 (see Fig. 9-30).

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 are marked by color-coded handles 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 preparations two larger 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[359,360]: canals in plastic blocks and in extracted teeth were prepared by the RaCe with less transportation from the original curvature than occurred with the ProTaper.[359] In a separate study, ProTaper and RaCe performed similarly when canals were prepared to an apical size #30.[293] When preparing to a size #40, RaCe prepared canals rapidly and with few aberrations or instrument deformities.[324] The newer BioRaCe instrument sequences attempt to utilize .02 tapered instruments to promote larger apical sizes; this is also possible in a hybrid technique.

EndoSequence

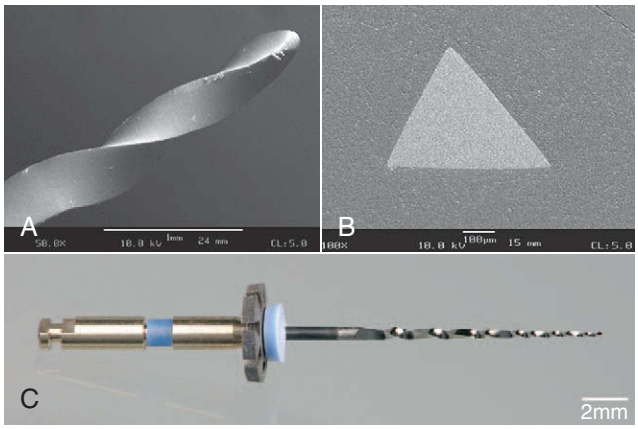
The Sequence rotary instrument is produced by FKG in Switzerland and marketed in the United States by Brasseler. This is another instrument that adheres to the conventional



D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
22	15-70 (.02) 15-40 (.04 and .06)	5; tapers of .02, .04, or .06	280 (150 to 300), Minimal axial force, low working torque	21, 25, 28 mm (.02 and .04 only)

FIG. 9-29 Design features of a FlexMaster instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.



D

No. of instruments/ set	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
15	15-60 (.02)	5 and 10	Up to 600	19
	25-35 (.04) 30, 40 (.06) 35 (.08) 40 (.10)	Varying tapers	Minimal axial force	25 mm

FIG. 9-30 Design features of an RaCe instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

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 (Fig. 9-31), but the manufacturer claims that a unique longitudinal design called *alternating wall contact points* (ACP) reduce torque requirements and keep the file centered in the canal. Another feature of the Sequence design is an electrochemical treatment after manufacturing, similar to RaCe files, that results in a smooth, polished surface. This is believed to promote better fatigue resistance, hence a rotational speed of 600 rpm is recommended for EndoSequence.[214]

Twisted File

In 2008, SybronEndo presented the first fluted NiTi file (Fig. 9-32, *A*) manufactured by plastic deformation, a process similar to the twisting process that is used to produce stainless steel K-files. 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 size #25 tip sizes only, in taper .04 up to .12.

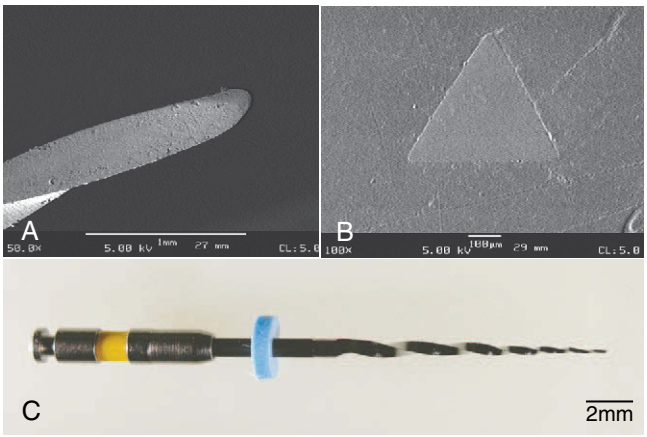
The unique production process is believed to result in superior physical properties; indeed, early studies suggested significantly better fatigue resistance of size #25 .06 taper Twisted File compared to K3 instruments of the same size and size #20 .06 GTX.[146] Moreover, as determined by bending tests according to the norm for hand instruments, ANSI/ADA No. 28 (ISO 3630), Twisted Files size #25 .06 taper were more flexible than ProFiles of the same size.[145]

The manufacturer recommends a conventional crown-down technique after securing a glide path with a size #15 K-file. Specifically, for a "large" canal, tapers .10 to .06 should be used, and in a "small" canal, tapers .08 to .04 are recommended. Although early reports suggest that the Twisted File is clinically resistant to fatigue, there are no reports available at this point that show improved healing outcomes compared to other rotary files.

The preceding descriptions covered only a limited selection of the most popular and widely used rotary instruments on the market. New files are continually added to the armamentarium, and older systems are updated. This is partly the reason for the scarcity of clinical outcome studies at this point.

To summarize, most systems include files with tapers greater than the #.02 stipulated by the ISO norm. The LightSpeed LS1 and LSX are different from all other systems; the ProTaper, RaCe, and Twisted File have some unique features; and most other systems have increased tapers. Minor differences exist in tip designs, cross sections, and manufacturing processes, but the clinical effects of these modifications currently are unknown. Even in vitro, tests have only begun to identify the effect of specific designs on shaping capabilities,[50,192,301] and differences in clinical outcomes in regard to these design variations appear to be minimal.[165,303,358]

Physical parameters governing rotary root canal preparation are crucial because NiTi rotary files are felt to have an increased risk of fracture compared with K-files. In a study using plastic blocks, as many as 52 ProFile Series 29 instruments became permanently deformed.[417] Three fractures were reported in a subsequent study on ISO-norm ProFile size #.04 instruments, and three other instruments were distorted.[73] An even higher fracture incidence was shown in a study on rotary instruments used in plastic blocks in a specially designed testing machine.[412]

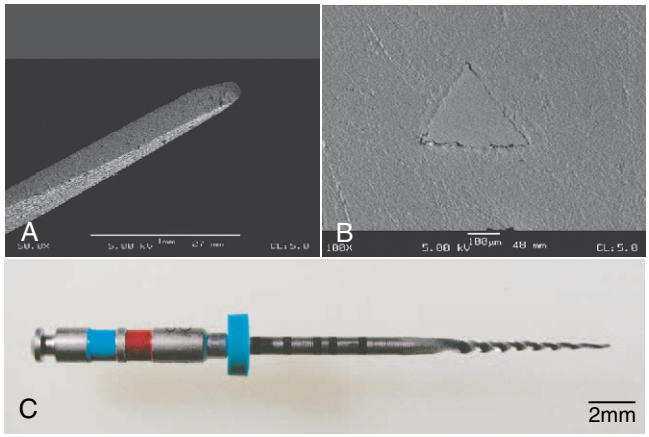


D

Number of instruments/ sets	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
4* plus Expeditor	15-60 (.04) 15-50 (.06)	5	500-600 rpm	21, 25, and 31 mm

*Separate packs exist for small, medium, and large canals.

FIG. 9-31 Design features of a Sequence instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.



D

Number of instruments/ sets	Tip sizes	Size increments	r.p.m. (recommended)	Lengths
3*	25, tapers .04-.12	None	500 rpm	23 and 27 mm

*Sets for large and small canals.

FIG. 9-32 Design features of a Twisted File (TF) instrument. **A,** Lateral view (scanning electron micrograph [SEM], ×50). **B,** Cross section (SEM, ×200). **C,** Lateral view. **D,** Design specifications.

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.[33,345]

On the other hand, a retrospective clinical study suggest similar outcomes with and without retained instrument fragments[400]; moreover, others' experiences suggest that the number of rotary instrument fractures is lower than previously estimated.[116,213,460] 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.[405,453]

Consequently, a benefit-versus-risk analysis should be carried out prior to attempts to remove NiTi instrument fragments, addressing the reasons and the clinical consequences of instrument fracture.

Physical and Chemical Properties of NiTi Alloys

During the development of the equiatomic nitinol alloy (55% [by weight] nickel and 45% [by weight] titanium), a shape memory effect was noted; this was attributed to specific thermodynamic properties of the new alloy.[75] The alloy sparked interest in dental research because of its "shape recovery" property after passage through critical temperatures.[100] Some researchers envisioned the manufacture of nondulling rotary instruments from an alloy called *60-nitinol*. However, NiTi wire was found to be difficult to bend into clamp retainers.[100]

Subsequently, researchers thought that the superelastic properties of 55-nitinol might prove advantageous in endodontics, and the first hand instruments produced from 55-nitinol were tested (Fig. 9-33).[445] That study found that size #15 NiTi instruments were two to three times more flexible than stainless steel instruments. Nickel-titanium instruments showed superior resistance to angular deflection; they fractured after 2½ full revolutions (900 degrees) compared to 540 degrees for stainless steel instruments (see Fig. 9-33, C).

Furthermore, hardly any plastic deformation of cutting flutes was recorded when an instrument was bent up to 90 degrees,[445] and forces required to bend endodontic files to 45 degrees were reduced by 50% with NiTi.[370] In the latter study,

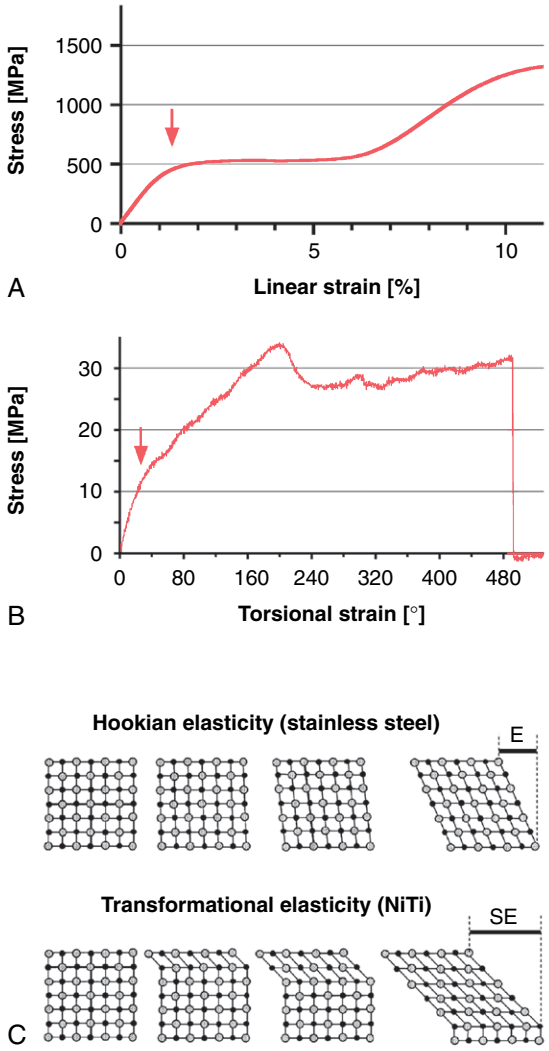


FIG. 9-33 Stress-strain behavior of nickel-titanium alloy. **A,** Schematic diagram of linear extension of a NiTi wire. **B,** Torque to failure test of a size #60, #.04 taper ProFile NiTi instrument. Note the biphasic deformation, indicated by arrows in **A-B. C,** Comparison of stainless steel and 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 superelastic *(SE)* behavior of NiTi alloy. *(**C** modified from Thompson SA: An overview of nickel-titanium alloys used in dentistry.* Int Endod J *33:297-310, 2000.)*

the authors speculated that heat, probably during sterilization cycles, could even restore the molecular structure of used NiTi files, resulting in an increased resistance to fracture.

Specific properties of NiTi can be explained by specific crystal structures of the austenite and martensite phases of the alloy.[413] Heating the metal above 212° F (100° C) may lead to a phase transition, and the shape memory property forces the instrument back to a preexisting form. Likewise, linear deforming forces are shunted into a stepwise transition from an austenitic to a martensitic lattice, and this behavior leads to a recoverable elastic response of up to 7% (see Fig. 9-33, A).

However, graphs such as those shown in Fig. 9-33, B are generated when larger NiTi instruments are subjected to angular deflection until failure. Such graphs show different results for stainless steel instruments, which produce a relatively steep stress-strain curve with less than 1.3% recoverable deformation.[413] As stated previously, the superelastic behavior of NiTi also dictates the production of NiTi instruments, which are usually milled.

Similarly to phase transformations induced by strain, heating and cooling NiTi can also result in conformational changes.[179,265] Thermal conditions during the production of the raw wire can be used to modify its properties—most importantly, its flexibility.

Recently, such a thermal process was harnessed to allow twisting of raw NiTi material into the shape of a nonlanded rotary instrument (Twisted File, SybronEndo). This process is believed to respect the grain structure of the material better and does not introduce milling marks or other surface irregularities.

Typically, NiTi instruments may have characteristic imperfections such as milling marks, metal flashes, or rollover.[125,370,426,445] Some researchers have speculated that fractures in NiTi instruments originate at such surface imperfections.[16,237]

Surface irregularities may also provide access to corrosive substances, most notably sodium hypochlorite (NaOCl). Some studies have suggested that chloride corrosion may lead to micropitting[344] and possibly subsequent fracture in NiTi instruments.[174] Not only immersion in various disinfecting solutions for extended periods (e.g., overnight) produced corrosion of NiTi instruments and subsequent decreased torsional resistance,[284,394] but for ProTaper,[51] RaCe, and ProFile[309] instruments, also short-term immersion. Other authors, however, did not find a corrosion-related effect on K3[35] or ProFile[257] instruments. Regular cleaning and sterilization procedures do not seem to affect NiTi rotary instruments.[231,266,406] In one study, only limited material loss occurred when NiTi LightSpeed instruments were immersed in 1% and 5% NaOCl for 30 to 60 minutes.[78] 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. Although sterilization procedures per se do not have an impact on NiTi integrity,[184,266,326,377,442,489] 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 work-hardening,[15] depending on the amount of torsional load the instrument was subjected to.

Over the last 4 years, several manufacturers have begun to utilize electropolishing, a process that removes surface irregularities such as flash and burr marks. It is believed to improve material properties, specifically fatigue and corrosion resistance; however, the evidence for both these claims is mixed. One study[24] found an extension of fatigue life for electropolished instruments, others found no improvement of fatigue resistance of electropolished instruments.[76,94,182] Still other researchers[59] suggested a change in cutting behavior with an increase of torsional load after electropolishing. Corrosion resistance of electropolished NiTi rotaries is also controversial. One study[62] found superior corrosion resistance for electropolished RaCe instruments, whereas another study[309] found similar corrosion susceptibility for RaCe and non-

electropolished ProFile instruments. A recent review[374] points out difficulties in assessing NiTi properties by conventional corrosion tests, since they do not take deformation and consequent surface deformation into account.

Fracture Mechanisms

In general, instruments used in rotary motion break in two distinct modes, torsional and flexural.[302,345,433] 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.[370] NiTi instruments can withstand several hundred flexural cycles before they fracture,[173,227,321,433,475] but they still can fracture in the endodontic setting after a low (i.e., below 10,000) number of cycles.[92]

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.[66] In GG burs, a 2-mm deflection of the instrument tip resulted in fatigue lifespans ranging from 21,000 revolutions (size #1 burs) to 400 revolutions (size #6 burs).[66] 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.[370] 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 size #.06 instruments using a similar device.[474,475] 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[474]; again, no significant differences were found in resistance to cyclic fatigue.

One study[173] used a different testing method involving tempered metal cylinders with radii of 5 mm and 10 mm that produced a 90-degree curve. They 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,[313] and although the radius of the curves was halved, fatigue-life was reduced by 400%. Another investigation[173] reported similar results for selected HERO instruments, and their 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.[306]

Reuse of rotary instruments depends on safety, specifically on assessment of fatigue and also the potential to properly clean NiTi surfaces.[34,61,284,289,376,394,396,427] Specific instruments perform differently in this regard, since fatigue depends more on the amount of metal in cross section at the point of stress concentration[159,425] than on the specifics of instrument design.[93]

On the other hand, manufacturers 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.[203] However, investigators[216] could not confirm these findings. Similarly, another study[223] did not find the Twisted File, which is not milled and hence believed to be fatigue resistant,[146] to perform better than conventionally manufactured ProFile rotaries. Another feature, electropolishing (see earlier) does not appear to confer a significantly increased fatigue resistance to EndoSequence[223,328] and RaCe.[425,427,471] One possible reason for these variable outcomes are the different testing environments used in vitro[95]; 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,[117] since 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. 9-34). These systems can simultaneously assess torque at failure, working torque axial force, and cyclic fatigue (Fig. 9-35).

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

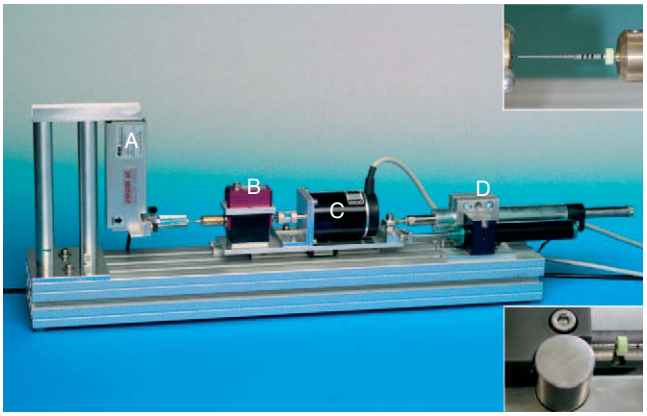


FIG. 9-34 Testing platform for analysis of various factors during simulated canal preparation with rotary endodontic instruments. Labeled components are a force transducer **(A),** a torque sensor **(B),** a direct-drive motor **(C),** and an automated feed device **(D).** For specific tests, a cyclic fatigue phantom or a brass mount compliant with ISO No. 3630-1 *(inserts)* may be attached.

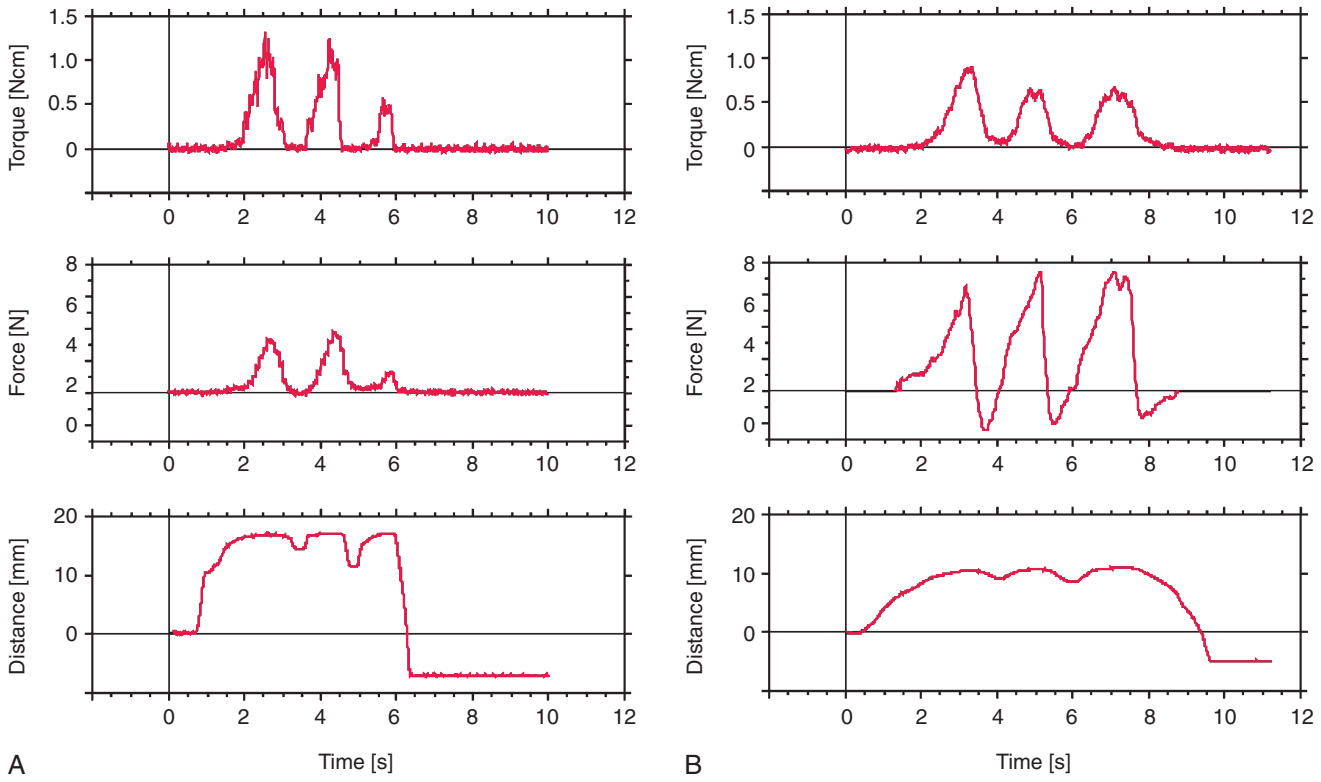


FIG. 9-35 Physical factors (torque, axial force, and insertion depth) that affect root canal instrumentation documented with a torque-testing platform. **A,** ProFile size #45, #.04 taper used in a mildly curved canal of a single-rooted tooth, step-back after apical preparation to size #40. **B,** FlexMaster size #35, #.06 taper used in a curved distobuccal canal of a maxillary first molar, crown-down during the initial phase of canal preparation.

rotary files in ISO sizes #25, #30, and #35 (#.04 taper) fractured at 0.78, 1.06, and 1.47 Ncm, respectively.[406]

Investigators[407] reported similar scores when instruments were forced to fracture in plastic blocks with simulated curved canals. 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.[306] These values are somewhat lower than recent data obtained from the same but slightly modified torque bench,[209] pointing towards the importance of experimental conditions for torque and fatigue measurements.

Compared with NiTi instruments with tapered flutes, LightSpeed instruments had lower torques to fracture (0.23 to 2 Ncm[254]). 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[345] (Fig. 9-36). However, these are not separate entities, especially in curved canals.[63] Working an instrument with high torque may lower resistance to cyclic fatigue.[142] Conversely, cyclic prestressing has been shown to reduce the torsional resistance of ProTaper finishing files,[433] as well as K3[30] and MTwo[313] (DENTSPLY VDW, Munich, Germany). 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.[364] 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*).[57,476]

The clinician can further modify torque by varying axial pressure, because these two factors are related[364] (see Fig. 9-35). 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.

Motors and Devices

Newer motors have been developed for rotary instruments since the simple electric motors of the first generation in the early 1990s (Fig. 9-37, *A*). Electric motors with gear reduction are more suitable for rotary NiTi systems because they ensure a constant rpm level; however, they also deliver torques much higher than those required to break tips. Some authors believe that torque-controlled motors (see Fig. 9-37, *B* to *D*), which

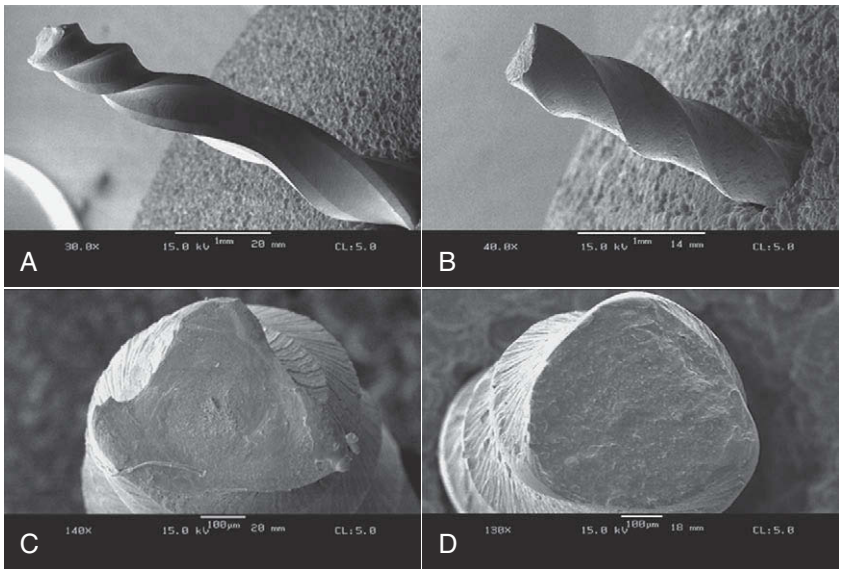


FIG. 9-36 Scanning electron micrographs of deformed or separated nickel-titanium rotary instruments. **A,** Lateral view of a ProTaper F3 instrument after application of torsional load (×25). **B,** Lateral view of a size #35, #.04 taper FlexMaster instrument after more than 500 rotations in a 90-degree curve with a 5-mm radius (see Figure 9-31) (×30). **C,** Cross section of the ProTaper instrument in **A.** Note signs of ductile fracture near center of the instrument core (×140). **D,** Cross section of FlexMaster instrument in **B** (×100).



FIG. 9-37 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,** Newest-generation motor with built-in apex locator and torque control.

have been used for several years, increase operational safety.[143] However, others have suggested that torque-control motors may be helpful mainly to inexperienced clinicians.[476] 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 D_3, a fracture at a smaller diameter (D_2) is still possible.

To complicate matters further, an obvious differential exists between torque at failure at D_3 and the working torque needed to operate an instrument effectively (Fig. 9-38 and Box 9-2).[58,187,302,307,346] In many cases, the working torque is greater than the torque required to fracture the instrument's tip. However, the tip will not break if a passive glide path has been verified.

This differential is especially large with files with a taper equal to or greater than #.06; therefore, these files are rather ineffective in most torque-controlled motors. Most motors allow adjustment of torque for the instrument used, either with a key or a system card that is inserted into the box.

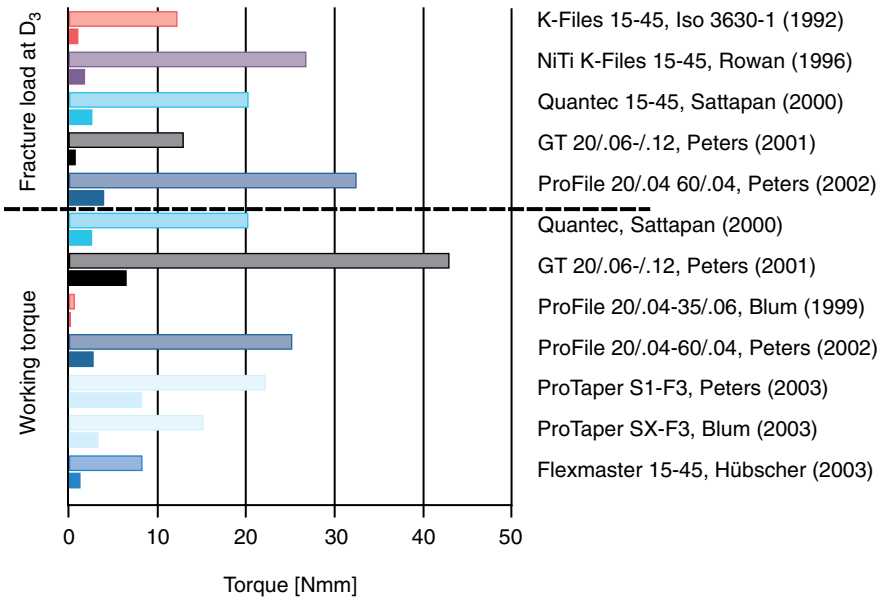


FIG. 9-38 Diagram comparing fracture loads at D_3 *(upper section of graph)* to torques occurring during preparation of root canals *(lower section of graph)*. Filled columns represent the largest file in each set, and open columns show the scores of the most fragile file (see text and Box 9-3 for details).

BOX 9-2

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.

BOX 9-3

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

One way to mitigate problems with continuous rotation (e.g., taper lock, fatigue fracture) is to return to oscillation. One researcher[472] has recently described such an approach. Based on experiments evaluating the maximal rotational angle before plastic deformation for the selected instrument, he recommends ProTaper F2, a forward angle of 144 degrees, followed by a reverse rotation of 72 degrees. This cycle continues at 400 rpm until working length is reached. Preliminary experiments and clinical results are encouraging, but more research is needed to optimize motor settings and instrument selection for this approach.

Other factors that may influence the incidence of fracture in motor-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, Norristown, PA) and Glyde (DENTSPLY Maillefer) have also been recommended, their benefit may be restricted to plastic blocks,[23] rather than relevant when rotary instruments engage dentin surfaces.[60,304] In fact, because of chemical interactions between NaOCl and ethylenediamine tetra-acetic acid (EDTA),[161] alternating irrigants and using lubricants that contain EDTA may even be counterproductive. Moreover, no data have been produced linking the use of lubricants to reduction of torque during clinical root canal preparation.

For instrument motion, most manufacturers usually recommend a pecking, up-and-down motion. This not only prevents threading in of the file, it is also believed to distribute stresses away from the instrument's point of maximum flexure where fatigue failure would likely occur.[227,321] However, such in-and-out movements did not significantly enhance the lifespan of ProFile size #.04 or GT rotary instruments rotated around a 5-mm radius cylinder with a 90-degree curve.[302,305] Furthermore, large variations were noted in the lengths of the fractured segments,[187,433] which suggests that ductile fractures may originate at points of surface imperfections.

Rotational speed may also influence instrument deformation and fracture. Some studies indicated that ProFile instruments with ISO-norm tip diameters failed more often at higher rotational speed,[119,141] whereas other studies did not find speed to be a factor.[113,206]

Clinicians must fully understand the factors that control the forces exerted on continuously rotating NiTi instruments (Box 9-3). To minimize the risk of fracture and prevent taper lock, they should not try to force motor-driven rotary instruments 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, a detailed knowledge of anatomic structures, and specific instrumentation sequences may also improve shaping results. Moreover, in a well-controlled study, clinical outcomes were statistically similar comparing teeth with and without retained instrument fragments.[400] This was true irrespective of

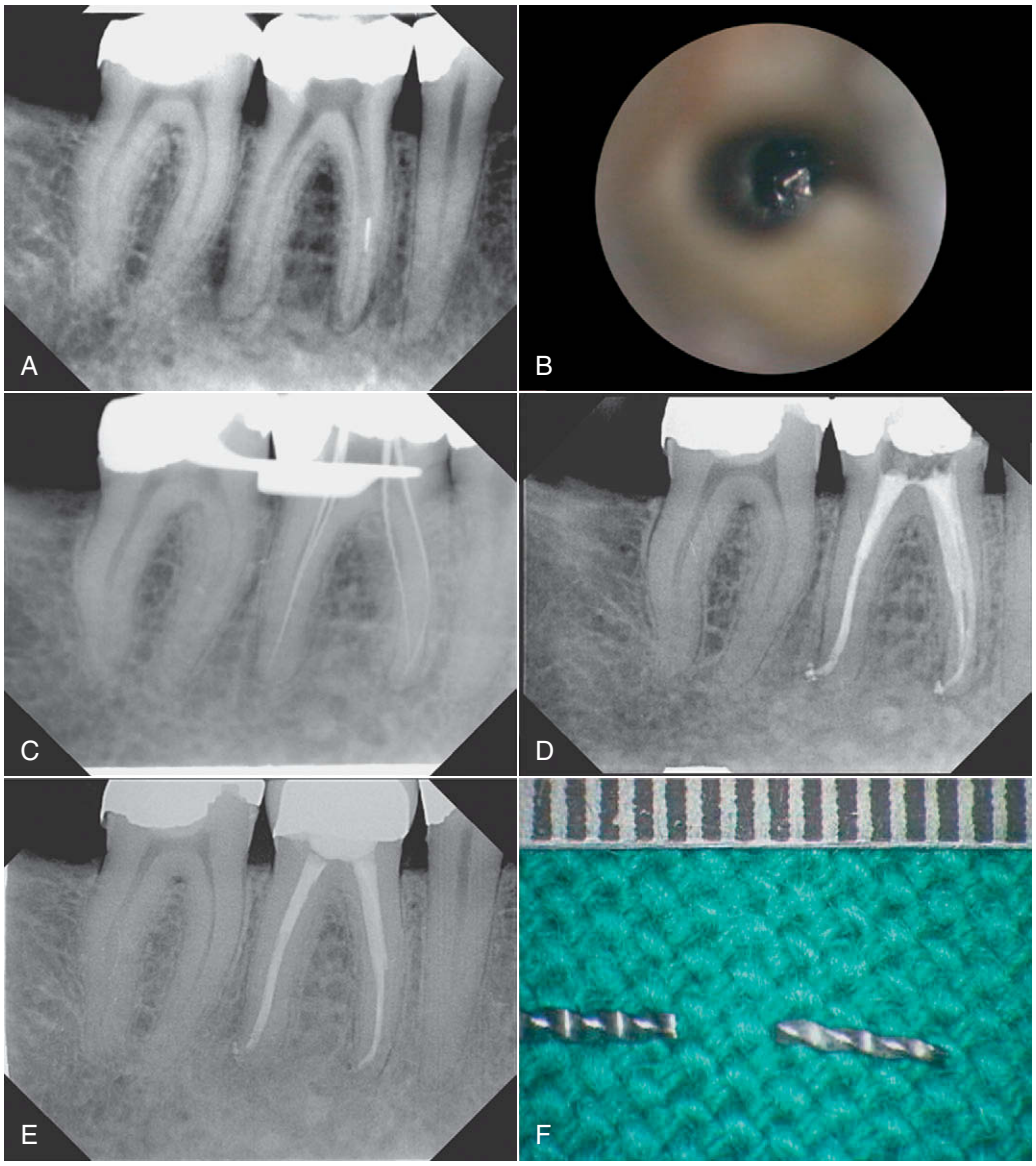


FIG. 9-39 Removal of a separated NiTi instrument from a mesiolingual canal of a mandibular molar. **A,** Fragment located in the middle third of the root. **B,** Clinical aspect of the fragment after enlargement of the coronal third of the root canal with modified Gates-Glidden drills, visualized with 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).

the pretreatment diagnosis; as stated before, this underlines the importance of a detailed case assessment before removal of a retained instrument fragment is attempted.

Specific procedures have been developed for removing fractured instruments from root canals (Fig. 9-39); these are discussed in detail elsewhere in this book (see Chapter 25). 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.

Disinfectants, Dentin Surface Modifiers, and Lubricants

Studies have demonstrated conclusively that mechanical instrumentation cannot sufficiently disinfect root canals, regardless of whether stainless steel[80] or NiTi[112] instruments are used (Fig. 9-40). Irrigation solutions are required to eradicate microbiota, and over time, a variety of chemicals have been promoted for this purpose. The ideal irrigant or combination of irrigants kills bacteria, dissolves necrotic tissue, lubricates the canal, removes the smear layer, and does not irritate healthy tissues.[153,191] Some formaldehyde-containing materials are no longer recommended for clinical use, but many irrigating solutions and varying concentrations of commonly used

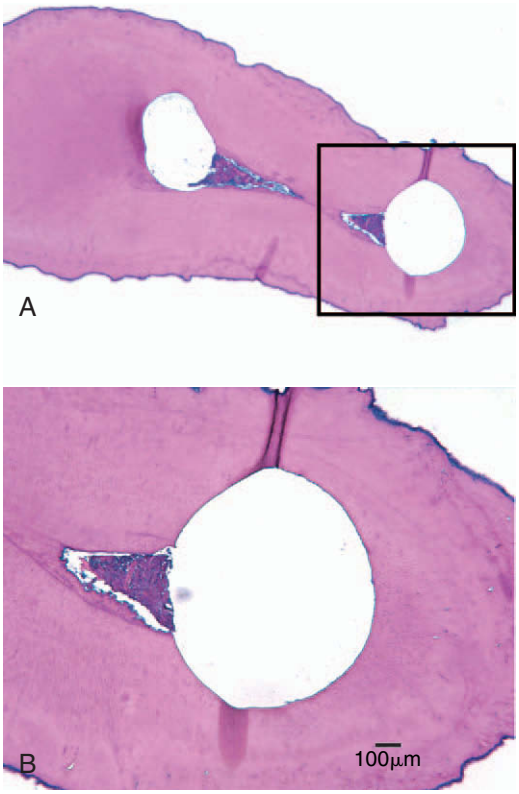


FIG. 9-40 Remaining potentially infected tissue in fins and isthmus configuration after preparation with rotary instruments. **A,** Cross section through a mesial root of a mandibular molar, middle to coronal third of the root. Both canals have been shaped; the left one is transported mesially (×10). **B,** Magnified view of rectangle in **A.** Note the presence of soft tissue in the isthmus area (×63). *(Courtesy Professor H. Messer.)*

materials are described in the literature. Some solutions used in the past were sterile saline, alcohol, hydrogen peroxide, NaOCl, and detergents (e.g., quaternary ammonium compounds, chlorhexidine, citric acids, and EDTA).[399] The following section describes current irrigation solutions and gives some recommendations for their clinical use.

Sodium Hypochlorite

NaOCl encompasses many desirable properties of a main root canal irrigant and has therefore been described as the most ideal of all available rinsing agents.[268,481] NaOCl has been in use for almost a century.[111,446] A 0.5% solution of NaOCl was used effectively during World War I to clean contaminated wounds.[111] In the endodontic field, NaOCl possesses a broad-spectrum antimicrobial activity against endodontic microorganisms and biofilms (Table 9-1), including microbiota difficult to eradicate from root canals, such as *Enterococcus, Actinomyces,* and *Candida* organisms.[175,320,338,481]

NaOCl dissolves organic material such as pulp tissue and collagen. If the organic portion of a smear layer is dissolved in NaOCl, and bacteria inside the main root canal, lateral canals, and dentinal tubules—if in direct contact with the irrigant—are destroyed, then to a minor degree, endotoxins may be eliminated.[256,444]

During endodontic therapy, NaOCl solutions are used at concentrations ranging from 0.5% to 6%. In infected dentin blocks, a 0.25% solution of NaOCl was sufficient to kill *Enterococcus faecalis* in 15 minutes; a concentration of 1% NaOCl required 1 hour to kill *Candida albicans*.[368] In infected extracted teeth, Ruff et al.[338] found that a 1-minute application of 6% NaOCl and 2% chlorhexidine were equally effective in eliminating microorganisms and statistically significantly superior to MTAD and 17% EDTA in eliminating *Candida albicans* infections.

Lower concentrations (e.g., 0.5% or 1%) dissolve mainly necrotic tissue.[482] Higher concentrations allow better tissue dissolution but dissolve both necrotic and vital tissue, which is not always a desirable effect. In some cases, full-strength NaOCl (6%) may be indicated, but although higher concentrations may increase antibacterial effects in vitro,[477] enhanced clinical effectiveness has not been demonstrated conclusively for concentrations stronger than 1%.[399]

Commercially available household bleach (Clorox, The Clorox Company, Oakland, CA) contains 6.15% NaOCl, has an alkaline pH of 11.4, and is hypertonic.[399,482] Some authors recommend dilution of commercially available NaOCl with 1% bicarbonate instead of water to adjust the pH to a lower level.[111,399] Others do not see any reduction of the aggressiveness on fresh tissue by buffering NaOCl and recommend diluting solutions of NaOCl with water to obtain less concentrated irrigation solutions.[383,482]

NaOCl only minimally removes dentin debris or smear layers (Fig. 9-41). Therefore, some authors recommend concurrent use of demineralizing agents to rid the root canal surface of a postinstrumentation smear layer and thus enhance cleaning of difficult-to-reach areas, such as dentinal tubules and lateral canals.[81,283] When using NaOCl over extended periods of time during treatment, it should be mentioned that NaOCl seems to have an undesired side effect on the flexural strength of dentin. One study[252] investigated the influence of irrigants on flexural strength of dentin bars and concluded that a 24-minute exposure time to a 2.5% hypochlorite solution caused a significant drop in flexural strength, while the modulus of elasticity was not altered during this time. Other authors discovered a lowering of both flexural and elastic strength after 2-hour submersion of dentin bars in NaOCl.[11,164] The loss of calcium ions appears to be both dependent on NaOCl concentration (5% showing the greatest amount of decalcification) and time of exposure.[351]

Chlorhexidine

Chlorhexidine (CHX) is a broad-spectrum antimicrobial agent effective against gram-negative and gram-positive bacteria (see Table 9-1). It has a cationic molecular component that attaches to negatively charged cell membrane areas, causing cell lysis.[26,156] CHX as a mouth rinse and periodontal irrigant has been used in periodontal therapy, implantology, and cariology for many years to control dental plaque.[130,232] Its use as an endodontic irrigant[40,96,131,181,454] is based on its substantivity and long-lasting antimicrobial effect, which arises from binding to hydroxyapatite. However, it has not been shown to have more superior clinical properties than NaOCl. In fact, an in situ disinfection study suggests no additive effect on typical endodontic flora.[259]

TABLE 9-1

Efficacy of Various Irrigants in Deactivating Microorganisms*

	NaOCl	CHX	IPI	MTAD	$Ca(OH)_2$
ENTEROCOCCI	**1 min** of 6% solution reduced biofilm by 7-8 orders of magnitude[459] **15 min** at 0.25% in contaminated dentin blocks[482] **30 min** at 0.5% and **2 min** at 5.25% in direct contact with bacteria[322]	**7 days** of 0.5% dressing resulted in complete killing in dentin blocks up to full depth of 950 μm[330] **24 hours** to reduce cultured bacteria below detection limit[318] and **5 min** when in direct contact with bacteria[122]	**24 hours** for iodine (2%) in potassium iodide (4%) resulted in complete killing in dentin blocks up to a depth of 700 μm[384] **1 hour** to reduce bacteria under 0.1% and **24 hours** below detection limit; however, loss of activity through dentin powder[317]	**5 min** application resulted in no growth on infected dentin[373] **MTAD** as efficient as 5.25% NaOCl in cultures[423]	**24 hours** to reduce cultured bacteria below detection limit, but inhibited by dentin powder, hydroxyapatite, and serum albumin[318] Showed little effect on *Enterococcus faecalis*[50] Complete killing in dentin blocks up to full depth of 950 μm[385] **7 days** of $Ca(OH)_2$ in 0.5% chlorhexidine acetate dressing resulted in complete killing in dentin blocks up to full depth of 950 μm[385]
***ACTINOMYCES* ORGANISMS**	**1 min** at 1% solution[37] **10 sec** at 0.5% in direct contact with bacteria[322]	No growth directly after rinsing with 2% CHX in patients with necrotic pulps and/or apical granuloma[127] **3 days** for 2% CHX to eliminate *Actinomyces israelii* from all samples of infected dentin[41]	After **60 days,** 25% of *Actinomyces israelii*–infected root canal walls and dentinal tubules in vitro treated with IKI still showed bacterial growth[41]	**ND**	After **60 days,** 50% of *Actinomyces israelii*–infected root canal walls and dentinal tubules in vitro treated with calcium hydroxide still showed bacterial growth[41]
***CANDIDA* ORGANISMS**	**1 hour** at 1% or 5% solution on root dentin with smear layer[368] **30 sec** for both the 0.5% solutions to kill all cells in culture[449] **1 min** of 6% solution: no growth[338]	**1 hour** at 0.12% on root dentin with smear layer[368] **10 sec** at 0.5% solution in direct contact with bacteria[322] **5 min** at 0.5% solution to kill all yeast cells and **1 hour** at 0.05% solution; less effective than IKI and NaOCl[449]	**30 sec** for both 2% and 4% solution to kill all cells in culture; 0.2% and 0.4% as effective as 0.5% CHX[449] **1 min** of 2% solution: no growth[338]	Not effective against *C. albicans* after 5 min exposure[338]	After **1 hour** and **24 hours,** only a small reduction of CFU observed[449] Not effective against *C. albicans* after 5 min exposure[338]

*Effects achieved by killing through antimicrobial action.

$Ca(OH)_2$, Calcium hydroxide; *CHX,* chlorhexidine acetate; *IKI,* iodine potassium iodide; *MTAD,* BioPure MTAD (*m*ixture of *t*etracycline, *a*cid, and *d*etergent); *NaOCl,* sodium hypochlorite; *ND,* not determined.

When compared to CHX as an irrigant, it was found that hypochlorite significantly more often achieved negative cultures than CHX.[333] When used as combination, NaOCl and CHX did not improve the antimicrobial activity of CHX against tested microorganisms.[443]

Some researchers found that CHX had significantly better antibacterial effects than $Ca(OH)_2$ when tested on cultures.[230] Combinations of CHX and $Ca(OH)_2$ are available and show antimicrobial activity against obligate anaerobes, the combination in some studies augmenting the antibacterial effect of either medicament on certain species.[315,385] However, 2% CHX gel alone was more effective than its combination with $Ca(OH)_2$ against several tested microorganisms in other investigations.[155,248] Medicaments containing 2% CHX have the ability

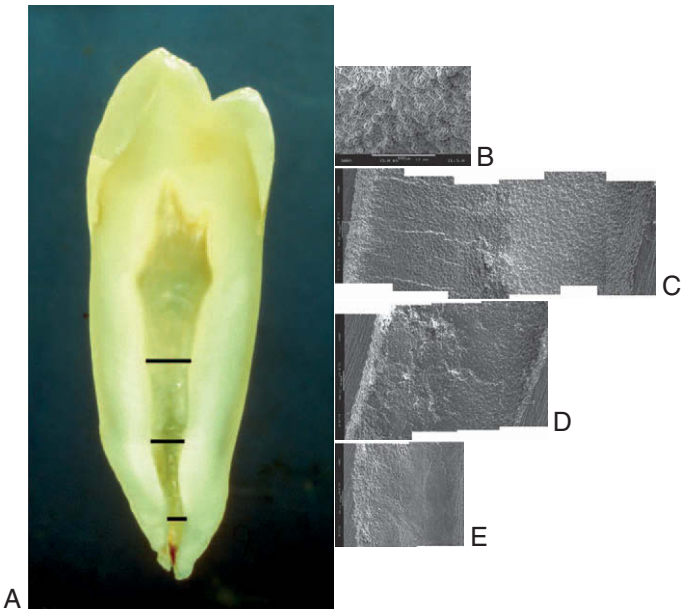


FIG. 9-41 Surface textures of an unprepared root canal at various levels. **A,** Ground section of a mandibular premolar. Areas viewed by scanning electron microscopy (SEM) are indicated by *black lines*. **B,** Canal surface at middle section, showing open dentinal tubules and typical calcospherites (×500). **C-E,** Coronal, middle, and apical areas as compound scanning electron micrographs. Note the numerous open tubules in **C-D,** whereas fewer tubules are visible in **E** (×200).

to diffuse through dentin and display antimicrobial action on the outer root surface. The addition of CHX or iodine potassium iodide to an intracanal dressing of $Ca(OH)_2$ in vitro did not affect the alkalinity (and hence the efficacy) of the $Ca(OH)_2$ suspensions.[385] Chlorhexidine (2%) has been advocated as a final rinse irrigant owing to its substantivity, which allows binding to dentin and sustained antimicrobial activity, especially in endodontic retreatment.[480]

Iodine Potassium Iodide

Iodine potassium iodide (IKI) is a traditional root canal disinfectant and is used in concentrations ranging from 2% to 5%. IKI kills a wide spectrum of microorganisms found in root canals (see Table 9-1) but shows relatively low toxicity in experiments using tissue cultures.[398] 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[385] evaluated the antibacterial activity of a combination of $Ca(OH)_2$ with IKI or CHX in infected bovine dentin blocks. 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[31] demonstrated that IKI was able to eliminate *E. faecalis* from bovine root dentin when used with a 15-minute contact time. An obvious disadvantage of iodine is a possible allergic reaction in some patients.

MTAD

BioPure (DENTSPLY Tulsa Dental), also known as *MTAD* (*m*ixture of *t*etracycline, *a*cid, and *d*etergent), is an irrigation solution that contains doxycycline, citric acid, and a surface-active detergent (Tween 80).[423] Chemicals and their combinations as root canal irrigants are developed constantly, including solutions based on antibiotics. However, doxycycline and other locally applied antibiotics have been unable to destroy microbiota organized in biofilms. One research group[282] investigated the effect of five antibiotics on a mature biofilm after 8 days of growth on dentin; in their experiment, none of the topically applied substances could eradicate the biofilm. Use of these irrigants is also controversial because of the emergence of increasingly resistant strains of bacteria (e.g., therapy-resistant enterococci), which may be due to overprescription of antibiotics in general. The increased risk of host sensitization by local antibiotics can be circumvented to some degree by using the antibiotic as a dressing. Because exposure to vital tissues is limited, higher microbicidal concentrations may be used.[269] A number of antibiotics, including erythromycin, chloramphenicol, tetracycline, and vancomycin, have been tested successfully against enterococci. In one study, investigators evaluated microbial susceptibility to different antibiotics in vitro and found that enterococcal isolates were resistant to benzylpenicillin, ampicillin, clindamycin, metronidazole, and tetracycline but sensitive to erythromycin and vancomycin.[110] Another investigation[202] used extracted teeth infected with *E. faecalis* and compared the efficacy of NaOCl and EDTA versus NaOCl and MTAD. They concluded that although the NaOCl/EDTA combination consistently disinfected the test specimens, almost half of the teeth rinsed with NaOCl/BioPure MTAD remained contaminated.[202] In a recent study,[371] doxycycline was replaced with CHX in one of the test groups of *E. faecalis*–infected teeth. Whereas no specimens that were cleaned with MTAD or MTAD + CHX showed the presence of residual bacteria, 70% of the samples rinsed with CHX as a substitute for doxycycline demonstrated growth of *E. faecalis*.

The citric acid component in MTAD effectively removed a smear layer.[44,372,421] Under these conditions, BioPure MTAD was more aggressive in eroding dentin than EDTA. In another investigation,[410] however, the addition of NaOCl was necessary to achieve dissolution of organic matter.

Ethylenediamine Tetra-Acetic Acid

EDTA came into use in endodontics in 1957[283]; chelators such as EDTA create a stable calcium complex with dentin mud, smear layers, or calcific deposits along the canal walls. This may help prevent apical blockage (Fig. 9-42) and aid disinfection by improving access of solutions through removal of the smear layer.

Neutral EDTA (17% concentration) showed a higher degree of decalcification of dentin surfaces than RC-Prep (a gel-type preparation of EDTA), although its effect was reduced in apical regions.[441] Similar to MTAD, RC-Prep did not erode the surface dentin layer.[421]

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.[191,485] Calcium binding results in the release of protons, and EDTA loses its efficiency in an

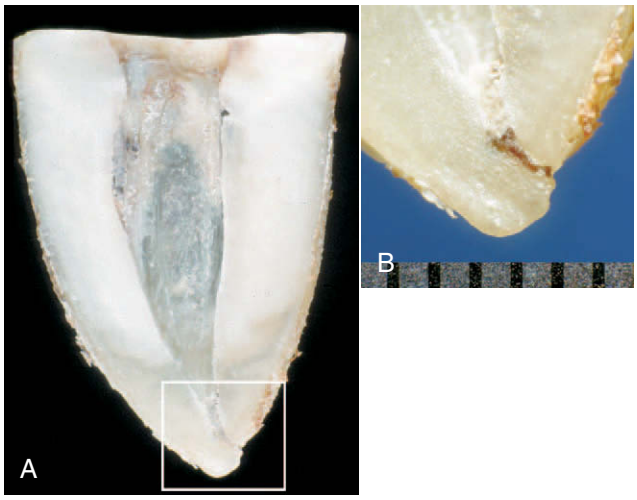


FIG. 9-42 Presence of dentin dust as a possible source of microbial irritation. Tooth #18 underwent root canal therapy. The clinician noted an apical blockage but was unable to bypass it. Unfortunately, intense pain persisted and at the patient's request, the tooth was extracted a week later. **A,** 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).

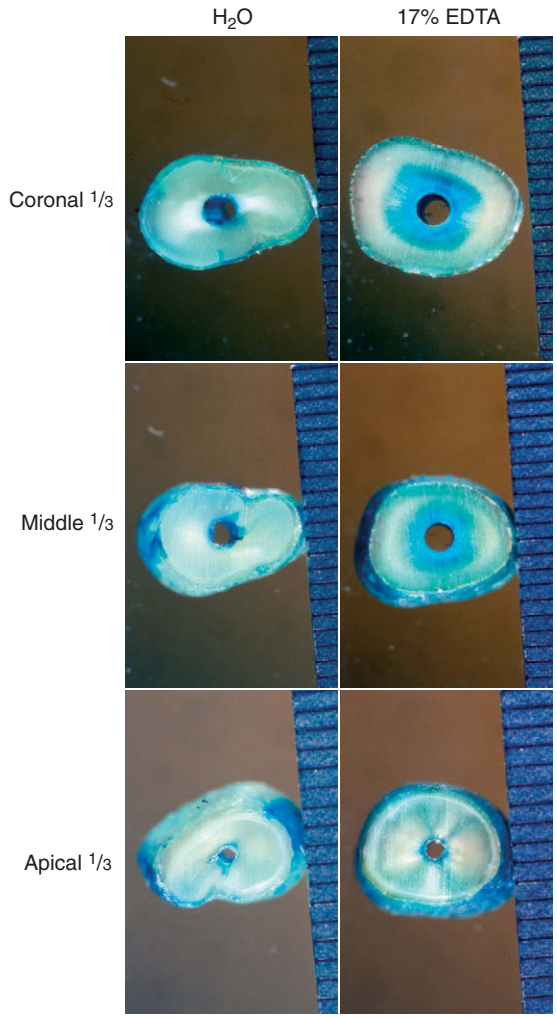


FIG. 9-43 Penetration of irrigants into dentinal tubules after root canal preparation with different dentin pretreatments. *Left column,* Irrigation with tap water and then with blue dye. *Right column,* Smear layer is removed with 17% EDTA, applied in high volume and with a 30-gauge needle, followed 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.

acidic environment. Thus the action of EDTA is thought to be self-limiting.[367] In one study, demineralization up to a depth of 50 µm into dentin was demonstrated for EDTA solutions[191]; however, other reports demonstrated significant erosion after irrigation with EDTA.[421]

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.[379] 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.[181]

Recent reports have indicated that several disinfecting agents such as $Ca(OH)_2$, IKI, and CHX are inhibited in the presence of dentin.[169,317,318] Moreover, chemical analyses indicated that chlorine, the active agent in NaOCl, is inactivated by EDTA.[161,483] In light of these facts, in addition to the unproven effect of lubricants containing EDTA on rotary-instrument torque, use of these solutions probably should be limited to hand instrumentation early in a procedure. Moreover, an EDTA solution preferably is used at the end of a procedure to remove the smear layer but does not prevent future bacterial penetration between root canal fillings and canal walls.[343,470,483] Clean, smear layer–free canal walls, tubulus openings, and entrances into lateral canals and isthmus areas, taken together with sufficient volume of NaOCl, ensures high disinfecting efficacy by enabling NaOCl to penetrate even into deeper dentin layers (Fig. 9-43).

BOX 9-4

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

Calcium Hydroxide

$Ca(OH)_2$ via its alkaline pH is generally very effective at eradicating intraradicular bacteria, with the exception of *E. faecalis*.[133] Increased effectiveness was observed when $Ca(OH)_2$ was mixed with some common irrigating solutions. Although additive effects could not be confirmed, and a reduction in the antimicrobial action of CHX was detected,[176] it appears that $Ca(OH)_2$ mixed with IKI or CHX may be able to kill $Ca(OH)_2$-resistant bacteria[385] (Box 9-4).

Unfortunately, it is not as effective when used short term and is not recommended as an irrigant but rather as an interap-

pointment dressing.[388] To achieve optimal antimicrobial activity, therefore, it requires prolonged exposure[36] or higher temperatures for use as an endodontic irrigant.[131]

Other Irrigants

Electrochemically activated water (also known as *oxidative potential water*) recently was tested as a potential irrigant.[166,250,391] Although this solution is active against bacteria[166] and removes the smear layer,[249] no assessment of its clinical potential is available, and in vitro research indicates that NaOCl is a superior disinfectant.[166]

Hydrogen peroxide traditionally has been used as an irrigant in conjunction with NaOCl; however, no additional benefit beyond NaOCl was registered.[181]

Some authors have advocated the use of 0.2% or 0.5% CHX in addition to NaOCl,[169,181] either as an irrigant or mixed with $Ca(OH)_2$ as an interappointment medicament. These combinations can overcome the inhibiting effect of dentin dust on conventional medicaments[169,318] and can optimize their antimicrobial properties against certain resistant bacteria and yeasts.[449,451]

Recently, one group[233] compared NaOCl with a final 17% EDTA rinse with an equal mixture of 2% NaOCl and 18% etidronic acid during and after instrumentation and a protocol involving 1% NaOCl during preparation and 2.25% peracetic acid after instrumentation. The results indicated that both acids had a similar effect on a smear layer as EDTA but with less demineralization of intratubular dentin. Strong demineralization has been shown to have a negative influence on canal sealability.[115]

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, 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.[478] The authors of this study cited the ability of a lubricant 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.[60,304]

In summary, irrigation is an indispensable step in root canal treatment to ensure disinfection. The tissue-dissolving and disinfecting properties of NaOCl currently make it the irrigant of choice. EDTA should be used at the end of a procedure to remove the smear layer, followed by another flush with NaOCl for maximum cleaning efficiency. This strategy also minimizes inactivation of NaOCl by chemical interactions.[161,481]

CLEANING AND SHAPING: CLINICAL ISSUES

Endodontists widely agree that a major biologic aim of endodontic therapy is to eliminate apical periodontitis by disinfection and sealing of root canal systems. However, considerable disagreement exists over the way this goal should be achieved (see Fig. 9-9). Although "cleaning and shaping" accurately describes the mechanical procedures,[337] it should be emphasized that "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.

Microorganisms in the pulp cavity and coronal root canal may be readily killed by irrigants early in a procedure, but bacteria in less accessible canal areas still can elicit apical periodontitis. These bacteria can be eradicated only after root canal preparation.

Biologic Objectives

Some have suggested that canals should be prepared to a uniform and continuous taper[361]; however, this mechanical objective likely addresses obturation rather than antimicrobial efficacy. The preparation shape and antimicrobial efficacy are intimately related through the removal of infected dentin and the delivery of irrigants.[185]

Traditionally, fluids have been delivered to root canals passively by syringe and needle (Fig. 9-44). Active systems such as the NIT are still in an experimental phase.[245] When delivered passively, irrigants have been shown to progress only 1 mm farther than the tip of the needle.[323] Enlarged apical canals are likely to allow increasingly deeper needle placement (see Fig. 9-44), and this improves débridement and disinfection of canals.[7,17,135,434] Nevertheless, thorough cleaning of the most apical part of any preparation remains difficult,[466] especially in narrow and curved canals.[180,295,329]

Mechanical Objectives

An important mechanical objective of root canal instrumentation is full incorporation of the original canals into the prepared shape, meaning that all root canal surfaces are mechanically prepared (*green areas* in Fig. 9-45, *A* and *B*); however, this goal is unlikely to be met with current techniques.[291,310]

Preparation errors such as zips and perforations should be absent. Although these and other procedural problems (Fig. 9-46) per se may not affect the probability of a favorable outcome,[229] they may leave parts of the root canal system inaccessible for disinfection.

Another important mechanical objective is to leave as much radicular dentin as possible so as not to weaken the root structure, thereby preventing vertical fractures. Although no definitive minimal radicular thickness has been established, 0.3 mm is considered critical.[228] Even pretreatment, wall thickness of 1 mm and below has been demonstrated.[147] Straightening of canal paths can lead to minimal remaining wall thicknesses (Fig. 9-47); this underlines the need for adequate access cavity preparation and optimal enlargement of the coronal third of the root canal.

Two primary mechanical elements are the apical width and the endpoint 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. This point is believed to coincide with the cementodentinal junction (CDJ) (see Chapter 7). This definition of working length is based on histologic sections and ground specimens. However, the

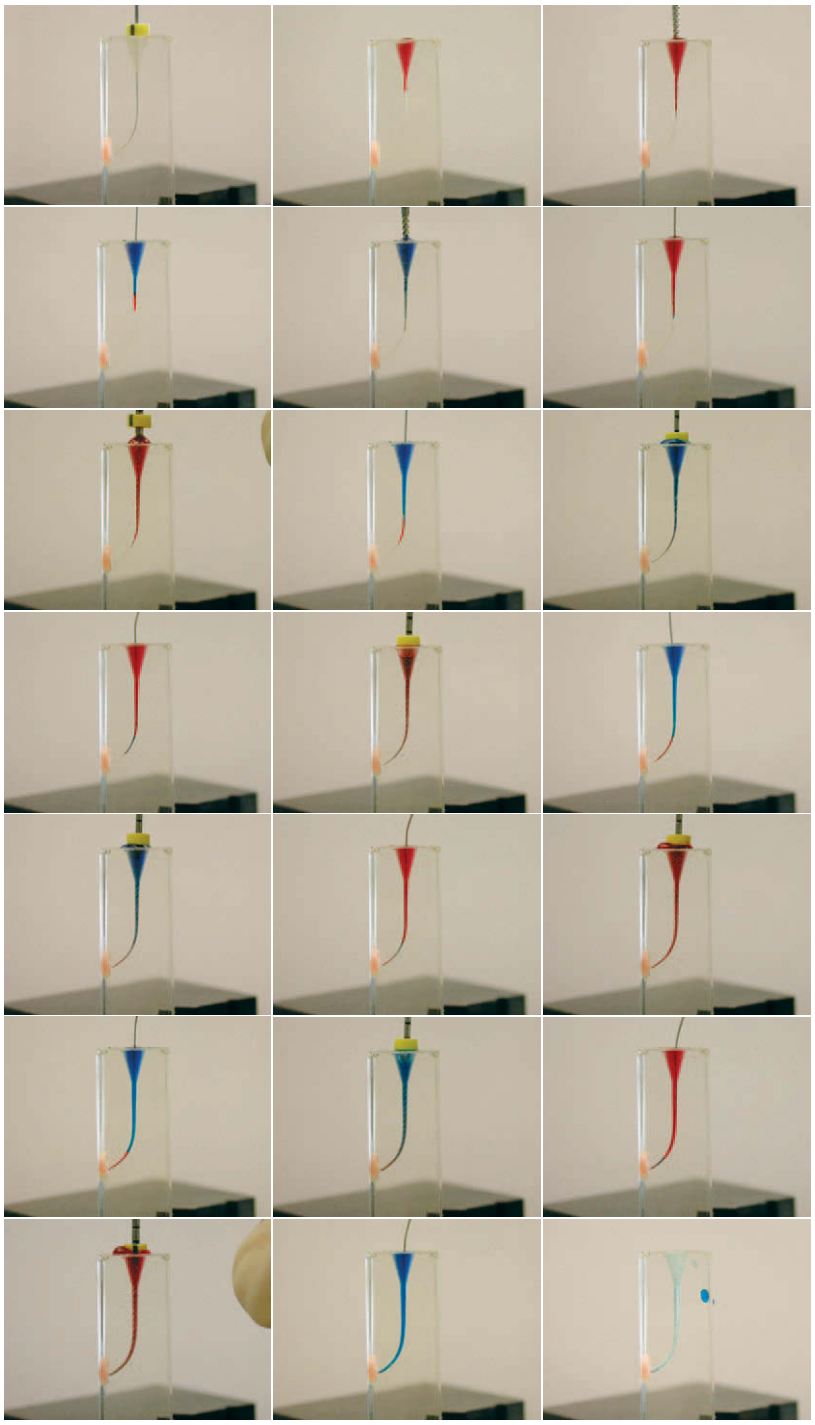


FIG. 9-44 Irrigation and movement of irrigants depends on canal shape. Sequential enlargement of a canal in clear plastic block was performed with a sequence of ProFile instruments in accordance with the manufacturer's recommendations. Alternating irrigation with blue and red fluid was done after each 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.

position and anatomy of the CDJ varies 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 0.5 to 1 mm short of the radiographic apex in necrotic cases and 1 to 2 mm short[177,332,468] in cases involving irreversible pulpitis. In this way, preparation would take place inside the root canal. Although there is no definitive validation for this strategy at present,[352] well-controlled follow-up studies seem to support it.[387,389]

However, working to shorter lengths could lead to the accumulation and retention of debris, which may result in apical blockage (see Fig. 9-42). Such blockage (which consists of collagen fibers, dentin mud, and most importantly, residual bacteria) inside apical canal areas is a major cause of persistent or recurrent apical periodontitis,[172,276,378] currently termed *post-treatment disease*[138,462] (also see Chapter 24). Additionally, because of the creation of apical blockage, working to short lengths may contribute to procedural errors such as apical perforations and fractured instruments.

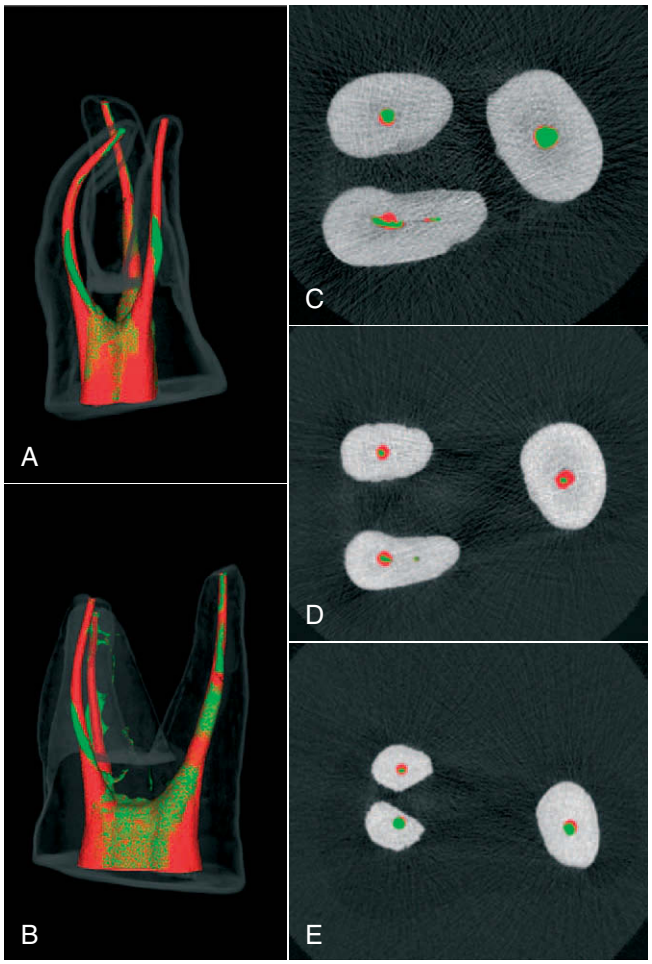


FIG. 9-45 Example of a desired shape, with the original root canal fully incorporated into the prepared outline. **A-B,** Micro–computed tomography reconstructions in clinical and mesiodistal views of a maxillary molar prepared with a NiTi rotary system. The *green area* indicates the pretreatment shape, 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 *36:740–747, 2003.)*

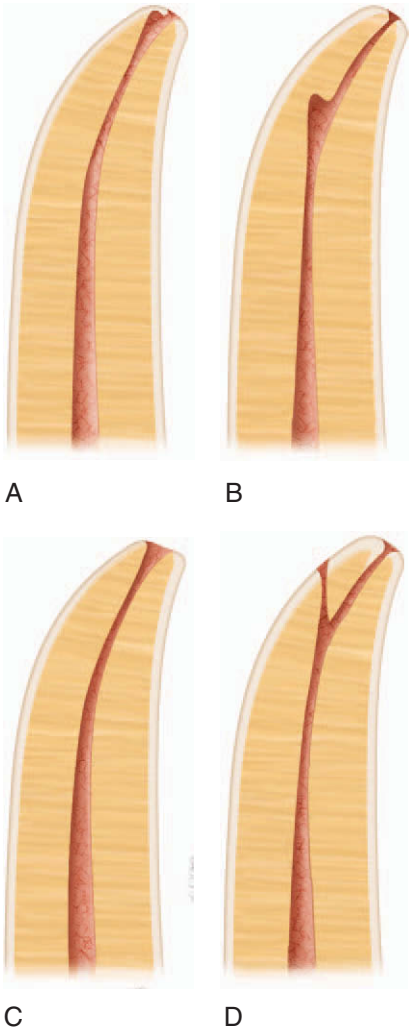


FIG. 9-46 Schematic diagrams showing the most common preparation errors. **A,** Apical zip. **B,** Ledge. **C,** Apical zip with perforation. **D,** Ledge with perforation.

The electronic apex locator has helped clinicians identify the position of apical foramina more accurately; the development of this instrument made it possible to work more precisely and routinely as close as 0.5 mm to the canal terminus (see Chapter 8).

Concepts and Strategies

Two factors are closely related to the preparation length: use of a patency file and the apical width. A *patency file* is a small K-file (usually a size #10 or #15) that is passively extended just through the apical foramen.

Use of a patency file has been suggested for most rotary techniques. This step is believed to remove accumulated debris and help maintain working length. However, the issue is controversial, and many U.S. dental schools did not teach this concept, at least not until recently.[82] Moreover, investigators[152] demonstrated that the use of patency files of varying sizes did not prevent preparation errors.

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.[198] Maintaining patency throughout an endodontic procedure does not lead to an increase in posttreatment symptoms.[25] No definitive evidence exists either favoring or disproving the use of a patency file, but clinical experience suggests that this technique involves relatively little risk and provides some benefit as long as small files are used carefully.

Like the position of the apical constriction, apical diameters are difficult to assess clinically.[220] 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.[458] This is a crucial point because the initial canal size is a major determinant for the desired final apical diameter.

TABLE 9-2

Characteristics of Wide and Narrow Apical Preparations

Root Canal Preparation	Benefits	Drawbacks
Narrow Apex	Minimal risk of canal transportation and extrusion of irrigants or filling material Can be combined with tapered preparation to counteract some drawbacks	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 Apex	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

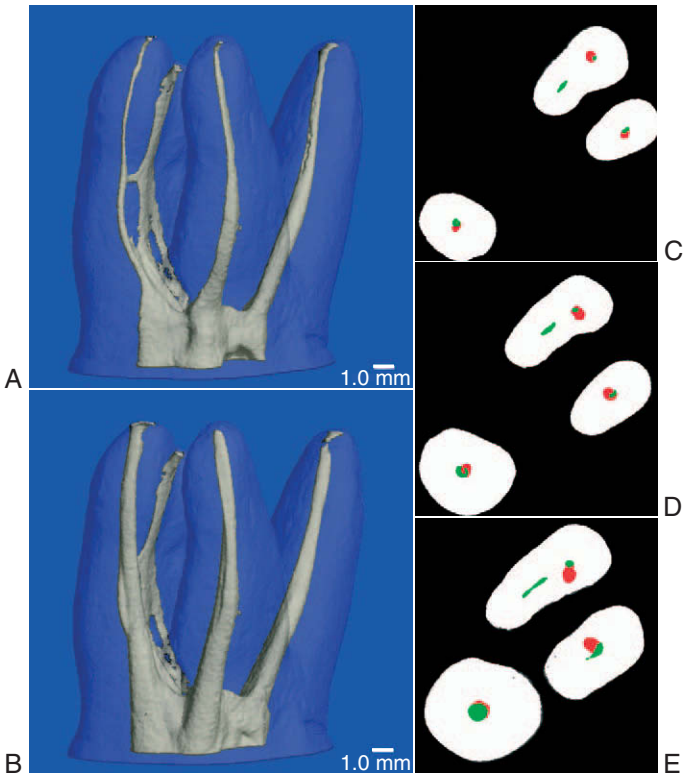


FIG. 9-47 Example of excessive thinning of dental structure during root canal treatment. **A-B,** Micro–computed tomography reconstructions show pretreatment and posttreatment root canal geometry of a maxillary molar. **C-E,** Cross sections of the coronal, middle, and apical thirds with pretreatment canal cross sections. Note the transportation and thinning, in particular, in the main mesiobuccal canal.

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. Both sides stress the importance of maintaining the original path of the canal during preparation; otherwise, bacteria infecting the apical third of the root canal may not be reached by a sufficient bactericidal concentration of an antimicrobial agent.[271] Investigators obtained a higher percentage of 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) can occur with wide preparations when either stainless steel or NiTi instruments are used (see Fig. 9-46).

Thorough disinfection of the apical part of a root canal is essential, because this area is likely to contain intraradicular bacteria.[277] Wider apical preparations remove potentially infected dentin, allowing the delivering needle and subsequently the antimicrobial irrigant to penetrate the root canal more deeply.[97,135]

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.[434] 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.[473] Another study compared step-down sequences with additional apical enlargement to ISO size #35 or a serial step-back technique with no apical enlargement. NaOCl and EDTA were used as irrigants. In that study, no significant difference was detected in colony-forming units with or without apical enlargement.[102] 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 (Table 9-2), the distillation of all 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. 9-45). This way, routine root canal treatment results in favorable outcomes at various levels of clinicians' expertise (Fig. 9-48).

CANAL PREPARATION TECHNIQUES

The traditional cleaning and shaping strategy (the step-back technique) focused on the initial preparation of the apical third of the root canal system, followed by various flaring techniques to facilitate obturation.[158,354,420] 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

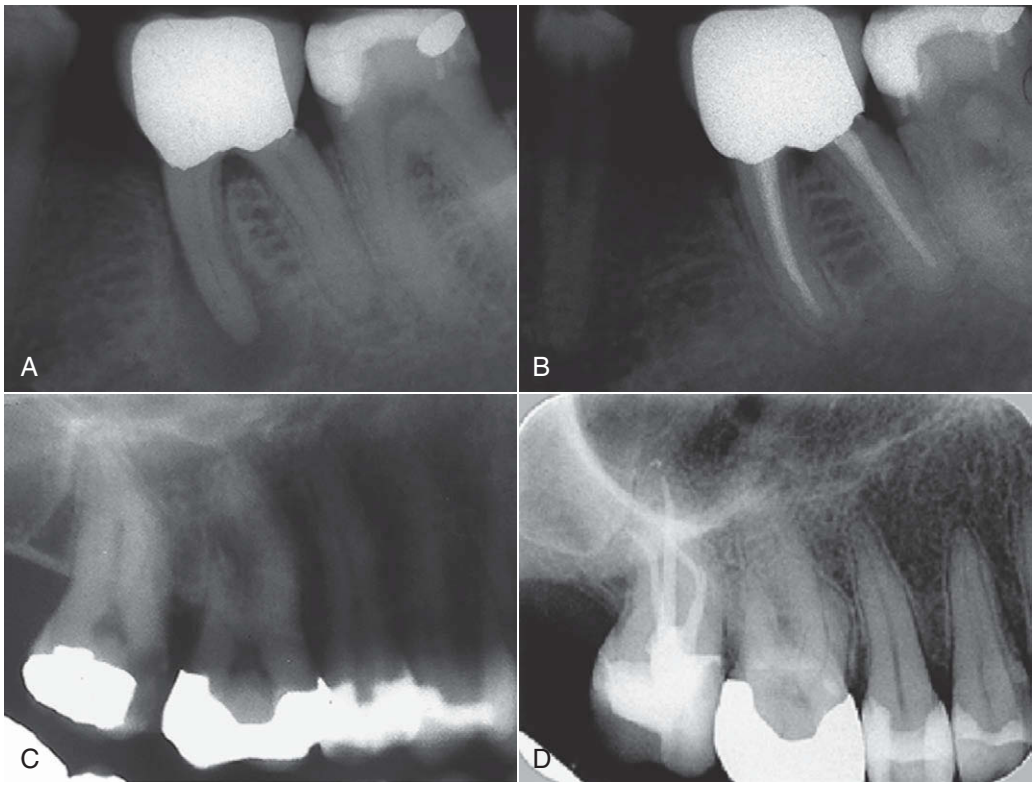


FIG. 9-48 Routine cases treated according to the principles discussed in this chapter. Biologic and mechanical aims were maintained at varying levels of expertise. **A,** Asymptomatic tooth #18 with periradicular lesion; endodontic treatment was indicated. **B,** Recall radiograph after 18 months (treatment was performed by fourth-year student at Zurich Dental School). **C,** Pretreatment radiograph of tooth #2, which was diagnosed with irreversible pulpitis. **D,** Recall radiograph 2 years later shows sound periradicular tissue; the tooth is clinically symptom free (treatment was performed by an endodontist).

infrequently full length may not be reached because of blockage or coronal binding.

Coronal binding is caused by overhangs at the orifice level and also occurs when the canal is less tapered than an instrument, thus binding somewhere coronally. Moreover, a straight root often has a curved canal; buccal and lingual curvatures that cannot be seen on radiographs also need to be detected.[103,312] Passing a precurved negotiating file through a coronally tight canal will straighten the instrument.[395] Nonflared canals do not allow efficient irrigation, which further predisposes to blockage.

Various instrumentation sequences have been developed for hand and rotary instruments; these are discussed later in the chapter. However, the shape of the access cavity is *the* prerequisite that must be optimized before any canal preparation can take place (see Chapter 7).

One approach to the preparation of an adequate access cavity (Fig. 9-49) involves 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 (Fig. 9-50, *A* and *B*) and other teeth. Preexisting restorations allow for ideal access cavities that serve as reservoirs of irrigants (see Fig. 9-50, *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 then is used to progress deeper into the canal; the third instrument follows, 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. Recently, apical enlargement has been broken down into three phases: preenlargement, apical enlargement, and apical finishing.[448]

Most rotary techniques require a crown-down approach to minimize torsional loads[57] and reduce the risk of instrument fracture. Used sequentially, the crown-down technique can help to enlarge canals further. All basic techniques described

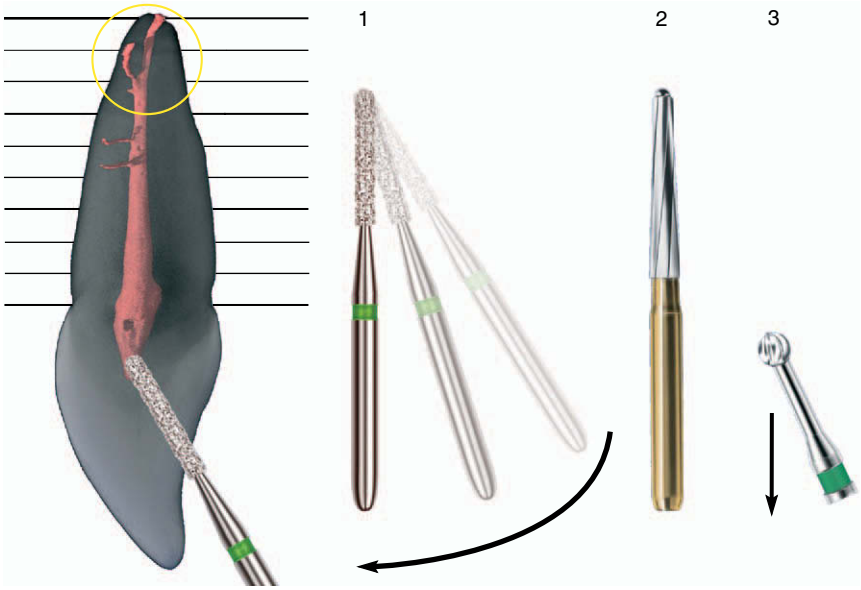


FIG. 9-49 Sequence of instruments used for optimal preparation of an access cavity (e.g., in an incisor). A parallel-sided diamond or steel bur is used to remove overlying enamel in a 90-degree angle toward the enamel surface *(1)*. The bur is then tilted vertically to allow straight-line access to the 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)*.

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. 9-47). Vertical root fractures and perforations are possible outcomes of excessive removal of radicular dentin in zones that have been termed *danger zones*.[20] Overenthusiastic filing, for example, may lead to more procedural errors (see Fig. 9-20). 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. 9-45).

Preenlargement of the coronal half to two thirds to allow files unimpeded access gives the clinician better tactile control in directing small, adequately precurved negotiating files into the delicate apical third (Fig. 9-51). Gates-Glidden drills can be used sequentially to enlarge the coronal third of the canal in teeth with straight roots (Fig. 9-52). Both step-back and step-down sequences have been recommended.

Besides GG burs, various 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 to and safer for more difficult cases (Fig. 9-53).

Once the coronal portions of a canal have been enlarged, the apical canal areas can be more efficiently prepared. Better clinical results are obtained with optimized access, regardless of the preparation technique used (Fig. 9-54). Only after preenlargement can fine scouting files (also used before rotary files in the coronal areas) provide information about the root canal paths. Preenlarged canals may accommodate hand files, which can be used to gather specific information about the apical third's cross-sectional diameter and anatomy.

Hand Instrumentation

Standardized Technique

The standardized technique adopts the same working length definition for all instruments introduced into a root canal and 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-used instrument. 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,[19] 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[18] (see Chapter 11).

Step-Back Technique

Realizing the importance of a shape larger than that produced with the standardized approach, one investigator[457] suggested the step-back technique, 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. 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.[273]

Although the step-back technique was primarily designed to avoid preparation errors in curved canals, it applies to the preparation of apparently[103,356] straight canals as well. Several modifications of the step-back technique have been described over the years. Another investigator[420] advocated the insertion of progressively larger hand instruments as deep as they would

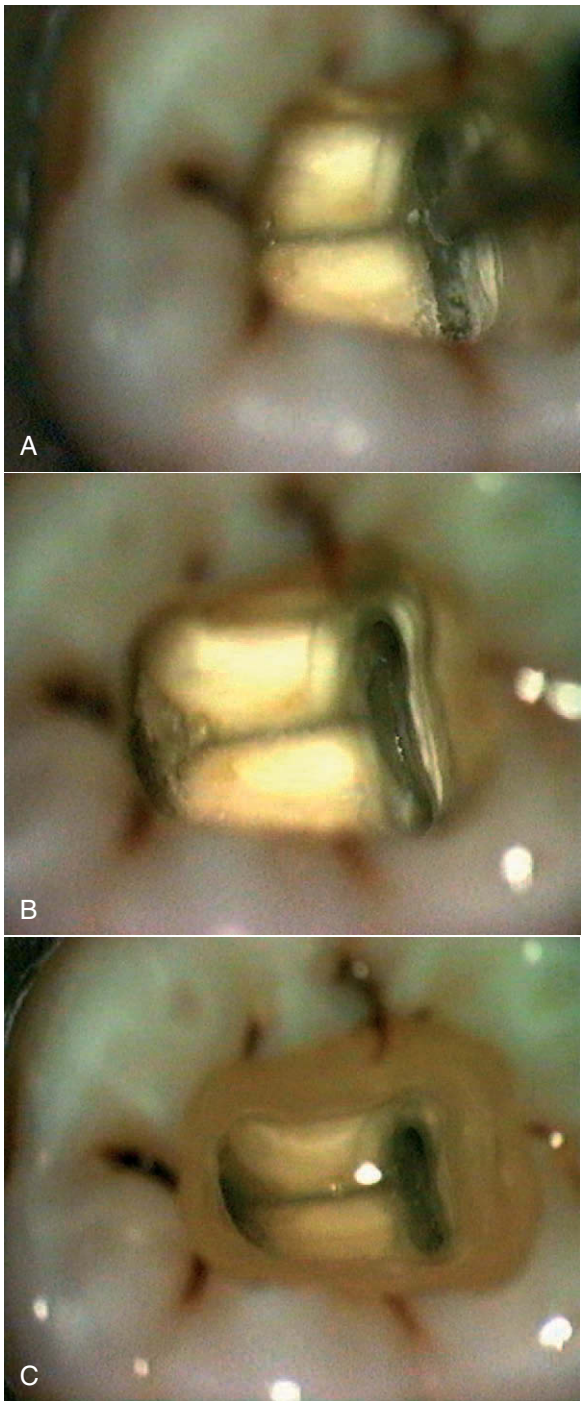


FIG. 9-50 Clinical views of an access cavity in a mandibular molar as seen through an operating microscope (×20). **A,** Modification with an ultrasonically activated tip. **B,** Access cavity after modification. **C,** Cavity is flooded with 1% sodium hypochlorite.

passively go in order to explore and provide some enlargement prior to reaching the working length.

Step-Down Technique

Other investigators[150] described a different approach. They advocated shaping the coronal aspect of a root canal first before apical instrumentation commenced. This technique is intended

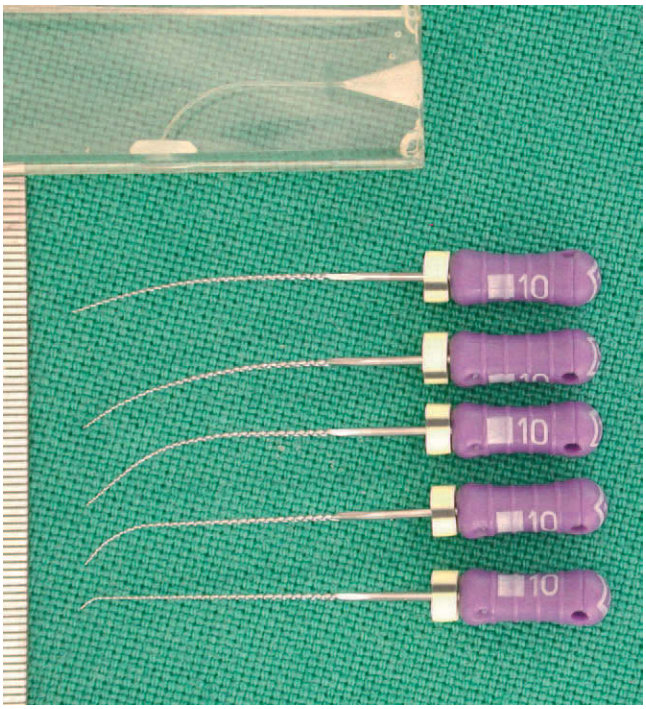


FIG. 9-51 Various precurved, stainless steel hand files for pathfinding and gauging. Compare the curves in the instruments to the ones in a plastic training block (gradation of ruler is 0.5 mm).

to minimize or eliminate the amount of necrotic debris that could be extruded through the apical foramen during instrumentation[134]; 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.[225]

Crown-Down Technique

Numerous modifications of the original step-down technique have been introduced, including the description of the crown-down technique.[136,253,348] The more typical step-down technique includes the use of a stainless steel K-file exploring the apical constriction and establishing working length. In 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[136] 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

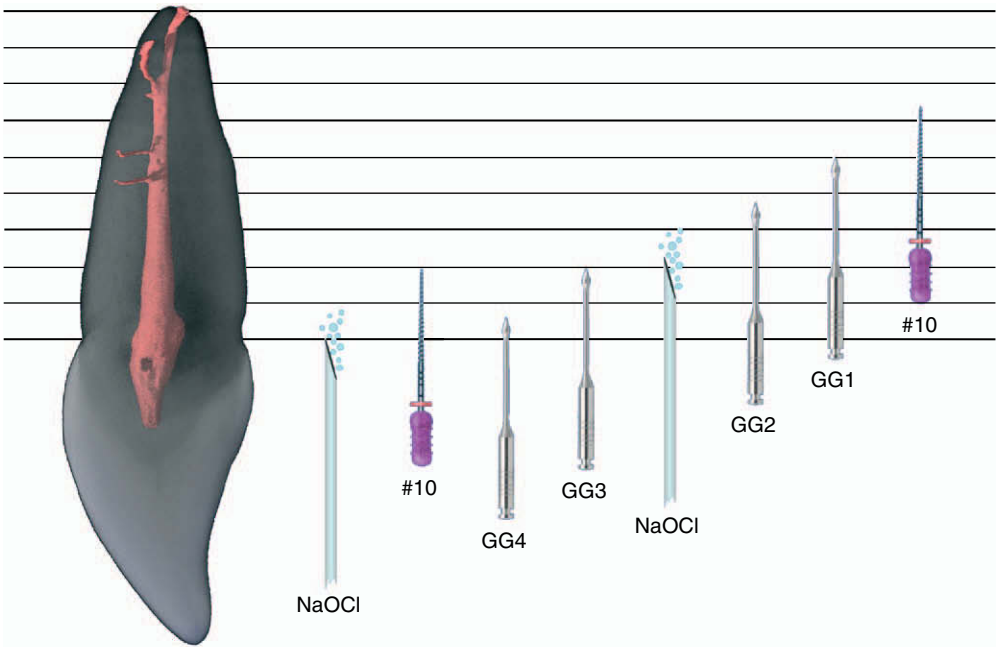


FIG. 9-52 Diagram of coronal enlargement in a maxillary anterior tooth. After preparation of the access cavity (see Figure 9-49) and copious irrigation, Gates-Glidden burs are used in a step-down manner to enlarge the orifice and provide straight-line access into the middle third of the canal. Precurved size #10 K-files are used to explore the canal path and dimension.

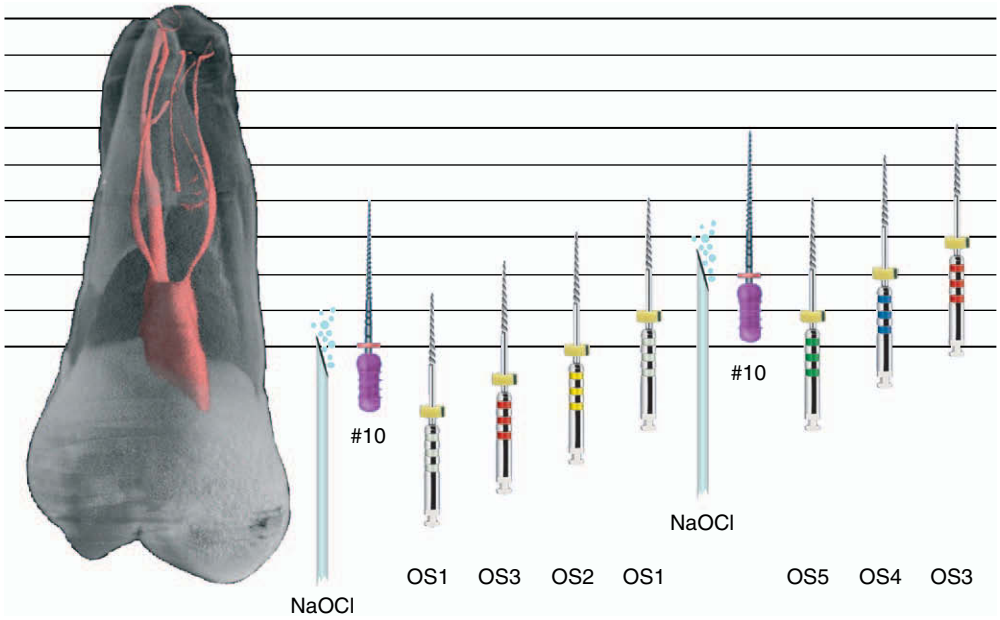


FIG. 9-53 Diagram of coronal enlargement in a more complicated maxillary posterior tooth. This maxillary molar presents several difficulties, including a narrow mesiobuccal canal that exits the pulp cavity at an angle. A possible approach in a case involving difficult entry into the root canal system 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.

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[347,348] 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. A double-flare technique was also suggested for ProFile rotary instruments.[363]

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. Researchers described this technique as a series of rotational movements for Flex-R files, but it can also be used for K-files and other hand instruments such as GT hand files. Many different explanations have been offered for the obvious and undisputed efficacy of the balanced force approach,[91,221,336] but general agreement exists that it provides excellent canal-centering ability, superior to other techniques with hand instruments.[29,69,226]

The balanced force technique involves three or four steps.[334] The first step (after passive insertion of an instrument into the canal) is a passive clockwise rotation of about 90 degrees to engage dentin (Fig. 9-55). In the second step, the instrument is held in the canal with adequate axial force and rotated

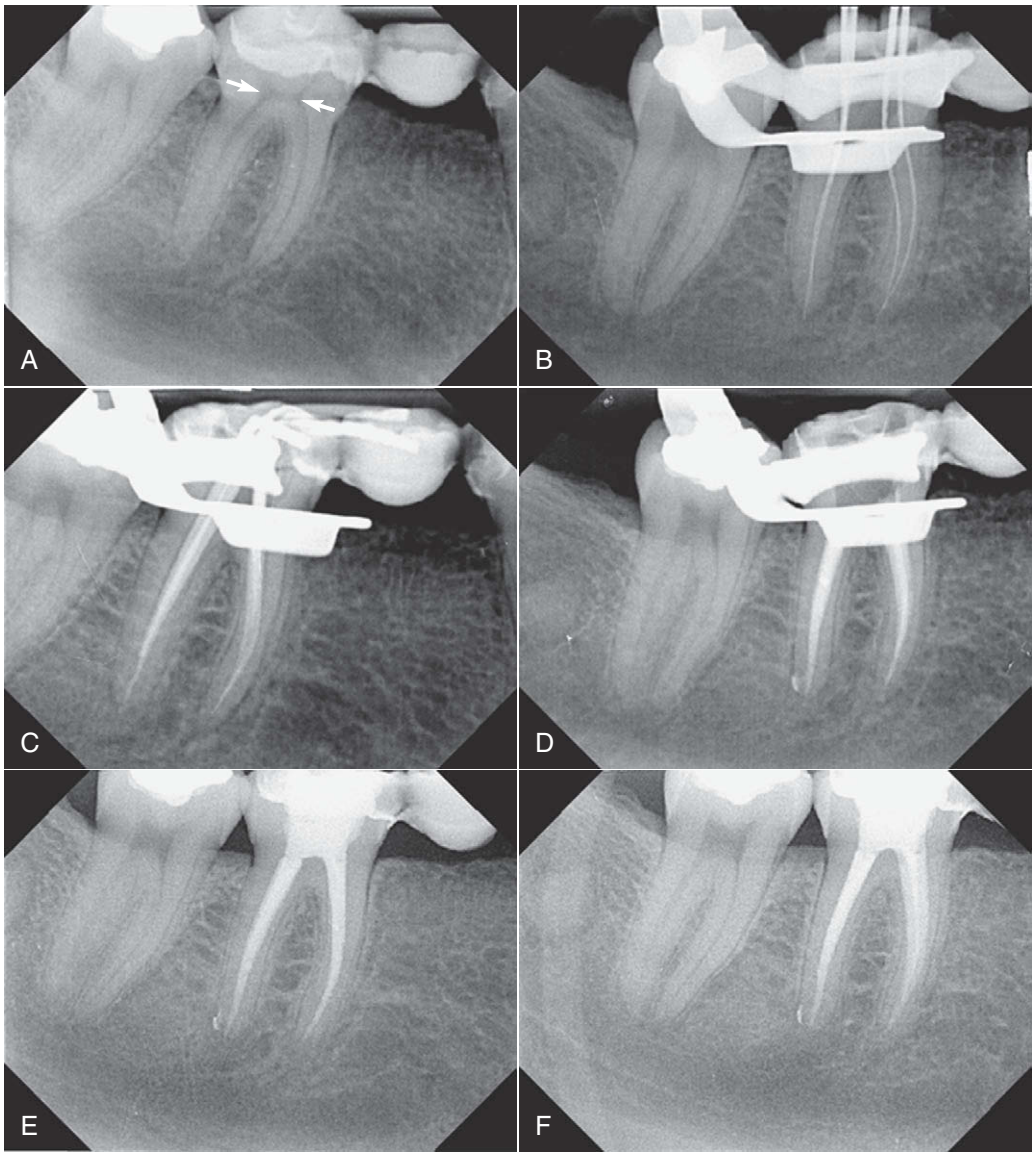


FIG. 9-54 Clinical example of the importance of straight-line access to the middle third of the root canal. **A,** Pretreatment radiograph of tooth #30, diagnosed with irreversible pulpitis. This tooth serves as a retainer for a metal-free, fixed partial denture. Note the prominent dentin shelves (*arrows*). **B,** Working length radiograph with hand instruments inserted into the mesial and distal canals. **C,** Cone-fit radiograph showing tapered preparations after removal of the dentin shelves. **D,** Posttreatment radiograph after thermoplastic compaction of gutta-percha. **E-F,** Follow-up radiographs at 2 and 4 years. The tooth is clinically symptom free, and the periodontal ligament appears to be within normal limits.

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 (Fig. 9-56). 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 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 enlargement to the desired size begins (Fig. 9-57). As stated previously, various apical preparation designs exist, and the choice is driven mostly by the desired obturation technique, whether an apical stop or an apical taper is prepared. Finally, canal taper is increased by decreasing the working length of larger instruments in 1-mm or 0.5-mm increments, producing 0.05 and 0.10 mm/mm tapers, respectively.

Copious irrigation and frequent recapitulations with a smaller file to working length may be required, and in some

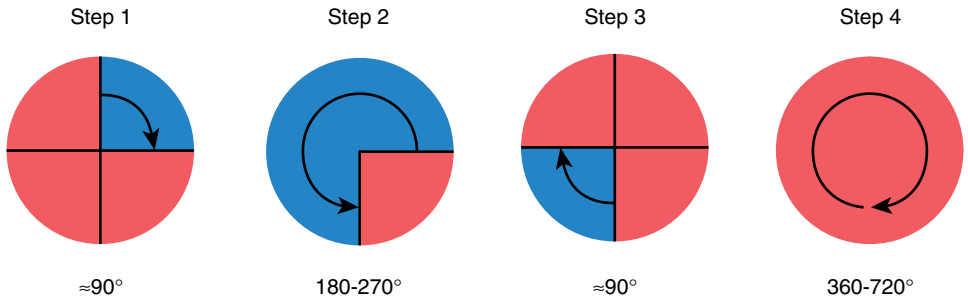


FIG. 9-55 Diagram of handle movements during balanced force hand preparation.
STEP 1: After pressureless insertion of a Flex-R or NiTiFlex K-file, the instrument is rotated clockwise 90 degrees, using only light apical pressure.
STEP 2: The instrument is rotated counterclockwise 180 to 270 degrees; sufficient 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.

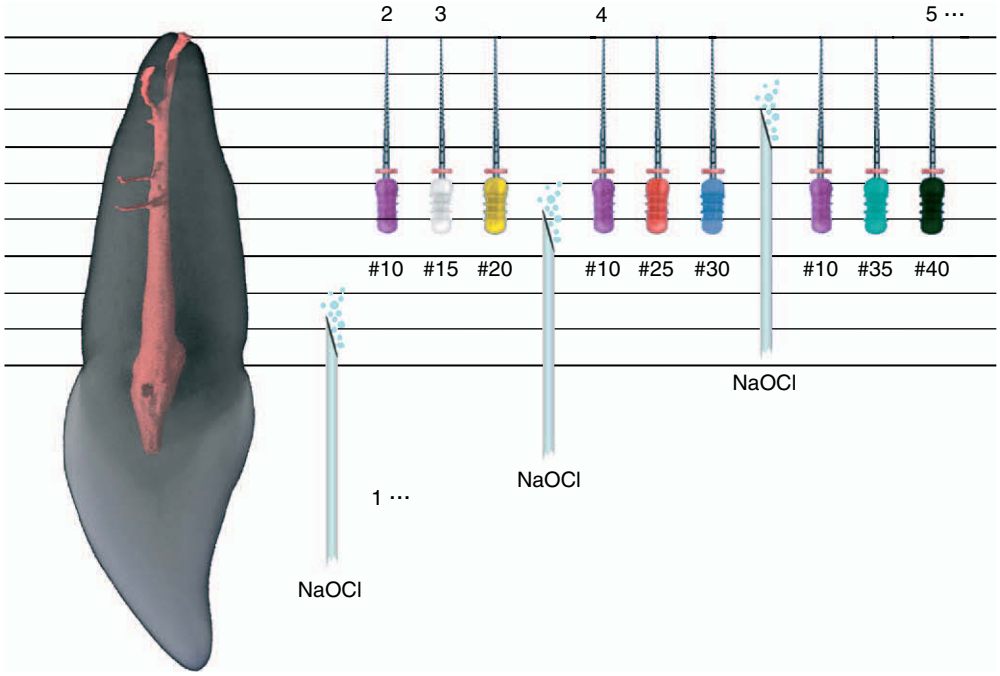


FIG. 9-56 Root canal instrumentation with hand files: Part I. After the orifice has been accessed (see Figs. 9-49 and 9-54) and copious irrigation performed *(1)*, the working length (WL) is determined. A size #10 and/or #15 K-file is advanced to the desired apical preparation endpoint, aided by an electronic apex locator *(2)*. The apical canal areas are then enlarged with K-files *(3)* used in the balanced force technique (see Figure 9-55). 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).

instances, clinicians must devise creative strategies using small crown-down and/or step-back sequences.

In many cases, hand instrumentation produces adequate shapes, but clinicians often choose NiTi rotary instruments either to enlarge curved canals or to produce wider tapers. Fig. 9-58 illustrates the development of these shapes in the mesial root canals of a mandibular molar, clearly showing that substantial areas of the root canal surface are not instrumented, even when apical size #50 or 0.09 taper are reached (see *red areas* in Fig. 9-58, *G* and *I*).

LightSpeed Instruments

Since the introduction of LightSpeed instruments, the manufacturer's guidelines have changed. This section presents a version used for the LightSpeed LS1[32] (Fig. 9-59).

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 while registering tactile feedback. The first instrument that experiences resistance 4 mm short of working length is the

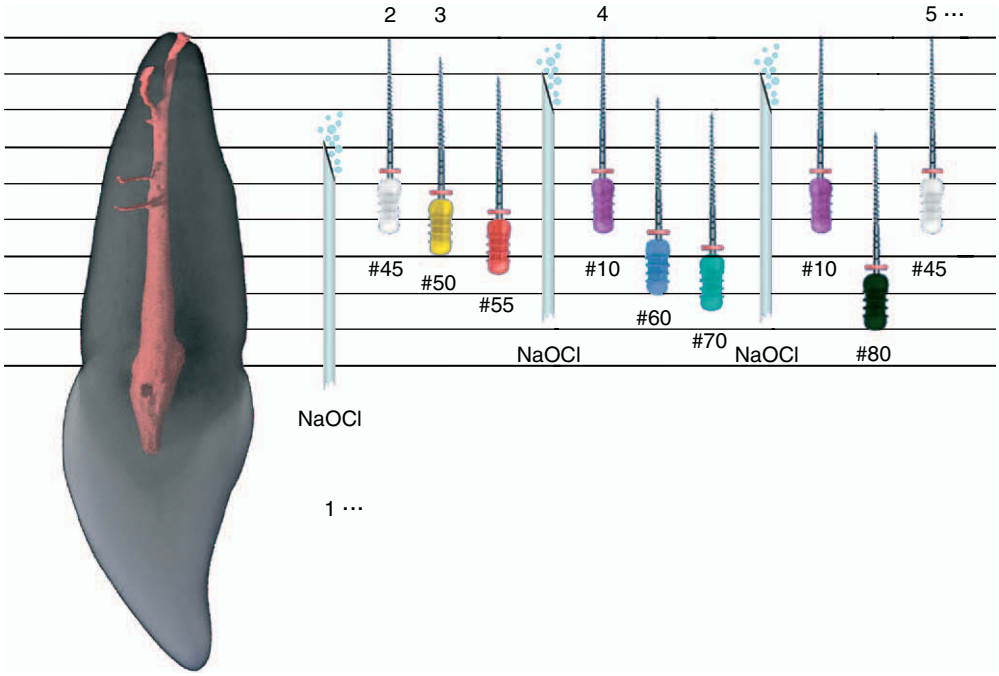


FIG. 9-57 Root canal instrumentation with hand files: Part II. Frequent irrigation with sodium hypochlorite *(1)* is more efficient after the working length (WL) is reached, because irrigation needles may penetrate deeper into the canal. Canal taper is increased to further improve antimicrobial efficiency 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 to 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.

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 (Discus Dental). Midroot shaping is then accomplished with sequentially larger LSX instruments. Finally, the 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.[120]

ProFile

Many different techniques have been advocated for the ProFile,[363] but the general pattern remains a crown-down approach with varying tapers and tip diameters. The ProFile instrument therefore can be used as an example for systems with this basic design (e.g., the HERO 642, K3, and FlexMaster). It must be noted that the manufacturers' instructions for those 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. That said, it also must be noted that the merits of specific instructions have not been scientifically elaborated.

As with other instruments, coronal preenlargement is suggested (see Figs. 9-53 and 9-54). The working length then is determined as described previously, 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] (Fig. 9-60). In more difficult small canals, #.06 tapers are followed by #.04-tapered instruments, also with descending tip diameters (Fig. 9-61). Apical preparation is performed either with multiple shaping waves, as suggested for GT rotary files,[74] or in a step-back manner.[361] Because of their superior resistance to cyclic fatigue, #.02-tapered ProFile instruments are useful for abrupt apical curves. Preparation is complete once a continuous #.06 taper with an adequate apical size is achieved. Recapitulation during the preparation with a small hand file is recommended.

ProTaper

The approach for ProTaper instruments differs from that for most other NiTi rotary files (except the MTwo instrument marketed by VDW in Europe) in that no traditional crown-down procedure is performed (Fig. 9-62).

Size #10 and #15 hand files are precurved to match the canal curvature and then passively inserted into the coronal two thirds of a root canal as pathfinding files, which confirm the presence of a smooth, reproducible glide path. This step is essential for ProTaper shaping instruments, because they are mostly side-cutting 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 pathfinding #10 or #15 file is reached.

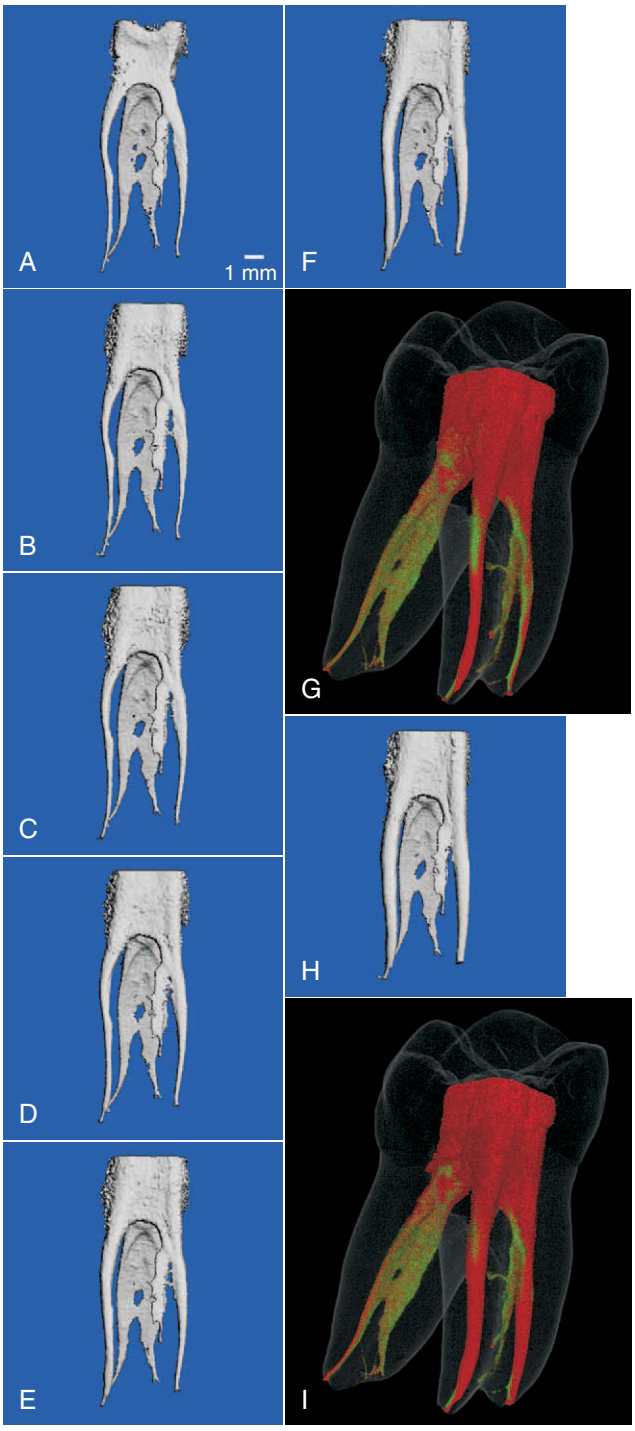


FIG. 9-58 Stepwise enlargement of mesial root canal systems in an extracted mandibular molar demonstrated with micro–computed tomography (μCT) reconstructions. The buccal canal *(left)* was prepared with a LightSpeed (LS) instrument, and the lingual canal *(right)* was shaped with a ProTaper (PT) instrument. **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.

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. 9-62). 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 (Fig. 9-63) 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 and/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. 9-62).

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. Fig. 9-64 presents two cases that involved different problems and therefore were approached differently. Mesiobuccal roots of the maxillary molar can show substantial curvature; rotary instrumentation and/or hybrid techniques allow preservation of the curvature (see Fig. 9-64, *A*) and optimal enlargement (see Fig. 9-64, *B*). Often hand instruments other than ISO-normed files (see Fig. 9-63) are used in these cases to ensure a smooth, tapered shape or to eliminate ledges.

Hybrid Techniques

For some time, combining various NiTi preparation systems have been suggested[89,448] to address certain shortcomings of current instruments (Box 9-5). 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,[408,456,464,469] and apical foramina may in fact be oval in most cases.[68] Naturally, a rotating file can produce a round canal at best, so a strategy must be devised for adequately shaping oval canals without overly weakening radicular structure (compare Figs. 9-45 and 9-47). One hybrid approach completely prepared 95% or more of all such canals and resulted in extremely wide apical sizes that may be difficult to achieve with most instrument systems.[210-212]

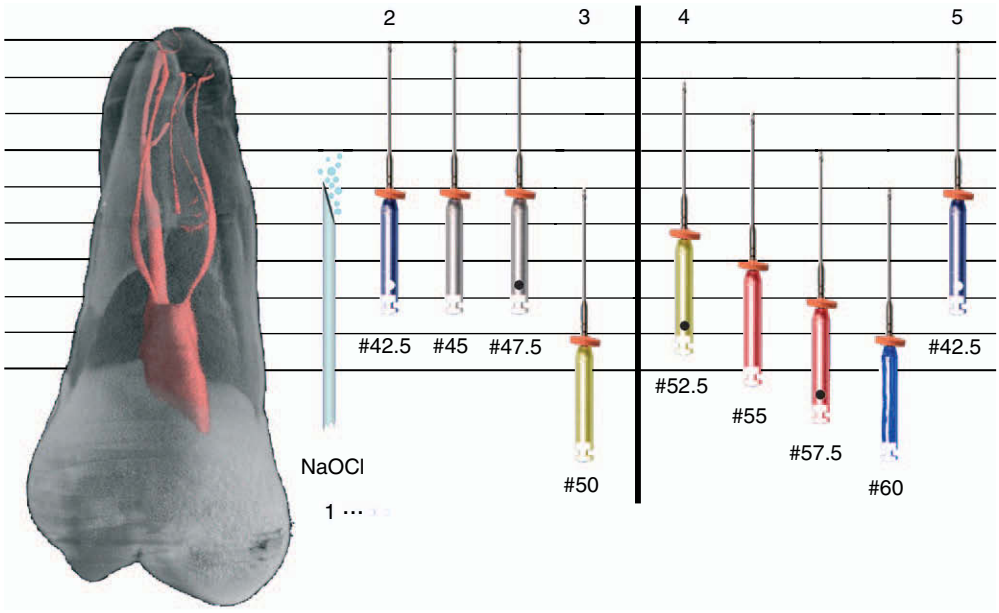


FIG. 9-59 Finishing of LightSpeed preparations to allow obturation. With the canal system flooded *(1)*, apical preparation *(2)* is continued until an LS instrument requires 12 pecks to reach the working length (WL). The next LS instrument *(3)* then is used to a point 4 mm short of the WL to prepare 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)*.

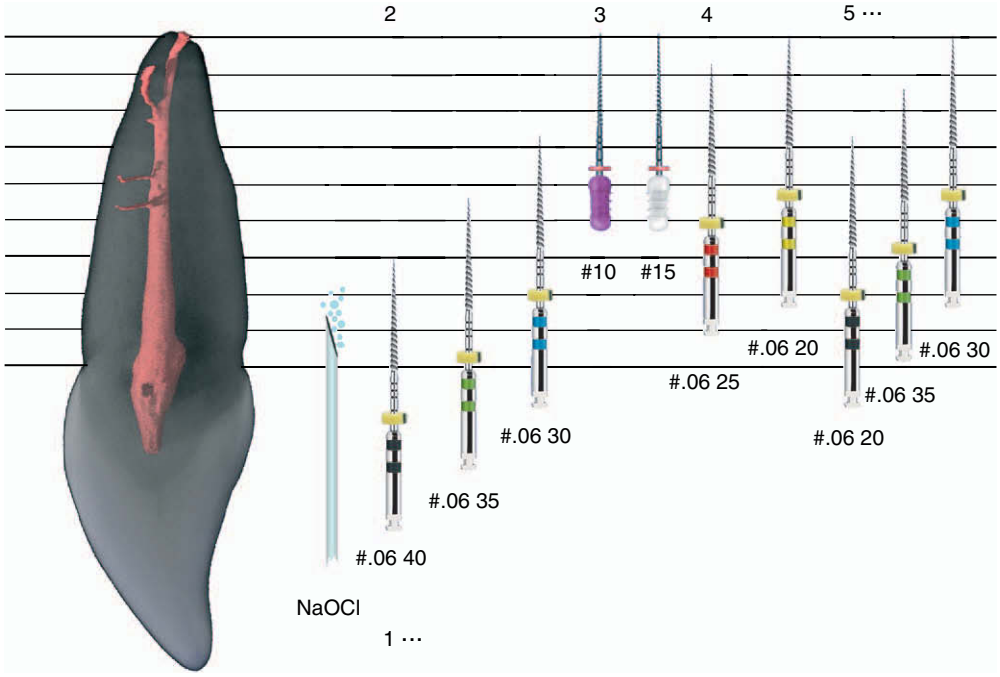


FIG. 9-60 ProFile instrumentation in a wider canal. In irrigated and flooded canals *(1)*, a crown-down preparation is done with a sequence of #.06-tapered ProFile instruments *(2)*. When the apical third is reached, the WL is determined and a glide path is secured *(3)*. Apical preparation is then completed by continuing the crown-down sequence *(4)* up to the desired apical width at the WL. Several shaping waves may be required *(5)*.

Histologic slides (see Fig. 9-41) and µCT reconstructions (see Figs. 9-45, 9-47, and 9-58) 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. 9-2, 9-5, 9-15, and 9-63). The technique involves the use of a variety of instruments: GG drills and K-files for establishing straight-line access; ProTaper instruments for body shaping and apical preenlargement; NiTi K-files or LightSpeed instruments

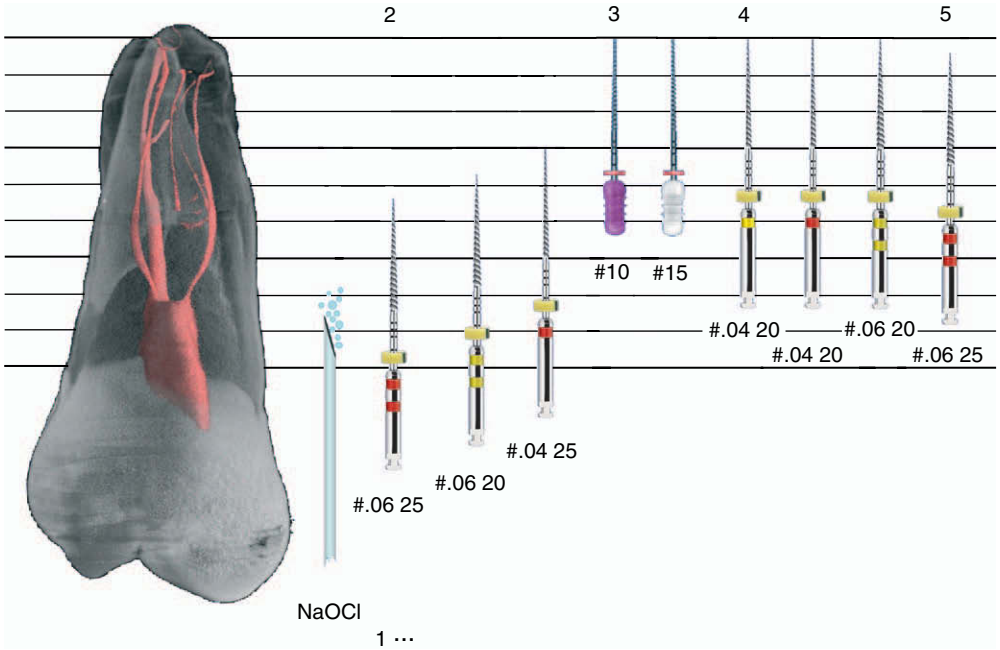


FIG. 9-61 Sequence of ProFile instruments used in constricted canals. After irrigation *(1)* and coronal preenlargement with orifice shapers (see Figure 9-51), ProFile instruments size #25, #.06 taper; size #20, #.06 taper; and size #24, #.04 taper are used as crown-down instruments *(2)*. After the WL has been determined and a glide path secured *(3)*, apical preparation to the desired size begins *(4)*. For additional taper, larger instruments may be used to a point short of the WL.

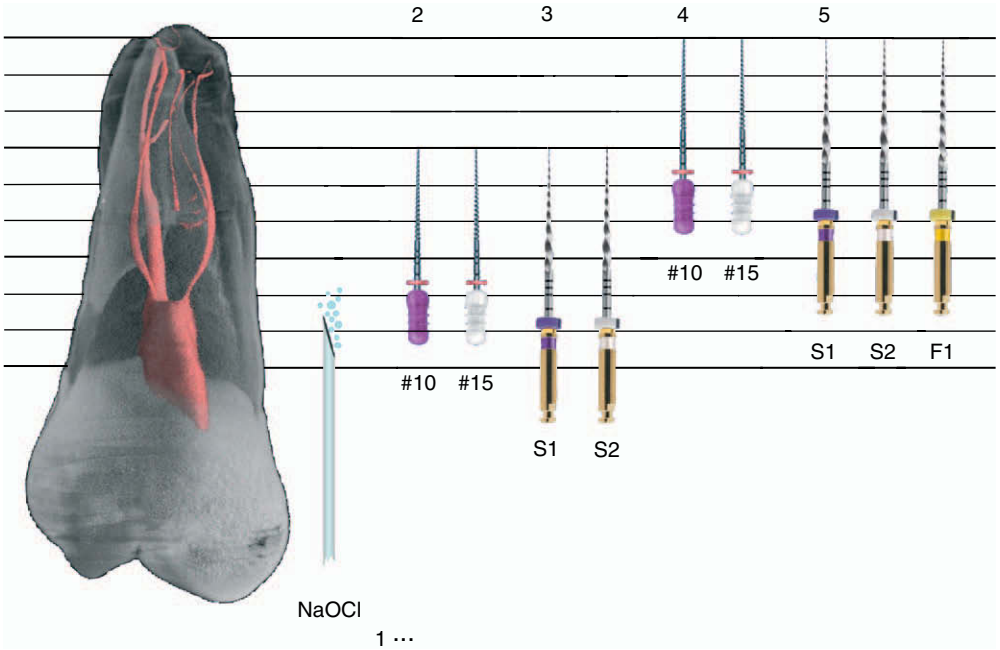


FIG. 9-62 Instrumentation of root canals with ProTaper instruments. After irrigation and scouting (*1* and *2*), the coronal thirds are enlarged with shaping files S1 and S2. Hand files then are used to determine the WL and to secure a glide path. Apical preparation is completed with S1 and S2. Finishing files are used to the desired apical width.

for apical widening; and various instruments for final smoothing.[448]

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. 9-65). Once the working length has been established, the apical third is flooded with 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. 9-66). Further enlargement is possible with the F4 and F5 instruments, but these files may

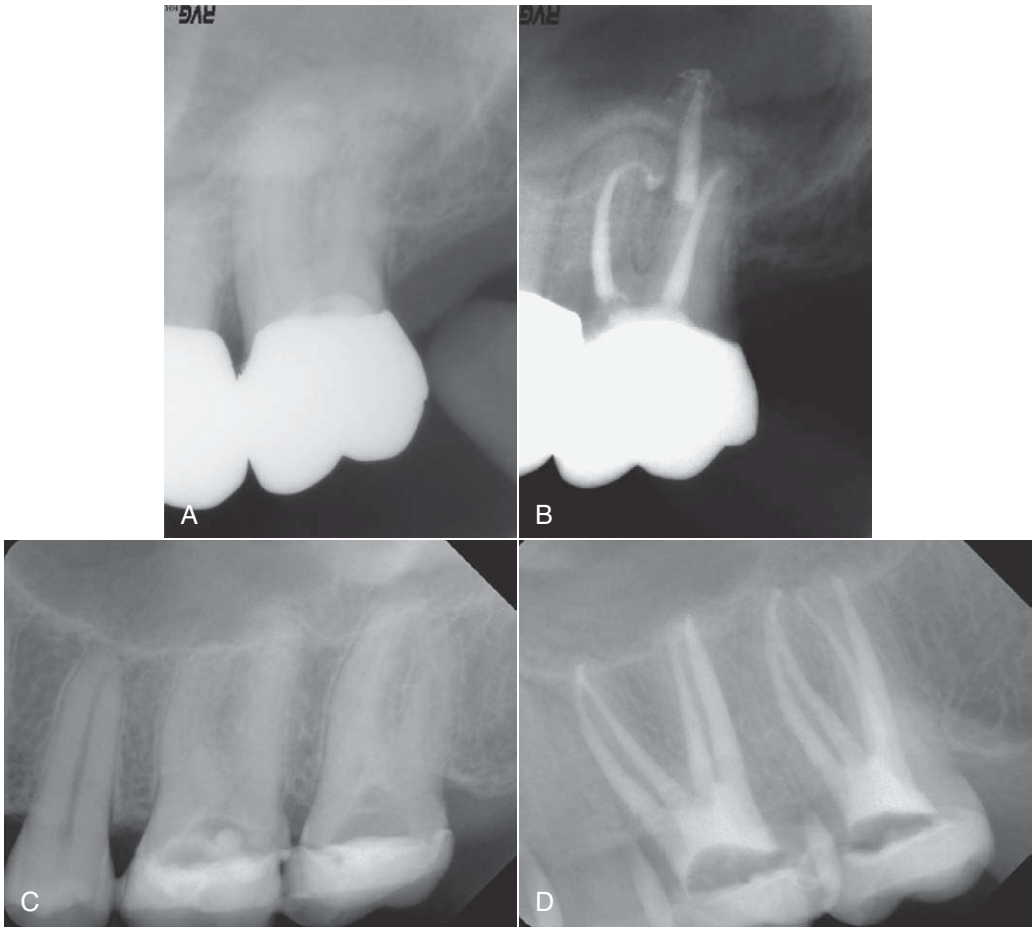


FIG. 9-63 Treatment performed with nickel-titanium rotary and hand instruments to eliminate instrument separation while maintaining biologic aims. **A,** Pretreatment radiograph of tooth #15. **B,** Posttreatment radiograph shows a significant curvature in the mesiobuccal canal and additional anatomy in the lingual root. **C,** Pretreatment radiograph of teeth #14 and #15. Both teeth were diagnosed with irreversible pulpitis. **D,** Posttreatment radiograph shows four canals in both of the treated maxillary molars. Note the wide apical preparation, particularly in the curved mesiobuccal canals. *(**A-B** courtesy Dr. T. Clauder; **C-D** courtesy Dr. H. Walsch.)*

BOX 9-5

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.

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. 9-67). 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. 9-68)—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. Some hybrid systems seem to work better than others, but the deciding factors are likely the root canal anatomy and an adequate preparation goal.

Other Systems

Ultrasonically activated files or alternating file movements with special handpieces may be used to work canal areas that rotary instruments cannot reach. However, to date no evidence shows that canal preparation with ultrasonic instruments is clinically superior. Similarly, neither traditional modified handpieces[192,412] nor a recently introduced system (EndoEZE AET; Ultradent, South Jordan, UT) have been shown to allow preparation of adequate canal shapes.[290]

Ultrasonic devices have been linked to a higher incidence of preparation errors and to reduced radicular wall thickness.[234,260,488] Newer analytic systems (e.g., μCT) allow tracking of the amount of dentin removed (Fig. 9-69); however, the

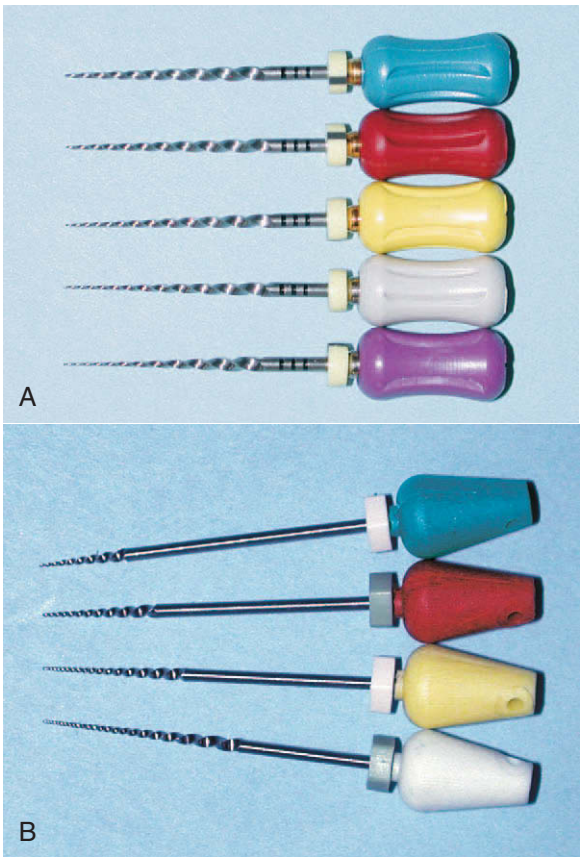


FIG. 9-64 Instruments with increased taper that can be used by hand. **A,** ProTaper instruments with special handles attached to rotary instrument shanks. **B,** GT hand instruments.

amount of potentially infected dentin that should be removed to maximize the chance of a successful outcome is unclear.

CANAL CLEANING TECHNIQUES

As stated earlier in this chapter, irrigants and other intracanal medicaments are necessary adjuncts that enhance the antimicrobial effect of mechanical cleansing and thus augment overall clinical efficacy.[79-81] It is well established that large areas of canal walls, particularly in the apical third but also in ribbon-shaped and oval canals, cannot be cleaned mechanically,[188,308,310,408,456,464] meaning that microbiota present in these untouched areas could survive (see Figs. 9-40, 9-45, and 9-47). Residual bacteria and other microorganisms exist both in these hard-to-reach spaces and in dentinal tubules.[171,300,342] Chemical disinfection is an important cornerstone of a successful outcome because it is directed towards elimination of microorganisms present in dentinal tubules and in the crevices, fins, and ramifications of a root canal system.[287,449] In one study, investigators prepared root canals, irrigated with saline solution, and sampled before, during, and after instrumentation.[112] They then cultivated and counted colony-forming units. These researchers found that with instrumentation alone, progressive filing reduced the number of bacteria regardless of whether rotary or stainless steel hand instrumentation was used. However, no technique resulted in bacteria-free canals. Siqueira et al.[380] confirmed this finding; they found that instrumentation combined with saline irrigation mechanically removed more than 90% of bacteria in the root canal. Many authors have stressed the importance of using antimicrobial irrigants during chemomechanical preparation to ensure complete disinfection.[384]

Substances that have been used to rinse and chemically clean root canals have different purposes, such as dissolution of soft and hard tissues, antimicrobial effect against bacteria or other microorganisms in the root canal, and inactivation of bacterial lipopolysaccharides. These substances also should be as nontoxic as possible to protect the periradicular tissues. Unfortunately, solutions that are toxic for bacterial cells frequently are toxic for human cells as well, so care must be taken to avoid extrusion of irrigants into periapical regions.[70,190]

Several factors are important for efficient root canal irrigation. One critical factor is the volume of irrigant. In a study evaluating the effect of different amounts of fluids, the volume of irrigant was found to affect the cleanliness of the root canal.[435,470] NaOCl and EDTA administered in larger volumes produced significantly cleaner root canal surfaces than smaller volumes.[470]

Irrigation Modes and Devices

There is a common consensus that root canal irrigants are indispensable aids in dissolving and inactivating organic debris and destroying microorganisms. In addition, some agents allow removal of a postpreparation smear layer in order to allow access to dentinal tubules. Several methods of employment of an irrigant inside the canal space are available.

Syringe Delivery

Application of an irrigant into a canal by means of a syringe allows exact placement, replenishing of existing fluid, rinsing out of larger debris particles, as well as allowing direct contact to microorganisms in areas that are reached by the needle tip. 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.[481] This was the case even if the needle tip diameter was three ISO sizes smaller than the diameter of the apical preparation.[64] Volume and speed of fluid flow are proportional to the cleansing efficiency inside a root canal. Therefore, both the diameter and position of the needle outlet determine successful chemomechanical débridement; placement close to working length is required to guarantee fluid exchange.[64,185,262]

The choice of an appropriate irrigating needle, therefore, is important. Although larger-gauge needles allow the irrigant to be flushed and replenished more quickly, the wider needle diameter does not allow cleaning of the apical and narrower areas of the root canal system (Fig. 9-70). Excess pressure or wedging of needles into canals during irrigation with no possibility of backflow of the irrigant should be avoided under all circumstances[190] to prevent extrusion of the irrigant into periapical spaces. In juvenile teeth with wide apical foramina or when the apical constriction no longer exists, special care must be taken to prevent resorption or overpreparation of the root canal.[107]

Another aspect is closeness of the needle tip or outlet to the apical endpoint of canal preparation to allow direct proximity of fresh irrigant to canal walls.[64,366] In that respect, size

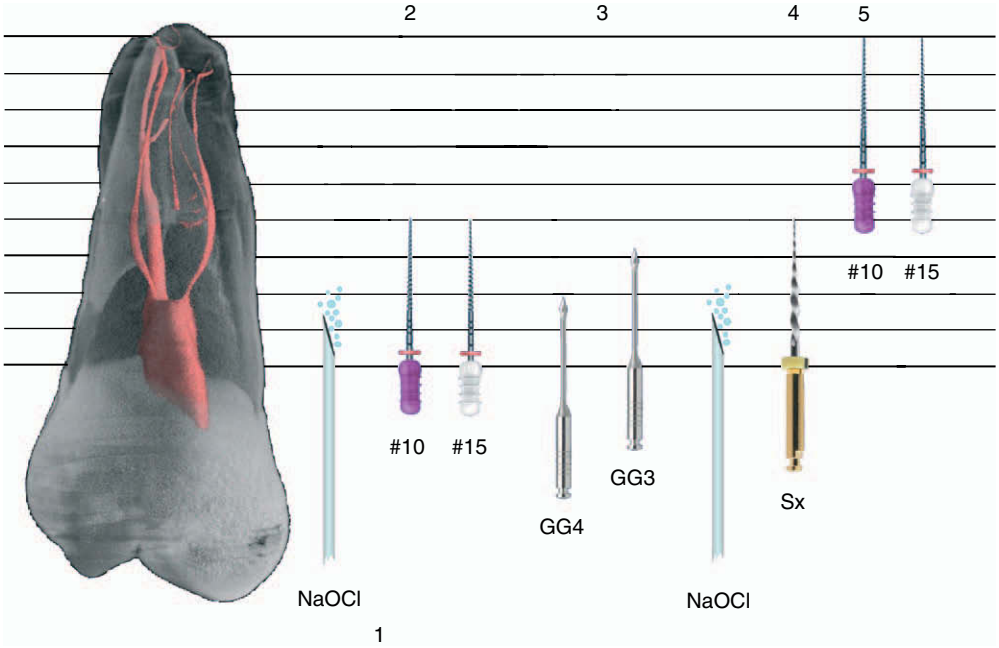


FIG. 9-65 Hybrid technique: Part I. After irrigation *(1)* and scouting *(2)*, GG drills *(3)* and/or ProTaper SX files *(4)* are used for coronal preenlargement and to secure straight-line access to the middle third. Precurved K-files are then used to explore and determine the working length *(5)*.

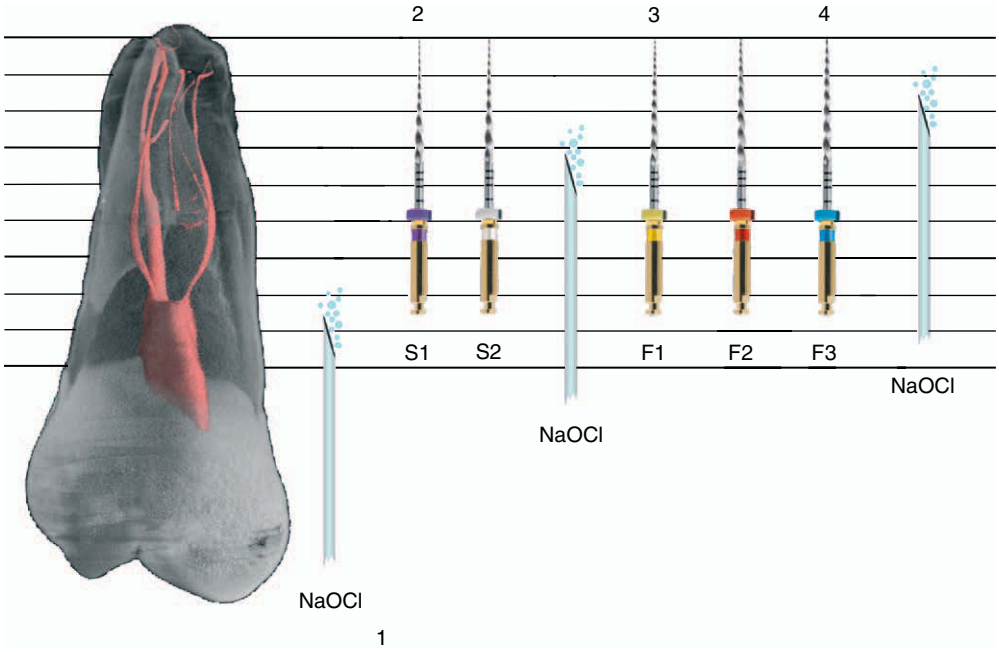


FIG. 9-66 Hybrid technique: Part II. In canal systems flooded with irrigant *(1)*, ProTaper shaping instruments S1 and S2 *(2)* and then finishing instruments F1 and F2 *(3)* are used to preenlarge the apical third, allowing irrigants access to the canals. Finishing instrument F3 may be used if feasible *(4)*.

of the irrigation needle,[97] as well as apical size and taper of root canal preparation,[89,264,280] play a role in allowing contact of irrigants to adjacent canal areas. Most root canals that have not been instrumented are too narrow to be reached effectively by disinfectants, even when very fine irrigation needles are used (see Figs. 9-44 and 9-70). Therefore, effective cleaning of the root canal must include intermittent agitation of the canal content with a small instrument[258,437] to prevent debris from accumulating at the apical portion of the root canal (see Fig. 9-42).

Preparation size[264] and taper[102] ultimately determine how close a needle can be placed to the final apical millimeters of a root canal. Some needles and suction tips may be attached to the air/water syringe to increase both the speed of irrigant

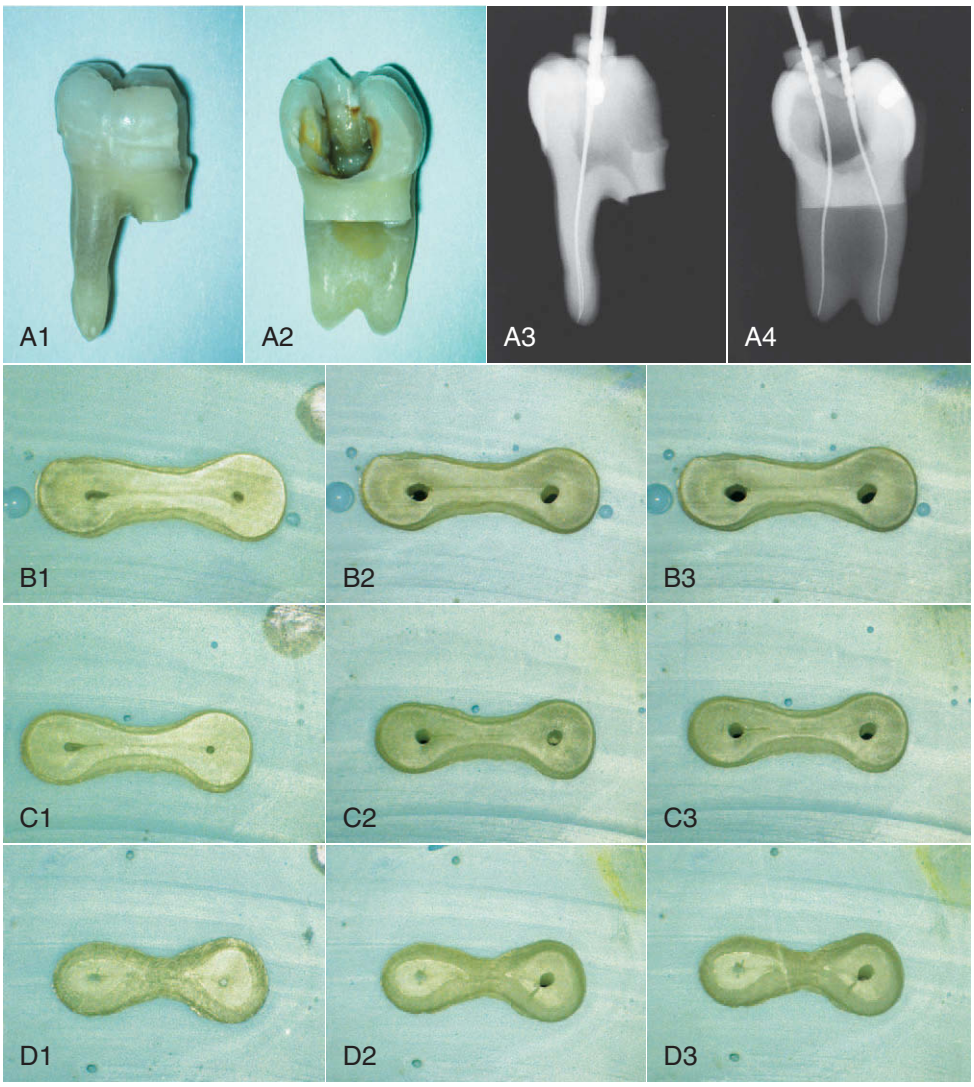


FIG. 9-67 Effect of a hybrid technique on root canal anatomy studied in a Bramante model. **A1-A4,** Both mesial canals of an extracted mandibular molar have been instrumented. Canal cross sections are shown before instrumentation **(B1-D1). B2-D2,** Cross sections after preenlargement with a ProTaper 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 Dr. S. Kuttler, Dr. M. Gerala, and Dr. R. Perez.)*

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

Irrigant that is placed inside the root canal more effectively reaches crevices and mechanically untouched areas if it is agitated inside the root canal. Coronoapical movements of the irrigation needle,[190] stirring movements with small endodontic instruments,[258,437] and manual push-pull movements using a fitted master gutta-percha cone have been recommended.[186]

Sonically Activated Irrigation

In one study,[341] investigators suggested that both passive sonic or ultrasonic irrigation rendered root canals significantly cleaner than manual preparation. Débridement of root canals supported by sonically and ultrasonically activated irrigation was superior to passive needle application of irrigants.[77,439] However, in comparison to sonic activation, ultrasonic irrigation produced significantly cleaner canals.[200,341] Other investigators discovered no significant difference in débridement between sonic or ultrasonic fluid activation inside a root canal. 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.[200,241,401] 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, Nyon, Switzerland) inserts, the EndoActivator System (DENTSPLY Tulsa Dental), and the Vibringe sonic syringe (Vibringe, Amsterdam, Netherlands).

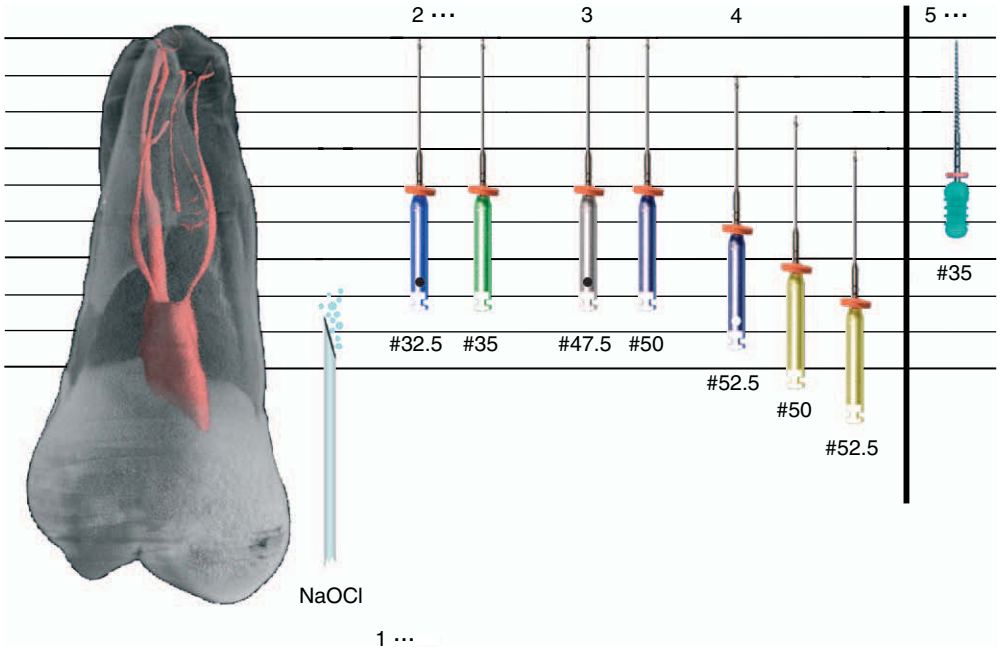


FIG. 9-68 Hybrid technique: Part III. Under irrigation *(1)*, LightSpeed instruments may be used to enlarge substantially (*2* and *3*) and to flare the apical section *(4)*. NiTi hand instruments *(5)* may be used similarly (see text for more detailed explanation).

Ultrasonically Activated Irrigation

Ultrasonically powered instruments have become indispensable now, with well-adapted tips from various manufacturers. During preparation, ultrasonic tips are able to remove minimal amounts of dentin, conserving as much tooth structure as possible. Visibility is better than with burs, and the tips can be diamond coated to increase their efficiency. However, all tips develop significant heat that is transferred through dentin walls and can cause necrosis of surrounding bone if used without coolant. The temperature elevation also takes place during use of ultrasonic power during root canal irrigation[10,195] and improves the antibacterial effect through warming of the irrigating solution.[486]

Ultrasonic action is most effective if the file is able to oscillate freely inside a given root canal.[240] *Passive ultrasonic irrigation* is defined as activation of the rinsing agent without simultaneous preparation of the root canal walls.[436,486] Passive ultrasonic irrigation is believed to promote tissue removal and tissue dissolution and may be done with a smooth wire insert that will avoid damaging canal walls and altering the shape in an undesirable way.[439] This strategy allows cleaning of isthmus areas, fins, or C-shaped canals by acoustic streaming and to a lesser extent cavitation, as well as (to some degree) other hard-to-reach areas such as dentinal tubules or lateral canals. Disinfection is rendered more effective, an important consideration in necrotic cases.[10,90] Kuah et al.[217] demonstrated that to eliminate smear layer and debris in the apical region of a prepared root canal, a 1-minute application of EDTA with ultrasonics followed by a final flush of NaOCl was the most proficient method.

Negative- and Positive-Pressure Irrigation

Another approach to afford better access of irrigation solution is so-called negative-pressure irrigation. 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.[281]

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. This system has been investigated by several authors and was superior to customary needle irrigation in cleaning and disinfection.[67,262] Researchers[262] used 30-gauge irrigation needles in canals shaped to ISO size #40 and compared irrigation with the RinsEndo system to conventional needle irrigation and so-called manual-dynamic irrigation that involves pumping action of a fitted master gutta-percha cone inside a root canal; in this study, 200 push-pull strokes were delivered. The cleaning effect of the RinsEndo unit was found to be superior to conventional needle irrigation, but the least amount of residual debris was found after hand agitation of a master cone.

Eradication of Microorganisms

Various types of microorganisms—bacteria,[205,215,270,272] yeasts,[449,450] and possibly viruses[339,340]—can infect the pulp and may lead to apical periodontitis (see Chapters 14 and 15 and Fig. 9-7). These microorganisms must be reduced or eliminated to reestablish periradicular health. When bacterial

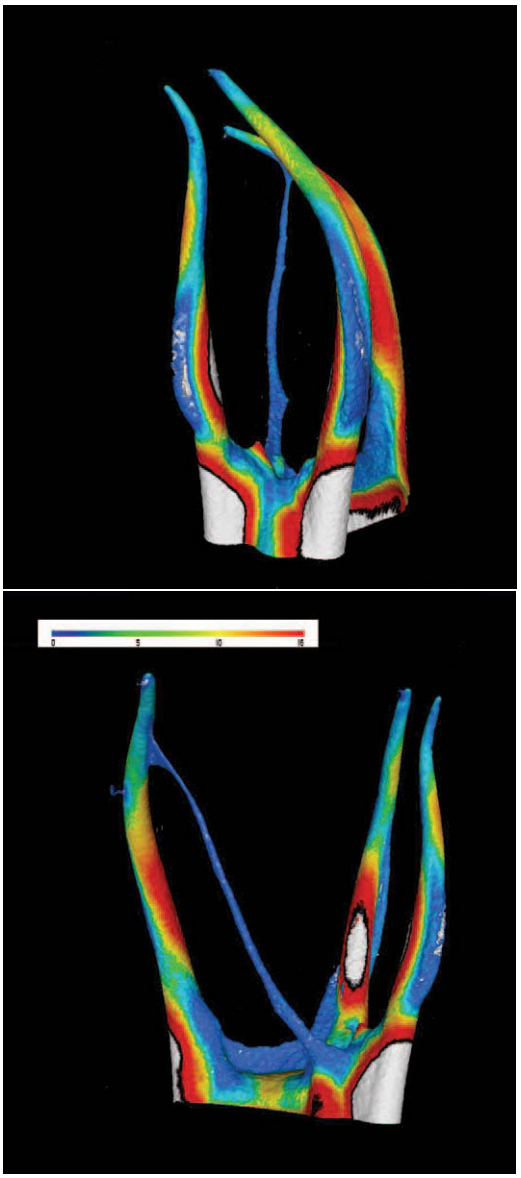


FIG. 9-69 Reconstruction from micro–computed tomography data (36 µm isotropic resolution) showing the amount of removed dentin by color coding. Maxillary molar shaped with ProTaper, apical size #25 (=F2) in mesiobuccal and distobuccal canals; palatal canal shaped to size #30 (=F3). The bar indicates the removed volume, expressed as the number of voxels. Note the *red areas,* which indicate dentin removal of more than 500 µm.

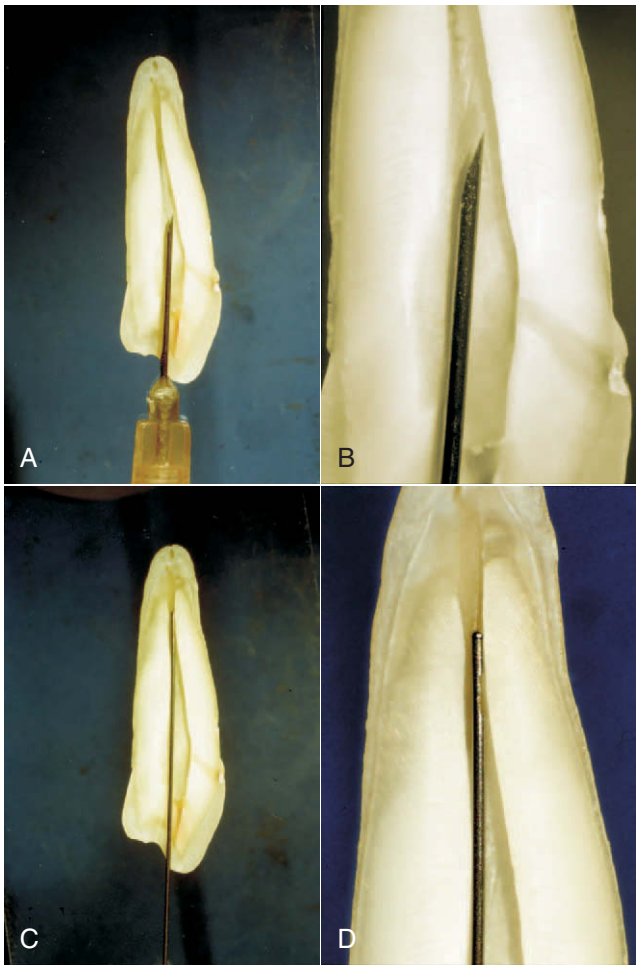


FIG. 9-70 Irrigation needles inserted into prepared root canals. **A-B,** A 27-gauge needle barely reaches the middle third. **C-D,** A 30-gauge, side-venting needle reaches the apical third (see Figure 9-43).

samples test negative after treatment, the prognosis is improved.[381,387] During mechanical root canal preparation, endodontic instruments are used to clean and enlarge root canal systems. Rotating instruments have an additional advantageous "Archimedes screw" effect by which debris is transported in an apicocoronal direction.[112] Even when simple saline was used as an irrigant, a 10-fold to 1000-fold reduction of the bacterial load through mechanical instrumentation was demonstrated.[80,112,288]

However, as noted earlier, instrumentation alone does not produce a bacteria-free root canal. In one study, dentin samples tested positive in most of the teeth after mechanical instrumentation, even though bacteria had been eliminated from the root canals in some cases.[80] In that study, bacteria persisted in seven root canals despite mechanical cleaning and saline irrigation during five consecutive appointments. Moreover, teeth with a high number of bacteria in the initial sample remained infected despite being treated five times.[80] In another study, teeth that caused symptoms tended to have more bacteria than teeth with no clinical symptoms.[288]

Other researchers[287] investigated the effect of endodontic irrigants and dressings in standardized bovine dentin specimens that were infected with test bacteria. They found that bacteria were capable of colonizing the canal lumen and dentinal tubules. In the specimens used, *E. faecalis* rapidly infected the whole length of the tubules, whereas *Escherichia coli* penetrated approximately 600 µm. They also found that IKI appeared to be more effective at destroying bacteria than NaOCl, which was more effective than CHX.

Other investigators have explored the effects of NaOCl (with and without EDTA), CHX, and hydrogen peroxide in varying concentrations when used in sequence or in combination as endodontic irrigants.[181] They found that CHX and NaOCl were similarly effective in eliminating the bacteria

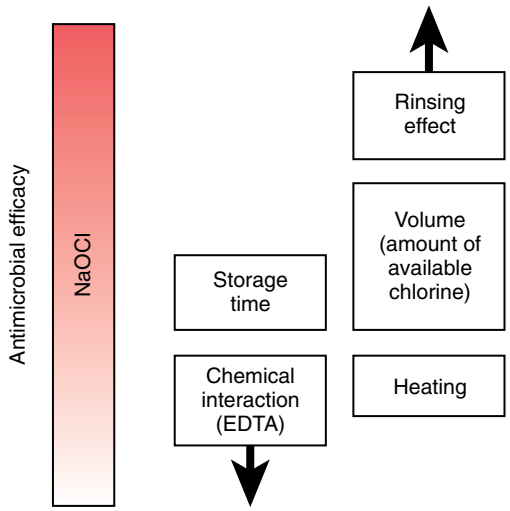


FIG. 9-71 Interaction of sodium hypochlorite with various factors that determine its efficacy.

BOX 9-6

Properties of an Ideal Irrigant for Root Canal Treatment

The irrigant should:
- Be a highly effective disinfectant
- Be nontoxic locally and nonallergenic
- Differentiate between necrotic and vital host tissue
- Retain its effectiveness with dental hard tissue and when mixed with other irrigants

tested. Synergistic effects were observed for some of the irrigants (e.g., CHX and IKI).

Both of the preceding studies used infected dentin specimens. When evaluating literature about antimicrobial efficacy, clinicians must keep in mind that most disinfecting solutions are inhibited or even inactivated by contact with dentin or dentin powder during root canal preparation.[169,317] Moreover, chemical interactions occur between irrigation solutions; for example, NaOCl can become ineffective if it comes in contact with EDTA[161] (Fig. 9-71).

Some of the more difficult to remove endodontic pathogens that can cause treatment failure are enterococci, *Actinomyces,* and *Candida* organisms[47,276,277,378,462] (see Chapter 15). Table 9-1 presents the results of a number of studies evaluating the effectiveness of some commonly used antimicrobial agents.

Currently, the endodontic irrigation solution with the best proteolytic effect is NaOCl,[481] even though it does not meet all the requirements of an ideal irrigant (Box 9-6). It is readily available, inexpensive, and consequently a widely used irrigation solution. As mentioned previously, necrotic tissue and debris are dissolved by the breakdown of proteins into amino acids through free chlorine in NaOCl. However, because unbound chlorine is the important component, the solution

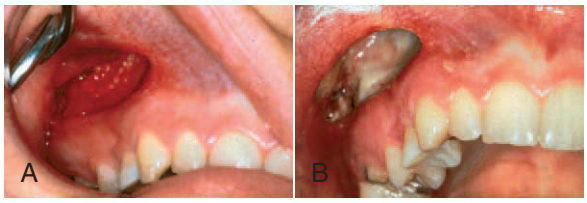


FIG. 9-72 Toxic effect of sodium hypochlorite on periradicular tissues. After root canal treatment of tooth #3, the patient reported pain. **A,** On a return visit, an abscess was diagnosed and incised. **B,** Osteonecrosis was evident after 3 weeks.

must be replenished frequently during preparation to compensate for lower concentrations and to constantly renew the fluid inside the root canal. This is even more important when the root canal is narrow and small and files must carry the NaOCl to the apical third during instrumentation (see Fig. 9-44). A 1% solution is effective at dissolving tissue and providing an antimicrobial effect. The use of 6% commercial household bleach in its undiluted form causes substantial necrosis of wound surface areas and may result in serious clinical side effects (Fig. 9-72). It is diluted in 1:1 or 1:3 ratios with water to produce a 2.5% or 1% solution; both are suitable for clinical endodontic use.[398,477,482]

Importantly, to avoid extrusion and serious damage to periapical tissues, irrigation needles should never be wedged into canals during irrigation.[70] Higher concentrations of NaOCl are more aggressive toward living tissue and can cause severe injuries when forced into the periapical area (see Fig. 9-72).

Such accidents can be prevented by marking the working length on the irrigation needle with a bend or a rubber stop and by passively expressing the solution from the syringe into the canal (see Fig. 9-70). The needle should be continuously moved in an up-and-down motion. It should remain loose in the canal, allowing a backflow of fluid. The goal is to rinse the suspended, concentrated dentinal filings out of the pulp chamber and root canals as new solution is brought down into the most apical areas by the endodontic instrument and the capillary effect.

As stated before, patency files should be used carefully and should not be extended farther than the periodontal ligament, because they are possible sources of irrigant extrusion.

In one study, heating increased the antibacterial action of NaOCl.[104] Heating can be done in several ways; for example, after the solution has been drawn into the irrigating syringe, a syringe warmer can be used (e.g., Syringe Warmer [Vista Dental Products, Racine, WI]) (Fig. 9-73). Heating also enhanced the antibacterial effectiveness of CHX and $Ca(OH)_2$ solutions.[131] A 0.5% NaOCl solution heated to 113° F (45° C) dissolved pulp tissue as efficiently as a 5.25% solution used as the positive control (Fig. 9-74).[386] Heating to 140° F (60° C) resulted in almost complete dissolution of tissue. Studies have shown that 1 minute at 116.6° F (47° C) is the cutoff exposure at which osteoblasts can still survive; however, higher temperatures may in fact be sufficient to kill osteoblasts and other host cells.[128,129] Also, warming of NaOCl to 122° F (50° C)[53] or 140° F (60° C)[5] increases collagen dissolution and disinfecting potential, but it may also have severely detrimental effects on NiTi instruments, causing corrosion of the metal surface after immersion for 1 hour[51] (Fig. 9-75).

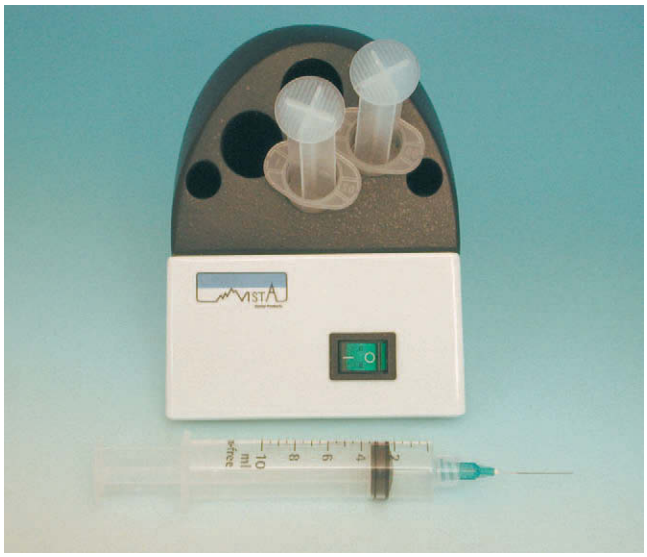


FIG. 9-73 Device for heating syringes filled with irrigation solution (e.g., sodium hypochlorite) before use.

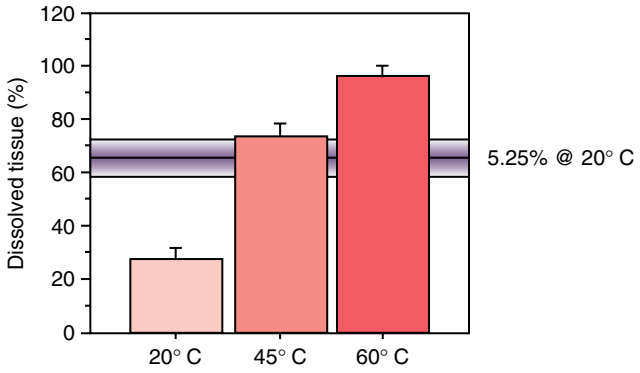


FIG. 9-74 Effect of heating on the ability of 0.5% sodium hypochlorite (NaOCl) to dissolve pulp tissue: NaOCl heated to 113° F (45° C) dissolved pulp tissue as well as the positive control (5.25% NaOCl) did. When the NaOCl was heated to 140° F (60° C), almost complete dissolution of tissue resulted. *(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 *31:669–671, 2005.)*

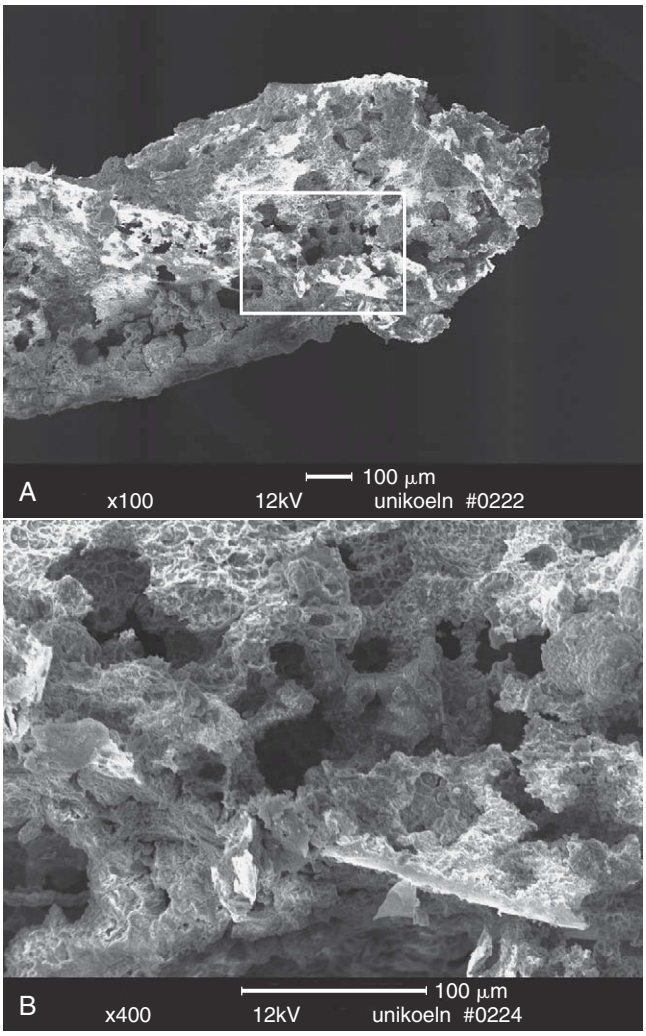


FIG. 9-75 Corrosion of nickel-titanium files in heated sodium hypochlorite (NaOCl). **A,** Rotary instrument immersed for 2 hours in NaOCl heated to 140° F (60° C). **B,** Magnification of rectangular area in **A,** showing severe corrosion.

As stated before, an increase in the temperature of the irrigant may be another reason to include ultrasonic devices in canal irrigation[10,486]; using these devices may also increase the tissue-dissolving capabilities of NaOCl via temperature elevation,[5] but this effect seems to be limited to the main canal.[10] In one study, peak irrigant temperatures during ultrasonic irrigation reached 113° F (45° C) near the file tip but remained at 89.6° F (32° C) on the outer root surface of teeth prepared to a size #45.[86] The effect seems to depend on the insert,[486] possibly due to its oscillation patterns.

Yet another reason for using ultrasonic devices might be enhancement of canal débridement. However, some authors have reported no significant effect with ultrasonics, neither in débriding root canal walls[96,108,258] nor in reducing bacterial counts.[118,382] However, the majority of available studies appear to find both bacterial reduction[194] and improved débridement,[167] as reviewed recently.[438]

Smear Layer Management

EDTA is a decalcifying chelating agent used as a 15% to 17% buffered solution during instrumentation of root canals. The decalcifying efficacy of EDTA-containing pastes is variable.[160,441] EDTA acts as a chelator with calcium ions and removes the dentinal debris produced on the root canal walls during preparation. It thus opens dentinal tubules, promoting better penetration of disinfectants.[154,191,422,485] Whenever the wall of a root canal is instrumented, whether by hand or rotating instruments, the parts of a dentin wall touched by an instrument are covered by a surface layer called the *smear layer*.[261,296] The

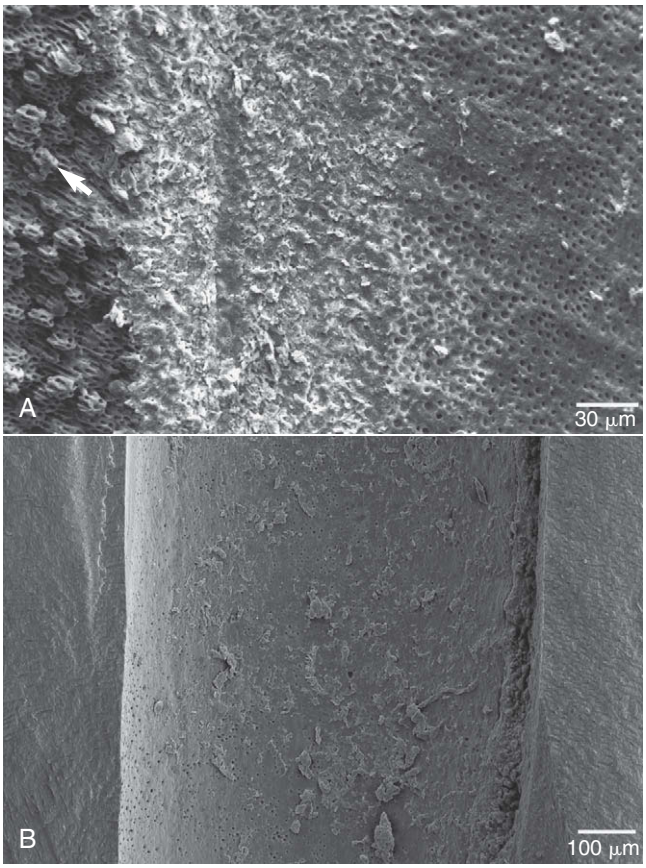


FIG. 9-76 Prepared root canal surfaces after irrigation, showing varying degrees of smear layer. **A,** Scanning electron micrograph (SEM, ×25) showing prepared areas with and without open dentinal tubules. The presence of calcospherites indicates that no mechanical preparation has been done laterally (*arrow*). **B,** SEM, ×400, showing thin, homogenous smear layer and scattered debris in a canal that received a final sequence of a high-volume flush of 17% ethylenediamine tetra-acetic acid (EDTA) followed by 2.5% sodium hypochlorite (NaOCl).

smear layer, which consists of dentin shavings, cell debris, and pulp remnants,[369] can be described as itself having two separate layers: a loose, superficial deposit and an attached stratum that extends into the dentinal tubules, forming occluding plugs.[87]

For some time, clinicians and researchers paid little attention to the smear layer, partly because it was a thin superficial layer (1 to 5 μm) that might be present or not, depending on the type of instrument and the sharpness of its cutting blades.[369] Also, because acids and chelating agents dissolve the smear layer, it was removed and escaped attention in routinely processed specimens (Fig. 9-76).[109] Smear layers are not seen in unprepared canal areas, which may have calcospherites, buttonlike structures that are abundant on intracanal surfaces.

Some authors have reported that an overlying smear layer delays but does not eliminate the effect of medicaments.[287] Others contend that a smear layer may adversely affect disinfection and may also increase microleakage after canal obturation.[369] Although organic substrate in a smear layer may serve as a nutrition source for some species of bacteria,[65,297] some have suggested the converse: that a smear layer can act as a beneficial barrier, preventing microorganisms from entering the dentinal tubules when a root canal is colonized by bacteria between appointments.[121] The potential of intracanal disinfectants has been evaluated in vitro after removal of the smear layer with a combination of 5.25% NaOCl and 17% EDTA.[171] The decalcifying effect of EDTA is self-limiting, so the solution must be replaced at intervals.[191] EDTA can help open very narrow root canals and can decalcify to a depth of approximately 50 μm. Because the smear layer consists of organic and inorganic components, the combined use of NaOCl and EDTA, with time frames of 30 seconds to 60 seconds each, is considered most effective for its removal.[83] In general, it seems beneficial to remove the smear layer in the later phases of endodontic therapy rather than during the early phases. Research continues on ways to improve the effectiveness of irrigation. For example, tensides were added to irrigants more than 20 years ago to reduce their surface tension, thereby improving wetability.[6] The rationale for this increased wetability was to improve the penetration of irrigants into the dentinal tubules,[409] and this concept is still pursued with MTAD today. One irrigation "cocktail" investigated was a mixture of 5% NaOCl and 17% EDTA with the tensioactive chemical Triton X-100; this solution was used with ProFile instruments.[7,144] The study reported that apical smear layer scores were significantly lower compared with those of control groups when the tensioactive agent was used throughout the preparation process. However, a more recent study found no effect of a wetting agent on the action of EDTA, measured as calcium in eluates from root canals in vitro.[484] Rather than surfactant, ultrasonic irrigation appears to promote smear layer removal by EDTA.[238]

Liquid disinfectants were effective against *E. faecalis* in dentinal tubules up to depths of 400 μm. Microbiologic analyses of split root halves showed that early removal of the smear layer resulted in significantly higher bacteria counts.[121] In contrast, other researchers have acknowledged that the smear layer, while acting as a barrier, might block irrigation solutions from entering the dentinal tubules.[42] Moreover, some bacteria (e.g., *Bacteroides gingivalis* and *Treponema denticola*) have the potential to dissolve smear layer proteins,[432] thereby producing gaps which could promote both coronal and apical microleakage and bacterial multiplication.

Fig. 9-77 shows root canal cross sections with very little debris; irrigating solutions can penetrate the dentinal tubules in this example. Some reported that the presence of the smear layer had no significant effect on apical leakage in dye penetration testing.[132,247] Others described an improved seal after removal of the smear layer.[43,411] The latter study, which used a coronal leakage model, found a significantly decreased incidence of bacterial penetration (30% versus 70%) when the canals were irrigated with 17% EDTA and 5.25% NaOCl before obturation. In obturated root canals, a remaining smear layer led to bacterial leakage in 60% of the samples versus no leakage when the smear layer was removed.[101] Other authors had similar results after smear layer removal with EDTA solution alone. Another investigation[421] found that many lateral canals in the apical third of root canal systems cleaned with a barbed broach wrapped with MTAD-soaked cotton showed less erosion than when EDTA was used. Other studies have found

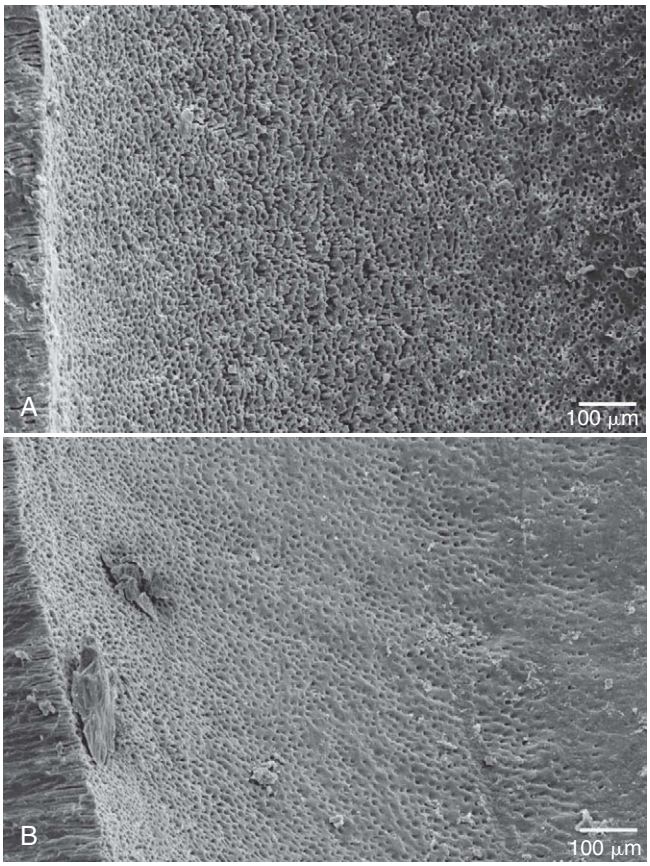


FIG. 9-77 Example of canals with minimal smear layer. **A,** Middle third after irrigation with 17% ethylenediamine tetra-acetic acid (EDTA) and 2.5% sodium hypochlorite (NaOCl). **B,** Apical third with some particulate debris.

that a stronger bond was present when the smear layer was removed,[148] and a statistically significant reduction of microleakage was also measured.[124,349,350] Still another investigation reported increased apical microleakage of the filled root canal after removal of the smear layer.[419]

As shown in Fig. 9-43, dye staining is improved when the dentinal tubules are opened and the smear layer is removed with EDTA, at least in the two more coronal levels. However, this effect is at least partly due to tubular sclerosis and the overall lower number of tubules apically, resulting in fewer diffusion pathways across dentin.[292]

Although the effect of the smear layer on leakage has been widely studied, its removal from root canal walls remains controversial. The apparently conflicting results of studies could stem from differences in the various microleakage test models[467] and from different obturation and irrigation techniques. The problem of coronal leakage has received much attention as a major factor in determining the success or failure in root canal therapy.[47,327,330,331,428]

In two in vitro studies, the noninstrumentation technique, which relies on activated irrigation solutions rather than mechanical preparation, produced excellent canal cleanliness[244,245] (see Fig. 9-10, A). However, preliminary clinical studies identified a need for improvement before this system can be used routinely to clean root canals.[27]

For the time being, root canals must be mechanically enlarged before irrigation. Larger apical preparations enhance the efficacy of irrigation, and the additional use of ultrasonic energy during cleaning and shaping may also increase the efficacy of endodontic irrigants.[87,88,105,224] Ultrasonics used passively in canals with sufficiently large apical preparations may reach and better clean any uninstrumented canal areas.[241,463,465] One investigation studied the débriding ability of 2.5% NaOCl in canal recesses.[105,106] In 10 of 11 cases, these researchers found significantly cleaner histologic sections after ultrasonically activated irrigation. In the ultrasonically treated group, the bacteria count was reduced by 99.8%, but hand filing alone reduced the bacteria count by 99.3%, so the improvement from ultrasonic therapy was limited. With ultrasonics, root canals are débrided by shear stresses produced between the irrigant and the canal wall, with subsequent cell disruption.[9]

Acoustic streaming of the irrigation fluid through ultrasonic treatment has been suggested as a method of improving cleanliness. However, this effect occurs mainly in the most coronal levels; the apical areas were least affected by activated irrigation[96,258] (see Fig. 9-43). Because the amplitude of the oscillation is greatest at the instrument's tip, attenuation and constraint most significantly affect the apical part,[447] where the diameter of the canal is smallest.

One investigator reported that the most effective regimen with ultrasonic energy was to activate every dose of irrigant placed in the canal.[85] With this approach, roughly 18 minutes of irrigation is required per canal. Other investigators used an irrigation time of 1 minute each for EDTA and NaOCl, which seems clinically more practical.[83,258] These authors stated that the use of ultrasonic energy for irrigant activation did not improve débridement compared with control groups. However, bacterial species show varying degrees of susceptibility to ultrasonication.[8,38,255,390]

Because of the conflicting evidence concerning the effectiveness of ultrasonics in root canal therapy, other methods of disinfecting and débriding canals properly must be studied. Such research might include better ways to deliver irrigants and disinfecting solutions.

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. 9-78) and more complicated (Fig. 9-79) 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.

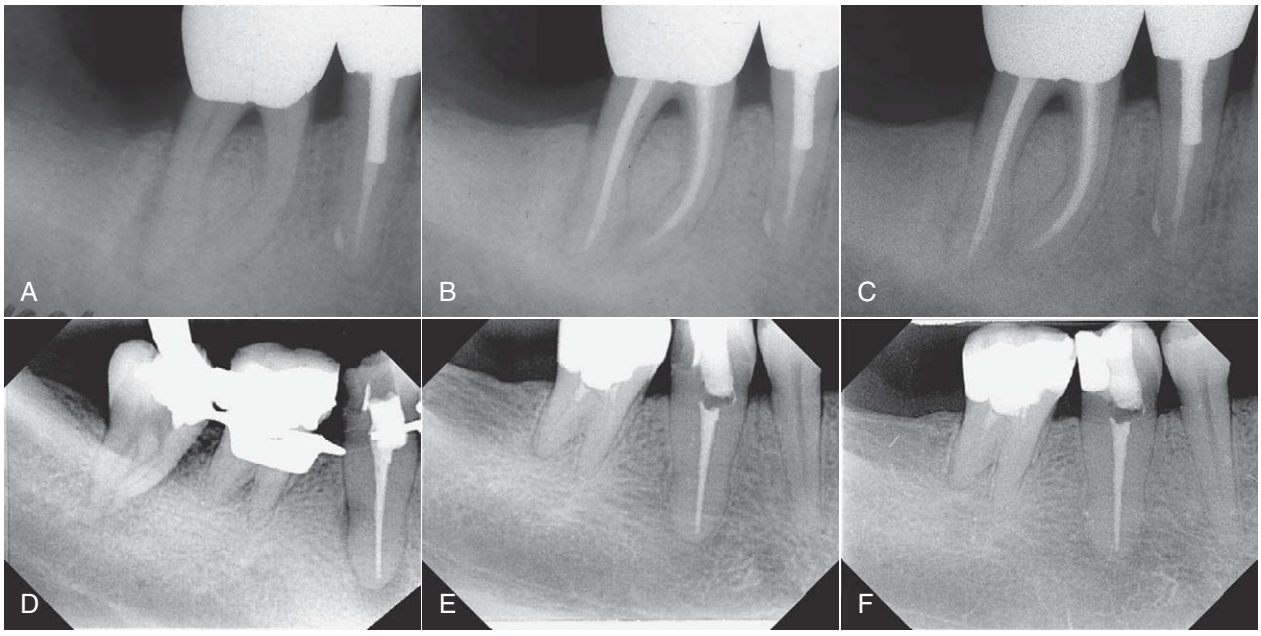


FIG. 9-78 Clinical cases treated according to the principles detailed in this chapter. **A,** Pretreatment radiograph of tooth #30 with a periradicular lesion. **B,** Postobturation radiograph. **C,** Two-year follow-up radiograph shows osseous healing. **D,** Immediate postobturation radiograph of tooth #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.

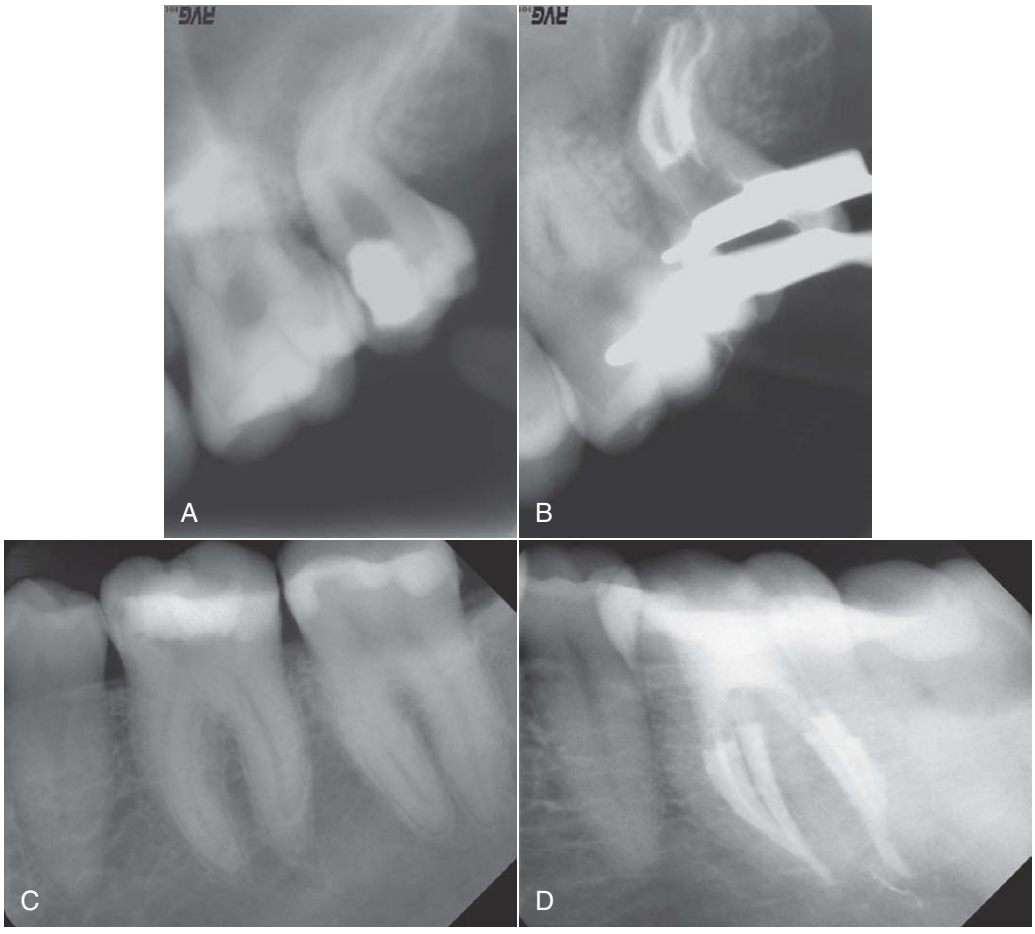


FIG. 9-79 Complicated clinical cases treated with hybrid techniques. **A,** Pretreatment radiograph of tooth #16 indicates laceration and significant curvature of all roots. **B,** Posttreatment radiograph shows multiple planes of curvature. **C,** Pretreatment radiograph of tooth #19, which was diagnosed 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 Dr. T. Clauder;* ***C-D*** *courtesy Dr. H. Walsch.)*

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CHAPTER 10

Obturation of the Cleaned and Shaped Root Canal System

WILLIAM T. JOHNSON and JAMES C. KULILD

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CORONAL ORIFICE SEAL

IMPORTANCE OF EFFECTIVELY SEALING THE ROOT CANAL SYSTEM

Success in endodontic treatment was originally based on the triad of débridement, 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 débridement, thorough disinfection, and obturation; and the coronal restoration. 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.[214]

In an early radiographic study of success and failure, Ingle and colleagues[145] 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 and have been shown to adversely affect the apical seal.[341]

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,[83] dyes,[151] bacteria,[56] proteins,[202] endotoxins,[56] glucose penetration,[225] and computerized fluid filtration.[309] These methodologies have employed a variety of in vitro conditions and experimental periods that often produce conflicting results. Fortunately, clinical success rates after endodontic treatment are high despite the varied conditions, materials, and techniques employed.[62,179,256] Circumstantial evidence indicates that the cleaning and shaping procedures provide an aseptic environment, and 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 nonhealing 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.[98] 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. Results emphasized the role of bacteria in apical pathosis and the importance of cleaning and shaping procedures.

In a controlled animal study,[253] 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, emphasizing 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.

At present 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.[326] 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.[146] Evidence indicates canals are often underprepared in the apical one third.[55] 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.[209] With vital pulp tissue, 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.[288,289] The role these organisms play in the disease process is not well understood. The reader is referred to Chapter 15 for a fuller discussion of this vexing issue.

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 (adequate provisional restoration) (Fig. 10-1). 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.[2,122] 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.[155,205] 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.[132,287,326,344] 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.[81]

Coronal leakage has also been demonstrated to contribute to treatment failure.[244,315] Maintaining an effective coronal seal and placing an appropriate restoration, as discussed in Chapter 22 should be considered an essential component of successful endodontic treatment.[134] Investigators suggest that it is more prudent to use a final restorative material versus a temporary material to prevent leakage.[319] One study[244] reported that good postendodontic restorations resulted in significantly more successful cases when compared with good endodontics (80% vs. 75.7%) and poor restorations resulted in significantly more periradicular inflammation cases when compared with poor endodontics (30.2% vs. 48.6%). The success rate for a good restoration and good endodontics was 91% compared with a success rate of 18% with the poor endodontic treatment and a poor restoration. In combination with technically good restorations the success rate was 81%. With poor restorations the success rate was 71%. Technically poor endodontics combined with either good restorations or poor restorations had significantly lower success rates of 56% and 57%, respectively. The radiographic quality of the endodontic treatment was significantly more important than the technical quality of the coronal restoration when the periapical status of endodontically treated teeth was evaluated. Investigators[12] noted that the prognosis for endodontically treated posterior teeth restored with crowns was enhanced sixfold. Thus, the ability to deliver combined high-quality endodontic and restorative treatment is a major factor in good clinical outcomes.

Using histologic and microbiologic techniques, investigators evaluated 39 teeth that were without proper restorations for at least 3 months and exposed to caries and the oral environment.[246] Thirty-four specimens were without radiographically discernible periradicular pathosis. Lesions were detected on five roots. Stainable bacteria were found in abundance at the orifice and in dentinal tubules but were absent midroot and apically in 37 roots. Inflammatory cell infiltrates were absent or sparse in 32 teeth whereas 7 teeth exhibited distinct inflammation. Despite pathosis involving five roots the results indicated that well-obturated root canals are resistant to bacterial penetration when exposed to the oral environment. Findings in this study were consistent with an observational study finding that coronal leakage was not a significant factor in root canal failure, using matched pairs of teeth for analysis.[247]

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.[346] Although a poorly obturated canal and leakage are correlated, radiographic evaluation of obturation does not correlate well with leakage.[124,164] An adequate radiographic appearance of the obturation may not correlate with an adequate seal (Fig. 10-2).[90] Variation in radiographic interpretation by the clinician, the overlying osseous structures, and the lack of uniformity in the obturation materials are significant variables.[29,86,87,164]

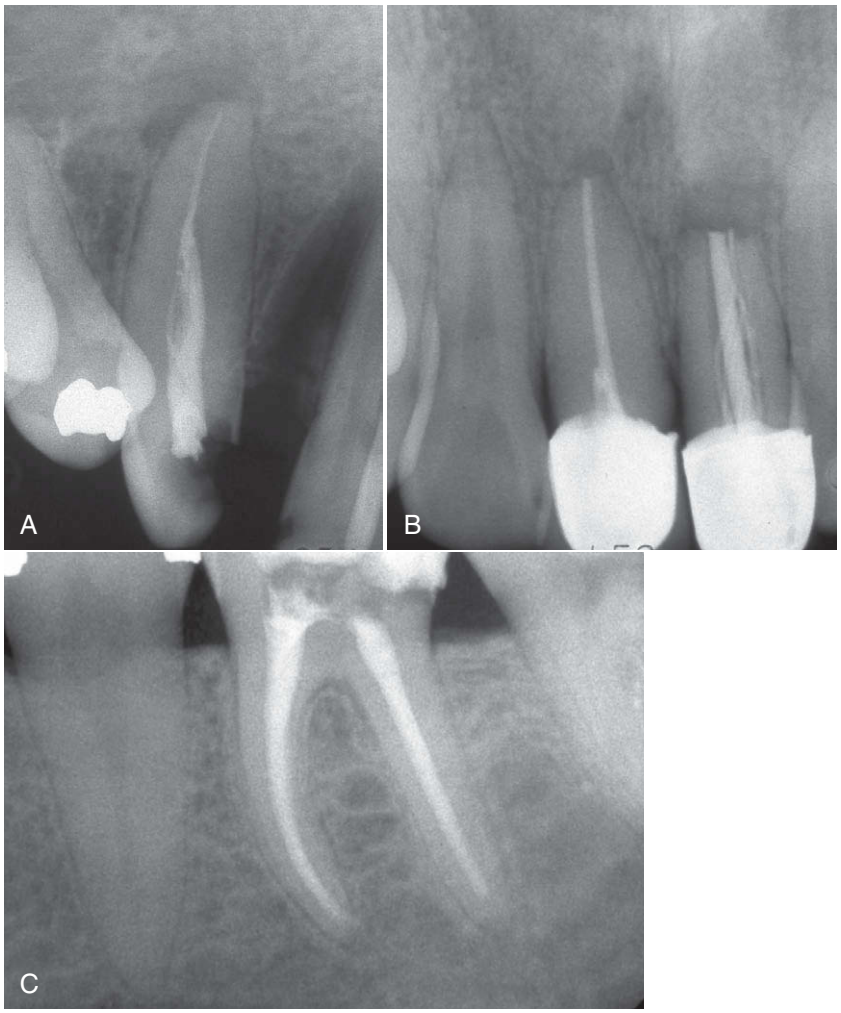


FIG. 10-1 Examples of inadequate obturation. **A,** Maxillary right canine with adequate length but lacking density and no coronal seal. Central incisor is filled to adequate length but obturation exhibits voids. **B,** Maxillary central incisors. Maxillary right central incisor exhibits a lack of density 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.

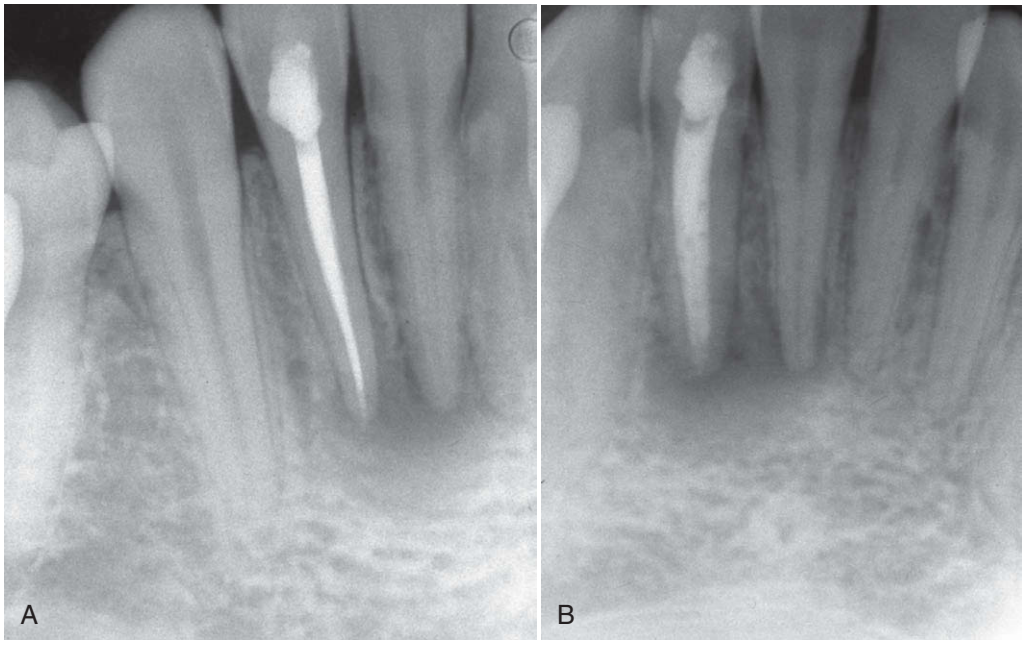


FIG. 10-2 A, Posttreatment radiograph of a mandibular left lateral incisor with ostensibly adequate obturation. **B,** Angled view reveals voids.

In a prospective study the Toronto group[100] evaluated success and failure of endodontic treatment at 4 to 6 years after completion of treatment. 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%). This study also agreed with previous studies[291,302] indicating preexisting apical pathosis as a factor reducing a favorable prognosis and highlighted the obturation technique as a factor influencing success and failure.[291]

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.[270] 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[129] 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." 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.[144]

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.[166] In 1847 Hill developed the first gutta-percha root canal filling material known as "Hill's stopping."[166] 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.[10]

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?).[234] 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.[159] In 1893 Rollins introduced a new type of gutta-percha to which he added vermilion.[328] 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 rosins, was introduced by Callahan in 1914.[52] 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.[83,252] 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.[318] 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 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 periapical pathosis (chronic 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.[296] 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, studies demonstrated that the incidence of pain was not increased in patients who were treated in one appointment versus those treated in multiple appointments.[107,210,217,220,249,294]

In contrast to teeth with vital pulp tissue, teeth exhibiting pulp necrosis frequently exhibit bacterial contamination and may require a different approach to treatment.[318] Sjögren and colleagues[290] raised questions regarding the long-term prognosis of teeth exhibiting necrotic pulp tissue and apical periodontitis treated in a single visit. 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 Chapters 9 and 15 for a fuller discussion of this issue.] Before obturation, cultures were obtained, using advanced 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.

However, other investigators were unable to confirm Sjögren's results, in a study of 39 patients with periapical lesions exhibiting both positive and negative canal cultures at the time of obturation. Twenty-one teeth were treated in one visit and 18 in two visits with an interappointment dressing of calcium hydroxide. Periapical healing was observed over a period of up to 4.5 years. Complete radiographic healing occurred in 81% of the cases in the one-visit group and in 71% of the cases in the two-visit group.[236] A randomized control trial comparing the difference in success between one- and two-visit procedures, using a standardized protocol, demonstrated no significant differences in success at 12 months in 63 patients.[230] A 2-year clinical and radiographic study demonstrated no statistical difference in healing between one- and two-visit treatments; however, negative cultures at obturation produced 80% success compared with 44% when cultures were positive. In addition, 52% of teeth with positive cultures were classified as uncertain with regard to healing compared with 7% of the negative culture group.[206]

In a prospective clinical study, investigators evaluated the effect of calcium hydroxide as an interappointment dressing on the periapical healing of lesions associated with necrotic pulps in 73 patients. Thirty-six teeth were endodontically treated in one visit. Thirty-one teeth were treated in two visits with calcium hydroxide as an intracanal medicament. Periapical healing increased with the length of the observation period. In both treatment groups the success rate exceeded 90%, and no statistically significant difference existed between the two groups.[327]

Controlled laboratory studies support the use of calcium hydroxide as an antimicrobial agent before obturation of teeth with pulp necrosis. Two studies[156,157] evaluated periapical healing of infected root canals in dogs. After inducing periapical pathosis the teeth were treated by immediate obturation or with calcium hydroxide for 1 week before obturation. Results of the radiographic[156] examination at 6 months indicated complete healing was similar for the one-visit (35.3%) and calcium hydroxide (36.8%) groups. The calcium hydroxide group, however, had fewer failed cases (15.8% vs. 41.2%) and more improved cases (47.4% vs. 23.5%) when compared with the one-visit group.[157] In histologic evaluations the calcium hydroxide group had significantly less inflammation than the one-visit group. One study also evaluated periapical healing of infected teeth in dogs after immediate obturation or with prior calcium hydroxide treatment for 7 or 14 days. Results indicated both calcium hydroxide groups were superior to the one-visit group, with the 14-day calcium hydroxide group being superior to the 7-day group.[139]

Other investigators overinstrumented and overextended teeth 45 minutes after extraction. After the procedures, teeth exhibiting vital pulps and no periradicular disease were without bacteria. Teeth exhibiting periradicular pathosis before extraction demonstrated a high percentage of bacteria at the root apices. The organisms were found to remain firmly attached to resorptive lacunae, indicating the instrumentation/obturation procedures were unable to eliminate the organisms.[121]

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.

A few studies suggest that complete cleaning and shaping should be accomplished and calcium hydroxide placed as an antimicrobial and temporary obturant in necrotic cases that cannot be treated in one visit[290] because investigators noted that bacteria in instrumented, unfilled canals can multiply and reach their pretreatment numbers in 2 to 4 days.[50]

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 psychologic 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. Early studies identified the dentinocemental junction as the apical limit for obturation.[70,117,118,221,292] 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 7 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[174] noted that the apical anatomy consists of the major diameter of the foramen and the minor diameter of the constriction (Fig. 10-3), 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.[48] Still another investigator noted in 92% of examined teeth that the apical constriction was between 0.5 and 1 mm from the apex.[61] One 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[88]; 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.[30] 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.

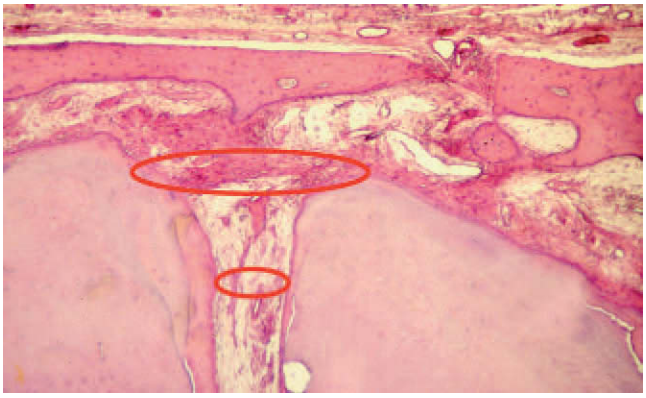


FIG. 10-3 Histologic section of a root apex, demonstrating anatomy of the classic foramen and constriction.

A later study found that no foramina coincided with the long axis of the root, with the distance ranging from 0.2 to 3.8 mm (Fig. 10-4).[121] Root resorption is an additional factor in length determination. Resorption is more common with necrosis and apical bone resorption, and this can result in loss of the constriction (Fig. 10-5).[154,195] On the basis of these findings it appears that canals filled to the radiographic apex reflect an overextension of the obturating material.

A study by the Toronto group[99] on the prognosis of retreatment identified perforation, 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 retreated could be cleaned and sealed. The success rate for negotiating the apical unfilled canal was 74%.

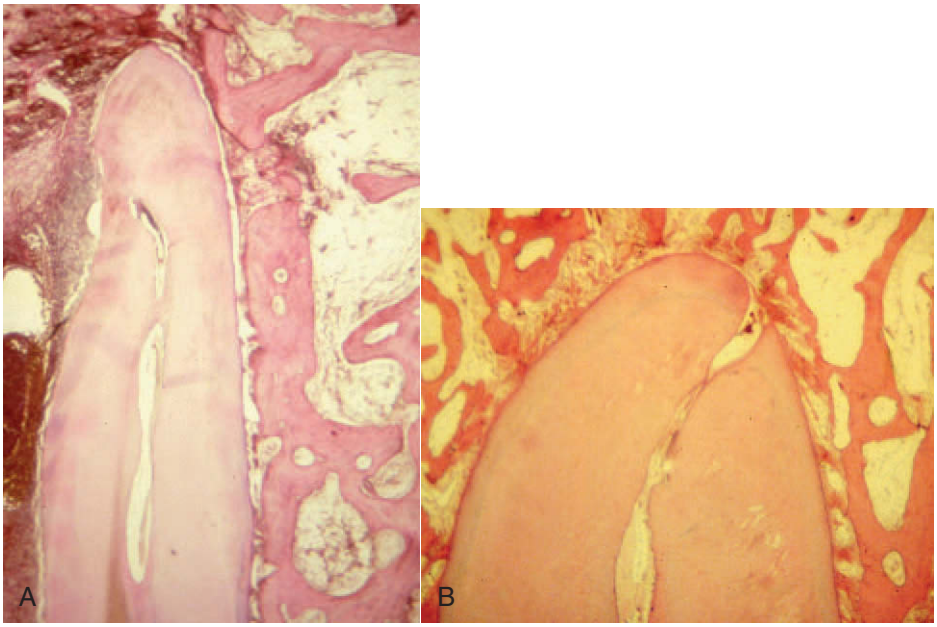


FIG. 10-4 Histologic sections demonstrating the foramen exiting short of the root apex.

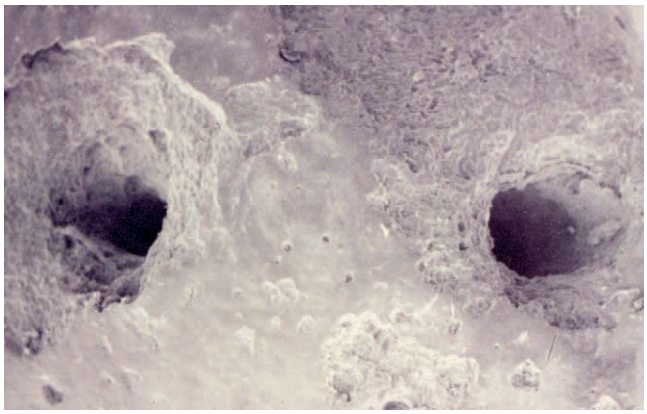


FIG. 10-5 Scanning electron microscopy of a tooth exhibiting a necrotic pulp and apical pathosis and resorption.

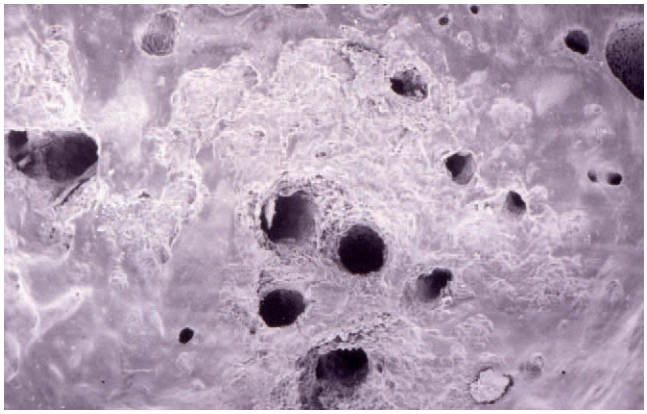


FIG. 10-6 Scanning electron microscopy of the apex of an extracted tooth that was removed because of pulp necrosis. Note the multiple accessory foramina and resorption.

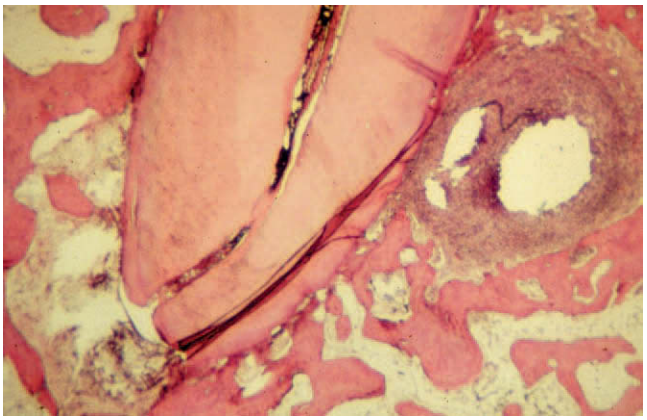


FIG. 10-7 Histologic section of a mesial root of a mandibular molar with a lateral canal present and associated lesion. Will the lesion resolve after the removal of the main canal contents, or will the lesion persist because of necrotic pulpal remnants in the lateral canal? The question remains unanswered.



FIG. 10-8 Posttreatment radiograph of a mandibular right first molar with a lateral canal associated with the distal root.

Controversy also exists regarding the role accessory canals play in success and failure (Fig. 10-6). For example, one scanning electron microscopy (SEM) study of the apical anatomy of each tooth group except the third molars noted no pattern for foraminal openings[120]; the number of accessory canals ranged from 1 to 16. Although lateral canals can be associated with pathosis, one study indicates that accessory canals are common but play little role in periradicular pathosis (Fig. 10-7).[17]

Further, other studies demonstrated that it is not possible to completely débride the canal space regardless of the technique or irrigant.[177,326] Investigators[329] noted that these anatomic structures are only demonstrated. Often, smaller apical accessory canals remain unfilled or partially filled.[322] Accessory/lateral canals are often obturated by chance and only serendipitously identified on the posttreatment radiograph (Fig. 10-8).

Investigators compared obturation of lateral canals, using six obturation techniques in resin blocks.[85] All techniques were able to obturate lateral canals with sealer. Warm vertical compaction, carrier-based thermoplastic gutta-percha, continuous wave compaction, and vertically compacted high-temperature gutta-percha filled lateral canals with gutta-percha significantly better than lateral compaction or warm lateral compaction. The use of sealer enhanced the ability of the gutta-percha to obturate the lateral canals.[85]

The importance of length control in obturation relates to extrusion of materials. Studies indicate that extrusion decreases the prognosis for complete regeneration.[27,105,271,272,291,293,301] One study evaluated the quality of root canal treatment in an American population.[47] Periapical disease was evident in 4.1% of all teeth and 31.3% of root-filled teeth, and the study noted that a periapical pathosis was found with 43% of the teeth with overfills. In another study of 1000 cases, investigators found that overfilling resulted in a failure rate of 37%. This was four times greater than for cases filled short.[301] A third study found that, in necrotic cases, better success was achieved when the procedures terminated at or within 2 mm of the radiographic apex.[348] Obturation shorter than 2 mm from the apex or past the apex resulted in a 20% lower success rate. For vital cases, termination between 2 and 3 mm was acceptable. Other investigators found that teeth obturated less than 2 mm from the apex had a higher success rate when compared with cases obturated more than 2 mm from the apex.[229]

On the basis of biologic and clinical principles, instrumentation and obturation should not extend beyond the apical foramen. This was demonstrated in one study that histologically evaluated 41 human root-filled teeth from 36 patients.[248] 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 empiric. 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. 10-9),[22,198,227] as shown in a study noting that the smear layer is superficial, with a thickness of 1 to 5 μm,[198] and this superficial debris can be packed into the dentinal tubules to various distances.[1]

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.[273] Another study noted that capillary action and fluid dynamics play a role in packing debris into the tubules[3] and yet another investigation noted a mean penetration of 479 μm after a 28-day incubation period.[232]

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.[84]

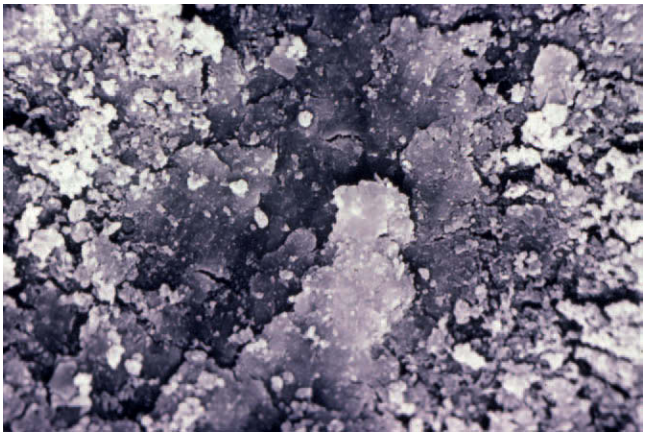


FIG. 10-9 Scanning electron microscopy of a prepared canal wall. The tubules are covered with a smear layer of organic and inorganic material.

The smear layer may also interfere with adhesion and penetration of sealers into dentinal tubules.[335] Evidence indicates that sealer penetration into dentinal tubules does not occur when the smear layer is present.[106,226] 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, OH), and Sealapex (SybronEndo, Orange, CA) to penetrate to between 35 and 80 μm, whereas the presence of the smear layer obstructed tubular penetration of all sealers.[171] Further, other studies found that smear layer removal increased bond strength and reduced microleakage in teeth obturated with AH-26 (DENTSPLY Maillefer, Ballaigues, Switzerland).[91,108] 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.[310]

There does not appear to be a consensus on removing the smear layer before obturation.[59,274,282] The advantages and disadvantages of the smear layer remain controversial; however, *growing evidence supports removal of the smear layer before obturation*.[143,282] The organic debris present in the smear layer might constitute a substrate for bacterial growth,[227] and it has been suggested that the smear layer prohibits sealer contact with the canal wall and permits leakage.[25] 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.[227] 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.[40] Removal of the smear layer introduces the possibility of reinfecting the dentinal tubules if leakage occurs.[274] However, one study demonstrated that bacteria present before obturation are not viable after obturation.[81]

The smear layer may also interfere with the action of irrigants used as disinfectants.[222] 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.[274]

The smear layer may interfere with the adhesion and penetration of root canal sealers. It also may prevent gutta-percha penetration during thermoplastic techniques.[123] Significant tubular penetration of gutta-percha and sealers has been shown with thermoplasticized obturations[123] and with dentin-bonded composite resins.[181] Removal of the smear layer also enhances the adhesion of sealers to dentin and tubular penetration.[181,219,274,333] Root canal filling materials adapt better to the canal walls after smear layer removal.[82,219,333-335]

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.[168] Another study found that removal of the smear layer reduced both coronal and apical leakage regardless of the sealer tested.[66]

Another study examined the smear layer and the passage of bacteria through and around obturating materials,[64] 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.

One study reported no difference in the apical and middle thirds of canals irrigated with 17% ethylenediaminetetraacetic acid (EDTA), using either the traditional syringe or a newer irrigation device, the Quantec-E irrigation system (SybronEndo).[276]

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.[72] Another investigator also demonstrated smear layer removal with ultrasonication and NaOCl.[54] 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.[254] 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.[21,325]

A new method for removing the smear layer employs the use of a mixture of a tetracycline isomer, an acid, and a detergent (MTAD) (BioPure; DENTSPLY Tulsa Dental Specialties, Tulsa, OK) as a final rinse to remove the smear layer.[312] MTAD removed most of the smear layer; however, some organic components of the smear layer remained on the surface of the root canal walls. The effectiveness of MTAD in completely removing the smear layer was enhanced when low concentrations of NaOCl were used as an intracanal irrigant before the use of MTAD as the final rinse. Further studies demonstrated that MTAD was superior to NaOCl in antimicrobial action.[277,278] Another study showed that MTAD was effective in killing *Enterococcus faecalis* at 200-fold dilution, which was more potent than NaOCl because it ceased being active when diluted 32-fold; EDTA had no antimicrobial activity.[313] One investigator found MTAD to be less toxic than eugenol, 3% H_2O_2, $Ca(OH)_2$ paste, 5.25% NaOCl, chlorhexidine gluconate (Peridex), and EDTA.[354] Other investigators found no significant difference in flexural strength and modulus of elasticity in dentin bars exposed to MTAD, indicating no alteration in the physical properties of the dentin, and noted that teeth treated with the MTAD protocol for clinical use (20 min of 1.3% NaOCl/5 min of MTAD) may not need any additional dentin conditioning before the application of bonding agents.[193]

After the completion of cleaning and shaping procedures, removal of the smear layer is generally accomplished by irrigating the canal with 17% disodium EDTA and 5.25% NaOCl (Fig. 10-10).[22] Chelators remove the inorganic components, leaving the organic tissue elements intact. NaOCl is necessary for removal of the remaining organic components. Fifty percent citric acid has also been shown to be an effective method for removing the smear layer,[20,131] as has tetracycline.[16,131]



FIG. 10-10 Scanning electron microscopy of the canal wall after removal of the smear layer with 17% EDTA and 5.25% sodium hypochlorite.

Chelating agents were introduced to endodontic treatment by Nygaard-Ostby in 1957 for treatment of calcified narrow root canals.[216] EDTA 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%.[143] 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.[323] The effectiveness of EDTA is related to time of application, the pH, and the concentration.[208,216]

Demineralization results in increased dentin permeability[119] 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.[141] The action of chelators and acids appears to be more effective in the coronal and middle thirds of the root and is reduced apically.[143,188] This reduced activity may be a reflection of canal size.[172] 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,[204] 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.[204]

EDTA appears to be biocompatible when used clinically[216]; however, irreversible decalcification of periapical bone and neuroimmunologic disturbances have been noted.[269] Extrusion of both NaOCl and EDTA in clinical treatment should be avoided.[130,228,306]

The recommended time for removal of the smear layer is 1 to 5 minutes.[53,143,264] 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.[53]

The use of EDTA in combination with NaOCl is recommended[300,307] and may enhance the cleaning[188,349] and antimicrobial effects of these solutions when compared with using them alone.[49]

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*[96] outlines contemporary endodontic treatment. 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. 10-11). Chapter 11 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 restoration or definitive (Fig. 10-12).

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,[353] the overlying bony anatomy, radiographic angulation, and the limited two-dimensional view of the obturated root canal or canals.

An often overlooked aspect in the assessment of root canal obturation is the density of the apical portion of the fill.[122] 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

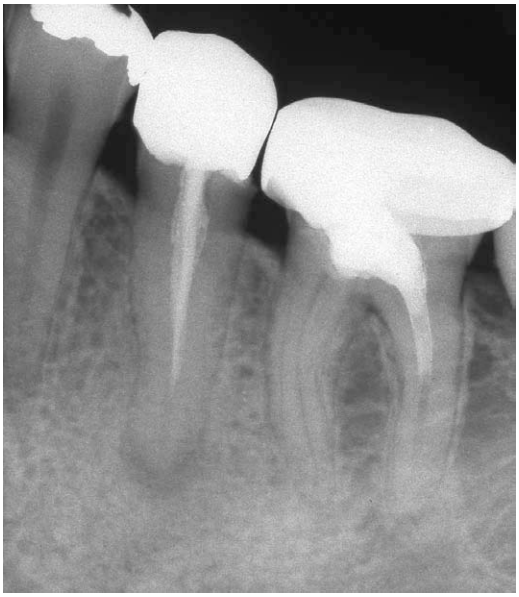


FIG. 10-11 A periapical radiograph of a mandibular left second premolar and first molar, demonstrating Sargenti paste root canal treatment. In addition to the toxic material, the technique often accompanies inadequate cleaning and shaping procedures.

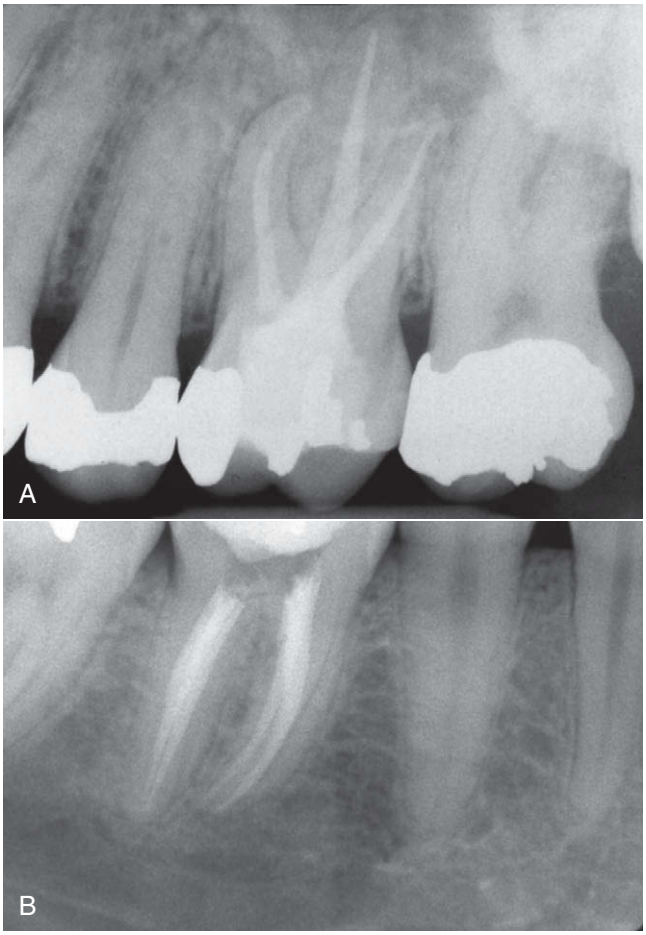


FIG. 10-12 A, Posttreatment radiograph of a maxillary right first molar, demonstrating adequate length, density, and taper. **B,** Posttreatment radiograph of a mandibular right first molar with an adequate obturation.

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.[243,305] 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.

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[115] outlined the properties of an ideal sealer (Box 10-1). At present no sealer satisfies all the criteria.

Sealers should be biocompatible and well tolerated by the periradicular tissues.[297] All sealers exhibit toxicity when freshly mixed; however, their toxicity is greatly reduced on setting.[176] Sealers are resorbable when exposed to tissues and tissue fluids.[13] Tissue healing and repair generally appear unaffected by most sealers, provided there are no adverse breakdown products of the sealer over time.[35,41-43,45] Breakdown products from the sealers may have an adverse effect on the proliferative capability of periradicular cell populations.[113] As a result, sealers should not be placed routinely in the periradicular tissues as part of an obturation technique.[176] Although an osteogenic response has been observed,[138,295,303,316] the ability of these sealers to sustain a high pH over time has been questioned.[170]

The most popular sealers are zinc oxide–eugenol formulations, calcium hydroxide sealers, glass ionomers, and resins. 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. 10-13).

BOX 10-1

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

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 absorb if extruded into the periradicular tissues.[13] They exhibit a slow setting time,[6] shrinkage on setting, solubility,[158,235] and they can stain tooth structure.[75,320] An advantage to this sealer group is antimicrobial activity.[4,15,133,203]

An early zinc oxide–eugenol sealer was introduced by Rickert and Dixon.[245] 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, PA) 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[116] modified the formulation and introduced a nonstaining formula in 1958 (Box 10-2). 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, IL) 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.

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. 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 R 972. Apexit and Apexit Plus (Ivoclar Vivadent, Schaan, Liechtenstein) consist of an activator (disalicylate, bismuth

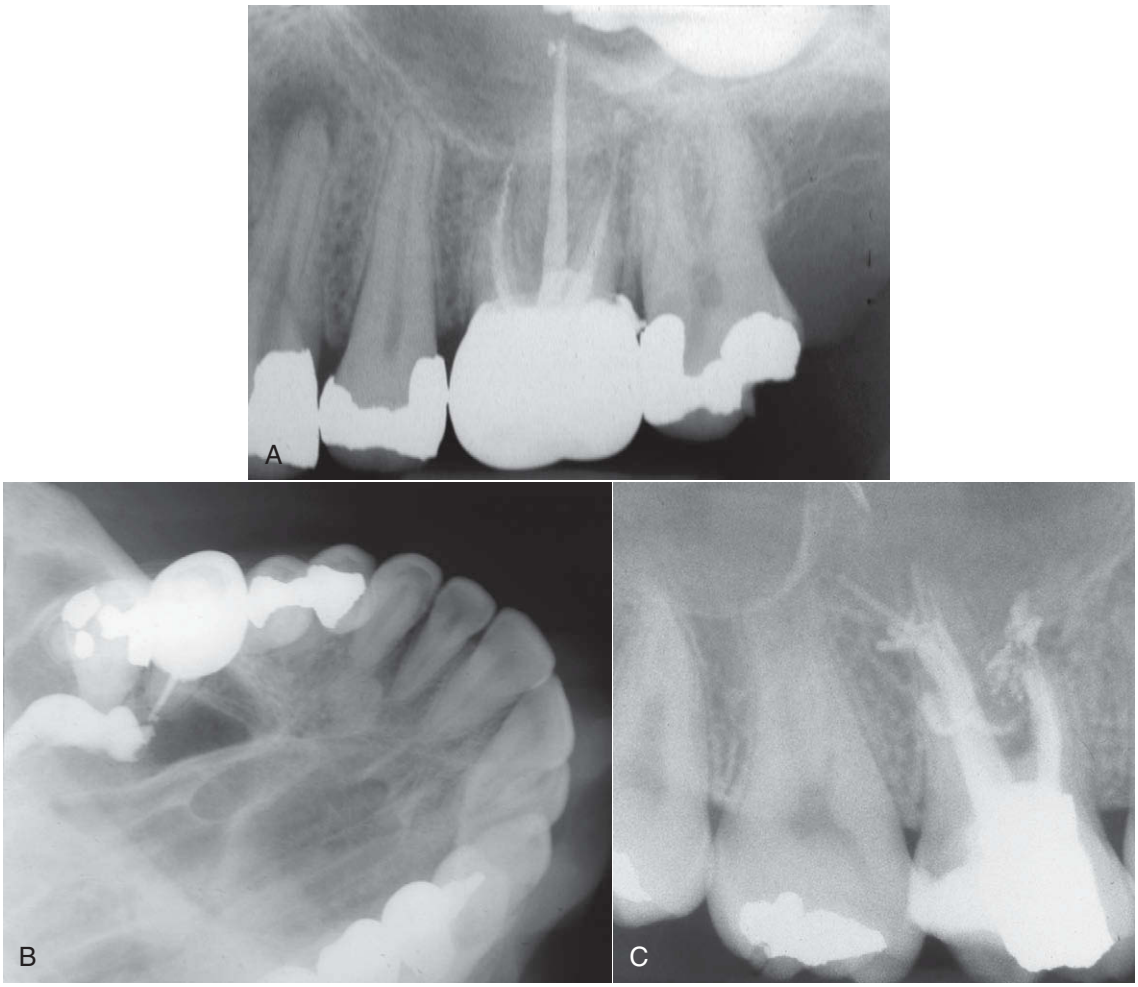


FIG. 10-13 A, Extrusion of sealer evident on the posttreatment radiograph of a maxillary first molar. The separated lentulo spiral in the mesiobuccal root indicates a possible method of sealer placement. **B,** Maxillary occlusal film demonstrates that the sealer is located in the maxillary sinus. Correction by nonsurgical techniques is not possible. **C,** Maxillary right first molar with extrusion of the sealer and gutta-percha.

BOX 10-2

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

hydroxide/bismuth carbonate, and fillers) and a base (calcium hydroxide, hydrated colophonium, and fillers).

Noneugenol Sealers

Developed from a periodontal dressing, Nogenol (GC America, Alsip, IL) is a root canal sealer without the irritating effects of eugenol. The base contains zinc oxide, barium sulfate, and bismuth oxychloride.

Glass Ionomer Sealers

The glass ionomers have been advocated for use in obturation because of their dentin-bonding properties. Ketac-Endo (3M ESPE, Minneapolis, MN) enables adhesion between the material and the canal wall.[105] It is also difficult to properly treat the dentinal walls in the apical and middle thirds with preparatory bonding agents to receive the glass ionomer sealer. A disadvantage of glass ionomers is that they must be removed if retreatment is required.[189] This sealer has minimal antimicrobial activity.[133]

Activ GP (Brasseler USA, Savannah, GA) consists of a glass ionomer–impregnated gutta-percha cone with a glass ionomer external coating and a glass ionomer sealer (Fig. 10-14). Available in .04 and .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.[103]



FIG. 10-14 Activ GP (Brasseler USA, Savannah, GA) glass ionomer–coated gutta-percha points and sealer.



FIG. 10-15 AH Plus sealer is a resin formulation. *(Courtesy DENTSPLY, Konstanz, Germany. All rights owned by and used with permission from DENTSPLY International, Inc.)*

Resin

Resin sealers have a long history of use, provide adhesion, and do not contain eugenol. AH-26 is a slow-setting epoxy resin that was found to release formaldehyde when setting.[167,298] AH Plus is a modified formulation of AH-26 in which formaldehyde is not released (Fig. 10-15).[182] The sealing abilities of AH-26 and AH Plus appear comparable.[79] AH Plus is an epoxy-bis-phenol resin that comes in two tubes. It exhibits a working time of approximately 4 hours.

EndoREZ (Ultradent Products, South Jordon, UT) is a methacrylate resin with hydrophilic properties. When used with EndoREZ resin-coated gutta-percha cones the dual cure EndoREZ sealer bonds to both the canal walls and the core material.

Diaket, a polyvinyl resin (3M ESPE), consists of a powder composed of bismuth phosphate and zinc oxide and a liquid consisting of dichlorophen, triethanolamine, propionylacetophenone, and copolymers of vinyl acetate, vinyl chloride, and vinylisobutyl ether. The material appears to be biocompatible.[212]



FIG. 10-16 GuttaFlow trituration capsule and injection syringe (Coltène/Whaledent, Cuyahoga Falls, OH).

Other resin-based sealers, Epiphany (Pentron Clinical Technologies, Wallingford, CT) and RealSeal (SybronEndo), have been introduced for use with a new core material, Resilon (Pentron Clinical Technologies). Advocates of these sealers propose that they bond to the canal wall and to the core material to create a "monoblock." One study indicated that the bond strength to dentin can be influenced by the irrigant used. Water and chlorhexidine decreased the bond strength compared with NaOCl, NaOCl/EDTA, and NaOCl/MTAD. The use of EDTA and MTAD did not improve the bond strength compared with NaOCl alone.[324]

Silicone Sealers

RoekoSeal (Coltène/Whaledent, Germany) is a polyvinylsiloxane that has been reported to expand slightly on setting.

GuttaFlow (Coltène/Whaledent) is a cold flowable matrix that is triturated. It consists of gutta-percha added to RoekoSeal (Fig. 10-16). 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[355] and is biocompatible,[36,95] but the setting time is inconsistent and may be delayed by final irrigation with sodium hypochlorite.[36] Sealing ability appears comparable to other techniques in some studies and inferior in others.[38,169,207,225]

Bioceramic

Bioceramic (BC) sealer is composed of zirconium oxide, calcium silicates, calcium phosphate monobasic, calcium hydroxide, and various filling and thickening agents. The material is available in a premixed syringe with calibrated intracanal tips. As a hydrophilic sealer it utilizes moisture within the canal to complete the setting reaction and it does not shrink on setting. It is biocompatible and exhibits antimicrobial properties during the setting reaction. The manufacturer advocates expressing the sealer into the coronal one third to one half of the canal and then seating the master gutta-percha cone.

Medicated Sealers

Sealers containing paraformaldehyde are strongly contraindicated in endodontic treatment (Fig. 10-17). 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[10] and are unacceptable under any circumstances in clinical treatment because of the severe and permanent toxic effects on periradicular tissues.[275] A paste containing 6.5% paraformaldehyde as well as lead and mercury was advocated for use by Sargenti[258-260] and originally marketed as N-2. Lead has been reported in distant organ systems when N-2 is placed within the radicular space.[223] In another study the investigators reported the same results regarding systemic distribution of the paraformaldehyde component of N-2.[31] Removal of the heavy metals resulted in a new formulation: RC2B. Other 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.[67,213]

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.[97,165] One clinician reported irreversible neurotoxicity, manifested as dysesthesia, in cases where paraformaldehyde pastes were forced through the apical foramen into the periapical tissues.[165] The reader is referred to Chapter 11 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. 10-18), an ultrasonic file, and coating the master gutta-percha cone.[336] Placement did not differ with the various techniques; however, the investigators noted the most variation in sealer coating was in the apical area.[336] 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.[125] Other investigators found that ultrasonics produced the best sealer distribution when used circumferentially.[299] These findings were supported by another study that found ultrasonic placement to be superior to manual techniques.[1]

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.[343] Another well-controlled study reported that none of five obturation techniques evaluated resulted in uniform sealer distribution along the entire length of the core obturation material.[142] Evidence indicates that the method of obturation affects the sealer penetration into tubules. This was exemplified by a study finding that thermoplastic techniques produced deeper sealer penetration into tubules.[78] Removal of the smear layer enhances sealer penetration into the dentinal tubules.[77]

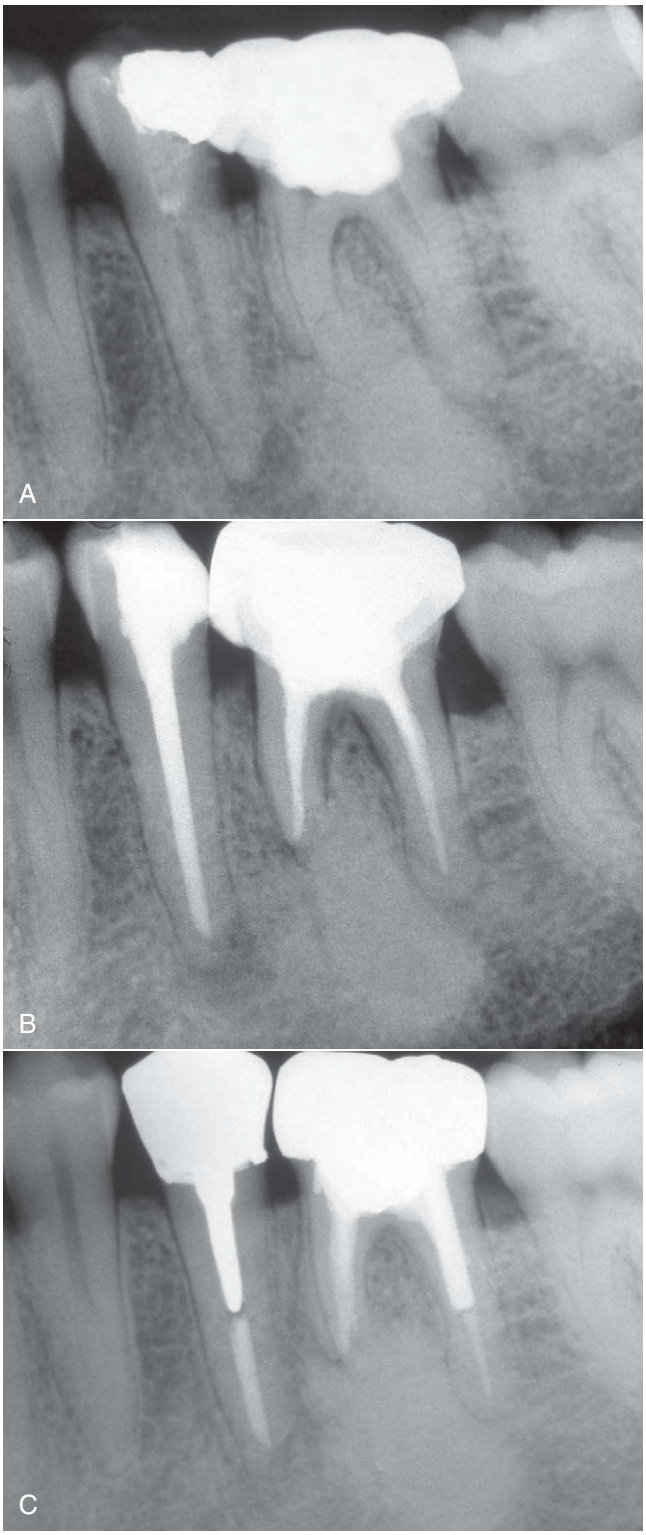


FIG. 10-17 Patient treated with Sargenti paste in her mandibular left second premolar and first molar. **A,** Pretreatment radiograph exhibits an osteolytic response associated with the premolar and a proliferative response associated with the molar. **B,** Posttreatment radiograph of the teeth. **C,** One year follow-up radiograph exhibiting osseous regeneration apical to the second premolar and appropriate restorative treatment.

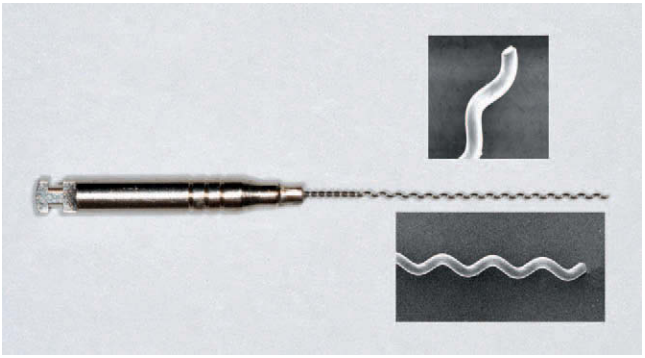


FIG. 10-18 Lentulo spiral used for sealer placement during obturation.

BOX 10-3

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

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 provide for an impervious seal of the canal system; all materials leak to some extent.[2] The choice of obturation techniques depends on the unique circumstances each case provides.

The properties of an ideal obturation material were outlined by Grossman[115] (Box 10-3). 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 was the silver cone.

Silver Cones

Jasper[148] introduced cones made of silver, which he claimed produced the same success rate as gutta-percha and were easier to use. The rigidity provided by the silver cones made them easy to place and permitted more predictable length control; however, their inability to fill the irregularly shaped root canal system permitted leakage (Fig. 10-19). When silver points contact tissue fluids or saliva, they corrode.[39] The corrosion products have been found to be cytotoxic and produced pathosis or impeded periapical healing.[271]

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 issue, the reader is referred to Chapter 11.

Gutta-Percha

Gutta-percha is the most popular core material used for obturation. Major advantages to 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 on cooling. Gutta-percha is the *trans* isomer of polyisoprene (rubber) and exists in two crystalline forms (α and β).[112] 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.[267]

Gutta-percha cones consist of approximately 20% gutta-percha, 65% zinc oxide, 10% radiopacifiers, and 5% plasticizers.[104] Attempts have been made to make gutta-percha more antimicrobial by the addition of materials such as iodoform,[63] calcium hydroxide,[190] chlorhexidene,[192] and tetracycline.[201] The clinical effectiveness of adding these materials has not been demonstrated.

Unlike rubber, room temperature gutta-percha cannot be compressed 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 (conventional) sizes. The nonstandard nomenclature refers to the dimensions of the tip and body (Fig. 10-20). 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. 10-21 and 10-22). A size 40/04 has a tip of 0.4 mm and a taper of 0.04 mm per millimeter. Unfortunately uniformity in manufacturing is not present, and the actual cone size varies.[111,197]

Although the points cannot be heat sterilized, a study found that gutta-percha points can be sterilized before use by placing the cones in 5.25% NaOCl for 1 minute. This study also found that 2% glutaraldehyde, 2% chlorhexidine, and 70% ethyl alcohol were not effective in killing *Bacillus subtilis* spores.[287]



FIG. 10-19 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.

Activ GP

Activ GP (Brasseler USA) consists of gutta-percha cones impregnated on the external surface with glass ionomer (see Fig. 10-14). Single cones are used with a glass ionomer sealer. Available in .04 and .06 tapered cones, the sizes are laser verified to 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.[103]

Resilon

The resin-based obturation systems Epiphany (Pentron Clinical Technologies), RealSeal (SybronEndo), and Resinate (Obtura Spartan, Earth City, MO) have been introduced as alternatives to gutta-percha (Figs. 10-23 and 10-24). Resilon is a high-performance industrial polyurethane 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. 10-25). With traditional techniques there is a gutta-percha–sealer interface and a tooth–sealer

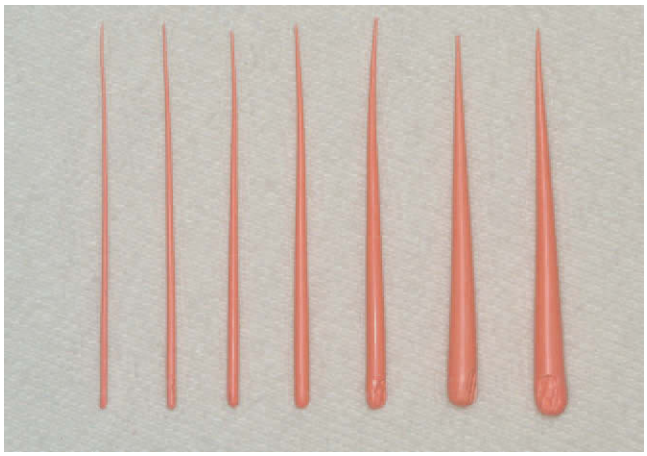


FIG. 10-20 Nonstandard gutta-percha cones: extra fine, fine fine, fine, medium fine, fine medium, medium, large, and extra large.

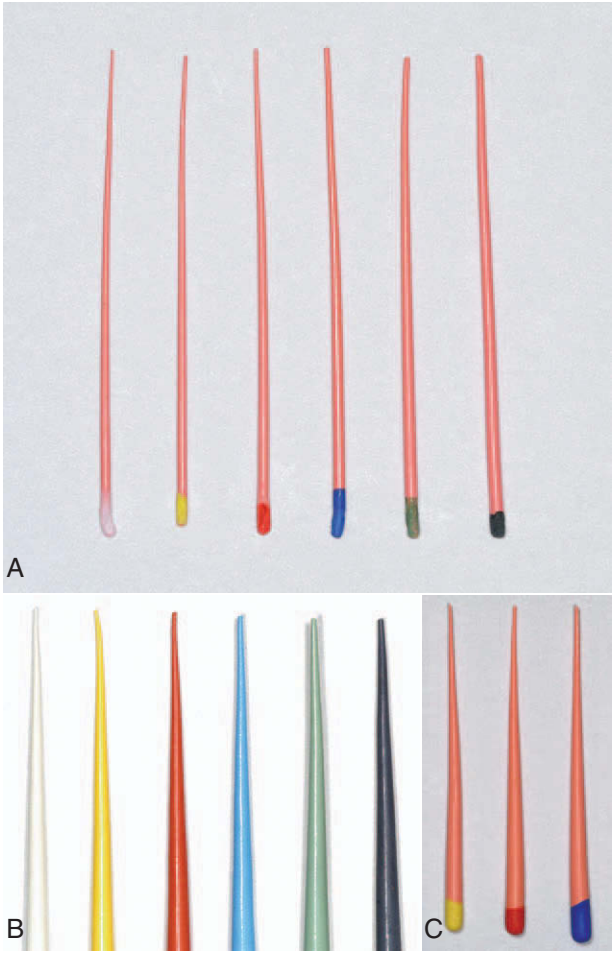


FIG. 10-21 A, Standard gutta-percha cone sizes #15 to #40. **B,** Standard cones #.06, taper sizes #15 to #40. **C,** Standard cones Protaper F1, F2, F3.

interface. With Resilon the resin sealer bonds to both the canal wall and the cone. Whether a monoblock is achievable remains controversial.[242,308] An in-depth review article on the subject of monoblocks indicates that with current materials and techniques, the monoblock has yet to be achieved.[308]

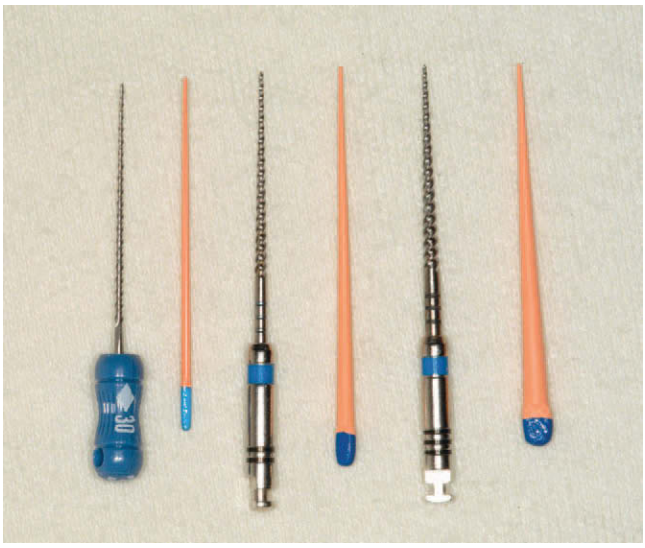


FIG. 10-22 Size #30 standard gutta-percha points exhibiting #.02, #.04, and #.06 tapers.



FIG. 10-23 Epiphany system (Pentron Clinical Technologies, Wallingford, CT) with the primer, thinning resin, sealant, and standard Resilon points. *(Courtesy SybronEndo, Orange, CA.)*

The system resembles gutta-percha and can be placed by lateral compaction, warm lateral or vertical compaction, or thermoplastic injection. 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. 10-24).

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

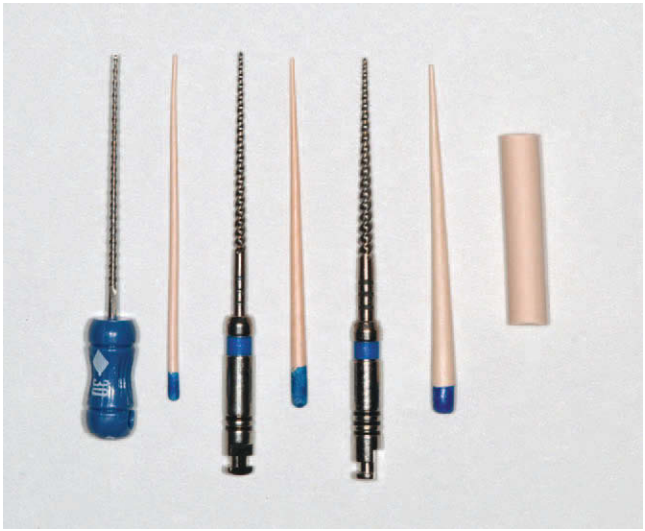


FIG. 10-24 Resilon #.02, #.04, and #.06 tapered points and a thermoplastic plug for use in the Obtura II system (Obtura Spartan, Earth City, MO).

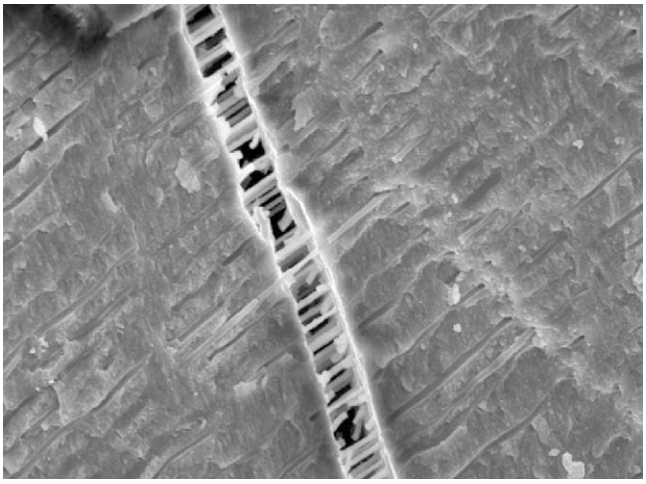


FIG. 10-25 Scanning electron microscopy view of Resilon tags extending into the dentinal tubules.

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.[19] 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.[283] 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 4 of 21 roots (19%).[284] Still another study demonstrated that teeth filled with Resilon were more resistant to fracture than roots filled with gutta-percha and AH-26 sealer.[311] More recent evidence suggests that Resilon does not strengthen roots.[114,338]

Resilon appears to be biocompatible. Implantation in the subcutaneous tissues of rats demonstrated fibrous encapsulation and negligible inflammation at 60 days.[34]

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.[71] 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.[69]

Custom Cones

When the apical foramen is open or a canal is large a custom cone may need to be fabricated (Fig. 10-26). This permits the adaptation of the cone to the canal walls, reduces the potential for extrusion of the core material, and may improve the seal.[24,160] 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. Only the outer superficial portion of the cone is softened. The central core of the cone should remain semirigid. The cone is then placed into the canal and gently tamped to 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.[161]

Large canals may necessitate fabricating a large master cone before canal adaptation. This can be accomplished by heating

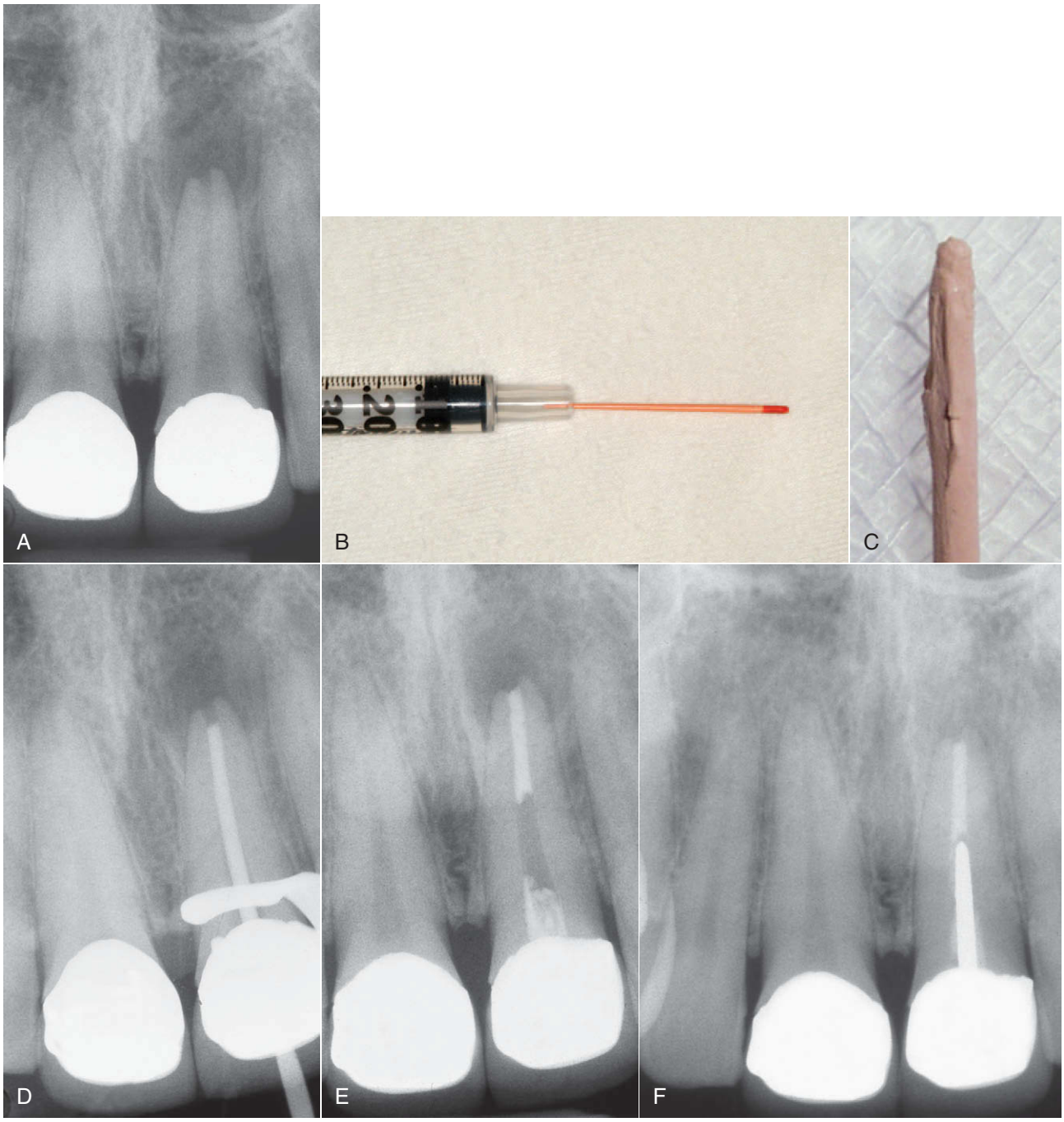


FIG. 10-26 Apical root resorption often results in an open apex requiring fabrication of a custom cone. **A,** Pretreatment radiograph of the maxillary left central incisor with pulp necrosis and chronic apical periodontitis. Apical root resorption is present. **B,** In fabricating a custom master cone 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.

several large gutta-percha cones and rolling the mass between two glass slabs until an appropriate size is obtained (Fig. 10-27). 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 and the influence of treatment technique on success/failure has yet to be determined.[11,215] The prospective Toronto studies have suggested that warm vertical compaction may be superior to lateral compaction; however, definitive evidence is lacking.[76,231]

Lateral Compaction

Lateral compaction is a common method for obturation (Fig. 10-28).[51] The technique can be used in most clinical situations

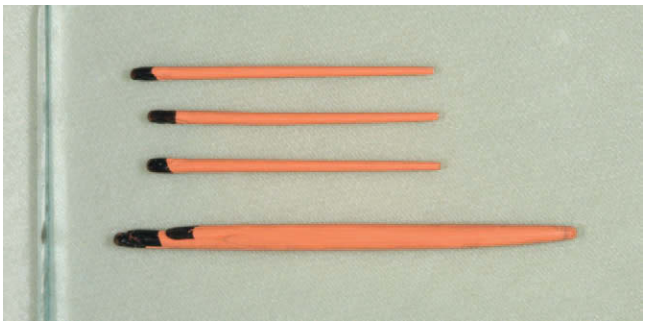


FIG. 10-27 For large canals, several gutta-percha points can be heated and rolled together, using a spatula or two glass slabs.

and provides for length control during compaction.[109] A disadvantage is that the technique may not fill canal irregularities[347] as well as warm vertical compaction or thermoplastic techniques.[345] The procedure can be accomplished with any of the acceptable sealers.

After canal 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 conventional cones and will permit deeper spreader penetration.[233] An alternative is to adapt an appropriately tapered nonstandard cone by cutting small increments from the tip. This "master cone" is measured and grasped with forceps so that the distance from the cone tip to 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 1.0 to 2.0 mm from working length. Appropriate accessory points are also selected to closely match the size of the spreader. The correlation between spreader size and nonstandard cones is variable,[44,357] 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.[135,321]

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.[74,183,184] 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.[238] Spreaders made from nickel–titanium are available and provide increased flexibility,[28] reduce stress,[89] and provide deeper penetration when compared with stainless steel instruments.[152,268,339] 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 establishing a seal and spreader penetration.[7,281]

After placement the spreader is removed by rotating it back and forth as it is withdrawn. An accessory cone is placed in the space vacated by the instrument. 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 plugger. Only light pressure is required during lateral compaction because the gutta-percha is not compressible, and because as little as 1.5 kg of pressure is capable of fracturing the root (Fig. 10-29). In addition to the force applied, investigators have noted that removal of dentin during preparation is a significant factor in root fracture.[337]

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 hoped that the space between each of the cones is filled with sealer.

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.[351] In anterior teeth the desired level is the cementoenamel junction on the facial surface.

An alternative to lateral compaction with finger spreaders is ultrasonics.[18] For example, one study found that the technique produced adequate obturation and a 93% clinical success rate.[356]

Another study used ultrasonic-energized files in a warm lateral compaction technique and found that the amount of gutta-percha by weight increased by 33% with two applications of ultrasonics when compared with lateral compaction.[80] 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.[302]

Warm Vertical Compaction

Schilder[266] introduced warm vertical compaction as a method of filling the radicular space in three dimensions. Preparation requirements for the technique include preparing a canal 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. 10-30).

The technique involves fitting a master cone short of the corrected working length (0.5 to 2 mm) with resistance to displacement (Fig. 10-31). This ensures that the cone diameter is larger than the prepared canal. 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. The cone is placed in the canal and the coronal portion is removed with heat. A heated spreader or plugger is used to remove portions of the coronal gutta-percha and soften the remaining material in the canal. The Touch 'n

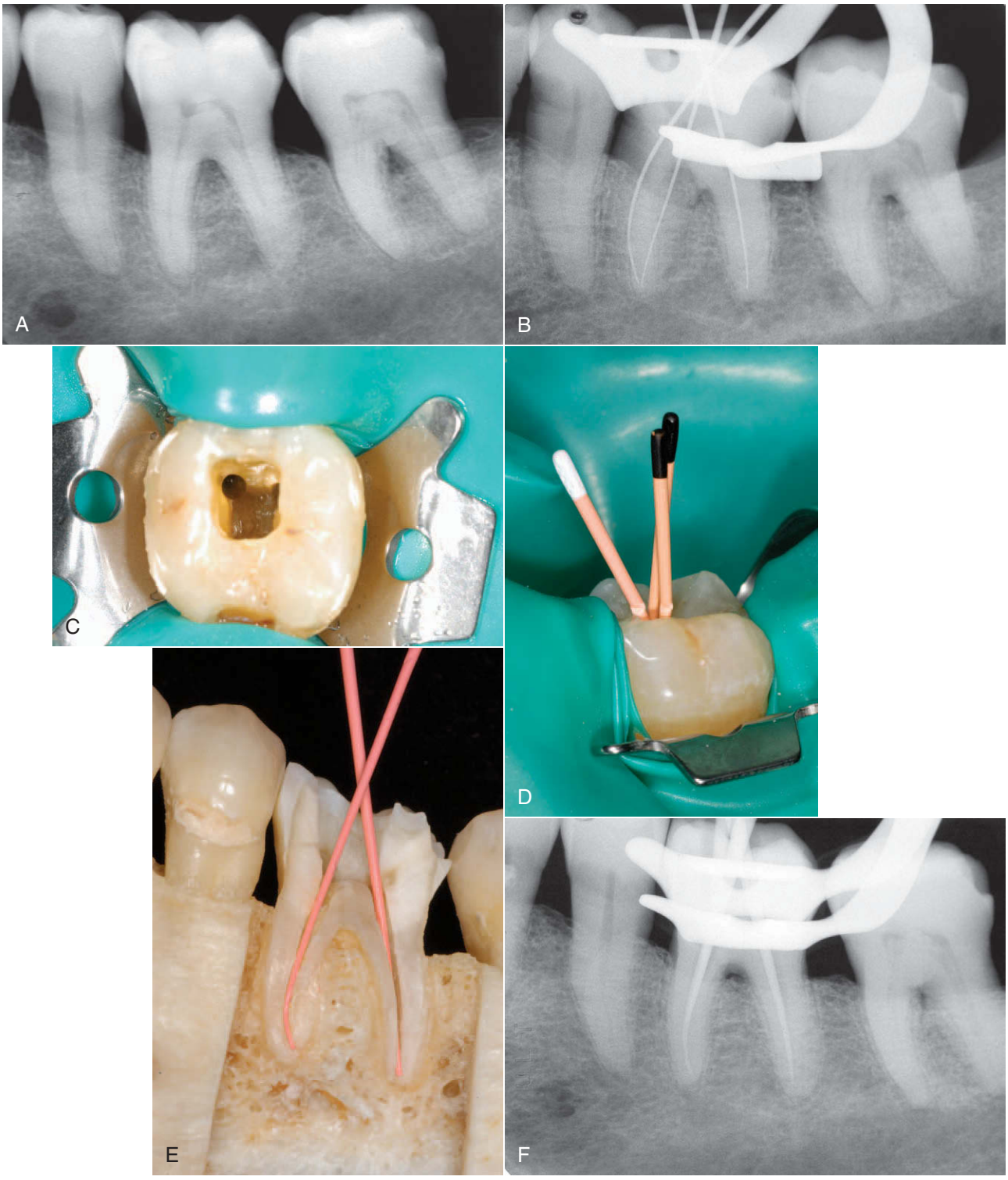


FIG. 10-28 Left first mandibular molar. **A,** Pretreatment radiograph. **B,** Working length radiograph. **C,** Coronal access opening, demonstrating the prepared mesiobuccal canal. **D,** Standardized master cones with coronal reference marked. **E,** Standard master cones fit to length as they exhibit minimal taper and permit deeper penetration of the spreader. **F,** Master cone radiograph.

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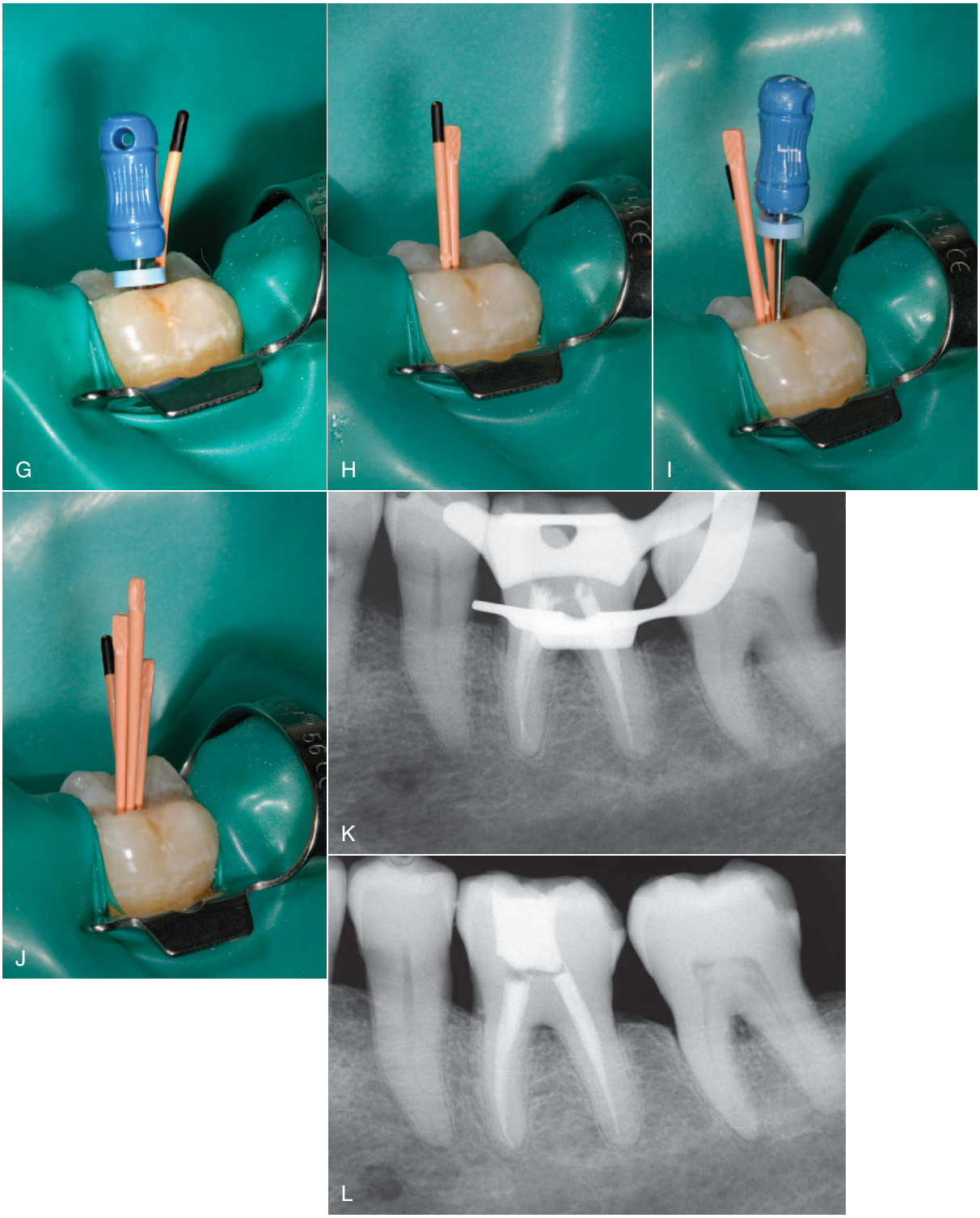


FIG. 10-28, cont'd G, Finger spreader in place. **H,** Fine-medium accessory cone placed in the space created by the spreader. **I,** Finger spreader placed in preparation, creating space for additional accessory cones. **J,** Additional cones are placed until the spreader does not penetrate past the 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.

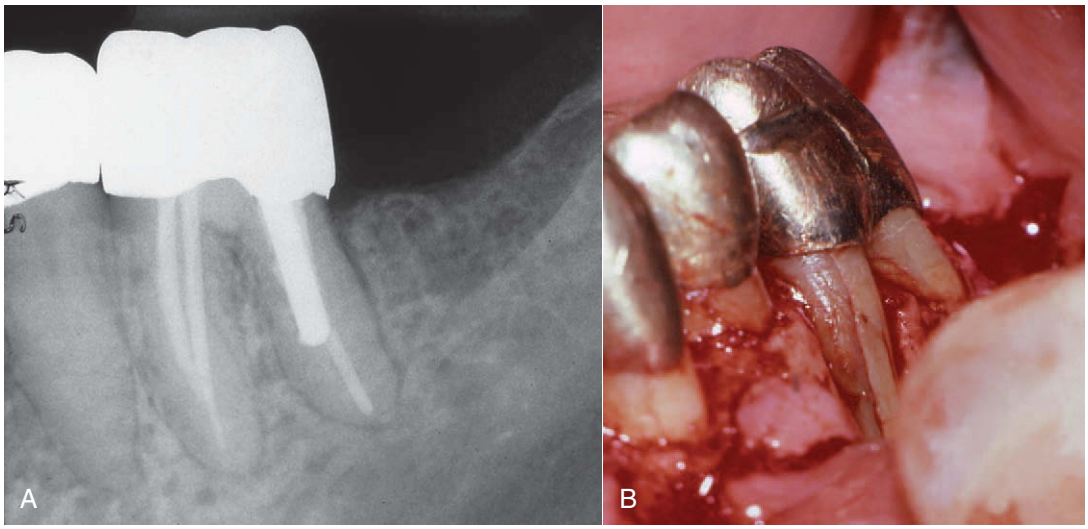


FIG. 10-29 Vertical root fractures can occur with excessive compaction forces. **A,** Follow-up radiograph of a mandibular left first molar. A deep isolated periodontal probing defect was associated with the buccal aspect of the mesiobuccal root. **B,** Flap reflection revealed a vertical root fracture.

Heat (SybronEndo) (Fig. 10-32), EI DownPak (EI/Hu-Friedy, Chicago, IL), and System B (SybronEndo) (Fig. 10-33) are alternatives to applying heat with a flame-heated instrument because they permit 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 filled. The coronal canal space is backfilled, using small 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.

One study[33] measured temperature changes in the canal with warm vertical compaction. 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 apex the maximal temperature 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 cementoenamel 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.[180]

The potential for vertical root fracture is also present with warm vertical compaction.[32] 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, filling irregularities and accessory canals better than lateral compaction.[85,342] 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.[341] However, thermoplasticized techniques result in more extrusion of obturation materials.[175] There appeared to be no consistent differences between the techniques in sealing the canal space.[342]

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 difficult in curved canals, where the rigid pluggers are unable to penetrate to the necessary depth. To allow the rigid carriers to penetrate within 4 to 5 mm of the apex, the canals must be enlarged and tapered more, in comparison with the lateral compaction technique; however, excessive removal of tooth structure weakens the root.

Continuous Wave Compaction Technique

A variation of warm vertical compaction is the continuous wave compaction technique.[46] The increasing use of nickel–titanium rotary preparation techniques and the fabrication of standard cones for files of greater taper have resulted in more clinicians using thermoplasticizing techniques. The manufacturing of 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 an electric heat carrier, the

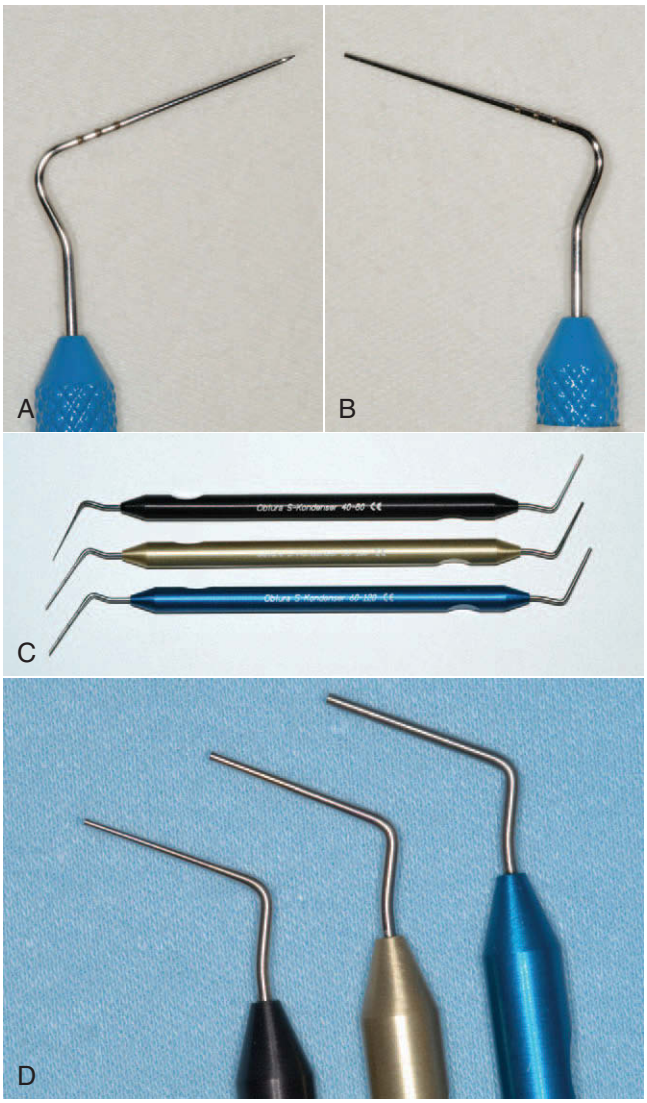


FIG. 10-30 Various pluggers are manufactured for compacting warm gutta-percha. **A,** ISO standard spreader. **B,** ISO standard plugger. **C,** Obtura S Kondensers (**C,** courtesy Obtura Spartan, Earth City, MO). **D,** Closeup of Obtura S-Kondensers.

System B unit, and #.04, #.06, #.08, #.10, and #.12 tapered stainless steel pluggers, each with a tip diameter of 0.5 mm (see Fig. 10-33). The #.06 tapered plugger approximates the fine nonstandard gutta-percha cone, the #.08 plugger the fine-medium cone, the #.10 plugger the medium cone, and the #.12 plugger the medium-large cone. Pluggers are consistent with the ProFile GT instruments (DENTSPLY Tulsa 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 the fine-medium plugger produced the highest mean internal temperature (74° C). The authors noted that the external temperature setting never exceeded the critical 10° C rise with any temperature setting or tip configuration.[302] 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.[322]

Another study reported that obturation temperature elevations produced during obturation with System B were significantly less ($P < 0.001$) than with warm vertical compaction. An elevation of external root surface temperature by more than 10° C was noted with vertical compaction.[285] 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.[200]

After selecting an appropriate master cone, a plugger is prefitted to fit within 5 to 7 mm of the canal length (Fig. 10-34). Placing the plugger deeper into the canal may enhance the flow of gutta-percha.[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 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.[37,153,345] Increasing the temperature settings does not seem to increase the effectiveness of obturation.[153]

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 the cold plugger against the gutta-percha in 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. 10-35). The heat is inactivated while firm pressure is maintained on the plugger for 5 to 10 seconds. After the gutta-percha mass has cooled a 1-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 is still present in the canal can be established with hand pluggers. 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.[265]

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.

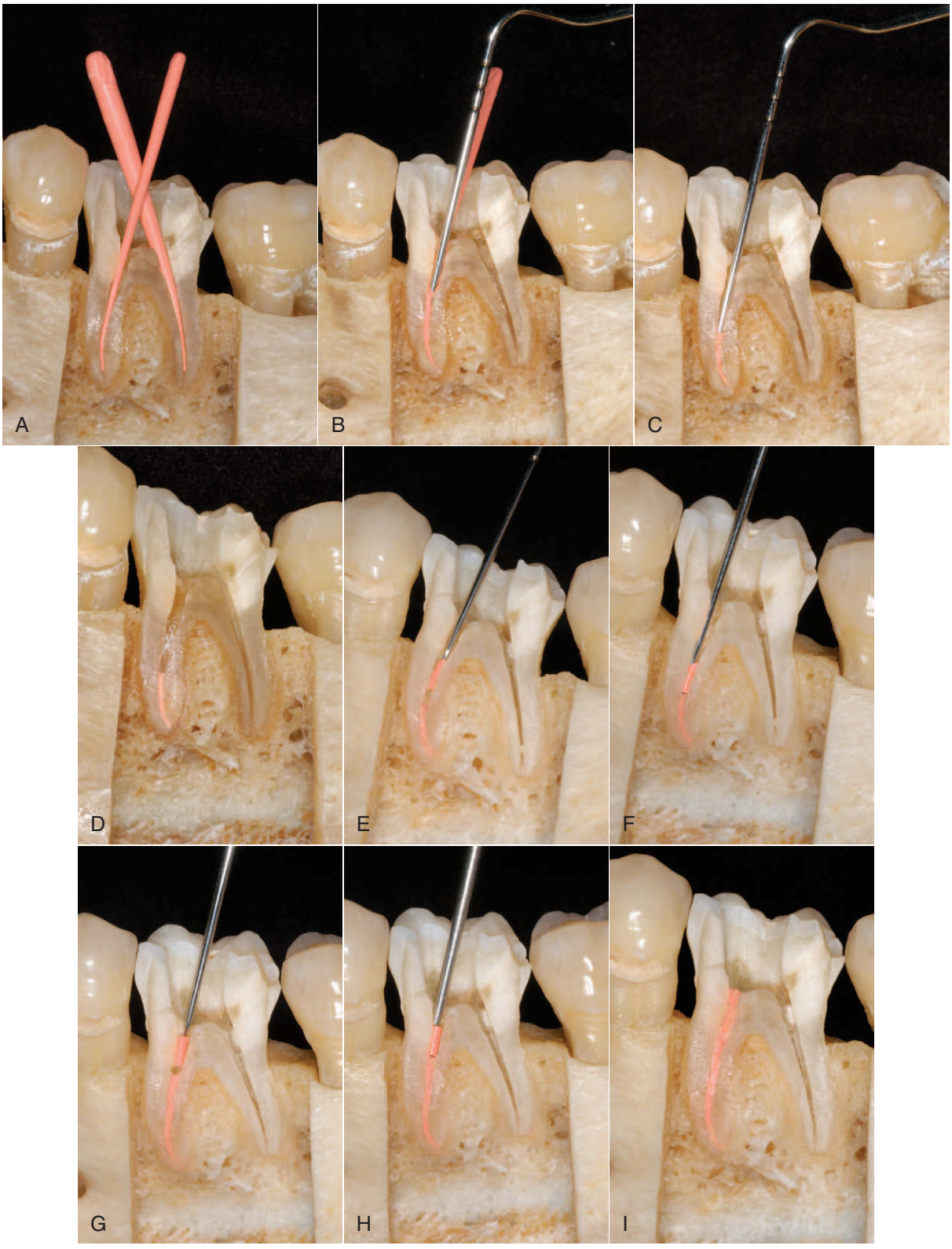


FIG. 10-31 Warm vertical compaction of gutta-percha employs heat and various condensers. **A,** Nonstandard cones are selected and fit short of the prepared length because they more closely replicate the prepared canal. **B,** Heated pluggers or spreaders are used to apply heat to the master cone and 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. 10-32 The Touch 'n Heat unit. *(Courtesy SybronEndo, Orange, CA.)*

Filling the space left by the plugger can be accomplished by a thermoplastic injection technique (Obtura II or Ultrafil 3D [Coltène/Whaledent], Calamus [DENTSPLY Tulsa Dental Specialties], Elements [SybronEndo, Orange, CA], or HotShot [Discus Dental, Culver City, CA]) (Fig. 10-36)[150] 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

Lateral compaction of gutta-percha provides for length control, which is an advantage over thermoplastic techniques. The Endotec II device (Medidenta) provides the clinician with the ability to employ length control while incorporating a warm gutta-percha technique (Fig. 10-37). Investigators demonstrated that the Endotec II produced a fusion of the gutta-percha into a solid homogeneous mass.[147] One study evaluated three thermoplasticized filling techniques and lateral compaction, using a bacterial metabolite model, and found the Endotec to be superior to lateral compaction alone, lateral thermocompaction, and the Ultrafil 3D.[162] The use of warm lateral compaction with the Endotec demonstrated an increased weight of



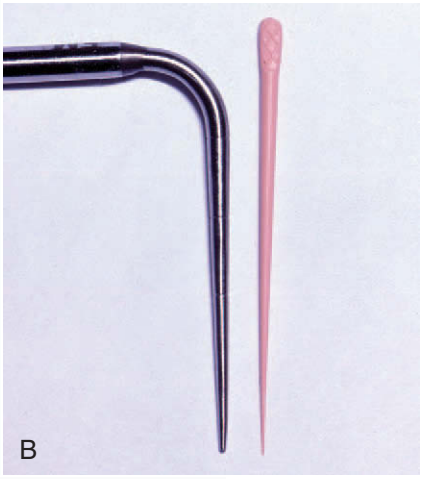




FIG. 10-33 Continuous wave obturation uses the System B unit. **A,** The System B unit. **B,** System B plugger with a nonstandard cone of similar taper. **C,** System B pluggers. *(Courtesy SybronEndo, Orange, CA.)*



FIG. 10-34 System B plugger fit.

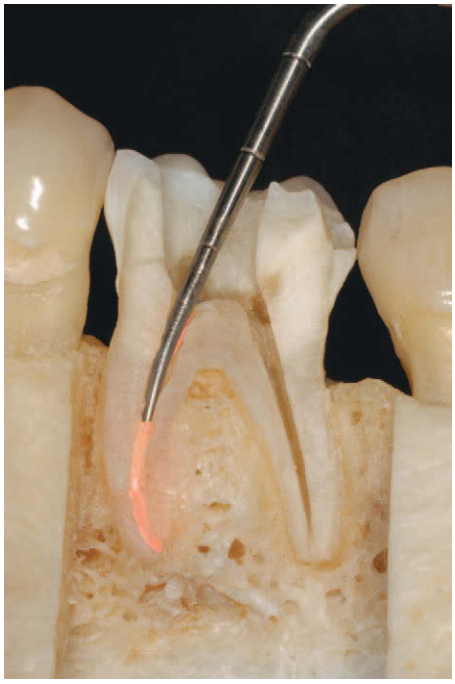


FIG. 10-35 System B activation and compaction.

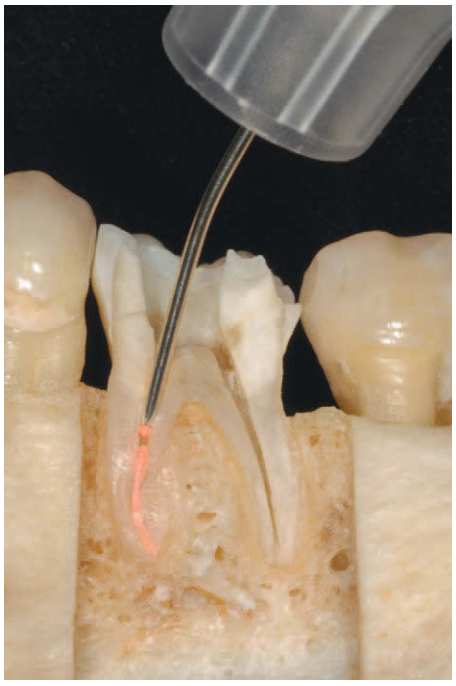


FIG. 10-36 Backfill by the Obtura II thermoplastic injection technique.

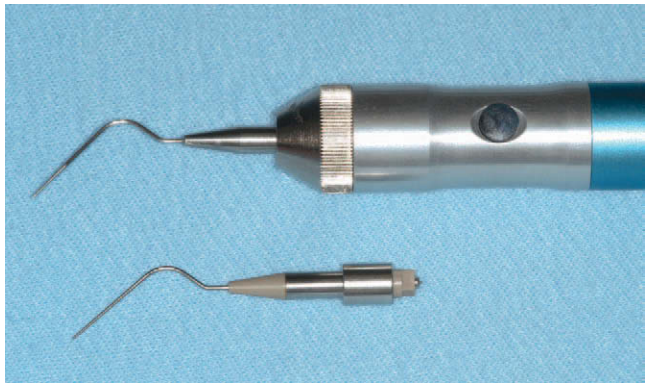


FIG. 10-37 Endotec II device (Medidenta, Woodside, NY) for warm lateral compaction.

gutta-percha mass, by 14.63%, when compared with traditional lateral compaction.[187] Another study found a 24% increase in weight with warm lateral compaction when using the System B device.[211] 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.[68] 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.[173] EI, a subsidiary of Hu-Friedy, has now introduced the EI DownPak (Fig. 10-38), 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, and found that the warm lateral compaction technique created less stress during obturation.[196] 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.[57]

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 #.02/20 and #.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. 10-39). The Obtura III (Fig. 10-40), Calamus (Fig. 10-41), Elements (Fig. 10-42), HotShot (Fig. 10-43), and Ultrafil 3D (Fig. 10-44) are available devices. The Obtura II 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, Earth City, MO) 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. 10-40). 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 II was 27° C.[302]



FIG. 10-38 The EI DownPak device for heat softening and vibrating gutta-percha (Courtesy EI/Hu-Friedy, Chicago, IL).



FIG. 10-40 Obtura III unit with silver tips, gutta-percha plugs, and cleaning solution (Obtura Spartan, Earth City, MO).



FIG. 10-39 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. 10-41 The Calamus thermoplastic unit (DENTSPLY Tulsa Dental Specialties, Tulsa, OK) for heating and injecting gutta-percha.



FIG. 10-42 The Elements obturation unit (SybronEndo, Orange, CA) for injecting and compacting gutta-percha. Note the System B heat source.



FIG. 10-43 The battery-powered HotShot unit (Discus Dental, Culver City, CA) for heating and injecting gutta-percha.



FIG. 10-44 The Ultrafil 3D system consists of an injection syringe, gutta-percha cannulas, and heating unit (Coltène/Whaledent, Cuyahoga Falls, OH).

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 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 posttreatment, 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%).[304] Another study compared lateral compaction with Thermafil (DENTSPLY Tulsa Dental Specialties) and Obtura II in root canal models and found that the Obtura II produced the best adaptation to the canal walls.[332] Other investigators found that continuous wave obturation with the Obtura II backfill initially produced a better bacterial seal when compared with lateral compaction, using bilaterally matched teeth and an anaerobic bacterial leakage model.[147]

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. 10-44). 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. 10-41). 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. 10-42). 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) is a cordless thermoplastic device that has a heating range from 150° C to 230° C (see Fig. 10-43). The unit is cordless and can be used with either gutta-percha or Resilon. Needles are available in 20, 23, and 25 gauges.

GuttaFlow

GuttaFlow (Coltène/Whaledent) consists of a cold, flowable matrix that consists of polydimethylsiloxane matrix filled with very finely ground gutta-percha (see Fig. 10-16). The material is provided in capsules for trituration in an amalgamator. The technique involves injection of the material into the canal and placing a single master cone to length. The material provides a working time of 15 minutes and it cures in 25-30 minutes. Evidence suggests that the material fills canal irregularities with consistency[355] and is biocompatible,[36,95] but the setting time is inconsistent and may be delayed by final irrigation with sodium hypochlorite.[36] Sealing ability appears comparable to other techniques in some studies and inferior in others.[38,169,207,225]

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. 10-45). 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.

Changes in the carrier systems include the development of a plastic core coated with α-phase gutta-percha (Fig. 10-46) and a heating device that controls the temperature (Fig. 10-47). 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. 10-46). Size verifiers are available to aid in selection of the appropriate carrier and should fit passively at the corrected working length (see Fig. 10-45).

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 Chapter 9) and has been shown to enhance the seal with Thermafil.[25] After drying the canal a light coat of sealer is applied

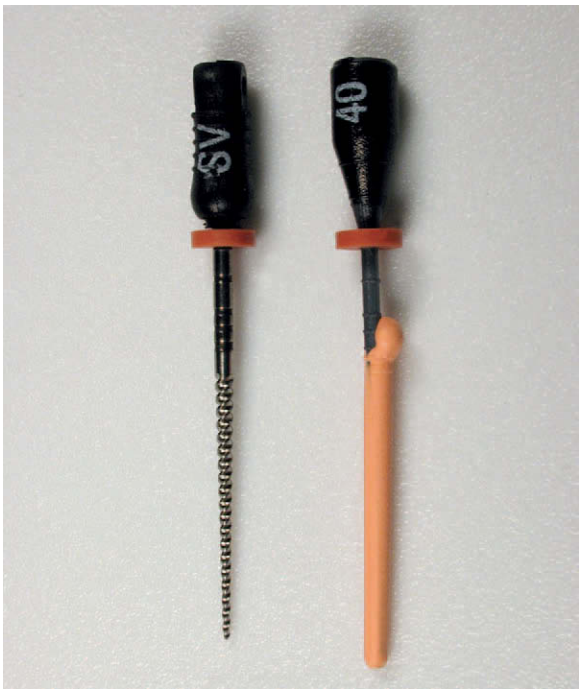


FIG. 10-45 Thermafil carrier and size verifier (DENTSPLY Tulsa Dental Specialties, Tulsa, OK).

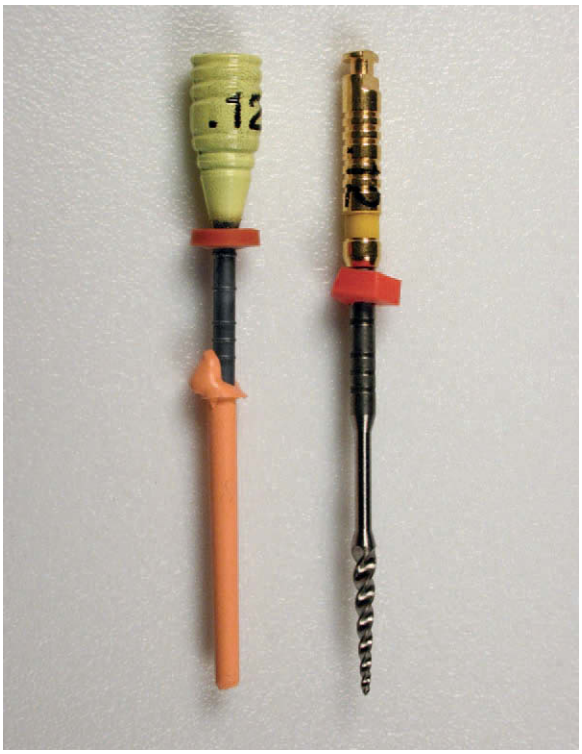


FIG. 10-46 GT obturator and instrument (DENTSPLY Tulsa Dental Specialties, Tulsa, OK).

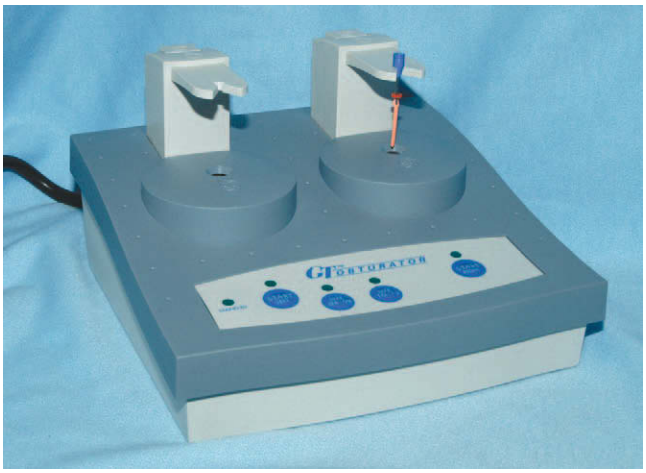


FIG. 10-47 The Thermafil oven with carrier in place (DENTSPLY Tulsa Dental Specialties, Tulsa, OK).



FIG. 10-48 Thermafil carrier placed in the distal canal.

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 has approximately 10 seconds to retrieve it and insert it into the canal (Fig. 10-48). 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.[186] A rapid insertion rate enhances obturation.[186]

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.

Vertical compaction of the coronal gutta-percha can 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. 10-49)[340]; however, extrusion of material beyond the apical extent of the preparation is a disadvantage.[65,73,175]

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.



FIG. 10-49 Apical obturation of accessory canals by the Thermafil technique. *(Courtesy DENTSPLY Tulsa Dental Specialties, Tulsa, OK. All rights owned by and used with permission from DENTSPLY International, Inc.)*

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.

Successfil

Successfil (Coltène/Whaledent) is a carrier-based system associated with Ultrafil 3D (Fig. 10-50); 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 (LightSpeed Technology, San Antonio, TX/Discus Dental) is gutta-percha or Resilon manufactured for use after canal preparation with LightSpeed instruments (Fig. 10-51). 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 (LightSpeed Technology/Discus Dental) to within 1 to 3 mm of the prepared length (Fig. 10-52). 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. 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



FIG. 10-50 Successfil is an additional carrier system (Coltène/Whaledent, Cuyahoga Falls, OH).

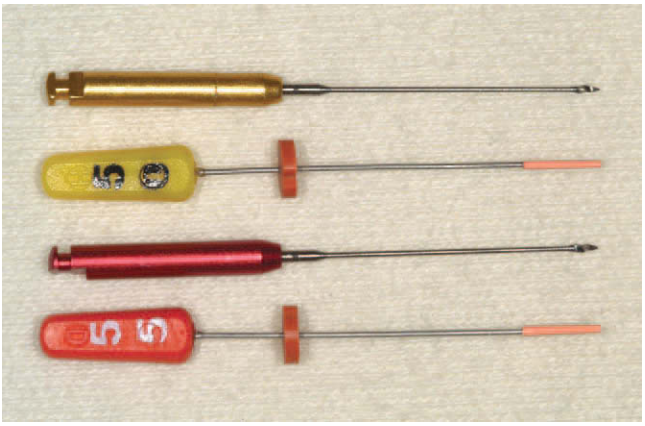


FIG. 10-51 SimpliFill carrier and LightSpeed file (LightSpeed Technology, San Antonio, TX/Discus Dental, Culver City, CA.)



FIG. 10-52 SimpliFill fitted to 1 to 3 mm from the prepared length.

same size as the SimpliFill carrier. This sectional technique is efficient, and leakage potential is similar to that of other common techniques.[257]

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 gutta-percha is heated by the friction of the rotating bur. 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,[128,163,191,261,262] and time. Disadvantages include possible extrusion of material, instrument fracture,[218] gouging of the canal walls, the inability to use the technique in curved canals, and heat generation.[23,102,126,199,262,263] Microseal condensers (SybronEndo) are a variation of this product.

Solvent Techniques

Gutta-percha can be plasticized with solvents such as chloroform, eucalyptol, and xylol. Disadvantages include shrinkage caused by evaporation, voids, the inability to control 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.[52] A gutta-percha cone was then softened and placed into the canal; the mass hardened as the solvent evaporated. Unfortunately, shrinkage occurred with the evaporation process.[241] The techniques using solvents have been abandoned and replaced with materials and methods that do not exhibit shrinkage.

Pastes

Pastes fulfill some of the criteria outlined by Grossman[115] 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.[255]

Dentin chips appear to confine materials to the canal space during instrumentation/obturation and may encourage development of a biologic seal.[94,255] Enhanced healing, minimal inflammation, and apical cementum deposition have been noted histologically.[224] A concern with this technique is contamination of the dentin with bacteria, because investigators found that infected dentin adversely affected healing.[139]

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.[239] Calcium hydroxide has the advantage of being free of bacterial contamination and may provide a better, although imperfect, apical seal.[330]

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.[9,251] Immediate obturation is an alternative to apexification. An apical barrier material should confine obturation materials to the canal space[94] and enhance healing by inducing cementum and bone formation.[137,224,239] Mineral trioxide aggregate (MTA) (ProRoot MTA; DENTSPLY Tulsa Dental Specialties) has been successfully employed as an apical barrier material before obturation (Fig. 10-53).[279]

After the cleaning and shaping procedures the canal is dried and MTA is placed. The material is compacted into the apical portion of the root to form a barrier. After the material sets, gutta-percha can then be compacted without extrusion (Fig. 10-54). Other investigators found that hand compaction of MTA provided better adaptation to the canal walls with fewer voids when compared with ultrasonic placement.[8] Another investigation reported that ultrasonic placement of a 4-mm apical plug of MTA improved the seal[352] and that placement of a composite resin as the obturation material enhanced the seal and strengthened the root.[178]

MTA is sterile, biocompatible, and capable of inducing hard tissue formation.[92,140] The technique has been shown to be clinically successful and can be accomplished quickly, eliminating the need for numerous visits and possible coronal recontamination during the months required for apexification.[110,185]

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.[93] A prospective clinical study found the success rate of MTA barriers and immediate obturation in 43 cases to be 81%.[286] In another study, 85% of 20 teeth with immature apical development were considered healed and 5% were considered healing after immediate barrier placement.[136] The time required for healing is comparable to traditional apexification with $Ca(OH)_2$ and treatment time is reduced.[240]

Another option for treating immature teeth with pulp necrosis involves regenerative techniques including revascularization (see also Chapter 16).[127,317] Advantages include continued increasing thickness of the canal walls, apical root development, and apical closure.[280] 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 to induce a clot that is covered with MTA. When the MTA is set a definitive restoration can be placed to ensure a coronal seal.[14]



FIG. 10-53 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, OK. All rights owned by and used with permission from DENTSPLY International Inc.)*

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,[56,314] 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 importance of maintaining a coronal seal to prevent bacterial leakage.[149] Leakage studies indicate that the coronal seal can be enhanced by the application of supplemental restorative materials over the canal orifice[237,250,331] and by placing a definitive coronal restoration as soon as is feasible.[244]

Cavit (Premier Dental Products, Plymouth Meeting, 3M Espe Dental Products) has traditionally been advocated as an acceptable material. One study demonstrated that placement of 3.5 mm of Cavit or SuperEBA cement (Bosworth, Skokie, IL) decreased bacterial leakage by 85% and 65%, respectively, when compared with unsealed controls, which all leaked at 45 days.[237] 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.[350] Another study demonstrated in a dog model that MTA placed in the orifice decreased inflammation in teeth inoculated with bacteria.[194]

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.[26,58,181] 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. Investigators found that this procedure resulted in none of the experimental canals showing bacterial leakage at 60 days.[60]

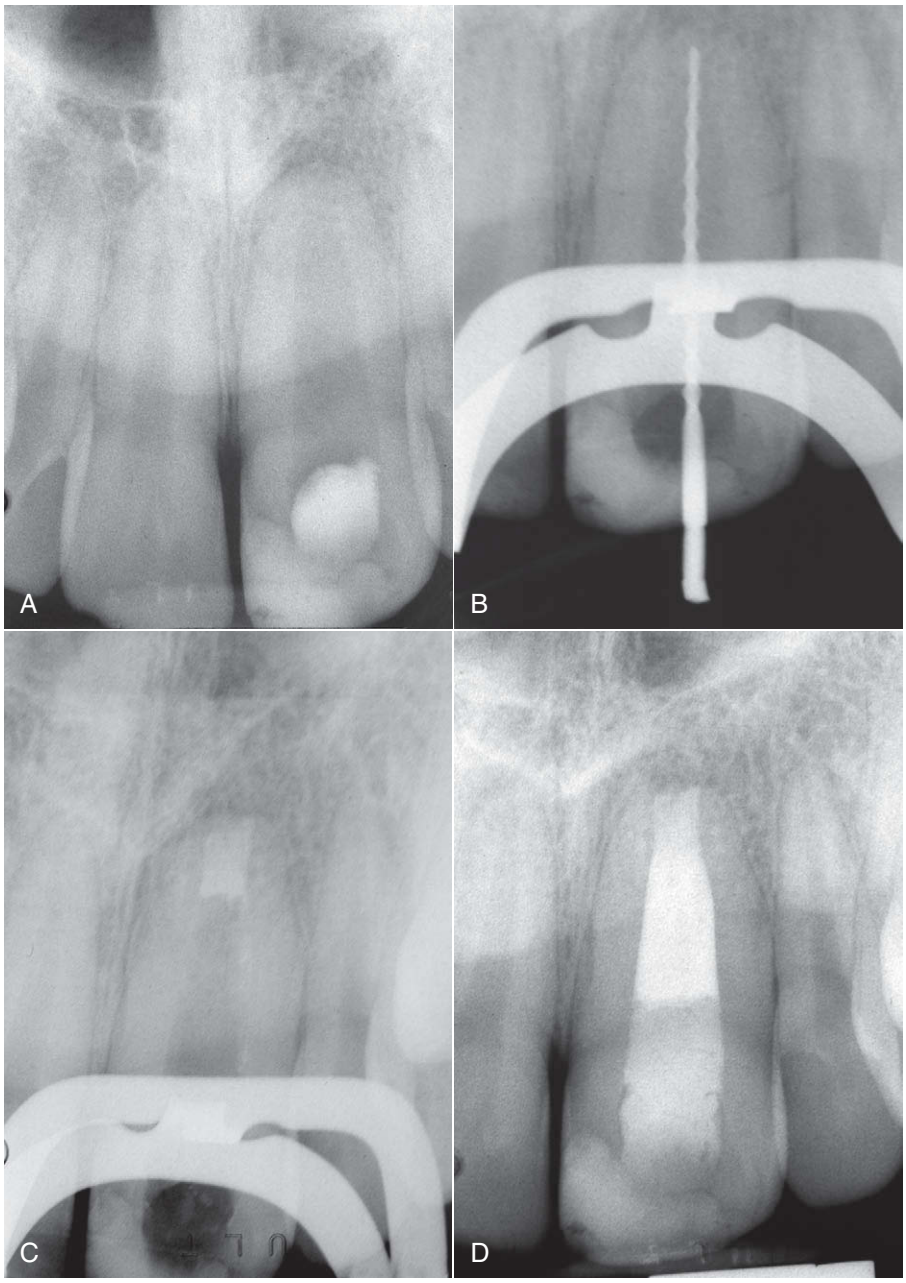


FIG. 10-54 Immediate obturation employs a barrier technique to prevent extrusion when the apex is open. This case involves a maxillary left central incisor with pulp necrosis caused by trauma. **A,** Pretreatment radiograph demonstrates a large canal with an open apex. **B,** Working length is established and the canal is prepared. **C,** Mineral trioxide plug is placed. **D,** The canal is obturated with gutta-percha.

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CHAPTER 11

Endodontic Records and Legal Responsibilities

EDWIN J. ZINMAN

CHAPTER OUTLINE

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- Posttrauma Therapy

Continuing Education
Millennium Management of Endodontic Advances
Statistical Research
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Peer Review
Human Immunodeficiency Virus and Endodontics
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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. Records also are fundamental means of communication among health care professionals, designed to protect the patient's welfare.

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 tests performed
8. Clinical examination findings
9. Differential and final diagnoses
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 forms
23. Accounting
24. Recall notifications
25. Author name or initials for each treatment entry
26. E-mail address
27. Emergency contact persons and addresses
28. Phone number including home, work, and cell

Function

Dental records should document the following information:

1. Course of therapy by recorded diagnosis, informed consent, 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. The patient's name; home, business, e-mail addresses; and telephone and fax numbers are needed to contact the patient for scheduling purposes and inquire about postoperative sequelae.[144] 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 installment payments are arranged (whether or not there are interest or late-payment charges).[79] Patient information should be updated periodically (Fig. 11-1).

Medical Health History

Past and present health status should be thoroughly reviewed by the clinician before proceeding with treatment 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 allergy mandates treatment modifications.[29,135,152] Current medications, medical therapy, and the name and location of the treating physician are 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. 11-2). 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 such contacts. In addition, the clinician should verify physician approvals for treatment by fax, with

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. 11-1 Patient information form.

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. 11-2 Medical history form.

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 and/or supplements. 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.[269] 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.[270] A clinician exclusively relying on staff members to obtain medical histories in a waiting room full of patients is making a mistake, because the accuracy of the histories must first be checked and then followed up by the clinician. 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 duration, frequency, type and intensity of any pain), 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 and/or present treating clinician.[188] After receiving positive responses, the clinician may also wish to obtain prior and concurrent treaters' radiographs for current comparison and notes of any progressive changes (Fig. 11-3).

Diagnostic and Progress Records

Diagnostic and progress records often combine fill-in and check-off types of forms. Fill-in or essay-type forms allow greater latitude of response to a question, resulting in a more detailed description. However, one drawback to these forms is that they are open to oversights unless a clinician is very 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 consideration or consultation.[29] 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.[221,231]

Electronic Records

Electronic records represent current methodology for recording patient information (see Chapter 28 for details).[246] 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.[120] 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.[70] 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 5).[114,219,279] 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 and retain all radiographs.

Digital radiography is recommended because it increases endodontic efficiency. 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 CCD (charged coupling device) chips rather than CMOS (complementary metal-oxide semiconductor) technology, and provide close-up zooming with improved image quality as described in Chapter 29.

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 or restorability of the tooth), the clinician should consider further consultation with the referring clinician or specialists, including a prosthodontist, periodontist, or both, 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 3).[5] For instance, in a long-term follow-up study of patients with chronic facial pain and without recognizable odontologic pathosis, some patients were subsequently diagnosed with maladies ranging from cracked-tooth syndrome to medication-mediated illnesses.[5] Accordingly, referral to other specialists is essential, lest a patient be misdiagnosed with idiopathic or atypical facial pain when instead, pain etiology was misdiagnosed. Cone-beam computerized tomography (CBCT) has greatly aided diagnosing pain sources caused by root fractures and incompletely filled, overfilled, or

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. 11-3 Dental history record.

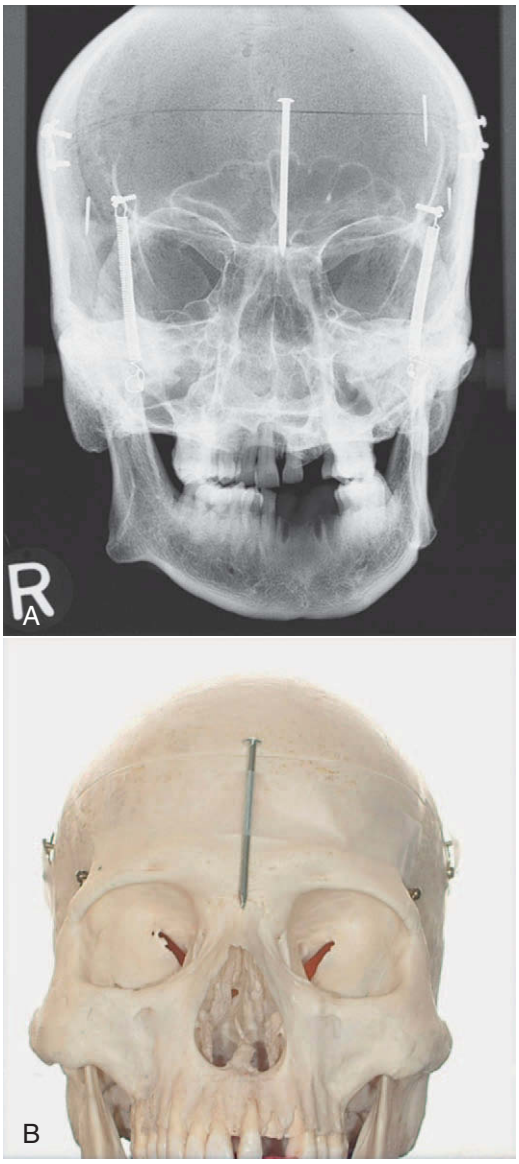


FIG. 11-4 A, Nail inside cranium? **B,** Nail in same patient as **(A)**, showing nail taped to outside of skull. Alas poor Yorick!

missed extra canals that two-dimensional (2D) periapical imaging may obscure.*

Fig. 11-4, *A-B* demonstrates the advantage of CBCT three-dimensional (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 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)

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.

Treatment Plan

Treatment records should contain a written plan that includes all aspects of the patient's oral health. Treatment plans should be coordinated, preferably in writing, with other concurrent or jointly treating clinicians. If an aspect of the patient's care is not under direct supervision, the clinician is proceeding improperly. Also, the clinician should initiate contact with the other treating clinician. The patient should also be advised of the problem. For instance, endodontic treatment will probably fail if underlying periodontal pathosis is ignored or untreated, so the clinician should assess the patient's entire dentition, not just a single root canal system. The clinician should also recommend a periodontal consultation to both the patient and referring clinician if periodontal therapy is required. Follow 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 Fig. 11-5. 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. Such discussion is documented by completing the checklist items.

*References 54, 73, 127, 138, 163, and 166.

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:			RCT Yes No
			pulsating provoked	biting head position	
	localized referred	constant momentary	steady reproducible	chewing activity	Sx Pre-Tx: Yes No
	diffuse radiating	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 ___ Treatment Plan ___ Restoration ___ Discoloration ___ Recall ___ Consent Form Referral: 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. 11-5 Diagnosis and treatment form.

Treatment

All treatment provided on a given date is documented by placing a check mark (✓) 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." Fig. 11-6 also gives 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 was superficial and treatment incomplete. If a suspicious apical lesion requires subsequent reevaluation, the clinician should record the future reevaluation date and differential diagnosis (e.g., "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 a potentially pathologic condition, 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.[154,246]

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 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. 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 believe 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.

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. 11-7). 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

Chart

A suggested chart is presented in Fig. 11-8 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. 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 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.

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 and/or recent past. Narrative facts regarding the presenting problem are then recorded. Additional details of the chief complaint obtained during

MEDICATION LOG: P. Ulp (415) 391-5353

Patient name 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
6/19/05	Penicillin VK	24 tabs	500 mg	N	W	N/A		EZ
6/29/05	Vicodin	10 caps	#3	N	P	Walgreens (415) 291-1234	KF	SC

FIG. 11-6 Medication log.

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 such 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.[57]

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.[29,231] Maximum

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. 11-7 True/false endodontic informed consent form.

LAST NAME	FIRST NAME	DR / MR. / MISS / MRS.	ADDRESS	HOME PHONE
Law	Murphy		132 Post Street Endo, MD.	800-872-3636 BUSINESS PHONE

REF. DR.	REF. DR. ADDRESS	REF. DR. PHONE
Good	911 Main Street Washington, D.C.	800-621-8099

TOOTH: R 30 L — FEE: $100 — TREATMENT PLAN: (CONSULT) EET — (RCT) AE PE (ME) — SURGERY: $Ca(OH)_2$ — SPECIAL INSTRUCTIONS: POST SPACE — (PREFORMED POST/B.U.) COMP AMAL TEMP. CR. — Distal Canal

S — MIDDLE INITIAL

INSURANCE S D	AMT DUE	REC'D	DATE	DAY	TIME	PRO-cedure	X-RAY	REMARKS:
PRE-AUTH Y N								
% COVERED								
VERIFIED								
FORM SIGNED								
PT. INFORMED								
PT. PORTION								
INS SENT								

Murphy — FIRST NAME

DATE MO	DAY	YR	REMARKS	SIGNATURE
9	14	09	Pt. urged to have a cast metal crown made as soon as possible. Confirming letter sent to referring doctor. S. Schwartz	EZ

Law — LAST NAME

FIG. 11-8 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.

Initials: Dr. SC Asst. EZ

SYMPTOMS	Location	Chronology	Quality		Affected By		Prior Tx		
Left side V1–V3	localized / (diffuse) / referred / (radiating)	Inception 6 days; Clinical Course: (constant) / intermittent / momentary / (lingering)	(sharp) / dull / (pulsating) / steady / enlarging	intensity + (++) +++ spontaneous / provoked / (reproducible) / occasional	(hot) + / (cold) ++ / biting / (chewing) / percussion	palpation / manipulation / head position / activity / time of day	Tx: restorative (Yes) No crown; emergency Yes (No); RCT Yes (No); Sx Pre-Tx: Yes (No); Sx Post-Tx: (Yes) No		

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

Tooth	Attachment Apparatus
WNL caries (restoration) (calcification) mesial 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

CLINICAL

Tooth	Soft Tissues
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 perio

B 2; M 3; D 3; L 2

DIAGNOSTIC TESTS

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:	2/14/06					

bruxism: yes ✓ no _____

nightguard: yes ✓ no _____ Initials: Dr. SC Asst. EZ

DIAGNOSIS

PULPAL	PERIAPICAL
WNL reversible pulpitis (irreversible pulpitis) necrosis prior RCTx/RCF	WNL (acute apical periodontitis) acute apical abscess chronic periapical inflammation phoenix abscess osteosclerosis

ETIOLOGY

idiopathic	trauma
caries	periodontal
(restoration)	orthodontic
attrition / abrasion	prior RCTx
developmental	intentional
sinusitis	systemic

PROGNOSIS

ENDODONTIC	PERIODONTAL	RESTORATIVE
(favorable)	(favorable)	(favorable)
questionable	questionable	questionable
poor	poor	poor
hopeless	hopeless	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	09	✓	✓	✓	✓			✓	✓	✓	✓	✓	✓	✓			✓	✓	w	(1)	+	glass ionomer							core							SC	EZ

CANAL	REF	Elec.	X-Ray	Adj.	Final	Size	Rx Date	MEDICATION	DOSE DISP	INSTR.
B F							2/14/01	Ibuprofen	600mg x 12	1 q 6 h
L P									x	q h
MB		21.0				25			x	q h
ML		20.5				25			x	q h
DB		21.0				30			x	q h
DL		21.0				30			x	q h

FIG. 11-8, cont'd B, Diagnosis and treatment form with notes.

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 assorted pathologic conditions. 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. 11-2). A review of the patient's medical status (including recent or current conditions, treatments, and medications) completes the medical history. Updated medical histories which the patient signs or initials should be done at least annually and at reevaluation visits.

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 anticoagulant drug reactions. About half of prescription users also concurrently take over-the-counter medications plus dietary supplements.[153,196]

Risks of drug interactions or side-effects incidence may not be discovered and/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.[21] Currently, pharmaceutical manufacturers budget more for marketing than research and development.[96] Some drug manufacturers have suppressed publication of adverse research.[38,170,272] U.S. Senators Chuck Grassley (R, IA) and Herb Kohl (D, WI) are probing the financial relationships among the drug industries, health care prescribers, and researchers (Senate Bill 2029 [http://thomas.loc.gov]). If their Physician Payments Sunshine Act passes, it would require companies to disclose payments to medical/dental professionals who tout their drugs in seminars or publications.

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.[272] For instance, mibefradil (Posicor) was withdrawn from the market after 1 year because it increased plasma concentrations of 25 other coadministered drugs, including erythromycin.[196,197] In another example, the popular prescription nighttime heartburn drug, Propulsid (cisapride), was linked to 70 deaths and more than 270 significant negative reactions.[263] Other examples include the risk of esophageal cancer with bisphosphonate[272] and bisphosphonate-associated osteonecrosis.[69,232] In 2008, the FDA added a black box warning label for Levaquin-associated risks of tendonitis (www.levaquin.com/levaquin/medication).

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 and/or severe side effects (risk outweighs the benefit).[23,257]

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.[199] In 2007, one in seven U.S. residents younger than age 65 skipped prescribed medications because of rising prescription cost.[81,123]

The Pharmaceutical Research and Manufacturers Association of America has created an online database that summarizes 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) forms the basis for safety information that accompanies every prescription label.[86,88]

For the first time, under a new federal law[89] 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 was under no federal legal obligation

*References 14, 147, 159, 178, 207, 216, 222, 229, 239, 254, and 272.

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.[254,273]

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 vast 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.[216] 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 requires 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, conflict potentials 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]).[86,273]

In 2009, the Government Accounting Office (GAO) concluded in GAO's 2009 High-Risk Series, *FDA Oversight of Medical Products*[259]:

> *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.[100] Clinical testing in developing countries help overcome stringent FDA regulatory barriers for FDA drug approval.[16]

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 and/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.[128] 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.[280]

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.[16] The Declaration of Helsinki 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; such 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.[105] 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 because labor, construction, regulatory, and environmental costs are lower there. The world's growing dependency on Chinese drug manufacturers become apparent in the heparin scare. In 2007,

Baxter Intentional 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.[259] 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 timely completed rather than delayed or evaded. Notwithstanding PUDFA, in 2008 the FDA was still missing target dates to act on NDAs because the FDA lacks sufficiently adequate staffing to handle PMA applications.[210]

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.[153,167,277] 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[167] 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.[134,277] 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 and/or deceptive.[167]

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.[277] 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. It 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.[28,117] Echinacea increases potential for liver damage when used with steroids. Gingko biloba and feverfew interfere with anticoagulants such as warfarin (Coumadin).[53] Dong quai root, willow bark, goldenseal, guarana, horse chestnut, and bilberry tablets/supplements also have antiplatelet properties.[242] Ginseng may cause rapid heartbeat and/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.

A Los Angeles police officer who experienced a crippling stroke after taking an ephedra-based energy supplement, Dymetradine Xtreme, was awarded $4.1 million.[20] The retail store owner had read but disregarded 30 journal articles warning of strokes, heart attacks, seizures, and other ailments. The stroke occurred before the FDA banned ephedra from sale in April 2004. Nonetheless, Internet sales proliferated.[242]

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 are proven efficacious for anything.

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.[87-90,196] 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 http://www.fda.gov/medwatch/getforms.htm and report suspected drug interactions, reaction, or adverse product events at http://www.fda.gov/medwatch/report.htm. Other electronic drug databases are available to report and access important drug interaction information.[275] 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, since clinician reporting is voluntary.[150] On the other hand, drug manufacturers must report known adverse drug incidents to the FDA.[88]

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.

ENDODONTICS AND HEART DISEASE

Associations between chronic infections such as periodontal diseases and coronary heart disease (CHD) have been suggested.[226] Conversely, supporting evidence linking root canal–filled teeth or teeth with periapical disease to CHD is lacking.[94]

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 (see Fig. 11-9) illustrates its proper use. Box 11-1 lists a key to the abbreviations.

Computerized Treatment Records

Clinicians are increasingly using computerized record storage as described in Chapter 29. To avoid a claim of record falsification, whatever computer system is used should be able to 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 not a virtual paperless office.[246]

Health Insurance Portability and Accountability Act

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*).[255]

BOX 11-1

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	=	Bisphosphate
BW	=	Bite wing
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	=	Epinepherine
final	=	Final file
Fx	=	Fracture
G.G.B.	=	Gates-Glidden bur
G.P.	=	Gutta-percha
I & D	=	Incision and drainage
I.P.	=	Irreversible puplitis
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

Pertinent HIPAA requirements stipulate that:

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.

Record Size

Although there is little harm in recording too much information, there is great danger in recording too little. Standard 8½ × 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.[39] 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 (Fig. 11-9).

Clinician and Staff Initials Log

Clinician	Staff	Initials	Date
Edwin J. Zinman		EZ	6-01-09
	Alyssa Malfatti	AM	6-01-09
Stephen Cohen		SC	6/1/09
	Kelly Fishman	KF	7/1/09

FIG. 11-9 Clinician and staff initials log.

Patient Record Request

Patient requests for record transfers or copies must be honored. It is unethical to refuse to transfer patient records, on patient request, to another treating clinician.[10]

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 on written request, even if an outstanding balance is owed.[42]

Patient Education Pamphlets

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* or 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, since this may prove useful should the patient later deny being informed of endodontic alternatives.

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.[4] Document that both written and oral postoperative instructions were provided. If treating in close proximity to the inferior alveolar nerve canal (IANC), place a precaution, such as seen in Fig. 11-10, in the 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

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. 11-10 Postoperative information sheet.

the patient's chart. The clinician or staff member should document the fact that the original referral card was given to the patient and record the name of the person who provided the referral card and the date on which it was provided. Document if mailed to the patient. 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.

Record Correction

Records must be complete, accurate, legible, and dated. All diagnosis, 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 make 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.[39] Professional liability insurance policies may defend but will usually not indemnify punitive damages if the clinician is found to have committed fraud or deceit.[43,45] 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. Fig. 11-11 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.

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).[78]

Spoliation occurs when the wrongdoer alters, changes, or substitutes dental records in an attempt to defeat a civil lawsuit.[47,258] 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 referral was made (Fig. 11-12, *A-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.[17]

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.[47,251,255,258,260]

Deliberate record alteration with intent to deceive may also subject the clinician to licensing[39] 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.[137,251] 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.

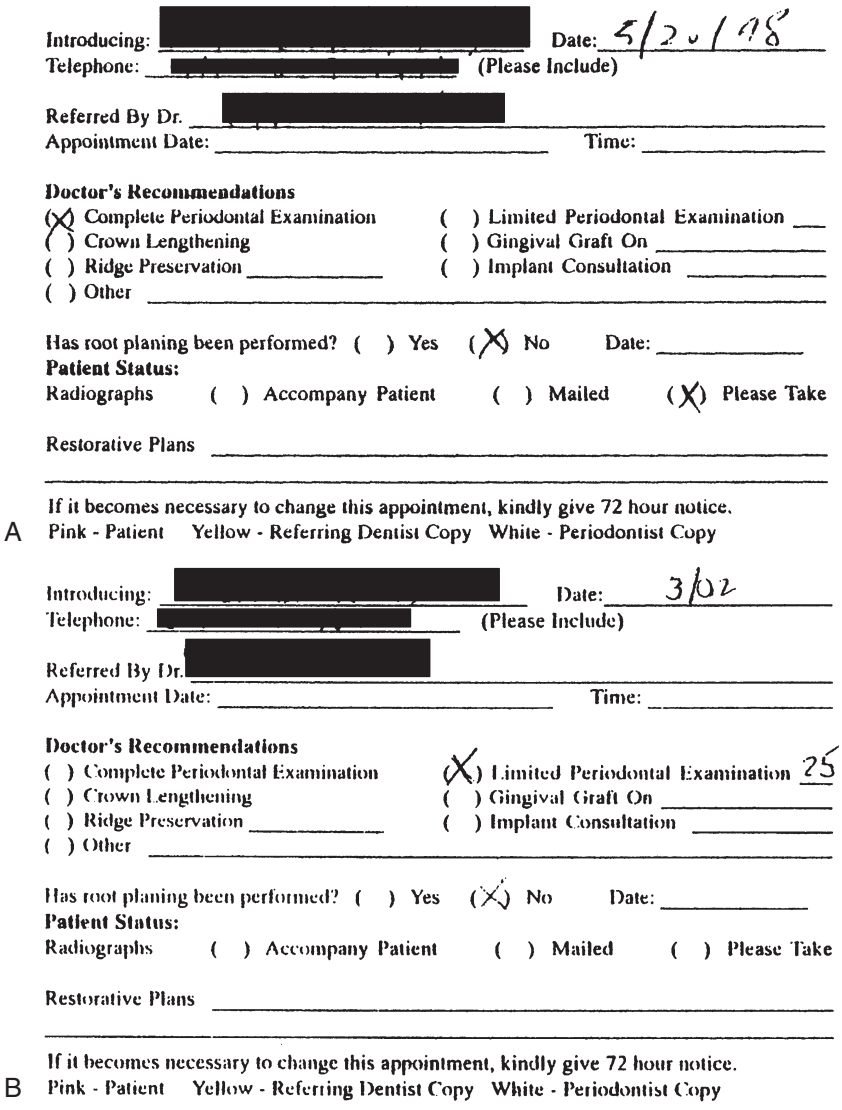
False Claims

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 treatment also ethically violates the Hippocratic Oath of "primum non nocere" (i.e., "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 CHAMPUS (Civilian Health and Medical Program of the Uniformed Ser-

- 10-13-09-Corrected entry for 10-12-09. (Entry should have recorded): Hot, cold, and percussion tests plus one. Recommend re-evaluation in 24 hours and repeat pulpal tests. Next visit scheduled 10/13/09 at 9 a.m.

FIG. 11-11 Belated record correction.



Introducing: [redacted] Date: 5/20/98
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. 11-12 A, Falsified records with belated entry of year of referral. **B,** Falsified records.

vices) 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.[258] 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.[252]

LEGAL RESPONSIBILITIES

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 endodontic diagnosis or therapy.

Standard of Care

Good endodontic practice, as defined by the courts, is the standard of reasonable care legally required to be performed by a reasonably careful clinician. 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.[140]

Although the standard of care is flexible to accommodate individual variations in treatment, it is objectively tested based on what a reasonable clinician should do. Reasonable conduct represents a minimum standard required for legal due care. Reasonable care is care based upon reasoning supported by sound science such as evidence-based peer-reviewed literature. Additional precautionary steps that rise above this minimal floor of reasonableness and reach towards the ceiling of ideal care are laudable, but they are 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, since 49% of clinicians practice as 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.

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.

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.[267] 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.[267]

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 or decline uninsured services. Informed choice is uninformed if the clinician fails to provide patients with all reasonable options and alternatives.[171]

If an insurance carrier denies endodontic therapy or limits endodontics to only certain clinical conditions, a prudent clinician must provide informed consent to the patient both legally and ethically that a tooth may be endodontically treated and retained rather than extracted.[51,181,233,245,281] 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."[41]

Clinicians may agree to a discounted fee with a health maintenance organization (HMO) carrier but must never discount the quality of care provided. Peer review 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 Three 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.[192] 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.[111]

Plans that delay treatment approval, resulting in endodontic complications or tooth nontreatability, may be subject to liability.[193,245] 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.[200]

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, since 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.[267] A patient's best interest is always paramount, both legally and ethically, to a clinician's financial interest.[97,268]

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 which 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 primarily based upon patient choice and not cost alone.

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).[140] *Negligence* is equated with carelessness or inattentiveness.[145] *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, or
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 should 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.

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.

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.[161,282]

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, since the AAE Board is national in scope.[235] 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 if an endodontic overfill enters the IANC.[206] 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.

*References 54, 73, 127, 138, 163, and 166.

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; to blame technologic ignorance on a clinician's rural location is inexcusable with modern media, computer technology, and travel ease.

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.[103,104] 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.[74,145] 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.

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.[97] Section 5 of the ADA Code of Ethics regarding veracity ("truthfulness") states that "Professionals have a duty to be honest and trustworthy in their dealings with people." Accordingly, both ethically and legally,[129] 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.[97]

Section 5.I of the ADA Code of Ethics provides: 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 which 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 versus a generalist, although each provide endodontics. Only a board-eligible or board-certified clinician is entitled to represent that the clinician is an endodontist. With this distinction in mind, the ADA-recognized endodontic specialty organization is entitled 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, the generalist should refer the patient to an endodontist.[55,103,104,149]

Endodontists should not forget their general clinician 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. Fig. 11-13 demonstrates a general clinician's radiograph showing apparent complete endodontic fill within the canal space, whereas Fig. 11-14 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.[163,166]

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 tested endodontically, is brought under periodontal disease control, including any crown-lengthening needs (see Chapter 18 for more information). Referral to a periodontist may be necessary before or contemporaneous with completion of endodontic treatment.



FIG. 11-13 A general clinician's radiograph that ostensibly shows a complete endodontic fill within the root canal space.



FIG. 11-14 Endodontist's radiograph demonstrating a transported canal.

In summary, it is necessary for the endodontist to:

1. Be alert to any contributory medical or dental condition within the operative area of endodontic treatment that can affect treatment.
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."[179] As expressed in the context of actions for negligence, however, ordinary care has assumed a technical legal definition somewhat different 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."[31]

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."[83] It has been equated with the reasonable care and prudence exercised by ordinarily prudent clinicians under similar circumstances.[107,213] 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.[213] 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."[227] Thus the standard of care equates with *reasonable* 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).[26] 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.[181,183] However, careful clinicians did follow the ADA's recommendation (Box 11-2 and Fig. 11-15).

Merely because a majority of clinicians in a community practice a particular method does not establish it as the standard of care if such practice is unreasonable or imprudent.[26] Ultimately, the courts determine what constitutes reasonable practice by considering available dental knowledge and evaluating the relative risks versus benefits of a particular procedure.

Again, the law does not require dental perfection.[249] 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 such standard.

In one case, it was not customary practice in the state of Washington for ophthalmologists to test patients under 40

BOX 11-2

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 and face masks
- Failing to use rubber dams for endodontics
- 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 and/or disinfection of environmental surfaces
- Failing to use a microscope for fractured file retrieval
- 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. 11-16 for implant "harpooning" of IANC).

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* 134:895–899, 2003; Simonsen R: Greed and gravy train: is this success? *J Esthet Dent* 11:287, 1999; Rosenbaum S, Frankford DM, Moore B: Who should determine medical care necessity? *N Engl J Med* 340:229, 1999; Occupational Safety and Health Administration (OSHA): Occupational Safety and Health Administration Directive, Washington DC, 1999. Available at www.OSHA.90V; Cheung G: Endodontic failures. *Int Dent J* 146:131, 1996; Lyon TC: Quaternary ammonia compounds: should they be used for disinfection in the dental office? *J Dist Columbia Dent Soc* 48:10–18, 1973; Kutsch V, Milicich G, Domb W, et al: How to integrate CAMBRA into private practice. *J Calif Dent Assoc,* 2007; Fontana M, Zero D: Assessing patients' caries risk. *J Am Dent Assoc* 137:1231–1239, 2006; Young D, Featherstone J, Roth J, et al: Consensus statement caries management by risk assessment: implementation guidelines to support oral health. *J Calif Dent Assoc* 35:799–805, 2007; ADA Caries Risk assessment forms. Assertion additional resources. *J Am Dent Assoc* Jan 12, 2009 update; Zeider S, Ruttimann U, Webber R: Efficacy in the assessment of intraosseous lesions of the face and jaws in asymptomatic patients. *Radiology* 162:691, 1987; Cotton TP, Geisler TM, Holden DT, et al: Endodontic applications of cone-beam volumetric tomography. *J Endod* 33:1121–1132, 2007; Kersten DD, Mines P, Sweet M: Use of the microscope in endodontics: results of a questionnaire. *J Endod* 34:804–807, 2008.



FIG. 11-15 Implant "harpooned" inferior alveolar nerve canal.

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, since a negligent custom is no defense.[121,175]

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 of tooth or endodontic pain when compared to the relatively low radiation risk. Thus the CBCT diagnostic benefit outweighs the radiation risk if a differential diagnosis can not accurately rule in or rule out a final diagnosis.[165]

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.[185] As a general maxim, "Don't be the first nor the last to adopt new technology."

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.[108,197,198,205] The unusual characteristics of a particular patient can place the patient at higher risk than the average patient.[124]

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.[118] 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.[15] 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.[14]

Evidenced-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.[198] 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 indicate that it works. 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.[108,159,205,239]

Nevertheless, 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.[187] 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.[4] Another example is one-visit

endodontics, which has proven as effective as two-visit endodontics.[84,186,202]

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. Postmarketing 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 Advance luting agent.[101]

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. 11-16). Accordingly, despite insufficient label warnings on some ultrasonic devices, ample water coolant, rest periods, and avoidance of prolonged ultrasonic device use are needed for safe post removal.[68,71,102]

FDA Approval

Despite FDA approval, some drugs are marketed with fraudulent concealment of the number and nature of adverse event incidents.[8]

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 which are exempt from premarket notification requirements under The Food and Drug Administration Modernization Act of 1997 (The Modernization Act),[86] which include the following endodontic products:

- Root canal posts
- Gutta-percha

Negligence Per Se

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.[58,248]

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.



FIG. 11-16 Results of overheating a post with an ultrasonic device during post removal.

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.[213]

Informed Consent Principles

In General

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.[51,141,182] This doctrine is based on the legal principle that individual 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 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 and/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.[51,141,182] 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 that disclosure a clinician knows (or should know) that 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.[221]

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.[51,141,182] 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 is not expected to consent to negligent care. Accordingly, informed consent applies only to treatment which complies within the standard of care. If the clinician is reasonably careful during treatment, but an untoward results occurs, the clinician is not liable if the patient was forewarned of the potential risk prior to the treatment.

A reasonably unavoidable risk which manifests during treatment represents a maloccurrence and not malpractice. For example, a small percentage of endodontic failures are due to 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.

Patient treatment refusals should be documented. Some states require informed refusal disclosures advising the patient about the consequences of refusing treatment or diagnostic modalities.

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.

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 which may result in 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, esthetics, 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 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 close radiographic observation at recall visits.[55,149,160] In Fig. 11-17, 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 and caused more deleterious chemical injury to the IAN. If retrieval was unsuccessful, the patient should have been referred to a microsurgeon within 48 hours from occurrence for optimal opportunity to prevent chemical cytotoxic injury to the IAN.[206]

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 avoid postoperative sensitivity. Long-term testing before marketing would have revealed that the two-step system is more reliable and forgiving.[250] Reasonable clinicians do not sacrifice patient safety for speed and profit and then compensate with desensitizing agents. Clinicians should be more sensitive to limited research on new products or devices lest the patient be the injured guinea pig.

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.[48] 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.[253] Accordingly, a patient who refuses necessary diagnostic radiographs should be refused treatment.

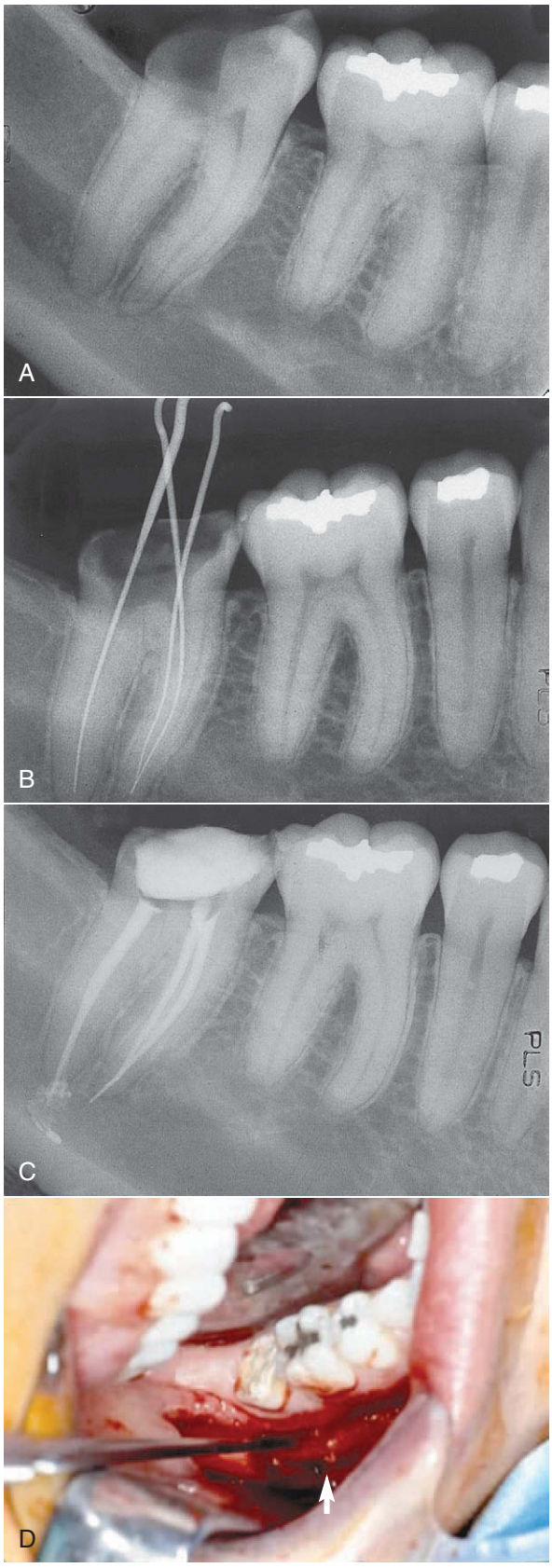


FIG. 11-17 A, Preoperative radiograph. **B,** Measuring film without a rubber dam. **C,** Postoperative radiograph. **D,** Decompression microsurgery showing protruding "dagger" of bone to right of retractor at inferior border of inferior alveolar nerve, resulting from prior perforation of the mandibular canal. This is probably due to file overinstrumentation. *(Courtesy Anthony Pogrel, DDS, MD.)*

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.[130]

Restorative Informed Consent

Patient choices for restorations may discount longevity and elect instead esthetics. In one study, amalgams lasted 15 years, composites 6 years, and conventional glass isomers 7 years.[91] 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.[261,281] For example, there may be two schools of thought concerning the optimal treatment for retrofilling and apicoectomy. One school posits that a retrograde with intermediate restorative material (IRM) mineral trioxide aggregate (MTA) is the appropriate procedure, whereas the minority review asserts that retrograde with Super EBA is the preferred treatment.[278] 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.[237] Long-term durability depends on an insoluble seal to reduce the risk of bacterial leakage.[48,189] Fig. 11-18 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 above 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

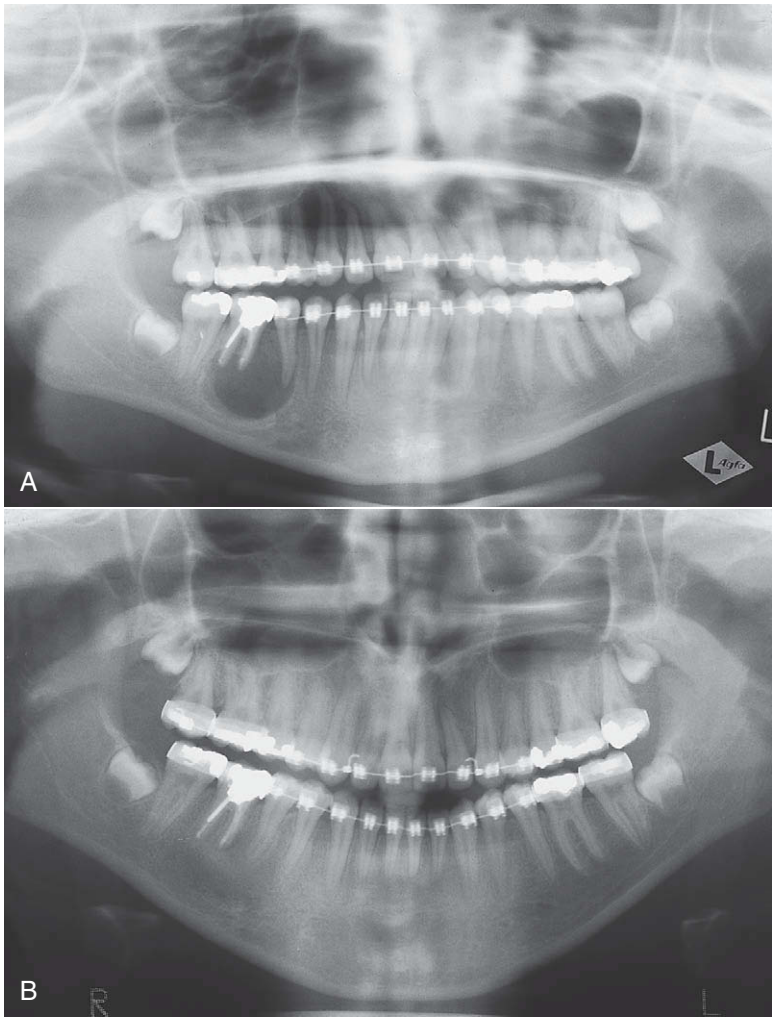


FIG. 11-18 A, Preoperative panoramic radiograph showing a successful apicoectomy and retrograde seal. **B,** Postoperative panoramic radiograph showing a successful apicoectomy and retrograde seal. (*Courtesy Edmond Bedrossian, DDS.*)

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. Such evaluation and decision constitute a nondental judgment reserved for the patient alone.[51,141] In this hypothetical situation, failure to disclose such material information would deprive the patient of the opportunity to weigh the risks. 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.[48] Finally, microscopic endodontics represents the current standard of care for apical surgery or fractured instrument retrieval.[55,136,149,160]

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 must be documented. Informed consent forms are very helpful, although not legally mandated, because a jury may believe that the patient was informed orally. 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."*

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).

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. 11-19).[116,236]

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[233]:

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 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. 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

Filling the root canal with the lateral compaction method or instead using vertical warm gutta-percha carrier delivery

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. 11-19 Informed consent form.

systems, including heat-transfer units for warm gutta-percha, are both currently taught methodologies.[160] 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 compaction has a long-term proven track record, particularly with less-experienced practitioners, and is less technique sensitive.

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.[98]

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.[214] 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.[184] 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, since 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).[142]

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.[13] Identification of microfractures in teeth[55,149,160,191] 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."*[136]

Successful root canal treatment requires the prevention of microorganisms and 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.[63,64,241]

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.

New Products

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[10]:

> *"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 or researched products only tested in vitro. Again, "Don't be the first nor the last to adopt new technology."

Separated Instruments

Risk reduction of broken files can be accomplished 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 is also increased, 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.[189]

The clinician should save broken or defective instruments (e.g., a file that was broken inside a canal) (Fig. 11-20). The instrument manufacturer may be liable because the product was defective, rather than the clinician being liable for dental negligence.[215] 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 suckback. The clinician should inspect check valves monthly and change clogged valves immediately.[152] 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.[11] The clinician or staff should consider using filters and should flush dental-unit water lines 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. Fig. 11-21 shows a student who was burned by an inadequately maintained handpiece that overheated. This case settled for $280,000.

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

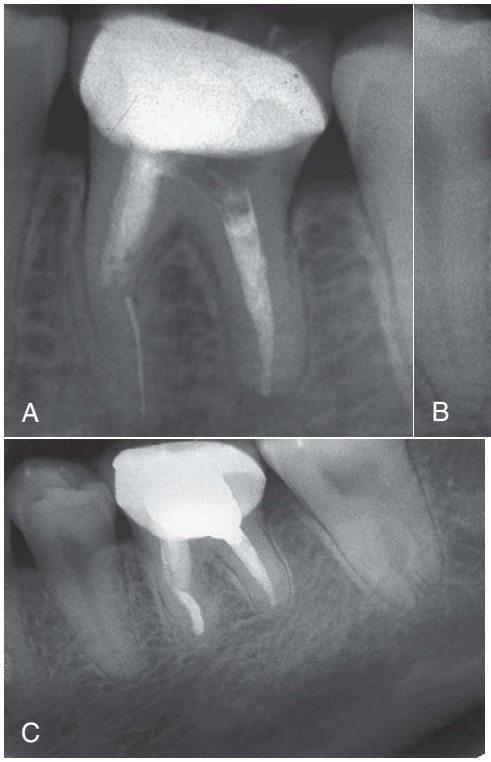


FIG. 11-20 A, Nickel-titanium file broken inside the mesiobuccal canal of the mandibular first molar. **B,** Due to patient discomfort, the segment was surgically removed and MTA was used as root-filling material. **C,** Periapical radiograph 2½ years post-treatment shows complete healing. (*From Torabinejad M, Walton RE: Endodontics: principles and practice, ed 4, St. Louis, Saunders/Elsevier, 2009.*)



FIG. 11-21 A, Before injury. **B-D,** A music student was burned by an inadequately maintained handpiece that overheated. This lawsuit was settled for $280,000.

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.[262] 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.[188] 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.[9]

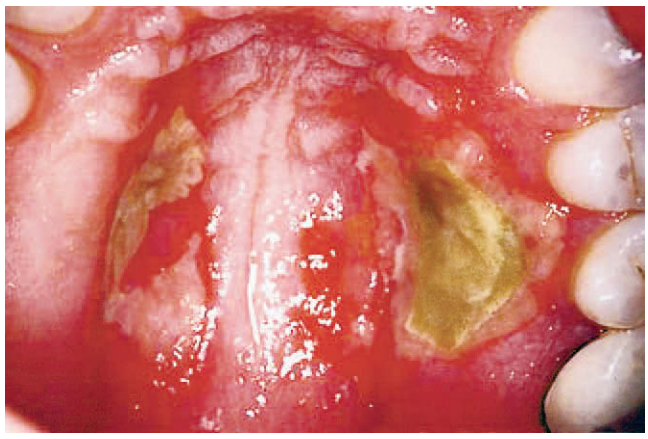


FIG. 11-22 Excessive local anesthetic resulting in palatal tissue sloughing with AMSA (wand) injection.

Product liability may include whether the manufacturer provides adequate hazard warnings for safe use of its product (Fig. 11-22).

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.[50,158] 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.[174,240]

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, and/or cell phones are 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 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.[33,264] 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 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.[190] 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:

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 the above 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.' "[139]

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.

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[59]) advises that 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 though continuing education and scientific journals, rather than an expert performing the research, is considered sufficient so long as the expert's opinion complies with the Daubert principle.

State courts are not bound to follow the Federal Rules of Evidence, but many do comply.[18] 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 end 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.*,[265] 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.*[59]

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.[61] The Public Citizens' Health Research Group report that more than half of U.S. hospitals have never reported an adverse incident to the National Practitioner Data Bank.[52]

Professional Negligence Incidence

In 2007, health care spending totaled $2.2 trillion, or 16.2% of the gross domestic product. Total spending for doctors' services was $393.8 billion.[259]

Harvard School of Public Health found that 97% of claims involving medical injury are meritorious.[203] 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.[156,256]

Tort filings in state courts, which comprise only 6% of all filings, declined by 21% over the decade 1997-2006.[194] Tort actions in U.S. District Courts also dropped 21% (2003-2007).[256] 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.[156,194]

A 2003 report submitted to Congress by the GAO found that malpractice awards are only one factor contributing to increases in premiums for medical professional liability insurance.[244] 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 report also stated that it is not possible to prepare a full analysis of the issue because comprehensive insurance data are not available.

Since 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 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.[22,244]

Some malpractice is inevitable. Even the best clinician is capable of making a 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. Forty-five percent of otolaryngologists admit medical errors in all phases of their practice. However, even this proportion of clinicians reporting medical errors is underestimated.[220] A recent study estimated the number of fatal medical errors doubled in the preceding 3 years.[61]

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 authorizations for treatment the clinician recommended as medically necessary. A recent 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

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.

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 and/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 confirms or rules out IANC penetration post treatment, promoting prompt removal of foreign materials piercing the IANC.

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 the IANC. 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.[85,146,168,234,271] Overfilling and/or overinstrumentation of the root ends can cause traumatic and/or chemical injury to the IAN bundle, resulting in potentially permanent altered or total loss of sensation from paresthesia (partial numbness), anesthesia (total anesthesia), and/or dysesthesia (burning pain).

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. 11-23, *A-C*), (2) inadequate measurements during cone measurement, and (3) not limiting filling materials to within the confines of the shaped root canal space as further evident in CBCT.

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[180] 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.[206] Better yet is to take precautions to prevent ANY significant injurious overfill.

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. In addition to 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.

Precise measurements during cleaning, shaping, and obturation compacting stages of endodontics minimizes significant overfill incidents. Obturating filling materials are not only toxic to nerve cells but also create a mechanical pressure injury to the nerve cells themselves. Immediate microsurgical referral for retrieval of any overfill materials which "harpoon" the IANC protect against irreversible neuropathies (see Fig. 11-25, *C*).[206]

Serious mechanical and chemical injury to the IAN results from a lack of recognition of the overfill piercing the IANC and/or disregard for neuropathic symptoms the patient reports postoperatively that are consistent with neurologic deficits.

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.[206] 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 nature of overfill errors.

After 3 months, microsurgical delay will almost close the window of opportunity to prevent permanent neurologic deficits of altered sensation, with resulting pain, numbness, and painful burning symptoms extrusion overfills into the IANC can cause. Stat referral for microsurgical removal of paraformaldehyde-containing root canal filling materials and sealants

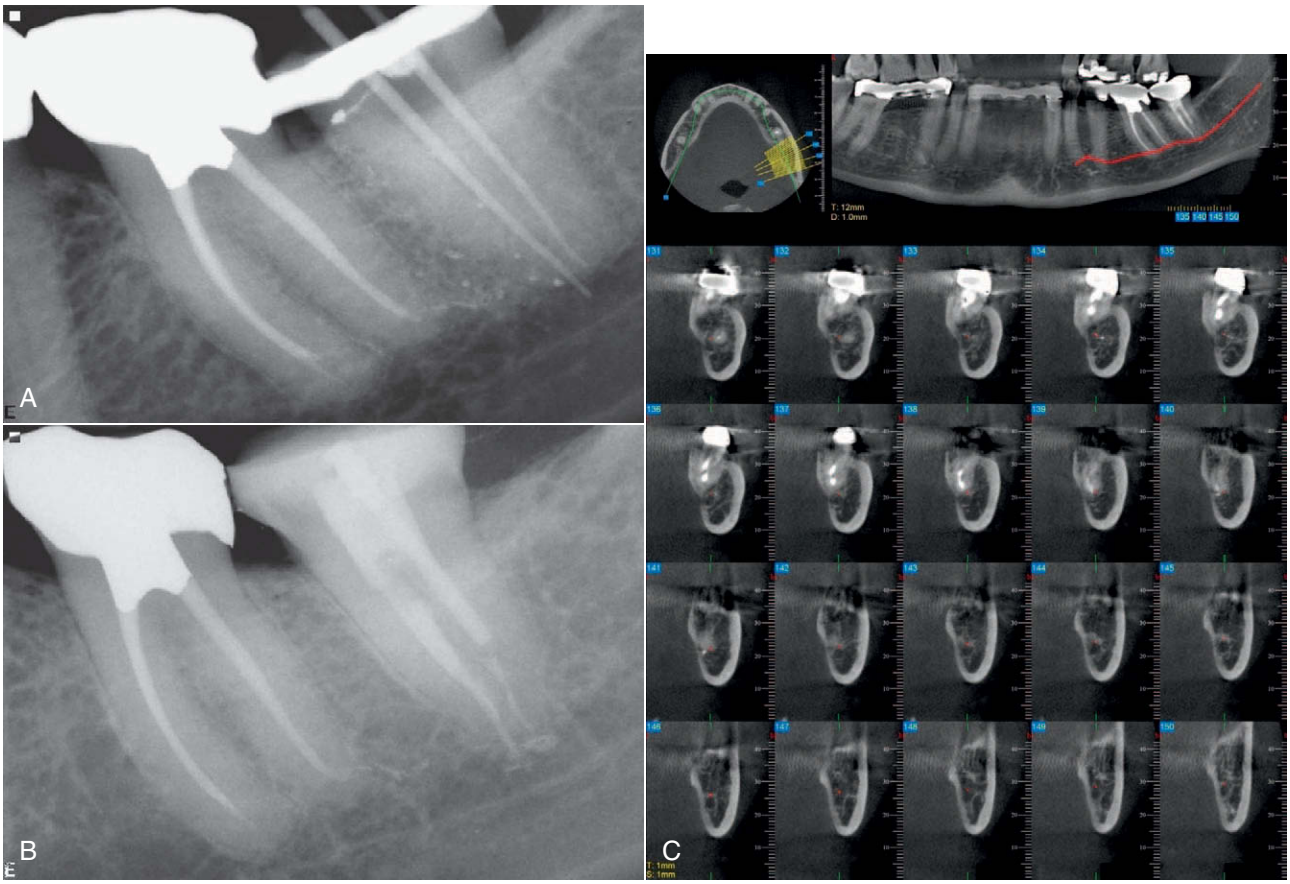


FIG. 11-23 A, Unclear periapical to show both cortical borders of inferior alveolar nerve canal (IANC). **B,** Cones placed into IANC, showing measurements beyond apices. **C,** Overfill into IANC better shown on cone-beam computed tomography (CBCT) than periapicals of **A** and **B.**

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.[34,99,115,155]

For needed stat referrals, the offending clinician should phone, write to, 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 accomplished.

Treatment Failure

A clinician should not guarantee treatment success. It is foolish to assure the patient of a perfect result.[49] Endodontic failures may occasionally occur despite adequate endodontic care.[131,249] 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,[48,237,241] overextension errors, and inadequate isolation of the tooth from salivary contaminants during instrumentation due to lack of a rubber dam.

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.[116,236] 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—for example, a rate that varies markedly from national statistics, which also suggests referral to an endodontist.

An endodontic treating clinician is also liable for failure to disclose evident 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.

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 paraphrase 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. A Senate bill was sponsored but has not yet been passed by 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 follow-up plastic surgery.

Leakage

Long-term seal of the root canal system is determined apically by the sealer and coronally by the final restoration.[24,106,189,237] Root canal–filled teeth should be permanently restored without undue delay to prevent leakage contamination of the previously obturated canal system, since varying canal shapes from round to oval prevent a 100% seal.[63,64,164] Bonded seals covering the canal surfaces should be used to control any leakage after compaction until a permanent restoration is cemented. 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.[63,241] Younger patients are more susceptible to bacterial penetration inside dentin tubules and thus recurring infections.[143]

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.

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.[112] 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 was readily apparent radiographically, the existence of a fourth canal should have been considered, and treatment should have extended to instrumenting and sealing it.

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.

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 loose the cement around posts. 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.[72,102,191] Fig. 11-16 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.[204]

Broken Files

Leaving broken files behind without referring the patient to an endodontist for attempted microscopic retrieval and also not advising the patient of leakage potential may constitute

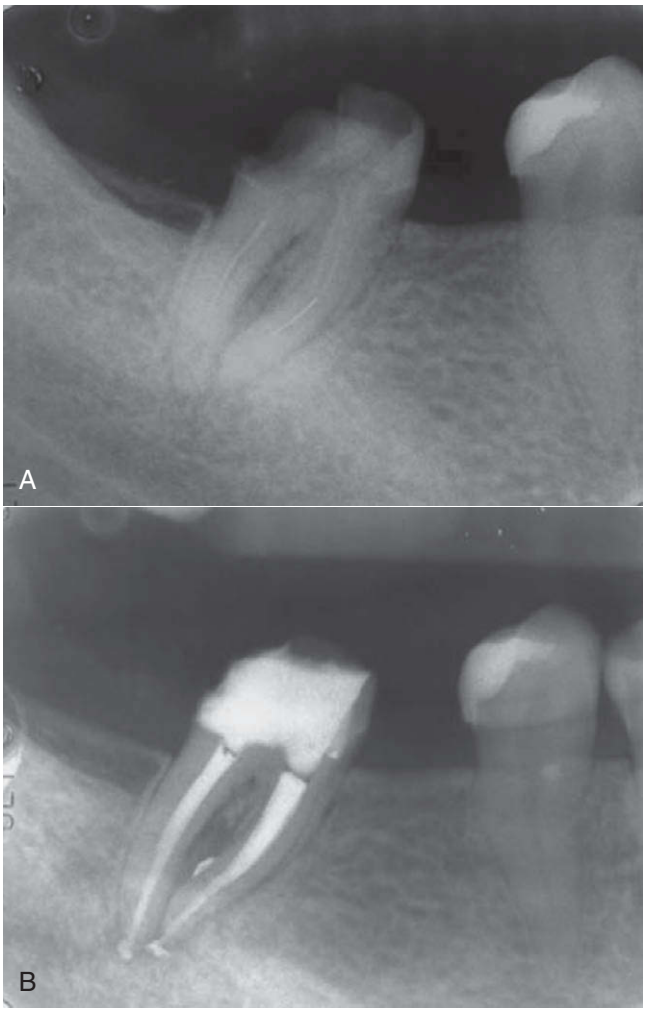


FIG. 11-24 A, Tooth #31 with three separated files, one in each canal. K-files used for cleaning canals and also obturation (1996). **B,** No apparent radiographic changes in 1998.

fraudulent concealment (Figs. 11-24 and 11-25). Patients should be informed of such mishaps for (1) referral consultation and/or treatment; (2) advising the patient, who on their own may seek a second opinion; or (3) disclosure, so as to return if a flare-up occurs.

Swallowing or Aspirating an Endodontic Instrument

Use of a rubber dam in endodontics is mandatory.[238] 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, the risk of a patient's aspirating or swallowing an endodontic instrument is eliminated (Fig. 11-26). 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 what occurred.
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

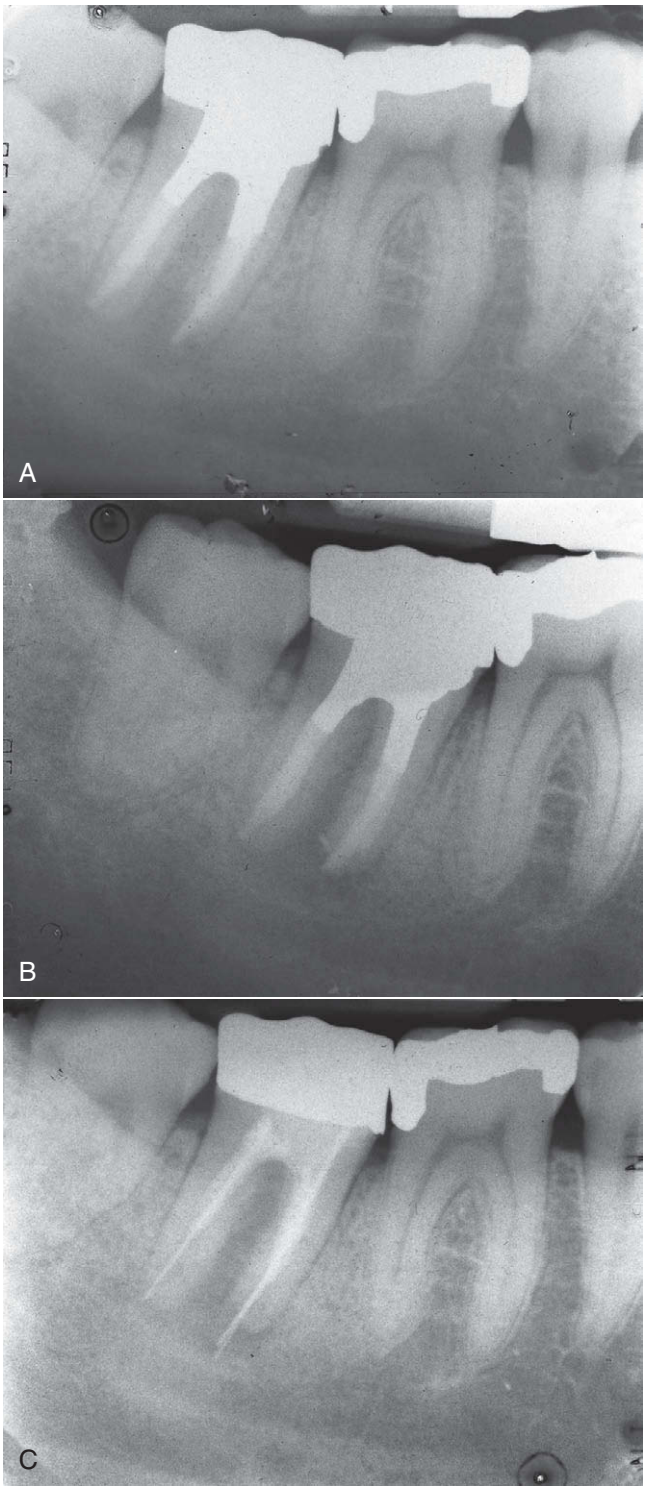


FIG. 11-25 A, Apical radiolucent changes with draining 9 mm buccal abscess (Aug. 2003). **B,** Endodontist retreatment (Aug. 2003). **C,** Successful retreatment with no buccal pocket (Sept. 2003). (*Courtesy James YS Ho, DMD.*)

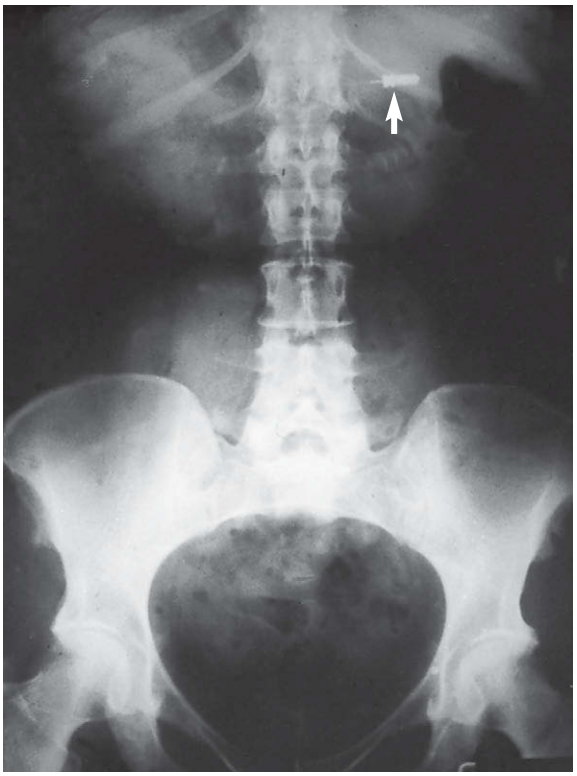


FIG. 11-26 Swallowed endodontic instrument demonstrates the wisdom of using a rubber dam.

policies will cover the incident as "medical payment" for an accident and not as a malpractice claim.

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.[168] (See Fig. 11-25, *B* periapical, which may appear to show only one cortical border, and Fig. 11-25, *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. Since 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 the CBCT confirms an overfill inside the IANC. In Fig. 11-25, *B*, the defendant endodontist misdiagnosed the overfill as being superior rather than inside the IAN canal, with resultant permanent paresthesia from only watching and waiting for sensory return rather than referring for a stat microsurgical removal.

A very slight overextension of root canal filling with conventional obturation or sealants can occur without violating the standard of care (see Chapter 10). Gross overfill usually indicates faulty technique. Fig. 11-27, *A-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 IAN or sinuses, permanent harm is unlikely unless the root canal is filled with a paraformaldehyde-containing sealant (causing a neurotoxic chemical burn type of injury which chemically diffuses through bone marrow and soft tissue spaces).

If, however, severe postoperative pain is foreseeable as a result of overextension, the patient should be advised of the likelihood of postoperative discomfort because of contact of the sealant material with surrounding tissue. Similarly, if the overextension is very slight and increased postoperative pain is unlikely, the patient need not be advised, lest it cause unnecessary alarm. A note should be made on the patient's chart of the overextension and the reason for not informing the patient, in case symptoms later manifest. The clinician should observe with close follow-up visits and patient phone calls that evening and the next morning to rule out severe postoperative persistent numbness and/or pain resulting from moderate to large overfills. Fortunately, slight to moderate overextensions with inert conventional endodontic sealers, such as gutta-percha with Grossman's sealant, often repair themselves and produce no irreversible changes without direct contact into the sinus or IANC.

Overextending the root canal filling material risks permanent consequences if the underlying IAN is initially "harpooned" with files. 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 showed more deviation (+0.6 mm) from the perioperative measurement.[85,234] As noted earlier, 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]).[224] On the other hand, conventional obturators and sealants usually require direct contact with the IAN before resulting permanent anesthesia, paresthesia, and/or dysesthesia occurs (Fig. 11-28). Consequently, the incidence of permanent sequelae with conventional filling materials is extremely low compared with the greater cytotoxic potential with paraformaldehyde-containing sealants.[195] 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. Permanent injury risk is substantially less likely with traditional eugenol-containing filling materials, a less toxic chemical than paraformaldehyde, which destroys and mummifies nerve tissues. When safer, less risky alternative 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 the overextended gutta-percha. 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

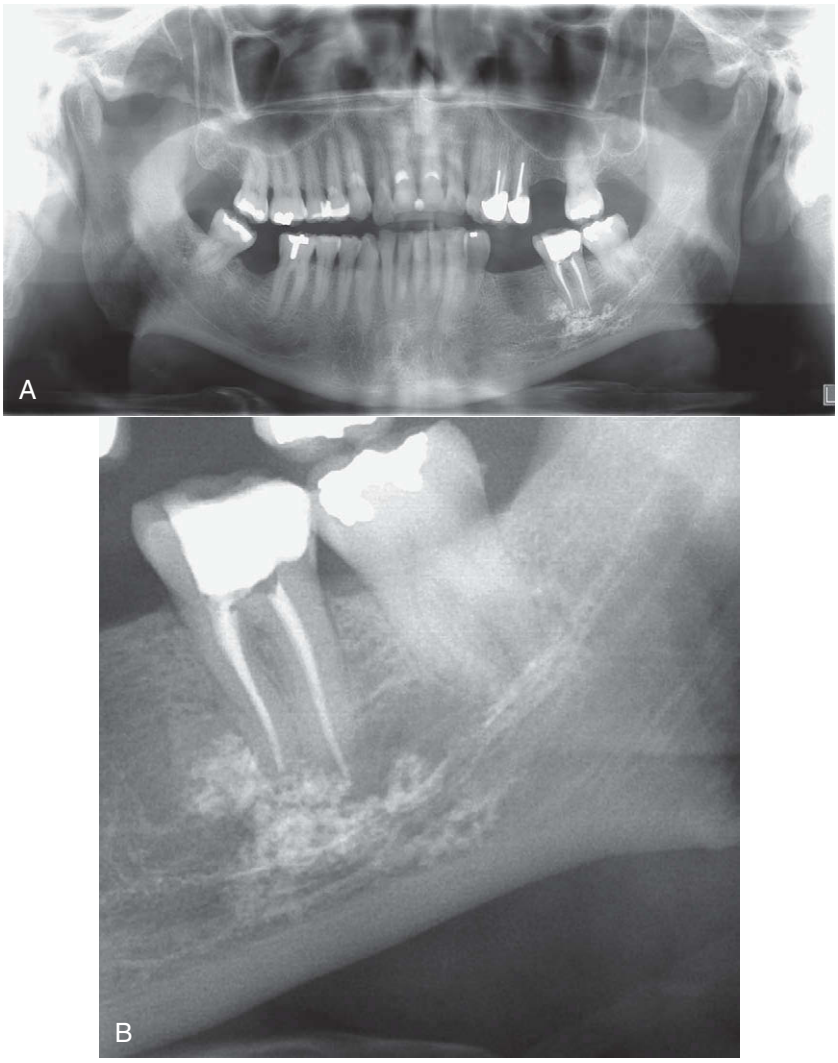


FIG. 11-27 A-B, Calcium hydroxide overfill, causing permanent paresthesia.

gutta-percha do not penetrate the cortical walls of the IANC unless 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 to 72 hours).[206] 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 to 72 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.[180,206]

Inflammatory edema that compresses and compromises blood supply to soft tissues and nerves in limited spaces with resulting ischemia is termed *compartment syndrome*.[230,274] 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 closed bony nerve compartment has the potential for

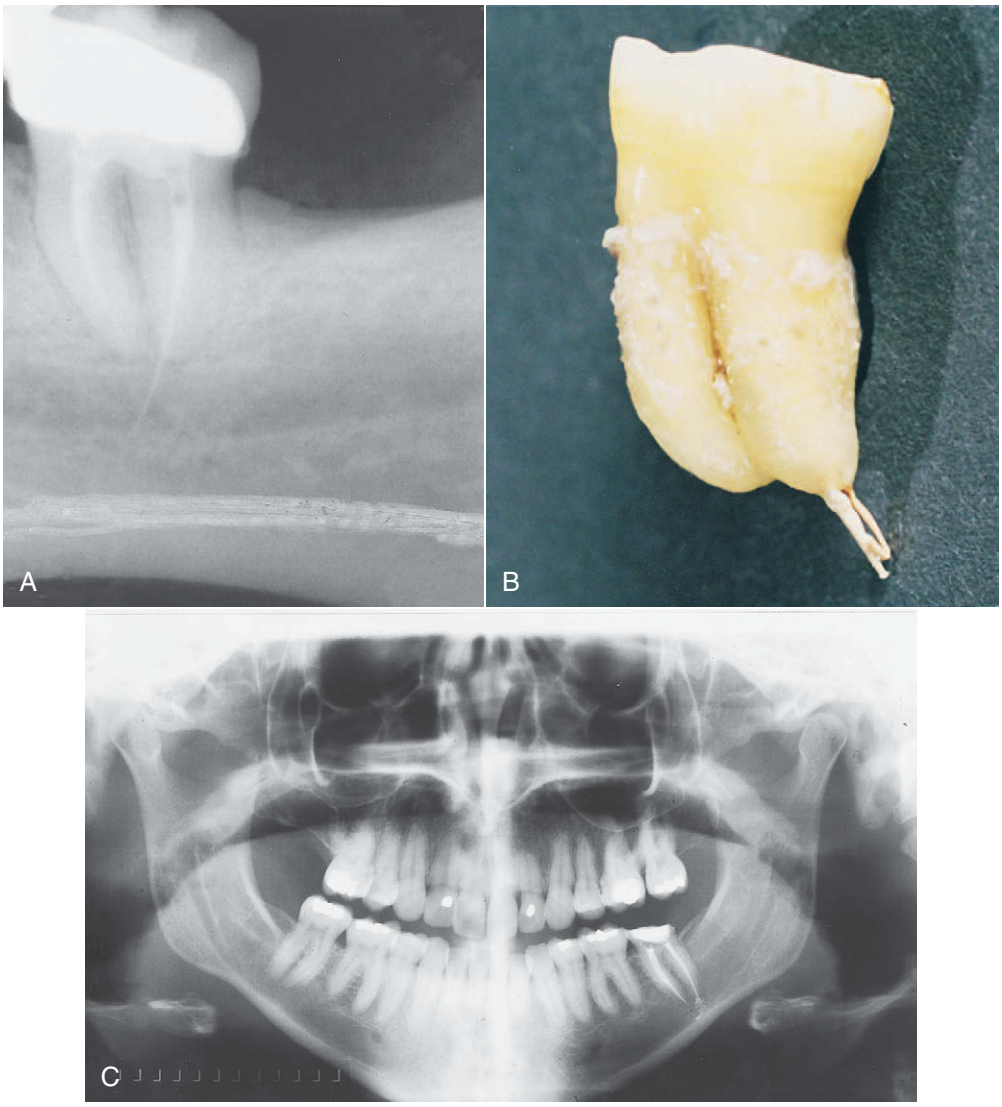


FIG. 11-28 A-C, Overextended gutta-percha and extracted tooth with gutta-percha intact.

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 IANC enclosed by bone on all sides.

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 current standard of care. This is because silver points corrode in time, and a tight 3D apical seal is lost. Fig. 11-29, *A-C* represents gross overextension with a silver point that ultimately caused the loss of tooth #14 as a result of endodontic failure.

N2 (Sargenti Paste)

Dental literature reports that permanent paresthesias are associated with gross overfilling with paraformaldehyde sealant (N2) that are not usually associated or reported with conventional sealants (Fig. 11-30).[162,195] 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 never makes it right.

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 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. Reasonable 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 upon pseudoscience is not controversial, it is fraudulent practice.[25,185] 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 regarding decortication and decompression surgery.

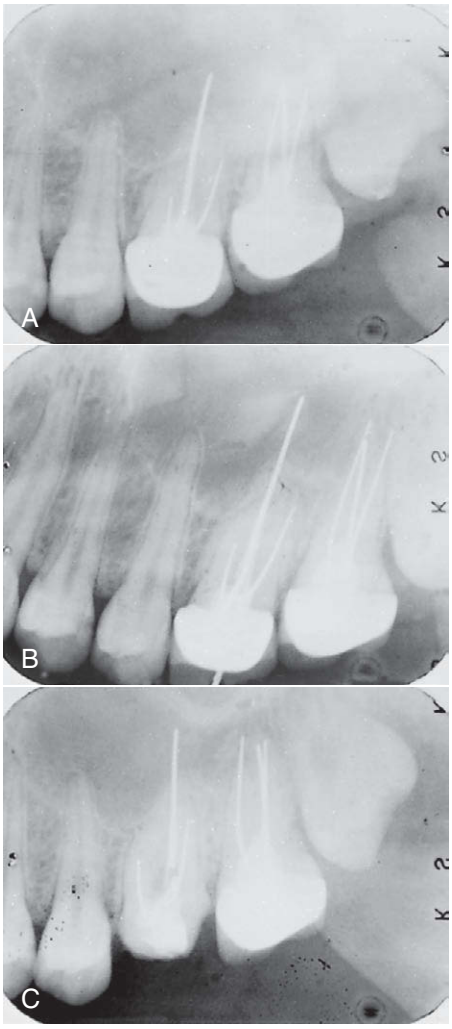


FIG. 11-29 Gross overfill into sinus with a silver point, which ultimately caused sinusitis and loss of tooth #14 as a result of endodontic failure.

The Federal Food, Drug, and Cosmetic Act prohibits interstate shipment of an unapproved drug or individual components used to compound the drug.[76] 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.[46] 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.[132]

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.[24] 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).[34]

PROPHYLACTIC ENDODONTIC PRACTICE

Malpractice Prophylaxis

Most negligently injured patients do not sue.[35] Nonetheless, litigation serves a prophylactic purpose since 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

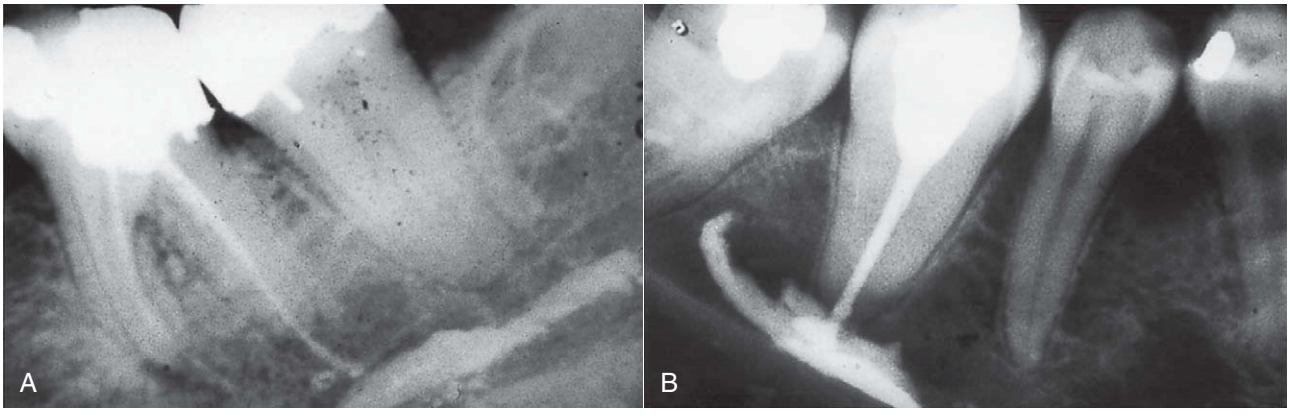


FIG. 11-30 A-B, Overextensions of Sargenti paste filling the inferior alveolar nerve canal. Both cases could have been avoided if the practitioners had selected a conventional sealing material and used a technique that emphasizes length control.

harm, deaths and professional liability premiums decreased dramatically.[204,228]

Disclosure Errors

Should dental negligence harm a patient, then full disclosure, including accepting the clinician 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.[172] 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.[148] Protecting the patient's best interest includes telling the patient when negligent errors occur.

Periodontal Examination

Competent endodontic treatment begins with adequate diagnostic procedures. An adequate periodontal evaluation must accompany each endodontic diagnosis, which requires a diagnostic radiograph, clinical visualization, mobility recordings, evaluation of the periodontal tissues, and probing for periodontal pockets with a calibrated periodontal probe, particularly in furcation areas.[7,8,173]

Although endodontic treatment may be successful, tooth loss may result from progression of any residual, untreated periodontitis. Consequently, periodontal evaluation and prognosis are mandatory so that the patient and clinician can make an informed and intelligent choice about whether to proceed with endodontics, a combination of periodontal and endodontic treatment, or extraction.

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.[7,8]

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.

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.[157] Hyperocclusion is a potential TMD trigger that results from open bite or a contralateral side, particularly when a terminal abutment tooth is taken out of occlusion.

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 is mandatory.
2. Measurement films or digital images are necessary to verify canal length (if an electronic canal-measuring device is not used) 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 10).

Digital Radiography

Digital radiographic endodontic applications are ever increasing in accuracy.[113] 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. Fig. 11-31, *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[37,113]:

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.)[3]
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 be devastating to the clinician's defense.

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.[54,97,127] In Fig. 11-32, *A-C* the PA does not show the

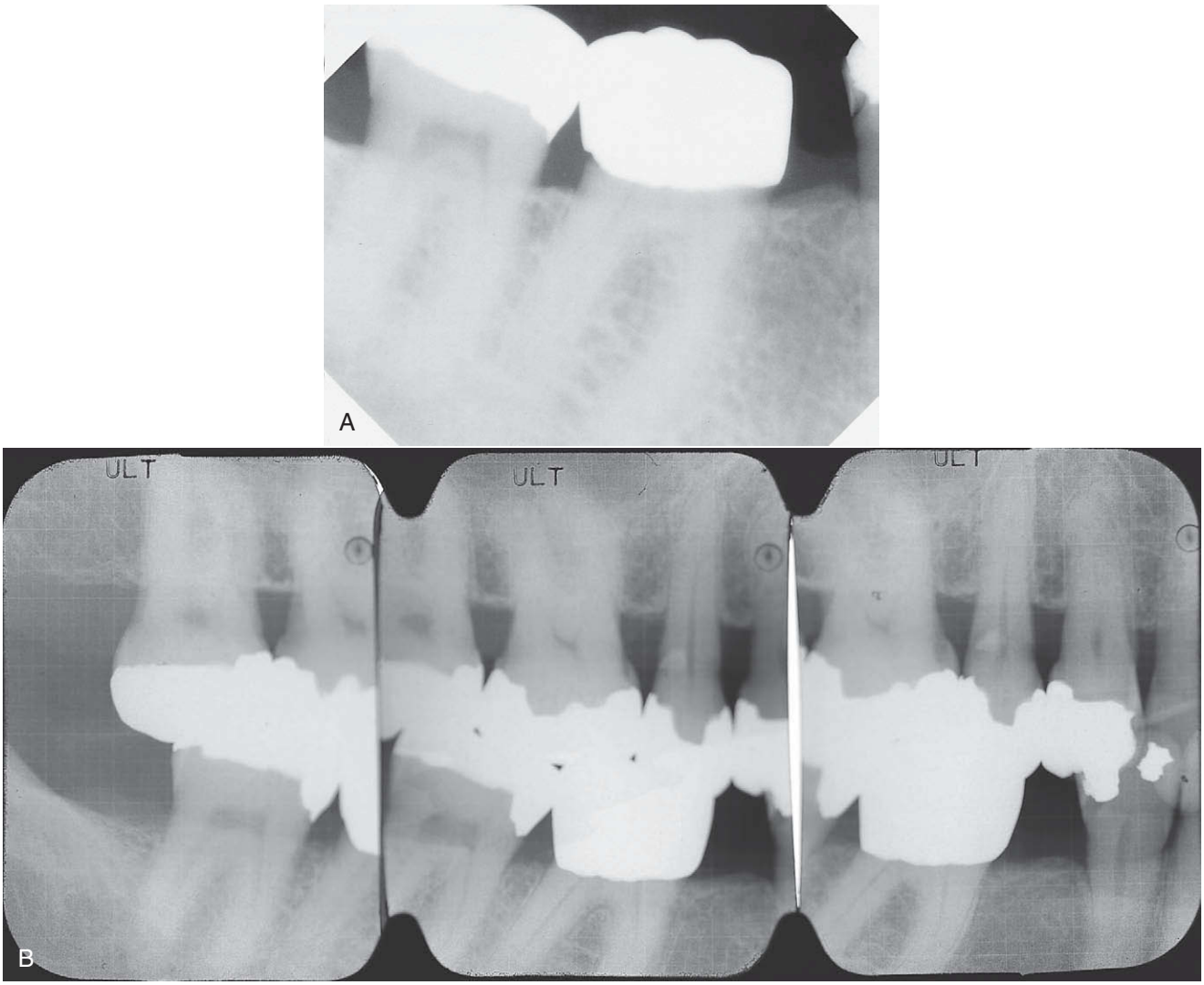


FIG. 11-31 A, Distal open margin on tooth #30 (shown digitally). **B,** Distal open margin on tooth #30 not evident with plain-film radiography.

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.

CT scans enable evaluation of the true extent of lesions and their spatial relationship to important anatomic landmarks.[73] Fig. 11-33, *A-C* shows a bifid IANC not apparent on panorexes. CT scans may provide information important for retreatment decisions, especially apical surgery. CBCT[54,163,166] showed significantly more lesions than PA radiography, including expansion of lesions into the maxillary sinus, sinus membrane thickening, and missed canals; 34% of such lesions were missed by PA radiography.[163,166] 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 maxillary premolars and molars for apical surgery,[166] 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.[163] 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 diagnosed with CBCT were more frequently detected with CBCT than with PA radiography.[163]

Apicomarginal communication is an important predictor for the success rate of apical surgery including evidence of undetected vertical fractures necessitating extraction; 83% of

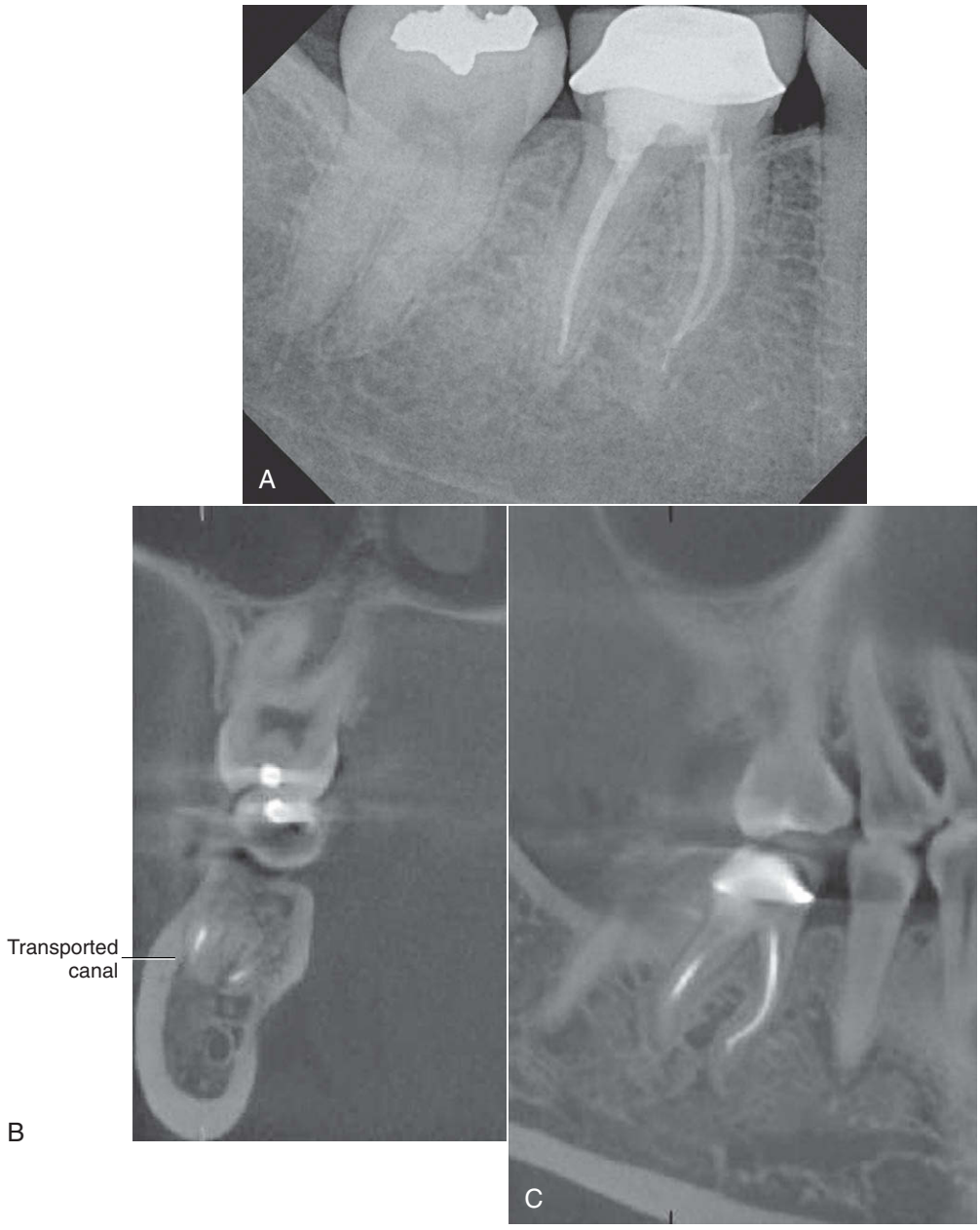


FIG. 11-32 A, Periapical (PA) view, which does not show full extent of transported and short-filled canal. Both deficiencies evident in **B** and **C** with cone-beam computed tomography (CBCT). **B,** CBCT Accuitomo of transported canal. **C,** CBCT Accuitomo of short-filled canal.

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, since 70% of cases studied revealed clinically relevant information not found in PA radiography.[166]

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. In summary, 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.[54,166]

Dental Fear

Dental fear may result in patients delaying or avoiding dental care. Frequent cancellations and missed appointments are characteristically associated with fearful dental

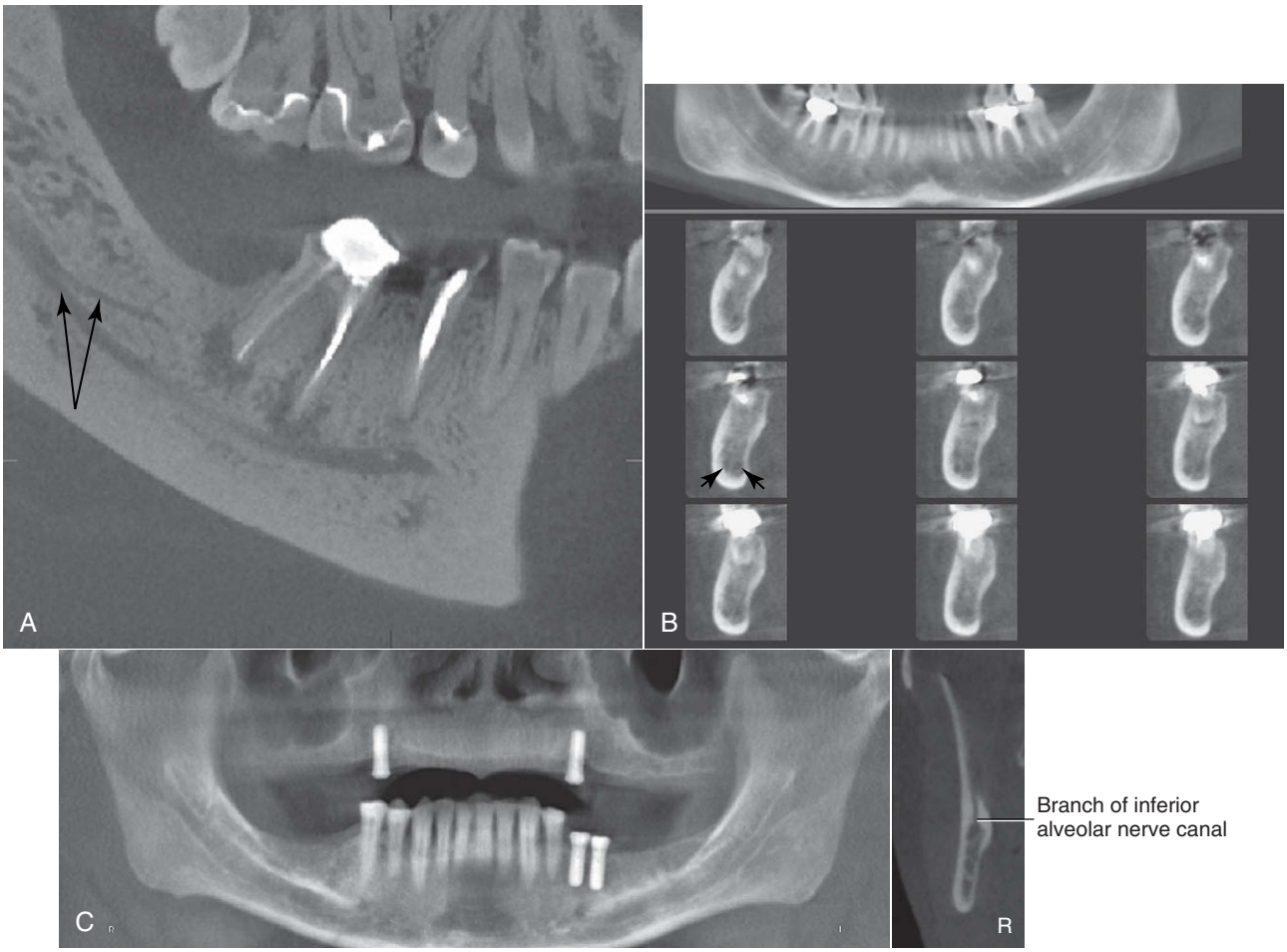


FIG. 11-33 A, Bifid inferior alveolar nerve canal (IANC) visible with cone-beam computed tomography (CBCT). **B,** Bifid canal visible with CBCT but not shown on panorex. **C,** Branch of IANC apparent on CBCT but not shown on panorex. *(Courtesy Gurminder Sidhu, DDS, MS.)*

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 cancelled 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 psychologic questionnaires, such as the Dental Anxiety Scale or the Modified Dental Anxiety Scale, may be helpful in identifying such individuals.[125] 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 psychologic 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.

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 reason-

able 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.

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

Full attention to the patient's complaints, good eye contact, and respectful addressing of the patient 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 upon 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 and/or reasonably suspected the negligent cause of the injury, not the date of the injury itself.[67,92,212,266] 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.[224] In 2008, a Spokane, Washington, jury awarded $14.2 million for unnecessary TMJ surgery. In Fig. 11-34, a Los Angeles jury awarded $1.7 million against a periodontist who "harpooned" the IANC with his implant.

Incidence of Negligence

Medical errors or adverse events are estimated to occur in almost one out of four patient visits to a family practitioner.[218] A U.S. government study of the Institute of Medicine found that 152,000 avoidable deaths occur annually because of medical negligence.[93,95] 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, since calculations were based solely on hospital chart review.



FIG. 11-34 Implant "harpooned" the inferior alveolar nerve canal.

Alcohol

Excessive alcohol consumption can contribute to a higher incidence of restoration corrosion from chronic regurgitation and vomiting from gastritis associated with alcohol abuse.[110] Corrosion can lead to coronal marginal leakage predisposing to endodontic failure. Accordingly, medical histories should include histories of gastric reflux disease, bulimia with associated habitual vomiting, gastritis, and excessive alcohol intake.

Post Perforation

Post selection is important for minimizing the risk of perforations. 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 23. When a tooth is already crowned, access to the root canal entrance 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. Fig. 11-35, *A-B* represents an endodontist's perforation of tooth #7 because of difficulty locating the entrance into the root canal apical to the pulp chamber.[82] Fig. 11-36 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 perforation is relatively small in size (i.e., 1 mm or less) and promptly diagnosed at the time of the post perforation, immediate treatment with intracanal sealants (MTA) 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 prognosticates a high failure risk.

Perforation Prevention

Irreversible endodontic complication such as furcation perforation during pulpal chamber access is usually preventable with due care.[189] 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.[65]

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. Irrigation

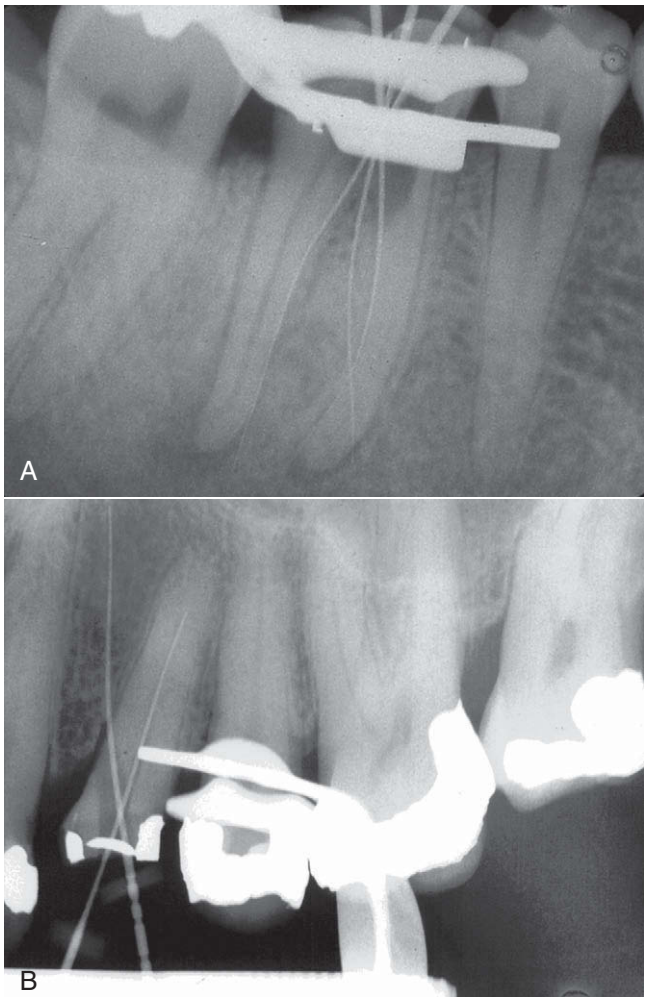


FIG. 11-35 Avoidable perforation. *(Courtesy Stephen Cohen, MA, DDS.)*



FIG. 11-36 Post perforation of tooth #5.



FIG. 11-37 Bleach injury.

with sodium hypochlorite is the usual disinfectant irrigant to minimize these remaining bacteria.[217] 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 the same visit, despite a bleach injury.[177,223] One article reports that subsequent flare-up may be related to the original bleach injury.[36]

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.[151] However, if the bleach diffuses into vital nerve tissue of the infraorbital or inferior alveolar nerves, permanent paresthesia and occasional dysesthesia may result.[75,176,201,271] Figs. 11-37 and 11-38, *A-C* represent bleach injuries secondary to a root canal perforation. Paresthesia persists. Note the PA does not show the perforation, but the Accuitomo does.

Cores

Incorrect choice of cores can contribute to failure, including fractures. Some manufacturers (e.g., ESPE Premier, Norristown, PA, 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

*References 2, 75, 151, 176, 201, 217, 211, and 271.

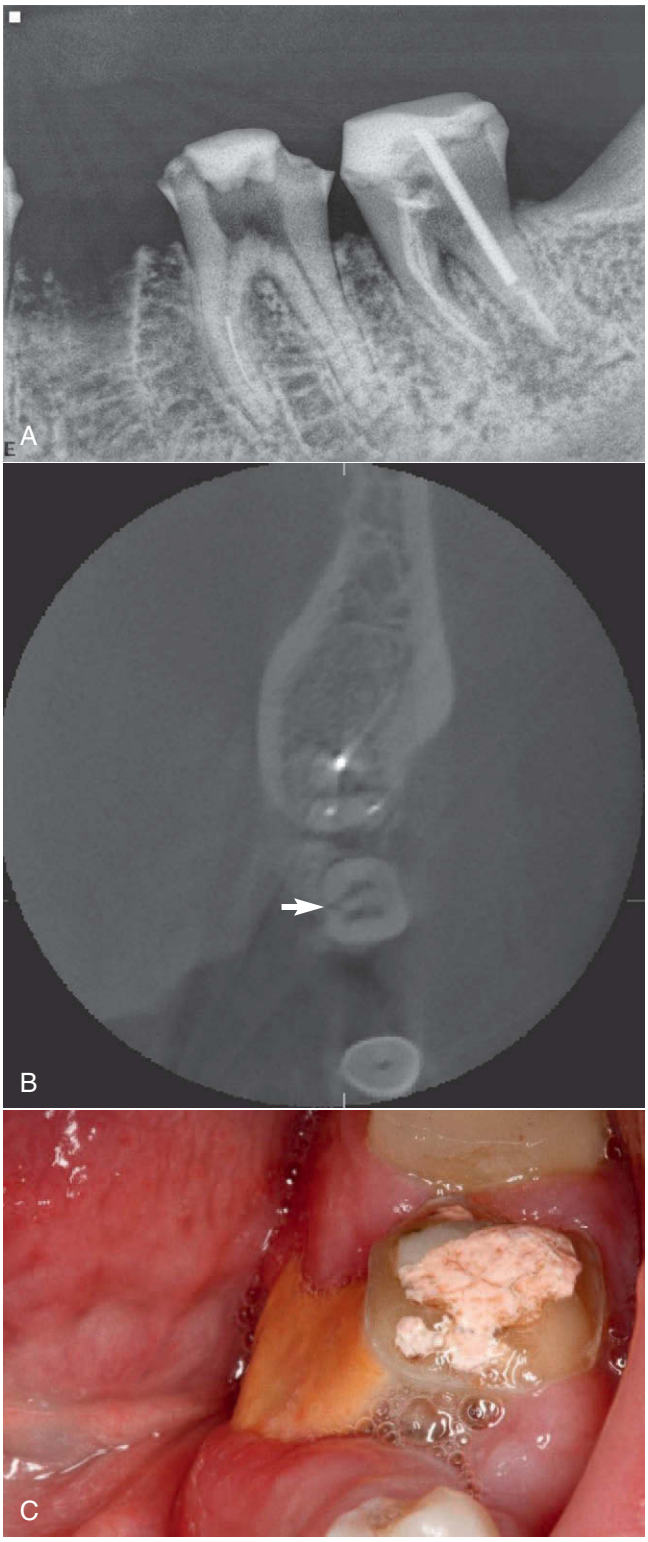


FIG. 11-38 A, Periapical tooth #19 showing fractured file but no perforation. **B,** Accuitomo cone-beam computed tomography (CBCT) showing perforation on the lingual side above the tooth #19 furcation. Perforation not evident in periapical view. Image demonstrates Accuitomo accuracy. **C,** Bleach extrusion through tooth #19 perforation, with resultant slough of lingual tissue.

not been reported. A ferrule or other counter-rotational core design is an important consideration for fracture resistance and retention,[126] although it has not been proved as statistically significant for the resin-reinforced core systems.[225]

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.[80,162] Also, as such 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.[80,162] 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.

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.[27,243]

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 manufacturing defect. Fig. 11-39 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.

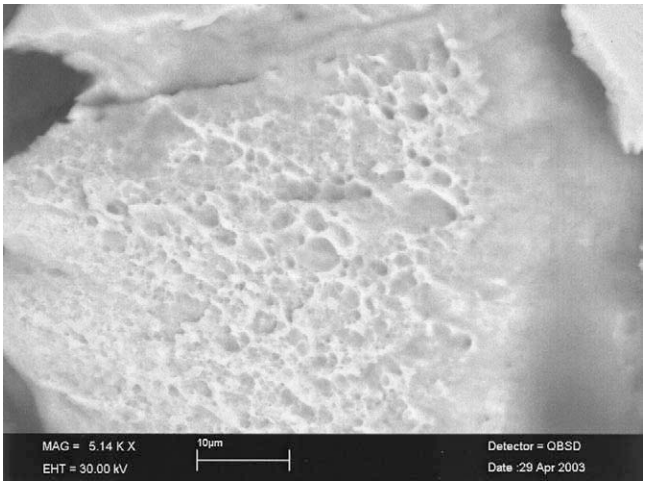


FIG. 11-39 Tensile overload shown in elongated ductile dimple voids.

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. Fig. 11-40 shows the needle tip bent and blunted as it hit a hard object. No manufacturing defects were found. Fig. 11-41, *A-B* shows SEM views of the fracture. Fig. 11-41, *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.[169]

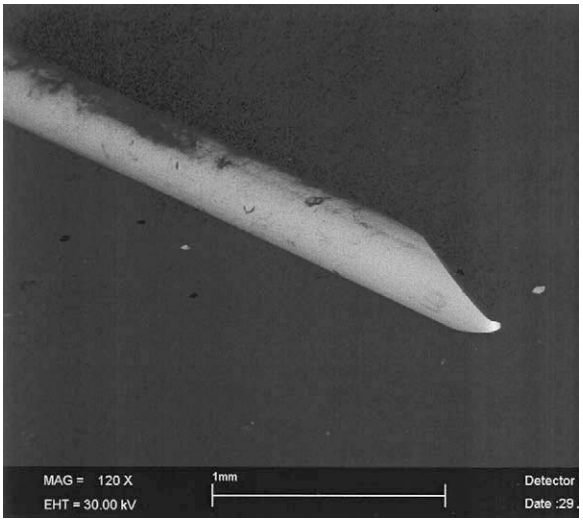


FIG. 11-40 Needle blunting of broken needle tip demonstrating striking hard object (ramus).

Sterilization

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.[152] 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.

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.[122]

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.[19]

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, which usually ascends as it approaches the mental foramen, com-

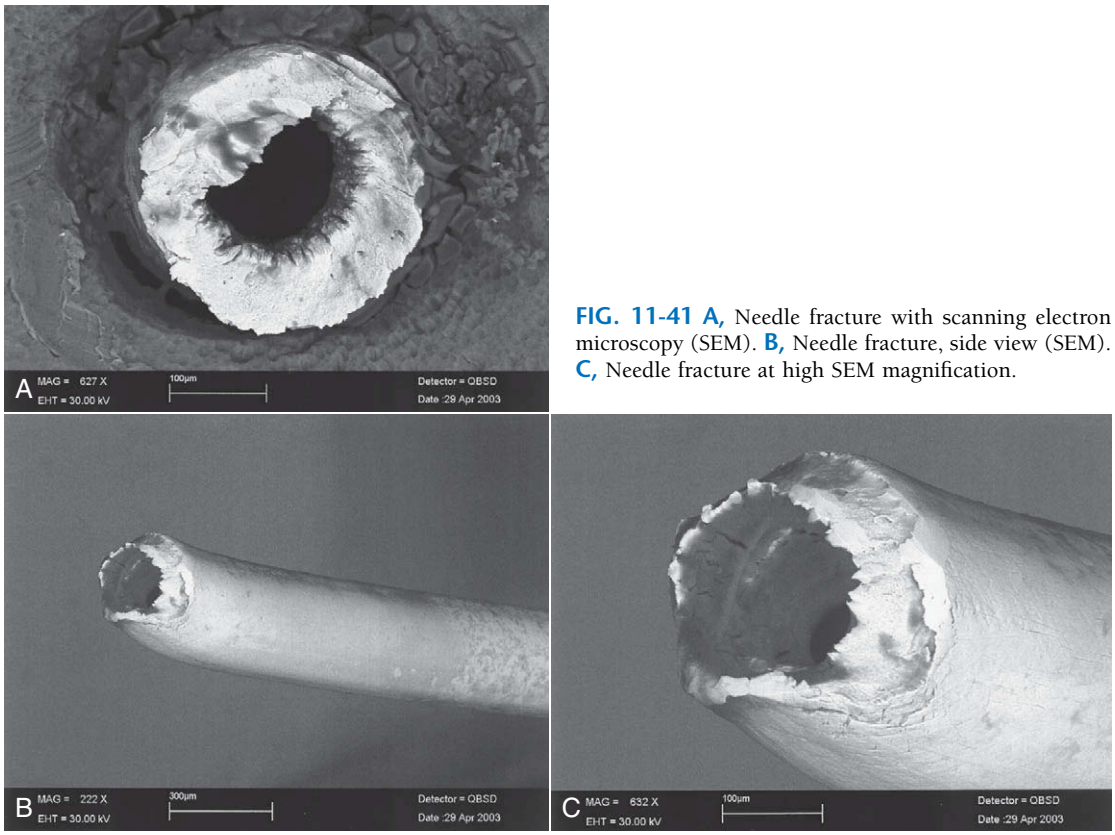


FIG. 11-41 A, Needle fracture with scanning electron microscopy (SEM). **B,** Needle fracture, side view (SEM). **C,** Needle fracture at high SEM magnification.

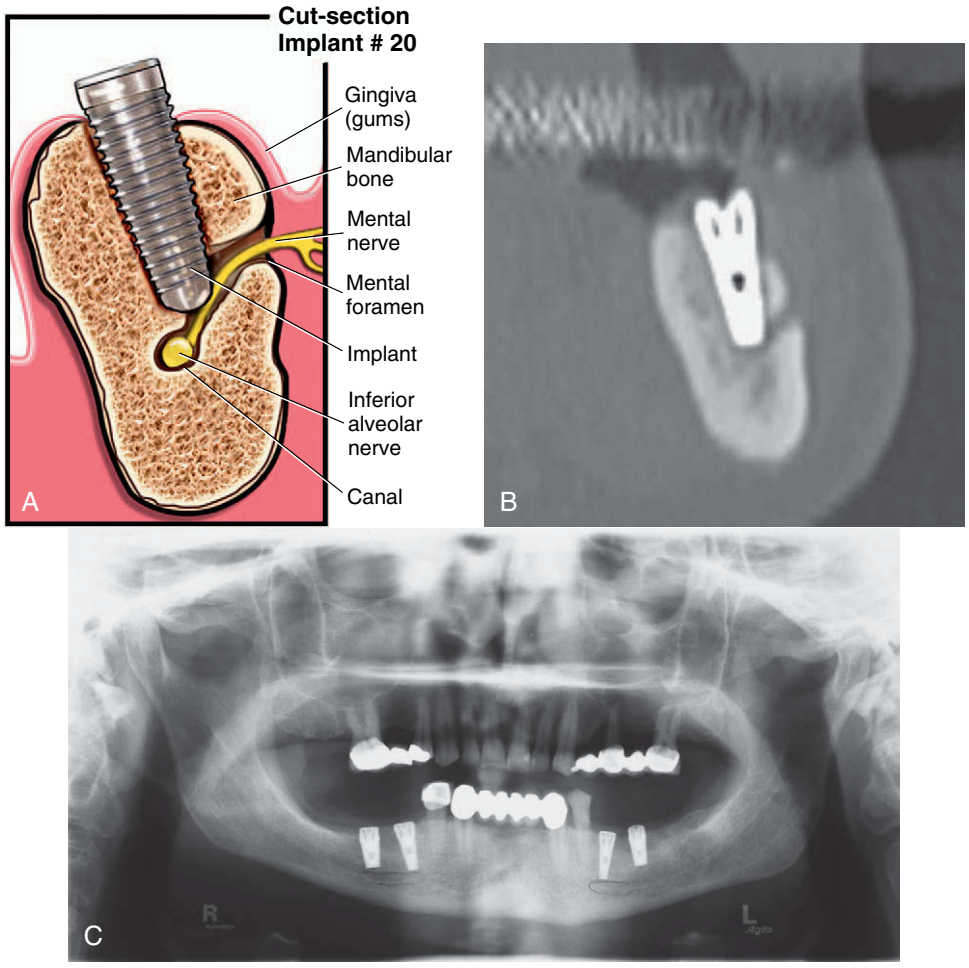


FIG. 11-42 A, Illustration showing implant placed into ascending inferior alveolar nerve canal (IANC). **B,** Cone-beam computed tomography (CBCT) image of implant into ascending IANC. Settlement of $915,000. **C,** Implant into IANC; immediate postoperative Panorex image. Same patient but with CBCT image.

pared with the IAN's more inferior location in the molar region (Fig.11-42, *A-C*[109]; this Los Angeles case settled for $915,000, with permanent dysesthesia). 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.[54,73]

Mental Nerve

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.[169] 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.[63,146]

Medication Errors

The FDA has received over 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.[153]

Medication errors are often preventable.[247] 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 17 for a full discussion of how to treat and manage patients who have sustained a traumatic injury.

Continuing Education

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.

Millennium Management of Endodontic Advances

Technologic advances are touted as ideal endodontics. However, the standard of care is a minimal standard of 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 endodontics 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 IAN or mental foramen. Fig. 11-43 shows an example of volumetric tomography usage for implant placement and/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.[77,209] Microscopic apical surgery has gained general acceptance, is performed by the majority of endodontists, and represents the current standard of care.[55,136,149]

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 three patients. Valid scientific principles mandate that a test result can be replicated by other competent scientists duplicating a particular research protocol. 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.



FIG. 11-43 Cone-beam computed tomography usage for implant placement and/or apical surgery.

Statistical Research

Statistical research conclusions may vary, since research protocols may not similarly control variables of population groups studied.[108,205] 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.[124]

Since 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 rather 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 investigator 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.[254]

*References 14, 88, 153, 159, 216, 222, 229, 239, 254, and 272.

Published clinical research is too often biased, usually by designing the studies in ways that will almost inevitably yield favorable results for the sponsor.[178,272] For example, comparator 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 compare the new drug with an older FDA-approved drug at equivalent doses. As the former editor for 2 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."[14]

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.[30,98] In general do not be the first nor 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 are pertinent examples.[54,127]

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 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.[10,97] If the patient is 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. Peer review proceedings, including the committee's decision, are not admissible in court.[43] 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. 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.[60] 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.[12] Many states already offer additional protection under state law.[40,62]

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 such information without authorization. Therefore, employees should sign the confidentiality agreement shown in Fig. 11-44. 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

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. 11-44 Dental auxiliary confidentiality agreement.

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. 11-45 Sample of written physician recommendations.

that patient. This information is essential to staff members who may come in contact with the infection. 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).[66] 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.[35]

Infective Endocarditis

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.[269] 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. A sample format is shown in Fig. 11-45.

Individuals who have undergone total joint-replacement surgery may arguably be at increased risk for bacterial infection to the artificial joint. Late joint infections (i.e., less than 6 months) may potentially occur as the result of bacteria introduced into the oral cavity. Antibiotic prophylaxis may be recommended for certain individuals with total joint replacements before specific dental procedures (as recommended by the AHA). The drugs (e.g., cephalexin, cephradine, amoxicillin) used to prevent the late infection of prosthetic joints differ slightly from those recommended by the AHA.[6,133] The treating orthopedist's recommendation is the guidepost clinicians should consider during the first 6 months after joint replacement surgery.

CONCLUSION

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 help lessen the likelihood of litigation by reducing, if not eliminating, avoidable risks associated with endodontic care.

Both the patient and clinician benefit from risk reduction.[94] 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 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 protect the patient's best interest as our paramount goal. No matter how many others do it wrong 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

12

Structure and Functions of the Dentin-Pulp Complex

KEIJO LUUKKO, PÄIVI KETTUNEN, INGE FRISTAD, and ELLEN BERGGREEN*

CHAPTER OUTLINE

DEVELOPMENT
- Stages of Development
- Development of the Tooth Nerve Supply
- Molecular Control of Trigeminal Axon Navigation
- Differentiation of Odontoblasts
- Root Development
- Accessory Canals in the Root

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- Primary, Secondary, and Tertiary Dentin
- Mantle Dentin
- Circumpulpal Dentin
- Predentin
- Dentinal Tubules
- Intertubular Dentin
- Intratubular Dentin
- Dentinal Sclerosis
- Interglobular Dentin
- Dentinal Fluid
- Dentin Permeability

MECHANICAL PROPERTIES OF ROOT DENTIN

MORPHOLOGIC ZONES OF THE PULP
- The Pulp-Dentin Complex
- Odontoblast Layer
- Cell-Poor Zone
- Cell-Rich Zone
- Pulp Proper

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- Odontoblast Process
- Relationship of Odontoblast Structure to Secretory Function
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- Regulation of Pulpal Blood Flow
- Circulation in the Inflamed Pulp

PULPAL REPAIR

PULPAL CALCIFICATIONS

AGE CHANGES

From many perspectives, the pulp is a unique tissue. It is a soft tissue of mesenchymal origin with specialized cells, the odontoblasts, arranged peripherally in direct contact with dentin matrix. The close relationship between odontoblasts and dentin, sometimes referred to as the *dentin-pulp complex*,[115,278,342] is one of several reasons dentin and pulp should be considered as a functional entity made up of histologically distinct elements. In particular, several unique properties are imposed on the pulp by the rigid mineralized dentin in which it is enclosed. It is situated within a low-compliance environment that limits its ability to expand during episodes of vasodilation and increased filtration. Because the pulp is relatively incompressible, the total volume within the pulp chamber cannot be greatly increased (although reciprocal volume changes can occur among arterioles, venules, lymphatics, and extravascular tissue). *As a consequence, inflammatory reactions result in an increase in tissue pressure due to a limited increase in fluid volume.*[141,143] In the pulp, therefore, careful regulation of blood flow is of critical importance.

*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.

The dental pulp is in many ways similar to other connective tissues of the body, but its special characteristics deserve serious consideration. Even the mature pulp bears a resemblance to embryonic connective tissue and is therefore a relatively rich source of stem cells (see also Chapter 16). The pulp houses a number of tissue elements, including axons, vascular tissue, connective tissue fibers, ground substance, interstitial fluid, odontoblasts, fibroblasts, immunocompetent cells, and other cellular components. These components respond dynamically to developmental, physiologic (e.g., orthodontic or masticatory forces) or pathologic stimuli. The overall dynamic response pattern plays a key role in whether pulp tissue adapts or undergoes tissue necrosis to these stimuli (see also Chapter 13).

The pulp is supported by a microcirculatory system, and its largest vascular components are arterioles and venules. Unlike most tissues, the pulp lacks a true collateral system and is dependent on relatively few arterioles entering through the apical foramina. The vascular system of the pulp decreases progressively with age.[24]

The tooth pulp is a unique sensory organ. Because it is encased in a protective layer of dentin, which in turn is covered with enamel, it might be expected to be quite unresponsive to stimulation. However, despite the low thermal conductivity of dentin, the pulp is undeniably sensitive to thermal stimuli, such as ice cream and hot drinks. The unusual mechanism that allows the dentin-pulp complex[115,278,342] to function as an exquisitely responsive sensory system is discussed later in the chapter.

After tooth development, the pulp retains its ability to form dentin throughout life. This enables the vital pulp to partially compensate for the loss of enamel or dentin caused by mechanical trauma or disease. How well it serves this function depends on many factors, but the potential for regeneration and repair is as much a reality in the pulp as in other connective tissues of the body.

This chapter brings together what is known about the development, structure, and function of the dentin-pulp complex in the hope that this knowledge will provide a firm biologic basis for clinical decision making.

DEVELOPMENT

Embryologic studies have shown that the pulp is derived from the cephalic neural crest. Neural crest cells arise from the ectoderm along the lateral margins of the neural plate and migrate extensively. Those that travel down the sides of the head into the maxilla and mandible contribute to the formation of the tooth germs. The dental papilla, from which the mature pulp arises, starts to develop as ectomesenchymal cells proliferate and condense adjacent to the dental lamina epithelium (Fig. 12-1). It is important to remember this migratory potential of ectomesenchymal cells later in the chapter when reviewing the ability of pulp cells to move into areas of injury and replace destroyed odontoblasts.

During the sixth week of embryonic life, tooth formation begins as a localized thickening of ectoderm associated with the maxillary and mandibular processes. This activity results in the formation of two horseshoe-shaped structures, one on each process. These structures are termed the *primary dental laminae*. Each primary dental lamina splits into a vestibular and a dental lamina (Fig. 12-2).

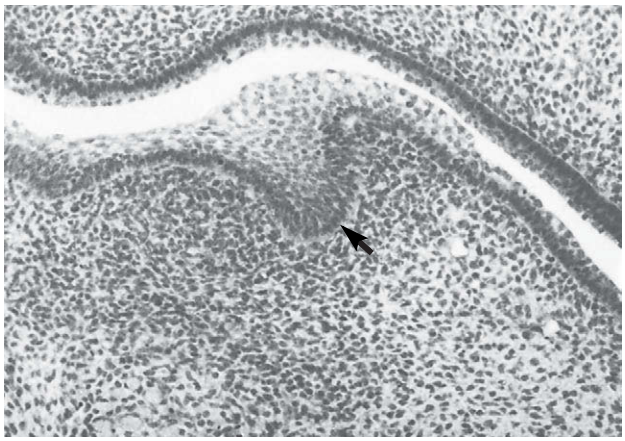


FIG. 12-1 Dental lamina *(arrow)* arising from the oral ectoderm.

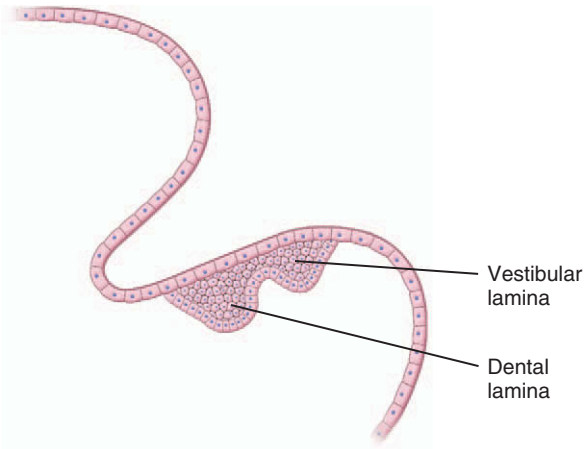


FIG. 12-2 Diagram showing formation of the vestibular and dental lamina from oral ectoderm.

Numerous studies have indicated that the embryonic development of any tissue is promoted by interaction with an adjacent tissue. The complex epithelial-mesenchymal interactions during tooth development have been studied extensively. Cell-to-cell and cell-to-extracellular matrix (ECM) interactions direct the differentiation of ameloblasts and odontoblasts by causing these cells to change gene expression.

Growth factors, often termed *signaling molecules,* are polypeptides that promote proliferation, migration, and differentiation of a variety of cells. It can be assumed that growth factors are involved in signaling during those epithelial-mesenchymal interactions that regulate tooth initiation, morphogenesis, and cell differentiation.[294,298-300,319-321]

From the onset of tooth formation, a dental basement membrane (DBM) exists between the inner dental epithelium and the dental mesenchyme. The DBM consists of a thin basal lamina, which is formed by the epithelial cells, and a layer of ECM derived from the dental mesenchyme. The basal lamina is made up of an elastic network composed of type IV collagen, which has binding sites for other basement membrane constituents such as laminin, fibronectin, and heparan sulfate proteoglycans. Laminin binds to type IV collagen and also to receptors on the surface of preameloblasts and ameloblasts.

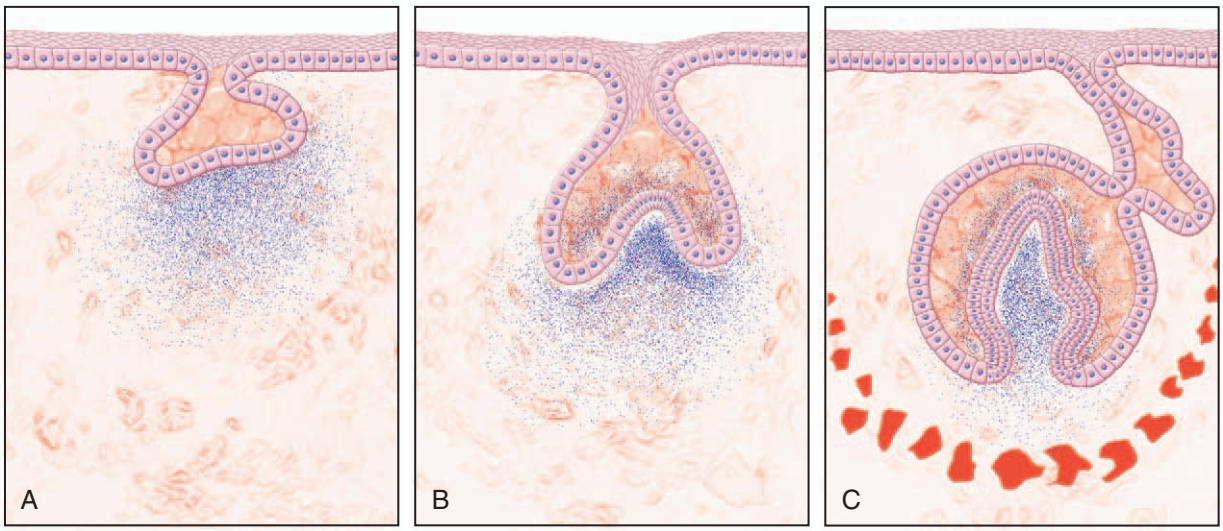


FIG. 12-3 Diagrammatic representation of **(A),** the bud, **(B),** the cap, and **(C)** the bell stages of tooth development.

The DBM also contains mesenchyme-derived type I and type III collagen, hyaluronate, heparan sulfate, and chondroitin 4- and 6-sulfates. Certain proteoglycans expressed on the cell surface of the odontoblast function as receptors for these matrix molecules. This permits signals from components of the matrix to influence the migration and differentiation of odontoblasts. The composition of the DBM changes during tooth development, and these alterations appear to modulate the successive steps in odontogenesis. With the differentiation of odontoblasts, type III collagen disappears from the predentin matrix, and fibronectin, which surrounds preodontoblasts, is restricted to the apical pole of mature odontoblasts.[196]

The initial stage of development of the dental papilla is characterized by proliferative activity beneath the dental lamina at sites corresponding to the positions of the prospective primary teeth. Even before the dental lamina begins to form the enamel organ, a capillary vascular network develops within the ectomesenchyme, presumably to support the increased metabolic activity of the developing tooth buds.

Stages of Development

Although formation of the tooth is a continuous process, as a matter of convenience tooth morphogenesis has been divided into three stages: (1) bud, (2) cap, and (3) bell (Fig. 12-3).[186] In the *bud stage* the epithelial cells of the dental lamina proliferate and produce a budlike projection into the adjacent ectomesenchyme. The *cap stage* is reached when the cells of the dental lamina have proliferated to form a concavity with a caplike appearance. The outer cells of the cap are cuboidal and constitute the outer enamel epithelium. The cells on the inner or concave aspect of the cap are somewhat elongated and represent the inner enamel epithelium. Between the outer and inner epithelia is a network of cells termed the *stellate reticulum* because of the branched reticular arrangement of the cellular elements. The rim of the enamel organ (i.e., where the outer and inner enamel epithelia are joined) is termed the *cervical loop*. As the cells forming the loop continue to proliferate, there is further invagination of the enamel organ into the mesenchyme. During this time, the organ assumes a bell shape, and tooth development then enters the *bell stage* (Fig. 12-4). During the bell stage, the ectomesenchyme of the dental papilla becomes partially enclosed by the invaginating epithelium.

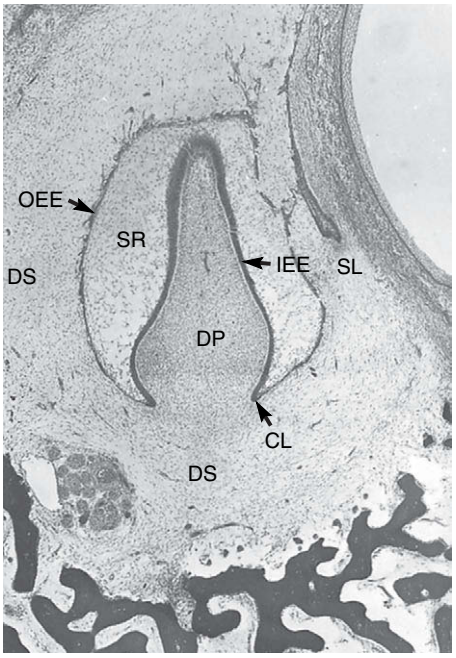


FIG. 12-4 Bell stage of tooth development showing the outer enamel epithelium (OEE), stellate reticulum (SR), inner enamel epithelium (IEE), dental papilla (DP), cervical loop (CL), successional lamina (SL), and dental sac (DS).

The condensed ectomesenchyme surrounding the enamel organ and dental papilla complex forms the dental sac and ultimately develops into the periodontal ligament (see Fig. 12-4). During the bell stage, the primary dental lamina degen-

erates and is invaded and replaced by mesenchymal tissue. In this way the epithelial connection between the enamel organ and the oral epithelium is severed. The free end of the dental lamina associated with each of the primary teeth continues to grow and forms the successional (secondary) dental lamina. It is from this structure that the tooth germ of the succedaneous tooth arises. As the maxillary and mandibular processes increase in size, the permanent first molar arises from posterior extensions of the dental lamina. After birth, the second and third molar primordia appear as the dental laminae proliferate into the underlying mesenchyme.

The morphogenesis (shape) of the crown is controlled by specialized signaling centers called *epithelial enamel knots* (EK). The enamel knots are transient clusters of nonproliferative epithelial cells located in the dental epithelium.[159,215,353] They express members of molecules belonging to key conserved fibroblast growth factor (Fgf), bone morphogenetic protein (Bmp), Hedgehog (Hh), and Wnt families that are well known to be essential for embryo and organ formation.[332] The signaling activity of the enamel knot and expressed molecules such as Fgf4 and Bmp4 regulate cellular processes of the surrounding epithelial and mesenchymal cells, leading to altered proliferation and expression of genes essential for tooth formation, such as transcription factors Mxs1 and Pax9.[172,354] The first appearing primary enamel knot (PEK) is present at the tip of the epithelial bud at the bud stage.[159] At the cap stage, the PEK is located in the middle part of the anterior-posterior axis of the enamel organ.[159,353] The secondary enamel knots (SEK), which appear later at the site of cusp tips, regulate the formation and number of the cusps of the multicusped teeth.[159] The recently found tertiary enamel knot (TEK) in turn prevents enamel formation by the underlying ameloblasts and thus controls the formation of the enamel-free areas in animals having these areas, such as mice.[215]

Development of the Tooth Nerve Supply

Peripheral innervation serves significant roles in pulp biology. The initial innervation of the tooth originates from the sensory trigeminal ganglia. The nerve fibers innervate specific sites in the adult tooth, and as a consequence, the development of dental innervation is not a random event but instead is a strictly spatiotemporally controlled process that is tightly linked to the advancing tooth morphogenesis and dental cell differentiation[173,209] (Fig. 12-5). In the well-analyzed mouse mandibular molar, the molar nerve[209] from the inferior alveolar nerve branch reaches the tooth at the early bud stage. The nerve follows a narrow mesenchymal pathway under the tooth germ, where it divides into the buccal and lingual branches.[173,209,243] During the cap and bell stages, nerve fibers establish a nerve plexus beneath the primary apical foramina. Nerve fibers wait here before they are allowed to enter the dental papilla. This takes place only after the crown shape is being defined by both dentin and enamel appearance in the crown.[237,238] The nerve fibers enter the papilla at both the mesial and distal ends, which are the sites of the future mesial and distal root of the molar tooth germ. The nerve fibers innervate the subodontoblastic region, the coronal odontoblast layer, and predentin layer with some fibers terminating in the dentin. The nerve fibers of the nerve plexus also contribute to the innervation of the periodontium.[146] Pioneer sympathetic innervation of the dental pulp follows the sensory one.[237]

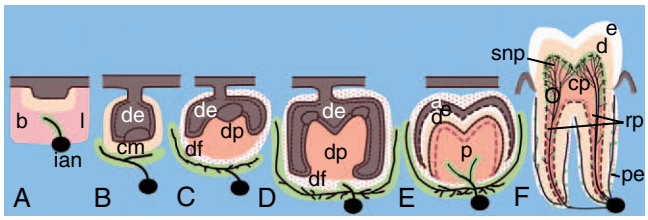


FIG. 12-5 Growth of the sensory trigeminal axons to the dental target areas during tooth formation. The schematic illustration is based on studies on the localization of nerve fibers in the developing mouse molar **(A-F).**[173,237,238] Pioneer dental axons follow a restricted mesenchymal pathway (*marked green in **B***) and grow around the developing tooth germ, termed the *mesenchymal dental follicle target field* (*green in **C-F***). Nerve fibers penetrate into the dental pulp (p) right after the onset of enamel formation **(E)** and innervate the major pulp-dentin border target area (*marked green in **F***). *a,* ameloblasts; *b,* buccal; *cm,* condensed dental mesenchyme; *cp,* coronal pulp; *d,* dentin; *de,* dental epithelium; *df,* dental follicle; *dm,* dental mesenchyme; *dp,* dental papilla; *e,* enamel; *ian,* inferior alveolar nerve; *l,* lingual; *o,* odontoblaster; *p,* dental pulp; *pe,* periodontium; *rp,* radicular pulp; *snp,* subodontoblastic nerve plexus; nerve fibers are marked in black. (*Modified from Kenntunen P, et al: Regularing av utvikling av tannens form og sensorisk nerveforsying er samordent. Nor Tannlaegeforen Tid 119:162–166, 2009.*)

Molecular Control of Trigeminal Axon Navigation

There is increasing evidence that the navigation of the growing dental axons involves concerted, redundant signaling activity by molecules of different families. The molecules exerting positive attractive effect on axon guidance or survival are expressed in the mesenchymal pathway or target areas around and within the tooth germ.[214] The molecules include neurotrophic factors, which promote survival, differentiation, and maintenance of neurons in developing and adult vertebrate nervous systems. NGF (nerve growth factor), which is essential for dental pulp nerve supply, and GDNF (glial cell line-derived neurotrophic factor) are expressed in the major target area of the developing tooth, namely the dental follicle and pulp-dentin border target areas. In addition, many other molecular families, such as laminins, neuregulin/ErbB, Ephs and Ncam are implicated in the development of the tooth nerve supply.[100,214]

Repulsive axon guidance molecules in turn, are produced in the areas next to the axon pathways. They control axon navigation by preventing nerve fibers from entering areas that should be avoided. The secreted chemorepellant, semaphorin3A (Sema3A), which is expressed in mesenchymal restriction areas,[173] controls dental trigeminal axon fasciculation as well as the timing and patterning of tooth innervation.[173] In addition, Sema3A appears to control the site of the nerve fiber penetration to the dental pulp (i.e., through the future roots).[216] Because Sema3A expression in mesenchymal restriction areas is controlled by signaling from the dental epithelium, this indicates that tooth innervation development, like the formation of the tooth germ proper, is controlled by local tissue interactions. Of note, Fgfr2b, which is essential to tooth morphogenesis by regulating epithelial proliferation and function of the enamel knot, also controls Sema3A expression and

therefore dental axon navigation. Thus, Fgf2b acts as a molecule that integrates tooth morphogenesis and dental trigeminal axon patterning.[197]

Differentiation of Odontoblasts

Differentiation of epithelial and mesenchymal cells into ameloblasts and odontoblasts, respectively, occurs during the bell stage of tooth development. This differentiation starts in the region where the cusp tip will develop and proceeds towards the cervical loop. The preameloblasts lining the dental papilla induce differentiation of odontoblasts. Consequently, odontoblast-secreted dentin matrix induces enamel secretion by ameloblasts.

During the bell stage of development, there is still mitotic activity among the relatively immature cells of the inner enamel epithelium in the region of the cervical loop. As they commence to mature into ameloblasts, mitotic activity ceases, and the cells elongate and display the characteristics of active protein synthesis (i.e., an abundance of rough endoplasmic reticulum [RER], a well-developed Golgi complex, and numerous mitochondria).

As the ameloblasts undergo differentiation, changes are taking place across the basement membrane in the adjacent dental papilla because of a coordinated exchange of growth factors and other soluble mediators. Before differentiation of odontoblasts, the dental papilla consists of sparsely distributed polymorphic mesenchymal stem cells with wide intercellular spaces (Fig. 12-6). With the onset of differentiation, a single layer of cells, the presumptive odontoblasts (i.e., preodontoblasts) align themselves along the basement membrane separating the inner enamel epithelium from the dental papilla. These cells stop dividing and elongate into short columnar cells with basally situated nuclei (Fig. 12-7). Several cytoplasmic projections from each of these cells extend toward the basal lamina. At this stage, the preodontoblasts are still relatively undifferentiated.

As the odontoblasts continue to differentiate, they become progressively more elongated and take on the ultrastructural characteristics of protein-secreting cells. Cytoplasmic processes from these cells extend through the DBM toward the basal lamina, and more and more collagen fibrils appear within the ECM. The first formed collagen fibers pass between the preodontoblasts and extend toward the basal lamina to form large, fan-shaped bundles 100 to 200 nm in diameter, often referred to as *von Korff fibers*. These fibers stain with silver stains and are associated with a high content of proteoglycans. Some smaller collagen fibers, approximately 50 nm in diameter, also pass between the odontoblasts and are thought to arise in the dental papilla subjacent to the odontoblasts.[29]

A study on collagen gene expression during rat molar tooth development found both types I and III collagen messenger

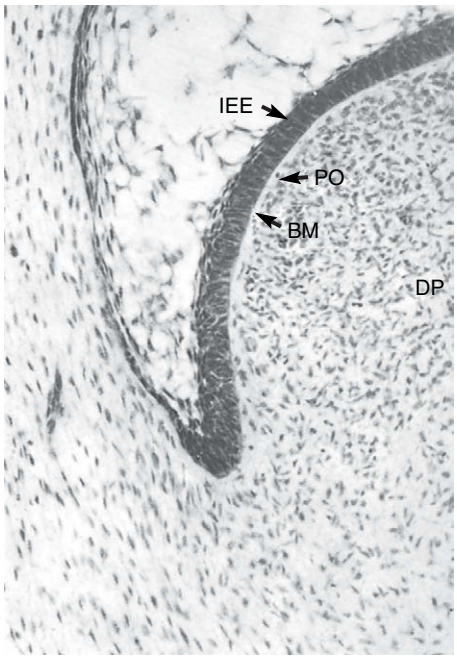


FIG. 12-6 Bell stage shows preodontoblasts (PO) aligned along the basement membrane (BM) separating the inner enamel epithelium (IEE) from the dental papilla (DP).

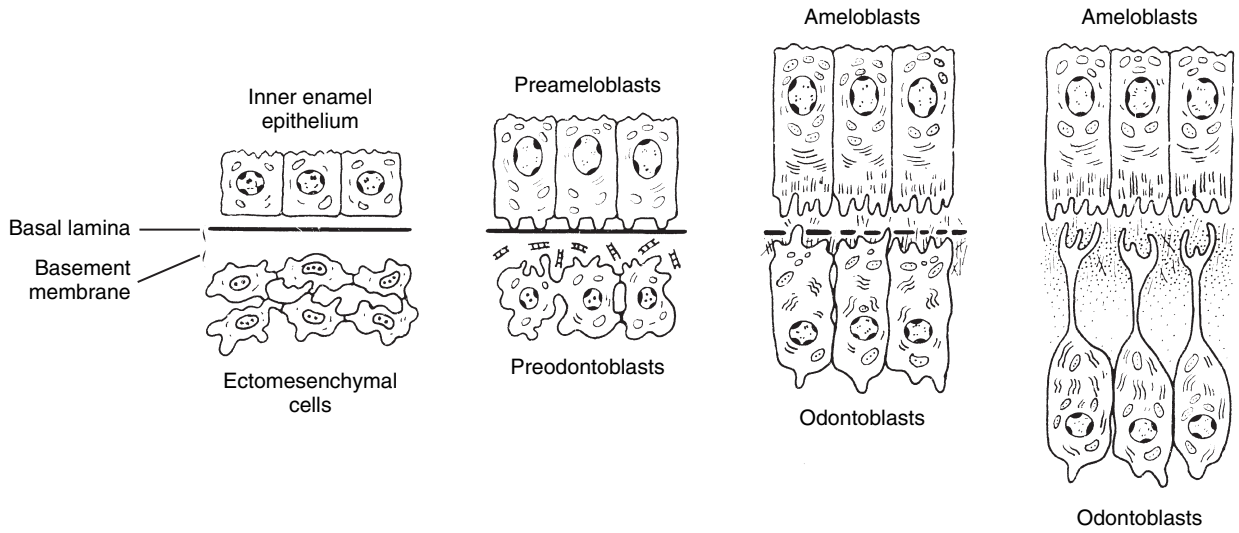


FIG. 12-7 Diagrammatic representation of the stages of odontoblast differentiation.

ribonucleic acid (mRNA) in developing odontoblasts.[75] Levels of type I collagen mRNA increased with the progression of odontoblast differentiation, whereas type III collagen gene expression decreased as dentinogenesis proceeded. Both type I and type III collagen mRNA were detected in dental pulp mesenchymal cells.

Dentinogenesis first occurs in the developing tooth at sites where the cusp tips or incisal edge will be formed. It is in this region that odontoblasts first reach full maturity and become tall columnar cells, at times attaining a height of about 50 µm (see Fig. 12-7). The width of these cells remains fairly constant at approximately 7 µm. Production of the initial dentin matrix involves the formation, organization, and maturation of collagen fibrils and proteoglycans. As predentin is formed, the odontoblasts commence to move toward the central pulp, depositing predentin at a rate of approximately 4 to 8 µm per day in their wake during early tooth development. Within predentin, a process from each odontoblast becomes accentuated and remains to form the odontoblast process. The dentinal tubules are formed around these cellular processes. Similar to tooth initiation and morphogenesis, dentinogenesis is controlled by epithelial-mesenchymal interactions that are mediated by a combination of EMC and signaling molecules of different families such as BMP and Tgf-β1.[197]

Root Development

Root development commences after the completion of enamel formation, when the cervical loop does not exist. The stellate reticulum and stratum intermedium cells disappear, and the inner and outer enamel epithelium in the distal part of the cervical loop forms Hertwig's epithelial root sheath (HERS), composed of two cell layers, the inner and outer enamel epithelium (Fig. 12-8). HERS determines the size and shape of the root or roots of the tooth. As in the formation of the crown, the cells of the inner enamel epithelium induce the adjacent mesenchymal stem cells in the pulp to differentiate into preodontoblasts and odontoblasts. As soon as the layer of dentin matrix mineralizes, gaps appear within the HERS-derived Malassez epithelium, allowing mesenchymal cells from the dental sac to move into contact with the newly formed dentin. These mesenchymal stem cells thereafter differentiate into cementoblasts and deposit cementum matrix on the root dentin. Some cells of the Malassez epithelium persist within the periodontal ligament and are known as the *epithelial cell rests of Malassez*. These clusters of epithelial cells surrounded by basement membrane appear as a meshlike network around the root. Although the number of these rests gradually decreases with age, it has been shown that at least some of them retain the ability to undergo cell division[347] (Fig. 12-9). In later life, if a chronic inflammatory lesion develops within the periapical tissues as a result of pulp disease, proliferation of the epithelial rests may produce a periapical (radicular) cyst.

Accessory Canals in the Root

Occasionally during formation of the root sheath, a break develops in the continuity of the sheath, producing a small gap. When this occurs, dentinogenesis does not take place adjacent to the defect. The result is a small "accessory" canal between the dental sac and the pulp. An accessory canal can become established anywhere along the root, creating a periodontal-endodontic pathway of communication and a possible portal of entry into the pulp if the periodontal tissues

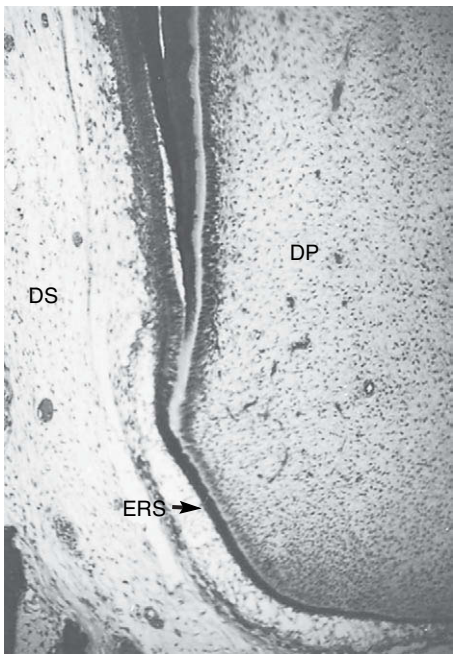


FIG. 12-8 Root development showing dental pulp (DP), dental sac (DS), and epithelial root sheath (ERS).

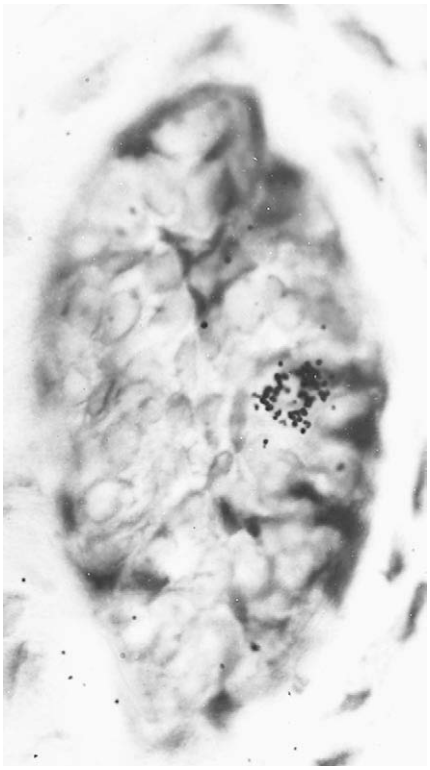


FIG. 12-9 Autoradiograph of an epithelial rest cell showing ^{3}H-thymidine labeling of the nucleus, indicating that the cell is preparing to divide. (*From Trowbridge HO, Shibata F: Mitotic activity in epithelial rests of Malassez.* Periodontics *5:109, 1967.*)

lose their integrity. In periodontal disease, the development of a periodontal pocket may expose an accessory canal and allow microorganisms or their metabolic products to gain access to the pulp. Conversely, in the case of teeth with infected, necrotic pulps, the inability to cleanse, shape, and obturate these canals could result in periodontitis (see also Chapter 18).

DENTIN

Fully mature dentin is composed of approximately 70% inorganic material, 20% organic material, and 10% water on a weight basis. The main inorganic component of dentin is calcium hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$.[204] Organic matrix consists of proteins, the most common of which is type I collagen; there is a minor component of type V collagen.[43] The most common noncollagenous proteins include dentin phosphoprotein (DPP), dentin matrix protein 1 (DMP1), dentin sialoprotein (DSP), osteopontin (OPN), osteocalcin, and bone sialoprotein (BSP).[43] In addition, dentin contains proteoglycans, small amounts of phospholipids, and numerous growth factors, including bone morphogenetic proteins (BMP), insulin-like growth factors (IGFs), and transforming growth factor beta (TGF-β). These growth factors have significance during demineralization of dentin since they may locally stimulate subsequent stem cell differentiation (see later and Chapter 16). The elasticity of dentin provides flexibility for the overlying brittle enamel and allows the impact of mastication to occur without fracturing enamel.[183,184,369] From an engineering perspective, the collagen can be thought of as steel rebar, and the mineralized crystals act as cement; the resulting composite structure has great tensile strength, particularly given its small size. Additionally, the enamel-dentin junction (EDJ) between dentin and enamel is an uneven scalloped border that increases contact and adherence between dentin and enamel, keeping the two hard tissues together during mastication.

Primary, Secondary, and Tertiary Dentin

Primary dentin is produced during tooth development until the teeth have erupted into the oral cavity. It is classified as *orthodentin,* the tubular form of dentin lacking of cells found in the teeth of all dentate mammals.[204] The primary dentin is secreted at a relatively high rate and constitutes the major part of the dentin in the tooth. It is regular in structure and contains dentin tubules that form an S-shaped primary curvature as a result of the directional movement of odontoblasts, which have a cell process that extends from a broad peripheral border towards a narrow, centrally located cell layer. After tooth eruption, the odontoblasts continue to lay down dentin but slightly change their direction, which contributes to bending of the dentinal tubules. It is referred to as *secondary dentin* and is synthesized at a much lower rate and is less regular in structure than primary dentin. Secondary dentin is deposited during the rest of the tooth's life. Unlike primary and secondary dentin, *tertiary dentin,* or reparative or reactionary dentin, is deposited as a result of a pathologic process such caries or occlusal abrasion. Tertiary dentin has been suggested to be secreted by original odontoblasts or in case of their death, by newly differentiated replacement odontoblasts originating from nearby mesenchymal stem cells. The function of the tertiary dentin is to protect the pulp from noxious influences. Tertiary dentin is disorganized in structure compared to primary and secondary dentin.

Mantle Dentin

The first layer of the primary dentin to be deposited is mantle dentin. It is produced by odontoblasts that are not yet fully differentiated. In the adult tooth, mantle dentin is the oldest dentin and is produced adjacent to the enamel in the crown. Odontoblasts first produce the collagen components, which are organized in a collagen fibril network. Electron microscopic analyses have shown that odontoblasts produce small membrane-closed matrix vesicles where precipitation of minerals starts.[33] After the rupture of the matrix vesicles, the crystals spread within the collagen matrix and are assumed to act as starting points for the growth of the apatite crystals. In addition to the crown, mantle dentin is also found in the root between cementum and Tomes granular layer. However, this is a controversial finding, and opposing data suggest that a hyaline layer or enameloid is found in the root instead of mantle dentin.

Later during the mineralization of the circumpulpal dentin, the matrix vesicles are no longer detected.[205] Mantle dentin can be recognized by the characteristic thick, fan-shaped collagen fibers deposited immediately subjacent to the basal lamina in histologic sections. These fibers run roughly perpendicular to the DEJ. Spaces between the fibers are occupied by smaller collagen fibrils lying more or less parallel with the DEJ or CDJ. Mantle dentin is only 150 µm thick and is slightly less mineralized (about 4% less mineral) than underlying dentin[139] and is therefore softer.[369] The mineralization of the mantle dentin may differ from mineralization of the rest of the dentin additionally, since dentinphosphoprotein (DPP), which regulates crystal growth in the circumpulpal dentin, does not appear to be present in mantle dentin.[246]

Circumpulpal Dentin

Circumpulpal dentin is formed after the layer of mantle dentin has been deposited, and it constitutes the major part of primary and secondary dentin. The odontoblasts first deposit the organic matrix, which is composed mainly of collagen fibrils approximately 500 nm in diameter, oriented at right angles to the long axis of the dentinal tubules. These collagen fibrils are closely packed together and form an interwoven network that will be embedded by the subsequent mineral crystals. Calcium is actively transported from the blood vessels by the odontoblasts into the collagen fibril template/network. There is evidence that mineralization of the dentin is under control of dentinphosphoprotein (DPP). DPP is able to bind calcium, since it is highly anionic. Conformational changes of DPP enable it to bind large numbers of calcium ions, which allows the formation and growth of mineral crystals in the collagen matrix network. By regulating the release and concentration of DPP, the odontoblasts may regulate the initiation and speed of dentin mineralization. Hydroxyapatite crystals are deposited on the surface and within the fibrils and continue to grow as mineralization proceeds, resulting in an increased mineral content of the dentin. The circumpulpal dentin is mineralized through calcospherites in the mineralization front between predentin and mineralizing dentin. The mineral crystals are organized in a radiating pattern in calcospherites, the size of which may vary in size and shape in different regions of dentin.

As the calcospherites enlarge, they fuse with adjacent calcospherites until the dentin matrix is completely mineralized.

Predentin

Predentin is a 15- to 20-μm unmineralized organic matrix layer of dentin situated between the odontoblast layer and the mineralized dentin.[204] Its prime protein constituents include type I and type II collagens.[42] Noncollagenous elements consist of several proteoglycans (i.e., dermatan sulfate, heparan sulfate, hyaluronate, keratin sulfate, chondroitin 4-sulfate, chondroitin 6-sulfate),[40,117] glycoproteins, glycosaminoglycans (GAGs),[83] Gla proteins, and dentin phosphoprotein (DPP)[42] DPP is a highly phosphorylated protein that is exclusively found in bone and dentin.[290] In dentin, DPP is produced by the odontoblasts and transported to the mineralization front. It is thought to bind to calcium and play a role in mineralization.[109,359] Growth factors such as TGF-β, insulin-like growth factor, platelet-derived growth factor, and angiogenic growth factor have also been identified in dentin.[294] The presence of growth factors in dentin may have profound clinical significance because resorption of dentin conceivably releases these growth factors into pulp, where they alter biologic functions of this tissue. Dentinal resorption may stimulate formation of tertiary dentin.

TABLE 12-1

Mean Number and Diameter per Square Millimeter of Dentinal Tubules at Various Distances from the Pulp in Human Teeth

Distance from Pulp (mm)	Number of Tubules (1000/mm²)		Tubule Diameter (μm)	
	Mean	Range	Mean	Range
Pulpal wall	45	30–52	2.5	2.0–3.2
0.1–0.5	43	22–59	1.9	1.0–2.3
0.6–1.0	38	16–47	1.6	1.0–1.6
1.1–1.5	35	21–47	1.2	0.9–1.5
1.6–2.0	30	12–47	1.1	0.8–1.6
2.1–2.5	23	11–36	0.9	0.6–1.3
2.6–3.0	20	7–40	0.8	0.5–1.4
3.1–3.5	19	10–25	0.8	0.5–1.2

(From Garberoglio R, Brännström M: Scanning electron microscopic investigation of human dentinal tubules. *Arch Oral Biol* 21:355, 1976.)

Dentinal Tubules

A characteristic of human dentin is the presence of tubules that occupy from 1% (superficial dentin) to 30% (deep dentin) of the volume of intact dentin.[107,277] The diameter of tubules vary from 1 μ to 2.5 μm and traverse the entire thickness of dentin from the DEJ or CDJ to the pulp. They are slightly tapered, with the wider portion situated toward the pulp. This tapering is the result of the progressive formation of up to 1 μm of peritubular dentin, which leads to a continuous decrease in the diameter of the lumen of the tubules to a final diameter of about 1 mm.[107] No peritubular dentin is present when the tubules reach the predentin and pulp chamber.

In coronal dentin, the tubules have a gentle S shape as they extend from the DEJ to the pulp. The S-shaped curvature is presumably a result of the crowding of odontoblasts as they migrate toward the center of the pulp. As they approach the pulp, the tubules converge because the surface of the pulp chamber has a much smaller area than the surface of dentin along the DEJ. This results in a progressive increase in dentin permeability (discussed later).[275-277,279]

The number and diameter of the tubules has been determined and varies depending on the location in the tooth (Table 12-1).[107] The number and diameter of dentinal tubules are similar among rats, cats, dogs, monkeys, and humans, demonstrating a remarkable consistency in mammalian orthodentin.[4]

Lateral microtubules containing branches of the main odontoblastic processes have been demonstrated by other researchers,[167,236] who suggested that they form pathways for the movement of materials between the main processes and the more distant matrix. It is also possible that the direction of the branches influences the orientation of the collagen fibrils in the intertubular dentin.

Near the DEJ, the dentinal tubules ramify into one or more terminal branches[236] (Fig. 12-10). This occurs because during the initial stage of dentinogenesis, the differentiating odontoblasts extended several small cytoplasmic processes toward the DEJ, but as the odontoblasts migrated, their processes converged into one major process (see Fig. 12-7).

Intertubular Dentin

Intertubular dentin is located between the dentin tubules and constitutes the bulk of dentin (Fig. 12-11). Its organic matrix consists mainly of collagen fibrils having diameters of 50 to 100 nm. These fibrils are oriented approximately at right angles to the dentinal tubules. They are well mineralized and provide tensile strength to dentin.[184] Intertubular dentin is found between the DEJ and the mineralization front.[204]

Intratubular Dentin

Dentin lining the inner walls of tubules is termed *intratubular* or *peritubular dentin* (see Fig. 12-11). Intratubular dentin represents a specialized form of orthodentin not found in all mammals. The matrix of peritubular dentin differs from that of intertubular dentin by having relatively fewer collagen fibrils and a higher proportion of sulfated proteoglycans and mineral. Because of its lower content of collagen,[183,374] peritubular dentin is harder than intertubular dentin and therefore is more quickly dissolved in acid than intertubular dentin. By preferentially removing peritubular dentin, acid-etching agents used during dental restorative procedures and ethylenediaminetetraacetic acid (EDTA) used in endodontic treatment enlarge the openings of the dentinal tubules, making the dentin more permeable. The absence of intrafibrillar mineral in patients with dentinogenesis imperfecta type II may be responsible for their softer dentin.[185]

Dentinal Sclerosis

Partial or complete obturation of dentinal tubules may occur as a result of aging or develop in response to persistent stimuli such as attrition of the tooth surface or dental caries. When

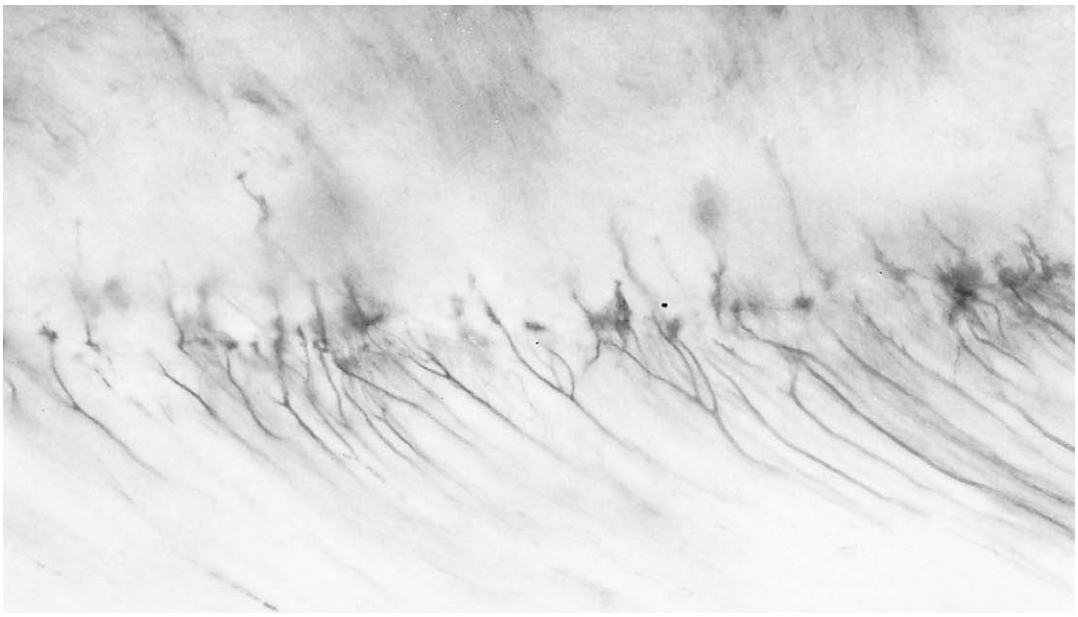


FIG. 12-10 Ground section of a tooth demonstrating branching of the dentinal tubules near the dentin and enamel junction (DEJ). This branching may account for the increased clinical sensitivity at the DEJ.

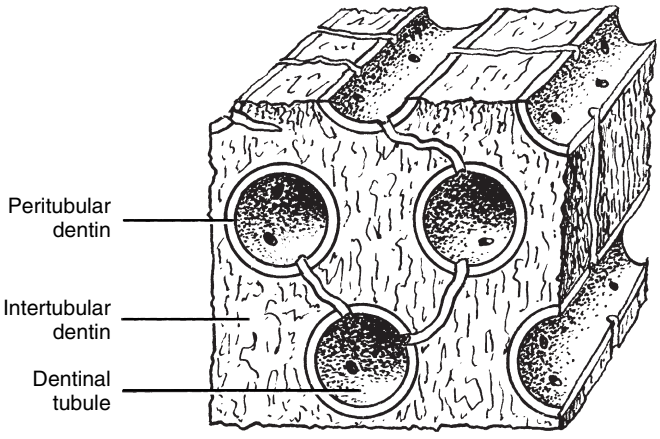


FIG. 12-11 Diagram illustrating peritubular and intertubular dentin. *(From Trowbridge HO: Dentin today: research reveals new information about the complexity of dentin.* Dentistry *2:22–29, 1982.)*

tubules become filled with mineral deposits, the dentin is termed *sclerotic*. Dentinal sclerosis is easily recognized in histologic ground sections because of its translucency (due to homogeneity of the dentin). Studies using dyes, solvents, and radioactive ions have shown that sclerosis results in decreased permeability of dentin,[92,276,328] so by limiting the diffusion of noxious substances through the dentin, dentinal sclerosis helps shield the pulp from irritation.

One form of dentinal sclerosis is thought to represent an acceleration of intratubular dentin formation. This form appears to be a physiologic process, and in the apical third of the root it develops as a function of age.[323,328] Dentinal tubules can also become blocked by the precipitation of hydroxyapatite and whitlockite crystals within the tubules. This type occurs in the translucent zone of carious dentin and in attrited dentin and has been termed *pathologic sclerosis*.[263,328,371]

Interglobular Dentin

Interglobular dentin refers to dentin that contains organic matrix that is unmineralized because the growing calcospherites failed to coalesce. This occurs most often in the circumpulpal dentin close to the mantle dentin in the crown and close to the Tomes granular layer in the root. In certain dental anomalies (e.g., vitamin D–resistant rickets, hypophosphatasia), large areas of interglobular dentin are a characteristic feature (Fig. 12-12).[108]

Dentinal Fluid

Free fluid occupies 1% of superficial dentin but about 22% of the total volume of deep dentin (i.e., near the pulp).[277] This fluid is an ultrafiltrate of blood from the pulp capillaries, and its composition not surprisingly resembles plasma in many respects.[61] Using calcium ion–specific electrodes, the ionized calcium concentration of dentinal fluid in predentin was two to three times higher than plasma.[212] Although it contains plasma proteins, their concentration is only about 10% that of plasma.[219] The outward fluid flows between the odontoblasts through the dentinal tubules and is blocked peripherally by enamel on the crown and cementum on the root. It has been shown that the tissue pressure of the pulp is approximately 14 cm H_2O (10.3 mm Hg).[67] Consequently, a pressure gradient exists between the pulp and the oral cavity that accounts for the slow outward flow of fluid if any dentin becomes exposed. Exposure of the tubules by tooth fracture or during cavity preparation often results in the outward movement of fluid to the exposed dentin surface in the form of tiny droplets.[157] Dehydrating the surface of the dentin with compressed air, dry heat, or the application of absorbent paper can accelerate this outward movement of fluid.[37-39,274] Rapid flow of fluid through the tubules is thought to be a cause of dentin sensitivity.[230] The slow outward movement of dentinal fluid is not sufficient to activate the mechanoreceptor nerves responsible for dentin



FIG. 12-12 Section showing interglobular dentin (ID) in a deciduous incisor from a 3-year-old boy with childhood hypophosphatasia.

sensitivity. This slow outward fluid flow, about 0.02 ml/sec/mm^2, must increase to 1 to 1.5 ml/sec/mm^2 before these nerves begin to discharge.[230]

Bacterial products or other contaminants may be introduced into the dentinal fluid as a result of dental caries, restorative procedures, or growth of bacterial biofilms beneath restorations.[37,234,284] Dentinal fluid may thus serve as a sink from which injurious agents can diffuse into the pulp, producing an inflammatory response. Conversely, dentinal fluid may serve as a vehicle for the egress of bacteria from a necrotic pulp into the periradicular tissue. This has particular significance in the apical portion of the root canal system in the development or maintenance of apical periodontitis because bacteria are the key etiologic factor (see Chapter 15). Fortunately, the density of dentinal tubules is much lower in the apical portion of the root compared to the cervical portion, and this anatomic feature may contribute to the success of nonsurgical root canal treatment. In addition, endodontic surgical techniques have been modified to reduce exposure of dentinal tubules during apical root resection by reducing the angle of root resection; as the angle of resection approaches 90 degrees to the long axis of the root, a corresponding decrease is seen in dentinal tubules exposed to the periapical tissue (see Chapter 21 for details).

Dentin Permeability

The permeability of dentin has been well characterized.[92,97,274-277,279,282] Dentinal tubules are the major channels for diffusion of material across dentin. Because fluid permeation is proportional to tubule diameter and number, dentin permeability increases as the tubules converge on the pulp (Fig. 12-13). The total tubular surface near the DEJ is approximately 1% of the total surface area of dentin,[274,277] whereas close to the pulp chamber, the total tubular surface may be nearly 45%. From a clinical standpoint, it should be recognized that dentin beneath a deep cavity preparation is much more permeable than dentin underlying a shallow cavity when the formation of sclerotic or reparative dentin is negligible.

One study[97] found that the permeability of radicular dentin is much lower than that of coronal dentin. This was attributed to a decrease in the density of the dentinal tubules from approximately 42,000/mm^2 in cervical dentin to about 8000/mm^2 in radicular dentin. These investigators found that fluid movement through outer radicular dentin was only approximately 2% that of coronal dentin. The low permeability of outer radicular dentin should make it relatively impermeable to toxic substances such as bacterial products emanating from plaque.

Factors modifying dentin permeability include the presence of odontoblast processes in the tubules and the sheathlike lamina limitans that lines the tubules. Collagen fibers have also been observed in most tubules.[71] The functional or physiologic diameter of the tubules is only about 5% to 10% of the actual anatomic diameter (i.e., the diameter seen in microscopic sections).[233] This is small enough to trap and remove bacteria from dentinal fluid,[234] permitting sterile fluid to enter the pulp chamber.

In dental caries, an inflammatory reaction develops in the pulp long before the pulp actually becomes infected with micorganisms.[345] This indicates that bacterial byproducts reach the pulp in advance of the bacteria themselves,[19,284] and the early inflammatory response is elicited by the accumulation of bacterial antigens or byproducts into the pulp, not by the bacteria themselves. Dentinal sclerosis (i.e., reparative dentin formation) beneath a carious lesion reduces permeability[328] by obstructing the tubules, thus decreasing the delivery of irritants into the pulp.

The cutting of dentin during cavity preparation or root canal therapy produces a microcrystalline debris that coats the dentin and clogs the orifices of the dentinal tubules. This layer of debris is termed the *smear layer*. Because of the small size

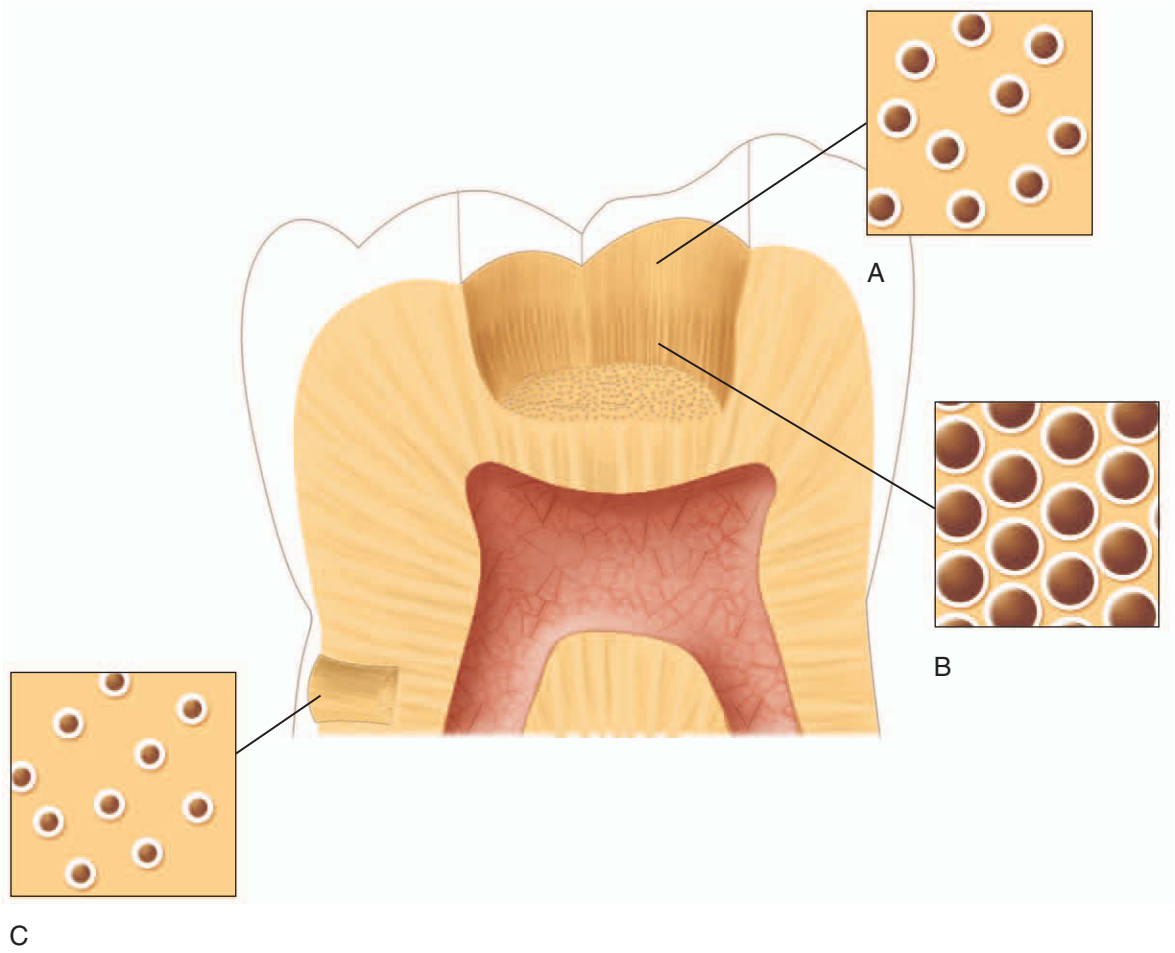


FIG. 12-13 Diagram illustrating the difference in size and density of tubules in the dentinal floor between a shallow **(A)** and a deep **(B)** cavity preparation, in coronal dentin versus **(C)** root dentin. *(Redrawn from Trowbridge HO: Dentin today: research reveals new information about the complexity of dentin.* Dentistry *22:22–29, 1982.)*

of the particles, the smear layer is capable of physically preventing bacteria from penetrating dentin.[234,244] Removal of the debris by acid etching or EDTA greatly increases the permeability of the dentin by widening the orifices of the tubules. Consequently, the incidence of pulpal inflammation may be increased significantly if cavities are treated with an acid cleanser unless a cavity liner, base, or dentin-bonding agent is used. Continued pulpal protection will then depend on the durability of the bond.

When the dentin surface of vital and nonvital teeth was exposed to the oral environment for 150 days, bacterial invasion of dentinal tubules occurred more rapidly in the nonvital teeth.[244] Presumably this was due to the resistance offered by the outward movement of dentinal fluid and the presence of odontoblast processes in the tubules of vital teeth.[280,361,362] Importantly, it has been shown that antibodies or other antimicrobial components are present within the dentinal fluid of teeth with vital pulps.[125]

MECHANICAL PROPERTIES OF ROOT DENTIN

Endodontic therapy has long been criticized for "weakening" teeth, especially roots, possibly making them more prone to fracture. Although some investigators have proposed that endodontically treated root dentin was more "brittle" than normal vital teeth,[80] subsequent punch shear tests of endodontically treated teeth showed only a 14% reduction in dentin toughness compared with vital dentin.[57] The controversy began when the moisture content of pulpless dog teeth was reported to be 9% less than that of vital controls.[137] However, endodontic therapy was shown not to change the Vickers hardness of human root dentin.[199] Much of the increased incidence of coronal fractures of endodontically treated teeth can be attributed to the loss of tooth structure produced by preparing adequate access openings.[152] Similarly, root fractures in endodontically treated teeth are in part due to physical weakening of roots by creation of post spaces.[123,156] In addition, fatigue life studies have shown that dentin from old patients has a much shorter fatigue life than dentin from young teeth.[15] The perceived increased incidence of root fractures in endodontically treated teeth may actually reflect a slow, spontaneous reduction in the fracture toughness of old dentin,[11] regardless of root canal treatment.

Any occlusal stress applied to the coronal dentin is magnified in the root because it has a smaller volume and cross-sectional area. Collagen in the dentin matrix does not turn over metabolically, so it is never repaired and may accumulate microcracks over decades of function. When collagen is

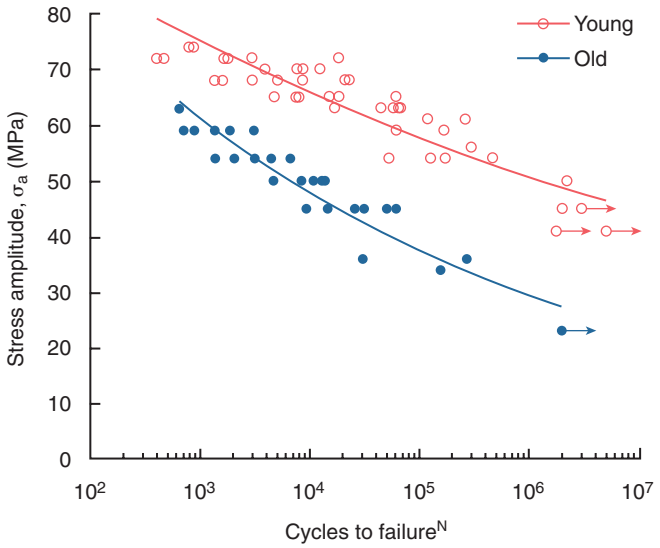


FIG. 12-14 The fatigue life diagram for cyclic loading of coronal dentin using 4-point flexure. The young dentin (average age = 25 years) and old dentin (average age = 62 years) were obtained from fully erupted second and third molars. Data points with *arrows* indicate specimens that did not fail. *(Courtesy Dr. Dwayne Arola, University of Maryland.)*

secreted into the forming dentin matrix, a number of noncollagenous proteins are secreted at the same time, some of which bind to collagen. For instance, dentin sialoprotein (DSP),[42,116] dentin phosphophoryn (DPP),[42,70,75] and matrix metalloproteinases (MMPs)[336] are all known to bind to collagen. MMPs such as gelatinase A have been extracted from dentin in their active form.[225] More recently, both collagenolytic activity and gelatinolytic activity have been measured in mineralized dentin powder.[281] These enzymes may slowly attack the collagenous matrix of dentin, thereby weakening it over many decades. The mineralized dentin surrounding post spaces in roots that had been under function for more than 10 years was shown to be disrupted and fragmented.[89] Functional stresses may cause microcracking of dentin in a manner similar to that seen in cortical bone.[358] Fatiguing of roots over decades[226,247,248] may be responsible for the fractured roots in endodontically treated teeth in older patients. Fatigue life experiments show that old coronal dentin tolerates fewer loading cycles before fracture (Fig. 12-14) than young dentin.[15] If this is confirmed in root dentin, and if the mechanism or mechanisms responsible for this increased susceptibility to fatiguing can be identified, then effective interventions may be developed to increase the fatigue life of roots that have been endodontically treated.

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.

Odontoblast Layer

The outermost stratum of cells of the healthy pulp is the odontoblast layer (Figs. 12-15 and 12-16). 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.[63] 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 spaces approximately 30 to 40 nm in width. Odontoblast cell bodies are connected by tight and gap junctional complexes.[30,63,149] Gap junctions are formed by connexin proteins[110] 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.[224] Whereas the odontoblasts of the mature coronal pulp are usually columnar, those in the midportion of the radicular pulp are more cuboidal (Fig. 12-17).[63] 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.[224] 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.[235]

There are 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 signal molecules can pass between cells (Fig. 12-18) to synchronize secretory activity that produces relatively uniform predentin layers (see Fig. 12-16). 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.[330] It appears that tight junctions determine the permeability of the odontoblast layer when dentin is covered by enamel or cementum by restricting the

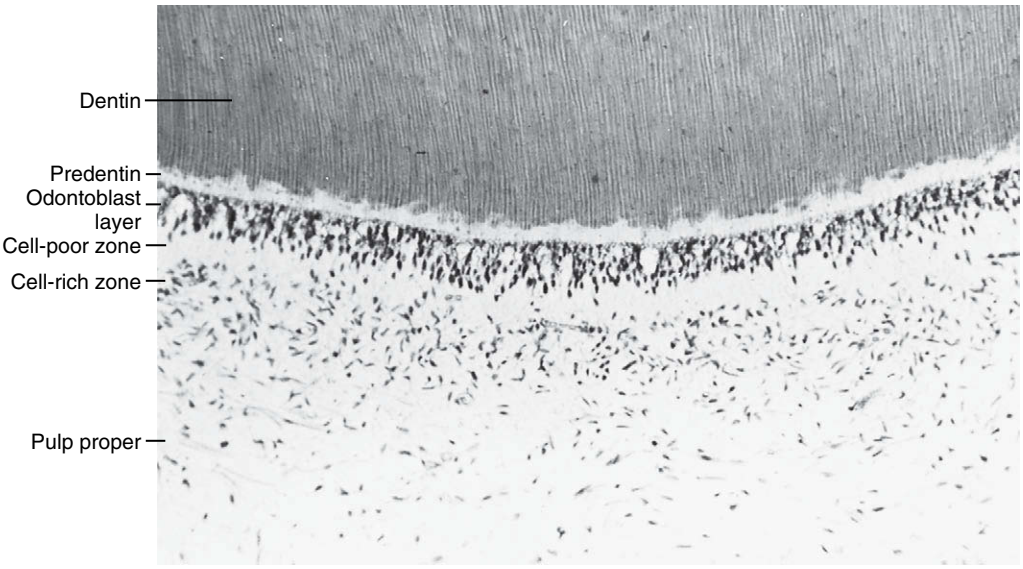


FIG. 12-15 Morphologic zones of the mature pulp.

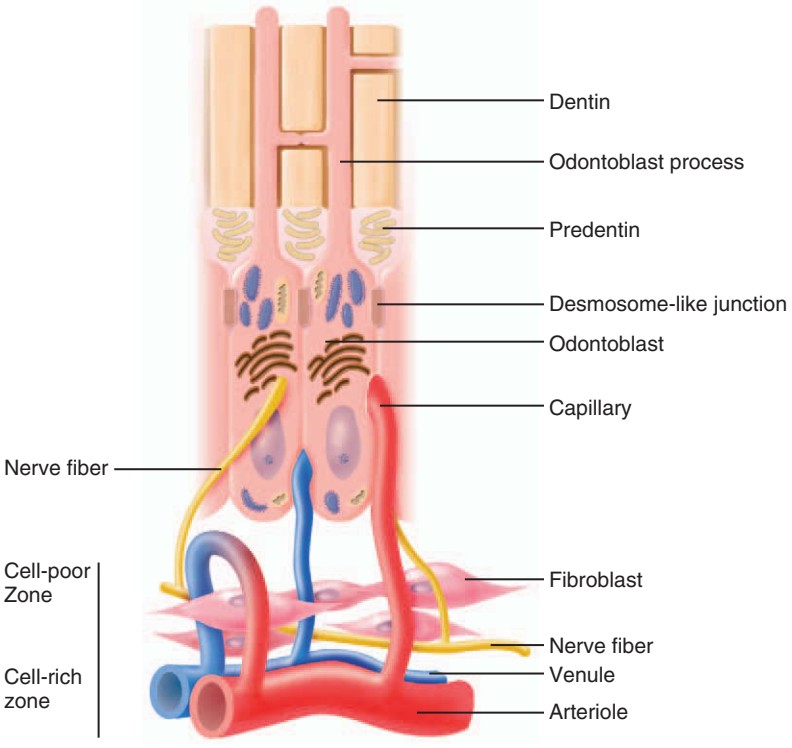


FIG. 12-16 Diagrammatic representation of the odontoblast layer and subodontoblastic region of the pulp.

passage of molecules, ions, and fluid between the extracellular compartments of the pulp and predentin.[30] During cavity preparation, these junctions are disrupted, thereby increasing dentin permeability.[350,351]

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. 12-15) 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. 12-16). The presence or absence of the cell-poor zone depends on the functional status of the pulp.[63] 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

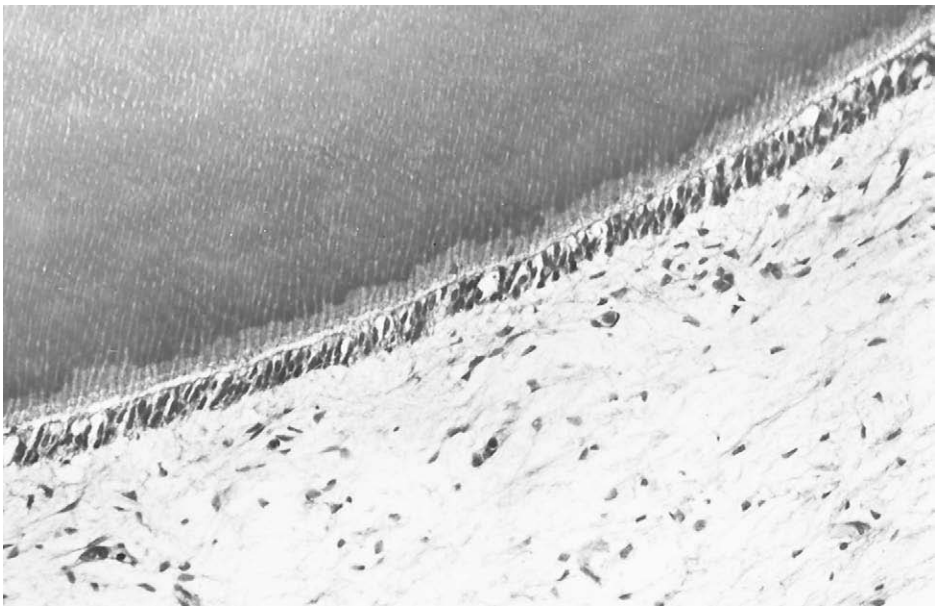


FIG. 12-17 Low columnar odontoblasts of the radicular pulp. The cell-rich zone is inconspicuous.

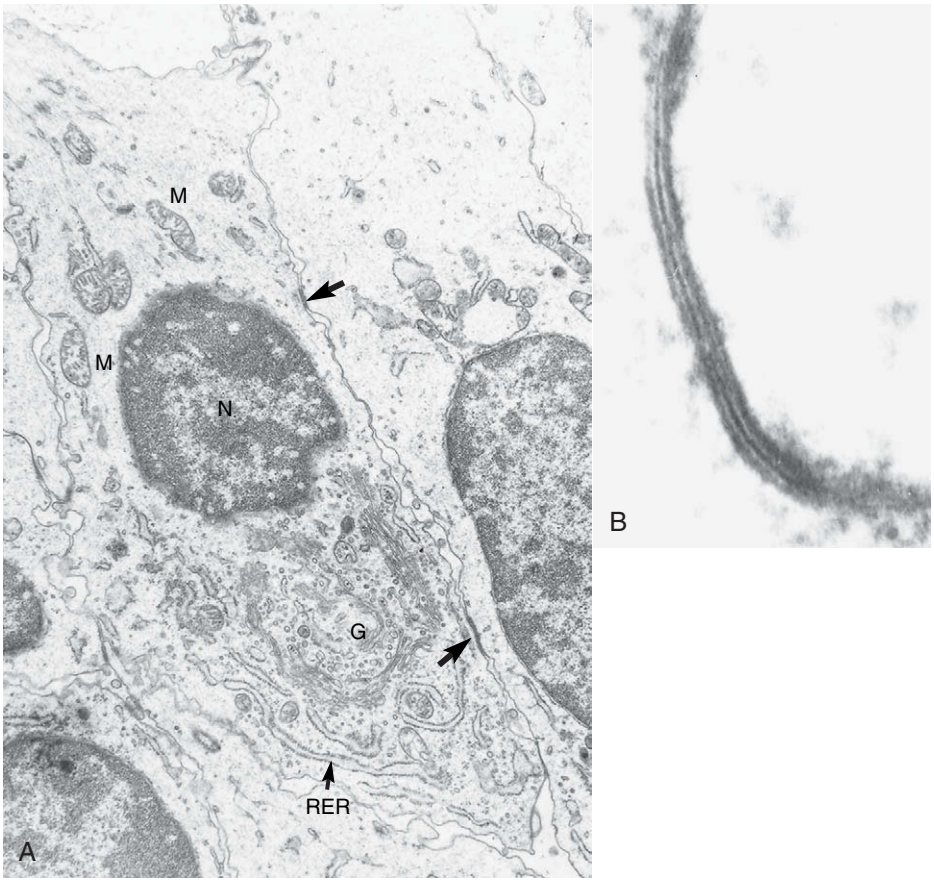


FIG. 12-18 A, Electron micrograph of a mouse molar odontoblast demonstrating gap junctions *(arrows)*, nucleus (N), mitochondria (M), Golgi complex (G), and rough endoplasmic reticulum (RER). **B,** High magnification of a section fixed and stained with lanthanum nitrate to demonstrate a typical gap junction. *(Courtesy Dr. Charles F. Cox, School of Dentistry, University of Alabama.)*

more central region of the pulp (see Fig. 12-15). 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[119] 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.[388] 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.[96] This mitotic activity is probably the first step in the formation of a new odontoblast layer.[73,240-242,320] Studies implicate stem cells as a source for these replacement odontoblasts.[326]

Pulp Proper

The pulp proper is the central mass of the pulp (see Fig. 12-15). 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 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.[63,224] 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[167,236] through canaliculi, just as osteocytes and cementocytes are linked together 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. 12-19). 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,[196,375] although small amounts of type V collagen have been found in the ECM. In addition to proteoglycans[40,82,117] and collagen,[196,202] the odontoblast secretes dentin sialoprotein[42] and phosphophoryn,[42,70] a highly phosphorylated phosphoprotein involved in extracellular mineralization.[42,75] Phosphophoryn is unique to dentin and is not found in any other mesenchymal cell types.[75] 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.[85]

In contrast to the active odontoblast, the resting or inactive odontoblast has a decreased number of organelles and may become progressively shorter.[63,224] These changes can begin with the completion of root development and eruption when dentin production shifts from primary to secondary dentin.

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.[106,150] Microtubules extend from the cell body out into the process.[106,151] 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 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. The peritubular

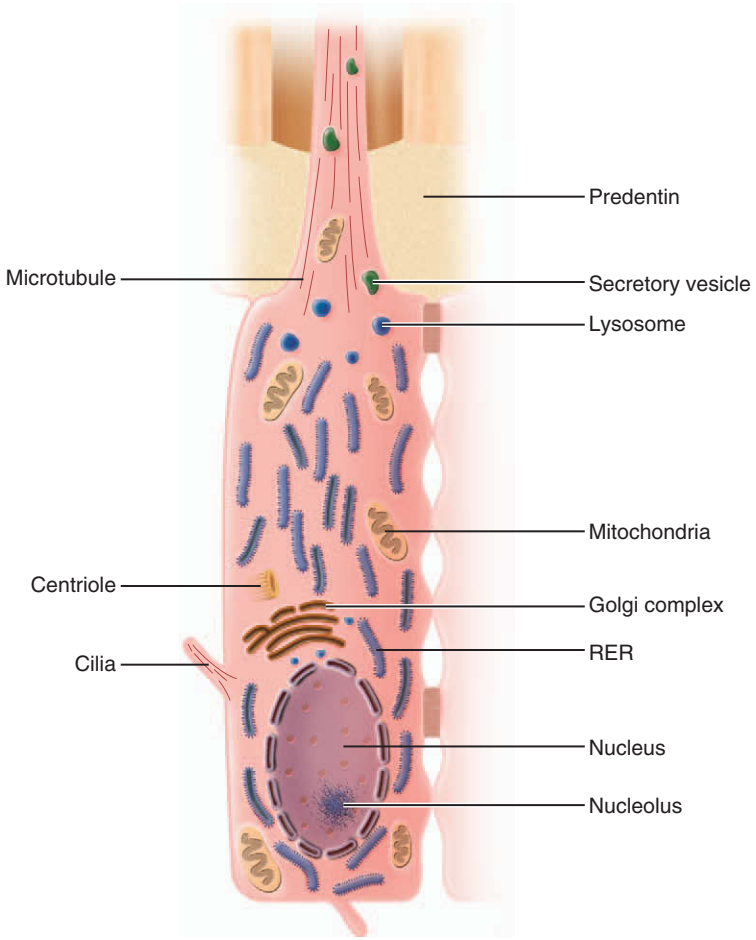


FIG. 12-19 Diagram of a fully differentiated odontoblast. *RER,* Rough endoplasmic reticulum.

dentin matrix within the tubule is lined by an electron-dense limiting membrane called the *lamina limitans*.[244,333,372] 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.* 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,[51,107,333,372] 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 DEJ,[121,168,318,381] but it has been suggested that what has been observed in scanning electron micrographs is actually the lamina limitans.[333,334,372]

*References 44, 47, 73, 77, 192, and 241.

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 entire thickness of dentin.[318] 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.[51] 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 does not turn over, 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 lifespan of the odontoblast coincides with the lifespan 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 organ-

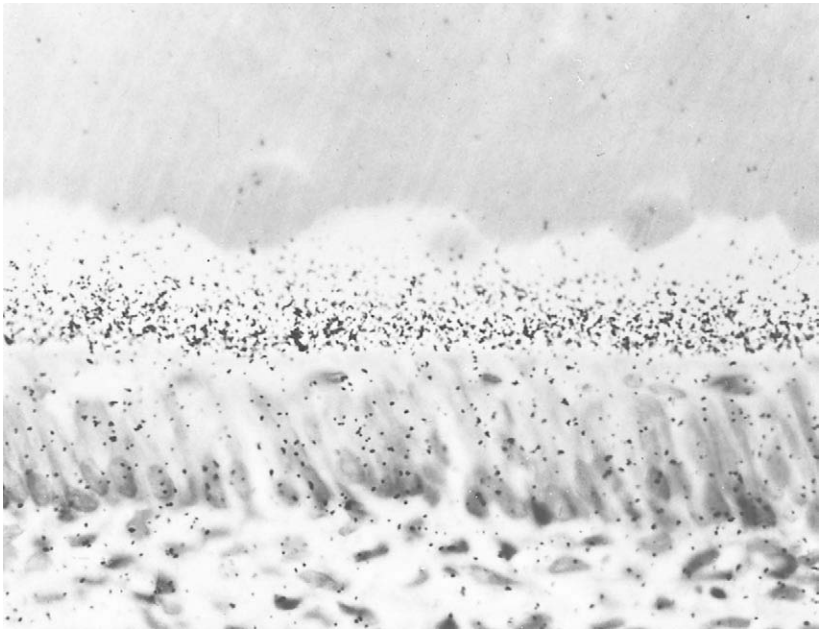


FIG. 12-20 Autoradiograph demonstrating odontoblasts and predentin in a developing rat molar 1 hour after intraperitoneal injection of ^{3}H-proline.

elles of the active odontoblast.[375,376] 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[375] (Fig. 12-20). 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.

Similar tracer studies[376] 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 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.[83]

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[128]). 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*.[17] 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[96] 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 is stimulated by neuropeptides; in turn, fibroblasts produce NGF and proinflammatory cytokines during inflammation.[32,380,382] NGF plays an important role not only in 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 Nerves Fibers in this chapter).[380]

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 quite active in endocytosis and phagocytosis (Fig. 12-21). 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.[265] 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 naïve or memory T cells.[124] 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. A recent study has shown that a subset of macrophages express lymphatic markers, indicating a link between macrophages and lymphatic function and development.[20]

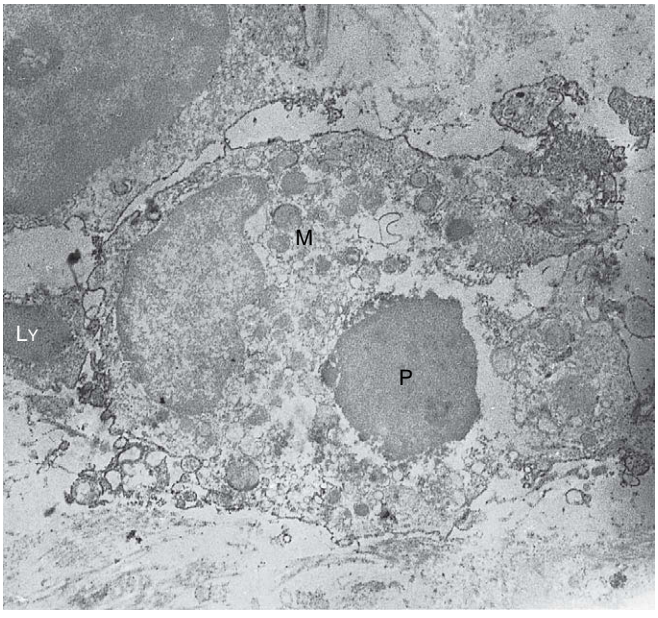


FIG. 12-21 Immunoelectron micrograph of an HLA-DR+ matured macrophage (M) in the human pulp, showing a phagosome (P). *Ly,* Lymphocyte.

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*.[164,264] Dendritic cells are primarily found in lymphoid tissues, but they are also widely distributed in connective tissues, including the pulp[303] (Fig. 12-22). 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. 12-23). In the normal pulp, they are mostly located in the periphery of the coronal pulp close to the predentin but migrate centrally in the pulp after antigenic challenge.[388] 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 occurs (Fig. 12-24). Fig. 12-25 shows a cell-to-cell contact between a dendritic-like cell and a lymphocyte.

Lymphocyte

Hahn et al.[124] 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.[191] 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.[164,303] B lymphocytes are scarcely found in the normal 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.[303] 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.[27,93-95,127,304] An investigation employed an oxygen-sensitive microelectrode inserted into a rat incisor pulp with a micromanipulator.[386] The authors



FIG. 12-22 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.

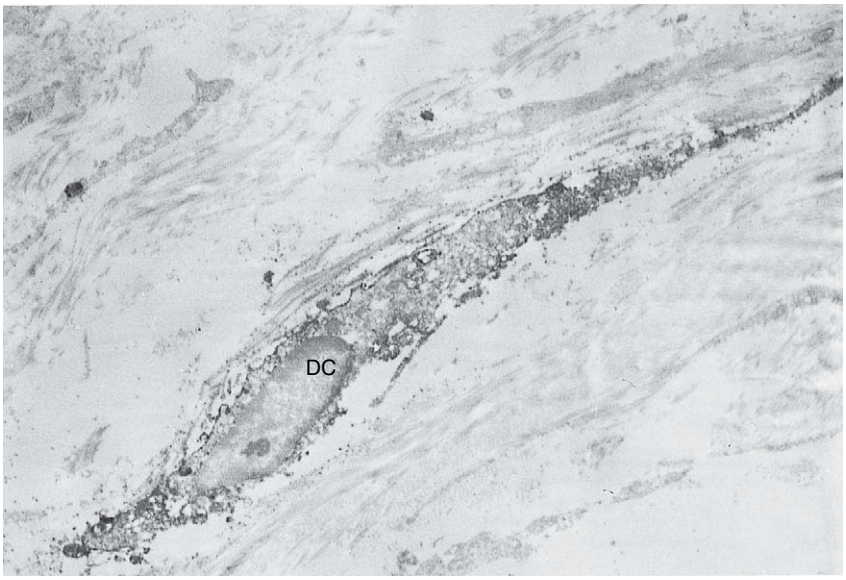


FIG. 12-23 Immunoelectron micrograph of a dendritic-like cell (DC) in the human pulp, showing a dendritic profile with a relatively small amount of lysosomal structures.

reported that odontoblasts consumed O_2 at the rate of 3.2 ± 0.2 ml/min/100 g of pulp tissue.[386]

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.[26]

In addition to the usual glycolytic pathway, the pulp has the ability to produce energy through a phosphogluconate (i.e., pentose phosphate) shunt type of carbohydrate metabolism,[95] 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.[175]

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.[94,163] One study[127] 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

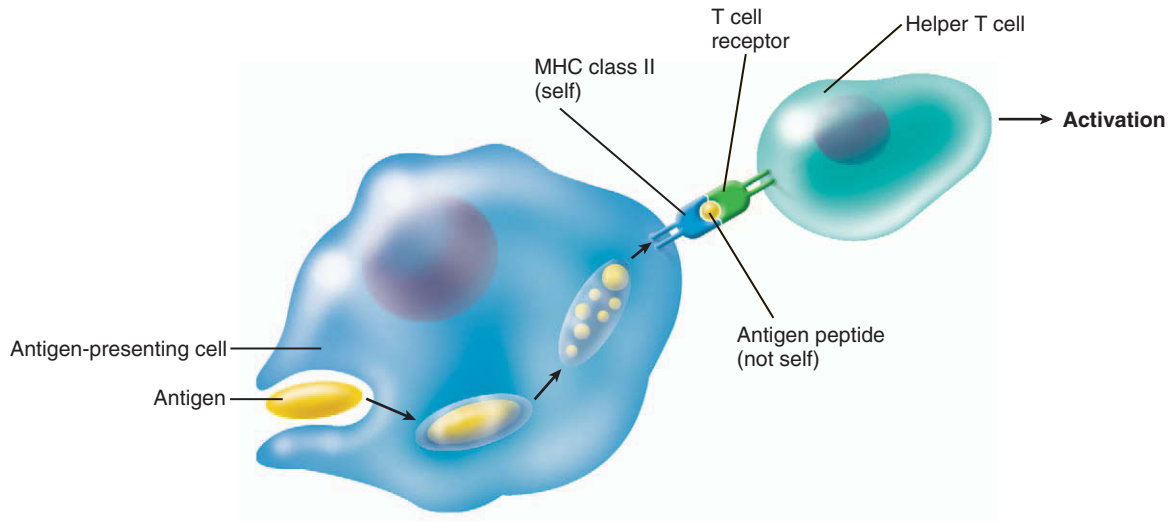


FIG. 12-24 Function of MHC class II molecule-expressing cells. They act as antigen-presenting cells that are essential for the induction of helper T cell–dependent immune responses.

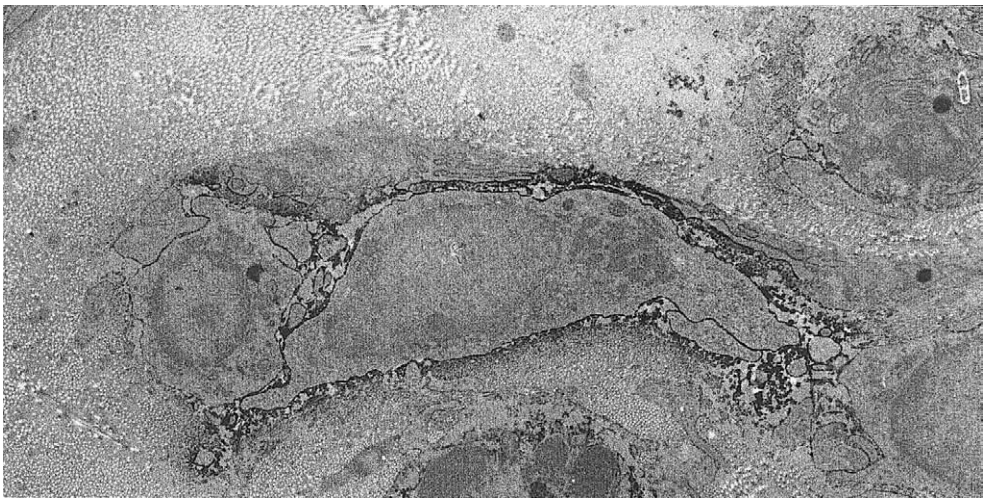


FIG. 12-25 Immunoelectron micrograph of a cell resembling a dendritic cell and a lymphocyte. They show cell-to-cell contact.

cells metabolize succinic acid, they produce $^{14}CO_2$ that can be trapped and quantitated by a liquid scintillation counter.[127] This technique requires only a few milligrams of tissue.

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. 12-26). 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

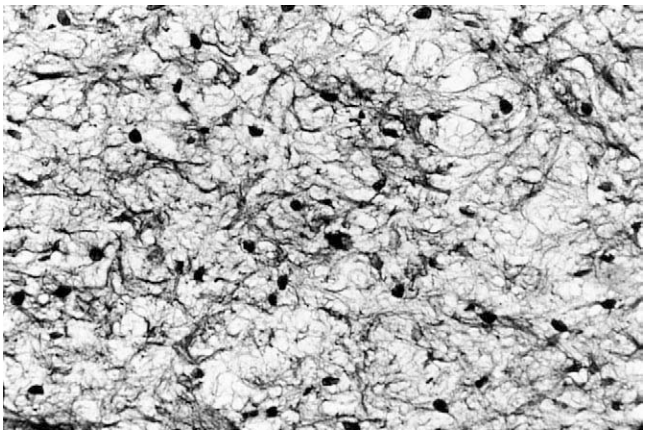


FIG. 12-26 Delicate network of pulpal collagen fibers as demonstrated by the Pearson silver impregnation method.

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.[378]

Connective tissue consists of cells and fibers, both embedded in ground substance or extracellular matrix (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.[378]

Nearly all proteins of the ECM are glycoproteins.[221] Proteoglycans are an important subclass of glycoproteins.[117] 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.[113] Tenascin is another substrate adhesion glycoprotein.

In the pulp, the principal proteoglycans include hyaluronic acid dermatan sulfate, heparan sulfate, and chondroitin sulfate.[221] The proteoglycan content of pulp tissue decreases approximately 50% with tooth eruption.[202] 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.[194] 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.[203,221] The hyaluronan receptor-1 is expressed on lymphatic vessels and also on immune cells in the dental pulp.[20] Hyaluronan is removed from the tissue by the lymphatics and metabolized in the lymph nodes[98] and by endothelial cells in the liver.[126,285]

Elastic Fibers

Elastic fibers comprise an elastin core and a surrounding microfibrillar network and provide elasticity to the tissue.[272] 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.[126,285]

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.[133,302] 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.[166]

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.[355] 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.[195,317] 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.[355]
- 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 aggregation of tropocollagen is somehow mediated by the GAGs. The conversion of soluble collagen into 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. 12-27). Pulpectomy procedures should engage the pulp with a barbed broach in the region of the apex, since it generally affords the best opportunity to remove this tissue intact.[342]

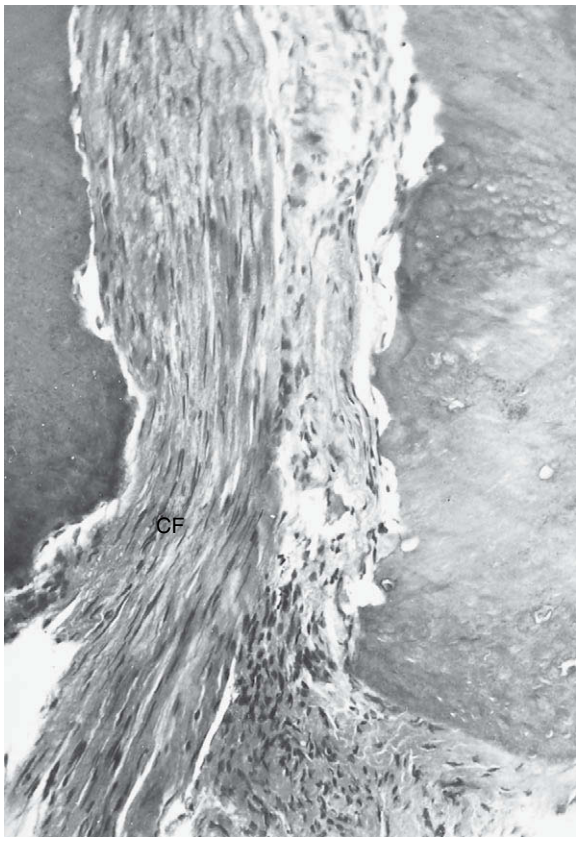


FIG. 12-27 Dense bundles of collagen fibers (CF) in the apical pulp.

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 brainstem to the brain (Fig. 12-28; see also Chapters 3 and 19). Transmission of sensory information consists of a cascade of events involving input, processing, and sensing,[313] 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 (see Chapter 19). The sensory system of the pulp appears to be well

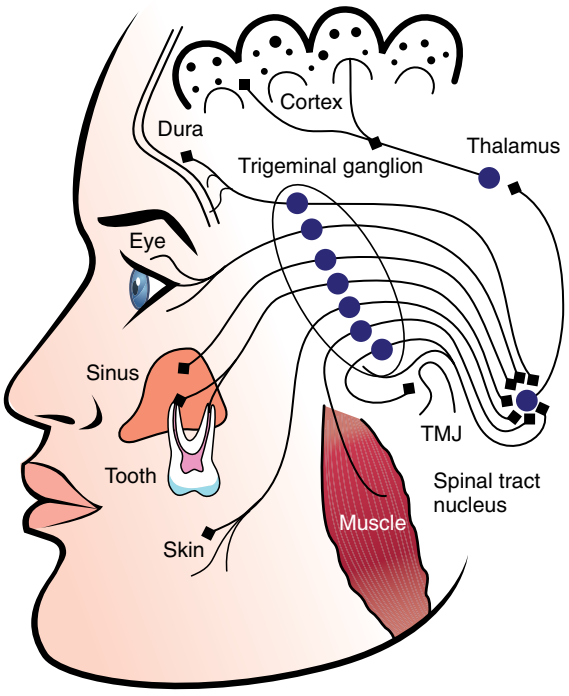


FIG. 12-28 Schematic drawing illustrating the convergence of sensory information from teeth to higher brain centers.

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.[87,160,161]

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.[231] 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,*[154] which provide neurogenic modulation of the microcirculation, inflammatory reactions,[136] and perhaps regulate dentinogenesis.[48]

The sympathetic innervation of teeth derives from the superior cervical ganglion (SCG).[10,286] 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.[223] Sympathetic fibers appear with blood vessels at the time the vascular system is established in the dental papilla.[101] In the adult tooth pulp, sympathetic fibers form plexuses, usually around pulpal arterioles (Fig. 12-29). Stimulation of these fibers results in constriction of the arterioles and a decrease in blood flow.[2,78] 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 to 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.[2,155]



FIG. 12-29 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.)*

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.[270,307] It has been reported that the neuropeptide, vasoactive intestinal polypeptide (VIP), is localized in the parasympathetic neurons.[210,211] The origin of these fibers is uncertain insofar as no form of surgical denervation has resulted in complete loss of VIP-containing fibers from the dental pulp.[366]

Sensory nerve fibers are usually classified according to their diameter, conduction velocity, and function as shown in Table 12-2. 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, since both fiber types can be nociceptors.[154,160,161,230,252] 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, since both innervate the dentinal tubules, and both are stimulated by dentinal fluid movement (Fig. 12-30). Approximately 90% of the A fibers in dental pulp are A-delta fibers.[230] Table 12-3 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.[101] Although only unmyelinated fibers are observed in the dental papilla, a proportion of these fibers are probably A fibers that have lost their myelin sheath. Myelinated fibers are the last major structures to appear in the developing human dental pulp.[13] The number of nerve fibers gradually increases, and some branching occurs as the fibers near the dentin. During the bell stage very few fibers enter the predentin.[101]

TABLE 12-2

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	0.5–2.0
Sympathetic	Postganglionic sympathetic	0.3–1.3	0.7–2.3

TABLE 12-3

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

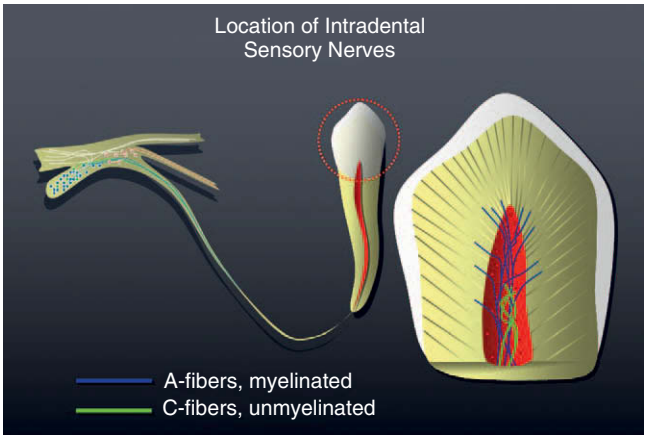


FIG. 12-30 Schematic drawing illustrating the location of A and C fibers in the dental pulp. Myelinated A fibers are located in the periphery of the pulp, penetrating the inner part of dentin. Unmyelinated C fibers are located in the deeper part of the pulp proper.

The sensory nerves of the pulp arise from the trigeminal nerve and pass into the radicular pulp in bundles by way of the foramen in close association with arterioles and venules (Fig. 12-31). 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. 12-32). 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. 12-30).[292] It should be noted that single neurons have been reported to innervate the pulps of multiple teeth in animal studies.[145] Assuming similar innervation patterns occur 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 patients have difficulty identifying the inflamed pulp until the inflammation reaches the periradicular tissue, which is highly innervated with proprioceptors. This is discussed in greater detail in Chapters 3 and 19.

In the human premolar, the number of unmyelinated axons entering the tooth at the apex reached a maximal number shortly after tooth eruption.[160] 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, since A fibers are more easily electrically stimulated than C fibers.[104]

A quantitative study of axons 1 to 2 mm coronal to the root apex of fully developed human canine and incisor teeth[160] 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.[160,161]

The nerve bundles pass upward through the radicular pulp together with blood vessels (see Fig. 12-31). 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. 12-33). Full development of this plexus does not occur until the final stages of root formation.[87] 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.[131,252-254,256] 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. 12-34 and 12-35). The extent to which dentin is innervated has been the subject of numerous investigations.[45,47,48,52,87,200] With the exception of the innervation of 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 very high concentration of lidocaine solution is needed to block the response of intradental nerves to mechanical stimulation of the dentin.[6]

One investigator[122] 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. 12-36). 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

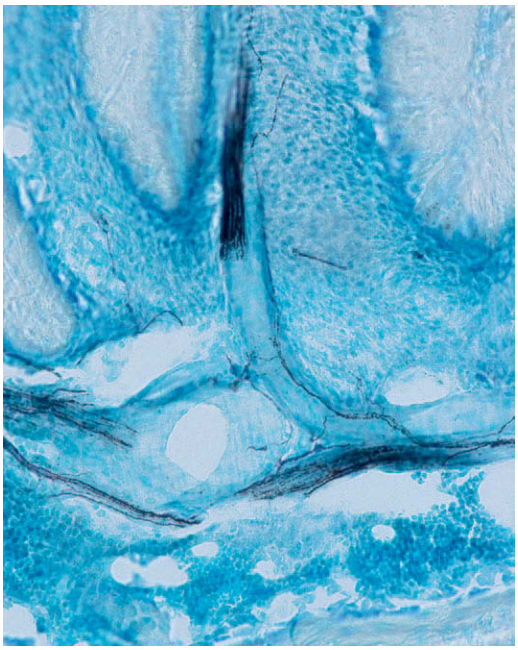


FIG. 12-31 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows distribution of sensory nerves in the apical area of a rat molar. Nerve fibers are seen associated with blood vessels and enter the dental pulp in nerve bundles. *(Courtesy Dr. Inge Fristad, Department of Clinical Dentistry, University of Bergen.)*

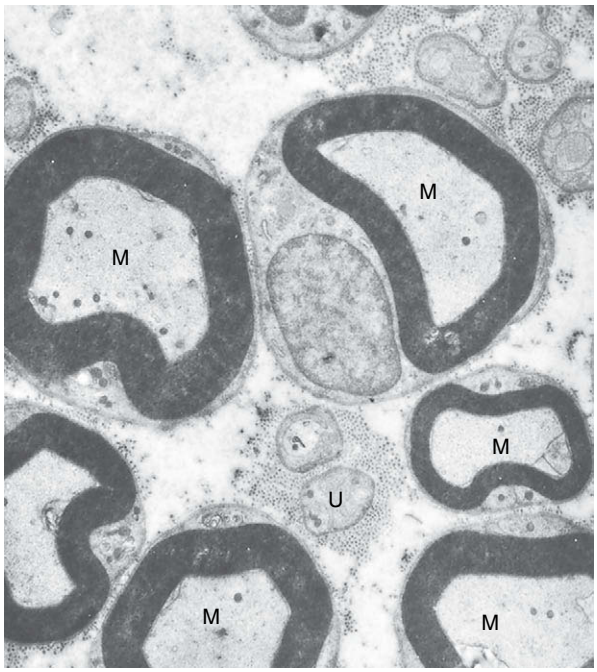


FIG. 12-32 Electron micrograph of the apical pulp of a young canine tooth, showing in cross section myelinated nerve axons (M) within Schwann cells. Smaller, unmyelinated axons (U) are enclosed singly and in groups by Schwann cells. *(Courtesy Dr. David C. Johnsen, School of Dentistry, Case Western Reserve University.)*

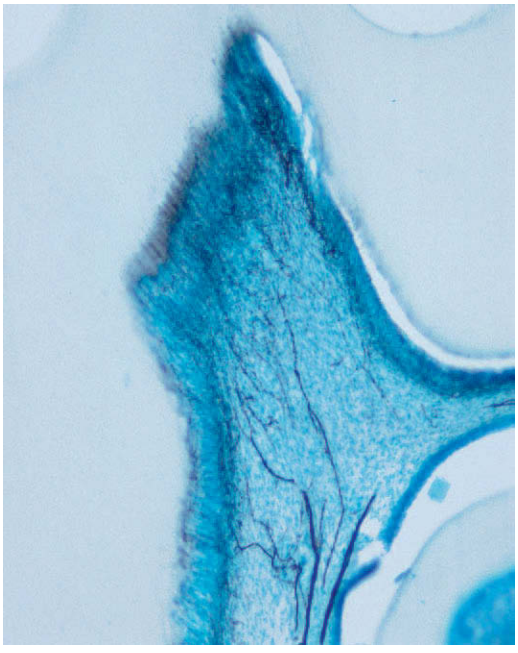


FIG. 12-33 Histologic section, immunohistologically stained for calcitonin gene-related peptide (CGRP), shows the distribution of sensory nerves in a rat molar. Nerves enter the coronal pulp in bundles, ramify in a network beneath the odontoblasts (i.e., plexus of Raschkow), before entering between the odontoblasts and the inner part of dentin.



FIG. 12-34 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. 12-35 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.*)

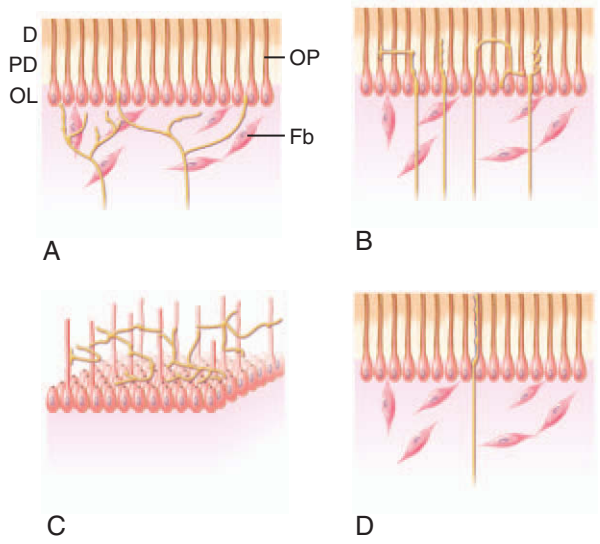


FIG. 12-36 Schematic drawing showing distribution of nerve fibers in the dentin-pulp border zone. **A,** Fibers running from the subodontoblastic plexus to the odontoblast layer. *D,* Dentin; *Fb,* fibroblast; *OL,* odontoblast layer; *OP,* odontoblast process; *PD,* predentin. **B,** Fibers extending into the dentinal tubules in the predentin. **C,** Complex fibers that branch extensively in the predentin. **D,** Intratubular fibers extending into the dentin.

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. 12-33). The area covered by a single such terminal complex often reaches thousands of square micrometers.[122,253]

Intratubular nerve endings are most numerous in the area of the pulp horns, where as many as 40% of the tubules may contain fibers.[48,200] The number of intratubular fibers decreases in other parts of the dentin, and in root dentin only about 1% of dentinal tubules contain a fiber. This notion has been challenged in a study that stained pulps for protein gene-product 9.5, a specific marker for nerves.[218] 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.[47] 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 are not synaptically linked.[149]

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 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),[189,212] and that the cell does not respond to electric stimulation.[189,379] 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.[39,201] 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.[78] The excitability of C fibers is less affected than that of A fibers by a reduction in blood flow.[341]

Of clinical interest is the evidence that nerve fibers of the pulp may be resistant to necrosis[84,239] because their cell bodies are found in ganglia outside the pulp. Since nerve bundles in general are more resistant to autolysis than other tissue ele-

*A receptor cell is a non–nerve cell capable of exciting adjacent afferent nerve fibers. Synaptic junctions connect receptor cells to afferent nerves.

ments, 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, since C fibers are often able to function in the presence of hypoxia.[341] This may offer an explanation as to 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 pain may be due to the transfer of noxious chemicals to terminals located in periapical tissues.[239]

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 brainstem.[46,367] Because each single-rooted tooth contains about 2000 nerve fibers,[160,161] extirpation of the pulp is shown to cause both neurochemical and degenerative changes of their cell bodies in the gasserian (trigeminal) ganglion.[120,174,315] The central projection of these nerves to the spinal nucleus of the trigeminal nerve are also affected,[343] and there is evidence for transsynaptic changes[314,315] 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.[368] Attempted regeneration by axonal sprouting may result in altered expression of various receptors, resulting in sensitivity to norepinephrine (via increased adrenergic receptor activity)[261] 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 Chapter 19). *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%.[222,360]

Following tissue injury or tissue inflammation, extensive changes occur in the gene expression of sensory ganglion nerves and, by way of transsynaptic mechanisms, in their central projections.[47,253,254,315] One example is the upregulation of inducible gene transcription factors such as c-*fos*[53] and different subsets of sodium channels.[114] 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 brainstem, but chronic pulpitis causes prolonged increases in c-*fos* expression is some brainstem neurons.

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,[52-54,182] these nerves may have been due to the transport of peripheral signaling molecules to the cell body by way of retrograde axoplasmic flow.[45,54] This could induce changes in the expression of many genes, resulting in central senstization[8,44] that may require many months to correct.[327] Reports of nerve sprouting in human inflamed pulps have been confirmed in different studies.[296,297,303] Such reactions might contribute to increases in dentin sensitivity as well as expansion of receptive fields.[253,254,256] Sprouting of sympathetic fibers has also been reported,[135] but the timing appears to be different. The functional implications and how this is related to pain mechanisms are unknown, but it has been suggested that these reactions are involved in healing and nociception following pulpal inflammation.[111,135,136]

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.[252] Bender et al.[18] found that in anterior teeth, the optimal placement site of the electrode is the incisal edge, since 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.[104] 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 in recent 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.[38,39,249,361,363] 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.[38,230] 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. Recently, classical mechanotransducers have been recognized on pulpal afferents, providing a mechanistic support to this theory.[138] Thus fluid movement in the dentinal tubules is translated into electric signals by receptors located in the axon terminals innervating dentinal tubules. Indeed, there is a positive correlation between rate of fluid flow in the tubules and discharge evoked in intradental nerves,[229,230,363] with outward fluid movements producing a much stronger nerve response than inward movements.[230]

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.[250,346] 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 to a barrage of impulses to the brain. Some ion channels are activated by voltage, some by chemicals, and some by mechanical pressure.[230,249,253] 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.*[107] 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. 12-37). According to Brännström,[38] 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.

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.[251,256] 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.[249] 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.[253,254] Capsaicin, the pungent active ingredient in hot peppers, is known to simulate C fibers and a subset of A-delta fibers.[154] Capsaicin activates transient receptor potential, subtype vanilloid 1, (TRPV1).[59] 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.[260] The ability of the TRPV1 antagonist, capsazepine, to inhibit acid-, heat-, and capsaicin-activated trigeminal neurons[206] 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.[384]

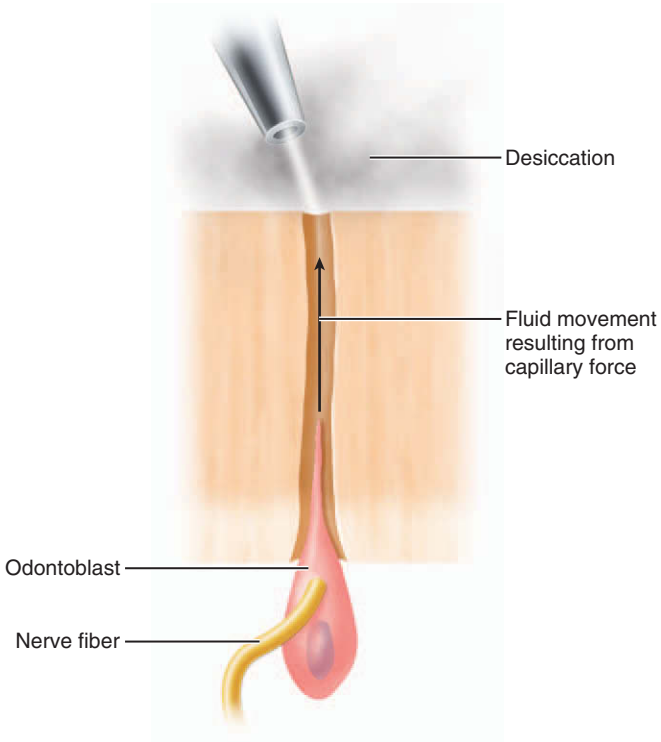


FIG. 12-37 Diagram illustrating movement of fluid in the dentinal tubules resulting from the dehydrating effect of a blast of air from an air syringe.

It has also been shown that pain-producing stimuli are more readily transmitted from the dentin surface when the exposed tubule apertures are open[148,162] and the fluid within the tubules is free to flow outward.[162,230,363] 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.[148,254]

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

*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.

*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.[55] This is far in excess of the compressive strength of dentin, listed as 245 MPa, as evidenced by the shallow grooves lined by smear layers created in dentin using this force.[56]

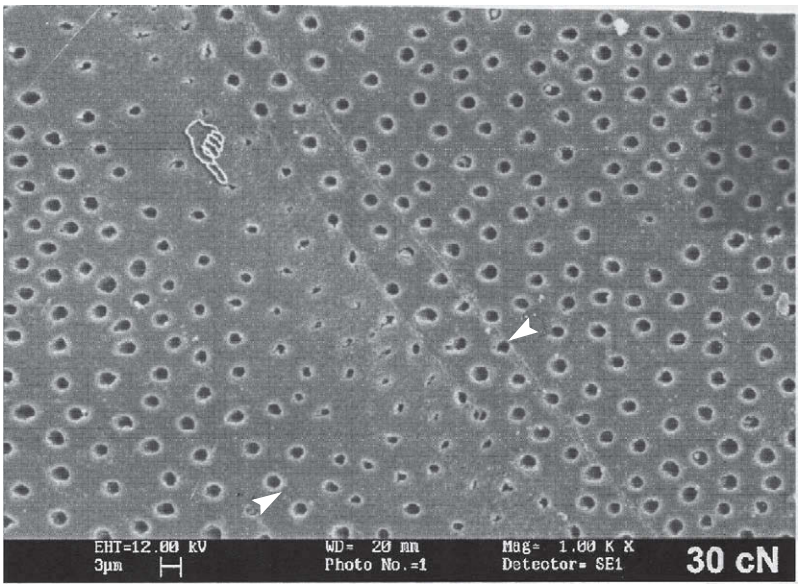


FIG. 12-38 Scanning electron micrograph of a shallow groove *(between white arrowheads)* created in polished dentin by a dental explorer tine under a force of 30 g (30 cN). Note the partial occlusion of the tubules by smeared matrix. *(From Camps J, Salomon JP, Meerbeek BV, et al: Dentin deformation after scratching with clinically relevant forces.* Arch Oral Biol *48:527, 2003.)*

the underlying pulp (Fig. 12-38).[55,56] Considering the density of the tubules in which hydrodynamic forces would be generated by probing, thousands of nerve endings would be simultaneously stimulated when a dental explorer is scratched across dentin.

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 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. 12-39).[76,192] 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.[37,39] There is controversy regarding whether exposed dentin is simply sensitive or becomes truly hypersensitive.[253,254,256] Growing evidence indicates that new sodium channels responsible for activating nerves are expressed in nerve tissue exposed to inflammation.[114,129,293] 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.[49] 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[271,348,365] can accomplish this objective:

1. Formation of a smear layer on the sensitive dentin by burnishing the exposed root surface[148,253,254]
2. Application of agents, such as oxalate compounds, that form insoluble precipitates within the tubules[148,278]
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[309]
4. Application of dentin bonding agents to seal off the tubules[287]

Dentin sensitivity can be modified by laser irradiation, but clinicians must be concerned about its effect on the pulp.[326,336]

Neuropeptides

Of immense importance in pulp biology is the presence of neuropeptides in pulpal nerves.[45,48,52,327] Pulpal nerve fibers contain neuropeptides such as calcitonin gene–related peptide (CGRP) (see Figs. 12-31, 12-33, 12-34),[44] substance P (SP),[253,268] neuropeptide Y (see Fig. 12-29), neurokinin A (NKA),[14] and VIP.[213,365] 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.[45,47] Release of these peptides can be triggered by numerous stimuli, including tissue injury,[14] complement activation, antigen-antibody reactions,[264] or antidromic stimulation of the inferior alveolar nerve.[268,269] Once released, vasoactive peptides produce vascular changes that are similar to those evoked by histamine and bradykinin (i.e., vasodilation).[269] In addition to their neurovascular properties, SP and CGRP contribute to inflammation and promote wound healing.[32,344] The release of CGRP can be mod-

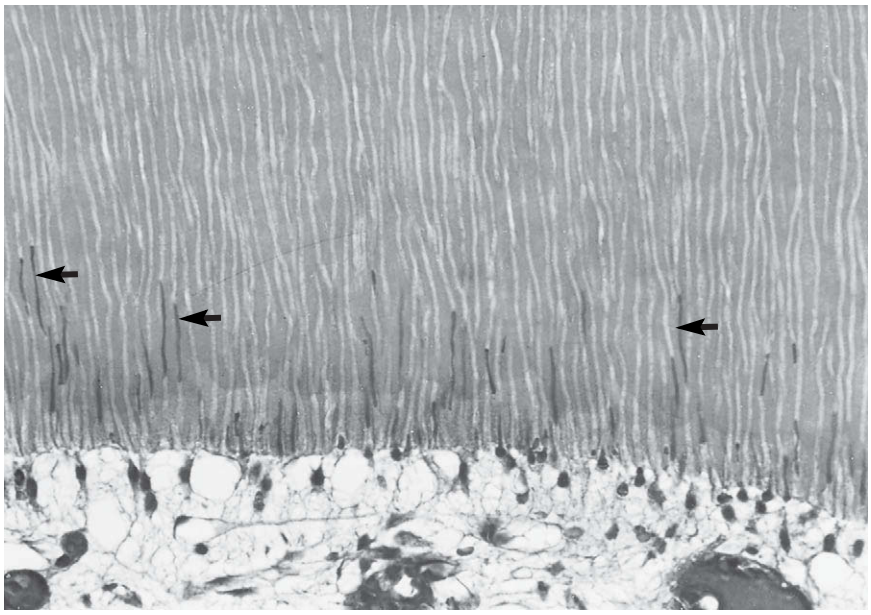


FIG. 12-39 Odontoblasts (*arrows*) displaced upward into the dentinal tubules.

ified by sympathetic agonists and antagonists,[111,130] offering the promise of using such agonists to treat dental pain. This latter point is important because sympathetic agonists are used by clinicians 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 on the nerves.[129,130] In cats, capsaicin acutely activates and chronically blocks the TRPV1-expressing classes of C and A-delta nociceptors in the pulp.[154] 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., towards the peripheral terminals) simply means that the afferent barrage goes in the opposite direction of the orthograde stimulation (towards 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, then the peripheral end of the nerve is electrically stimulated. This causes an action potential to travel backward toward the periphery, which causes release of neuropeptides in the pulp.[268] All branches of the nerve also depolarize and release neuropeptides (the so-called axon reflex).[306]

It has been reported[230,268] that mechanical stimulation of dentin produces vasodilation within the pulp, presumably by causing the release of neuropeptides from intradental sensory fibers.[268] Electric stimulation of the tooth has a similar effect.[142] The pulpal concentrations of CGRP, SP, and NKA are elevated in painful human teeth over healthy control teeth extracted for orthodontic treatment.[14] These peptides are also elevated in pulps beneath advancing carious lesions.[295,296]

Plasticity of Intradental Nerve Fibers

It has become apparent that the innervation of the tooth is a dynamic situation in which the number, size, and cytochemistry of nerve fibers can change because of aging,[99,207,327] tooth injury,[45,47,48,52] and dental caries.[296] 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.[45,47,48,52,54] When inflammation subsides, the number of sprouts decreases. Fig. 12-40 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,[227] 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.[190,289] This suggests that GDNF may function as a neurotrophic factor for the subset of larger nerves in the tooth, which apparently mediate mechanosensitive stimuli, whereas NGF is suggested to support neurons responsible for nociception.[243] NGF is the most extensively investigated among the trophic factors.[198] 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.[50,54] NGF receptors are found on intradental sensory fibers and Schwann cells.[47] 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.[47] Maximal sprouting of CGRP- and SP-containing nerve fibers corresponds to areas of the pulp where there is increased production of NGF.[54] Fig. 12-41 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, since a coordinated increase of pulpal

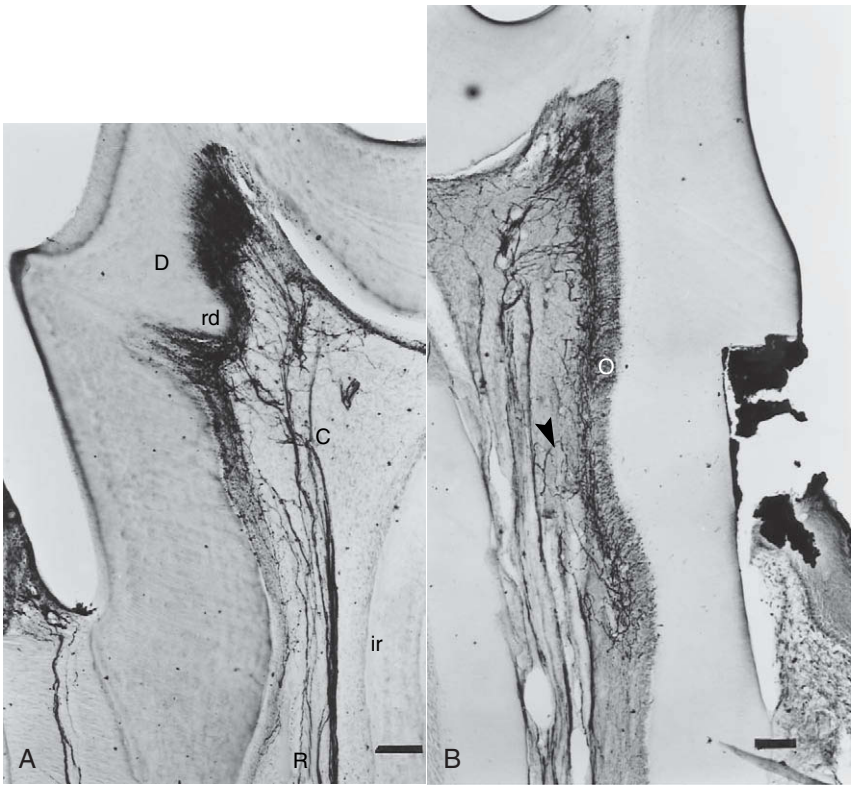


FIG. 12-40 A, Normal distribution of calcitonin gene–related peptide (CGRP)-immunoreactive sensory fibers in adult rat molar. Nerve fibers typically are unbranched in the root (R), avoid interradicular dentin (ir), and form many branches in coronal pulp (C) and dentin (D). Nerve distribution is often 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. **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. **A,** ×75. **B,** ×45. *(From Byers MR: Dynamic plasticity of dental sensory nerve structure and cytochemistry.* Arch Oral Biol *39[suppl]:13S, 1994.)*

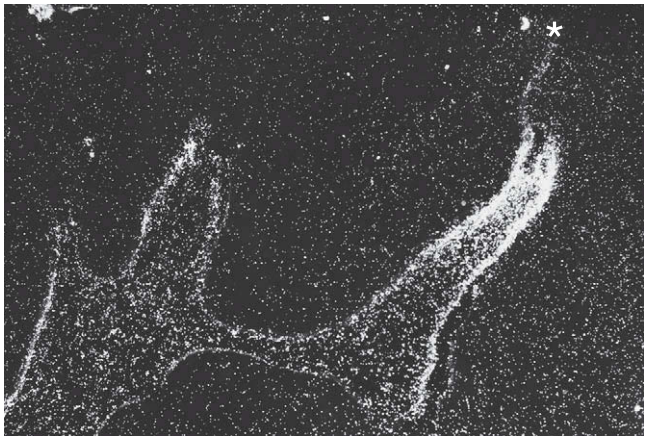


FIG. 12-41 NGF-mRNA is upregulated in the mesial pulp horn 6 hours after cavity preparation. *(From Pimental E: Handbook of growth factors, vol 3, London, Taylor & Francis, 1994).*

nerves and immune cells has been demonstrated.[164,303] In addition, recruitment of immunocompetent cells has been demonstrated in the dental pulp after electrical tooth stimulation.[66,103] Similar responses have been seen in periapical bone in rats with radicular pulpitis.[182] Neurogenic inflammation is generally thought to enhance healing, because denervated teeth show poorer healing following pulp exposures than innervated teeth.[45,53]

Hyperalgesia

Three characteristics of hyperalgesia are (1) spontaneous pain, (2) a decreased pain threshold, and (3) an increased response to a painful stimulus.[129] 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 is often increased 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 inflamed neural tissue.[114,129] NGF seems to play an important role in hyperalgesia. NGF regulates chronic inflammatory hyperalgesia by controlling gene expression in sensory neurons,[257] 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 (see Chapter 19 for additional detail).[141,143,324,339,356] 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.

In addition, certain mediators of inflammation (e.g., bradykinin, 5-hydroxytryptamine [5-HT], prostaglandin E2) are capable of producing hyperalgesia.[253,256] For example, 5-HT and CGRP are able to sensitize intradental fibers to hydrodynamic stimuli such as cold, air blasts, and osmotic stimulation.[256] Unmyelinated fibers are activated by a number of inflammatory mediators. Bradykinin produces a dull, aching pain when placed in a deep cavity in a human tooth.[187]

Leukotriene B_4 (LTB_4) was shown to have a long-lasting sensitizing effect on intradental nerves, suggesting that it may potentiate nociceptor activity during pulpal inflammation.[217]

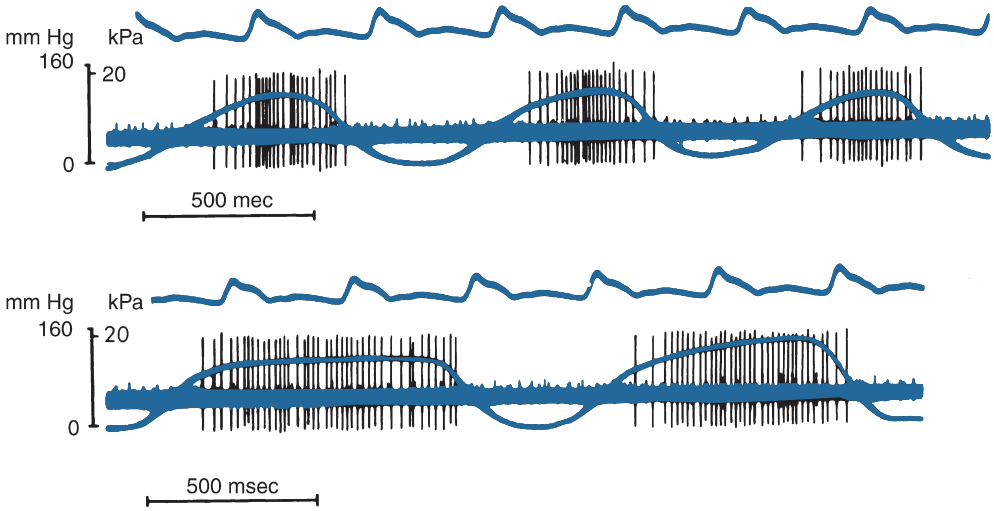


FIG. 12-42 Response of a single dog pulp nerve fiber to repeated hydrostatic pressure stimulation pulses. Lower solid wavy line of each recording indicates the stimulation pressure applied to the pulp. Upper line (kPa) is the femoral artery blood pressure curve recorded to indicate the relative changes 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.)*

Both LTB_4 and the complement component, C5a, stimulate neutrophils to secrete additional pain-producing cytokines.

Many silent nerve fibers are present in the normal pulp[253,254] 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.[48,253,254,256] 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.[14,47,114]

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,[230,252] and painful pulpitis is more likely to be associated with nociceptive C fiber activity indicative of pulpal tissue injury.[252-254,256] The clinician should carefully examine symptomatic teeth to rule out the possibility of hypersensitive dentin, cracked or leaky fillings, or tooth fracture—each of which may initiate hydrodynamic forces—before establishing a diagnosis of reversible or irreversible pulpitis (see also Chapters 1 and 2).

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).[8] On the other hand, the tooth may commence to ache spontaneously in the absence of any external stimulus.[129] Spontaneous, unprovoked pain generally indicates a pulp that is seriously damaged and generally will not respond to noninvasive therapy. For a more complete discussion of pain mechanisms of the dentin-pulp complex, the reader is referred to Chapter 19.

Several studies have elucidated the role of hydrostatic pressure changes in the activation of pulpal nerve fibers.[249] In experiments involving cats and dogs, both positive and negative pressure changes were introduced into the pulp by means of a cannula inserted into the dentin. 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. 12-42 and 12-43).[230,250,362]

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[1,23,181,267,340] (Fig. 12-44).

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[188] (Fig. 12-45). 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[329] (Fig. 12-46). This network provides the odontoblasts with a rich source of metabolites.

Capillary blood flow in the coronal portion of the pulp is nearly twice that in the root portion.[176] Moreover, blood flow in the region of the pulp horns is greater than in all other areas of the pulp.[232] In young teeth, capillaries commonly extend into the odontoblast layer, thus ensuring an adequate supply of nutrients for the metabolically active odontoblasts (Fig. 12-47). In the subodontoblastic capillaries, fenestrations are observed in the vessel wall.[291] These fenestrations are thought to promote rapid transport of fluid and metabolites from the

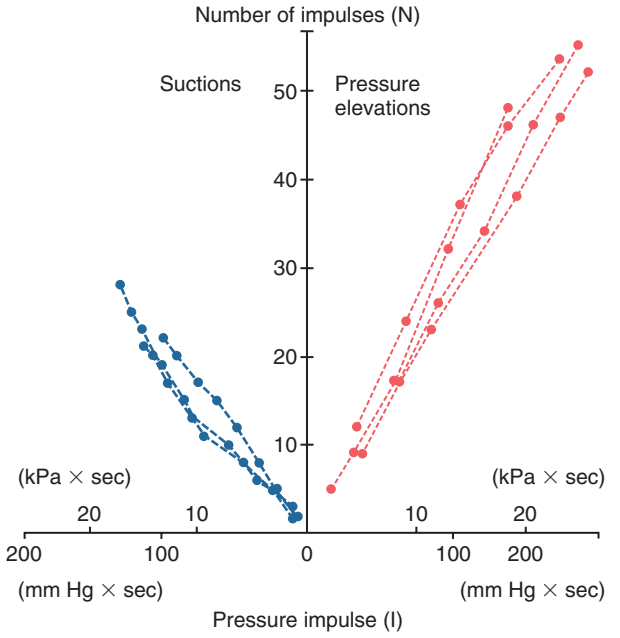


FIG. 12-43 Relationship between the number of nerve impulses (N) and the pressure impulse (I) of a small group of pulpal cat nerve fibers with three suction stimuli and four pressure elevations (pressure impulse is labeled as both mm Hg × sec and kPa × sec). *(Modified from Närhi M: Activation of dental pulp nerves of the cat and the dog with hydrostatic pressure.* Proc Finn Dent Soc *74[suppl 5]:1, 1978.)*

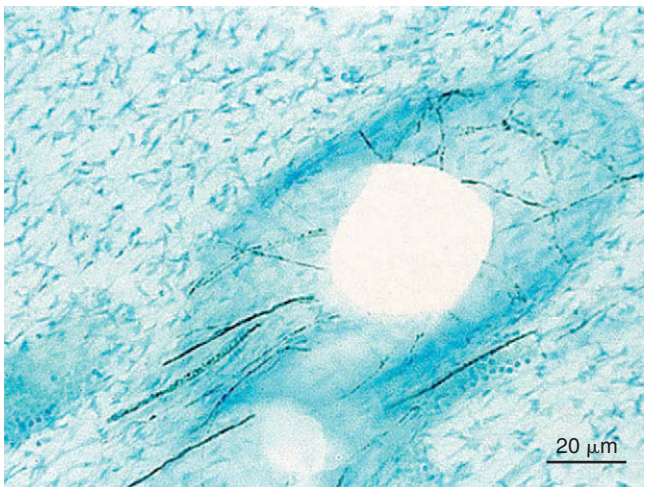


FIG. 12-44 Substance P–positive nerve fibers in the wall of pulpal blood vessels. *(Courtesy Dr. K.J. Heyeraas.)*

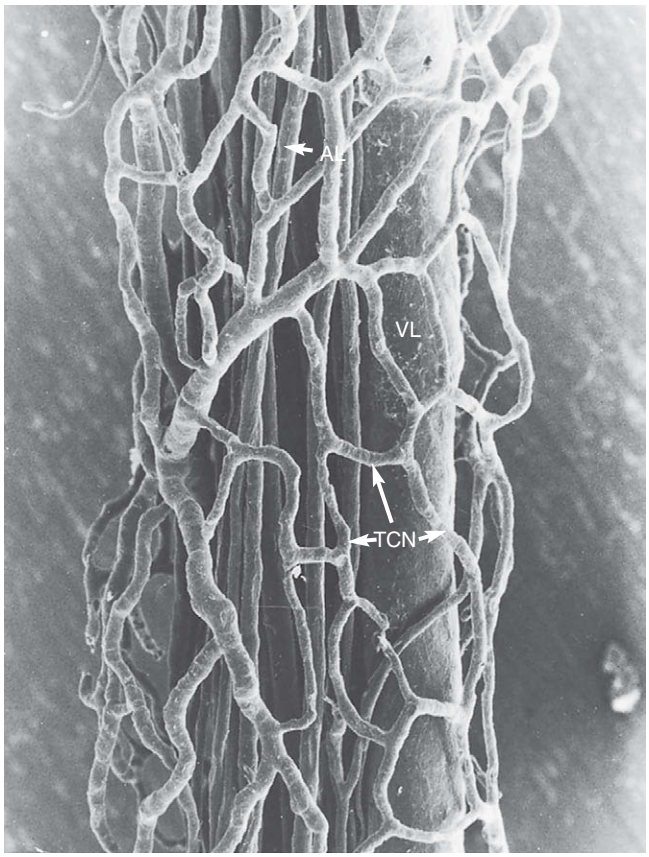


FIG. 12-45 High-power scanning electron micrograph of vascular network in the radicular pulp of a dog molar showing the configuration of the subodontoblastic terminal capillary network (TCN). Venules (VL) and arterioles (AL) are indicated. *(Courtesy Dr. Y. Kishi, Kanagawa Dental College, Kanagawa, Japan.)*

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.[364]

Blood passes from the capillary plexus, first into postcapillary venules (see Fig. 12-46; Fig. 12-48) and then into larger venules.[188] Venules in the pulp have unusually thin walls, and the muscular layer is discontinous,[69] 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,[228,337] and blood volume represents about 3% of pulpal wet weight,[31] approximately the same as in mammary tumour tissue.[377] As would be anticipated, pulpal blood flow is greater in the peripheral layer of the pulp (i.e., the subodontoblastic capillary plexus)[188] where the oxygen consumption has been shown to be higher than in the central pulp.[27]

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.[91,305] Because up to 80% of the Doppler signal originates from periodontal tissue, it is helpful to cover periodontal tissues with a black rubber dam.[132] Laser Doppler flowmetry can be used to detect revascularization of traumatized teeth.[76,86] Although measurement of pulpal blood flow would be an ideal tool for determining pulp vitality, the use of laser Dopplers and other techniques is limited due to sensitivity, specificity, reproducibility, and costs.

Regulation of Pulpal Blood Flow

Under normal physiologic conditions, pulpal vascular tone is controlled by neuronal, paracrine, and endocrine mechanisms

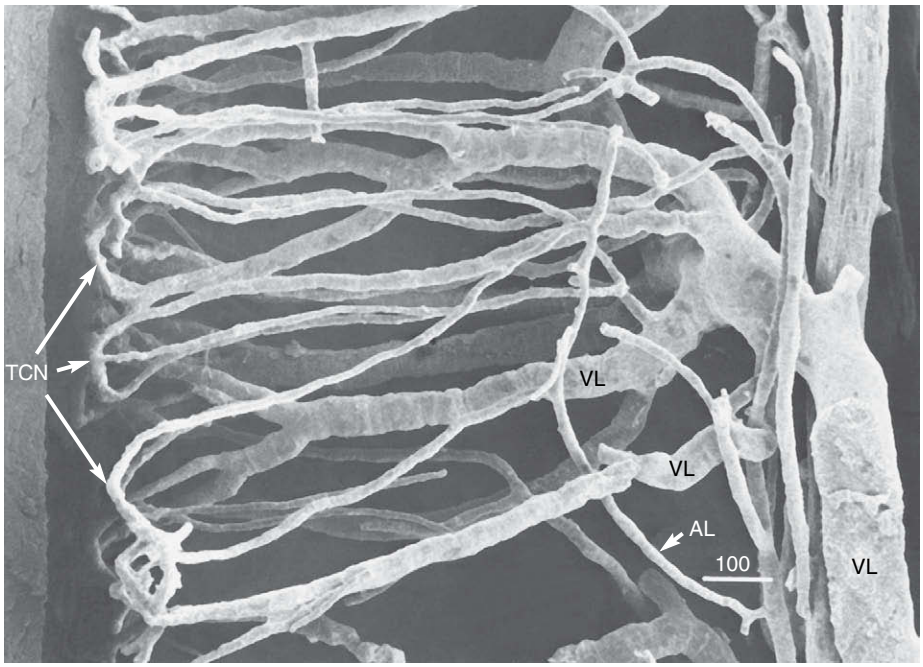


FIG. 12-46 Subodontoblastic terminal capillary network (TCN), arterioles (AL), and venules (VL) of young canine pulp. Dentin would be to the far left and the central pulp to the right. Scale bar: 100 µm. *(From Takahashi K, Kishi Y, Kim S: A scanning electron microscopic study of the blood vessels of dog pulp using corrosion resin casts.* J Endod *8:131–135, 1982.)*

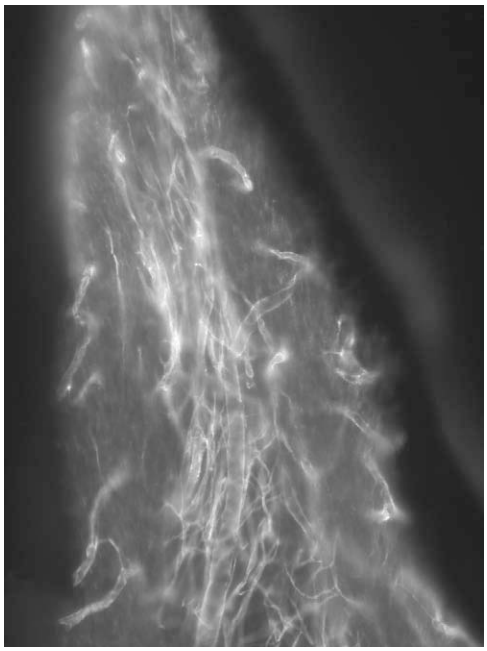


FIG. 12-47 Blood vessels in the pulp horn fan out into the odontoblast layer. *(Courtesy Dr. S.R. Haug.)*

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.[338] 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,[158,340] but a neuronal vasodilator tone caused by release of sensory neuropeptides has been demonstrated (Fig. 12-49).[21,23]

There are α-adrenergic receptors in the pulp,[153] and stimulation of the cervical sympathetic trunk causes vasoconstriction and fall in pulpal blood flow that can be partially reversed by α-receptor blockade.[181,340] NPY, colocalized with norepinephrine in pulpal sympathetic nerve fibers, contributes also to vasoconstriction in the pulp.[79,178]

Increase in pulpal blood flow is observed after electrical tooth stimulation and is caused by release of sensory neuropeptides followed by vasodilatation.[23,144,169] CGRP released from sensory nerve fibers is mainly responsible for the observed vasodilatation.[21,23]

There is evidence for sympathetic modulation of sensory neuropeptide release in the dental pulp[130]; presynaptic adrenoceptors are found on the sensory nerve terminals and attenuate the release of vasodilators from the sensory nerves.[36,171]

Muscarinic receptors have been identified in the pulp,[34] and the parasympathetic neurotransmitter acetylcholine (ACh) causes vasodilatation and increases blood flow in the tissue.[385] 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[102,352] and has been demonstrated to cause vasodilatation and increase in pulpal blood flow in cats.[267]

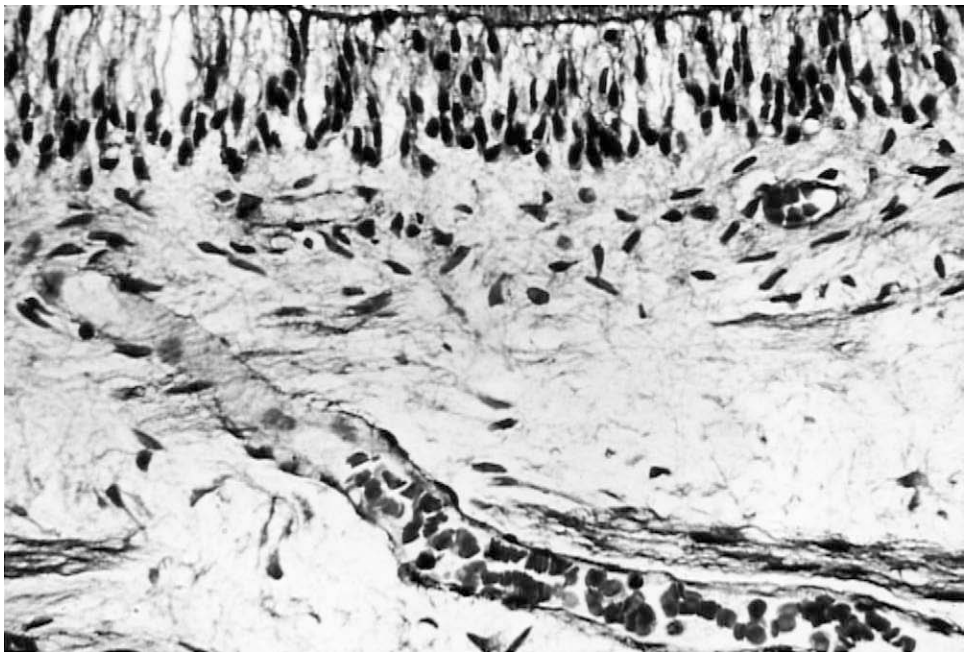


FIG. 12-48 Postcapillary venule draining blood from subodontoblastic capillary plexus.

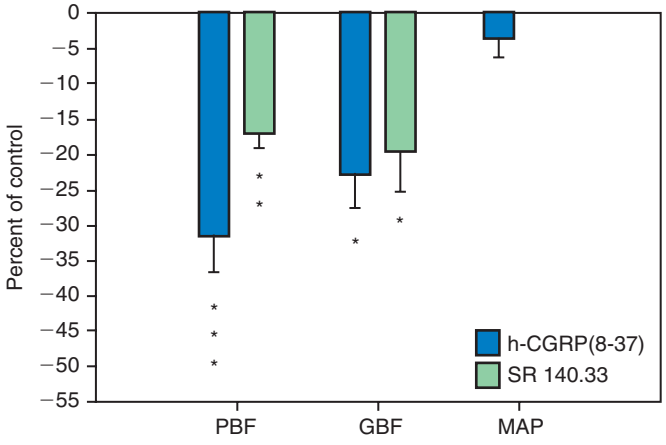


FIG. 12-49 Effect of antagonist infusion of h-CGRP(8-37) (calcitonin gene–related peptide inhibitor) and SR 140.33 (substance P inhibitor) on basal pulpal blood flow (PBF) and gingival blood flow (GBF). *(From Berggreen E, Heyeraas KJ: Effect of the sensory neuropeptide antagonists h-CGRP[8-37] and SR 140.33 on pulpal and gingival blood flow in ferrets.* Arch Oral Biol *45:537–542, 2000.)*

On the other hand, Sasano and co-workers[307] 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,[58] and close intraarterial infusions of endothelin-1 reduce pulpal blood flow.[22,112,385] 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.[23,208] 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.[385]

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, were recently identified in the rat pulp.[322]

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.[385]

Lymphatics

The lymphatic vasculature forms a vessel network in the interstitium which drains filtered fluid and proteins and returns it to the blood through the larger lymphatics.[147,301] In addition, it serves an important role in the body's immune defense, since dendritic cells enter the blind-ended lymphatic capillaries in the dental pulp and transport captured antigen to regional

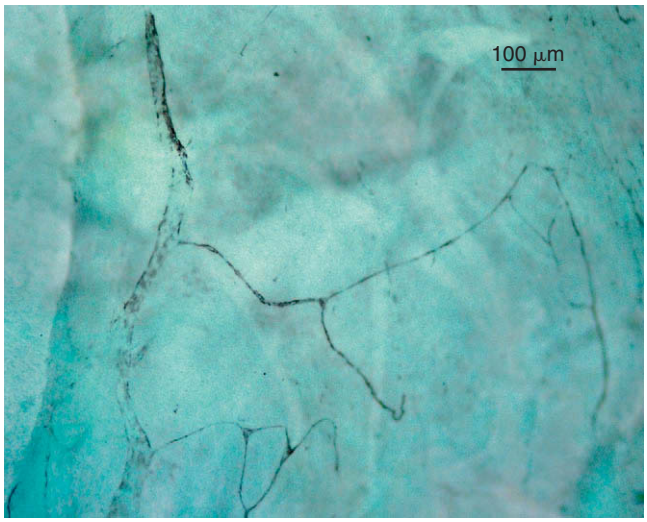


FIG. 12-50 Lymphatic capillaries joining into larger lymphatic vessels in a rodent incisor. The lymphatic endothelium is visualized by whole mount immunohistochemistry utilizing an antibody against vascular endothelial growth factor receptor 3. *(From Berggreen E, Haug SR, Mkonyi LE, Bletsa A: Characterization of the dental lymphatic system and identification of cells immunopositive to specific lymphatic markers.* Eur J Oral Sci *117(1):34–42, 2009.)*

lymph nodes where they present the antigen to lymphocytes. The lymphatic fluid removes foreign invaders such as bacteria and their byproducts from the peripheral tissue and represents a transport system for infectious agents. During inflammation, new lymphatic vessels are formed to meet the demand of increased fluid transport and antigen presentation.[5]

Historically, 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.

Recently, specific lymphatic markers have been applied (Fig. 12-50), and an extensive system of lymphatic vessels in the pulp is now recognized to exist (Fig. 12-51).[20,283] These new data indicate that true lymphatics travel from the coronal pulp throughout the roots.[28,262] Lymphatic capillaries are found in the pulp horn but not in the odontoblast layer.[20] Moreover, bundles of lymphatics leave the pulp via the apical foramen and through lateral canals in the root. In the rodent incisor pulp, lymphatic capillaries are found only in the apical third. In the middle and coronal third of the periodontal ligament, lymphatic vessels seem to drain mainly into the alveolar bone, whereas in the apical third, vessels drain toward the apex, where they join into bundles of lymphatic vessels.

Lymphatic capillaries are filled when pulpal interstitial pressure increases after bulk swelling of the interstitium and by strain on the extracellular matrix. When lymph is formed, deformation of interstitial tissue leads to propulsion of lymph by compression of the lymphatics,[308] and inside the rigid pulp chamber, the only force that can cause tissue deformation and strain on the extracellular matrix is the arteriolar pulse pressure converted to a relatively high interstitial pulsatile pressure. It was demonstrated more than 70 years ago in the rabbit ear that the removal rate of an intravital stain injected subcutaneously was higher with than without arterial pressure pulsations in the tissue.[273]



FIG. 12-51 The lymphatic system in a mouse molar immunoreacted with antibody to vascular endothelial growth factor receptor 3. Note that the lymphatic vessels travel from the coronal pulp through the root toward apex. *(From Berggreen E, Haug SR, Mkonyi LE, Bletsa A: Characterization of the dental lymphatic system and identification of cells immunopositive to specific lymphatic markers.* Eur J Oral Sci *117(1):34–42, 2009.)*

As lymphatics from the pulp drain through the periodontal ligament, it is logical to assume that normal tooth movement during chewing will act as an additional extrinsic propulsion mechanism for the lymph.

Transcapillary Fluid Exchange

As in all other tissues in the body, the fluid transport between the pulpal blood vessels and the interstitial space is regulated by differences in colloid osmotic and hydrostatic pressures in the plasma and interstitium in addition to lymph flow (Fig. 12-52).

During normal conditions, a steady state is achieved as the fluid filtered into the interstitial space equals the amount of

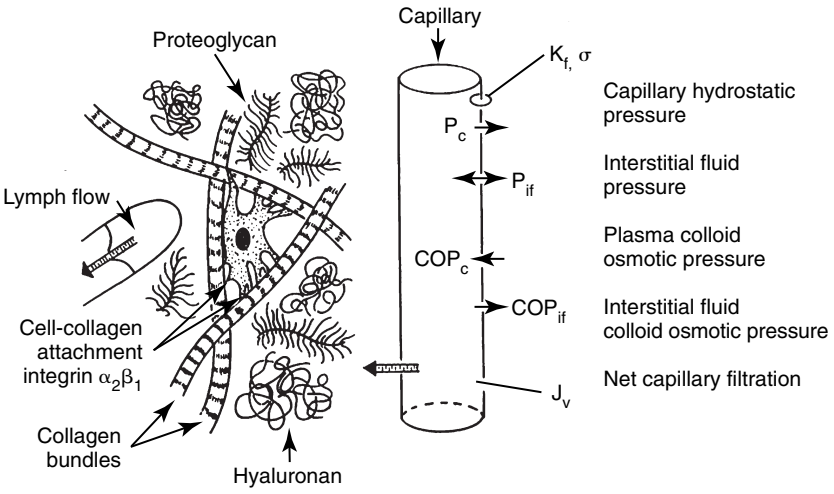


FIG. 12-52 Interstitial structure and pressures that govern transcapillary fluid transport. K_f, Capillary filtration coefficient; σ, capillary reflection coefficient for plasma proteins. *(From Wiig H, Rubin K, Reed RK: New and active role of the interstitium in control of interstitial fluid pressure: potential therapeutic consequences.* Acta Anaesthesiol Scand *47:111–121, 2003.)*

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,[31] 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 in the range from 6 to 10 mm Hg,[23,140] but higher values measured with different methods have also been reported.[41,357,361]

Colloid osmotic pressure (COP) measurements in interstitial fluid isolated from rat incisors has shown a relatively high pulpal COP, reaching 83% of plasma COP.[31] The high value may imply that the normal permeability of pulpal vessels to plasma proteins is relatively high and/or the drainage of plasma proteins is ineffective. The fact that lymphatic vessels are only found in the apical part of the rodent incisor may support the latter possibility. Filtered plasma proteins must be removed from the interstitial fluid to maintain homeostasis, and the only possibility for transport is by lymphatics, since proteins cannot be reabsorbed into blood vessels.

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 = Δ V/ Δ 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[141,143,340,356] and may tend to compress blood vessels and counteract a beneficial blood flow increase (Fig. 12-53).

Classical studies have demonstrated that an increase in intrapulpal tissue pressure promoted absorption of tissue fluid back into the blood and lymphatic vessels, thereby reducing the pressure.[141,143] This observation can explain why pulpal tissue pressure in inflamed pulps may persist in local regions for long observation periods,[339] contradicting the old concept of a wide, generalized collapse of pulpal venules and cessation of blood flow (pulpal strangulation theory).

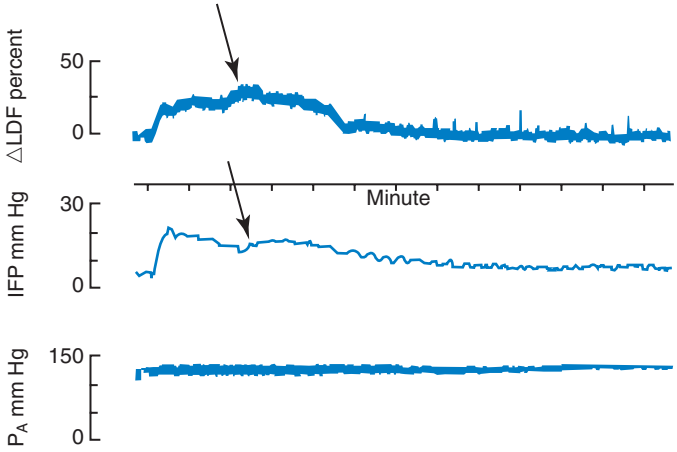


FIG. 12-53 Original simultaneous recordings of percent change in pulpal blood flow (ΔLDF%), interstitial fluid pressure (IFP), and systemic blood pressure (P_A mm Hg) in cat during electrical tooth stimulation. Note that when IFP is first decreasing after an initial rise, the pulpal blood flow reaches its maximal level *(arrows)*, demonstrating compression of vessels in the first phase. *(Courtesy Dr. K.J. Heyeraas.)*

The delivery of dental restorative procedures may lead to substantial increases or decreases in pulpal blood flow, depending upon the precise procedure and time point sampled.[180] 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.[179,266] In contrast, serotonin (5-HT) is released primarily from the platelets, and given intraarterially, it has been shown to reduce pulpal blood flow.[177,385]

Acute inflammation in the dental pulp induce an immediate rise in blood flow and can reach a magnitude of up to nearly 200% of control flow followed by increased vascular permeability.[142,143]

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.[31]

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[265] 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.[25,255] The activated endothelium also participates in procoagulant reactions that promote fibrin clot formation.[325] 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.[12] 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.

The role of lymphatic vessels in dental inflammation is unknown, but Pimenta et al., found an increased number of lymphatic vessels in inflamed pulps from teeth with caries compared to noninflamed pulps, indicating that lymphangiogenesis takes place in the pulp.[283]

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.[170,179,220]

LPS and lipoteichoic acid (LTA) from gram-negative and gram-positive bacteria, respectively, cause upregulation of VEGF in activated pulpal cells.[35,331] VEGF increases vascular permeability,[88,312] and it is likely that it also causes leakage in pulpal vessels. It is a very potent agent, since its ability to enhance microvascular permeability is estimated to be 50,000 times higher than that of histamine.[316] Cytokines such as IL-1 and TNF-α are released into the pulp interstitial fluid during inflammation[31] 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 barrier between plasma and interstitium can be eleminated.[31]

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 and thereby increases 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,[141,143,324,339,356] which is then sufficient to activate sensitized pulpal nociceptors and initiate spontaneous pulpal pain. Thus the "throbbing" sensation of a toothache is due to the pulsation in the pulp that follows heart contractions (systole), 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*.[320] Generally, the rate of formation of dentin is increased, but the tubules remain continuous with the secondary dentin.[323] 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 such 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.[90,294,320] 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, 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.[319] 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.[299,300,321] 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,[299] it was unsuccessful in inflamed pulps.[298] 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.[116] 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 such dentin impermeable and protecting the pulp from the inward diffusion of noxious substances that might leak around restorations.[277] 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.[259]

It will be recalled that secondary dentin is deposited circumpulpally at a very slow rate throughout the life of the vital tooth.[323] 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. 12-54). 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.[345]

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. 12-55). The quality of tertiary dentin (i.e., the extent to which it resembles primary or secondary dentin) is quite

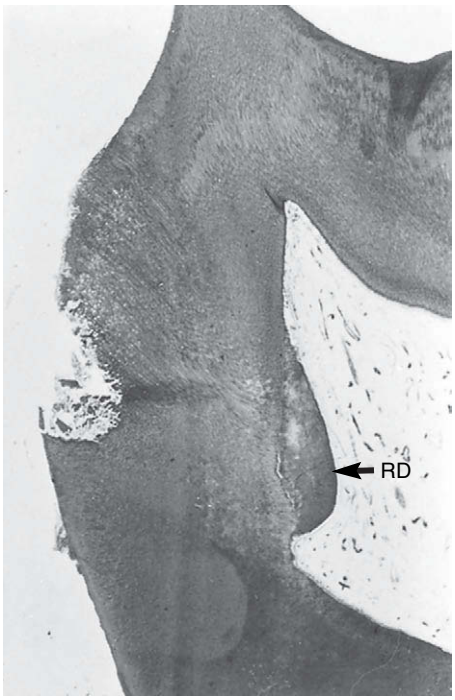


FIG. 12-54 Reparative dentin (RD) deposited in response to a carious lesion in the dentin. *(From Trowbridge HO: Pathogenesis of pulpitis resulting from dental caries.* J Endod *7:52–60, 1981.)*

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.[64,345] 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. 12-56). As the entrapped soft tissue degenerates, products of tissue degeneration are released that further contribute to the inflammatory stimuli assailing the pulp.[345]

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[242] and human teeth.[240] 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.[96]

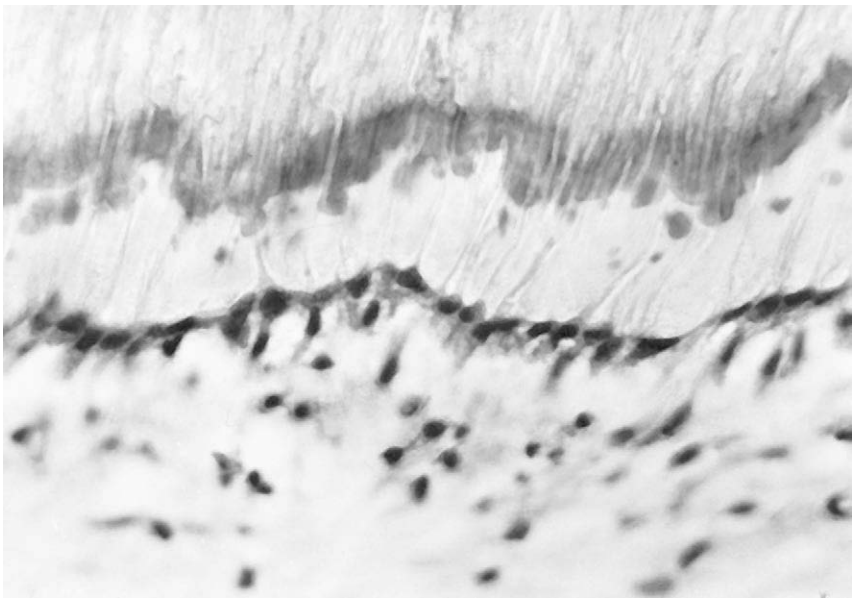


FIG. 12-55 Layer of cells forming reparative dentin. Note the decreased tubularity of reparative dentin compared to the developmental dentin above it.

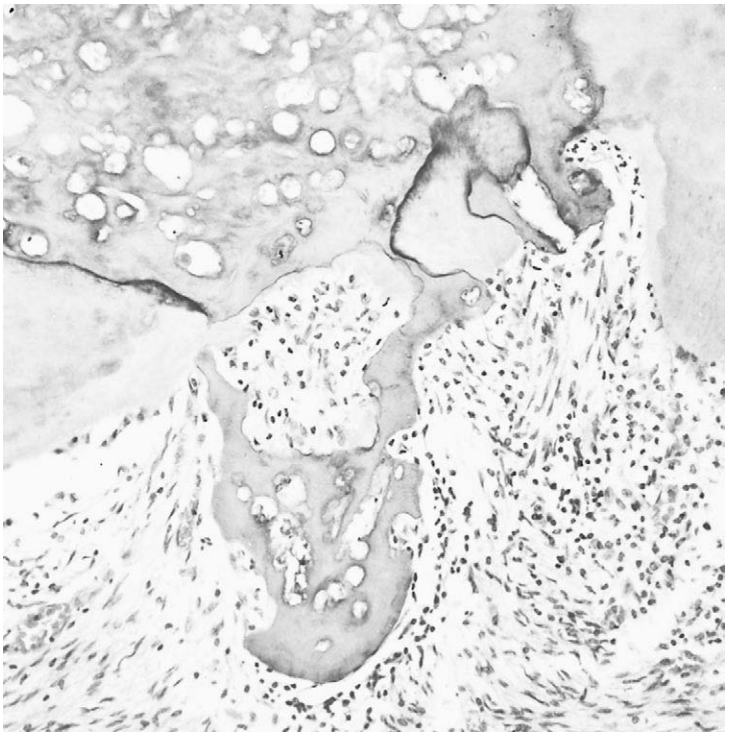


FIG. 12-56 Swiss-cheese type of reparative dentin. Note the numerous areas of soft-tissue inclusion and infiltration of inflammatory cells in the pulp.

Investigators[383] 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. 12-57). The similarity of primary odontoblasts to replacement odontoblasts was established by D'Souza et al.[75] 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,[77,192] from bacterial products such as endotoxins shed from deep carious lesions,[19,370] or from mechanical exposure of pulps.[241] 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

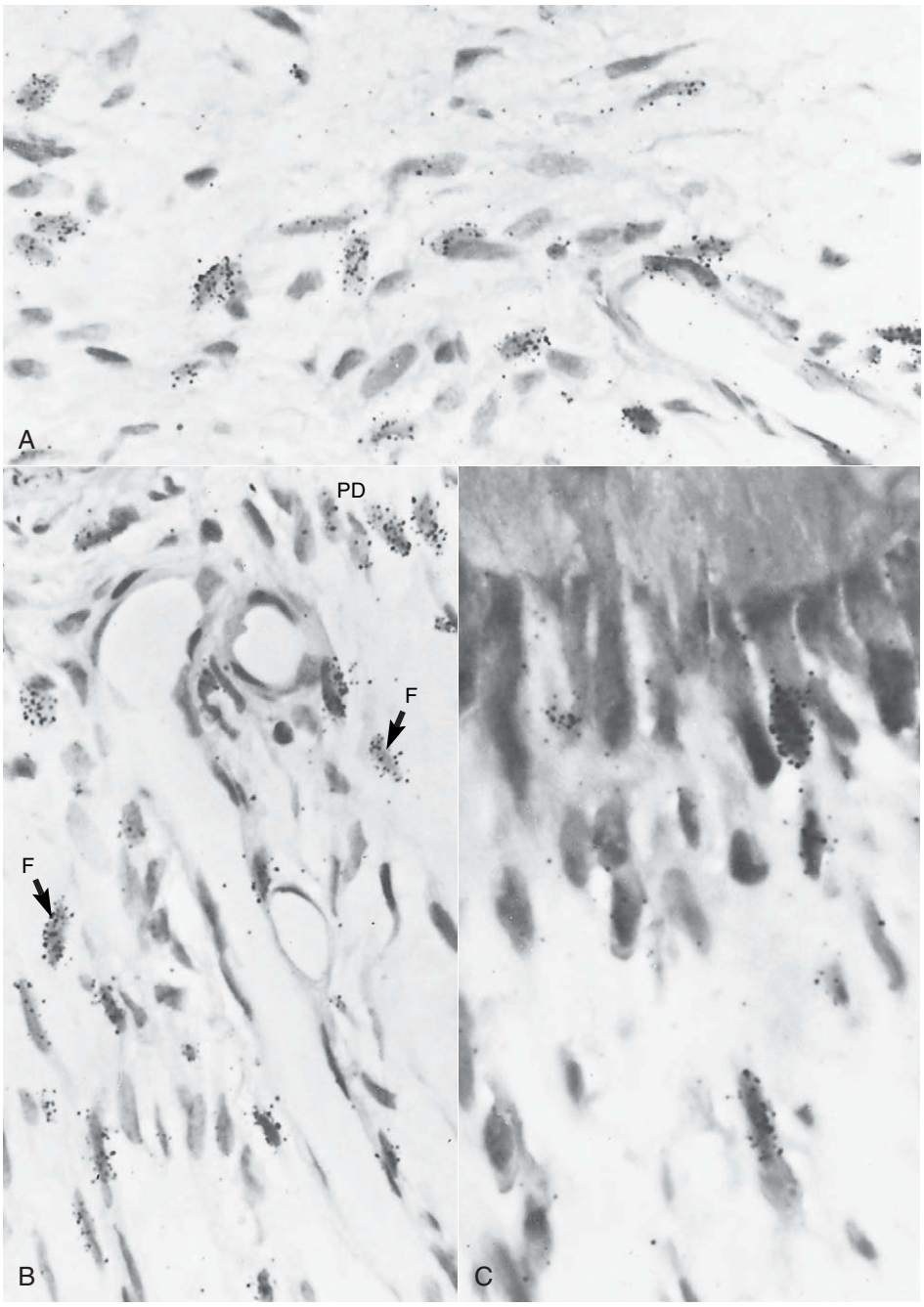


FIG. 12-57 Autoradiographs from dog molars illustrating uptake of ^{3}H-thymidine by pulp cells preparing to undergo cell division after pulpotomy and pulp capping with calcium hydroxide. **A,** Two days after pulp capping. Fibroblasts, endothelial cells, and pericytes beneath the exposure site are labeled. **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.)*

immune response within the pulp tissue that would also modulate the repair process.[118]

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.[62] When small mechanical pulp exposures are inadvertently made in healthy teeth, the current recommendation is 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.[165] Like calcium hydroxide, mineral trioxide aggregate (MTA) has also been recognized to promote hard-tissue formation.[3,7,245]

The formation of atubular "fibrodentin" is another potential product of newly differentiated odontoblasts, provided that

a capillary plexus develops beneath the fibrodentin.[17] This is consistent with the observation made by other researchers[64,96] 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[323] 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. 12-58).[92] 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.[310] 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.[303]

One group[335] 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 were lacking such a deposit. It would thus appear that reparative dentin can protect the pulp,[19] 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. 12-55). 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.

Periodontally diseased teeth have smaller root canal diameters than teeth that are periodontally healthy.[193] The root canals of such teeth are narrowed by the deposition of large quantities of reactionary dentin along the dentinal walls.[311] 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.



FIG. 12-58 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, Ltd.)*

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.[134] However, given that normal rat root dentin is only 100 µm thick, such 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. 12-59). However, fibrosis and reparative dentin formation often go hand in hand, indicating that both are expressions of a reparative potential.

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 pulp-like tissues with tubular-like dentin,[85] 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.[288]

A case report indicates that it might be possible to revascularize the pulp in infected necrotic immature roots (Fig. 12-60; see also Chapter 16).[16] A young patient presented with an immature second lower right premolar with radiographic and clinical signs of apical periodontitis with the presence of a sinus tract. The canal was disinfected without mechanical

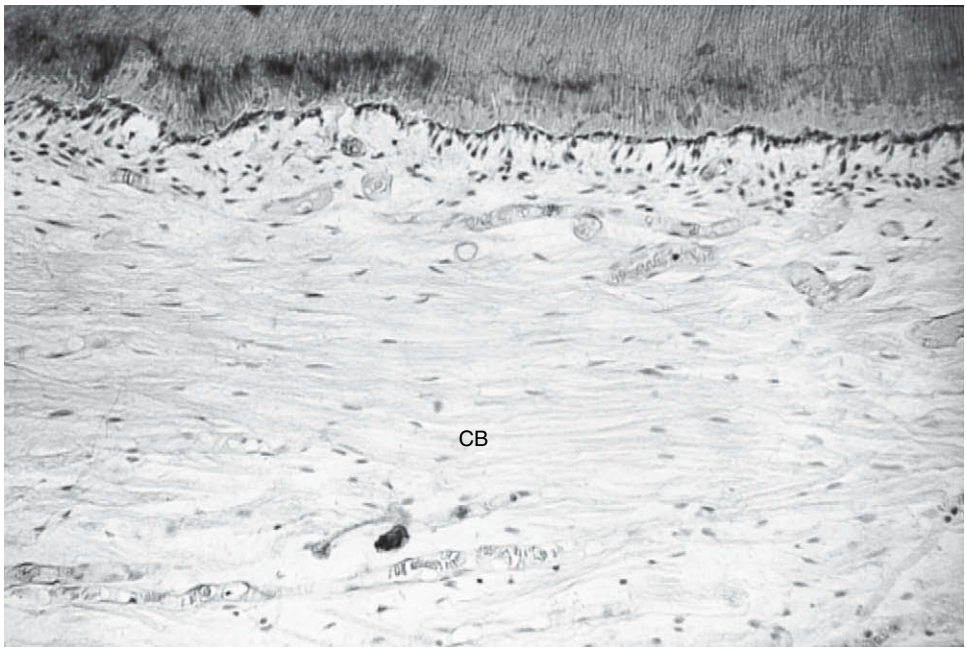


FIG. 12-59 Fibrosis of dental pulp showing replacement of pulp tissue by large collagen bundles (CB).

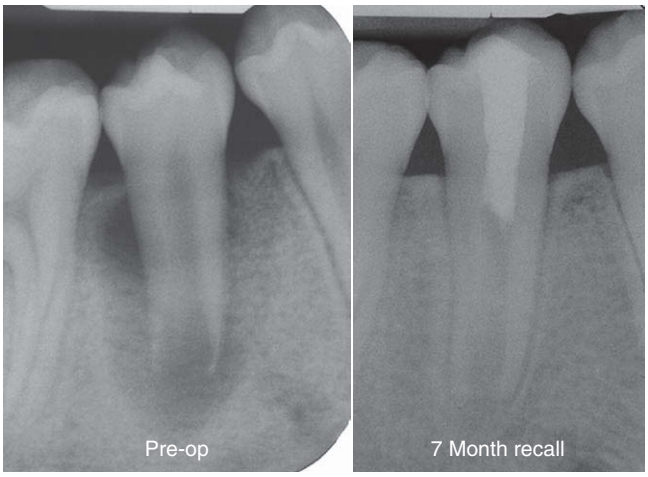


FIG. 12-60 Immature tooth with a necrotic infected canal with apical periodontitis. The canal is disinfected with copious irrigation with sodium hypochlorite and an antibiotic paste. Seven months after treatment, the patient is asymptomatic, and the apex shows healing of the apical periodontitis and some closure of the apex. *(From Banchs F, Trope M: Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol?* J Endod *30:196–200, 2004.)*

instrumentation, with copious irrigation and the use of a mixture of antibiotic agents. Later, a blood clot is created in the canal space and the access is filled with an MTA base. The treatment allows revascularization of the immature tooth and regain of a vital state of the pulp chamber, as well as normal root development below the restoration similar to the adjacent and contralateral teeth.

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 very 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. 12-61), whereas in the radicular pulp, calcification tends to be diffuse (Fig. 12-62).[349] There is no clear evidence 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. 12-63). The mineral phase of pulp calcifications has been shown to consist of typical carbonated hydroxyapatite.[349] Histologically, two types of stones are recognized: (1) those that are round or ovoid, with smooth surfaces and concentric laminations (see Fig. 12-61) and (2) those that assume no particular shape, lack laminations, and have rough surfaces (Fig. 12-64). 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. 12-65). 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.

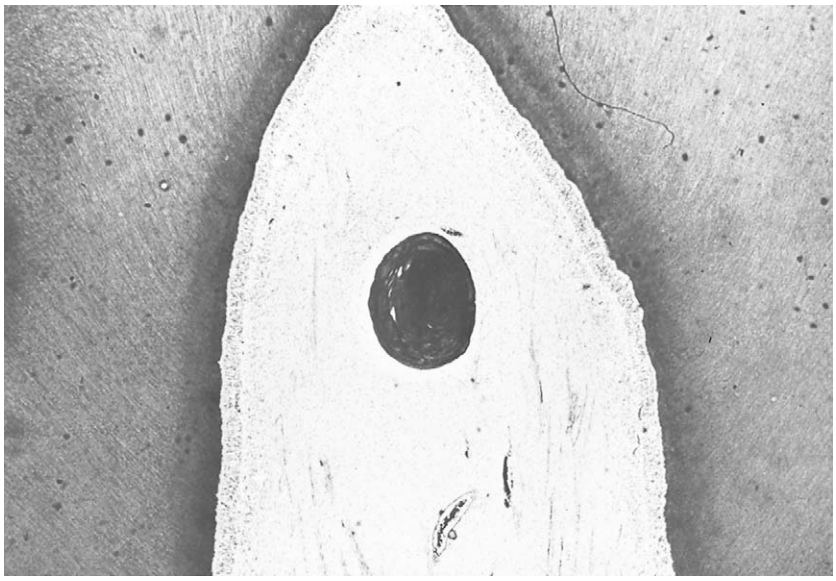


FIG. 12-61 Pulp stone with a smooth surface and concentric laminations in the pulp of a newly erupted premolar extracted in the course of orthodontic treatment.



FIG. 12-62 Diffuse calcification near the apical foramen.

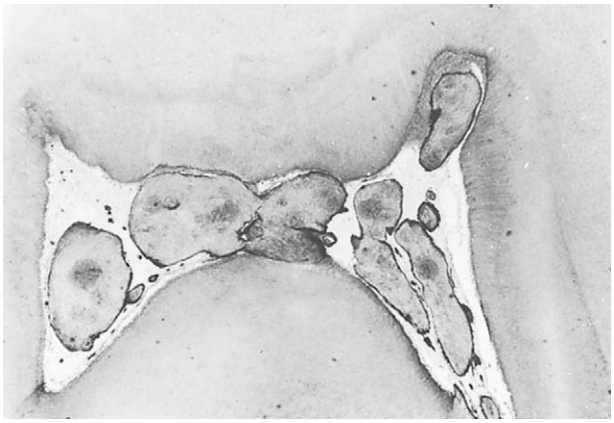


FIG. 12-63 Pulp stones occupying much of the pulp chamber.

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.[259] 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).[259]

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 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

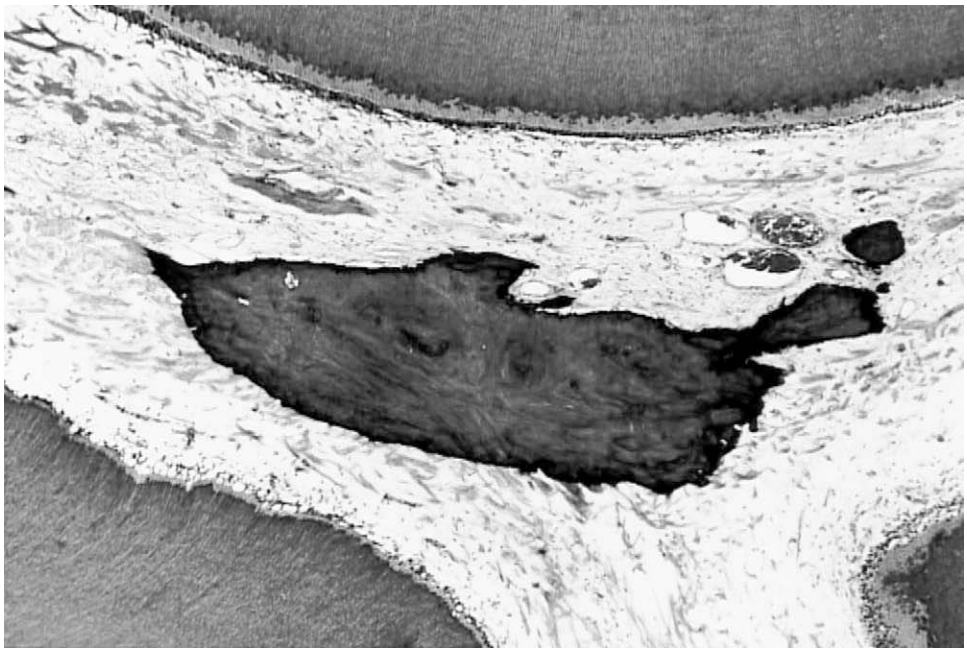


FIG. 12-64 Rough surface form of pulp stone. Note hyalinization of collagen fibers.



FIG. 12-65 High-power view of a pulp stone from Fig. 12-57, showing the relationship of mineralization fronts to collagen fibers.

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 very 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. 12-66). Histologic examination invariably reveals the presence of some soft tissue, and cells

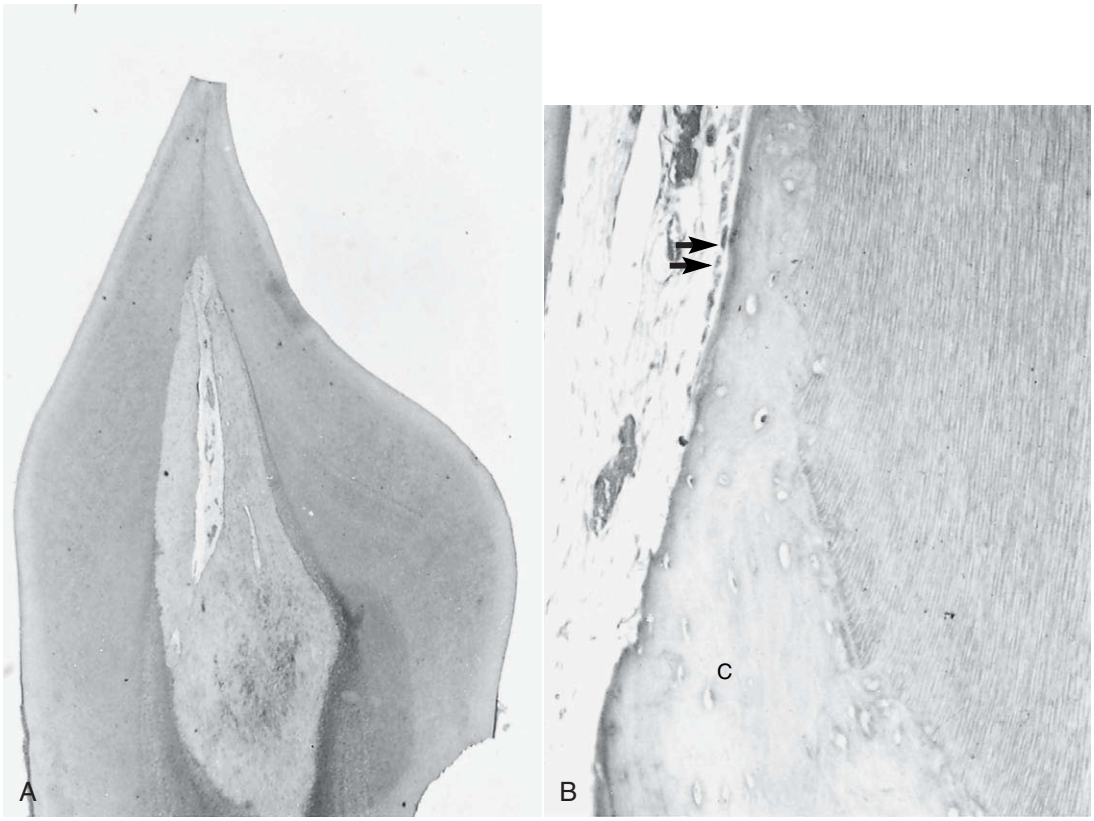


FIG. 12-66 A, Calcific metamorphosis of pulp tissue after luxation of tooth as a result of trauma. Note presence of soft-tissue inclusion. **B,** High-power view showing cementoblasts *(arrows)* lining cementum (C), which has been deposited on the dentin walls.

resembling cementoblasts can be observed lining the mineralized tissue. Such calcific metamorphosis of the pulp has also been reported in replanted teeth of the rat.[258]

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[9] found only 7% of the pulps that underwent calcific metamorphosis exhibited secondary infection. Since the success rate for nonsurgical endodontic therapy, not only in general[373] but also for obliterated teeth,[68] 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.[105,323] In addition, certain regressive changes in the pulp appear to be related to the aging process. 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. 12-64). 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[99] and blood vessels.[24,26] Evidence also suggests that aging results in an increase in the resistance of pulp tissue to the action of proteolytic enzymes,[387] hyaluronidase, and sialidase,[26] 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.*[323] Dentinal sclerosis produces a gradual decrease in dentinal permeability as the dentinal tubules become progressively reduced in diameter.[328]

*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

ASHRAF F. FOUAD and LINDA LEVIN

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
- Degree of Pretreatment Pulp Inflammation
- Degree of Physical Irritation Caused by Procedure
- Proximity of Restorative Procedures to Dental Pulp and Surface Area of Dentin Exposed
- Permeability of Dentin and Odontoblastic Layer Between Area Being Restored and Pulp
- Patient Age

PULPAL REACTIONS TO RESTORATIVE MATERIALS
DIRECT PULP CAPPING WITH MINERAL TRIOXIDE AGGREGATE
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

PULPAL REACTIONS TO CAVITY PREPARATION USING AIR ABRASION TECHNIQUES
PULPAL REACTIONS TO VITAL BLEACHING TECHNIQUES
PULPAL REACTIONS TO PERIODONTAL PROCEDURES
MECHANICAL IRRITANTS: ORTHODONTIC MOVEMENT
PULPAL REACTIONS TO ORTHODONTIC SURGERY
BIOMECHANICAL IRRITATION: PARAFUNCTIONAL HABITS
PULPAL REACTIONS TO IMPLANT PLACEMENT AND FUNCTION
REGENERATIVE POTENTIAL OF THE DENTAL PULP

The dental pulp is a very dynamic tissue that responds to external stimuli in a variety of different 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 very 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 promotes the effects of inflammation and may lead to rapid degeneration and necrosis, a condition considered very serious in any tissue in the body. The treatment of dental caries and other tooth abnormalities involves the cleaning and shaping of the enamel and dentin, the hardest tissues in the body, thus adding to the irritation of the pulp. In this chapter, the response of the pulp to all these variables will be discussed, and recent advances in our understanding of dental procedures and their effects on the pulp will be presented.

PULPAL REACTION TO CARIES

Dental caries is a localized, destructive, and progressive infection of dentin; if left unchecked, caries can result in pulpal necrosis and potential tooth loss. Both bacterial byproducts 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. These responses occur concomitantly, and their robustness is highly dependent on the aggressive nature of the advancing lesion.

In the advancing infection front of the carious lesion, multiple intrinsic and extrinsic factors are released that are stimulatory to subjacent pulpal parenchyma. Bacterial metabolites such as acids have been thought to be initiators of pulpal reactions, yet the buffering capacity of dentin fluid likely attenuates the pH before it can directly effect a deleterious response, except when the remaining dentin thickness is minimal.[214]

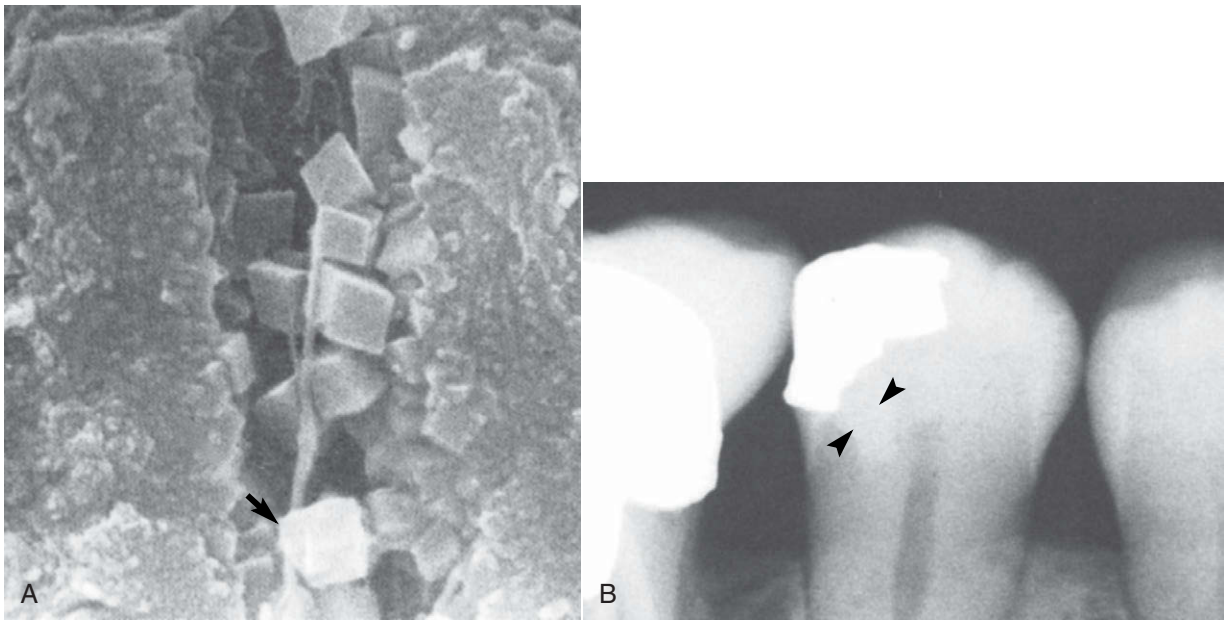


FIG. 13-1 A, Whitlockite crystals occlude the dentinal tubules in sclerotic dentin. **B,** Dentinal sclerosis is radiographically apparent beneath a deep class II lesion. (***A*** *from Yoshiyama M, Masada J, Uchida A, Ishida H: Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin.* J Dent Res *68:1498–1502, 1989.)*

When relatively unhindered access to pulpal tissue is present, both bacterial metabolites and cell wall components induce inflammation. In initial to moderate lesions, current evidence suggests that acidic byproducts of the carious process act indirectly by degrading the dentin matrix, thereby liberating bioactive molecules previously sequestered during dentinogenesis. Once liberated, these molecules once again assume their role in dentin formation, this time stimulatory for *tertiary* dentinogenesis.[216] This theory is supported by the findings that demineralized dentin matrix implanted at the site of pulpal exposure can induce dentinogenesis.[243] 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.[217,242]

Recent evidence offers several candidate molecules that are stimulatory for reparative dentinogenesis. Heparin-binding growth factor, transforming growth factor (TGF)-β1, TGF-β3, insulin-like growth factors (IGF)-1 and -2, platelet-derived growth factor, and angiogenic growth factors have been shown to be stimulatory for dentinogenesis in vitro. The TGF-β superfamily in particular seems to be important in the signaling process for odontoblast differentiation 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.[39] 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.[214]

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 that occurs in a relatively short period of time. Classic studies have noted that while sclerosis is observed in the dentin of disease-free and attrition-free teeth, there is a 95% increase in the incidence in carious teeth.[222] In vitro studies with cultured tooth slices implicate TGF-β1 as a central player in the increased deposition of intratubular dentin.[213] 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[140,239] (Fig. 13-1).

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.[135] 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 the aggressive lesion, the carious process may prove cytocidal to subjacent odontoblasts and require repopulation of the disrupted odontoblast layer with differentiating progenitors. The appearance of the resultant matrix is 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, owing to its irregular con-



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.



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.

figuration and tissue inclusions, is more permeable than physiologic dentin[252] (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.[107] 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.[33]

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).[109] These receptors recognize pathogen-associated molecular patterns (PAMPs) on invading organisms and initiate a host defense through the activation of the nuclear factor (NF)-κB pathway.[91] One class of the PAMP recognition molecules is the toll-like receptor family (TLRs). Odontoblasts have been shown to have increased expression of certain TLRs in response to bacterial products. Under experimental conditions, odontoblast expression of TLR3, 5, and 9

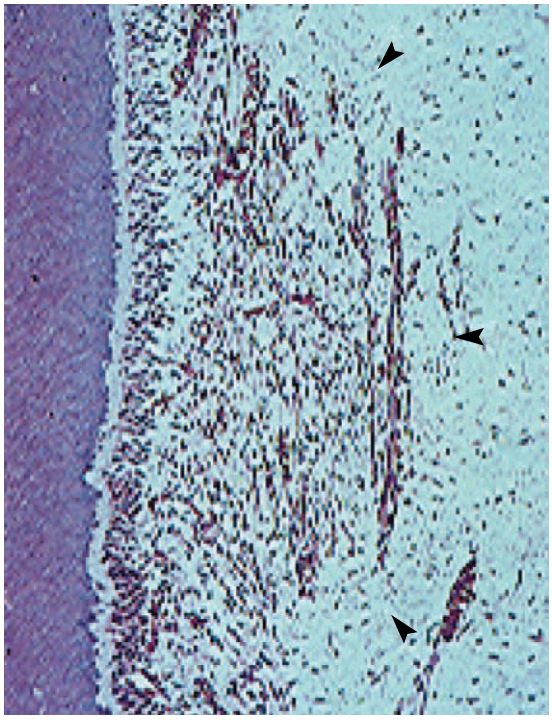


FIG. 13-4 The early pulpal response to caries is represented by a focal accumulation of chronic inflammatory cells. Note that peripheral to the inflammation, the pulpal parenchyma is relatively unaffected.

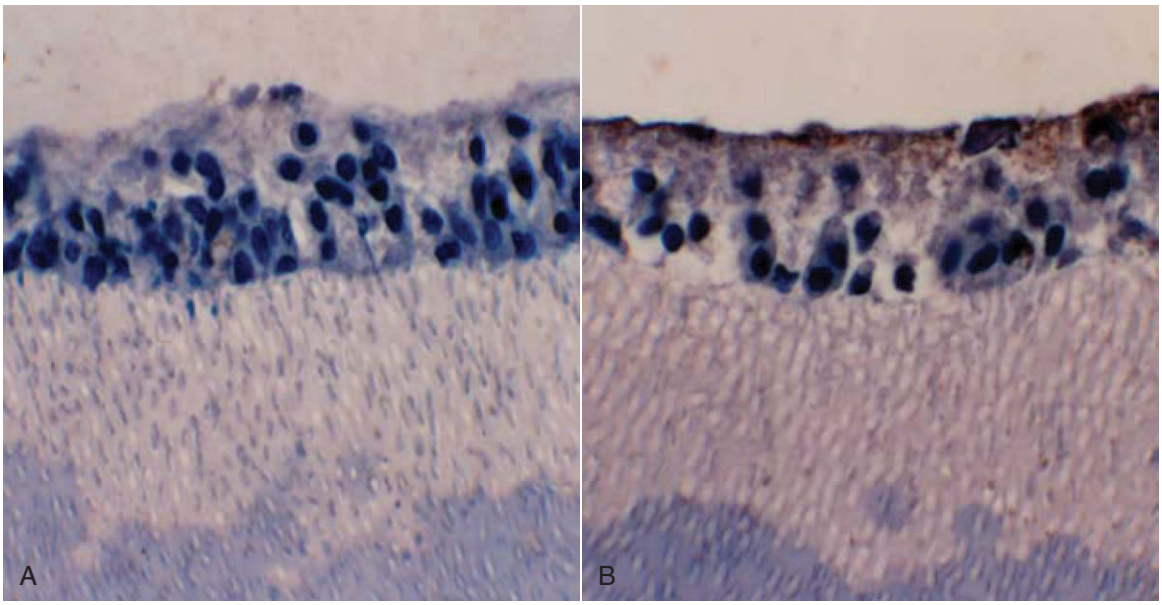


FIG. 13-5 Odontoblasts exposed to lipopolysaccharide (LPS) in an in vitro culture model express interleukin 8 (IL-8), evidenced by immunostaining with anti-IL-8 antibodies.

was increased in response to lipoteichoic acid, whereas lipopolysaccharide increased TLR2 expression.[59,166] 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.[67]

Many cells constitutively produce chemokines at low levels. Unstimulated odontoblasts express genes coding for CCl2, CXCL12, and CXCL14, three genes known to code for factors chemotactic for immature dendritic cells.[40] They also produce CCL26, a natural antagonist for CCR1, CCR2, and CCR5, which are chemokines normally produced by monocytes and dendritic cells.[264] Stimulation with bacterial cell wall constituents has been shown to upregulate the production of several chemokines, suggesting that odontoblasts sense pathogens and express factors that recruit immune effector cells[40,136] (Fig. 13-5). These data suggest a scenario whereby stimulated odontoblasts express high levels of chemokines such as interleukin (IL)-8 (CXCL8) that act in concert with the release of formerly sequestered TGF-β1 from carious dentin, the result of which is a focal increase in dendritic cell numbers, with additional release of chemotactic mediators.[68] 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 coaggregation of nerve fibers and HLA-DR-positive dendritic cells.[257,258]

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[259] (Fig. 13-6). There are

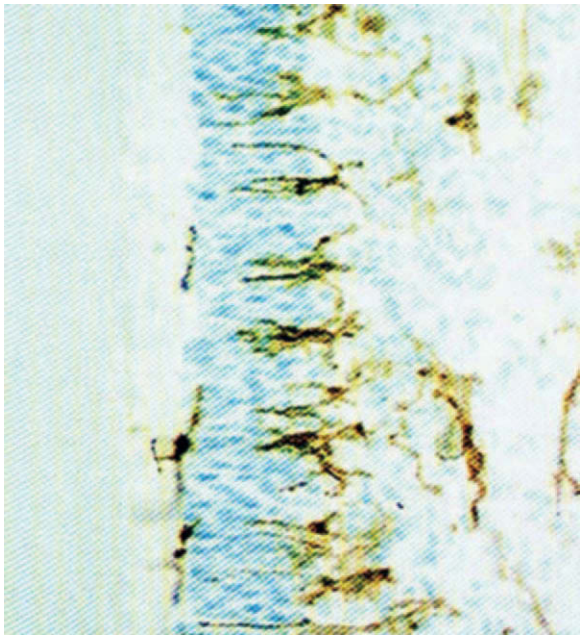


FIG. 13-6 Dental caries stimulates the accumulation of pulpal dendritic cells in and around the odontoblastic layer. *(Courtesy Mats Jontell.)*

two distinct populations of dendritic cells that have been identified in the dental pulp. CD11c+ are found in the pulp/dentin border and subjacent to pits and fissures. F4/80+ dendritic cells are concentrated in the perivascular spaces in the subodontoblastic zone and inner pulp.[264] 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 den-

dritic cells under the carious lesion have led to speculation that dendritic cells may play a role in odontoblast differentiation and/or secretory activity in the immune defense and dentinogenesis. Pulpal Schwann cells have also been shown to produce molecules in response to caries, which is indicative of the acquisition of the ability for antigen presentation.

Evidence suggests that odontoblasts also play a role in the humoral immune response to caries. Immunoglobulin (Ig)G, 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.[171] 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.[170]

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] These findings support the contention that antigen-antibody complex formation, in addition to various products of the inflammatory cascade, gives rise to a nonspecific response that, while designed to rid the body of pathogens, effects destruction of parenchymal tissues as well.

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 (for a review see Ref. 43). External stimulation of dentin causes the release of pro-inflammatory neuropeptides from pulpal afferent nerves.[35] Substance P, calcitonin gene-related peptide (CGRP), neurokinin A (NKA), NKY, and vasoactive intestinal peptide are released and affect vascular events such as vasodilatation 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. Both receptor field studies and anatomic studies have shown sprouting of afferent fibers in response to inflammation.[35]

Neuropeptides can act to modulate the pulpal immune response. It has been demonstrated that substance P 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 decrease in H_2O_2 production by macrophages and a diminution of class II antigen presentation and lymphocyte proliferation.

Substance P and CGRP are mitogenic for pulpal and odontoblast-like cells and thereby initiate and propagate the pulpal healing response.[237] CGRP has been shown to stimulate the production of bone morphogenic protein by human pulpal cells. The result of such stimulation has been postulated to induce tertiary dentinogenesis.[37]

As the carious lesion approximates the pulp, there is an acute exacerbation of the precedent chronic inflammation. This is characterized by an influx of neutrophils. The accumulation of inflammatory cells becomes marked when the infection front reaches tertiary dentin.[128,129,200] In the presence of severe pulpal inflammation, focal microabscesses form and eventually coalesce, leading to progressive pulpal necrosis (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

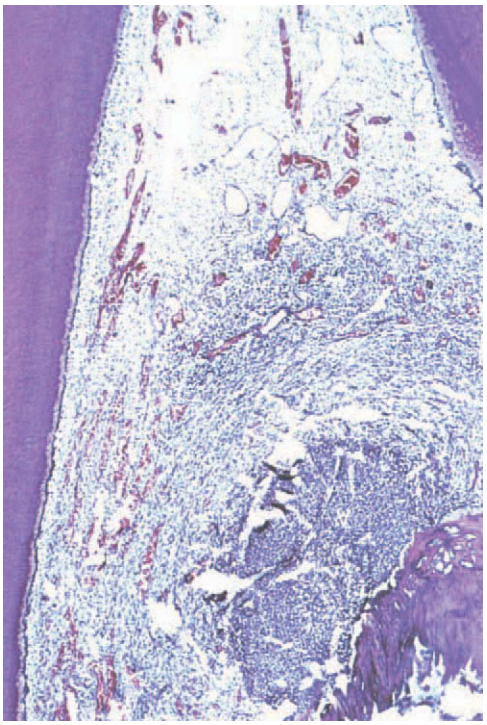


FIG. 13-7 Hematoxylin- and eosin-stained section of a dental pulp, displaying pulpal necrosis.



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.)*

a rich blood supply coupled with ample lymphatic and oral drainage allows this proliferative response. Conventional root canal therapy or progressive vital pulp therapy is indicated.

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 the patient's presenting symptoms. 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, such 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 clinician to determine clinically whether the inflammation is reversible or irreversible.

Many clinicians 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. The findings of these studies revealed that in the vital pulp, clinical symptoms generally did not correlate with gross histomorphologic findings.[90,150,209] 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.[209] In a few studies, prolonged or spontaneous severe symptoms were associated with chronic partial or total pulpitis or pulp necrosis.[57,209] However, in these as well as other studies, it was common to find cases with histologic evidence of severe inflammatory responses, including partial necrosis, but little or no clinical symptoms—the so-called painless pulpitis.[57,90,150,209] Based on these studies and more recent data, it was reported that the incidence of painless pulpitis that leads to pulp necrosis and chronic periradicular periodontitis is about 40% to 60% of pulpitis cases.[149]



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.)*

Objective clinical findings are essential for determining the vitality of the pulp and whether the inflammation has extended into the periapical tissues. Lack of response to electric pulp testing is generally indicative that the pulp has become necrotic.[202,209] Thermal pulp testing is valuable in reproducing a symptom of thermal sensitivity and allowing the clinician 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.[57,209] 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.[190,191] Pulp necrosis could be verified by a test cavity and/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 recent advances in the science of pulp biology. In the last few decades, studies have shown that numerous molecular mediators may act in synchrony to initiate, promote, and/or modulate the inflammatory response in the dental pulp. Without the use of very specialized staining techniques, the nature and quantity of these inflammatory mediators cannot be determined from histologic analysis. Many of these molecular mediators tend to reduce the pain threshold, either directly by acting on peripheral nerve cells or through 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[47,204]; the vasoactive amine, bradykinin[134]; tumor necrosis factor alpha (TNF-α)[123]; neuropeptides such as substance P,[29] CGRP, and NKA[11]; and catecholamines.[167] 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.[11] In another study, trained volunteers had an incisor stimulated with a constant current threefold the threshold value for 90 seconds.[10] This resulted in a significant increase in crevicular matrix metalloproteinase 8 (MMP8), one of the collagenases involved in tissue destruction.

It has also been determined that peripheral opioid receptors are present in the dental pulp,[108] 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[61] and its Y1 receptor,[62] compared to normal teeth. Neuropeptide Y is a sympathetic nervous system neurotransmitter 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.[60]

With recent advances in molecular biology, efficient simultaneous detection of hundreds of molecular mediators 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.[147] Gene microarrays have been used in several studies to obtain an accurate mapping of candidate genes that show elevated expression in inflamed pulp, the odontoblastic cell layer.[148,176,177] Development of more accurate chairside diagnostic methods is potentially feasible, particularly 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 represents a special situation in which a significant, usually chronic, pulpal problem arises that does not seem to be associated with irreversible pulpal pathosis in the majority of cases. Dentin hypersensitivity is characterized by short, 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 pathology.[94] 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[2,3,44] and may be increased following scaling and root planning.[44,251] The dentin is hypersensitive most likely due to the lack of protection by cementum, loss of smear layer, and the hydrodynamic movement of fluid in dentinal tubules.[4,31] 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[261] (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[146] or tubule blocking agents such as strontium chloride, oxalates, or dentin bonding agents (Fig. 13-11)[4,184] usually alleviates the condition, at least temporarily. However, the placement of passive molecules or crystals may provide only temporary relief, so there has been a need to provide biocompatible materials that bond to the root surface to provide a more lasting solution. One such material was a calcium sodium phosphosilicate bioactive glass,[141] 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.[256] However, a recent study found that BisBlock and two other products (Seal&Protect, DENTSPLY DeTrey GmbH, Konstanz, Germany, and Vivasens, Ivoclar Vivadent AG, Schaan, Liechtenstein) were effective several weeks after treatment, compared to placebo.[180] 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 macromolecules are all thought to reduce the problem[183] (illustrated by Mechanism of Dentin Hypersensitivity animation on the Expert Consult Site).

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 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,[185] 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[118] (Fig. 13-12), and that this effect was more severe with periodontal ligament injections[117] (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,[5,46,160] even if the infiltration was palatal to maxillary premolars.[193] 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 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.[169] It is important to note a limitation of studies using laser Doppler flowmetry: a large proportion of the signal measured may be from sources other than the dental pulp.[192,219] 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.[89] Human studies on the effects of periodontal ligament or intraosseous injections on pulpal blood flow are unavailable, but from animal studies, it is probable that these supplemental anesthetic techniques cause more severe reduction or even transient cessation

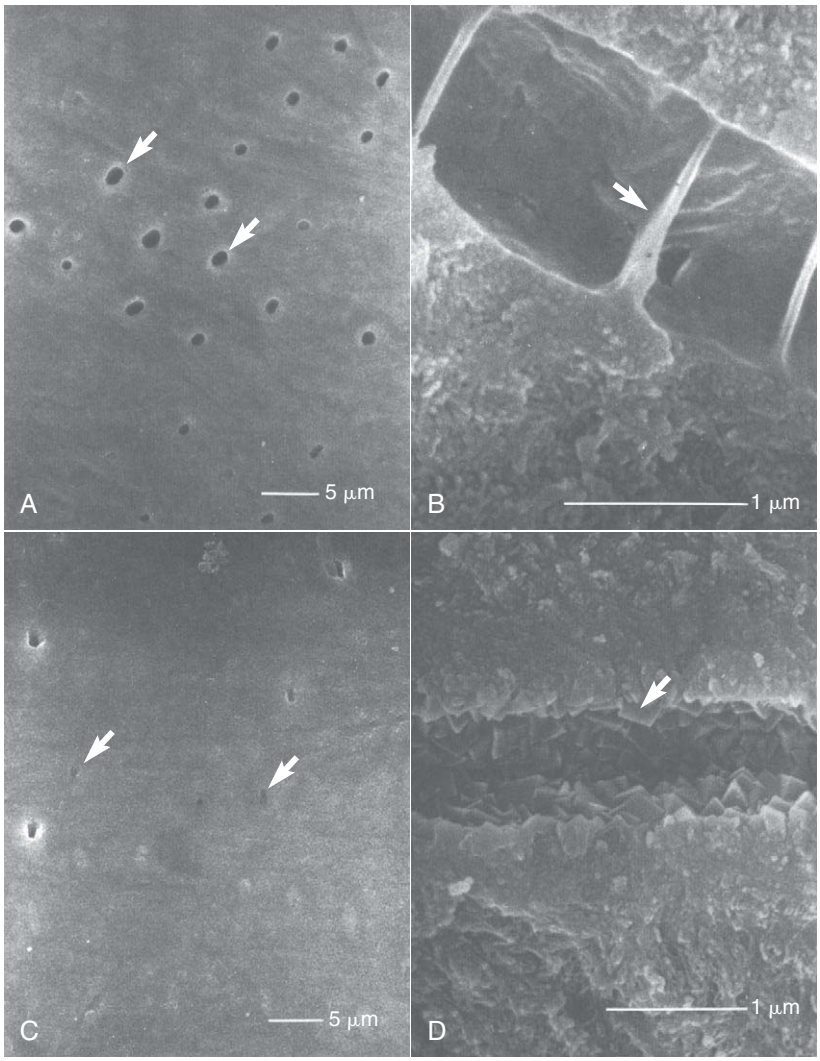


FIG. 13-10 A, Scanning electron microscope (SEM) image of an exposed dentin surface of a hypersensitive area. A large proportion of dentinal tubules *(arrows)* are seen to be open. **B,** SEM image of a fractured dentinal tubule of a hypersensitive area. The lumen of the dentinal tubule is partitioned 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 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.)*



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 (×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.)*

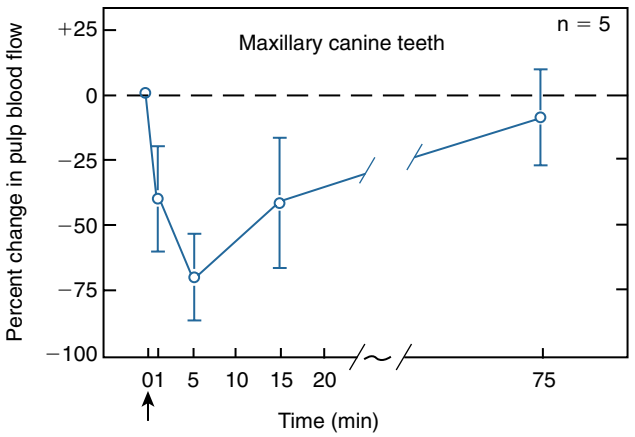


FIG. 13-12 Effects of infiltration anesthesia (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the maxillary canine teeth of dogs. There is a drastic decrease in pulpal blood flow soon after the injection. *Arrow* indicates the time of injection. *Bars* depict standard deviation. *(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.)*

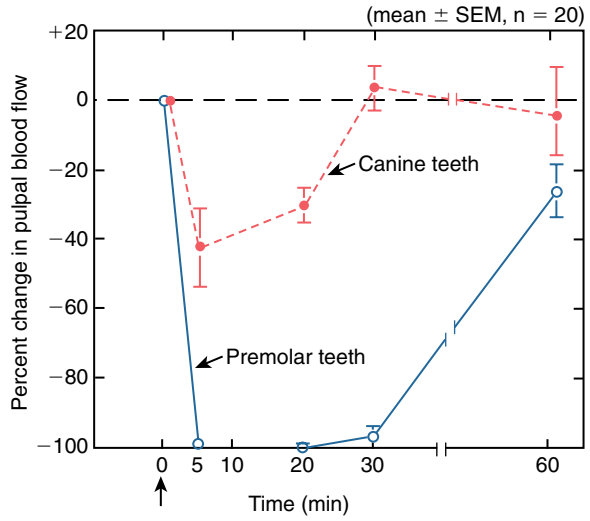


FIG. 13-13 Effects of ligamental injection (i.e., 2% lidocaine with 1:100,000 epinephrine) on pulpal blood flow in the mandibular canine and premolar teeth of dogs. Injection was given in the mesial and distal sulcus of premolar teeth. Injection caused total cessation of pulpal blood flow that lasted 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.)*

of pulpal blood flow. It was also shown that intraosseous injection of Depo-Medrol (a corticosteroid) in patients with symptomatic irreversible pulpitis causes 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.[104] Taken together, these findings suggest that local anesthesia may compromise the inflamed pulp's ability to recover from inflammation, particularly if it is severely inflamed, if the tooth is subjected to extensive restorative procedures, or 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, since 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.[233]

PULPAL REACTIONS TO RESTORATIVE PROCEDURES

A voluminous 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, 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 that would not potentially convert the diagnosis to irreversible pulpitis. As was discussed previously, irreversible pulpitis may present clinically with severe spontaneous postoperative pain, but it may also be asymptomatic, leading to the quiet demise of the pulp. The additive effects of restorative procedures are particularly critical in borderline cases, such as those of mildly symptomatic teeth with deep caries but no pulp exposure. There are still many factors whose influence on the response of the dental pulp to 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. The question of the fate of pulps beneath single unit metal-ceramic (MC) crowns or MC bridge abutments was addressed.[45] Patients who had received either treatment from 1981 to 1989 at the Prince Philip Dental Hospital in Hong Kong were randomly selected and invited to attend a recall appointment that involved both clinical and radiographic examinations. One hundred twenty-two teeth with preoperatively vital pulps treated with single unit MC crowns and 77 treated as bridge abutments were examined. The mean observation period was 169 months for the former and 187 months for the latter. Pulpal necrosis had occurred in 15.6% of the teeth treated with single unit crowns after 10 years, while 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 several key factors discussed in the following sections.

Degree of Pretreatment Pulp Inflammation

As stated previously, the dental pulp is compromised in its ability to respond to external irritants because (1) it is enclosed in a noncompliant environment, and (2) it lacks sufficient collateral circulation. Thus the more severely the pulp is inflamed, the less will be its ability to respond to further irritation, such as in the form of restorative procedures.[130]

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 pulp is

already inflamed by the carious lesion and pulpal blood flow is reduced by local anesthetic injection. Classic experiments have documented that different levels of irritation induce different degrees of inflammation in the pulp,[154] and more severe inflammation may take longer to heal.[155] In a study that evaluated the response of the pulp to capping procedures as a function of duration of exposure, it was shown that the pulp responds favorably to exposures of up to 24 hours after exposure, but not as favorably after longer periods of exposure to oral environment.[52] It may be that the longer exposure periods lead to the formation of a bacterial biofilm that is difficult for the pulpal immune responses to eliminate. 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. 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,[17,95] but newer studies in which MTA was used show more favorable results in these cases.[16,26]

In the absence of severe spontaneous symptoms or pulp exposure, the clinician currently cannot accurately determine the degree of preoperative pulpal inflammation. Thus, every effort should be made to minimize added irritation during restorative procedures, because it is possible that excessive irritation could convert the inflammatory status of the pulp from a reversible to an irreversible condition.

Degree of Physical Irritation Caused by Procedure

The physical irritation of the pulp during restorative procedures, such as from heat, desiccation or vibration, may adversely affect the dental pulp.

Heat

Restorative procedures such as cavity preparation, crown preparation, or curing of resins during direct fabrication of provisional restorations[234] 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.[262] 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, 13-15, 13-16). However, a 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.[12] In this study, an increase of intrapulpal temperature of about 11° C, followed for 2 to 3 months of evaluation, did not induce 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.[7] 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.[168] It was common in that study to see 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,[80] presumably because of the poor conductive properties of dentin and the cooling effect of compressed air from the high-speed handpiece. 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. 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 excessive heat with other irritation factors and its proximity to the pulp may induce pathologic changes.

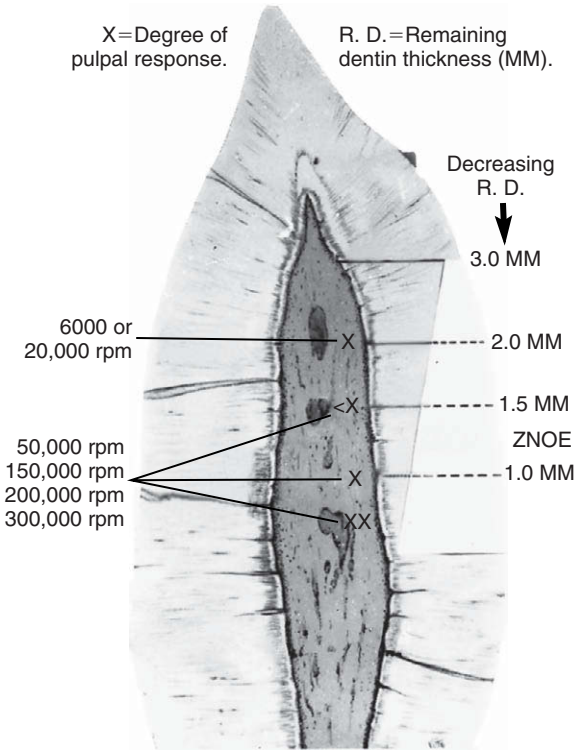


FIG. 13-14 With adequate water and air spray coolant, the same cutting tools, and a comparable remaining dentin thickness, the intensity of the pulpal response with high-speed techniques (i.e., decreasing force) is considerably less traumatic than with lower-speed techniques (i.e., increasing force). *(From Stanley HR, Swerdlow H: An approach to biologic variation in human pulpal studies.* J Prosthet Dent *14:365, 1964.)*

Desiccation

Desiccation during cavity and crown preparation has long been known to cause aspiration of odontoblastic nuclei into dentinal tubules and pulpal inflammation.[30] 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.[51]

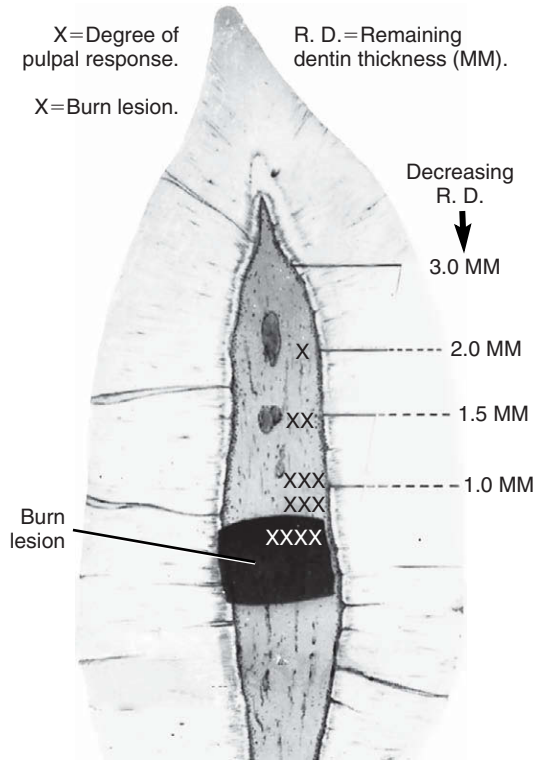


FIG. 13-15 Without adequate water coolant, larger cutting tools (e.g., No. 37 diamond point) create typical burn lesions within the pulp when the remaining dentin thickness becomes less than 1.5 mm. *(From Stanley HR, Swerdlow H: An approach to biologic variation in human pulpal studies.* J Prosthet Dent *14:365, 1964.)*

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.[138] 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.[36]

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.[33] However, despite the elimination of visible caries during cavity preparation, the cavity floor is undoubtedly left with some contamination by caries bacteria. Whereas 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.[50] An earlier study showed that the amounts of residual bacteria following adequate restoration are not significant.[151] Once dentin is exposed, there is constant outward flow of dentinal fluid that minimizes the inward flow of any noxious agents.[252] This may aid in the reduction of irritation from residual microbial factors in dentinal tubules.

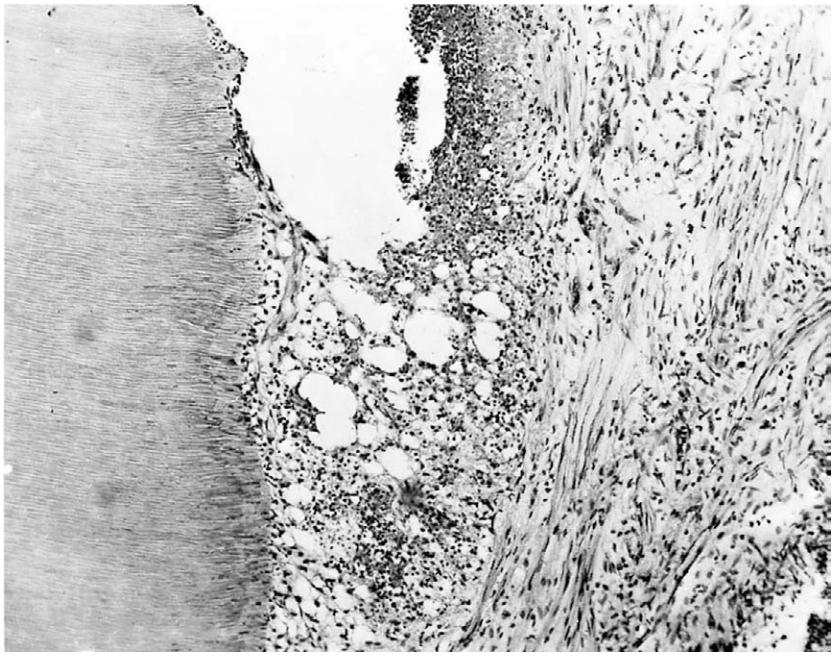


FIG. 13-16 Burn lesion with necrosis and expanding abscess formation in a 10-day specimen. Cavity preparation dry at 20,000 rpm, with remaining dentin thickness 0.23 mm. *(From Swerdlow H, Stanley HR Jr: Reaction of the human dental pulp to cavity preparation. I. Effect of water spray at 20,000 rpm.* J Am Dent Assoc *56:317, 1958.)*

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.[81,179] 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 promote permeation of 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% ethylenediamine tetra-acetic acid (EDTA) or not etched.[159] Pulpal inflammation in this study was not correlated with the etching treatments but with bacterial presence; in cases of etching with phosphoric acid, if bacteria were also present, severe pulpal inflammation and necrosis could be seen.

In recent years, 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 compared to total-etch systems.[28] However, studies have shown no significant differences between the two adhesive systems in long-term in vitro bond strength, postoperative sensitivity,[188] long-term in vivo degradation,[125] or long-term in vitro bond strength.[9] One clinical study showed no differences between the two systems with respect to bacterial leakage and the inflammatory response in the pulp.[164] 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[113] and monocytes/macrophages[133,196,197,208] in vitro. These components may leach directly into the pulp in deep cavities and cause chemical irritation.[98] 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.

Proximity of Restorative Procedures to Dental Pulp and Surface Area of Dentin Exposed

It has been known for several decades that as the carious lesion progresses towards 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.[201] The diameter and density of dentinal tubules increase closer to the pulp (Fig. 13-17). Based on the dentinal tubule density at the dentin-enamel junction (DEJ) (about 65,000/mm^2) and the pulp (about 15,000/mm^2),[74,78] 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.[183] Thus it is not surprising that several studies have shown that pulpal inflammation in response to restorative procedures increases with the reduction in RDT.[1,53,158] A recent 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.[157] 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 on pulpal injury.

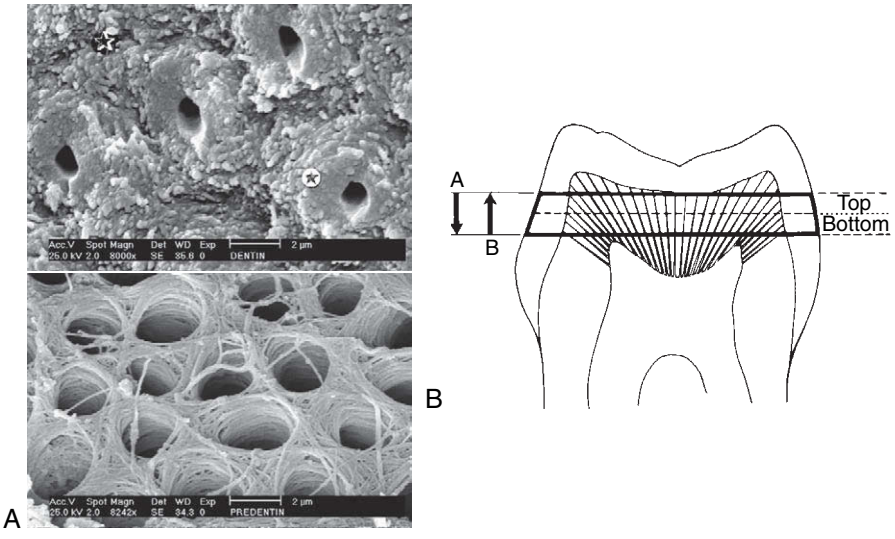


FIG. 13-17 Convergence of tubules toward the pulp. **A,** Periphery of the dentin. Most surface area is occupied by intertubular dentin (☆), with a few tubules surrounded by hypermineralized peritubular dentin (✪). **B,** Near the pulp, the increase in tubule diameter has occurred largely at the expense of the peritubular dentin. This substrate has a high protein content. As the remaining dentin is made thinner (from **A-B**), the permeability of the dentin increases because both diameter and density of dentinal tubules are increased. *(Bouillaguet S: Biological risks of resin-based materials to the dentin-pulp complex.* Crit Rev Oral Biol Med *15:47–60, 2004.)*

With the passage of time following cavity preparation, there is reduction in the permeability of RDT.[186] This may be due to rapid deposition of reactionary dentin, the migration of large proteins into the tubules, and/or the diminution of tubule diameter as dentin becomes more sclerotic. Using a primate model, one study showed that the basic rate of secondary dentin deposition is about 0.8 µm/day, and this rate increases to an average of 2.9 µm/day following restorative procedures. Interestingly, dentin deposition was more rapid next to shallow cavities than deep cavities in one study,[254] but another study showed that total reactionary dentin deposited was thicker in deeper and wider cavities.[156]

Clinically it has been observed that postoperative sensitivity is common with many restorative procedures. In one study, it was shown that following resin composite restorations on patients, postoperative sensitivity was related to the depth of the cavity but not to the presence or absence of liners or bases.[244]

In addition to the depth and/or 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 imperfect adaptation of temporary restorations, leading to 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). There are few studies available on the effects of modern crown and bridge techniques on the pulp. However, some long-term outcome studies have documented that the incidence of pulp necrosis following crown placement ranges from 10% to 18%.[70,245] As noted earlier, a more recent paper of work done in the 1980s in Hong Kong showed these figures to be much larger in bridge retainers compared to single crowns, reaching a value of up to 54% in anterior teeth.[45]

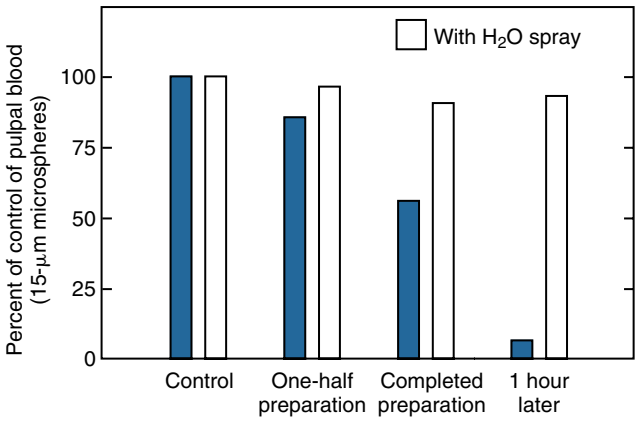


FIG. 13-18 Effects of crown preparation in dogs, with and without water and air spray (at 350,000 rpm) on pulpal blood flow. Tooth preparation without water and air spray caused substantial decrease in pulpal blood flow, whereas that with water and air spray caused insignificant changes in the flow.

Permeability of Dentin and Odontoblastic Layer Between Area Being Restored and Pulp

The permeability of dentin plays an important role in the ingress of potential irritants to the pulp. It is clear from research done in the last 3 decades 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 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.[86] 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.

With age, the width of peritubular dentin increases, causing 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[184] and could be considered a protective mechanism, since it may delay the progress of the carious lesion.

It has been reported that the odontoblastic cell layer itself may contribute to the reduction of permeability to large molecules such as bacterial proteins or toxins. Experiments were conducted in which large protein markers were allowed to permeate the odontoblastic layer in experimental animals before and after cavity preparation. It was shown that irritation from cavity preparation increased the odontoblastic permeability only at the site of the cavity preparation.[106,240] 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[136,178] or recognize bacteria through toll-like receptors.[27,59,67,112]

Patient Age

It was reported that resting pulpal blood flow (PBF), as well as the changes in PBF in response to cold application, decrease with age.[100] Age may also be associated with reduction in pulpal neuropeptides.[75] However, a recent animal study failed to show any 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.[116] Thus in humans with advancing age, the net result of the ability of the pulp to cope with external stimulation or irritation 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.[230] As noted before with respect to restorative procedures and bacterial ingress, perhaps the

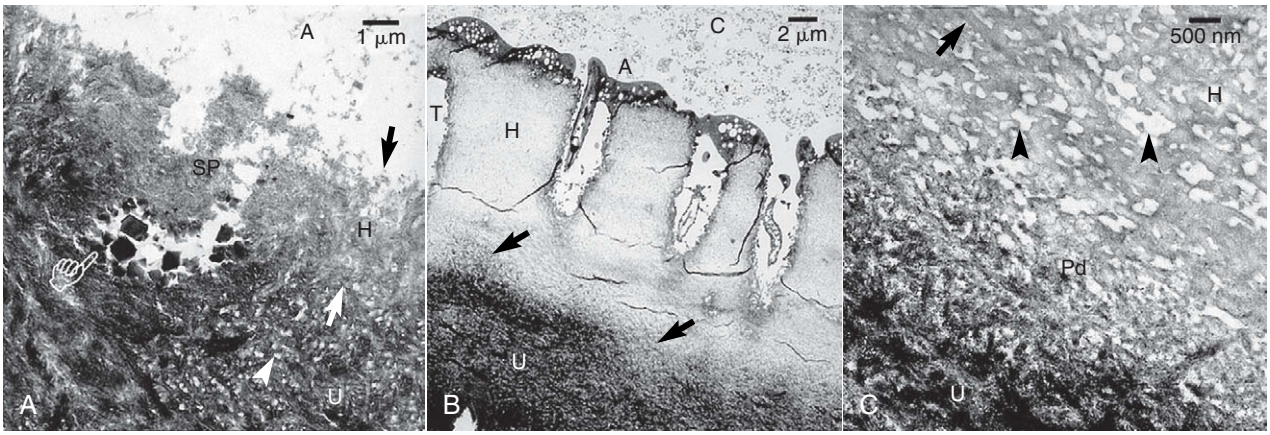


FIG. 13-19 Transmission electron micrographs (TEM) of undemineralized specimens of resin-bonded caries-affected dentin. **A,** Stained TEM of undemineralized specimens following the application of the self-etch ABF system to caries-affected dentin. The hybrid layer *(H, between arrows)* was about 3 µm 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.)*

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]

Unbound components of resin materials and preparative agents such as acid etchants can affect the subjacent pulp by inducing an inflammatory response.[77,195] The indirect effects of desiccation and/or demineralization of dentin, as well as direct effects of the material itself when in contact with pulpal tissue, mediate this inflammatory response. Studies have shown that certain cytotoxic components of resin monomers (e.g., triethylene glycol dimethacrylate and 2-hydroxyethyl methacrylate) readily penetrate dentin.[72] Similarly, eugenol and components of Ledermix (triamcinolone and demeclocycline) have been shown to pass through dentin into the subjacent pulp.[97,99] In vivo data show that these chemicals have an effect on the pulp, but the effect seems to be short lived and, in the absence of bacteria, reversible.[21]

The mechanisms whereby restorative materials exert an injurious effect on the dental pulp are varied. Evidence exists that supports direct and, in some instances, prolonged cytotoxicity, stimulation of hypersensitivity reactions, or impairment of the host immune response to bacteria. 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.[71] Furthermore, even subtoxic concentrations of certain agents are capable of eliciting allergic reactions in humans.[98] 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.[115] 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.[102,103] Histologic examination of such 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.[113] 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; disease, débridement, 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.[55,207] The degree of inflammation is dependent on the preparation of calcium hydroxide used. Aqueous suspensions of calcium hydroxide applied to exposed pulps causes superficial necrosis of pulpal tissue followed by tissue displaying low-grade inflammation. Within 30 days, the tissue subjacent to the necrotic zone has reorganized and resumed normal architecture. Hard-setting calcium hydroxide preparations are effective in eliciting dentinal bridge formation with a much smaller to nonexistent necrotic zone.[92] 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[54] (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

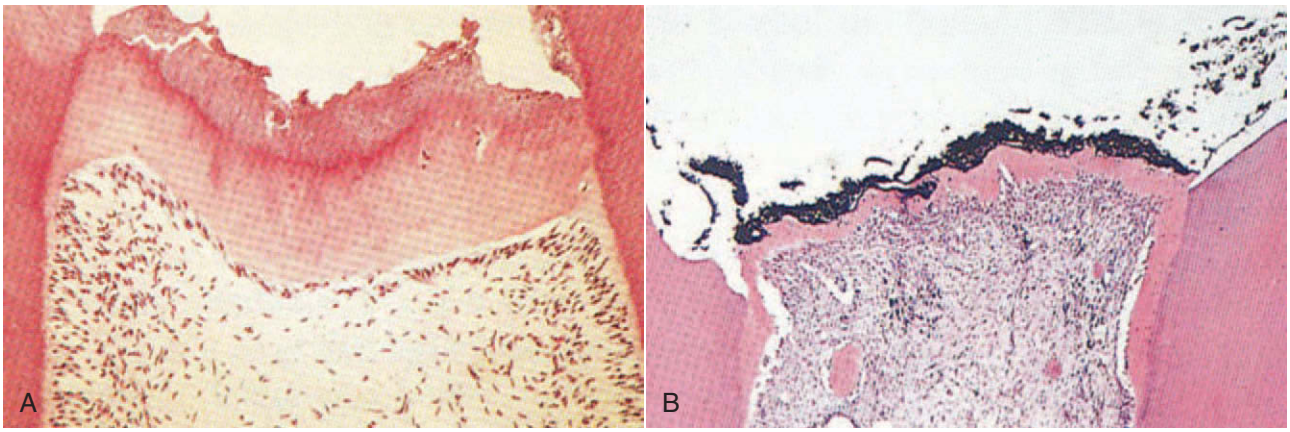


FIG. 13-20 Calcium hydroxide produces an inflammatory response that stimulates dentinal bridge formation. The dentin bridge forms lower in the tooth with calcium hydroxide paste **(A)** versus hard-setting calcium hydroxide **(B)**.

of calcium hydroxide to intact dentin appears to induce sclerosis by promoting crystal precipitation within the tubules, accompanied by reductions in permeability.[153]

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.[82,152] Applied resin infiltrates the exposed collagen mesh, creating a layer 5 to 10 µm thick referred to as the *hybrid layer*.[161] 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. The optimal degree of hydration of the preparation surface can vary from material to material, so 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.[69] 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.[238] 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 long-term restorations; ZOE temporaries have been shown to leak after only a few weeks in situ.[263]

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.[229] Although calcium hydroxide has historically been the preferred dressing agent on mechanically exposed pulps, the use of mineral trioxide aggregate (MTA) has recently been proposed, even on carious pulp exposures.[16,26] 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.[66,73,121] Although the results are generally favorable, one concern is of tooth discoloration if the gray MTA formulation is used on anterior teeth.

In a recent investigation, white MTA was compared to gray MTA in the capping of direct mechanical pulp exposures created in dogs' teeth.[182] No significant differences were found in the healing response to either material. At 1 week, none of the capped pulps showed necrosis close to the exposure site, and odontoblast-like cells were observed at the periphery and under the calcified bridge. At 2 weeks, almost all of the specimens from both types of MTA showed complete calcified bridge formation just below the exposure site. In a recent clinical study, MTA was used as a pulp capping material for carious pulp exposures.[26] Forty patients aged 7 to 45 were diagnosed with reversible pulpitis and 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. 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. It must be emphasized, however, that not only must the pulp capping agent be biocompatible and hopefully stimulate the formation of reparative dentin, but the prevention of bacterial ingress by the placement of a well-sealed restoration must also be provided.

USE OF HEMOSTATIC AGENTS AND DISINFECTANTS ON DIRECT PULP EXPOSURES

As noted before, the use of direct capping procedures in an attempt to maintain the vitality of an exposed dental pulp has been a controversial area of pulp biology. In particular, the impact of preoperative diagnosis and the effects of various dental medicaments and materials on the long-term prognosis of exposed pulps have been questioned.[53,95,179,215,221] With regard to the former, it has generally been accepted that carious pulp exposures offer poor therapeutic opportunities for continued pulp vitality with direct capping techniques.[17,95] In the case of the latter, there is still debate as to whether the toxicity of medicaments and materials placed on vital pulp tissues determines outcome, or if their ability to seal the cavity from further bacterial ingress is of prime importance. It is likely a combination of the two, as was shown in the recent 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.[221] 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.

A recent 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.[211] 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. The teeth were restored with class I bonded resin restorations, extracted after 7, 30, or 90 days, and processed for routine histologic evaluation. 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 (full concentration is now 6.0%), 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. There are several different types of laser technologies 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. It has the longest wavelength

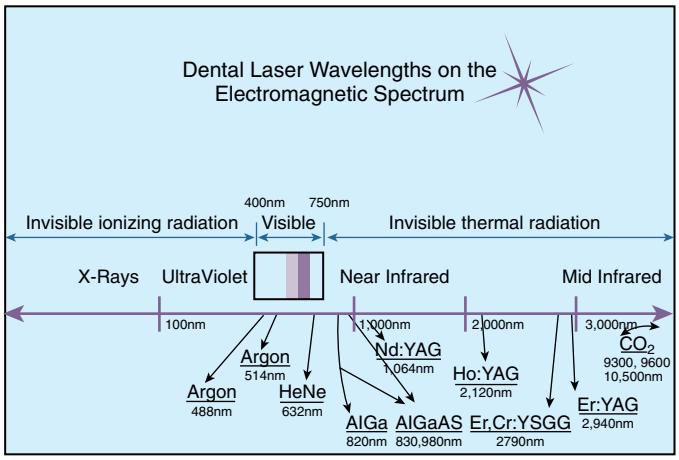


FIG. 13-21 Currently available dental wavelengths on the electromagnetic spectrum. Note all of the wavelengths are nonionizing. *(From Coluzzi DJ: An overview of laser wavelengths used in dentistry.* Dent Clin North Am *44:753, 2000.)*

(10,600 nm). It cannot be delivered in an optic fiber, thus must be used in a hollow-tubelike 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, delivered using a solid optic fiber. It has a high affinity for water and hydroxyapatite, thus can be used for removal of caries and cutting 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 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 high affinity for water and pigmented tissues and offers good hemostasis, thus is used extensively in surgery.[48] In addition, there are lasers with low power output, such as *HeNe,* or helium neon (632 nm), and *GaAlAs,* or gallium-aluminum-arsenide (diode; semiconductor) (720-904 nm) lasers, which have been used in laser Doppler flowmetry and in treating dentin hypersensitivity.[119]

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 apex, 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, but this was shown to not add any advantage following acid etching—a necessity for

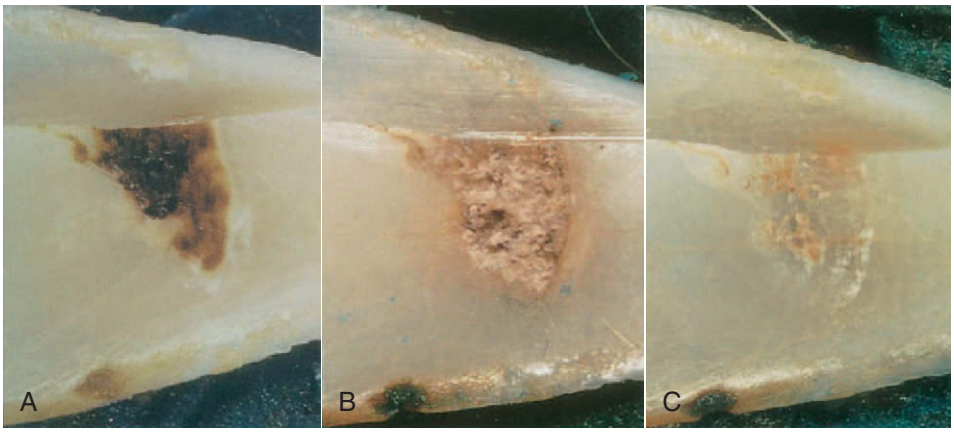


FIG. 13-22 A, Dental caries. **B,** Pulsed Nd:YAG ablation byproducts (160 mJ, 10 Hz). **C,** Debris removed with acid etch and polishing. Enamel surface was faceted for reflection spectroscopy. *(From Harris DM, White JM, Goodis H, et al: Selective ablation of surface enamel caries with a pulsed Nd:YAG dental laser.* Lasers Surg Med *30:342, 2002.)*

adhesion.[143] 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,[144] more recent work suggests that they are best used as adjunctive devices to radiography and visual examination.[6,49,126]

Laser ablation of superficial carious lesions may be more conservative than bur preparation. A controlled clinical trial supported this tenet when free-running pulsed Nd:YAG laser was used to ablate superficial pit and fissure caries in third molars scheduled for extraction[88] (Fig. 13-22). In this study, there were no histologic differences in the pulp response between the two groups.

From the perspective of effects on the pulp, most laser applications 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).[220] 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.[255] 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 Er:YAG laser[235,255] (Fig. 13-23). More recently it was reported that water cooling was as necessary for laser ablation as it is for high-speed bur preparations.[41] Studies have shown that 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.[65,231,232,255] However, the findings with electron microscopy were different. It was reported that whereas shallow cavities ablated in rat molars using Er:YAG lasers did not show changes from baseline using light microscopy, transmission electron microscopy (TEM) showed disruption and degeneration of pulpal peripheral nerve endings and of myelin sheath in the immediate postoperative period (Fig. 13-24).[101] 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 in cavity preparation compared with traditional methods at this time.

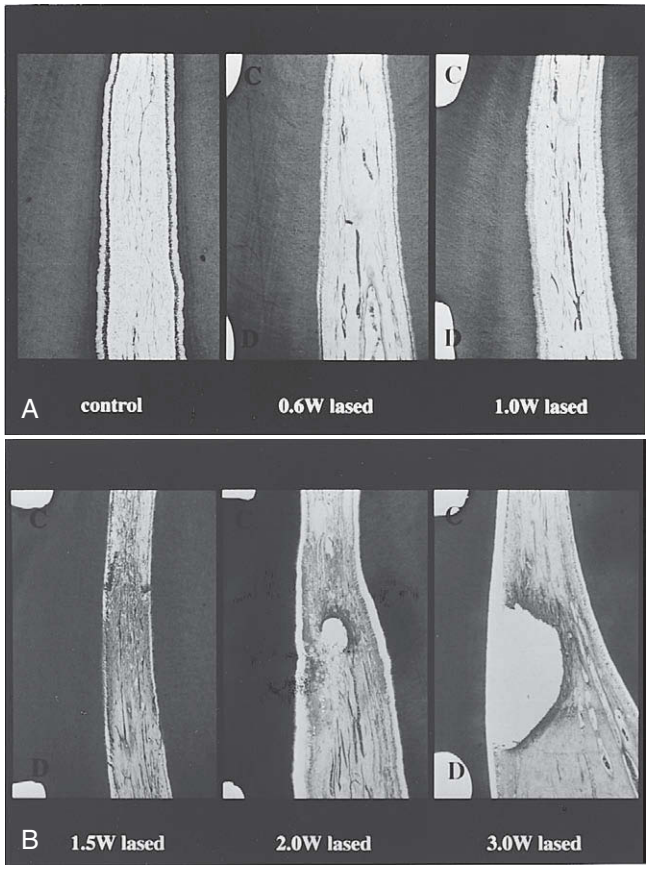


FIG. 13-23 A-B, Histopathologic picture of Nd:YAG laser specimen with an increasing power. There is a direct relationship between the degree of pathologic changes and the increasing power of the laser. In fact, 1.5 W and the greater power cause permanent damage to the pulp.

For pulpotomy procedures, such as in primary teeth, permanent teeth with immature apex, or pulps that are exposed due to fracture and treated promptly, the lasers (particularly CO_2 lasers) may be useful in achieving precise surgical excision of coronal pulp and immediate hemostasis. A controlled

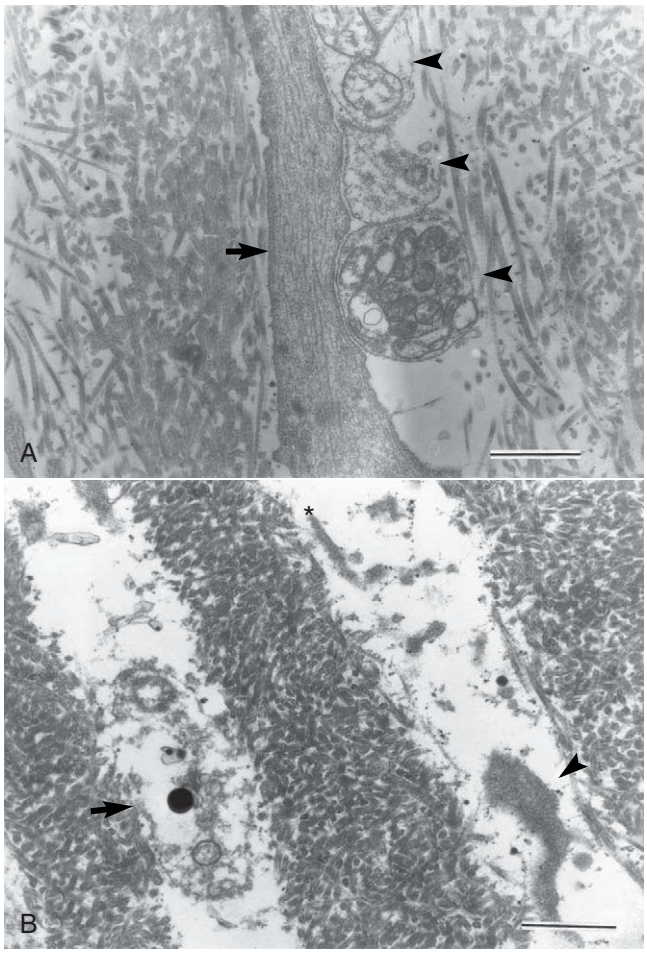


FIG. 13-24 A, Control; normal odontoblastic process *(arrow)* and a few nerve terminals *(arrowheads)* are seen in a dental tubule of rat upper first molar. **B,** Six hours after Er:YAG laser irradiation; disrupted cell membrane of a nerve terminal that contains some granular vesicles *(arrow)* and shrinkage of an odontoblastic process *(arrowhead)* are noted in dentinal tubules just under the ablated area. *Asterisk* indicates the irradiated side (transmission electron micrograph, ×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.)*

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.[63] However, an animal study in which CO_2 and Nd:YAG lasers were compared revealed poor response with both lasers compared with calcium hydroxide.[114] A more recent animal study using Er:YAG laser showed that the results are dependent on the power settings, in that lower energy delivered with this laser produced favorable results.[120]

Lasers in the Treatment of Dentin Hypersensitivity

Earlier studies have shown effectiveness ranging from 5% to 100% of low-output lasers on dentin hypersensitivity.[119] 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.[119] Low-output lasers do not have any effects on the morphology of enamel or dentin, but they are thought to cause transient reduction in action potential mediated by pulpal C-fibers, but not Aδ-fibers,[253] although this finding was not consistent.[174] 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,[142] in addition to action potential blockage within the pulp in vitro or in experimental animals.[173,174] However, a placebo-controlled clinical trial has shown that both Nd:YAG and placebo caused significant reduction in dentin hypersensitivity for up to 4 months postoperatively but were not different from each other.[137] Therefore, the collective available evidence does not support the use of lasers for treating dentin hypersensitivity. Other more conservative and economical therapies are recommended.

Use of Lasers as a Protective Measure for Dentin

A recent 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 because its permeability and bacterial content may decrease. In this study, two patients were scheduled to have six teeth extracted during orthodontic treatment.[79] 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 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 CAVITY PREPARATION USING AIR ABRASION TECHNIQUES

Air abrasion is another modality that has been adopted for conservative, nonpainful cavity preparations. Aluminum oxide particles are forced in a rapid stream onto the tooth structure and simultaneously evacuated from the field. An earlier study evaluated the effects of air abrasion in class V preparations using two different particle sizes (25 and 50 μm) and two different forces (80 and 160 psi) on the dental pulp of dogs. The findings showed that histologically, higher pressures and smaller particles yielded significantly fewer pulpal effects than the high-speed treated teeth, whereas lower pressures and larger particles were not significantly different.[132] Air abrasion cavity preparations were more recently shown to be equivalent to high-speed bur preparations in microleakage studies if both techniques are followed by acid etching.[250] Because of the lack of tactile control, the use of air abrasion is limited to specific situations such as shallow caries or pediatric applications.

PULPAL REACTIONS TO VITAL BLEACHING TECHNIQUES

Vital bleaching techniques employ the use of 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 are present. An earlier study identified the toxicity of these chemicals on cell cultures in vitro.[84] 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.[8,76] 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 post-treatment symptoms, these tend to be reversible and could be prevented by treating the teeth with fluorides and by correcting restorative pretreatment deficiencies.[163] Clinical symptoms are likely to be due to increases in neuropeptides in the pulp. A recent report showed that light- and laser-activated bleaching systems increase substance P expression in the pulp to significant levels.[42] An earlier clinical trial showed that vital bleaching using 10% carbamide peroxide in a custom tray for 6 weeks was safe for the pulp health for up to 10 years post treatment, although the bleaching effectiveness may decline with time.[203]

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 15). Periodontal disease causes attachment loss, which exposes the root surface to the oral cavity. Occasionally, pulpal inflammation secondary to severe periodontitis is observed.[131] Some reports have described bacterial infiltration through dentinal tubules of exposed root surface, causing mild inflammatory changes in the pulp (Fig. 13-25).[129] 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.[236] It is much more common for pulp necrosis or failed healing of periradicular 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, as happens in cases of vertical root fractures or congenital tooth defects such as palatal groove defect. 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 is 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

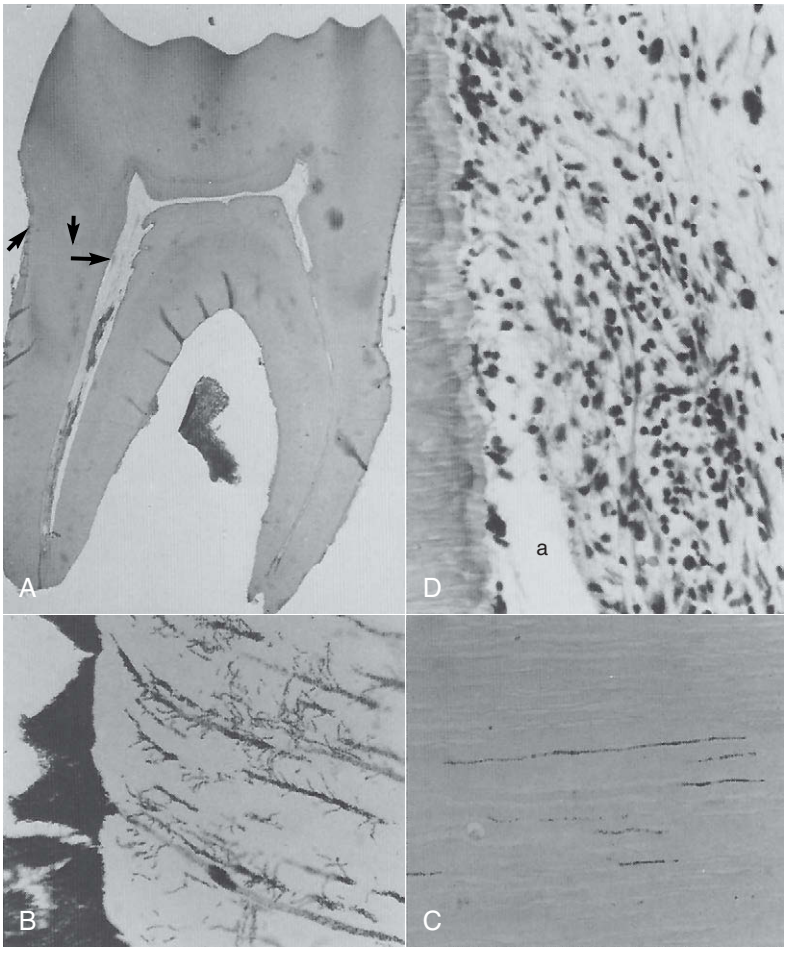


FIG. 13-25 Mandibular first molar of 49-year-old female, no pain. Clinically caries free. Calculus, periodontal disease, and bone loss from two thirds to three fourths of root. **A,** Note narrowness of pulp chamber seen throughout all serial sections (×8.5). **B,** Bacterial plaque and adjacent dentinal tubules with bacteria *(oblique arrows in A)* (×400). **C,** Farthest penetration of bacteria in dentinal tubules *(vertical arrow in A)* (×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 can be seen. *a,* Artifact, pulp tissue torn away from dentin during processing (×400). *(From Langeland K: Tissue response to dental caries.* Endod Dent Traumatol *3:149–171, 1987.)*

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,[110] and the prognosis of endodontic and periodontal treatment may be related,[111] since 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 increased incidence of pulpal pathosis. In an older study, investigators induced periodontal disease in a primate model (using ligatures) and 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, but 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 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.[56] 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.[87,205]

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 result in an increase in pulpal blood flow.[127,165,172] 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 and result in pulpal necrosis.[34]

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.[83] Biochemical and molecular markers confirm that apoptosis and necrosis of pulpal cells is also increased subsequent to movement.[189] However, it was shown that the inflammatory mediators IL-1α and TNF-α show 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.[224-226] Most if not all of these effects are due to circulatory changes, and the consensus is that they are transient, provided the movement forces are not excessive.[124] It was recently shown, however, that pulp necrosis of teeth that had suffered trauma prior to undergoing orthodontic treatment was significantly increased, compared to teeth that had either of these conditions alone—particularly in lateral incisors and when the traumatized tooth had pulp canal obliteration.[18,19]

PULPAL REACTIONS TO ORTHODONTIC 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 and/or necrosis.[15,122,162,187] Occasionally the teeth affected show postoperative manifestations common with traumatic injuries, such as pulp canal obliteration.[247] 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.[58,260] Using laser Doppler flowmetry, a number of studies have documented actual reduction in PBF immediately following maxillary Le Fort I osteotomy,[175,198,206] particularly if segmental osteotomy is performed.[64] In most cases, the blood flow is regained within months of the surgery. Recently a modification of the Le Fort I osteotomy technique was described in which the Le Fort I sectioning was combined with a horseshoe palatal osteotomy to spare any disruption to the descending palatine artery.[85] 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.[212]

BIOMECHANICAL IRRITATION: PARAFUNCTIONAL HABITS

Occlusal loading of teeth affects deformation to varying degrees.[86] Enamel is largely resistant to flexure, but the underlying dentin demonstrates considerable elastic and viscoelastic characteristics. As a result, defects in enamel secondary to cracks, decay, and/or restorative preparation allow cuspal flexure, with subsequent pulpal responses presumably due to dentinal fluid flow secondary to compression and microleakage.[93] 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

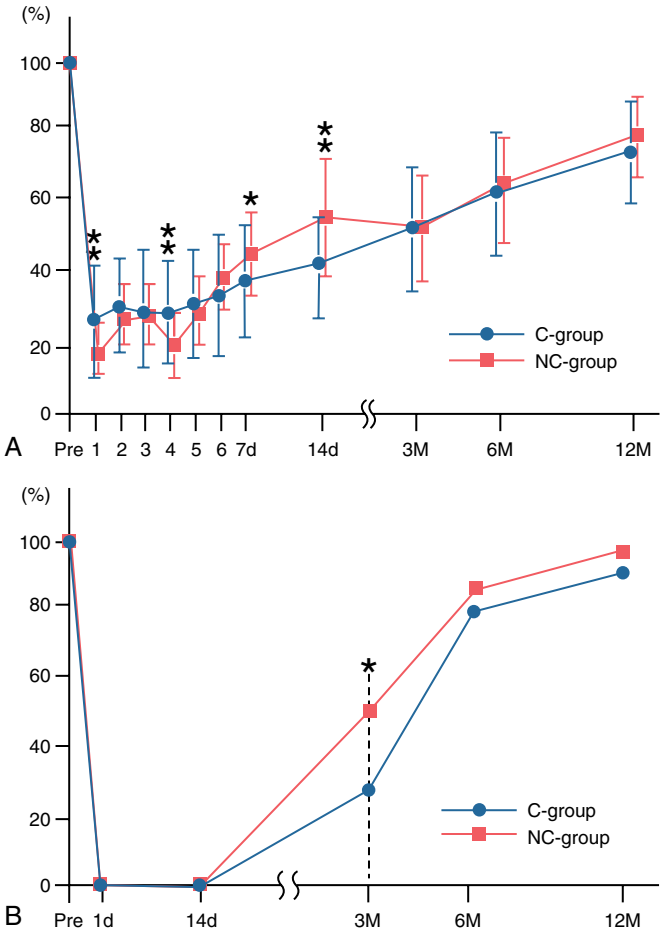


FIG. 13-26 A, Postoperative change of the mean pulpal blood flow in the upper incisors of the two groups. *Pre,* Before the operation; *d,* day(s) after the operation; *M,* months after the operation; *bars,* SD; *, $P < 0.05$; **, $P < 0.01$. **B,** The postoperative change in the percentages of teeth (upper incisors) with positive pulpal sensibility in the two groups. *Pre,* Before the operation; *d,* day(s) after the operation; *M,* months after the operation; *, $P < 0.05$. *(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 *97:12–17, 2004.)*

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.[246,199,181] 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.[248,249]

Symptomology from cuspal flexure can result from two primary sources. It has been theorized that cuspal flexure results in dentin deformation, thus promoting dentinal fluid flow that activates nerve endings in the odontoblastic 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.[93] 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 byproducts 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 and 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.

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 2 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 ablation of symptoms. Clinical interventions include the application of materials that occlude the tubules and extracoronal restorations to 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.[241] This assures that implant placement fully rests in bone and does not compromise neighboring structures, including teeth. The lack of attention to (1) the three-dimensional anatomy of the site of implant placement and (2) the orientation of neighboring teeth may lead to the implant perforating the root and devitalizing the pulp.[145,228]

It is usually recommended that implants not be placed directly at a site where a periradicular lesion, particularly one with signs of purulence, exists; microbial irritants may interfere with osseointegration.[194] However, recent data suggest that immediate implant placement in sites that have been adequately débrided is successful.[38]

Case reports have also claimed that teeth with periradicular lesions may reduce the success of neighboring implants, even if adequate endodontic treatment is performed.[227] To address this issue, a study was reported in which implants were placed to replace premolars in dogs, and periradicular lesions were then induced. Some were treated nonsurgically or with combined surgical and nonsurgical treatment.[210] 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. Following adequate endodontic treatment, complete resolution of periapical lesions that also involve neighboring implants has been reported.[139,223] Furthermore, implants that are not mobile but have apical periimplant lesions have been successfully treated with local débridement and "implant-apex resection" procedures.[13]

REGENERATIVE POTENTIAL OF THE DENTAL PULP

Recent studies and case reports suggest that the dental pulp has great regenerative potential, particularly in the immature permanent tooth (see also Chapter 16).[14,105] This has been attributed to the store of stem cells resident in the dental papillae of developing teeth.[218] Mesenchymal stem cells (MSCs) have been found in a variety of tissues and have proven to be pluripotential. Stem cells from the apical papilla (SCAP) of immature teeth have likewise been identified and have been shown to develop into dentinogenic cells, given the proper stimulation.[96,218] The results of these studies, coupled with case reports of continued root development subsequent to apical periodontitis in young teeth, suggest the potential for bioroot engineering in the future.[96]

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CHAPTER

14

Pathobiology of the Periapex

LOUIS M. LIN and GEORGE T-J HUANG

CHAPTER OUTLINE

APICAL PERIODONTITIS
- Prevalence

ETIOLOGY

INFECTION: A CONFLICT BETWEEN HOST AND PARASITES

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- Adaptive/Specific Immune Response
- Neurogenic Inflammation

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- Correlation Between Radiographic and Histologic Findings

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- Inflammatory Mediators
- Histopathology
- Clinical Features
- Outcomes

ASYMPTOMATIC APICAL PERIODONTITIS: APICAL GRANULOMA, CHRONIC APICAL PERIODONTITIS
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- Histopathology
- Clinical Features
- Outcomes

ASYMPTOMATIC APICAL PERIODONTITIS WITH CYST FORMATION: RADICULAR CYST, CHRONIC APICAL PERIODONTITIS WITH CYST FORMATION
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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

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

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 cemento-enamel 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.[171] 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.[41,81,147] These growth factors may be released under certain conditions, since they have been shown to be associated with cementoblast proliferation, migration, and differentiation during cementum wound healing.[81]

The *periodontal ligament* is a soft, specialized connective tissue that connects the cementum to the alveolar bone. Periodontal ligament contains heterogeneous cell populations[128] and extracellular matrix (ECM). The cells of the periodontal ligament include osteoblasts, osteoclasts, fibroblasts, epithelial cell rests of Malassez, macrophages, cementoblasts, and undifferentiated mesenchymal cells (stem cells).[171] Fibroblasts, osteoblasts, and epithelial cells are differentiated cells that have retained the ability to undergo cell division and proliferation upon stimulation by appropriate signals. Multipotent stem cells of the periodontal ligament are capable of differentiating into cementoblast-like cells and periodontal ligament cells as well as osteoblasts.[102,161,209] The ECM of the periodontal liga-

ment 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's 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 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 Hertwig's epithelial root sheath that disintegrates after tooth development, are present in the periodontal ligament near the root surface in all teeth after root formation.[187] They are nests of epithelial cells connected as a network and surrounded by a basal lamina.[171] ERM are quiescent in normal periodontal ligament[171] but can be stimulated to proliferate in apical periodontitis.[139] They are believed to be the cellular source that when properly stimulated can form radicular cysts in certain apical periodontitis lesions.[139,171,178]

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.[171] Bone matrix contains IGFs, TGF-β, BMPs, FGF, and PDGF.[36,222] These growth factors are essential for osteoblast proliferation, migration, and differentiation during bone wound healing.[145]

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 immunoinflammatory reaction. Although microbial infection of the pulp in the root canals is the primary cause of apical periodontitis,[108,158] 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 byproducts, and disintegrated pulp tissue in the root canal system. These irritants are capable of inducing both innate and adaptive immune responses; they can activate either nonantigenic pathways or serve as antigens to activate adaptive responses. The host's immunoinflammatory responses are quite 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 immunoinflammatory 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, since bacterial toxins and noxious metabolic byproducts 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 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,[70] and this recognition has spurred considerable research into treating biofilms (see Chapters 9 and 15).

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%).[181] Most studies on the prevalence of apical periodontitis are from European and Scandinavian countries.[64,106,212] 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.[8] Apical periodontitis is a very prevalent problem.[65]

ETIOLOGY

The etiology, pathogenesis, and histopathology of apical periodontitis are very similar to that of marginal periodontitis (see also Chapter 15). 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 byproducts, chemical agents, mechanical irritation, foreign bodies, and trauma. Endogenous factors include the host's metabolic products, such as urate and cholesterol crystals, as well as cytokines or other inflammatory mediators that activate osteoclasts. 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.[108,158,232] The classic study of Kakehashi et al.[108] 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 trauma-

tized teeth with intact crowns and necrotic pulps, radiographic evidence of periradicular bone destruction was observed.[232] 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.[137,158] 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 byproducts that egress from the root canal system into the periapical tissues are capable of inducing a periapical immunoinflammatory reaction.[47,58,203,280] 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.[1,154] 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.[154] For example, LPS can stimulate sensory nerve fibers to release calcitonin gene–related peptide (CGRP) and substance P (SP)[55,96] to cause vasodilation and increased vascular permeability. LPS and lipoproteins can also activate TLRs on dendritic cells to stimulate T lymphocyte differentiation.[4] The common shared structural features of various toxins (i.e., PAMPS) are recognized by certain subtypes of TLRs. Since 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 byproducts 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, physical, or chemical injury.[148] In diseases caused by infection, bacteria are usually present in the involved tissues or organs,[265] 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,[123,162,267] except in certain cases of apical periodontitis associated with abscess formation,[179,262,274] a draining sinus tract,[83,181,272] or extraradicular endodontic infection.[231,255] One major current hypothesis is that apical periodontitis is triggered by entry into the periapical tissues of bacterial toxins, enzymes, and noxious metabolic byproducts.[240] 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 perhaps combinations of microorganisms in the periapical tissues.[235] 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.[181]

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 15).[233,235] 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.[156,236] A high prevalence of *E. faecalis* is frequently observed in filled root canals associated with persistent apical periodontitis.* These issues are described in greater detail in Chapter 15.

Physical (overinstrumentation, overfilling) and chemical (irrigants, intracanal medication, root canal filling materials) insults,[205] as well as traumatic injury[10] 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.[91,118,166,183,282] 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 because they cause granulocytes to develop phagocytic defect, loss of ammunition.[283] 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.[91]

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 and resulting infection.[265] 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.[265] 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.[265] 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

*References 87, 112, 156, 184, 218, and 236.

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 differentiation and activation.[21] 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.[152] 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, and natural killer (NK) cells with the former two types as the majority.[135,151] The characteristic features of innate and adaptive immunity are summarized in Table 14-1.

TABLE 14-1

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	Lymphocytes Neutrophils 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; and Abbas AK, Lichtman AH, Pober JS: *Cellular and molecular immunology,* ed 5, Philadelphia, Saunders, 2003.

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.[5,153] 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.[104] There are numerous microbial constitutive and conserved products, the pathogen-associated molecular patterns (PAMPs), also noted earlier. Importantly, the PRRs of the innate immune system recognize PAMPs.[5,153]

The specificity of innate immunity is due to the recognition of PAMPs of microorganisms by PRRs, also called *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.[104] 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*.[42] 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+).[94] In addition, LPS is shown to be capable of inducing pain via direct activation of TLR4/CD14 expressed on nociceptive sensory neurons.[264] 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*.[60,241] LTA also stimulates leukocytes to release inflammatory mediators, including TNF-a, IL-1b, IL-6, IL-8, and prostaglandin (PG) E2, which are known to play a role in various phases of the inflammatory response. All 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.[4,154] The innate

immune system is capable of recognizing nonself and self-antigens, while the adaptive immune system does not; thus, many autoimmune diseases are disorders of adaptive immunity.[154]

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.[12] Phagocytosed microbes or foreign particles are exposed to a very toxic environment containing specific and azurophil granules and oxygen-derived free radicals and are eventually degraded.[173] 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.[27,71] Besides their role in the innate immunity as professional phagocytes, macrophages also serve as antigen-presenting cells by expressing MHC 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.[1,208] 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 receptor or BCRs; also termed *immunoglobulins*). TCRs on T cells interact with antigens that are presented by 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^{18} and for BCR is approximately 10^{14} that generates the repertoire of different individual T and B cell clones.[1,105] 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 serves to reduce the potential for autoimmune disorders. Only those that do not interact with self-antigens are released into the lymphatic system and blood circulation. The naïve 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 activate 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 other 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.[1,50,261] 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, naïve CD4 T cells proliferate and differentiate into T_H0, which are subsequently committed to develop into T_H1 or T_H2 cells. Monocytoid DC (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. 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 comprise 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, since 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.[1,105]

Dendritic cells and macrophages phagocytose foreign antigens, while 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.

While 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.[44,285] 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.[1,105] 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.[242,266] Using RAG-2 SCID mice (both T- and B-cell-deficient), it was found that approximately a third of the RAG-2 mice developed endodontic abscesses, while no immunocompetent controls had abscesses.[244] 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.[95] Therefore, both T and B cells mediate the observed immune responses in apical periodontitis lesions.[152]

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*. 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[89] and local increases in inflammatory mediators that trigger neuropeptide release, leading to neurogenic inflammation.[23,38] 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.[225] 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, suggests a complex interplay between these molecules in the immune response.[77,189,225]

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.[38]

The major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis caused by root canal infection is illustrated in Fig. 14-1.

DIAGNOSIS

Correlation Between Clinical and Histologic Findings

Clinical diagnosis of inflammatory periapical disease is mainly based on clinical signs and/or symptoms, duration of disease, pulp tests, percussion, palpation, and radiographic findings. 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 upon signs, symptoms, and testing results, whereas the histologic diagnosis is a definitive diagnosis of tissue disease.

Similar to pulpitis,[206] 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,[89,90] other mediators such as endogenous opioids and somatostatin released by inflammatory cells during inflammation are able to inhibit firing of sensory nerve fibers.[93,211] 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.[24,143,213] For example, many teeth with apical periodontitis are free of symptoms.

Correlation Between Radiographic and Histologic Findings

Radiography is designed to detect pathologic changes at tissue not cellular levels. Even using very 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

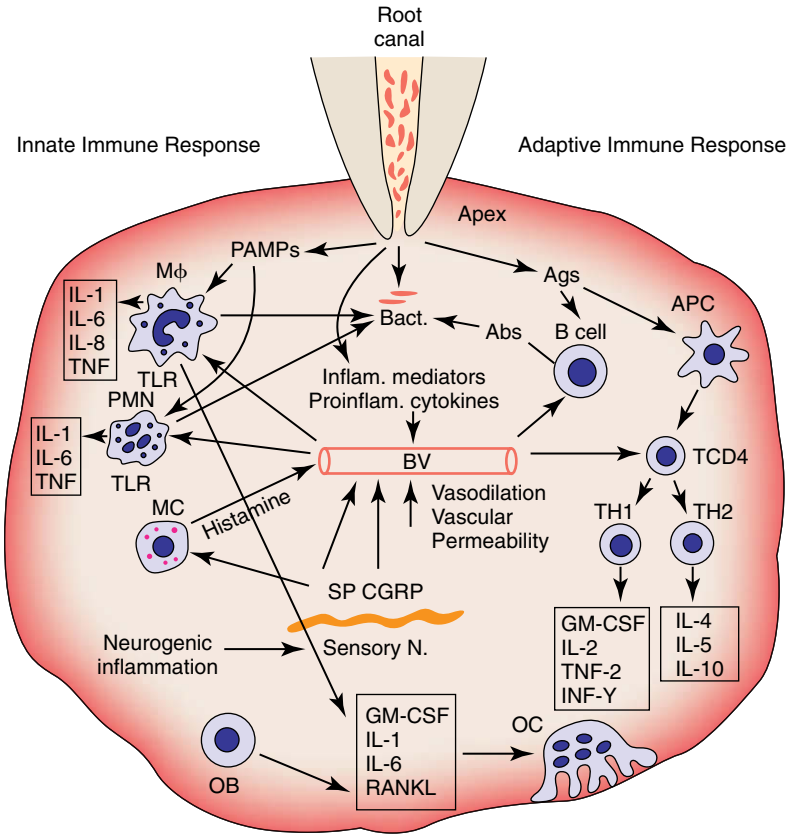


FIG. 14-1 Major innate and adaptive immune responses and neurogenic inflammation in the pathogenesis of apical periodontitis. *APC,* Antigen presenting cell; *GM-CSF,* granulocyte/monocyte colony-stimulating factor; *MC,* mast cell; *Mϕ,* macrophage; *OB,* osteoblast; *OC,* osteoclast; *PAMPs,* pathogen-associated molecular patterns; *PMN,* polymorphonuclear leukocyte; *RANKL,* receptor activator of nuclear factor κB ligand; *TKR,* toll-like receptor. *(Courtesy Dr. Lin.)*

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.[15,32,78] This finding is supported by the fact that lesions localized in the cancellous bone may not be visible radiographically unless they involve cortical bone.[17,19,98] In addition, radiographic findings are unable to predict asymptomatic apical periodontitis (granuloma) from asymptomatic apical periodontitis with cyst formation (radicular cyst).[125,238] 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. 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 and/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 is focused 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.[148]

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.[278] There are also many classifications of inflammatory periradicular disease in several endodontic textbooks[99,181,253] 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. Since the focus of this chapter is on pathobiology of the periapex, 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 4).

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 immunoinflammatory 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.[121,148,220,270] Acute immunoinflammatory response is characterized by participation of neutrophilic leukocytes and chronic immunoinflammatory 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 immunoinflammatory responses.[121,148] Acute and chronic immunoinflammatory responses are in fact not rigid phases of a single programmed event but two overlapping responses with partially different triggering mechanisms and programs.[148]

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[89]; and (2) animal and human studies that concluded that uncontaminated necrotic pulps which are intentionally devitalized or accidentally traumatized are generally incapable of inducing periapical inflammation unless they are infected.[137,158,232] 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. 14-2).* For example, Kovacevic et al.[119] studied the transition from pulpitis to apical periodontitis in dogs' teeth by artificial exposure of pulps to the oral cavity and observed that pulpitis was coupled with an acute apical periodontitis. Similarly,

*References 119, 140, 142, 192, 193, and 239.

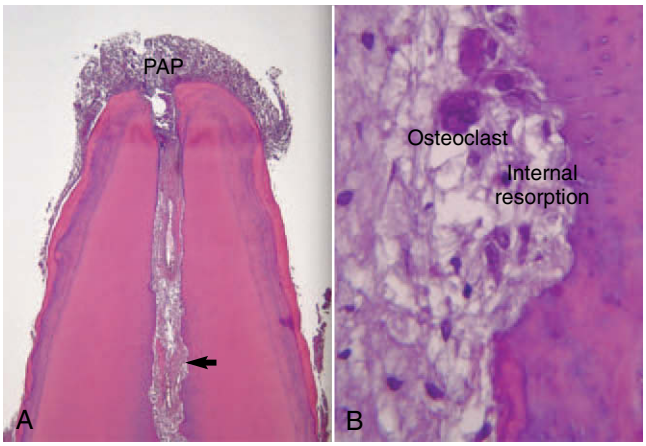


FIG. 14-2 A, Inflammation of the pulp tissue in the apical root canal extends into the periapical tissues in a mature tooth (H & E, ×100). **B,** Arrow in **(A).** High magnification of the pulp tissue in the apical root canal in **A.** The pulp tissue is vital and infiltrated with chronic inflammatory cells. Note resorption of canal wall and multinucleated clast cells (H & E, ×200). *(Courtesy Dr. Domenico Ricucci, Rome, Italy.)*

Kimberly and Byers[115] 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 et al.[279] and Stashenko et al.[226] 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[140] 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 consists of vasodilatation, 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 byproducts.[121,148]

Cell Biology

The immunoinflammatory 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 immunoinflammatory 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.[1,121,148] *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, 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).[1,26,121,148] 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.[1,33,114]

Endothelial Cells

Endothelial cells are important players in the immunoinflammatory response. Without 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.[1,121,148] 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.[1,121,132,148] 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.[3,111,180] 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.[114,121,148] 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.[148] 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.[1,121,148,220]

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 their 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.[82,121,148] 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.[121,148] 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.[121,148] 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.[148] 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 adhe-

sion 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.[148]

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.[121,148,220] 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.[148] Tissue macrophages are not terminally differentiated cells and are able to undergo mitosis. Their lifespan is from several weeks to months.

Activated neutrophilic leukocytes and macrophages are very 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.[1,121,148] 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.[1,121,220] 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.[1,121,148,220] Some antimicrobial peptides and other oxygen-independent mechanisms possessed by phagocytes are specialized for killing of certain groups of microbes.[1,220]

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.[1,148]

Platelets

Platelets normally circulate in the blood but also play an important role in inflammation. They are small cytoplasmic fragments derived from the megakaryocyte.[114] Platelets are essential for blood clotting, hemostasis, and fibrinolysis. Platelets produce vasoactive amines (PAF, serotonin), chemokines, and growth factors (PGDF, FGF, TGF) during inflammation.[148]

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.[196,221] 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.

Inflammatory Mediators

Numerous biochemical mediators are involved in the acute innate immunoinflammatory response. They are mainly derived from 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 immunoinflammatory response are listed in Table 14-2.

All listed inflammatory mediators have been shown to be present in apical periodontitis.[249,250] 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.[121,148]

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

TABLE 14-2

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 tissue

CGRP, Calcitonin gene–related peptide; *fMLP,* formyl-methionyl-leucyl-phenylalanine; *IL-1,* interleukin-1; *PAF,* platelet activation factor; *NK cell,* natural killer cell; *SP,* substance P; *TNF,* tumor necrosis factor.

Data from Kumar V, Abbas AK, Fausto N, et al: *Robbins and Cotran pathologic basis of disease*, ed 8, Philadelphia, Saunders, 2010; Slauson DO, Cooper BJ: *Mechanisms of disease*, ed 3, St Louis, Mosby, 2002; Majno G, Joris I: *Cell, tissues, and disease*, ed 2, Oxford, Oxford University Press, 2004; Abbas AK, Lichtman, Pober JS: *Cellular and molecular immunology*, ed 5, Philadelphia, Saunders, 2003.

anaphylatoxins which 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.[121,148]

Besides inflammatory mediators, the immunoinflammatory 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.[1,84,148] 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.[224]

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.[1,84,214] 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 immunoinflammatory process, are described in detail in Neurogenic Inflammation in this chapter. Inflammatory mediators such as histamine, kinins, prostaglandins, and proinflammatory cytokines are capable of sensitizing and activating sensory nerves to release neuropeptides.[26,89,90,191,202]

Histopathology

The acute immunoinflammatory 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.[3,180] In addition, sprouting of sensory nerve fibers has been shown early in the inflamed periapical tissues.[35,115] 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.[140,192,193,226,279] 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 et al.[226] 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.[113] Pain is induced by sensitization and activation of nociceptive sensory nerve fibers by inflammatory mediators, proinflammatory cytokines, nerve growth factor, and pressure.[113] Sprouting of sensory nerve fibers in inflamed periapical tissues could also increase receptive field size in teeth with apical periodontitis.[34,35] Radiographic examination usually does not show periapical bone destruction of 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 immunoinflammatory response is to restore the structural and functional integrity of damaged tissue by eliminating irritants as soon as possible.[270] 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.[148] 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.[121] It is a morphologic variation of acute and chronic inflammation.[121,148,270] 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.[29,179,234,262] 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 deprivation of oxygen and interference of respiratory burst. The purpose of respiratory burst is to generate bactericidal agents.[12,148] However, the phagocytic activity of leukocytes is not impaired in aerobic conditions.[148]

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 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. 14-3).[39,193,205]

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 very 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

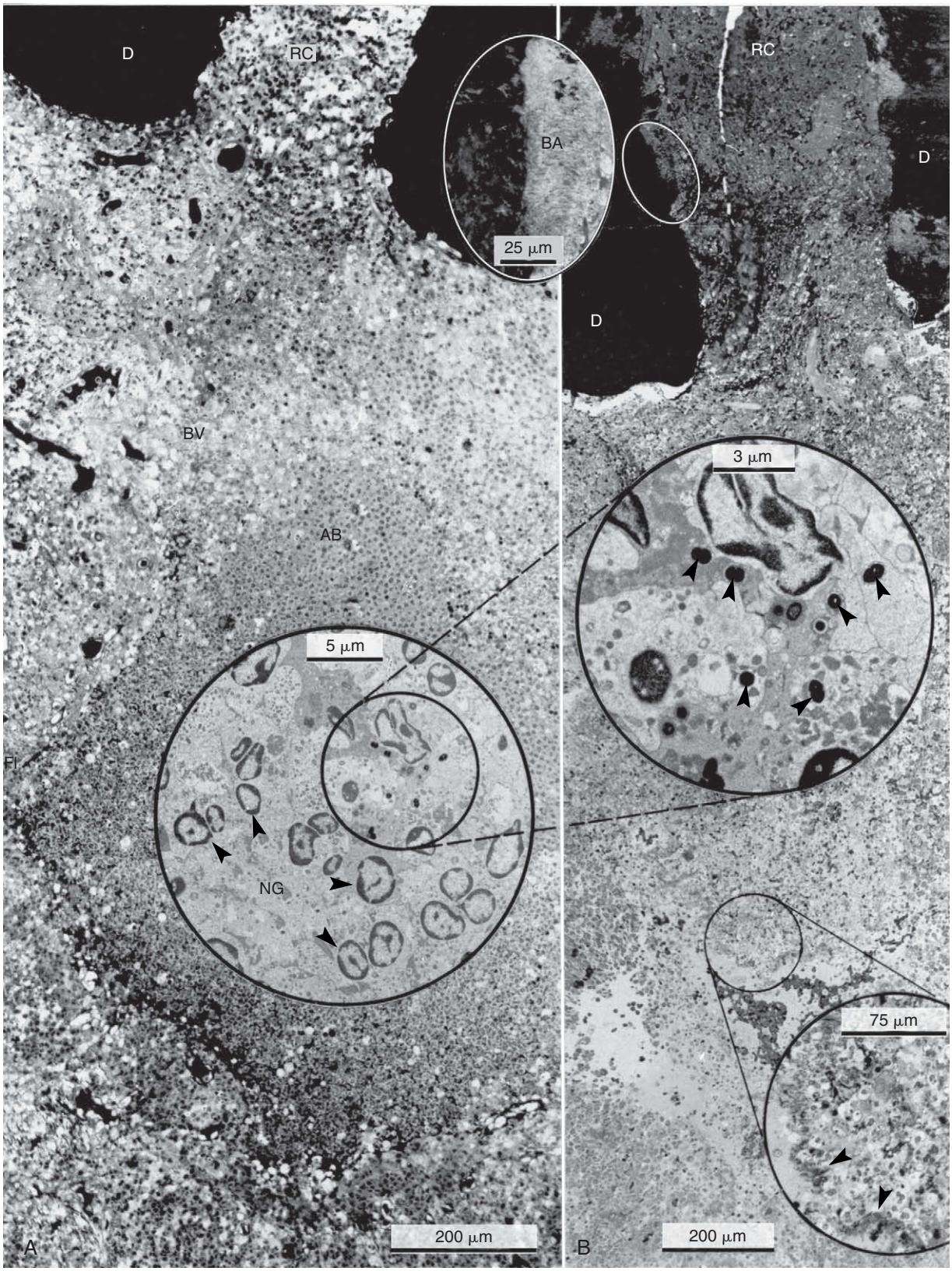


FIG. 14-3 Structure of a secondary periapical abscess. **A,** Axial section of an abscessed apical periodontitis. The microabscess (AB) contains a focus of neutrophils (NG inset in **A**). Note the phagocytosed bacteria in one of the neutrophils (further magnified in large inset in **B**). A secondary 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 *(arrowhead in lower inset). BV,* Blood vessels; *D,* dentin. (**A** ×130; **B** ×100; oval inset, ×400; inset in **A,** ×2680; upper inset in **B,** ×4900; lower inset in **B,** ×250.) *(From Nair PNR:* Periodontol *2000 13:121, 1997.)*

by inflammatory mediators and sensitization to mechanical stimuli due to increased interstitial pressures.[207] 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 15).[126,205]

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.[121,148,220,270] 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.[148]

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, *asymptomatic chronic apical periodontitis* and *periapical granuloma* are terms used interchangeably. A granuloma is a focal area of *granulomatous inflammation,* which is a histologic term for a chronic inflammatory reaction.[121,148,258] 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.[92,121,148,275] The presence of poorly digestible irritants (nonantigenic or antigenic), T cell–mediated immunity to irritants, or both appears to be necessary for granuloma formation.[1,121,148] A granuloma is relatively avascular,[121,148] whereas a chronic apical periodontitis is very vascular. Histologically, some but not all chronic apical periodontitis lesions may show some features of granulomatous inflammation,[121,148] 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.[121,148]

The foreign body reaction is a specific subtype of chronic inflammation.[121,148,270] Foreign materials such as root canal filling materials, paper points, cotton fibers, and surgical sutures can trigger a foreign body giant cell granuloma.[118,166,282] 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.[148] In addition, foreign bodies can favor infection in several ways, since they can be a source of bacterial biofilm[45,176] 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 10^8 bacteria (i.e., a million-fold increase in dose) fails to produce an abscess in normal guinea pig skin.[284] 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.[107,148]

Cell Biology

Macrophages and Lymphocytes

Macrophages and lymphocytes are the primary players in asymptomatic apical periodontitis.* Lymphocytes are bloodborne and have counterparts in the connective tissue.[148] 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 Cell

Dendritic cells play a vital role in asymptomatic apical periodontitis. They have been shown to be present in apical periodontitis lesions in rats.[109,180] Dendritic cells are accessory immune cells derived from bone marrow stem cells and may

*References 46, 73, 117, 229, 230, 252, and 281.

be related to the mononuclear phagocyte lineage.[1] They function as antigen-presenting cells for naïve 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 are decreased,[268] 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.[268] The stationary phase of bone resorbing activity was correlated to asymptomatic apical periodontitis.[268] Increased expression of bone resorptive cytokines such as IL-1, IL-6, and TNF was related to the period of active bone resorption.[269] 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.[227]

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 differentiation.[59,131,159,186,243] 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.[114]

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.[169] 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.[75] 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.[13,14,25,243] (See Fig. 14-5 for the mechanism of bone resorption by osteoclasts in apical periodontitis.)

The resorption of root cementum and/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*.[197] It has been shown in ultrastructure and gene expression studies that odontoclasts and osteoclasts are similar.[198,199] Therefore, it is believed that the cellular mechanisms of bone, cementum, and dentin resorption are similar.[198] 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. 14-4.

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.[164] It is a form of pathologic (inflammatory) hyperplasia. This type of hyperplasia is caused by stimulation of growth factors and cytokines produced during the immunoinflammatory response. Hyperplasia is a self-limiting process and is reversible when the causative stimulus is eliminated.[121,148,220] 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,[31] proinflammatory cytokines,[80,155,201] and growth factors* are capable of stimulating epithelial cell rests to proliferate (Fig. 14-5). The extent of cellular proliferation appears to be related to the degree of inflammatory cell infiltration.[192,193]

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.[114] 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.[121,148] 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.[148,258]

Fibrovascular granulation tissue is also a prominent feature of chronic apical periodontitis as a repair process. Migration

*References 40, 72, 100, 144, 157, 177, and 247.

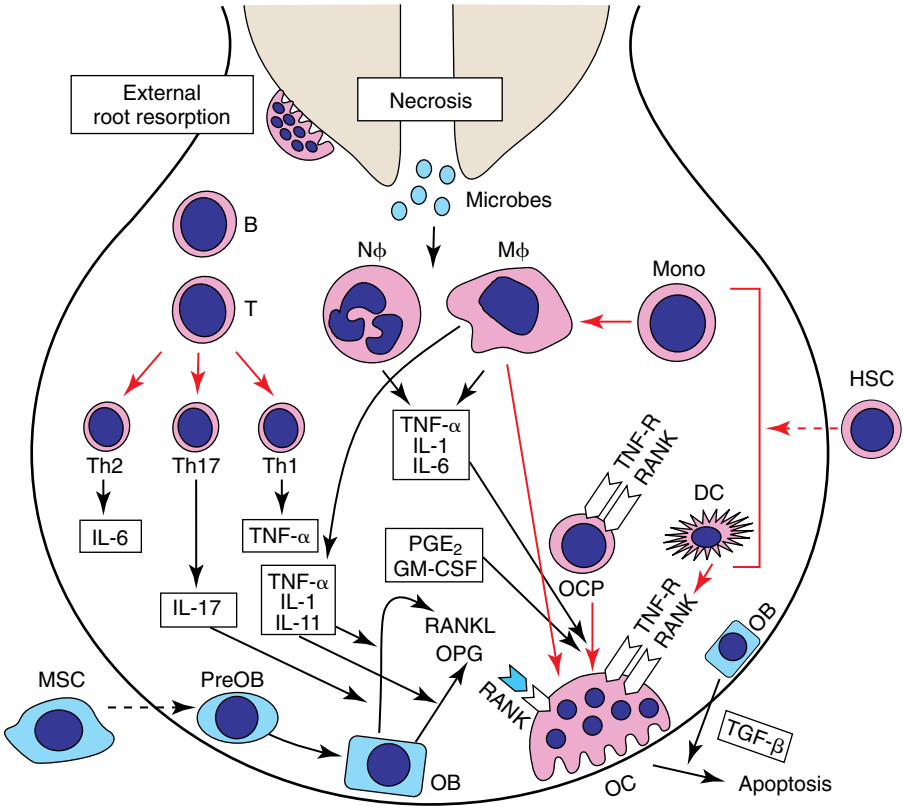


FIG. 14-4 Bone resorption by osteoclasts in apical periodontitis. *DC*, Dendritic cell; *HSC*, hematopoietic stem cell; *Mϕ*, macrophage; *MSC*, mesenchymal stromal cell; *Nϕ*, neutrophil; *OB*, osteoblast; *OC*, osteoclast; *OCP*, osteoclast precursor; *OPG*, osteoprotegerin; *RANK*, receptor activator of nuclear factor κB; *RANKL*, receptor activator of nuclear factor κB ligand. *(Courtesy Dr. Huang.)*

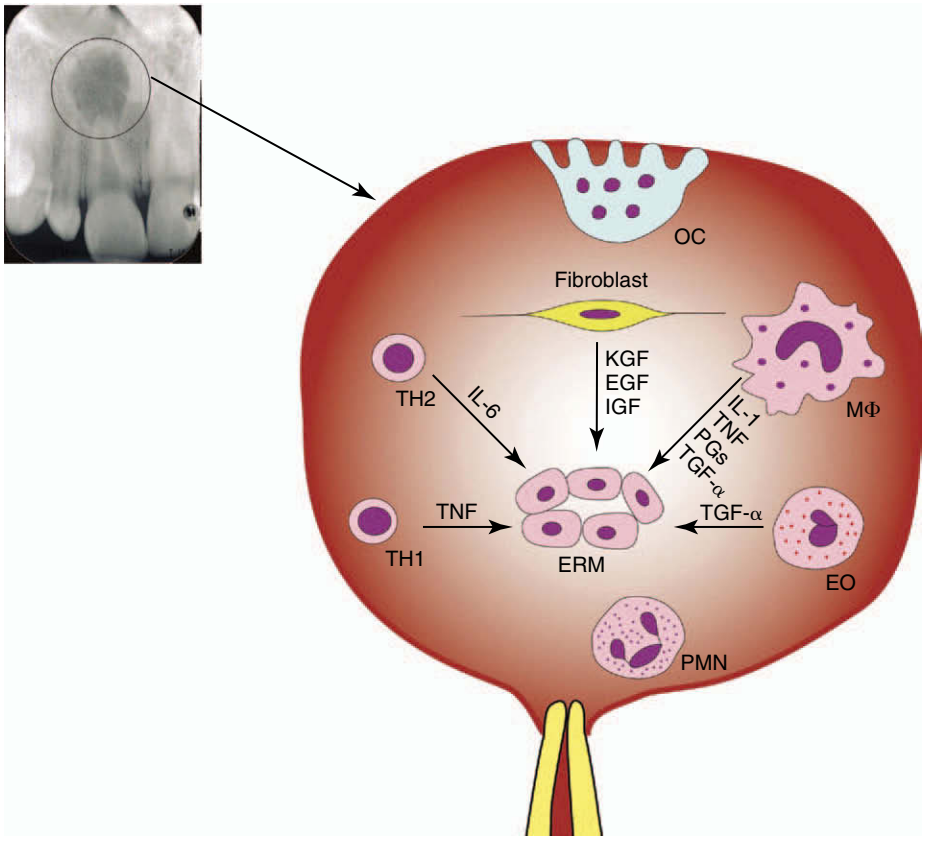


FIG. 14-5 Schematic illustration of the major mechanisms that activate proliferation of epithelial cell rests in apical periodontitis. *(From Lin LM, Huang GT-J, Rosenberg P: Proliferation of epithelial cell rests, formation of apical cysts, and regression of apical cysts after periapical wound healing.* J Endod *33:908, 2007.)*

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.[148,220] Neovascularization supplies oxygen and nutrients 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 immunoinflammatory response described earlier.

Histopathology

Macrophages and lymphocytes are predominant cells in asymptomatic apical periodontitis lesions (Fig. 14-6). 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.[30,210,257] Bone resorption is a hallmark of asymptomatic apical periodontitis. Multinucleated osteoclasts may sometimes be seen in resorptive Howship's 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. 14-6, *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 et al.[216] 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. 14-7).[141,150] 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.[127,263] Fortunately, cementum and dentin appear to be less readily resorbed than bone by inflammatory processes.[85] 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.[2,101,162,255,271] Inadvertent contamination is a major concern when culturing bacteria from these lesions. Under meticulous transmission electron microscopic examination, Nair[162] 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. 14-8).[140] 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 18 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.[139,168,178,245] 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 neoplastic lesion in the WHO Histological Typing of Odontogenic Tumors, Jaw Cysts and Allied Lesions.[120] The prevalence of radicular cysts in apical periodontitis lesions from extracted teeth ranges from 15% to 20%.[164]

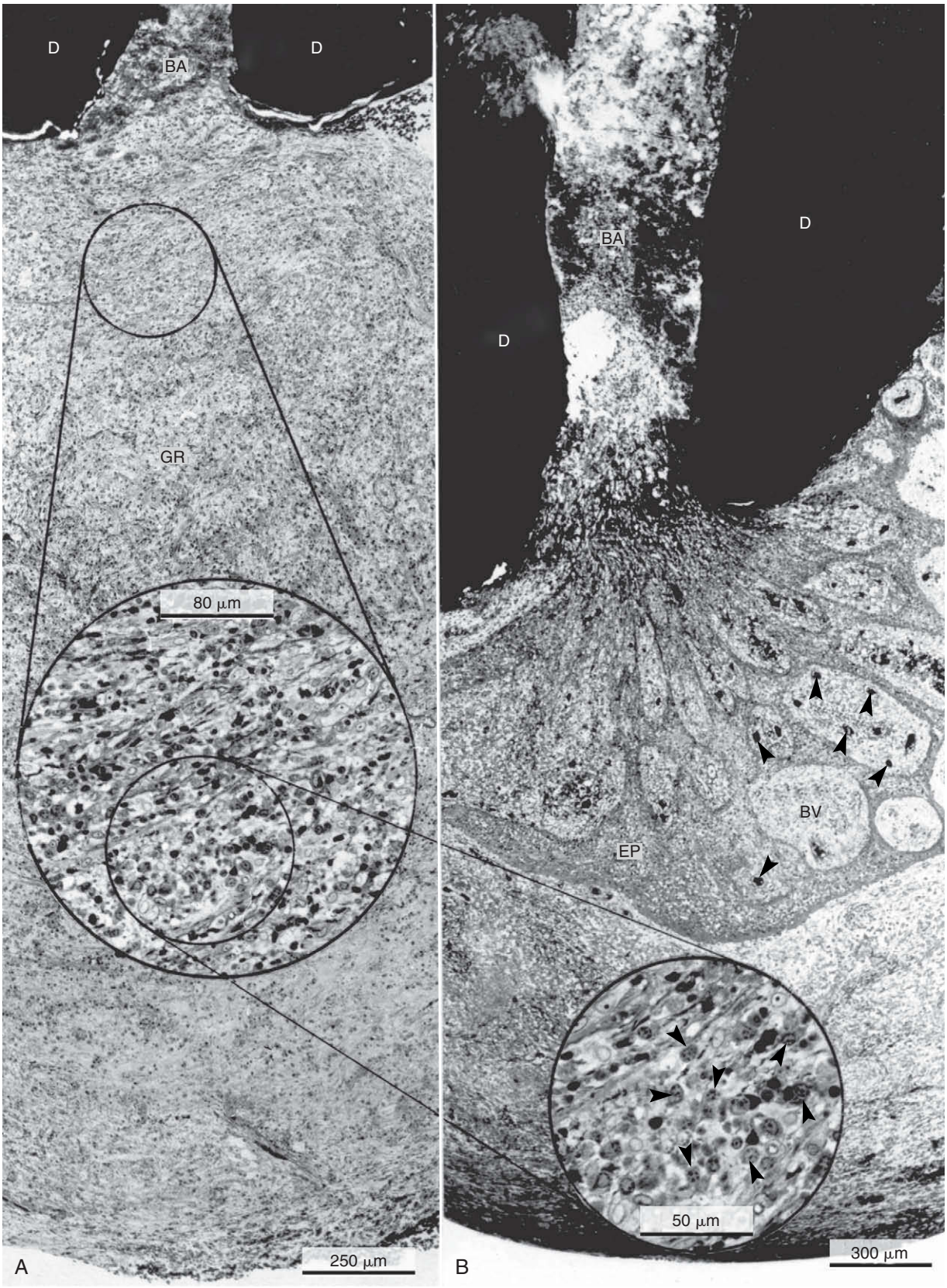


FIG. 14-6 Chronic asymptomatic apical periodontitis without **(A)** and with **(B)** epithelium (EP). The root canal contains bacteria (BA) in **A** and **B.** The lesion in **A** has no acute inflammatory cells, even at the mouth of the root canal with visible bacteria at the apical foramen (BA). Note the collagen-rich maturing granulation tissue (GR) infiltrated with plasma cells and lymphocytes (inset in **A** and **B**). *BV,* Blood vessels; *D,* dentin. (**A** ×80; **B** ×60; inset in **A,** ×250; inset in **B,** ×400.) *(From Nair PNR:* Periodontol *2000 13:121, 1997.)*

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.



FIG. 14-7 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, ×1600). *(From Lin L, Langeland K: Innervation of inflammatory periapical lesions.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *51:535, 1981.)*

They can be stimulated to divide symmetrically and asymmetrically into basal cells and suprabasal squamous cells of lining epithelium of radicular cysts.[139] 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.[245] 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.[168,178] 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.[139] 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,[248] but this hypothesis overlooks the cellular aspects of cyst growth and the biochemistry of bone destruction.[66,155] 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[246] and bone resorption. The ERM have also been shown to be capable of secreting a bone resorbing factor.[22] Most inflammatory mediators and proinflammatory cytokines that stimulate proliferation of epithelial cell rests also mediate bone resorption in apical periodontitis lesions.

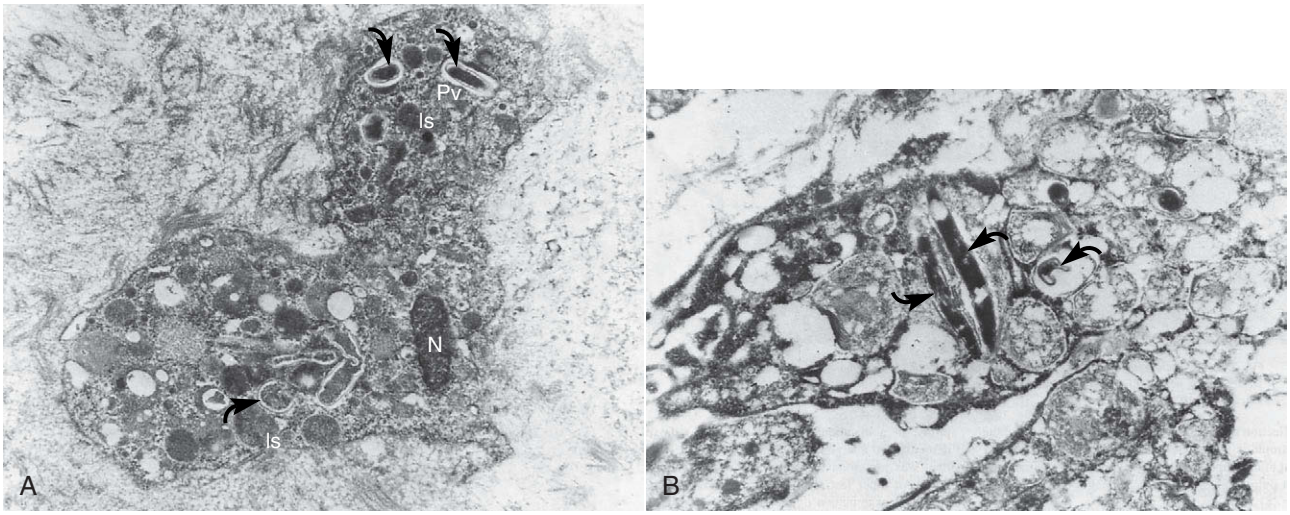


FIG. 14-8 Bacteria phagocytosed by phagocytes in chronic apical periodontitis lesion. **A,** Bacteria *(arrows)* in phagosome of a neutrophil. *ls,* Lysosome; *N,* part of nucleus; *pv,* phagocytic vacuole (EM, ×6300). **B,** Part of the cell structure of a macrophage. Note longitudinally and cross-cut bacteria cells with unit membrane *(arrows)* in phagocytic vacuoles (EM, ×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.)*

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. 14-9).[164,215] 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. 14-10).[164,215] 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, indicating that inflammatory cells are attracted to these tissues by chemotactic irritants, either in the root canal system and/or in the periapical tissues. In nonproliferating epithelium, there is less inflammatory cell infiltration.[39,205] Occasionally, mucous cell metaplasia or ciliated respiratory-type cells are present in cystic epithelial lining.[174] 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 and adaptive immune cells during periapical inflammation. The lumen of cysts may contain inflammatory exudates, cholesterol crystals, clear fluid, or bacterial colonies (Fig. 14-11; Fig. 14-12).[164,193]

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.

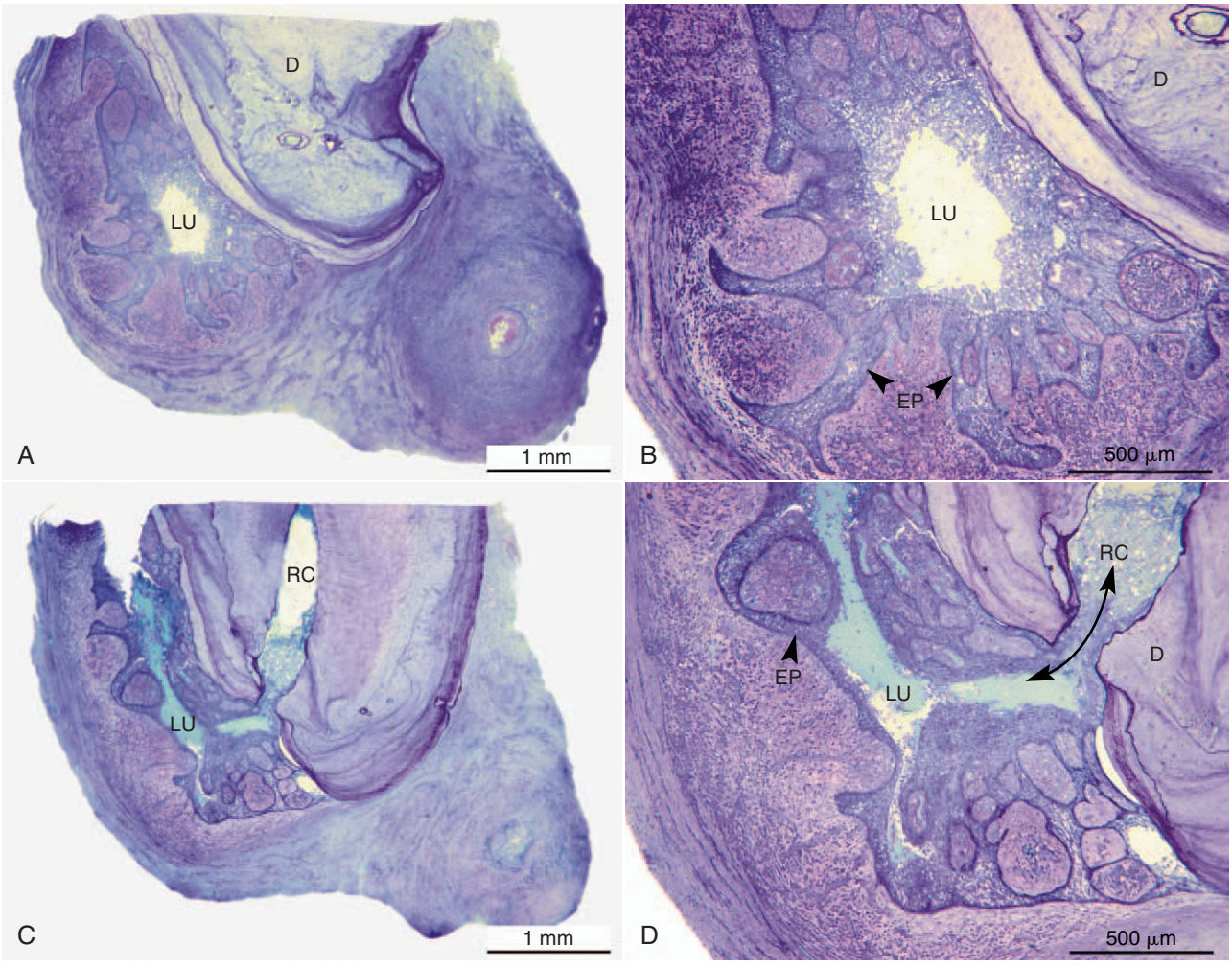


FIG. 14-9 A, A well-developed pocket cyst in apical periodontitis lesion. **B,** Note the saclike epithelial lumen. **C-D,** Sequential axial serial section passing through the apical foramen in the root canal plane (RC) shows the continuity of the lumen with the root canal. *D,* Dentin (**A, C,** ×16; **B, D,** ×40). *(From Nair PNR:* Endod Topics *6:96, 2003.)*

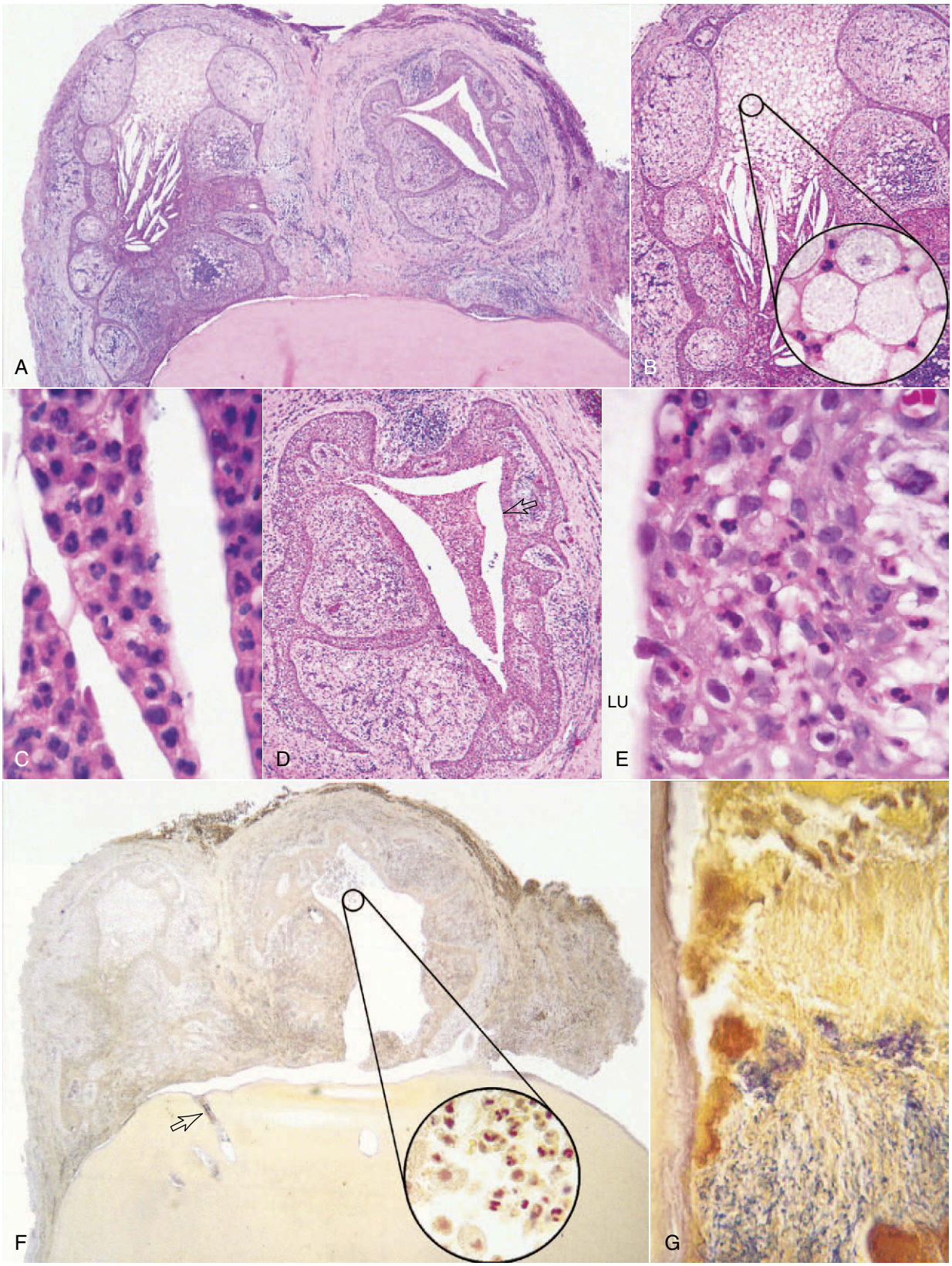


FIG. 14-10 A, Two distinct cysts lined by epithelium in apical periodontitis lesion. There is no evidence of communication with the foramen in serial sections (H & E, ×25). **B,** Cyst cavity on the left side in **A.** Accumulation of foamy macrophages (H & E, ×50; inset ×1000). **C,** Lower part of cavity in **B.** Dense infiltration of neutrophilic leukocytes (H & E, ×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, ×50). **E,** Cystic wall in **D.** Cyst's lining epithelium is infiltrated with acute and chronic inflammatory cells. *Lu,* Lumen (H & E, ×1000). **F,** Foamy macrophages, neutrophilic leukocytes, and necrotic tissue inside the cyst *(inset)*, but no bacteria (Brown & Brenn, ×25, inset ×1000). **G,** Area indicated by an *open arrow* in **F.** Bacterial colonies in the apical foramen (Brown & Brenn, ×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.)*

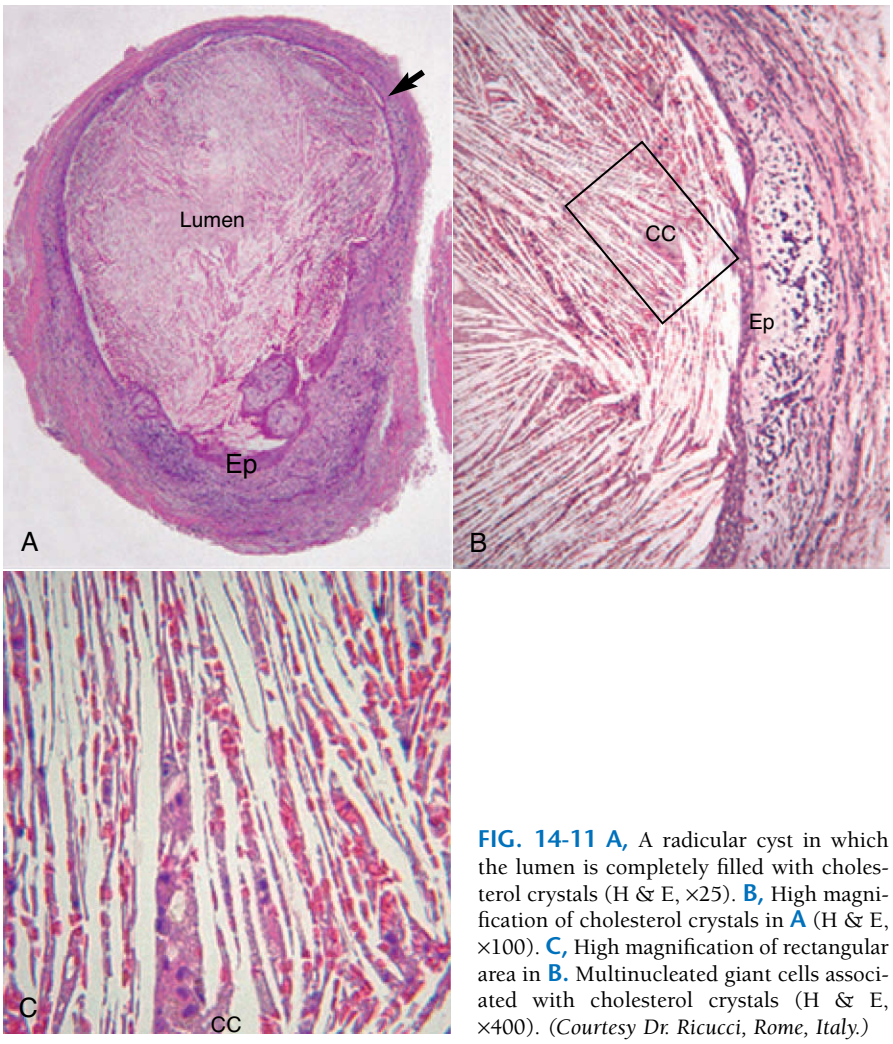


FIG. 14-11 A, A radicular cyst in which the lumen is completely filled with cholesterol crystals (H & E, ×25). **B,** High magnification of cholesterol crystals in **A** (H & E, ×100). **C,** High magnification of rectangular area in **B.** Multinucleated giant cells associated with cholesterol crystals (H & E, ×400). *(Courtesy Dr. Ricucci, Rome, Italy.)*

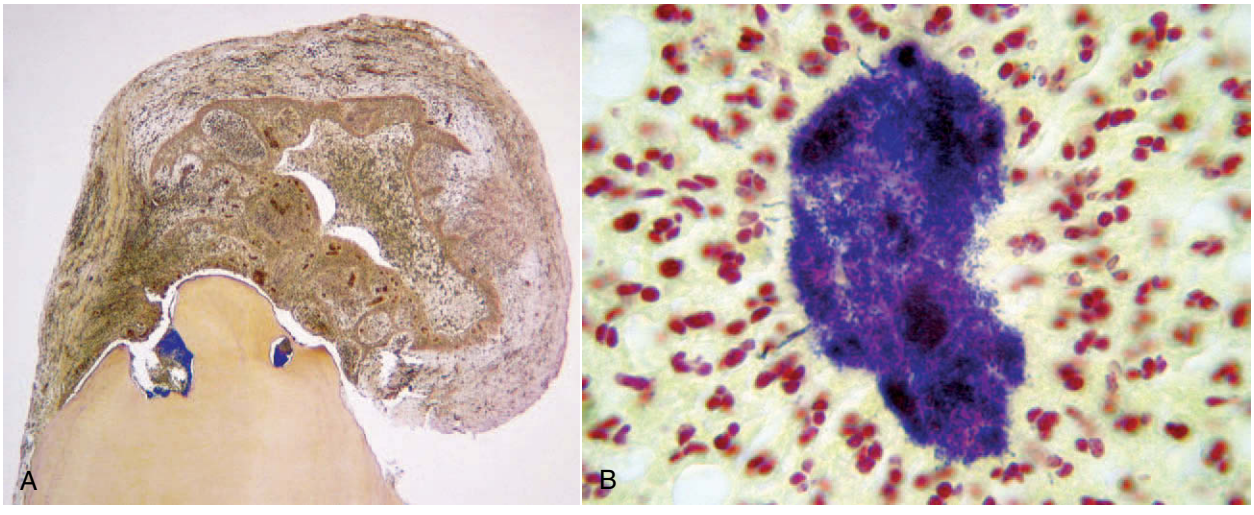


FIG. 14-12 A, An apical true cyst in apical periodontitis lesion. Bacterial colonies are seen in the foraminal areas (Brown & Brenn, ×25). **B,** Bacterial colonies in the cyst lumen, surrounded by inflammatory cells (Brown & Brenn, ×400). *(From Ricucci D, Bergenholtz G: Histologic features of apical periodontitis in human biopsies.* Endod Topics *8:68, 2004, Fig. 6.)*

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, regardless of whether preobturation bacterial cultures are negative or positive.[219] Therefore, it is speculated that some cysts, especially pocket cysts, may heal after nonsurgical root canal therapy.[163] Apical true cysts are less likely to heal after nonsurgical root canal therapy because of their self-sustaining nature; surgical intervention is necessary.[163] 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.[174,188] 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 is decreased.[268] 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).[114]

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 which 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.[39,174,188]

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.[63]

PERIAPICAL LESIONS OF NONENDODONTIC ORIGIN

There are many periapical lesions that 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,[9] foreign bodies,[118,166,282] host metabolic byproducts,[167] advanced periodontal disease,[124] fibro-osseous lesions, and benign and malignant tumors.[174] 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.[162,217,231,255] 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, since 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 lack of blood circulation and areas resistant to access (e.g., isthmi; see Chapter 7). In our opinion, the independent extraradicular infection probably does not occur often and is usually associated with apical actinomycosis.[88,165,237] Bacteria have been observed as biofilm on the apical external root surface.[217,254] In addition, viruses have also been isolated from apical periodontitis lesions.[133,196,221] If microbiologic root canal cultures are not completely reliable (because bacteria may remain in the complex root canal system),[200] 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?[29,83,179,234,262] 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. Systemic conditions affecting the severity of inflammatory periapical lesions have been documented and include diabetes, smoking, hypertension, cytokine gene polymorphisms, and nonspecific disorders of the immune system. In general, diabetic patients are prone to infections due to generalized vascular disorders caused by accumulation of atheromatous plaques in the vascular walls, particularly of capillaries. Atherosclerotic vessels cause impaired leukotactic response and failure in delivering humoral and cellular components of the immune system.[18] Clinical and radiographic studies have shown that there is a greater prevalence of periapical lesions in diabetics than in nondiabetics.[18] Type 2 diabetes mellitus is significantly associated with an increased prevalence of apical periodontitis.[204] Other findings demonstrated that type 2 diabetes is associated with increased risk of a poor response by the periradicular tissues to odontogenic pathogens.[28] In rodent models, diabetic conditions enhance the development of periradicular lesions and increase mortality.[67,103,116] Mice deficient in P/E-selectin are shown to be more prone to infection-mediated early-onset periodontal disease.[175]

Genetic polymorphisms may also play a role in the individual's response to various diseases. Haplotype analyses indicated that a variant of the IL-1β gene was likely to be most important for early-onset periodontitis risk.[56] 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.[134] 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.[146] Those displaying high-producer *IL6* genotype, intermediate and high-producer *IL1B* genotypes, and low-producer *TNFA* genotype tend to exhibit symptomatic dental abscesses.[54]

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.[97] 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.[16,51,52] Microorganisms in the blood circulation are rapidly eliminated by the host's humoral and cellular defense components within minutes.[16,52] However, bacteremia may pose a potential danger to immunocompromised patients or patients with congenital heart valve disease.[51] No correlation between apical periodontitis and coronal heart disease and rheumatoid arthritis has been observed.[37,160] There is insufficient evidence to support that apical periodontitis could serve as a focus of infection and cause significant systemic diseases.[62,182]

WOUND HEALING OF APICAL PERIODONTITIS

Periapical Wound Healing After Nonsurgical Root Canal Therapy

Understanding of 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 9 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 reduction of immunoinflammatory 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 (e.g., opioids, somatostatin, glucocorticoids, etc.); (3) relative balance between intracellular levels of cyclic AMP and cyclic GMP; (4) the antiphlogistic role of histamine; (5) inhibitors of the complement system[258]; and (6) antiinflammatory cytokines, such as IL-4, IL-10, IL-13 and TGF-β.[86,172] In addition, the major cellular inducer of inflammation, neutrophilic leukocytes, undergo apoptosis,[82] and the major cellular player of wound healing, macrophages, secrete antiinflammatory molecules.[172]

The process of wound healing is tightly regulated by cell-to-cell and cell-to–extracellular matrix interactions, as well as

TABLE 14-3

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

Modified from Majno G, Joris I: *Cell, tissues, and disease*, ed 2, Oxford, Oxford University Press, 2004; Slauson DO, Cooper BJ: *Mechanisms of disease*, ed 3, St Louis, Mosby, 2002; Werner S, Grose R: Regulation of wound healing by growth factors and cytokines. *Physiol Rev* 83:835, 2003.

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.

the temporal and spatial expression of a variety of cytokines, growth factors, and neuropeptides and apoptosis (Table 14-3).[26,79,82,228,273] 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.[69,108,251] 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,[11,129,130] no studies of wound healing of apical periodontitis lesions with cyst formation after nonsurgical root canal therapy exist in the literature.

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 and/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 stromal cells in alveolar bone and multipotent stem cells in periodontal ligament.[209] During periapical wound healing, many unwanted hyperplastic cells (e.g., endothelial cells, fibroblasts, epithelial cells) are deleted by apoptosis,[53,79,82] and the extracellular matrix is remodeled by metalloproteinases. Pathologic processes such as extensive fibrosis do not occur, and the damaged periapical tissues can be ideally restored 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 initial histogenesis 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 periodontal ligament, new cementum, and new alveolar bone.[61,256] Both nonsurgical root canal therapy and guided tissue regeneration therapy are able to remove irritants and provide a favorable environment 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 Hertwig's epithelial root sheath (i.e., enamel matrix proteins) are required for cementoblast differentiation from ectomesenchymal stem cells in the dental follicle during tooth development.[171] The cells of Hertwig's epithelial root sheath are absent in mature teeth.[147,223]

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.[81,209] 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.[170,195,260] 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.[209]

Bone has a remarkable capacity for regeneration in response to injury. During periapical wound healing, the osteoblasts 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.[6,145] When one of the cortical bone plates (buccal or lingual/palatal) is destroyed, mesenchymal cells in the inner layer of perios-

teum 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.[114,145] 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.[11] 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.[48,49] The cellular and molecular mechanisms of excess scar tissue formation in periapical wound healing is not completely understood. Growth factors/cytokines may play an important role in regulating fibroblast gene expression and excess scar tissue formation.[259]

The newly regenerated periodontal ligament will finally undergo remodeling into mature periodontal ligament, with one group of collagen fibers (Sharpey's 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 quite similar, but the kinetics of healing of the periapical wound after endodontic surgery is much faster than nonsurgical endodontic therapy.[122] In surgical endodontic therapy, a surgeon performs removal of irritants, such as necrotic cells, tissue debris, and bacteria in the periapical lesions, which is called *surgical débridement*. In contrast, in nonsurgical endodontic therapy, activated macrophages perform bacterial killing and cleanup of periapical lesions, which is called *biologic débridement*.[138] Surgical débridement is very effective and of course quite rapid, whereas biologic débridement 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.[139] In contrast, apical true cysts may be less likely to heal after nonsurgical root canal therapy because of their self-sustaining nature.[163] Histologically, inflammatory cell infiltration is always present in the lining epithelium and/or fibrous connective tissue capsule of apical true cysts.[164,192,193] 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 and/or fibrous connective tissue capsule.[164,192,193] 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 very 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.[148,265] 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.[139] 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 offer tantalizing clinical implications and clearly support 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

Local and systemic factors may affect periapical wound healing. Infection will complicate and prevent wound healing, foreign bodies can impair wound healing,[166,282] and nutrition can also affect wound healing.[276] Diabetes was reported to reduce the likelihood of healing of apical periodontitis lesions after nonsurgical root canal therapy.[68] 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.[149] However, immunocompromised patients, such as HIV patients, responded as well as counterpart patients after nonsurgical endodontic

therapy.[43,76,185] Although smoking has not been shown to be associated with increased incidence of apical periodontitis and prognosis of nonsurgical root canal therapy,[20,57] smoking may increase complications of periapical surgery, such as pain and swelling.[74]

Patients receiving radiotherapy of jaws and bisphosphonate therapy have a risk of developing osteonecrosis of jawbones,[190,194,277] so nonsurgical root canal therapy is recommended for these patients.[110,136] 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.[7] For asymptomatic patients who have taken an oral bisphosphonate for less then 3 years and have no clinical risk factors, endodontic surgery is not contraindicated.[7] Nevertheless, the patient's physician should be consulted. Any kind of surgical procedures should be avoided for patients receiving intravenous bisphosphonate.[7] 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 initiation of radiation or bisphosphonate therapy.[7]

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CHAPTER 15

Microbiology and Treatment of Endodontic Infections

JOSÉ F. SIQUEIRA, JR AND ISABELA N. RÔÇAS

CHAPTER OUTLINE

APICAL PERIODONTITIS AS AN INFECTIOUS DISEASE
ROUTES OF ROOT CANAL INFECTION
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- Biofilm Community Lifestyle
- Resistance to Antimicrobial Agents
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- Symptomatic Infections
- Geographic Influence
- Spatial Distribution of the Microbiota—Anatomy of Infection
- Microbial Ecology and the Root Canal Ecosystem
- Other Microorganisms in Endodontic Infections

PERSISTENT/SECONDARY ENDODONTIC INFECTIONS
- Secondary/Persistent Infections and Treatment Failure
- Bacteria at the Root Canal–Filling Stage
- Microbiota in Root Canal–Treated Teeth

EXTRARADICULAR INFECTIONS
THE FOCAL INFECTION THEORY
TREATMENT OF ENDODONTIC INFECTIONS—RATIONALE
FASCIAL SPACE INFECTIONS
MANAGEMENT OF ABSCESSES AND CELLULITIS
SYSTEMIC ANTIBIOTICS FOR ENDODONTIC INFECTIONS

Apical periodontitis is essentially an inflammatory disease of microbial etiology primarily caused by infection of the root canal system.[233] 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.[23,110,160,287] Endodontic infection develops in root canals devoid of host defenses, either as a consequence of pulp necrosis (as a sequel to caries, trauma, periodontal disease, or iatrogenic operative procedures) or pulp removal for treatment.

Although fungi and most recently archaea and viruses have been found in association with endodontic infections,[217,264,273,305] 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.[161,167,257] Therefore, there is a current trend to include apical periodontitis in the roll of biofilm-induced oral diseases. Bacteria colonizing the root canal system enter in contact with the periradicular tissues via apical/lateral foramens 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 or chronic apical periodontitis.

The ultimate goal of the 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. Because apical periodontitis is an infectious disease, the rationale for endodontic treatment is unarguably to eradicate the occurring infection and/or prevent microorganisms from infecting or reinfecting the root canal or the periradicular tissues. The cardinal principle of any healthcare 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 of high quality and founded on solid scientific insight. This chapter focuses on diverse aspects of endodontic microbiology, including pathogenetic, taxonomic,

morphologic, and ecological issues. Special emphasis is also placed on the underlying principles for sound antimicrobial treatment of endodontic infections.

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 him to be alive,[54] but the role of Leeuwenhoek's "animalcules" in disease causation was unsuspected at that time. It took almost 200 years before 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 on the association between bacteria and apical periodontitis after the analysis of samples collected from root canals.[155] 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. 15-1). 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 an etiologic role was suspected for these bacteria. Most of the bacteria Miller had seen under light microscopy could not be cultivated using the technology available at his 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,[6,200] 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 et al.[110] These authors investigated the response of the dental pulps of conventional and germ-free rats after exposure to the oral cavities. Histologic evaluation was performed and revealed that whereas pulp necrosis and apical periodontitis lesions developed in all conventional rats, the pulps of germ-free rats not only remained vital but also repaired themselves by hard-tissue formation. Dentinlike tissue sealed the exposure area and isolated the pulps again from the oral cavity.

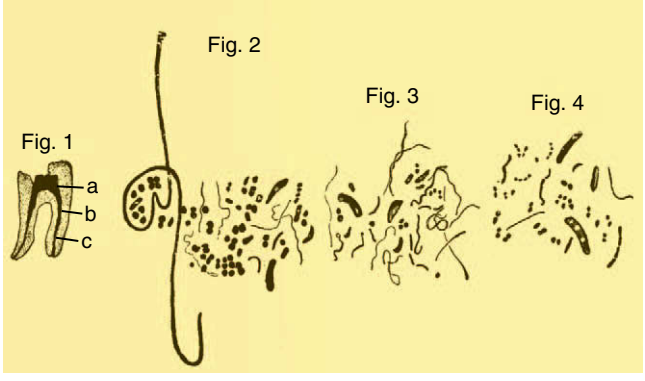


FIG. 15-1 Drawings from Miller's classic paper showing different bacterial forms in a root canal sample observed by microscopy.

The important role of bacteria in the etiology of apical periodontitis was further confirmed by Sundqvist's classic study.[287] 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 etiology of this disease. Anaerobic bacteria accounted for more than 90% of the isolates. Findings from Sundqvist's study also served to demonstrate that in the absence of infection, the necrotic pulp tissue itself and stagnant tissue fluid in the root canal cannot induce and perpetuate apical periodontitis lesions.

Möller et al.[160] 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.

ROUTES OF ROOT CANAL INFECTION

Under normal conditions, the pulpodentin complex is sterile and isolated from oral microbiota by overlying enamel 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 and then challenged by microorganisms present in caries lesions, in saliva bathing the exposed area, or in dental plaque formed onto 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 and apical foramens. Microorganisms may also have access to the root canal any time during or after professional endodontic intervention.

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 (Fig. 15-2). 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).[75] 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 well assume that once exposed, dentin offers an unimpeded access pathway for bacteria to reach the pulp via tubules. This is not necessarily the

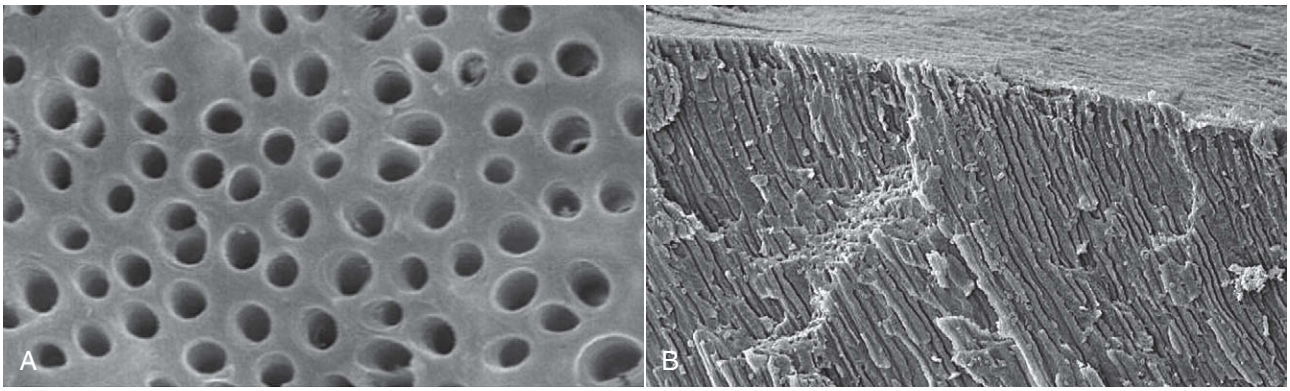


FIG. 15-2 Scanning electron micrographs of dentin showing tubules in cross-sectional **(A),** and longitudinal **(B),** views (**A-B,** ×850 and ×130, respectively).

case. It has been demonstrated that bacterial invasion of dentinal tubules occurs more rapidly in nonvital teeth than in vital ones.[164] In vital teeth, outward movement of dentinal fluid and the tubular contents (including 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.[152] 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.[183] 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.[3,172,173] So long as the pulp is vital, dentinal exposure does not represent a significant route of pulpal infection, except when dentin thickness is considerably reduced and 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.[153] Bacteria inside tubules under a deep carious lesion can reach the pulp even before frank pulpal exposure.[101] As mentioned, it has been assumed that the pulp will not be infected if it is still vital, and the few bacteria that reach the pulp can be of no importance, since 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.[182] On the other hand, if the vitality of the pulp is compromised and the defense mechanisms are impaired, even very few bacteria may initiate infection.

Bacteria have been isolated from traumatized teeth with necrotic pulps and apparently intact crowns.[287,318] It has been suggested that bacteria from the gingival crevice or periodontal pockets might reach the root canals of teeth whose pulps became necrotic after trauma through severed blood vessels of the periodontium—a phenomenon called *anachoresis*.[86] However, this theory has never been supported by scientific evidence. In fact, trauma can induce exposure of dentin by fracturing the crown or inducing the formation of enamel cracks. Macro- and microcracks in enamel can be present in most teeth (not only traumatized ones) and do not necessarily end at the enamel-dentin junction but extend deep into the dentin.[142] 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.

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 enters in direct contact with oral bacteria from caries lesions, saliva, and/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.[45]

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

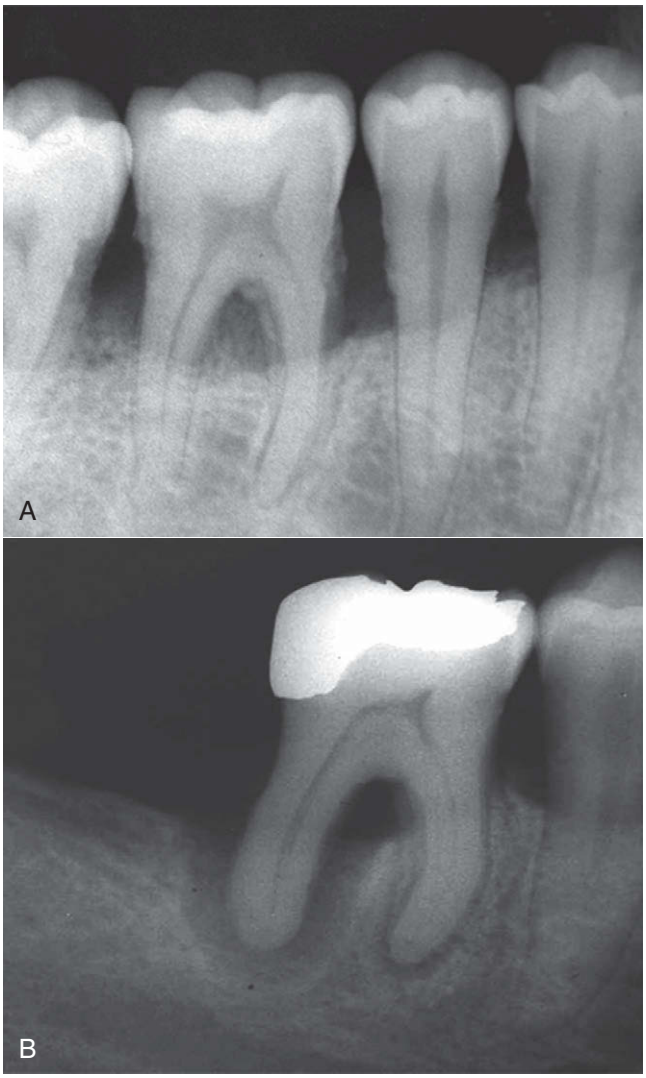


FIG. 15-3 Periodontal disease **(A)** mostly affects the pulp vitality when the subgingival biofilm reaches the apical foramen **(B).**

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 to irrigate the pulp[127] (Fig. 15-3). 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. Theoretically, microorganisms are 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[77,203]; 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.[51] Another argument against anachoresis as a route of pulpal infection comes from the study by Möller et al.,[160] 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. Although anachoresis has been suggested to be the mechanism through which traumatized teeth with seemingly intact crowns become infected,[86] current evidence indicates the main pathway of pulpal infection in these cases is dentinal exposure due to enamel cracks.[141,142]

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, it can be very 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 where the pulp was removed for treatment. Microbial penetration in the canal can occur during treatment, between appointments, or even after root canal filling. 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 touch 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 filling* 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, exposing the root canal filling material; or delay in placement of permanent restorations.[242]

MECHANISMS OF MICROBIAL PATHOGENICITY AND VIRULENCE FACTORS

The ability of a microorganism to cause disease is regarded as *pathogenicity*. *Virulence* denotes the degree of pathogenicity of a microorganism, and *virulence factors* are the microbial products, structural components, or strategies that contribute to pathogenicity. Bacterial virulence factors comprise structural cellular components and released products. Bacterial strategies that contribute to pathogenicity include the ability to coaggregate and form biofilms, which confers protection against 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 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

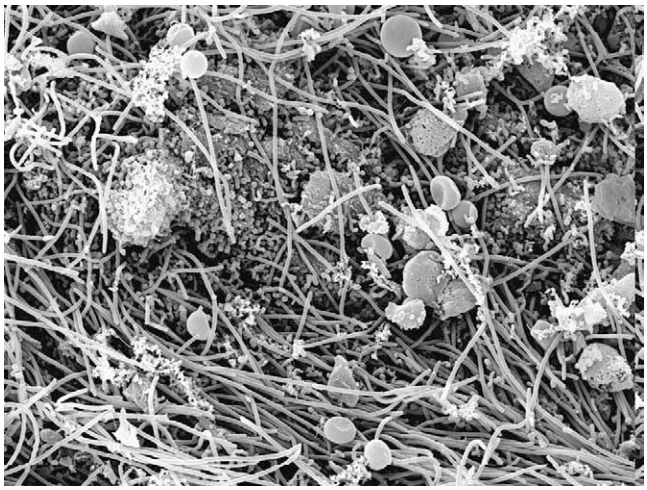


FIG. 15-4 Scanning electron micrograph showing a bacterial biofilm covering dentin in a deep carious lesion. Note the presence of different bacterial morphotypes (×3500). *(From Torabinejad M, Walton RE: Endodontics: principles and practice, ed 4, Saunders/Elsevier, St. Louis, 2009.)*

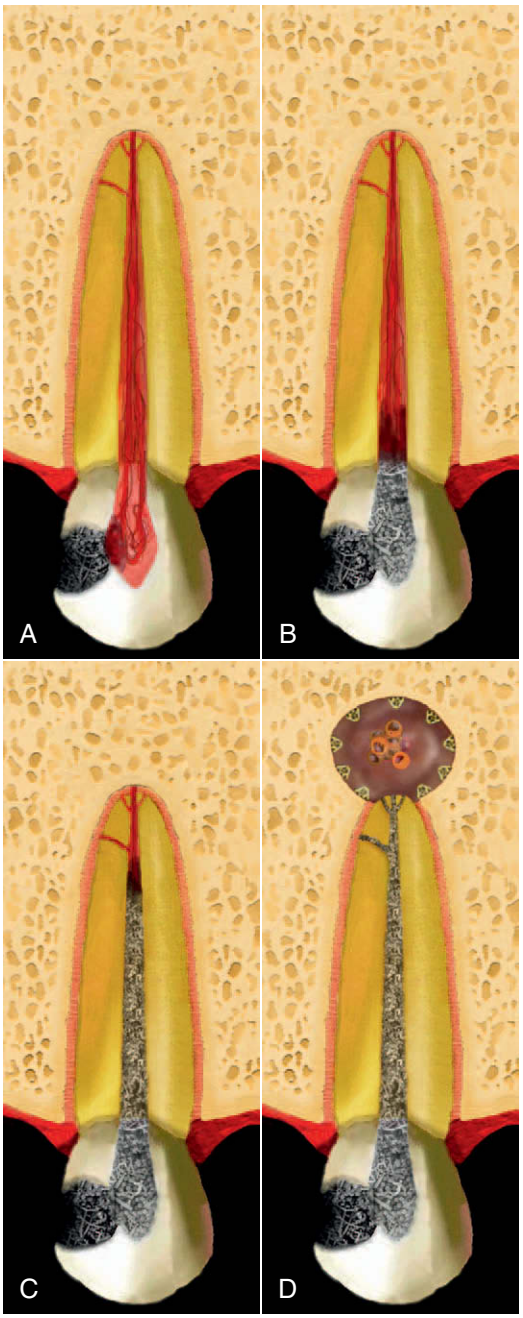


FIG. 15-5 Dynamics of pulp inflammation, necrosis, and infection from caries exposure **(A)** to apical periodontitis formation **(D).**

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 and causing endogenous infections.

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. 15-4). 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, colonize the necrotic tissue. These events advance through tissue compartments, coalesce, and move towards the apical part of the canal until virtually the entire root canal is necrotic and infected (Fig. 15-5). At this stage, involved bacteria can be regarded as the early root canal colonizers or pioneer species.

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 entire root canal is necrotic.[7,161,279,320] Therefore, the early colonizers play an important role in the initiation of the apical periodontitis disease process. The environmental conditions in the root canal are modified by pioneer species and the disease process and may then be conducive to the establishment of bacterial groups different from the early colonizers. Once the pulp is necrotic, species other than those that participated in the initial infectious process may also have access to the canal via coronal exposure or exposed dentinal tubules, and a shift in the microbiota is observed. 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 in advanced disease. With the passage of time, the endodontic microbiota becomes more and more structurally and spatially organized.

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 evincing 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.[248]

Bacteria exert their pathogenicity by wreaking havoc on the host tissues through direct and/or indirect mechanisms. Bacterial virulence factors that cause direct tissue harm include those that damage host cells and/or the intercellular matrix of the connective tissue. These factors usually involve secreted products, including enzymes, exotoxins, heat-shock proteins and metabolic end products.[231] 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 host immune reactions capable not only of defending the host against infection but also of causing severe tissue destruction[98,248,301] (Fig. 15-6). 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 chronic apical periodontitis lesions.[278] 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 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.[300] Although bacterial products may certainly be involved in the pathogenesis of diseases related to endodontic infections, bacterial indirect destructive effects seem to be more significant in the tissue damage associated with acute and chronic apical periodontitis lesions.

Depending on several factors, apical periodontitis can be chronic or acute. A chronic disease is usually associated with low virulence of the bacterial consortium involved, but this represents a persistent source of aggression to the tissues. Persistence in chronic infections is usually related to community organization in biofilms and the inaccessibility to host defenses because of the anatomic location of the infection. In chronic infections, the juxtaposition of bacterial biofilms to susceptible tissues that are not adapted to their presence triggers harmful inflammatory responses.[42] Thus, bacteria in the necrotic root canal cause chronic infection by forming biofilms on the canal walls and maintaining a close contact with the apical periodontal ligament, which reacts in a damaging way because periodontal ligament and bone cells are never adapted

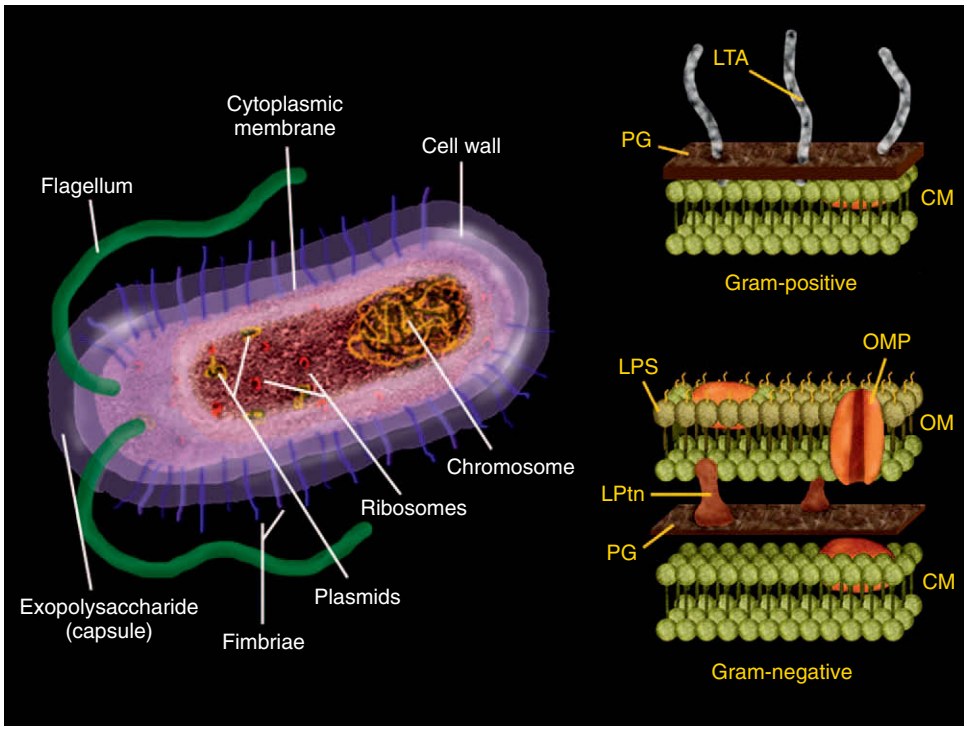


FIG. 15-6 Bacterial cell and its structural components that can act as virulence factors. *Right,* a detailed scheme of the bacterial cell walls from gram-positive and gram-negative bacteria. *CM,* Cytoplasmic membrane; *LPS,* lipopolysaccharide (endotoxin); *LPtns,* lipoproteins; *LTA,* lipoteichoic acid; *OM,* outer membrane; *OMP,* outer membrane protein; *PG,* peptidoglycan.

to the presence of bacteria. An acute infection in turn is usually caused by a high virulent bacterial consortium. High virulence may be a result of synergism between species. Acute infections are usually related to bacterial cells in a planktonic state, at high numbers, with tissue invasion ability, counteracted by a diminished host resistance. It has been demonstrated for some pathogens that genes coding for many virulence factors are much more expressed in planktonic cells than in sessile (biofilm) cells, suggesting that planktonic cells are more likely to participate in acute infections.[73]

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.[248]

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 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. To sum up, 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 ecological 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.[146]

Historically, microbiologists have faced periods of "reductionism" and "holism."[121] *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.

Traditionally, the study of the etiology of the main human diseases (including oral diseases) has been based on reductionism. More recently, 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 be due to collaborative activities of a biofilm community.

Community profiling studies[143,209,261] revealed that bacterial composition of the endodontic microbiota differs consistently between individuals. This indicates that apical periodontitis has a heterogeneous etiology, where multiple bacterial combinations can play a role in disease causation. Moreover, community structure differs significantly between different disease forms (e.g., chronic apical periodontitis versus acute apical abscess),[219,261] suggesting existence of a pattern associated with each form. Differences are still observed between individuals suffering from the same disease, disclosing an interindividual variability even more pronounced when different geographic locations are studied. There is seemingly a geography-related pattern, where individuals from a given location tend to cluster together based on similarities of the microbial community profiles.

The high interindividual variability observed for samples from the same clinical disease indicates that different compositions of bacterial communities can result in similar disease outcomes. Identification of community members can reveal the presence of some species or groups of species that may be important for the causation of some forms of disease. It is reasonable to realize that different magnitudes of disease, based on intensity of signs and symptoms, may be related to the species composition of the community. Indeed, it has been demonstrated that, despite individual uniqueness, bacterial communities associated with acute abscesses (severe pain and swelling) are more similar among them in comparison to bacterial communities associated with chronic apical periodontitis (no pain).[219,261] Although there may be no specific species related to some forms of disease, specificity seems to be related to community level, and certain communities (species composition) are specifically associated with some forms of apical periodontitis. From the perspective of the single-pathogen concept, apical periodontitis can be considered as having no specific microbial etiology. However, based on the community-

as-a-pathogen concept, it is possible to infer that some communities are more related to certain forms of the disease.[219,261]

Biofilm and Bacterial Interactions

The community-forming ability can be regarded as essential for microbial survival in virtually all environments. Indeed, the vast majority of microorganisms in nature invariably grow and function as members of metabolically integrated communities, or biofilms.[42,147] *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 polysaccharide[42,55] (Fig. 15-7). The ability to form biofilms has been regarded as a virulence factor,[90] and biofilm infections account for an estimated 65% to 80% of bacterial infections that affect humans in the developed world.[41] 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.

Biofilms are not merely passive assemblages of bacterial cells that are stuck to surfaces; they are structurally and dynamically organized complex biologic systems. 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.[44,55,276,281] Microcolonies are usually shaped as "towers" or "mushrooms." Dental biofilms can reach up to 300 or more cell layers in thickness.[276] Individual microcolonies may consist of a single bacterial species but more frequently are composed of several different species in a mixed community.

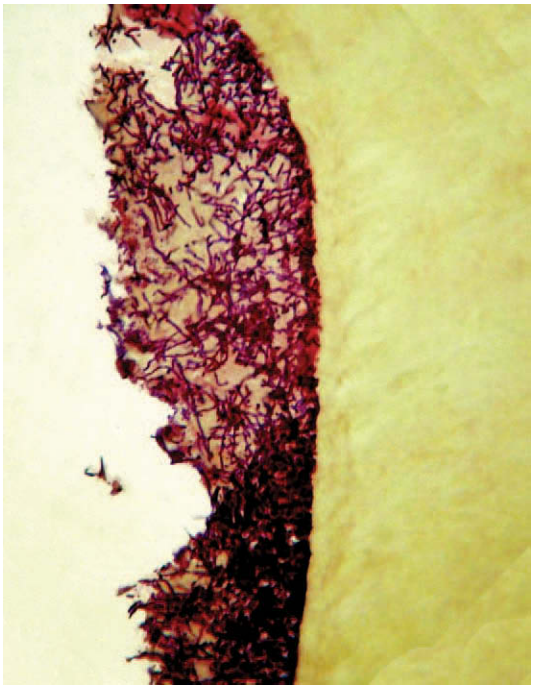


FIG. 15-7 Mixed bacterial biofilm adhered to the tooth surface (Brown and Brenn staining, ×1000).

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.[41] Although the matrix is primarily composed of polysaccharides, it can also contain proteins and nucleic acids.[90] 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.[43] Fluid in these channels carries substrate, end products of bacterial metabolism and signal molecules involved in bacterial interactions.[27] 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.[20,174,222] Thus, bacteria in biofilms adopt a radically different phenotype compared to their planktonic counterparts. Within biofilms, sophisticated systems of cell-cell communication (quorum sensing) are also used by some bacteria 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

As a community of bacteria, biofilms have a collective physiology, responding in concert to environmental challenges. Many naturally occurring biofilms have a highly diverse microbiota. The component species form populations (microcolonies) that are not randomly distributed but are spatially and functionally organized throughout the mixed community.

The properties displayed by a mixed biofilm community are mostly dictated by the interactions between populations, which create novel physiologic functions that cannot be observed with individual components. Populations are strategically positioned for optimal metabolic interaction, and the resultant architecture favors the ecological role of the community in the ecosystem. The biofilm community lifestyle affords a number of advantages to colonizing bacteria, including those discussed in the sections that follow.[148]

A Broader Habitat Range for Growth

The metabolism of early colonizers alters the local environment, setting the stage for attachment and growth of latecomers (including more fastidious species). This may include increased availability of some types of nutrients, removal of harmful metabolic end products, and the development of

appropriate physicochemical conditions (e.g., reduced oxygen tension and redox potential). As a consequence, the diversity of the microbiota increases over time because of bacterial succession.

Increased Metabolic Diversity and Efficiency

Nutrients are limited in most ecosystems, so the communities that can fix and process their resources most efficiently will thrive and be perpetuated. Bacteria living in mixed communities take part in a number of nutritional interrelationships, and food webs develop. For instance, products of the metabolism of one species may become the main source of nutrients for other species. Also in food webs, the concerted action of interacting species is required for the sequential breakdown of complex host-derived substrates to simpler products, which can favor less competent species. Many complex host macromolecules, especially glycoproteins, can only be degraded efficiently by mixed consortia of oral bacteria. Byproducts of the degradation of complex nutrients are trapped in the biofilm matrix and shared with other community members.

Protection From Competing Microorganisms, Host Defenses, Antimicrobial Agents, and Environmental Stress

Neighboring cells of a different species can produce enzymes such as beta-lactamases, catalase, and proteinases that are retained in the biofilm matrix and protect other bacteria against antimicrobials and host defenses. The same is true for metabolites and bacteriocins, which can inhibit competing species. The biofilm matrix also confers physical protection against phagocytosis.

Genetic Exchanges

Cell-cell associations and the biofilm architecture facilitate horizontal gene transfer in the community. Potential mechanisms mediating genetic exchange in biofilms include conjugation, transformation, and transduction. The main consequences of genetic exchanges among community members are related to the acquisition and dissemination of virulence and antibiotic-resistance genes.

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 likely 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 mixed 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).[30]

Resistance to Antimicrobial Agents

From a clinical standpoint, biofilm increased resistance to antimicrobial agents is of special concern. Bacteria arranged in biofilms are 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.[145] There are several possible mechanisms involved with biofilm resistance to antimicrobials.

Biofilm Structure May Restrict Penetration of Antimicrobial Agents

The agent may adsorb to and inhibit the bacteria at the biofilm surface, leaving cells deeply located in the biofilm relatively unaffected. The matrix in biofilms can also bind and retain neutralizing enzymes at concentrations that could inactivate the antimicrobial agent.[280]

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.[90]

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*.[113] It remains unclear whether these bacteria actually represent a distinct phenotype or are simply the most resistant cells within a population.[90]

Quorum Sensing—Bacterial Intercommunication

Quorum sensing is a term currently used for systems based on cell-cell communication that regulate gene expression in a cell density–dependent manner.[180] Quorum-sensing systems capacitate bacteria to behave collectively as a group. This phenomenon has been described in both gram-positive and gram-negative bacterial species.[10,57,317] Quorum sensing involves the production, release, and subsequent detection of diffusible signaling molecules called *autoinducers*. Two predominant types of autoinducers, *N*-acyl-L-homoserine lactones (AHLs) and posttranslationally modified peptides, are used by gram-negative and gram-positive bacteria, respectively. For example, the human pathogens *Pseudomonas aeruginosa* and *Staphylococcus aureus* employ AHLs and peptides, respectively, to control the expression of multiple virulence genes in concert with cell population density. Several other small-molecule quorum-sensing autoinducers have recently been discovered, including *Pseudomonas* quinolone signal (PQS) and AI-2.[35]

As members of a bacterial population producing and releasing autoinducers multiply, the extracellular concentration of these signaling molecules also increases. When autoinducers

reach a crucial threshold level, the group responds with a population-wide alteration in gene expression, activating or repressing target genes.[181] Since alterations in gene expression are linked to the presence of autoinducers, bacteria can perform specific functions only when living in groups. Such behavior provides some advantages to a bacterial population, affording adaptability to and protection against threatening environments.

Quorum-sensing systems are known to regulate virulence, competence for DNA uptake, entry into stationary phase, and biofilm formation.[35,46,184] With regard to virulence, quorum sensing allows the community to regulate expression of virulence genes in accordance with cell density. Thus, some opportunistic pathogens express virulence factors in response to sensing their own cell density. Using quorum sensing, bacteria can amass a high cell density before virulence factors are expressed, and in so doing, the bacteria are able to make a consolidated attack to overcome the host defenses and establish infection.[116] Teleologically, it appears bacteria were able to strategically wait for a critical number of cells to be reached and then start inducing damage, not giving the host sufficient time to mount an effective defense response. In reality, with regard to energy yield, it would be difficult for bacteria to produce some virulence factors before a critical community density is reached. For instance, production of proteinases by a few cells are not expected to have significant impact on the host environment, representing "wasted energy" for the growing population.[180]

The process of biofilm formation, including surface attachment, proliferation, matrix production, and detachment, is also partially controlled by quorum sensing.[74] Moreover, bacteria can monitor crowding as a measure of competition for nutrients. At high cell density, there may be too much competition to utilize available nutrients for growth, and starvation is anticipated. If bacteria are already starved and crowded, as they often are when growing as a biofilm, it may be advantageous to enter stationary phase. Entry into stationary phase dramatically alters patterns of gene expression to allow extended cell survival in the absence of nutrients.[128] Control of cell density in biofilms is also important to prevent water circulatory channels, essential for biofilm physiology, from being clogged and then inactivated by cells under excessive growth.

Several oral bacteria have been demonstrated to produce quorum-sensing signaling molecules,[71,194,321] indicating that bacteria in endodontic biofilms may also communicate with one another through this system.

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.[272] Essentially, culture analyses involve the following steps: sample collection and transport, dispersion, dilution, cultivation, isolation, and identification. 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.[61] 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 15-1. As one can tell, 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

BOX 15-1

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

extant population.[6,314] 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.[2,58,120,185,282]

That a given species has not been cultivated does not imply that this species will remain indefinitely impossible to cultivate. A myriad of obligate anaerobic bacteria were uncultivable 100 years ago, 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.[83] While we remain relatively unaware of the requirements for many bacteria to grow, identification methods not based on cultivability are required.

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.[19,25,295] As a consequence of all these factors, phenotype-based identification does not always allow an unequivocal identification.

To sidestep the limitations of culture, tools and procedures based on molecular biology have become available and have been substantially improved to achieve a more realistic description of the microbial world without the need for cultivation (Fig. 15-8). Molecular biology techniques have been widely used to directly identify microorganisms in samples without the need for cultivation. In addition, 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.*

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.[319] Similarly, the 18S rRNA gene of fungi and other eukaryotes has also been extensively used for identification of these organisms.

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 (see Fig. 15-4).

*References 26, 56, 195, 249, 277, and 294.

Many molecular methods for the study of microorganisms exist; the choice of a particular approach depends on the questions being addressed (see Fig. 15-5). Broad-range PCR followed by cloning and sequencing can be used to unravel the breadth of microbial diversity in a given environment. Microbial community structures can be analyzed via fingerprinting techniques, such as denaturing gradient gel electrophoresis (DGGE) and terminal restriction fragment length polymorphism (T-RFLP). Fluorescence in situ hybridization (FISH) can measure 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 15-2).

Impact of Molecular Methods in Endodontic Microbiology

Traditionally, endodontic infections have been studied by means of culture approaches. Such studies identified a set of species thought to play an important role in the pathogenesis of apical periodontitis.[288] More recently, 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.[236] Molecular methods have confirmed and strengthened the association of many cultivable bacterial species with apical periodontitis and have also revealed new suspected endodontic pathogens.[250] 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. Of these, about 45% were exclusively reported by molecular biology studies, compared to 32% detected by culture studies alone. Twenty-three percent of the total bacterial species richness has been detected by application of both culture and molecular studies (Fig. 15-9). As a consequence, it becomes quite apparent that the endodontic microbiota has been refined and redefined by molecular methods.[250]

TYPES OF ENDODONTIC INFECTIONS

Endodontic infections can be classified according to anatomic location (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 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

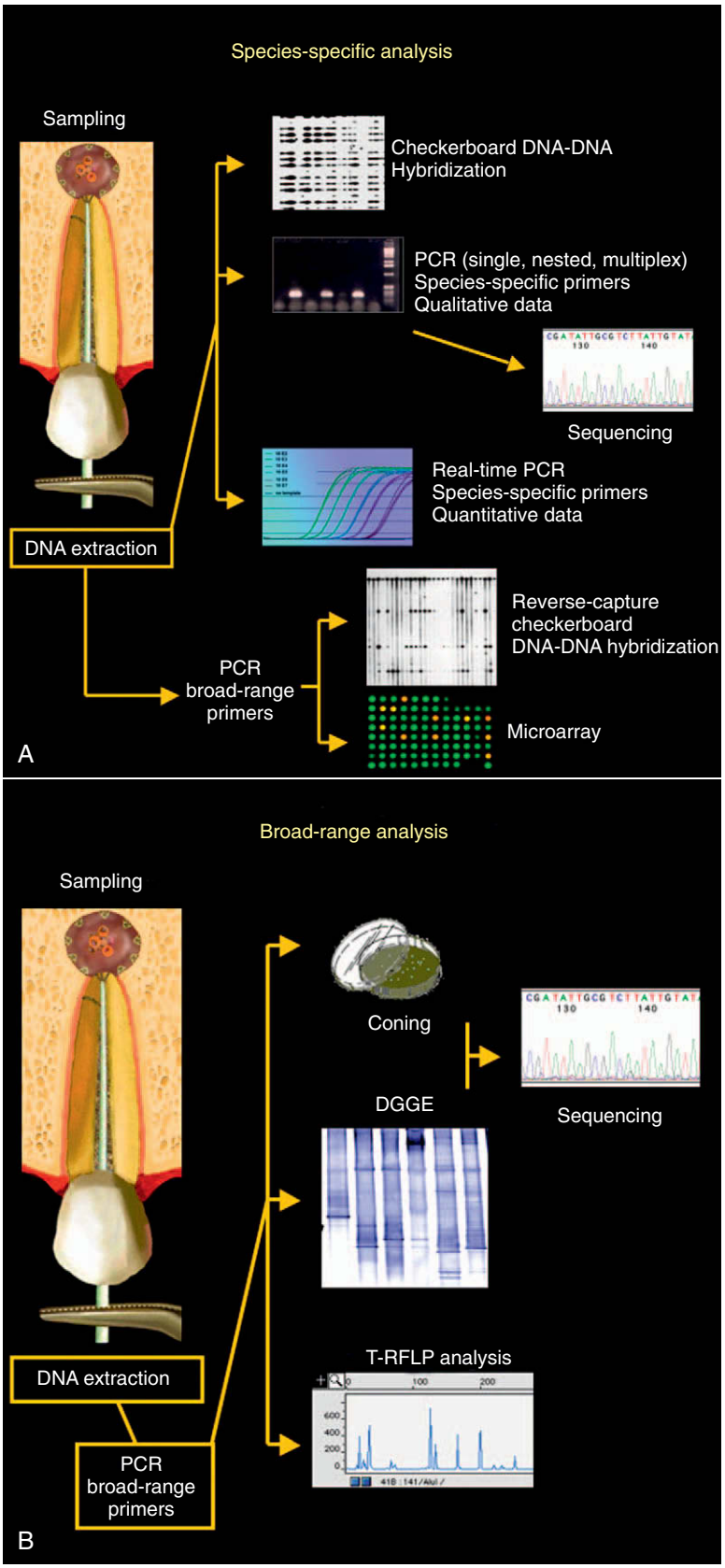


FIG. 15-8 Molecular biology methods used (or with potential to be used) in the study of endodontic infections. The choice for a particular technique will depend on the type of analysis to be performed. Some methods can be used for detection of target microbial species or groups **(A)** and others for a broader analysis of the microbiota **(B).**

BOX 15-2

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, microarray)
◆ 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	◆ Detect dead microorganisms
◆ Samples can be stored frozen for later analysis	
◆ DNA can be transported easily between laboratories	
◆ Detect dead microorganisms	

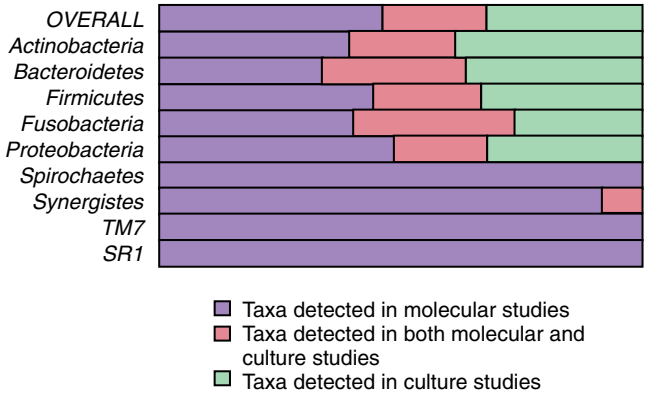


FIG. 15-9 Percent distribution of bacterial species/phylotypes found in endodontic infections according to the detection method. Data refer to the percentage of species or phylotypes overall or in each of the nine phyla that have endodontic representatives.

infection and in some way resisted intracanal antimicrobial procedures and were able to endure periods of nutrient deprivation in treated canals. Both persistent and secondary infections are for the most part clinically indistinguishable, except for cases where signs and/or symptoms of infection arise in a previously noninfected tooth—a typical example of secondary infection. *Extraradicular infection* is characterized by microbial invasion of the inflamed periradicular tissues and is a sequel to the intraradicular infection. Admittedly, 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.[52] *Diversity* refers to the number of different species present (richness) and their relative abundance (evenness) in a given ecosystem.[105]

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,[156] 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.[2,275] A high diversity of bacterial species has been revealed in the oral cavity by culturing approaches, 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. Over 700 bacterial taxa have been found in the human oral cavity, and phylogenetic studies have revealed that these taxa fall into 13 separate phyla: *Firmicutes, Bacteroidetes, Actinobacteria, Fusobacteria, Proteobacteria, Spirochaetes, Synergistes,* TM7, Sulphur River 1 (SR1), *Chloroflexi, Cyanobacteria, Deinococcus,* and *Acidobacteria.*[1,112,133,185,186]

Modern anaerobic culture and sophisticated molecular biology techniques have disclosed important aspects of the endodontic microbiota in the different types of infection. Of particular interest, they have allowed the assessment of the microbial diversity in endodontic infections, collectively revealing that more than 400 different microbial species/phylotypes have been found in infected root canals, usually in combinations involving many species in primary infections and a few ones in secondary/persistent infections. Endodontic bacteria fall into 9 of the 13 phyla that have oral representatives* (Fig. 15-10). Fungi and archaea 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 have emerged as candidate or putative endodontic pathogens. The following sections discuss specific aspects of each type of endodontic infection.

*References 162, 208, 218-220, 253, 260, and 307.

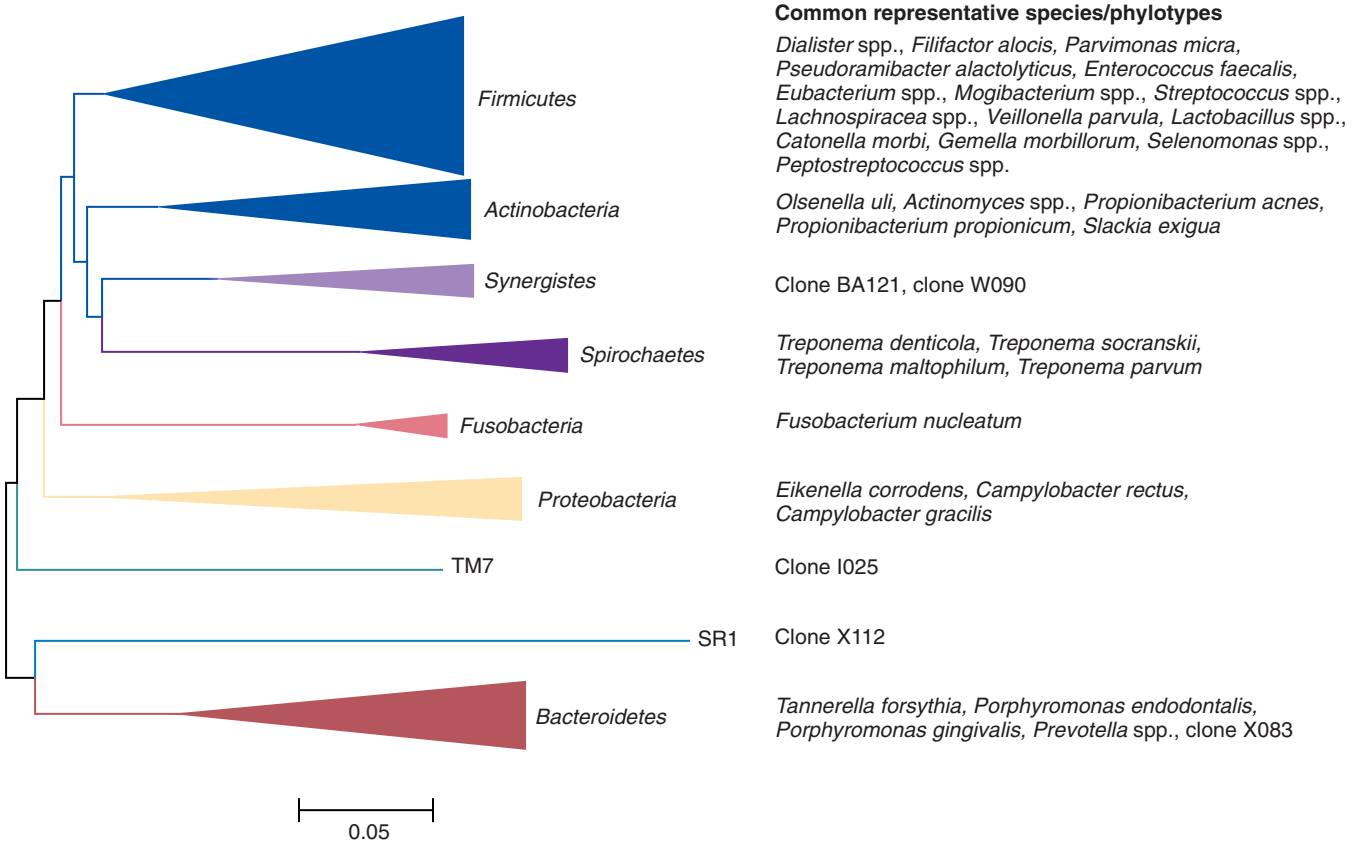


FIG. 15-10 Bacterial phyla with their respective endodontic representatives. *Right,* example species or phylotypes for each phylum.

PRIMARY INTRARADICULAR INFECTION

Microbial Composition and Diversity

Primary intraradicular infection is infection of the necrotic pulp tissue (Fig. 15-11). Participating microorganisms can have been 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 community conspicuously dominated by anaerobic bacteria. The number of bacterial cells may vary from 10^3 to 10^8 per root canal.[220,265,287,306] Molecular studies have disclosed a mean of 10 to 20 species/phylotypes per infected canal.[162,208,250,261] Canals of teeth with sinus tracts exhibit a mean number of 17 species.[208] The size of the apical periodontitis lesion has been shown to be proportional to the number of bacterial species and cells in the root canal.[208,260,287] A molecular study[208] 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[208]; 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 (*Fusobacterium, Dialister, Porphyro-*

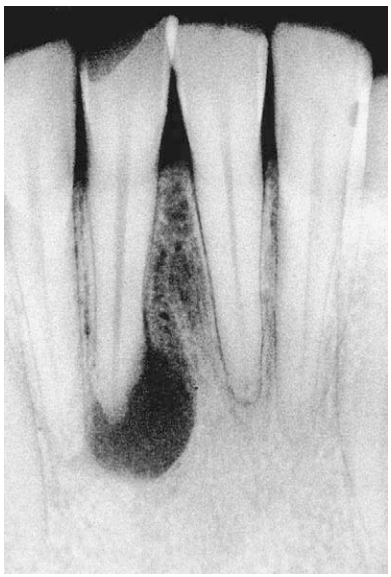


FIG. 15-11 Apical periodontitis due to primary intraradicular infection. Pulp is necrotic and lesion size is usually directly proportional to complexity of the microbiota involved.

monas, Prevotella, Tannerella, Treponema, Campylobacter, and *Veillonella*) and gram-positive (*Parvimonas, Filifactor, Pseudoramibacter, Olsenella, Actinomyces, Peptostreptococcus, Streptococcus, Propionibacterium,* and *Eubacterium*) bacteria.* 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. 15-12 to 15-14 display the most frequently detected species associated with chronic apical periodontitis, acute apical periodontitis, and acute apical abscesses, respectively, as revealed by studies from the authors' group using a highly sensitive molecular biology technique.

About 40% to 55% of the endodontic microbiota in primary infections is composed of species still uncultivated.[162,219] A molecular study revealed that 55% and 40% of the taxa detected in association with chronic apical periodontitis and acute apical abscesses, respectively, were uncultivated phylotypes.[219] As for their abundance in these infections, as-yet-uncultivated phylotypes corresponded to 38% and 30% of the clones sequenced from samples of chronic apical periodontitis and abscesses, respectively.[219] Molecular studies investigating the breadth of bacterial diversity in infected root canals have disclosed the occurrence of uncultivated phylotypes belonging to several genera, including *Synergistes, Dialister, Prevotella, Solobacterium, Olsenella, Fusobacterium, Eubacterium, Megasphaera, Veillonella,* and *Selenomonas,* as well as phylotypes related to the family *Lachnospiraceae* or the TM7 phylum.† Some uncultivated phylotypes can even be among the most prevalent bacteria in primary intraradicular infections, and others may be associated with pain.[219] Two of the most prevalent such phylotypes found in endodontic infections include *Synergistes* clone BA121 and *Bacteroidetes* clone X083.[207,208,251,253,255] Detection of these 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

Acute apical periodontitis and acute apical abscesses are typical examples of symptomatic endodontic infections. 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 the infected root canal and invade the periradicular tissues to establish an extraradicular infection and evoke purulent inflammation. Clinically, the disease leads to pain and/or swelling and has the potential to diffuse to sinuses and other facial spaces of the head and neck to form a cellulitis or other complications (Fig. 15-15). The microbiota involved in endodontic abscesses is mixed and dominated by anaerobic bacteria[47,115,122,219,261] (see Fig. 15-14). Direct comparisons using molecular technology reveal an average of 12 to 18 taxa per abscess case, compared to 7 to 12 taxa present in root canals from chronic cases.[219,261] 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.[219]

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 symptomatic lesions,* but the same species may also be present in somewhat similar frequencies in asymptomatic cases,† so factors other than the mere presence of a given putative pathogenic species may play a role in the etiology of symptomatic endodontic infections.[232,237] These factors could 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 (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.[232,237]

Cross-sectional studies suggest that bacterial succession occurs before symptoms arise.[261] At a given moment during the endodontic infectious process, the microbiota may reach a certain degree of pathogenicity that elicits acute inflammation at the periradicular tissues, with consequent development of pain and sometimes swelling. Molecular studies using DGGE or T-RFLP analysis revealed that the structure of the endodontic bacterial communities in symptomatic teeth is significantly different from that of asymptomatic teeth.[261] Differences are represented by different dominant species in the communities and larger numbers of species in symptomatic cases. A shift in the structure of the microbial community is likely to occur before appearance of symptoms. Such a shift is probably due to the arrival of new pathogenic species or variations and rearrangements in the number of members of the bacterial community. Differences in the type and load of dominant species and the resulting bacterial interactions may affect virulence of the whole bacterial community. This is in agreement with the community-as-a-pathogen concept discussed earlier.

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 different geographic locations and suggested that significant differences in the prevalence of some important species can actually exist.[14,204,241] In a more holistic approach, analysis of the bacterial community profiles of the microbiota associated with acute apical abscesses from the United States and Brazilian patients also revealed a geography-related pattern, with several species being exclusive for each

*References 15, 28, 67, 69, 79, 81, 89, 115, 126, 129, 162, 208, 218, 219, 250, 260, 262, 263, 286, 287, and 307.

†References 162, 207, 212, 213, 218, 219, 253, and 255.

*References 79, 84, 210, 219, 287, 302, and 322.

†References 15, 69, 89, 109, 262, and 263.

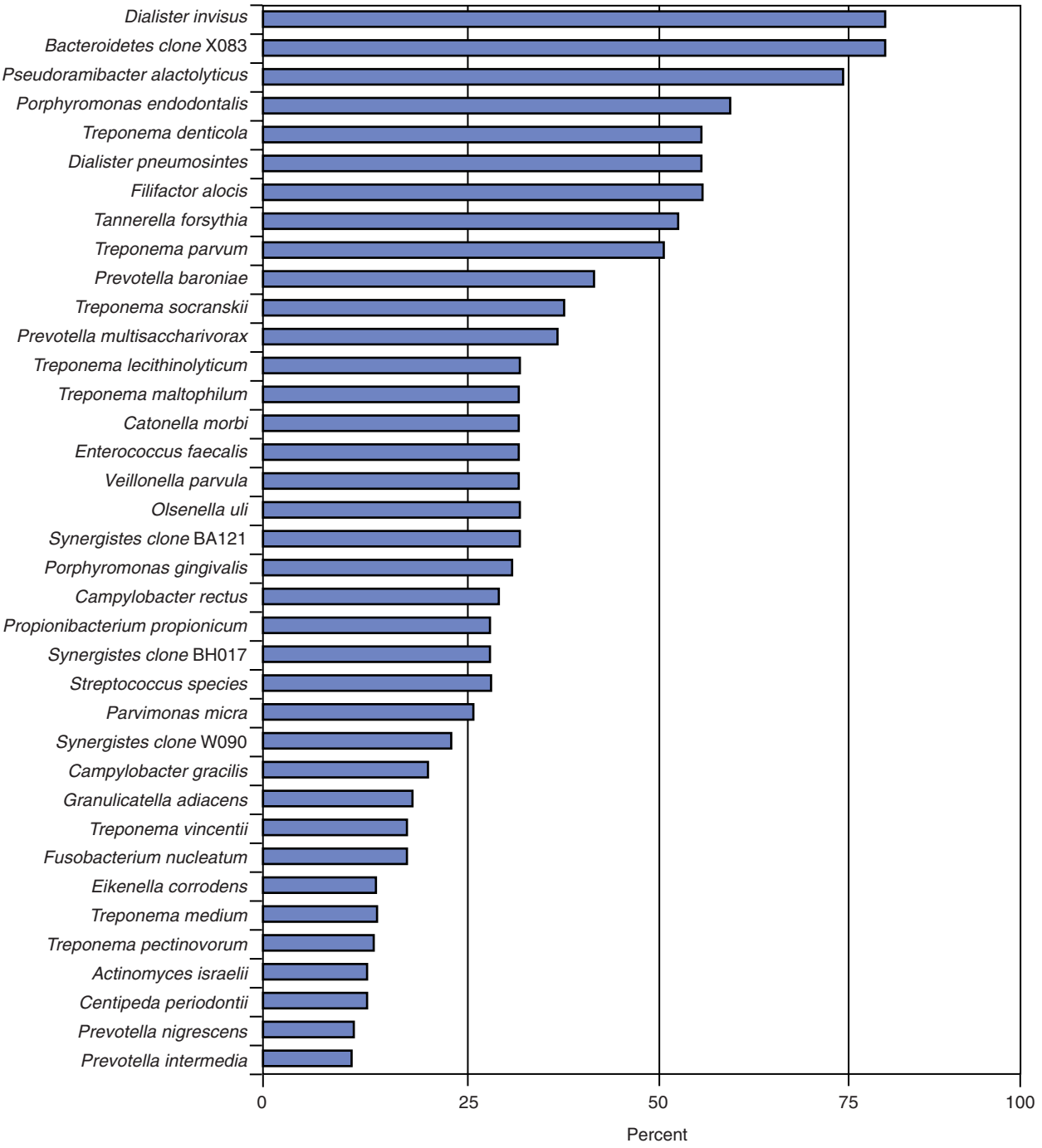


FIG. 15-12 Prevalence of bacteria detected in primary infections of teeth with chronic apical periodontitis. Data from the authors' studies using a taxon-specific nested-polymerase chain reaction protocol. *(From references 207, 211, 250, and 253.)*

location and others shared by the two locations, showing great differences in prevalence.[143] 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.

Spatial Distribution of the Microbiota—Anatomy of Infection

Knowledge of microbial location and organization within the root canal system assumes special importance to understanding the disease process and establishing effective antimicrobial therapeutic strategies. Most of the knowledge of the structure of the endodontic microbiota comes from morphologic studies,[161,167,227,257] but these do not usually provide information about bacterial identity and numbers. Consequently, it is not possible to delineate the role of the visualized bacteria in the disease process. Because every bacterial cell observed in the root canal system might be an endodontic pathogen, findings from morphologic studies should be used to understand the topography of the root canal infection and establish thera-

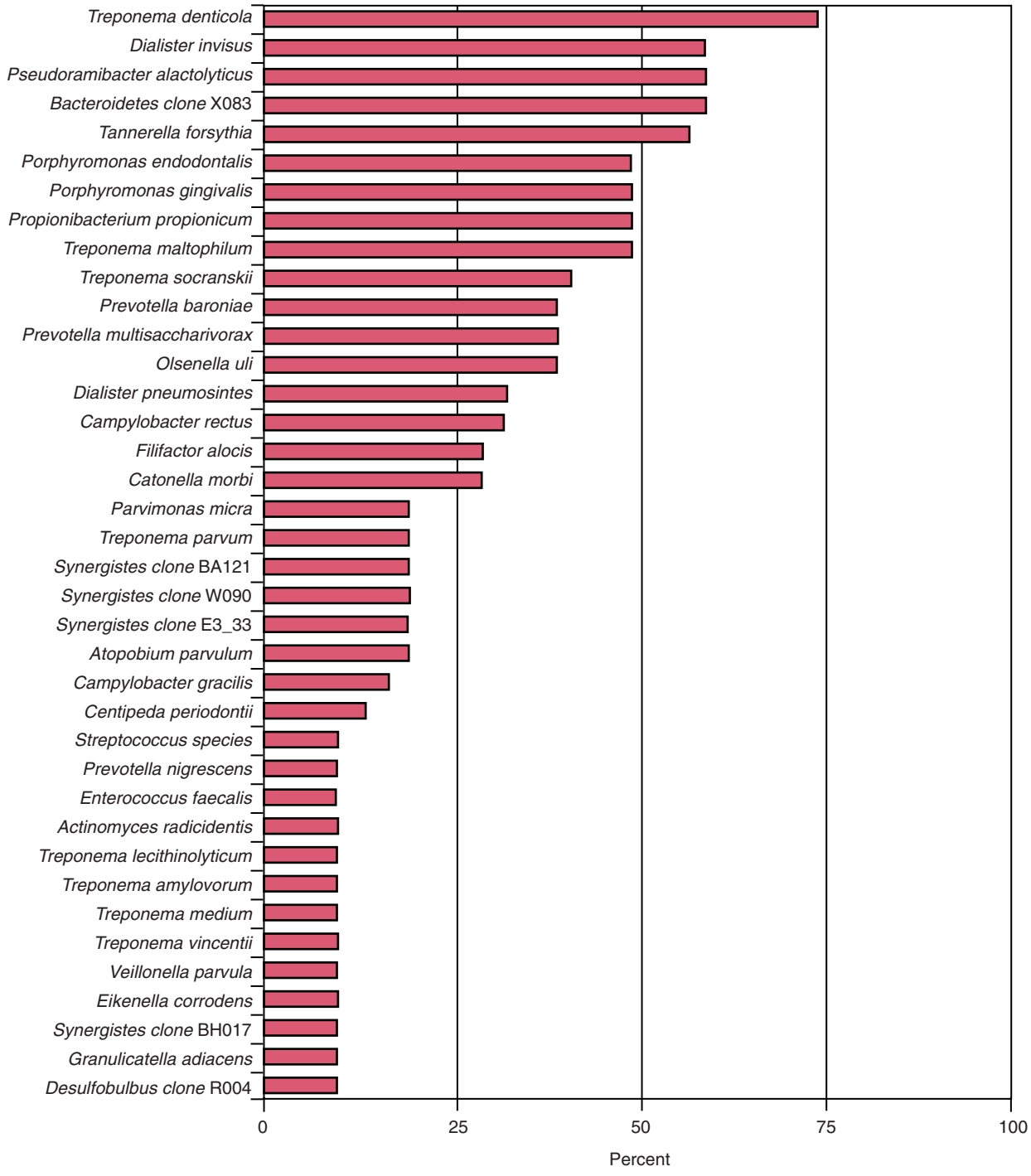


FIG. 15-13 Prevalence of bacteria detected in primary infections of teeth with acute apical periodontitis. Data from the authors' studies using a taxon-specific nested-polymerase chain reaction protocol. *(From references 207, 211, 250, and 253.)*

peutic measures in an attempt to completely eradicate the infection or at least reduce the bacterial load to thresholds compatible with periradicular tissue healing.

Again, mounting evidence suggests that apical periodontitis, like caries and periodontal diseases, is also a biofilm-induced 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. 15-16), and fungal cells are sporadically found[227,257] (Fig. 15-17). Bacteria colonizing the root canal system usually grow in sessile biofilms adhered to the dentinal walls and also as coaggregates (cells with distinct morphologies), aggregates (cells with the same morphology), and planktonic cells suspended in the fluid phase of the main canal[161,167,257] (Fig. 15-18). Lateral canals and isthmuses connecting main canals may be clogged with bacterial cells primarily organized in biofilms[166,202] (Figs. 15-19 and 15-20).

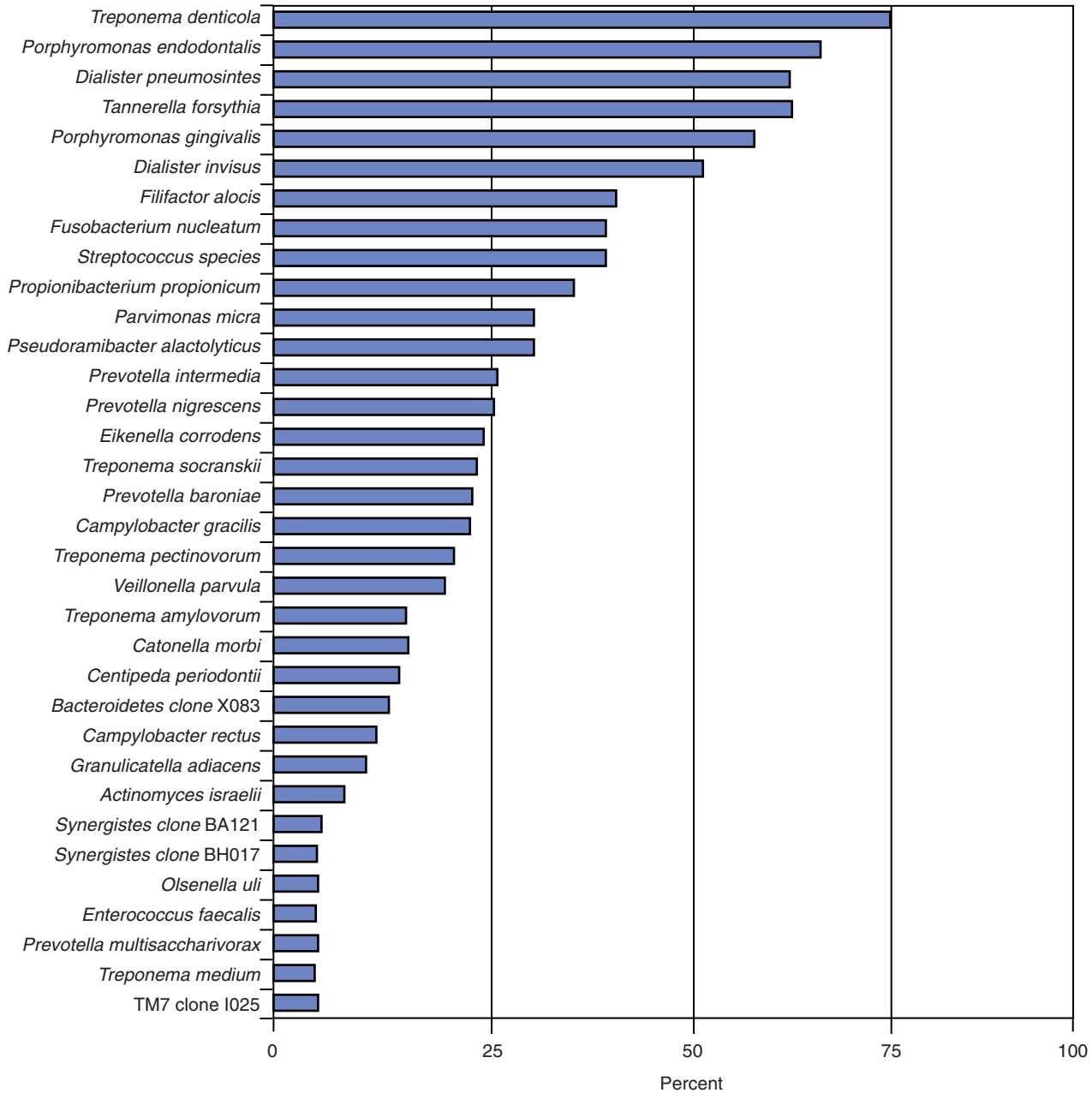


FIG. 15-14 Prevalence of bacteria detected in primary infections of teeth with acute apical abscesses. Data from the authors' studies using a taxon-specific nested-polymerase chain reaction protocol. *(From references 207, 211, 250, and 253.)*

Bacterial cells from endodontic biofilms are very often seen penetrating the dentinal tubules (Fig. 15-21). The diameter of dentinal tubules is large enough to permit penetration of most oral bacteria. It has been reported that dentinal tubule infection can occur in about 70% to 80% of the teeth evincing apical periodontitis lesions.[149,193] A shallow penetration is more common; bacterial cells can be observed reaching approximately 300 µm in some teeth[257] (Fig. 15-22). Dividing cells can be frequently observed within tubules in in situ investigations[257] (see Fig. 15-22), 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 acnes, Enterococcus faecalis, Candida albicans,* and streptococci.[142,191,239,258,310] In their clinical study, Peters et al.[193] 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 strep-



FIG. 15-15 Acute apical abscess. The infection has spread to other anatomic spaces to form cellulitis.

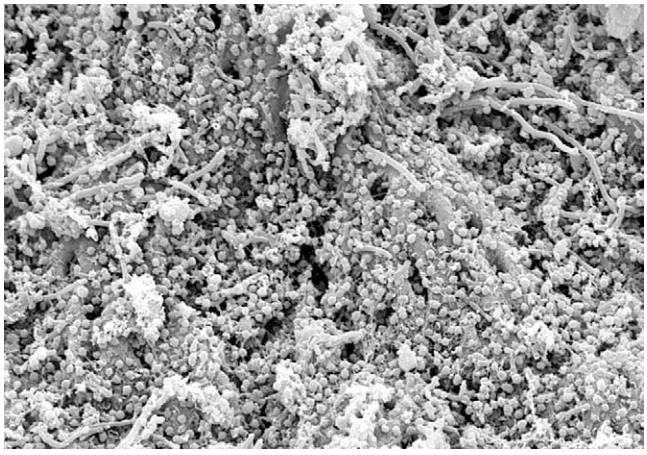


FIG. 15-16 Mixed bacterial population colonizing the root canal wall. Cocci are the predominant forms, but rods, filaments, and spirochetes are also observed. In some areas, coccoid cells are relatively apart from each other (×2200). *(From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *93:174, 2002.)*

tococci. Using immunohistologic analysis, Matsuo et al.[149] observed the intratubular 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.

Whereas bacteria present as planktonic cells in the main root canal may be easily accessed and eliminated by instruments and substances used during 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 and may require special therapeutic strategies to be eradicated.

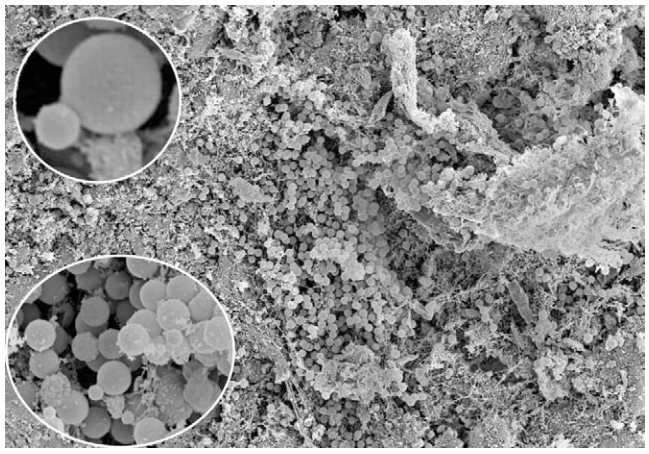


FIG. 15-17 Heavy colonization of yeast cells in the root canal of an extracted tooth with primary infection associated with apical periodontitis (×300). Note that some cells are in the stage of budding. A daughter cell is growing on the surface of the mother cell *(insets)* (insets: bottom ×2700, top ×3500). *(From Siqueira JF Jr, Sen BH: Fungi in endodontic infections.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *97:632, 2004.)*

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, and colonization not 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,[187] only a limited assortment of these species (about 10 to 40) is consistently selected out for growth and survival within a root canal containing necrotic pulp tissue from the same individual. This indicates that ecological determinants operate in the necrotic canal and dictate which species will succeed in colonizing this previously sterile environment. The major ecological 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. 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 very initial phases of the pulpal infectious process, facultative bacteria predominate.[63] 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 survival and growth of obligate anaerobic bacteria. With the passage of time, anaer-

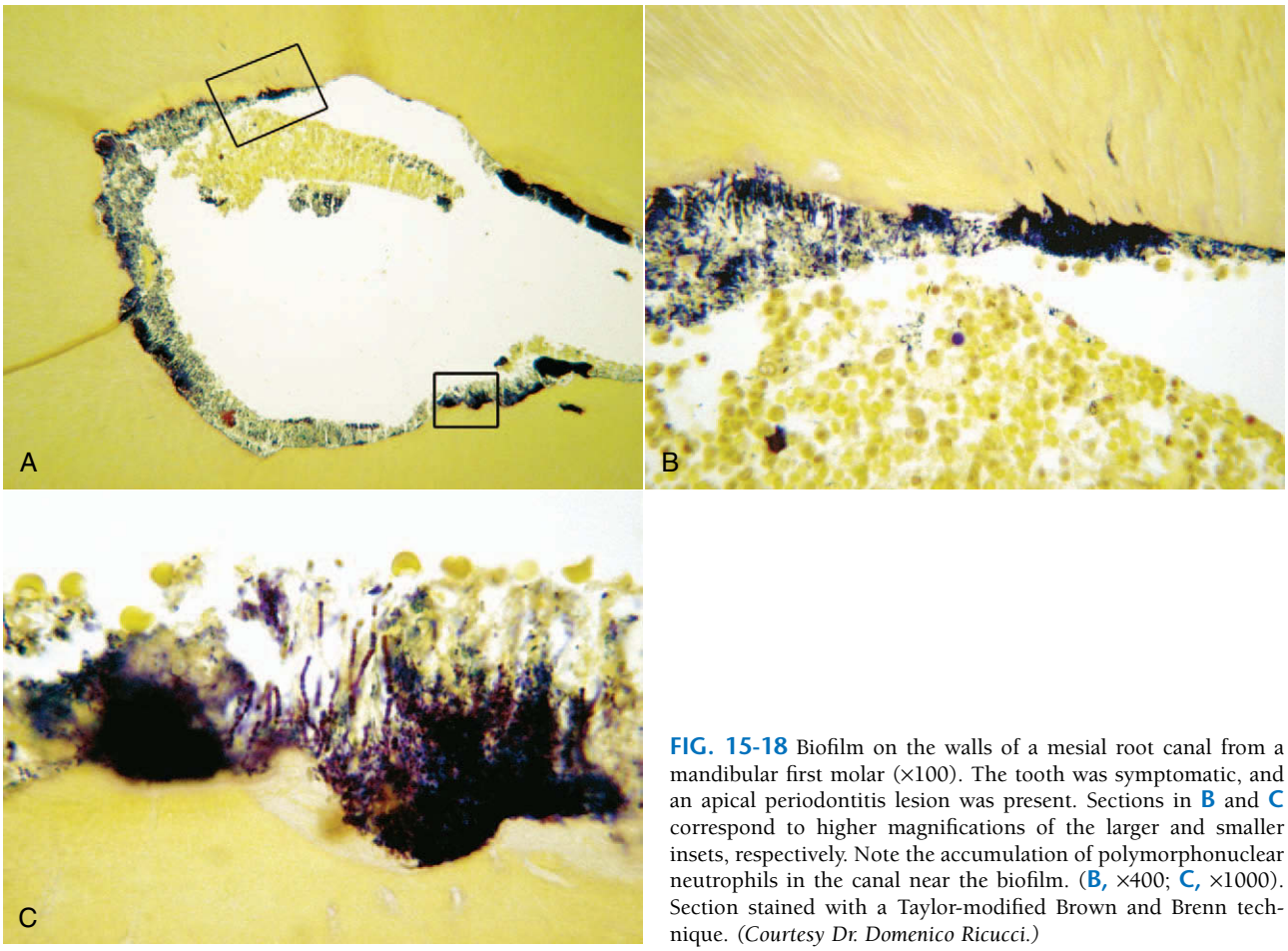


FIG. 15-18 Biofilm on the walls of a mesial root canal from a mandibular first molar (×100). The tooth was symptomatic, and an apical periodontitis lesion was present. Sections in **B** and **C** correspond to higher magnifications of the larger and smaller insets, respectively. Note the accumulation of polymorphonuclear neutrophils in the canal near the biofilm. (**B,** ×400; **C,** ×1000). Section stained with a Taylor-modified Brown and Brenn technique. *(Courtesy Dr. Domenico Ricucci.)*

obic 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. 15-23).

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 foramens; (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 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.[289] 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 gradually 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 and/or amino acids in their metabolism (see Fig. 15-23).

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 ecological 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

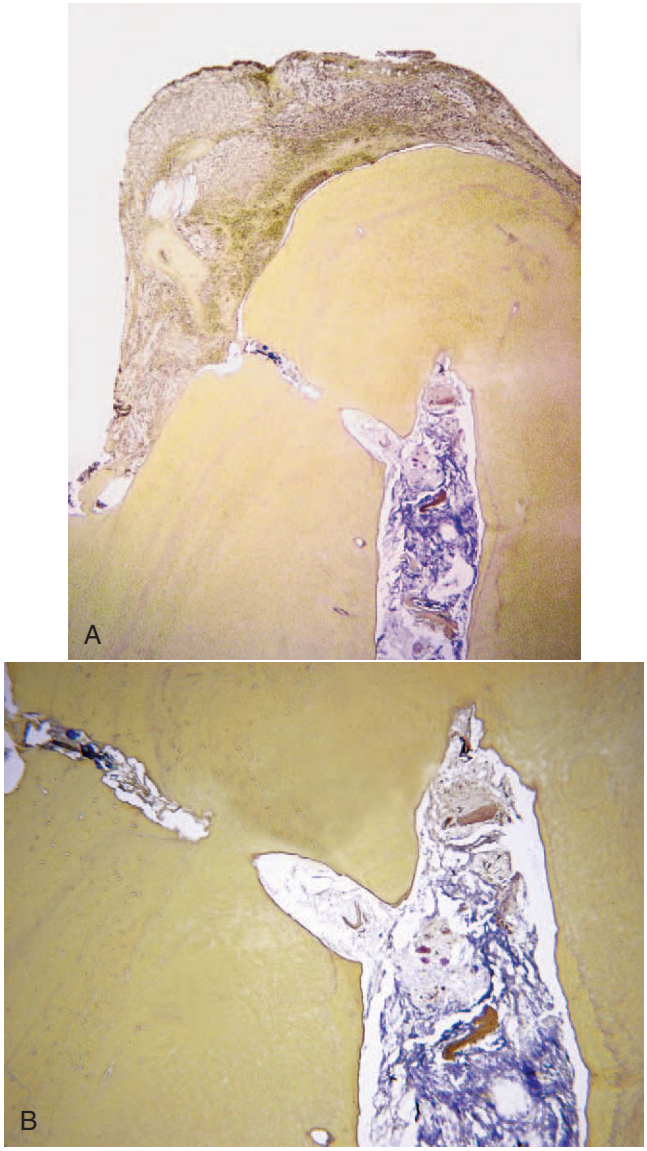


FIG. 15-19 A, Bacterial biofilm in the necrotic canal and in an apical ramification contiguous to inflamed periradicular tissues (×25). **B,** Higher magnification of **A** (×100). Sections stained with a Taylor-modified Brown and Brenn technique. *(Courtesy Dr. Domenico Ricucci.)*

breakdown of complex host-derived substrates. Fig. 15-24 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 upon products of 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 substance that inhibits another species).

Many species adhere directly to host surfaces, 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.[117] Coaggregations differ from aggregations and agglutinations in that the latter two interactions occur between genetically identical cells.[118] A given pair of species can attach to each other by means of specific receptor-adhesin interactions, which are usually lectinlike 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.[114]

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.[60,126,159,259] One molecular study[16] has reported the occurrence of *C. albicans* in 21% of samples from primarily infected canals.

Archaea

Archaea comprise 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.[59] To date, no member of the *Archaea* domain has been described as a human pathogen. Although one study failed to detect archaea in samples from primary endodontic infections,[254] other studies detected a *Methanobrevibacter oralis*–like phylotype in some primarily infected canals.[305,307]

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 and need to infect living cells to use the cell's machinery to replicate the viral genome. 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 the root canal only in noninflamed vital pulps of patients infected with the human immunodeficiency virus.[78] Human cytomegalovirus (HCMV) and Epstein-Barr virus (EBV) have been detected in apical periodontitis lesions,[215] where living host cells are present. 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.[273] Bacterial challenge emanating from the canals may cause an influx of virus-infected cells into the periradicular tissues. Reactivation of HCMV and/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. Herpesvirus-infected inflammatory cells are stimulated to release proinflammatory

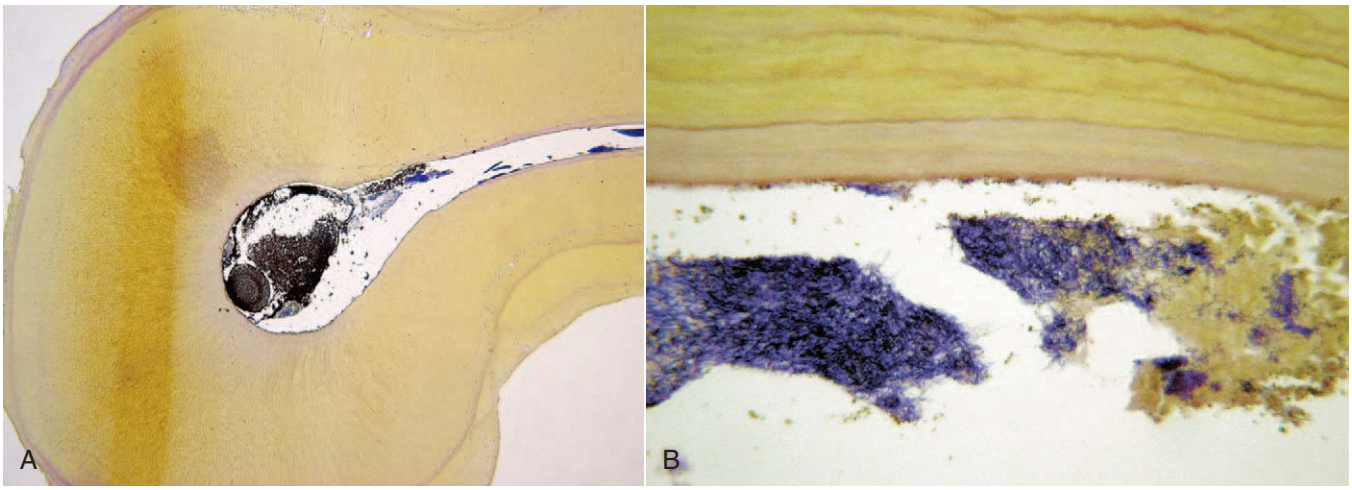


FIG. 15-20 Transverse sections of a maxillary second molar with fused mesial and palatal roots. **A,** Heavy bacterial infection of the canal, spreading to an isthmus (×25). **B,** Higher magnification of the isthmus clogged with bacteria (×400). Section stained with a Taylor-modified Brown and Brenn technique. *(Courtesy Dr. Domenico Ricucci.)*

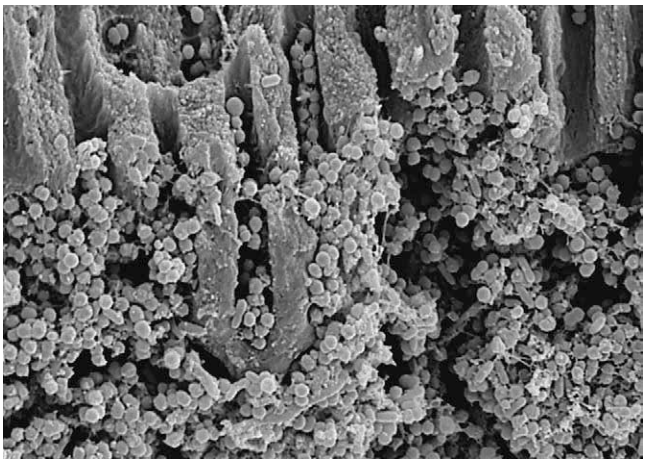


FIG. 15-21 Heavy infection of the root canal walls, mainly by cocci, but some small rods are also seen. Cocci are penetrating into dentinal tubules (×3500). *(From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *93:174, 2002.)*

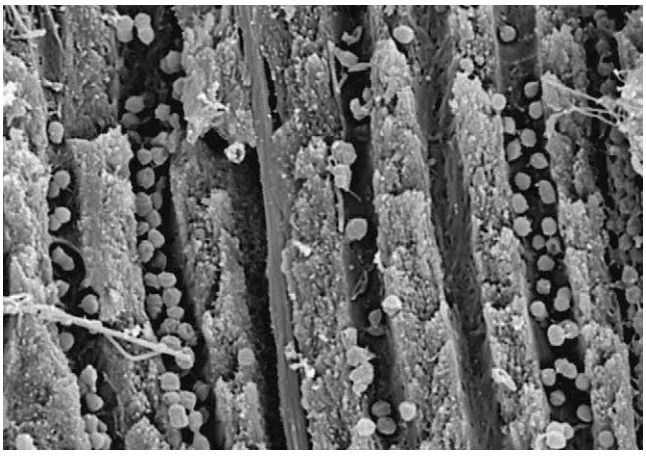


FIG. 15-22 Cocci in dentinal tubules approximately 300 µm from the main root canal (×5000). *(From Siqueira JF Jr, Rôças IN, Lopes HP: Patterns of microbial colonization in primary root canal infections.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *93:174, 2002.)*

cytokines,[157,313] and evidence of HCMV and/or EBV infection has been more frequently observed in symptomatic apical periodontitis lesions,[214,216] large lesions,[215,216] and lesions from HIV-positive patients.[217] However, the role of herpesviruses in the pathogenesis of apical periodontitis 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 professional 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.

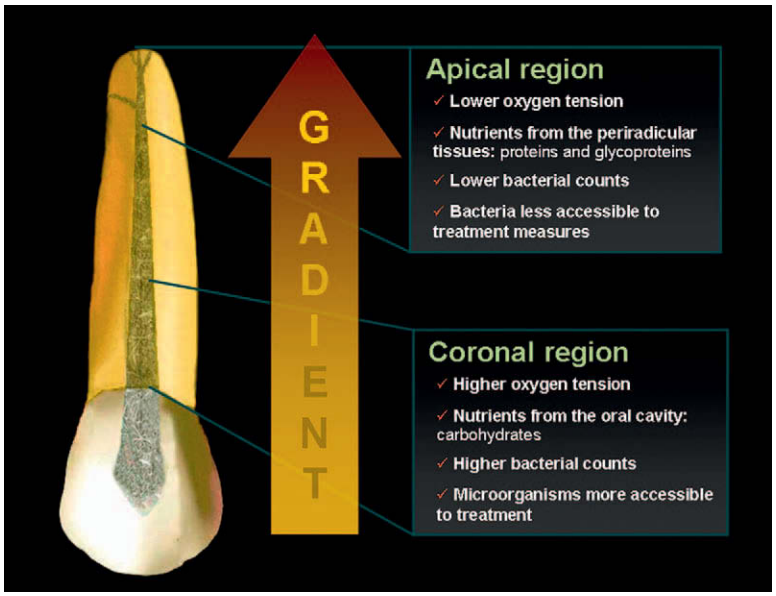


FIG. 15-23 Ecological conditions in different areas of the root canal. A gradient of oxygen tension and nutrients (type and availability) is formed. Consequently, the microbiota residing in different parts can also differ in diversity, density, and accessibility to treatment procedures.

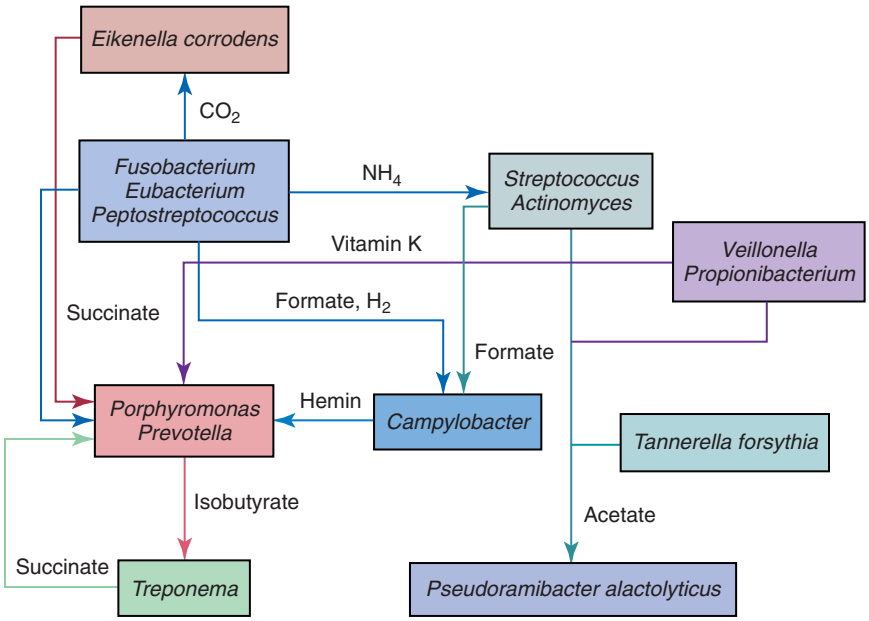


FIG. 15-24 Interbacterial nutritional interactions that can take place in infected root canals where growth of some species can be dependent upon products of metabolism of other species.

Secondary/Persistent Infections and Treatment Failure

Persistent or secondary intraradicular infections are the major causes of endodontic treatment failure (Fig. 15-25). 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 filling.[64,267,308] Second, most (if not all) root canal–treated teeth evincing persistent apical periodontitis lesions have been demonstrated to harbor an intraradicular infection.* Based on these arguments, studies investigating bacteria remaining in the root canals at the filling stage serve the purpose of disclosing 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 evincing apical periodontitis serve to show the association of species with treatment failure, since the microorganisms detected are likely to be participating in the etiology of persistent disease (*outcome already established*).

*References 134, 135, 197, 206, 221, 252, and 290.

Bacteria at the Root Canal–Filling Stage

Diligent antimicrobial treatment may still fail to completely eliminate bacteria from the infected root canal system. This is because persisting bacteria are either resistant or inaccessible to treatment procedures. Whatever the cause of persistence, bacterial diversity and density 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 1 to 5 bacterial



FIG. 15-25 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.

species per case, and the number of bacterial cells usually varies from 10^2 to 10^5 per sample.*

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.† 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, Actinomyces* species, *Propionibacterium* species, *P. alactolyticus,* lactobacilli, *E. faecalis,* and *Olsenella uli.*‡ This gives support to 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. About 40% of the taxa found in posttreatment samples are as-yet-uncultivated bacteria,[220] indicating that along with other reasons (such as inaccessibility to sampling or low bacterial levels), attainment of negative cultures after treatment procedures does not necessarily imply sterility.

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 found in the canal at the filling stage.[64,267] There are some possible explanations:

- Residual bacteria may die after filling because of toxic effects of the filling material, 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 filling can influence the outcome of the endodontic treatment provided they:

- Have the ability to withstand periods of nutrient scarcity, scavenging for low traces of nutrients and/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 Root Canal–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 30 species, which is very similar to untreated canals (see Fig. 15-25).[197,209,221,252,290] The number of bacterial cells in treated teeth varies from 10^3 to 10^7 per canal.[190,225]

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* (Fig. 15-26). Root canal–treated teeth are about nine times more likely to harbor *E. faecalis* than cases of primary infections.[211] This suggests that this species can be inhibited by other members of a mixed bacterial consortium commonly present in primary infections, and that the bleak environmental conditions within filled root canals do not prevent its survival. The fact that *E. faecalis* has been commonly recovered from cases treated in multiple visits and/or in teeth left open for drainage[266] suggests that this species may be a secondary invader that succeeds in colonizing the canal, resists treatment, and causes a secondary infection that then becomes persistent.

*References 34, 220, 245, 246, 267, and 306.

†References 34, 80, 192, 220, 265, and 267.

‡References 34, 36-38, 40, 80, 190, 192, 220, 240, 245, 246, 267, and 293.

*References 158, 197, 206, 211, 225, 252, 290, and 324.

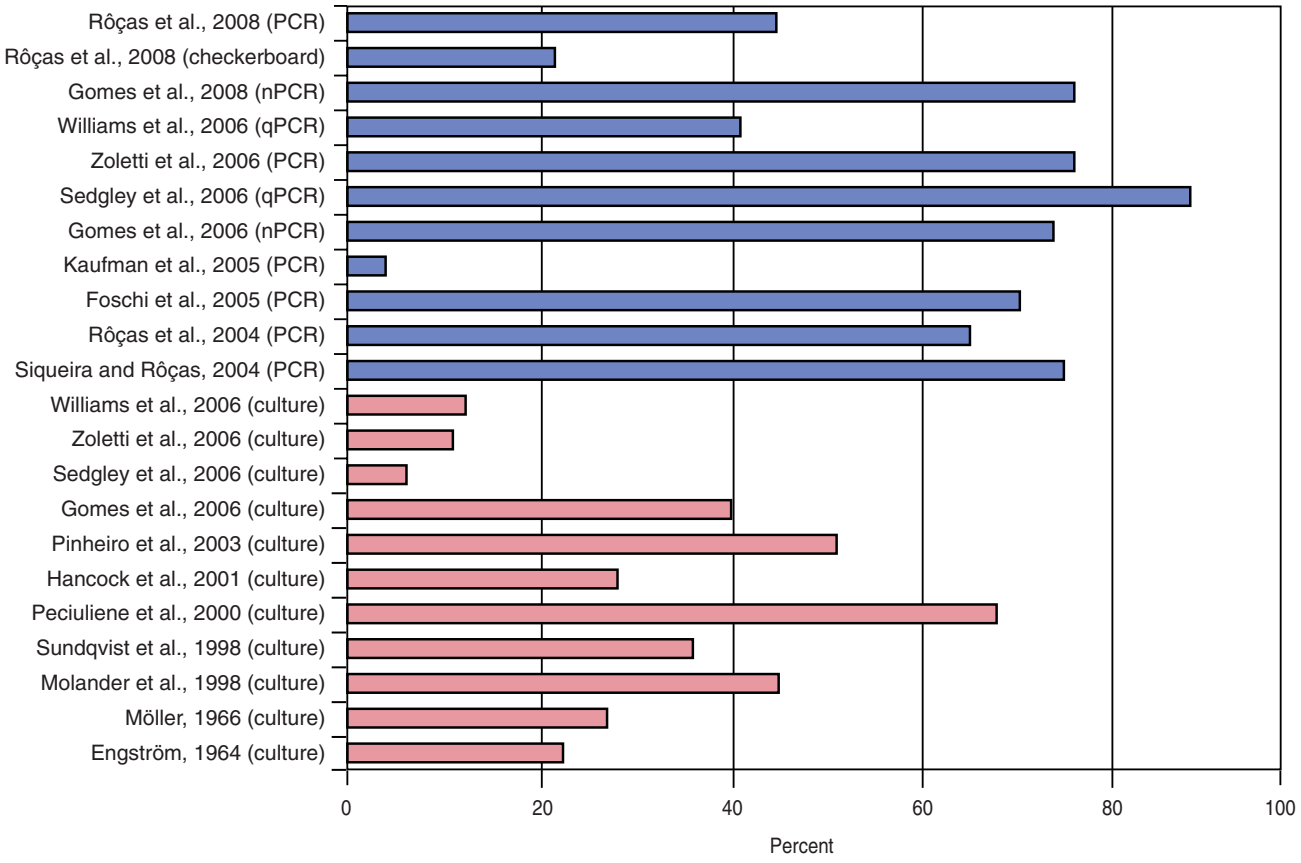


FIG. 15-26 Prevalence of *Enterococcus faecalis* in samples from root canal–treated teeth with apical periodontitis. Data from culture *(orange bars)* and molecular *(blue bars)* studies below.

Molecular Studies:

1. *Foschi F, Cavrini F, Montebugnoli L, et al: Detection of bacteria in endodontic samples by polymerase chain reaction assays and association with defined clinical signs in Italian patients.* Oral Microbiol Immunol *20:289, 2005.*
2. *Gomes BP, Pinheiro ET, Jacinto RC, et al: Microbial analysis of canals of root-filled teeth with periapical lesions using polymerase chain reaction.* J Endod *34:537, 2008.*
3. *Gomes BP, Pinheiro ET, Sousa EL, et al:* Enterococcus faecalis *in dental root canals detected by culture and by polymerase chain reaction analysis.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *102:247, 2006.*
4. *Kaufman B, Spangberg L, Barry J, Fouad AF:* Enterococcus *spp. in endodontically treated teeth with and without periradicular lesions.* J Endod *31:851, 2005.*
5. *Rôças IN, Hulsmann M, Siqueira JF Jr: Microorganisms in root canal-treated teeth from a German population.* J Endod *34:926, 2008.*
6. *Rôças IN, Siqueira JF Jr, Santos KR: Association of* Enterococcus faecalis *with different forms of periradicular diseases.* J Endod *30:315, 2004.*
7. *Sedgley C, Nagel A, Dahlen G, et al: Real-time quantitative polymerase chain reaction and culture analyses of* Enterococcus faecalis *in root canals.* J Endod *32:173, 2006.*
8. *Siqueira JF Jr, Rôças IN: Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *97:85, 2004.*
9. *Williams JM, Trope M, Caplan DJ, Shugars DC: Detection and quantitation of* Enterococcus faecalis *by real-time PCR (qPCR), reverse transcription-PCR (RT-PCR), and cultivation during endodontic treatment.* J Endod *32:715, 2006.*
10. *Zoletti GO, Siqueira JF Jr, Santos KR: Identification of* Enterococcus faecalis *in root-filled teeth with or without periradicular lesions by culture-dependent and -independent approaches.* J Endod *32:722, 2006.*

Culture Studies:

1. *Engström B: The significance of enterococci in root canal treatment.* Odontol Rev *15:87, 1964.*
2. *Gomes BP, Pinheiro ET, Sousa EL, et al:* Enterococcus faecalis *in dental root canals detected by culture and by polymerase chain reaction analysis.* Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontology *102:247, 2006.*
3. *Hancock HH 3rd, Sigurdsson A, Trope M, Moiseiwitsch J: Bacteria isolated after unsuccessful endodontic treatment in a North American population.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *91:579, 2001.*
4. *Molander A, Reit C, Dahlen G, Kvist T: Microbiological status of root-filled teeth with apical periodontitis.* Int Endod J *31:1, 1998.*
5. *Möller AJR: Microbial examination of root canals and periapical tissues of human teeth.* Odontol Tidskr *74(supplement):1, 1966.*
6. *Peciuliene V, Balciuniene I, Eriksen HM, Haapasalo M: Isolation of* Enterococcus faecalis *in previously root-filled canals in a Lithuanian population.* J Endod *26:593, 2000.*
7. *Pinheiro ET, Gomes BP, Ferraz CC, et al: Microorganisms from canals of root-filled teeth with periapical lesions.* Int Endod J *36:1, 2003.*
8. *Sedgley C, Nagel A, Dahlen G, et al: Real-time quantitative polymerase chain reaction and culture analyses of* Enterococcus faecalis *in root canals.* J Endod *32:173, 2006.*
9. *Sundqvist G, Figdor D, Persson S, Sjogren U: Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment.* Oral Surg Oral Med Oral Pathol Oral Radiol Endod *85:86, 1998.*
10. *Williams JM, Trope M, Caplan DJ, Shugars DC: Detection and quantitation of* Enterococcus faecalis *by real-time PCR (qPCR), reverse transcription-PCR (RT-PCR), and cultivation during endodontic treatment.* J Endod *32:715, 2006.*
11. *Zoletti GO, Siqueira JF Jr, Santos KR: Identification of* Enterococcus faecalis *in root-filled teeth with or without periradicular lesions by culture-dependent and -independent approaches.* J Endod *32:722, 2006.*

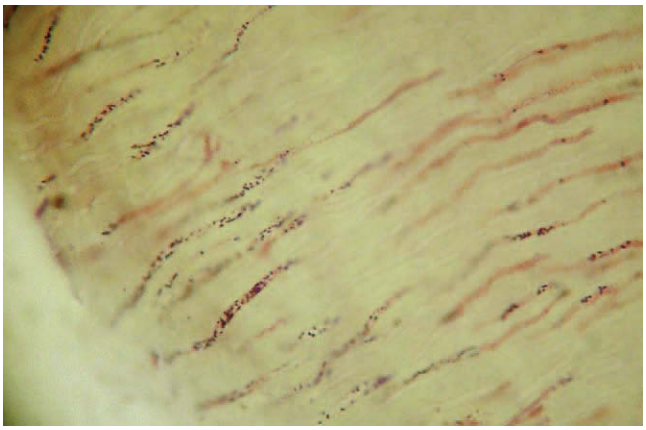


FIG. 15-27 Dentinal tubule infection by *Enterococcus faecalis* in dog's teeth after experimental infection. Notice that cells invaded the entire extent of some tubules up to the cementum (Brown and Brenn staining, ×1000).

For a given microorganism to survive in treated canals, 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[88,239] (Fig. 15-27), can enable it to escape the action of endodontic instruments and irrigants used during chemomechanical preparation.[88,238] Moreover, its ability to form biofilms in root canals can be important for its resistance to and persistence after intracanal antimicrobial procedures.[53] *Enterococcus faecalis* is also resistant to calcium hydroxide[32]; 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.[62] Unlike most putative endodontic pathogens frequently found in primary infections, *E. faecalis* may colonize root canals in single infections,[290] 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.[107] Indeed, *E. faecalis* can enter a so-called viable but non-cultivable (VBNC) state,[136] which is a survival mechanism adopted by several bacteria when exposed to unfavorable environmental conditions.[137] In 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.[66] It also has the capacity to recover from a prolonged starvation state in obturated canals,[226] suggesting that viable cells of this species entombed at the time of root filling 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 not proven. In fact, the status of *E. faecalis* as the main causative agent of endodontic failures has been questioned by the following findings from studies carried out in independent laboratories:

- Despite being easily cultivated, *E. faecalis* is not detected in all studies evaluating the microbiota of root canal–treated teeth with posttreatment disease.[39,213]
- Even when present, *E. faecalis* is rarely one of the most dominant species in retreatment cases.[205,209,221]
- *E. faecalis* has been found not to be more prevalent in root canal–treated teeth with lesions when compared to treated teeth with no lesions.[111,324]

Other bacteria found in root canal–treated teeth with apical periodontitis include streptococci and some fastidious anaerobic bacterial species: *P. alactolyticus, Propionibacterium propionicum, Filifactor alocis, Dialister pneumosintes, Dialister invisus, Tannerella forsythia, P. micra, Prevotella intermedia,* and *Treponema denticola** (Fig. 15-28). 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.[221] Some still uncultivated phylotypes, such as *Bacteroidetes* clone X083 and *Synergistes* clone BA121, can be among the most prevalent taxa in treated canals[205,221] (see Fig. 15-28). As-yet-uncultivated bacteria can also dominate the community, helping explain why culture studies failed 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.[209,221] 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.† 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.[264] *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[228,229,258] and resistance to calcium hydroxide.[309,311]

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

*References 82, 158, 197, 205, 209, 221, 252, 253, and 290.

†References 39, 60, 158, 159, 190, 197, 252, and 290.

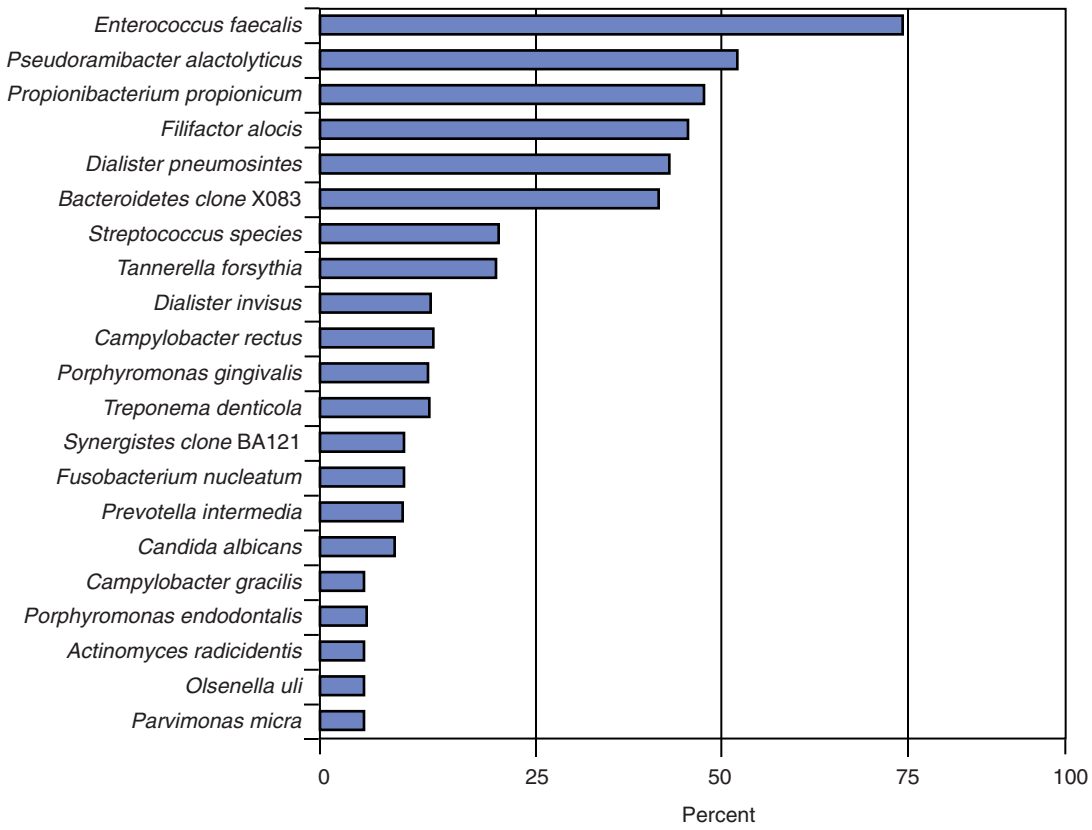


FIG. 15-28 Prevalence of microorganisms detected in root canal–treated teeth with posttreatment disease. Data from the authors' studies using a taxon-specific polymerase chain reaction assay. *(From references 252 and 253.)*

bacteria from the root canal. There is, however, another form of extraradicular infection which, unlike the acute abscess, is usually characterized by absence of overt symptoms. This condition entails the establishment of microorganisms in the periradicular tissues, either by adherence to the apical external root surface in the form of biofilm structures[169,297] or by formation of cohesive actinomycotic colonies within the body of the inflammatory lesion.[92] These extraradicular microorganisms have been discussed as one of the etiologies of persistence of apical periodontitis lesions in spite of diligent root canal treatment.[234,299]

Certain situations permit intraradicular bacteria to reach the periradicular tissues and establish an extraradicular infection.[235] Some may 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 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. 15-29). 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. 15-29). It has been suggested that in some cases, the extraradicular segment becomes independent of the intraradicular component of the infectious process.
- Due to bacterial persistence in the apical periodontitis lesion after remission of acute apical abscesses. 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, usually associated with an actively draining sinus tract.
- 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.

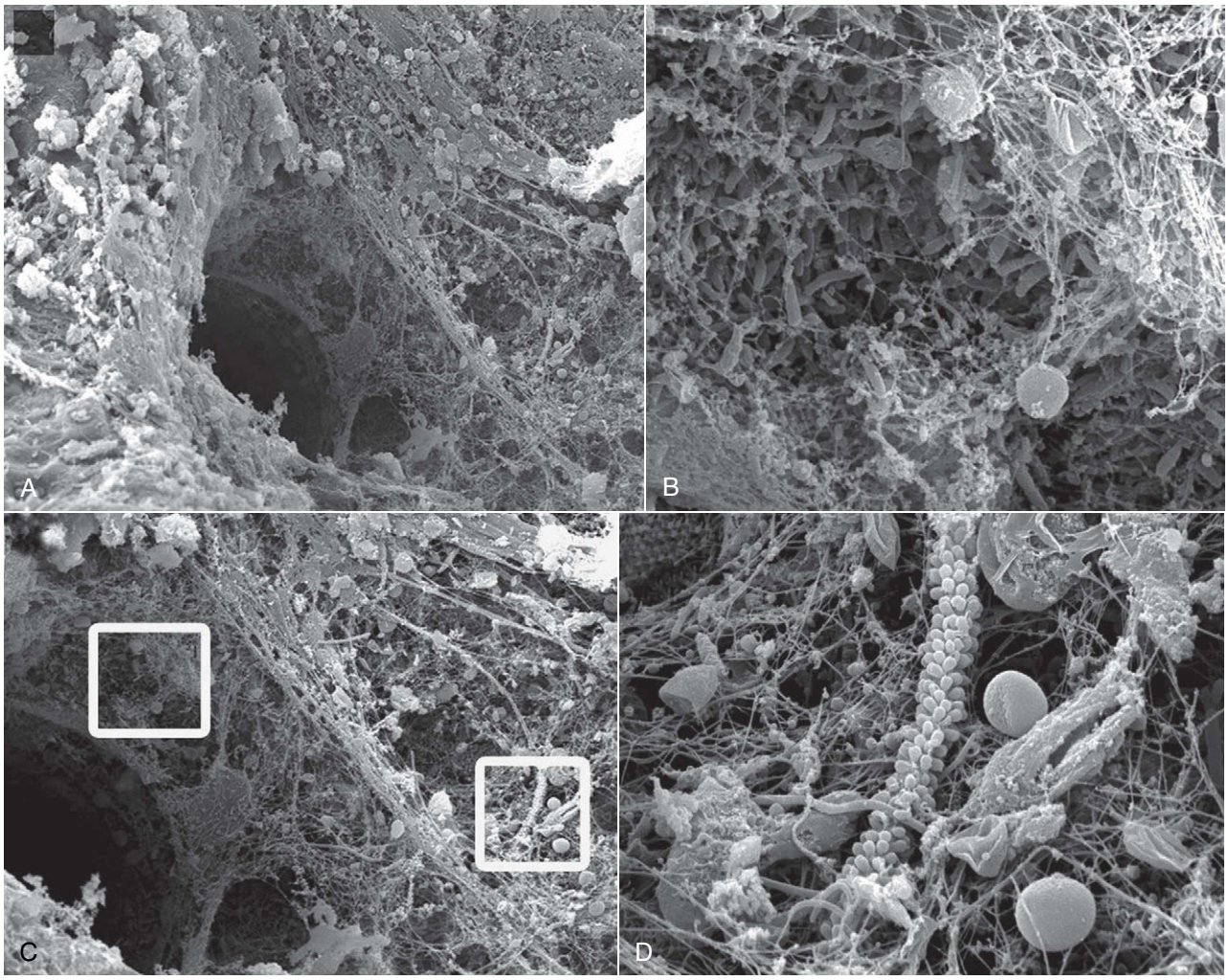


FIG. 15-29 Scanning electron micrograph showing extensive bacterial colonization in the very apical part of the canal, near and at the apical foramen (**A-B,** ×550 and ×850, respectively). **C,** Higher magnification of *left inset* in **B** showing a bacterial biofilm adhered to the very apical canal walls (×3700). **D,** Higher magnification of *right inset* in **B** showing a fully developed "corn cob" in tissue meshwork adjacent to the apical foramen (×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.)*

Conceivably, the extraradicular infection can be dependent on or independent of the intraradicular infection.[234] The acute apical abscess is the main example of dependent extraradicular infection. 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 suggested that the main bacterial species implicated in independent extraradicular infections are *Actinomyces* species and *P. propionicum,* in a pathologic entity named *apical* (or periapical, periradicular) *actinomycosis*[33,92,270,291] (Fig. 15-30). These bacteria form cohesive colonies that may be collectively resistant to phagocytosis.[65] 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 proven.[202,247]

Except for apical actinomycosis and cases evincing sinus tracts, it is still controversial whether chronic apical periodontitis lesions can harbor bacteria for very long beyond initial tissue invasion.[11] Studies using culture-dependent[283,298,315] or culture-independent molecular biology methods, such as checkerboard hybridization[76,285] and FISH,[284] have reported the extraradicular occurrence of a complex microbiota associated with apical periodontitis lesions that do not respond favorably to the root canal treatment. Anaerobic bacteria have been reported to be the dominant microorganisms in several of those lesions.[283,285] 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 extraradicular infections were dependent on or independent of an intraradicular infection.

The incidence of extraradicular infections in untreated teeth is conceivably low,[167,243] which is congruent with the high success rate of nonsurgical root canal treatment.[269] Even in root canal–treated teeth with recalcitrant lesions in which a higher incidence of extraradicular bacteria has been reported,

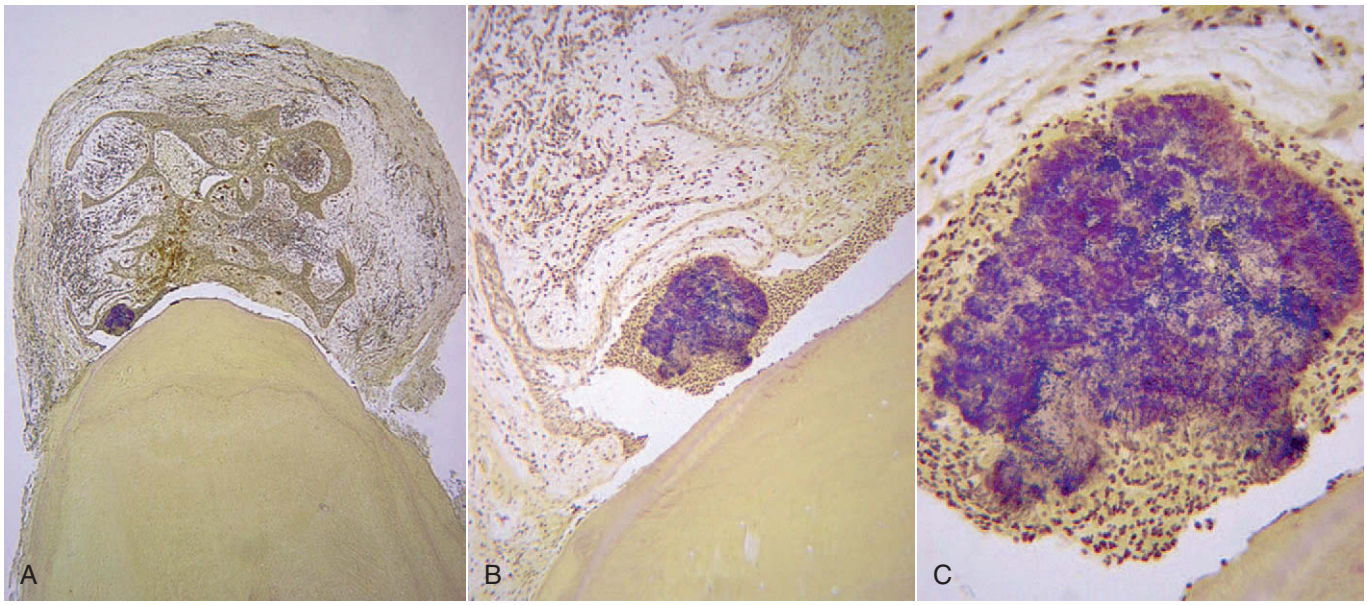


FIG. 15-30 Bacterial aggregate in an epithelialized periradicular lesion, suggestive of actinomycosis. This case presented a severe flare-up in the past, but the tooth was asymptomatic at the time of extraction. **A,** Section not passing through the canal; note cyst cavity and bacterial aggregation on the *left* (×25). **B,** Detail of area with colony (×100). **C,** Higher magnification of colony, which is surrounded by neutrophils (×400). Sections stained with a Taylor-modified Brown and Brenn technique. *(Courtesy Dr. Domenico Ricucci.)*

a high rate of healing following retreatment[269] 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 studies investigating the microbiologic conditions of root canals associated with persistent apical periodontitis.[158,197,209,252,290] Based on such data, it is reasonable to assume that most extraradicular infections observed in association with root canal–treated teeth must have been fostered by an intraradicular infection.

THE FOCAL INFECTION THEORY

Focal infection consists of an infectious disorder caused by microorganisms or their products that have disseminated from a distant body site (focus of infection).[179] Microorganisms from an infected oral site might spontaneously or after dental procedures gain entry into the blood circulation and cause disease in remote sites. The relationship of oral infections and systemic diseases is not a new issue; in 400 BC, Hippocrates reported that a patient was cured of arthritis after tooth extraction.

In a paper published in 1891, Miller described several human diseases, including non-oral diseases, which had been traced to the action of oral microorganisms.[154] He did not necessarily recommend extracting teeth considered to be a focus of infection, and indeed suggested treatment of infected root canals. Conservative dentistry suffered a severe attack at the beginning of the 20th century, after spread of the concept of oral sepsis into the medical literature by the English physician William Hunter in 1900.[103] Hunter believed that oral sepsis was responsible for several infectious diseases in the body, including osteomyelitis, gastritis, middle-ear suppurations, tonsillitis, endocarditis, meningitis, nephritis, and other septic conditions.[104] In 1900, he wrote:

> *The more I study it [oral sepsis as a cause of medical diseases] the more impressed I am, at once with its importance, and with the extraordinary neglect with which it is treated alike by physicians and surgeons.… I confess I think it urgent, in the interests of the many sufferers from gastritis as well as in the interests of those suffering from pyogenic conditions generally, that some similar steps be taken with regard to the mouth—the chief channel of access, in my judgement, of all pyogenic infections.*[103]

In 1911, he stressed the oral sepsis concept still more emphatically and attacked American dentists:

> *It is an all-important matter of sepsis and antisepsis that concerns every branch of the medical profession, and concerns very closely the public health of the community.… My clinical experience satisfies me that if oral sepsis could be successfully excluded, the other channels by which medical sepsis gains entrance into the body might almost be ignored.… No one has probably had more reason than I have had to admire the sheer ingenuity and mechanical skill constantly displayed by the dental surgeon. Such are the fruits of this baneful so-called "conservative dentistry." … The title that would best describe the dentistry here referred to would be that of "septic dentistry." Conservative it is, but only in one sense. It conserves the sepsis which it produces by the gold work it places over and around the teeth, by the satisfaction which it gives the patient, by the pride which the dentist responsible for it feels in his "high-class American" work, and by his inability or unwillingness to recognise the septic effects which it produces.*[104]

As a result of the focal infection theory, the avoidance of conservative dentistry in favor of tooth extractions was what

followed in dentistry for years. Fortunately, improvements in dental treatment (including root canal therapy), accompanied by advances in epidemiologic and biologic research giving scientific support to treatment modalities, significantly contributed to rescuing the fate of the dental profession. In 1952, an editorial in the *Journal of the American Medical Association* heralded the apparent end of the focal infection theory as it was acknowledged at that time:

> *After exerting a tremendous influence on the practice of medicine for a generation, the theory of focal infection in the past 10 or 15 years has fallen in part into disfavor. This has been due partly to the following observations that seem to discredit it: 1) Many patients with diseases presumably caused by foci of infection have not been relieved of their symptoms by removal of the foci; 2) many patients with these same systemic diseases have no evident focus of infection; 3) foci of infection are, according to some statistical studies, as common in apparently healthy persons as in those with disease.*[274]

Nevertheless, the last 2 decades have witnessed a revived interest in the focal infection theory, thanks to reports from epidemiologic studies suggesting the involvement of oral microorganisms with systemic diseases.[150,292] Although a plethora of focal infections having the oral cavity as their suspected source have been described, the most documented examples are bacterial endocarditis,[22] brain abscess,[132] and orthopedic joint infections.[179] Oral bacteria have also been implicated in aspiration pneumonia,[224] preterm low birth weight,[170] and coronary heart disease.[18] Infected root canals and apical periodontitis lesions have long been suggested to be potential foci of infection. Once again the focal infection concept is knocking on our doors. Fortunately, this time it can be evaluated and discussed on a scientific basis and has to go through the scrutiny of scientific methods.

To cause a focal disease, bacteria must travel from the focus of infection to a distant body site. This occurs via bacteremia, a condition in which viable bacteria are present in the blood circulation. There is no evidence showing that bacteremia spontaneously arises from infected root canals associated with a chronic apical periodontitis lesion. On the other hand, bacteremia can occur in cases of acute apical abscesses and during the treatment of infected root canals or periradicular surgery.* It has been shown that it is far more probable that bacteremia occurs if root canal procedures are performed beyond the apical foramen than when maintained within the root canal.[21] Bender et al.[21] reported that the incidence of bacteremia was zero if the instrumentation remained within the canal and 15% if it extended beyond the apical foramen. Even so, the bacteremia following endodontic procedures was demonstrated to last no longer than 10 minutes, as a result of competent clearance of bacteria from within the circulation. Using improved anaerobic cultivation methods, Baumgartner et al.[12,13] revealed that nonsurgical root canal treatment resulted in a lower incidence of bacteremia (3%, as a result of overinstrumentation) than surgical flap reflection (83%), periradicular curettage (33%), or tooth extraction (100%). In a study that performed intentional instrumentation beyond the apex, a 34% to 54% incidence of bacteremia was reported.[108]

However, it has been demonstrated that bacteremia can occur even if instrumentation is maintained within the root canal system. Debelian et al.[49] investigated the incidence of bacteremia following endodontic treatment of teeth with apical periodontitis lesions. In the treatment of half of the patients, the first three reamers (sizes 15, 20, and 25) were used to a level 2 mm beyond the root apex, while in the other half, instrumentation ended inside the root canal, 1 mm short of the apex. Bacteremia was observed in 54% of the cases where instrumentation through the foramen occurred and in 31% of the cases where instrumentation was confined to the canals. No statistically significant difference was found when the frequency of bacteremias in the two groups was compared. This may be explained by the fact that all instrumentation techniques induce apical extrusion of debris, some more than others, even when instrumentation is confined to the interior of the canal.[4,102,140,201] If debris is infected, bacteria are propelled into the periradicular tissues and then can enter the circulation. In a study where instrumentation was accomplished short of the apical foramen and patency files were not utilized,[223] culture analysis revealed detectable bacteremia in 30% of the patients who had no positive preoperative control blood sample.

There is no doubt that root canal procedures can induce bacteremia. The questions now are at what magnitude (number of bacterial cells in the blood) bacteremia occurs, how long it persists, and whether endodontic bacteria can cause disease at distant sites. It is very difficult to prove that a given oral microorganism is the causative agent of a focal infection. Although it has been demonstrated that the microbial species present in the blood of patients undergoing root canal treatment are of the same clonal types as those present in their root canals,[48] such findings only mean root canal treatment can cause bacteremia, not that microorganisms from the root canal cause disease in remote sites of the body. For bacteria present in the bloodstream to reach other body sites and induce disease, they must (1) survive the host defenses in the blood vessels as well as in the distant anatomic site, (2) encounter predisposing conditions in the distant site for their attachment and further colonization, (3) reach sufficient numbers to induce disease, and (4) possess an array of virulence factors that can inflict direct or indirect damage to the host tissues.

Establishing a causal link between oral disease and disorders in remote parts of the body depends on some important factors. Perhaps one of the most important is determining whether the microorganism associated with the medical disorder is genotypically the same as the suspected oral microorganism. In addition, the onset of the medical disease should follow the onset of the oral disease. In cases where a dental procedure is suspected of causing bacteremia, the onset of the medical disease should be within the incubation period.[271]

It is apparent from well-conducted studies that oral bacteria are rarely a cause of systemic disease.[179] Periodontal pathogens seldom cause endocarditis, with about 100 reported cases due to *Aggregatibacter actinomycetemcomitans,* two due to *Prevotella oralis,* one due to *Prevotella bivia,* one due to black-pigmented anaerobic bacteria, and five due to *Veillonella* species.[175,189] Except for *A. actinomycetemcomitans,* which has been infrequently found in endodontic infections,[256] the other

*References 12, 13, 21, 50, 131, and 163.

species have been isolated from infected root canals or apical abscesses. Obligate anaerobic bacteria from the oral cavity do not appear to survive well in other body locations, and viridans group streptococci, considered the principal oral culprits in endocarditis, are not primary pathogens but rather opportunistic bacteria that usually require altered biologic tissues to induce disease.[175]

Culture-independent techniques for bacterial identification have revealed previously unsuspected degrees of diversity in the oral microbiota.[119,185] Since many oral (and endodontic) bacteria remain to be cultivated and then fully characterized, their pathogenicity and involvement in causation of disease have yet to be elucidated. A study using molecular technology revealed a large amount of bacterial DNA in blood specimens from healthy individuals,[168] and many of the DNA sequences detected were from uncultivated and unknown bacteria. The presence of bacterial DNA in the blood has important implications for a possible role of previously uncharacterized bacterial species in some diseases, sometimes distant from the focus of infection. It would appear that to date, no molecular study has been performed to detect the occurrence of uncultivated bacterial phylotypes from the oral cavity in bacteremias following endodontic procedures. In addition, no study has evaluated the presence of those phylotypes in remote diseases in the body. Therefore, future research is warranted to ascertain whether uncultivated bacteria from the oral cavity can be involved in focal diseases.

Although there is no definitive evidence that bacteria from infected root canals can cause systemic diseases after bacteremia, there is a potential risk in certain patients, so it would be prudent to avoid situations that could predispose to bacteremias, such as overinstrumentation. Overinstrumentation induces damage to the periradicular tissues, affecting cells, extracellular matrix, and vessels; when it occurs during preparation of infected root canals, large numbers of bacteria can also be carried into the periradicular tissues. Bacteria introduced into periradicular tissues can enter injured vessels, and a bacteremia ensues. It has been postulated that lymphatics and not blood vessels (where the pressure gradient is outward and not inward after trauma) may be the primary means of entry of oral bacteria into the blood.[179] In addition to a higher risk of bacteremia, as alluded to earlier, bacteria pushed into the periradicular tissues during overinstrumentation may cause postoperative pain or even the failure of root canal treatment.[235] For all these reasons, overinstrumentation should be avoided.

The focal infection theory has remained controversial owing to the lack of indisputable evidence regarding the causal relationship between endodontic infections and other medical conditions. A cross-sectional study[72] revealed that there was no association between coronary heart disease and root canal–treated teeth or teeth with apical periodontitis. Nonsurgical endodontics is perhaps the least likely dental treatment procedure to produce a significant bacteremia in either incidence or magnitude.[179]

One should bear in mind that bacteremia can occur naturally as a result of normal daily activities, including toothbrushing and mastication.[8,87,139] Because of the 1000 to 8000 times greater chance of any bacteremia originating from normal daily activities, determining whether bacteremia has emanated from an endodontic intervention or a time before or after it is impossible.[179] Whatever the origin, bacteremias are usually transient. Detection of bacteria after dental procedures is significant after 10 minutes, and some reports indicate that positive blood cultures can be detected for as long as 30 minutes after the procedure.[9,96,138,198,199] However, bacteremia caused by some pathogenic species has been observed to persist for at least 60 minutes after brushing and extraction without antibiotic prophylaxis.[139]

In the absence of clear evidence regarding the effects of bacteremia in some compromised patients, and before elucidation of the speculative involvement of uncultivated bacteria, empirical consensus indicates that antibiotic prophylaxis should be performed in patients at risk of developing infective endocarditis. Antibiotic prophylaxis should also be considered for immunosuppressed patients, individuals with indwelling catheters, or patients with orthopedic prosthetic devices. Such individuals may receive some benefit from antibiotic prophylaxis, although there is unclear evidence in this regard.[175]

TREATMENT OF ENDODONTIC INFECTIONS—RATIONALE

The ultimate goal of endodontic treatment is either to prevent the development of apical periodontitis or, in cases where disease is present, to create adequate conditions for periradicular tissue healing. Taking into account the microbial etiology of apical periodontitis, the logical goals of the endodontic treatment are then to eliminate or at least substantially reduce the microbial population within the root canal system (through disinfection means) and to prevent microorganisms from infecting or reinfecting the root canal or the periradicular tissues (through aseptic means and a tight coronal seal provided by both the root canal filling and the permanent coronal restoration). The success of the endodontic treatment will depend on how effective the clinician is in accomplishing these goals.

Inasmuch as apical periodontitis has a polymicrobial etiology, bacterial community profiles significantly vary from one individual to another,[219] and differences are even more pronounced when samples are taken from different geographical locations,[143,204] endodontic infections should be treated using a broad-spectrum, nonspecific antimicrobial strategy with the potential to reach the most likely members of the endodontic bacterial community.

The privileged anatomic localization of the root canal system places bacteria entrenched in it beyond the reach of the host's defenses. Endodontic infections can only be treated by means of professional intervention using both chemical and mechanical procedures. The main steps of endodontic treatment involved with infection control are represented by the chemomechanical preparation and the interappointment medication. In this regard, the chemomechanical preparation is of paramount importance for root canal disinfection, since instruments and irrigants act primarily on the main canal, which is the most voluminous area of the system and consequently harbors the largest numbers of bacterial cells. Bacterial elimination from the root canal is carried out by means of the mechanical action of instruments, the flow and backflow of the irrigant solution, and the antibacterial effects of irrigants. Several irrigants have been proposed over the years, but sodium hypochlorite (NaOCl) remains the most widely used, although

studies have revealed that instrumentation and irrigation with NaOCl per se do not suffice to predictably render root canals free of cultivable bacteria. About 40% to 60% of canals are still positive for bacterial presence after chemomechanical preparation using different NaOCl concentrations.* Chlorhexidine has been proposed as an alternative, but clinical studies showed that it is not superior to NaOCl with regard to antibacterial effectiveness.[265,306]

Because residual bacteria can adversely affect treatment outcome,[64,267] the use of an interappointment medication has been recommended to complement the antibacterial effects of chemomechanical procedures and eliminate persisting bacteria.[151,245,268] Calcium hydroxide is arguably the most used intracanal medication. Addition of other antimicrobial mixing substances can enhance the effectiveness of calcium hydroxide.[244] Studies have shown that intracanal medication with a calcium hydroxide paste may be necessary to supplement the antibacterial effects of chemomechanical procedures and predictably render root canals free of cultivable bacteria before filling.[230,240,245,246,268]

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 (see Fig. 15-15). 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 very 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 is serving as a *focus of infection* because it leads to periradicular infection and secondary (i.e., metastatic) spread to the fascial spaces of the head and neck, resulting in cellulitis and systemic signs and symptoms of infection.

In such cases, treatment involves incision for drainage and root canal treatment of the involved tooth to remove the source of the infection. Antibiotic therapy may be indicated with 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.

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. 15-31, *A*). For example, if the source of the infection is a mandibular molar and the apices of the molar lie closer to the lingual cortical plate and above the attachment of the mylohyoid muscle of the floor of the mouth, the purulent exudate breaks through the lingual cortical plate into the sublingual space. If the apices lie below the attachment of the mylohyoid muscle, the infection spreads into the submandibular space.

As described by Hohl et al.,[100] 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 between the buccal cortical plate, the overlying alveolar mucosa, and the buccinator muscle (posterior) or the mentalis muscle (anterior) (see Figs. 15-31, *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. 15-31, *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. 15-31, *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. 15-31, *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 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. 15-31, *G*) is the potential space between the mylohyoid muscle superiorly and the

*References 34, 151, 240, 245, 267, and 268.

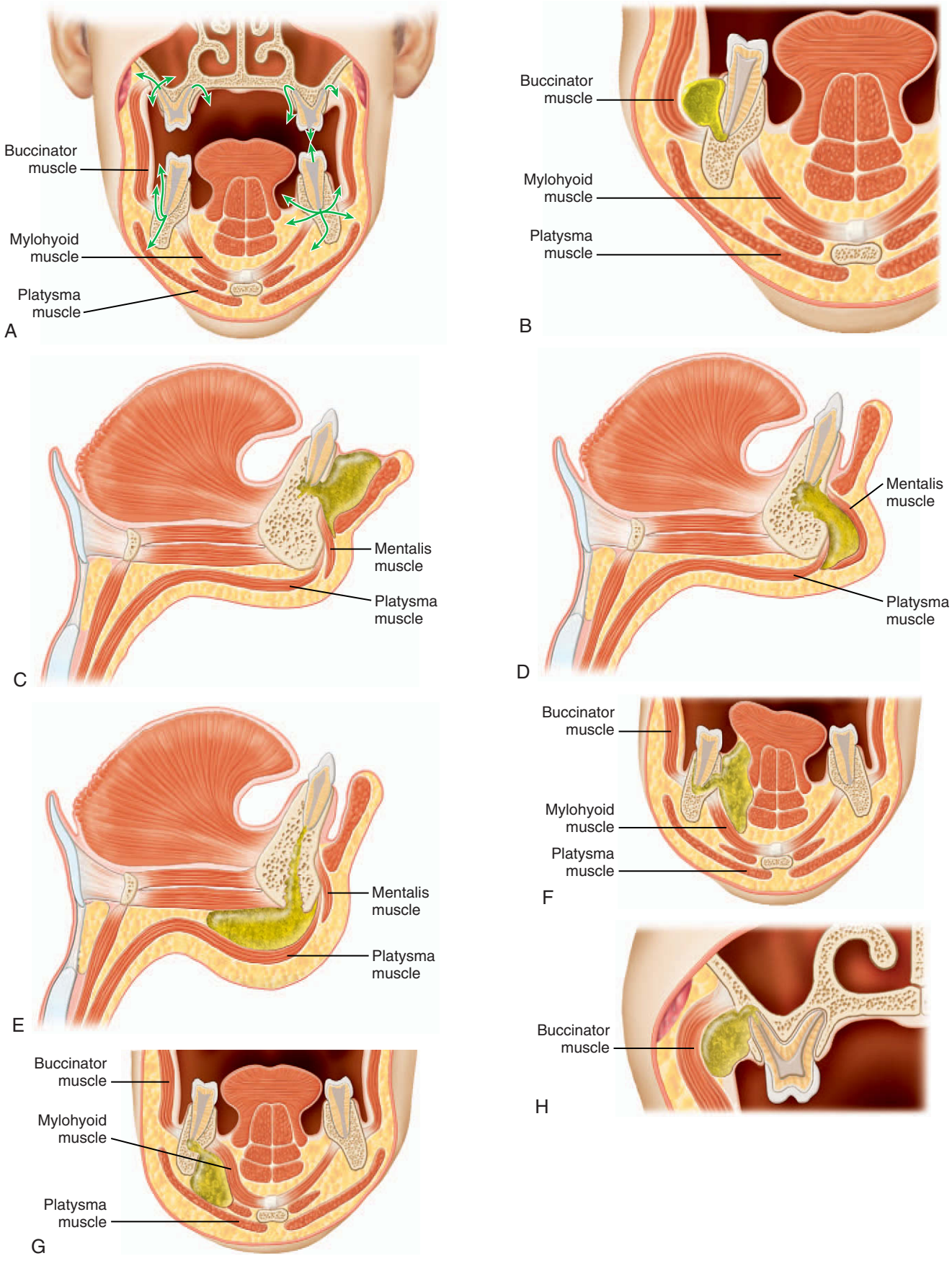


FIG. 15-31 A, Spread of odontogenic infections. **B,** Mandibular buccal vestibule (posterior tooth). **C,** Mandibular buccal vestibule (anterior tooth). **D,** Mental space. **E,** Submental space. **F,** Sublingual space. **G,** Submandibular space. **H,** Maxillary buccal vestibule.

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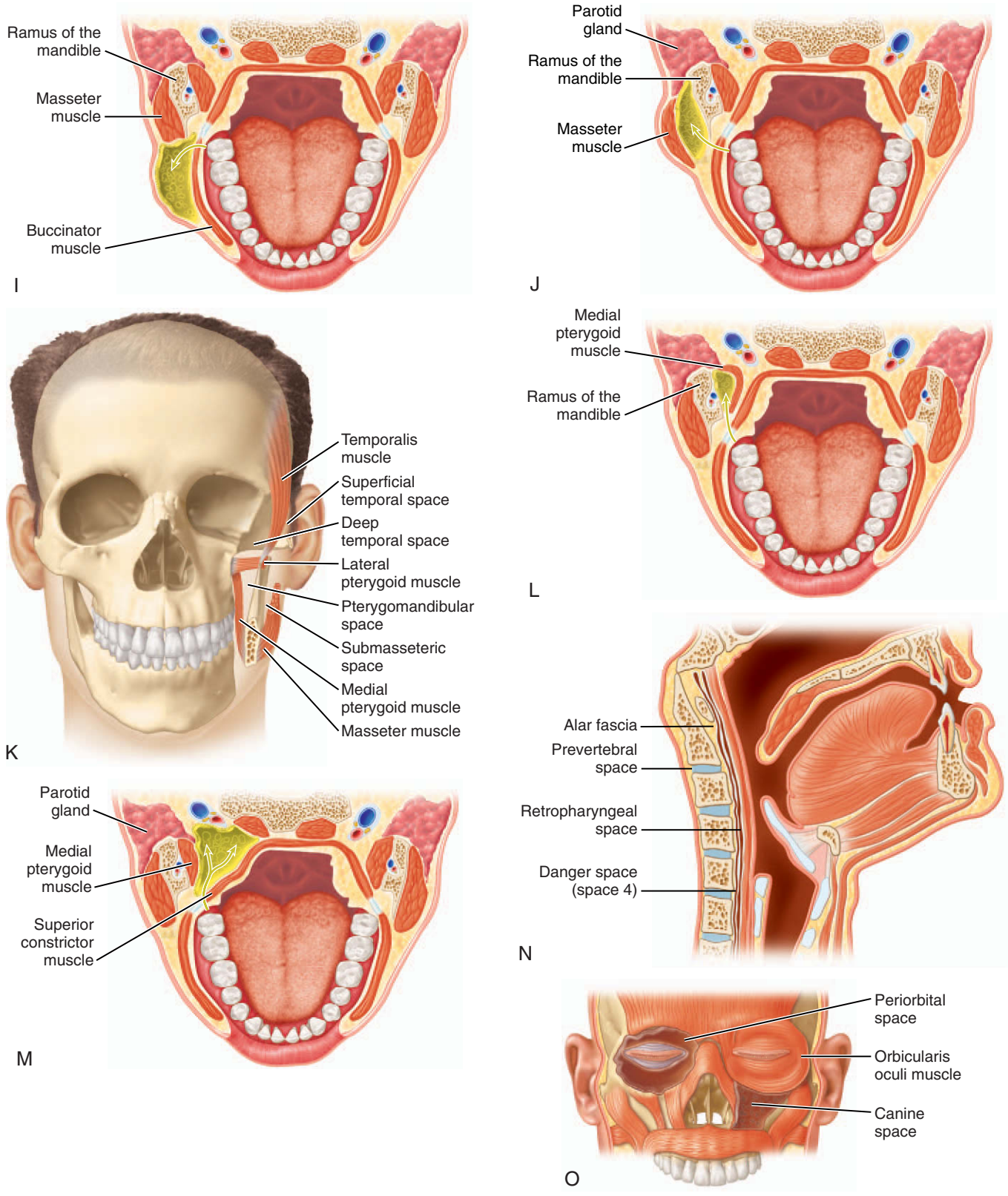


FIG. 15-31, cont'd I, Buccal space. **J,** Submasseteric space. **K,** Temporal space. **L,** Pterygomandibular space. **M,** Parapharyngeal spaces. **N,** Cervical spaces. **O,** Canine (infraorbital) and periorbital space.

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's 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. 15-31, *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. 15-31, *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. 15-31, *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. 15-31, *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. 15-31, *L*) is the potential space between the lateral surface of the medial pterygoid muscle and the medial surface of the ramus of the mandible. 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. 15-31, *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 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. 15-31, *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)[85] 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. 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).[130]

Swellings of the midface consist of four anatomic areas and spaces:

1. The palate
2. The base of the upper lip
3. The canine spaces
4. 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. 15-31, *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.

The *periorbital space* (see Fig. 15-31, *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 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.[171,323]

MANAGEMENT OF ABSCESSES AND CELLULITIS

The two most important elements of effective patient management are correct diagnosis and removal of the cause by controlling the endodontic infection. In an otherwise healthy patient, chemomechanical preparation of the infected root canals and incision for drainage of periradicular swelling usually prompt rapid improvement in clinical signs and symptoms. The majority of cases of endodontic infections can be treated effectively without the use of adjunctive antibiotics. The appropriate treatment is removal of the cause of the inflammatory condition.

Antibiotics are *not* recommended for irreversible pulpitis, acute 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).[70,99,165,196,312] 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 the treatment of pain.

Antibiotics in conjunction with appropriate endodontic treatment are recommended for progressive or persistent infections with systemic signs and symptoms such as fever (100° F [37.8° C]), malaise, cellulitis, unexplained trismus, and progressive or persistent swelling (or both). In such cases, antibiotic therapy is indicated as an adjunct to effective débridement of the root canal system, which is the 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 and/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 stab incision is made through the periosteum 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. Patients should improve rapidly after removal of the cause of the infection and drainage.

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 usually can 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 infections or persistent infections. Likewise, patients requiring extraoral drainage should be referred to a clinician trained in the technique.

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.[93] 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 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 in detriment of the susceptible portion of community. The emergence of multidrug-resistant strains of several bacterial species capable of causing life-threatening infections has been reported.[93,144,188,296,316] Antibiotic resistance among obligately anaerobic bacteria is increasing, with resistance to penicillins, clindamycin, and cephalosporins noted at community hospitals and major medical centers.[94,95]

Among oral bacteria, there have been a handful of 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 tetracy-

cline and amoxicillin, and in *A. actinomycetemcomitans* for amoxicillin and azithromycin.[124,303] Macrolides (erythromycin and azithromycin) have presented decreased activity against *Fusobacterium* and nonpigmented *Prevotella* species.[97,123,124] 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*.[24,31,68,91,304] Kuriyama et al.[123] 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.[124] 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.[31,68,91,304] Facultative bacteria such as *Capnocytophaga* and *Neisseria* species have also been detected among the beta-lactamase producers.[91] 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.[29]

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 and/or duration of therapy, and excessive use in prophylaxis.[177,178] 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.[144]

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.[177] Based on the earlier discussion, it becomes clear that the most important decision making in antibiotic therapy is not so much *which* antibiotic should be employed but *whether* antibiotics should be used at all.[176] One should bear in mind that antibiotics are very useful drugs classically used to treat or help treat infectious disease and provide prophylaxis in carefully selected cases.

The vast 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, so the source of infection is often unaffected by systemic antibiotic therapy. Antibiotics can, however, help impede spread of the infection and development of focal infections in medically compromised patients and be 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 replantation of avulsed teeth.

Selection of antibiotics in clinical practice is either empirical or based on the results of microbial susceptibility 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,[17,106,115,124] which make them first-line drugs of choice. Since 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. 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.[124] In patients allergic to penicillins or in cases refractory to amoxicillin therapy, clindamycin is indicated. Clindamycin has strong antimicrobial activity against oral anaerobes.[122,124,125]

To sum up, the risk/benefit ratio should be always weighed before prescribing antibiotics. Appropriately selected patients will benefit from systemically administered antibiotics. A restrictive and conservative use of antibiotics is highly recommended in endodontic practice, but indiscriminate use (including cases of pulpitis—no infection!) is contrary to sound clinical practice. This may cause a selective pressure and consequent overgrowth of intrinsically resistant bacteria, predisposing patients to secondary and superinfections and rendering drugs ineffective against potentially fatal medical infectious diseases. Future generations will thank us for today's conscientious and judicious use of antibiotics.

ACKNOWLEDGMENT

The authors acknowledge the outstanding work of Drs. J. Craig Baumgartner and Jeffrey W. Hutter in previous editions of this text. The sections on fascial space infections and management of abscesses and cellulitis were built upon their foundation work.

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CHAPTER 16

Regenerative Endodontics

KENNETH M. HARGREAVES and ALAN S. LAW

CHAPTER OUTLINE

OVERVIEW OF REGENERATIVE DENTISTRY
- Overview of Regenerative Endodontics

PRECLINICAL STUDIES ON REGENERATIVE ENDODONTICS
- Stem Cells
- Growth Factors/Morphogens
- Scaffolds
- Delivery System
- Summary of Basic Research on Regenerative Endodontics

CLINICAL STUDIES ON REGENERATIVE ENDODONTICS
- Historical Development of the Field
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OVERVIEW OF REGENERATIVE DENTISTRY

From a biologic perspective, the goal of endodontics is to prevent or treat apical periodontitis. An optimal way to accomplish this goal is to either maintain pulpal health in cases of pulpal inflammation or to regenerate healthy pulpal tissue in cases of pulpal necrosis. Thus, there is considerable interest in research and clinical studies aimed towards regeneration of a functional and healthy pulp-dentin complex.[68,109] This chapter will review the current status of regenerative endodontic procedures with an emphasis on biologic principles and the advantages and limitations of currently available clinical procedures.

The history of dentistry has largely involved the prosthetic replacement of lost or diseased tissues, including endodontic obturation of root canal systems with inert materials (see Chapter 10). 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 growth and differentiation factors which hasten or induce natural biologic regeneration. Many of these concepts have emerged from the burgeoning field of tissue engineering, which emphasizes the spatial assembly of distinct stem cells, growth factors/morphogens, and scaffolds to form a functional tissue or organ.[62]

Pioneering work supporting the concept of regenerating dental tissues was reported more than 50 years ago when Dr. B.W. Hermann described the application of calcium hydroxide ($Ca[OH]_2$) for vital pulp therapy,[47] and Professor Nygaard-Østby evaluated a revascularization method for reestablishing a pulp-dentin complex in permanent teeth with pulpal necrosis (see later).[82] Over the last several decades, the scope and clinical application of regenerative dental procedures has continuously advanced to now include guided tissue or bone regeneration (GTR, GBR) procedures and distraction osteogenesis,[15,17,86] the application of platelet-rich plasma for bone augmentation,[60] Emdogain for regeneration of periodontal tissues,[44] recombinant human bone morphogenic protein (rhBMP) for augmentation of bone,[34] and preclinical trials on the use of fibroblast growth factor 2 (FGF-2) for periodontal tissue regeneration.[118] At the beginning of the 21st century, we are seeing the potential of these therapies for dental practice, including endodontics. For example, pulp, dentin, and enamel can be regenerated using scaffold material and stem cells,[29,51,141] and tooth crowns can be regenerated using the primordium of embryonic oral epithelium and adult bone-marrow stem cells.[87] Adult bone marrow stem cells are capable of differentiating directly into ameloblasts when mixed with embryonic epithelial cells,[49] dissociated cells from tooth germs can be mixed with agar and collagen (i.e., scaffolds) to form crown, root, and periodontal structures in adult hosts,[49,71] and postnatal stem cells isolated from extracted wisdom teeth can be used to regenerate tooth roots and periodontal ligaments that can support synthetic crowns.[113] Regenerative dental procedures are emerging as a vital, evolving field of dental care.

Overview of Regenerative Endodontics

Regenerative endodontics has been defined as biologically based procedures designed to replace damaged structures such as dentin, root structures, and cells of the pulp-dentin complex.[68] Similar to most rapidly evolving fields, the scope and depth of basic research has outpaced the translation of this knowledge into clinical trials and established dental procedures. It is important to review both basic and clinical studies for a full understanding of the scope, limitations, and potential of this field. To understand the implications of preclinical research for clinical practice, it is important to briefly review the development of teeth.

The forming tooth progresses through a succession of morphologic stages—bud, cap, and bell—resulting in the formation of the crown, root, and periodontium (see also Chapter 12). Tooth development is regulated by a series of reciprocal interactions involving both ectodermal and mesenchymal stem cells.[119] Ectodermal stem cells differentiate into ameloblasts, and mesenchymal stem cells, also known as *ectomesenchymal stem cells* owing to their neural crest origin, differentiate into odontoblasts. One important requirement for regeneration of pulp tissues is to obtain stem cells capable of differentiating into odontoblasts.

Stem cells are often viewed as relatively undifferentiated cells capable of self-renewal/expansion (i.e., can divide many times) and able to differentiate into specialized cell types. The range of differentiation ("potency") is an important property of stem cells and serves as the basis for classifying cells as *totipotent*—capable of differentiating into any cell type—or *pluripotent, multipotent,* or *unipotent*—characterizing more restricted abilities for differentiation. In general, considerable research has focused on totipotent embryonic stem cells that have maximal capacity for differentiation, but ethical, legal, and medical (tissue-rejection) issues can render these cell types unsuitable for clinical development.[68] In contrast, postnatal (i.e., adult) stem cells are more readily available, are already applied in clinical practice (e.g., bone marrow transplants, etc.), and when used in an autologous application, will not induce immune responses such as tissue rejection. Many of these stem cells appear to satisfy criteria for multipotency, since they can differentiate into cells that express phenotypic markers for odontoblasts, neurons, and muscle or adipose cells.[52]

Nakahara has summarized potential methods for regenerating an entire tooth. The first approach, incorporating principles of tissue engineering, involves seeding appropriate stem cells onto scaffolding materials, such as has been used for periodontal regeneration.[70] This approach may be controlled by the addition of specific growth factors and/or signaling molecules. The second approach involves replicating the natural developmental processes of embryonic tooth formation. In this approach, artificial tooth germs are transplanted into the bodies of animal hosts where there is enough blood flow to support tissue formation. Although either approach may evolve into a method capable of generating entire teeth, it should be appreciated that the natural development of permanent human teeth takes years to complete. Regenerating an entire tooth from a patient's own stem cells may not be clinically practical.

One alternative approach would be to regenerate a functional pulp-dentin complex *within* a patient's existing permanent tooth. This would restore natural functions, such as formation of replacement dentin and maintenance of tissue immunity and neural sensation, to teeth that otherwise would require endodontic obturation with inert materials or possibly extraction. One key advantage is that the pulp occupies a relatively small volume (about 0.1 to 2 ml) and, compared to complex organs such as the heart or liver, has a relatively simple cytoarchitecture that can be approximated as a core of loose connective tissue surrounded by a layer of odontoblasts attached to dentin. From a tissue-engineering perspective, the dental pulp may be a relatively easier tissue to regenerate.

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.[42,62,74] In this section, we will review each of these critical components in turn.

Stem Cells

As described earlier, 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). Pulpal mesenchymal stem cells are thought to be localized in the perivascular region and the cell-rich zone of Hohl adjacent to the odontoblastic layer; both have been proposed to serve as cell sources for replacement odontoblasts.[32,106] Thus, several cell types must be developed in order to form this core of loose connective tissue. The regeneration of loose connective tissue has been proposed to be similar to the process of the granulation phase of wound healing, with the local release of angiogenic growth factors such as vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF) playing key roles in coordinating angiogenesis and the development of loose connective tissue.[8] Interestingly, platelet-rich plasma has about a three- to sixfold increase in VEGF and PDGF and has been reported to increase rates of wound healing.[8,81,94,132] Additional studies have demonstrated that the application of recombinant human VEGF substantially increases the degree of revascularization of human dental pulp slices implanted into immunocompromised mice.[36] Given the large existing knowledge base on the regeneration of loose connective tissue, recent research has mainly focused on generation of the surrounding layer of odontoblasts.

At least five different types of postnatal mesenchymal stem cells have been reported to differentiate into odontoblast-like cells, including dental pulp stem cells (DPSC),[39] stem cells of human exfoliated deciduous teeth (SHED),[66] stem cells of the apical papilla (SCAP),[113,114] dental follicle progenitor cells (DFPC),[67] and bone marrow-derived mesenchymal stem cells (BMMSC).[12] Although many of these studies involve human samples collected from patients younger than 25 years of age, it should be noted that multipotent stem cells have been collected from the pulps of patients as old as 41.[50] Regenerative procedures may be applicable to at least young and middle-aged patients. To our knowledge, no study has evaluated the formation of odontoblast-like cells from patients older than 50 years of age, and this represents an important gap in knowledge. The identification of a differentiated cell as an odontoblast is not always straightforward, since this cell is similar to osteoblasts 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 osteoblasts.[131] Measuring only one or two characteristics of a cell might not conclusively identify whether the resulting cell is a true odontoblast. Even among odontoblasts, the phenotype varies in cells located in the apical (squa-

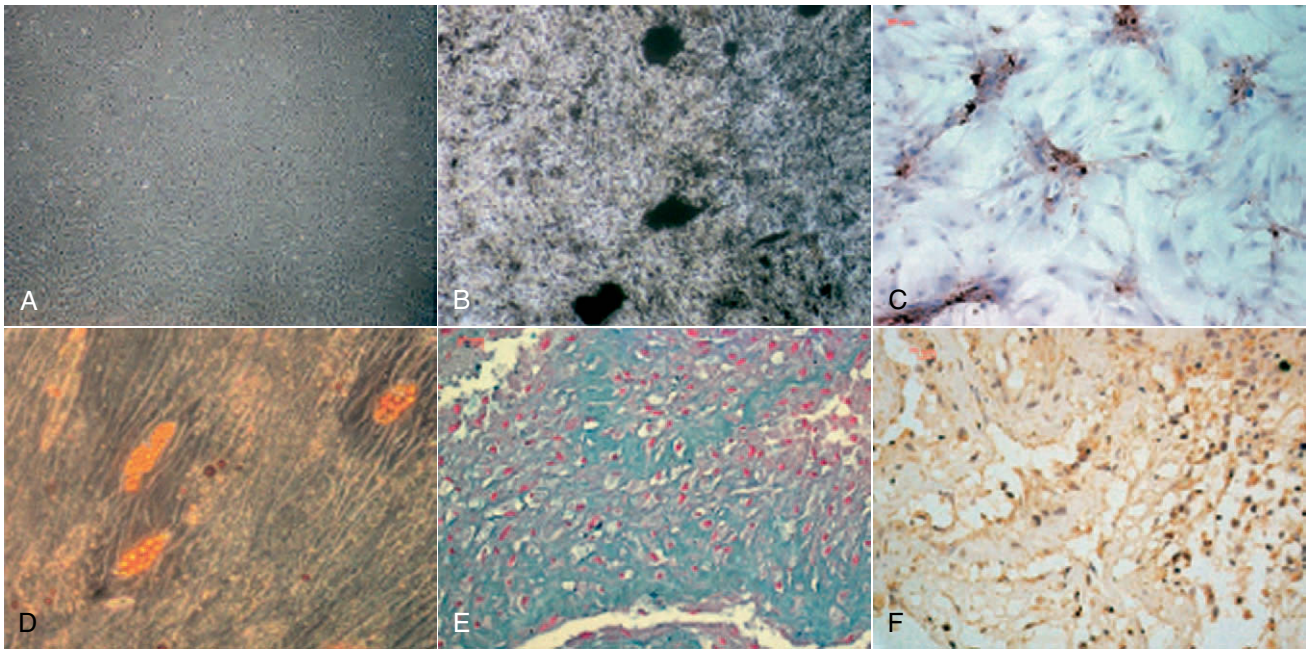


FIG. 16-1 Multilineage differentiation capacity of human dental pulp cells (DPCs). **A,** DPCs cultured in control medium (×40). Differentiation into odontoblast-like cells was shown by the deposition of a mineralized matrix indicated by von Kossa stain (**B;** ×100) and by the positive immunostaining of DSP (**C;** ×200). **D,** Adipogenic differentiation was shown by the accumulation of neutral lipid vacuoles stainable with Oil Red O (×100). Chondrogenic differentiation was shown by the secretion of cartilage-specific proteoglycans stainable with Alcian blue (**E;** ×400) and by the positive immunostaining of collagen type II (**F;** ×400). *(From Wei X, Ling J, Wu L, et al: Expression of mineralization markers in dental pulp cells.* J Endod *33:703, 2007.)*

mous shape) versus coronal (tall columnar) pulpal tissue. Importantly, molecular studies have identified many of the genes selectively expressed in odontoblasts,[64,89,90] and this knowledge is expected to aid future studies characterizing the conditions necessary for mesenchymal cells of multiple origins to differentiate into true odontoblasts. It is likely that definitive cellular identification depends upon both the morphology of the cell and an assessment of the expression of multiple genes. For accuracy, we will refer to cells that express mineralized nodules and DSP as *odontoblast-like*.

Growth Factors/Morphogens

Several growth factors have been evaluated for their ability to trigger the differentiation of selected mesenchymal stem cell populations into odontoblast-like cells. 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.[77,136] Although these patients were medically complex patients (e.g., renal failure) taking multiple drugs, the use of corticosteroids appeared to be associated with the observed increased activity of human odontoblasts. More recent studies have extended this general observation by demonstrating that the application of dexamethasone greatly increased the differentiation of human dental pulp cells into odontoblast-like cells.[54,131] This was particularly evident when dexamethasone was combined with 1,25-dihydroxyvitamin D_3.[54] 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 (Fig. 16-1).[131] 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 16-1).

Several of the approaches listed in Table 16-1 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.[95] 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.* Although human dentin contains many types of noncollagenous proteins, it is notable that transforming growth factor

*References 10, 72, 111, 112, 125, and 127.

TABLE 16-1

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[54]
Dexamethasone and Vitamin D_3	Human dental pulp	Odontoblast-like	In vitro × 8 weeks	Huang et al., 2006[54]
Dexamethasone and Ascorbate-2-phosphate and β-Glycerophosphate	Human or rat dental pulp	Odontoblast-like	In vitro × 3 weeks	Wei et al., 2007[131] Zhang et al., 2005[143]
Insulin and Indomethacin and 3-Isobatyl-1-methylxanthine (IMBX)	Human dental pulp	Adipocyte	In vitro × 19 days	Wei et al., 2007[131]
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[131]
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[130]
Bone morphogenetic proteins	Dental pulp	Odontoblast-like	In vitro	Saito et al., 2004[99] Sloan et al., 2000[108] Chen et al., 2008[19]
TGF-β1-3	Rat/monkey dental pulp	Odontoblast-like	In vitro	Sloan et al., 1999[110]
Demineralized dentin	Human or rodent pulp	Odontoblast-like	In vitro/in vivo	Smith et al., 1990[112] Smith et al., 2001[111] Tziafas, 2004[124]
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]

β 1 (TGF-β1) is the only TGF subtype detectable in human dentin.[144] Moreover, the application of ethylenediamine tetraacetic acid (EDTA) strongly exposed immunoreactive TGF-β1 from human dentin, with appreciably smaller activities released after treatment with $Ca(OH)_2$, sodium hypochlorite (NaOCl), mineral trioxide aggregate (MTA), or citric acid.[38,122,144] Moreover, dentin contains additional noncollagenous proteins that promote odontoblast differentiation[1] or angiogenesis.[97] Collectively these findings suggest that future clinical regenerative trials should evaluate whether an EDTA irrigation of the dentinal walls prior to an endodontic regenerative procedure results in improved clinical outcomes.

Scaffolds

An important component of tissue engineering is a physical scaffold.[76,104] 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. It is known that extracellular matrix molecules control the differentiation of stem cells,[16,138] and an appropriate scaffold might selectively bind and localize cells,[128] contain growth factors,[137] and undergo biodegradation over time.[142] Thus, a scaffold is far more than a simple lattice to contain cells.

Scaffolds can be classified as either natural or synthetic. Examples of natural scaffolds include collagen,[55,73] glycosaminoglycans, demineralized or native dentin matrix,[10,40,55,72,127] and fibrin. From the perspective of focusing on practical clinical applications, platelet-rich plasma (PRP) appears to satisfy several of these criteria. PRP is autologous, fairly easy to prepare in a dental setting, rich in growth factors, degrades over time, and forms a three-dimensional fibrin matrix.[8,9,57,85] In addition, these properties may play a role in the reported outcomes of classical revascularization methods applied to patients (see later).

The second major category of scaffolds is based upon synthetic materials. Examples include polylactic acid (PLA), polyglycolic acid (PGA), polylactic-coglycolic acid (PLGA),

polyepsilon caprolactone,[140] hydroxyapatite/tricalcium phosphate,[4] bioceramics,[139] titanium (in some[143] but not all studies[126]), and hydrogels such as alginate[28,35] or variants of polyethylene glycol (PEG). Moreover, the combination of scaffolds with certain growth factors/morphogens appears to be an important combination for optimal generation of an odontoblast-like cell. This is a critical domain of research for the development of regenerative endodontics as a predictable clinical procedure.

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.[37,46] If one were to inject cells along the entire coronal-apical extent of a root canal system, the vast majority of cells would be expected to succumb to tissue hypoxia. One alternative approach would be to inject a cell/scaffold/growth-factor mixture into the apical 1 mm of the root canal system and then "back-fill" the root canal system with a scaffold/growth-factor combination. Since 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. The questions require additional research effort.

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, it should be noted that emerging studies conducted in animal models are quite promising for regeneration of this pulp tissue. A recent preclinical study involved surgical placement of human root tips filled with various human stem cell/growth factor/scaffold combinations into immunocompromised mice.[51] This model permits 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 for odontoblast-like cell differentiation and the spatially appropriate formation of dentin-like material onto the root canal walls (Fig. 16-2). 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.[38] This and related

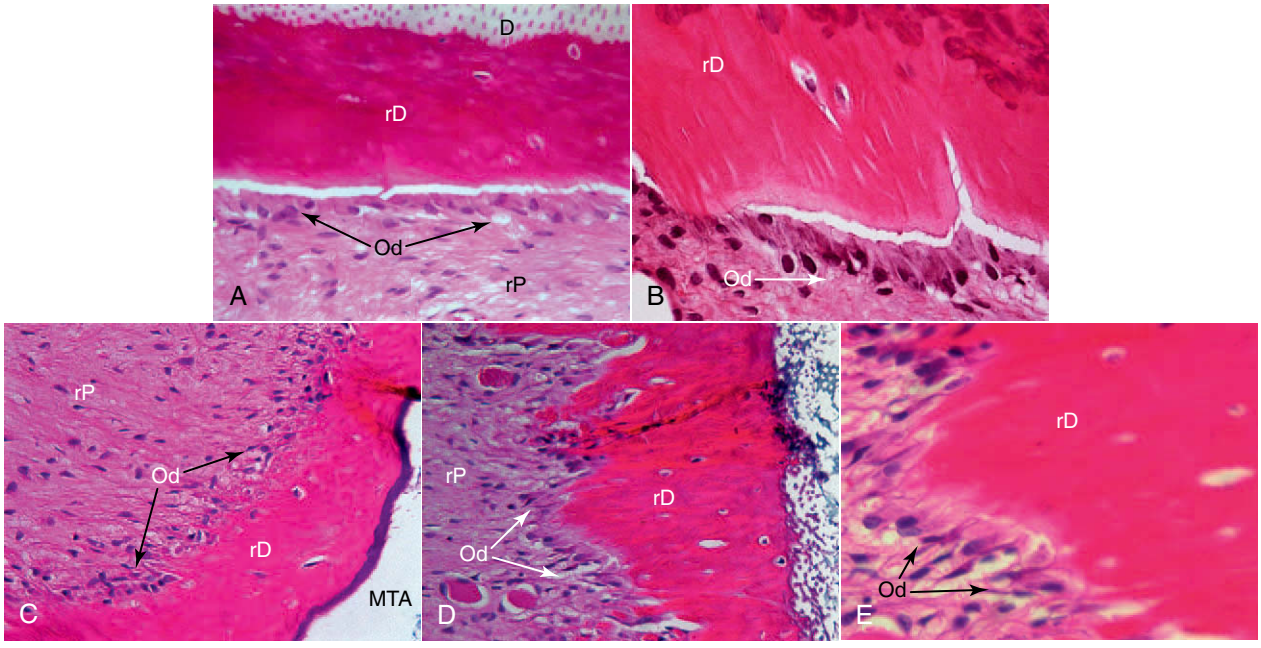


FIG. 16-2 Histologic analysis of a preclinical experiment evaluating regeneration of pulp tissue using dental pulp stem cells (DPSC) implanted with a resorbable polylactic-coglycolic acid (PLGA) scaffold into human root segments that were sealed on the coronal side with mineral trioxide aggregate (MTA) and implanted into immunocompromised mice for 4 months. *D,* Original dentin; *rD,* regenerated dentin-like tissue; *rP,* regenerated pulplike tissue. *Green arrows* in **A** indicate rD; *blue arrows* in **A** indicate the entrance of blood supply; *blue arrow* in **B** and **C** indicate the thin layer of rD under MTA cement; *blue arrows* in **F** and **G** indicate the junction of D and rD; *black arrow* in **G** indicates well-aligned odontoblast-like cells. Scale bars: **A** = 1 mm; **B** = 200 µm; **C-E** = 100 µm; **F** and **G** = 50 µm. *(From Huang G, Yamaza T, Shea LD, et al: Stem/progenitor cell-mediated de novo regeneration of dental pulp with newly deposited continuous layer of dentin in an in vivo model.* Tissue Eng *Part A, 2009 Sep 8. [Epub ahead of print].)*

studies provide strong impetus for clinical translational research evaluating various potential regenerative endodontic therapies.

CLINICAL STUDIES ON REGENERATIVE ENDODONTICS

To date, no published clinical trials have fully incorporated the tissue-engineering concepts described. Instead, studies over the last 50 years have focused on revascularization techniques, which share some features with the principles of regenerative tissue engineering. The key distinction is that in contrast to the focused delivery of cells/growth factors/scaffolds employed in tissue-engineering approaches, revascularization focuses on triggering bleeding into an empty root canal space with the hope that this will trigger a process similar to the role of the blood clot in triggering wound healing in surgical procedures.[82] These revascularization case series will be reviewed in this section.

Historical Development of the Field

Nearly 50 years ago, Nygaard-Østby evaluated whether pulp tissue could be regenerated in root canal systems. His approach was based on the known importance of the formation of a blood clot in wound healing[82,83] and involved laceration of the periapical tissue with an endodontic file. His first case series reported on nine patients aged 21 to 42 and a total of 17 teeth; there was a 13-day to 3-year follow-up prior to tooth extraction and histologic analysis.[82] Some of the cases had vital pulp tissue. In these cases, there was an aseptic access, the pulp was extirpated, and the apex was overinstrumented to evoke bleeding. Then the canals were obturated with Kloroperka N-O paste and a gutta-percha point. In the teeth with necrotic pulp tissue, the procedure was the same as in the teeth with vital pulp tissue, but the canals were irrigated and had interappointment treatment with sulfathiazole and 4% formaldehyde. Teeth were then extracted, and both clinical and histologic findings were reported (Fig. 16-3). In general, apical inflammation from

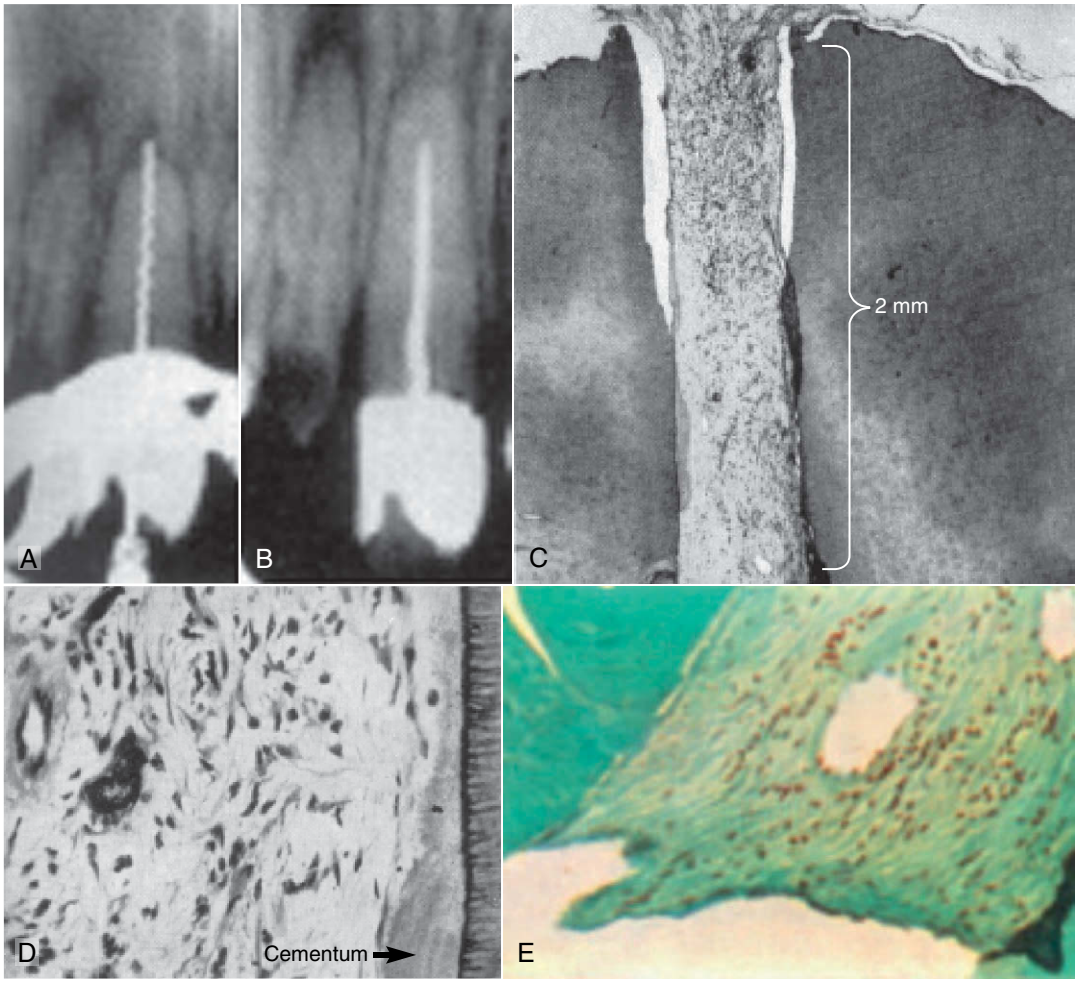


FIG. 16-3 Radiographic and histologic findings from a central incisor with a necrotic pulp, from Nygaard-Østby.[82] **A,** Evidence of a file going beyond the apex. There is also evidence of a radiolucency in the apical area. **B,** Second radiograph at 14 months, taken shortly before tooth was extracted, 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.)*

the overinstrumentation resolved by about 2 weeks. At the 1-month postoperative time point, the periodontal ligament tissue was healed. At 10 months, periapical bone was still being formed. The blood clot in the root canal systems gradually was replaced with granulation tissue, and then fibrous connective tissue. However, the growth of this tissue into the root canals was incomplete, and there was also histologic evidence of variable resorption of the dentinal walls along with the deposition of cementum. No newly formed dentin was observed. In his second case series on 47 teeth, histologic evidence of an ingrowth of vascularized fibrous connective tissue was observed in 80% (28 of 35) of teeth with vital pulps but only 8% (1 of 12) of teeth with necrotic pulps.[83] Although newly mineralized tissue was evident on some dentinal walls, it appeared to be cementum and not newly formed dentin. This finding of cementum deposition is consistent with recent histologic studies using contemporary revascularization procedures.[129] Taken together, most* but not all[101] studies of this period suggested that it is possible to create an environment that favors ingrowth of connective tissue into the débrided root canal system that is capable of depositing mineralized tissue on canal walls. However, the extent of tissue ingrowth was limited in most cases to the apical 2 to 3 mm of the root canal system, the mineralized tissue was often described as cementum, and inflammatory elements and/or resorption were also reported. These findings clearly did not replicate the pulp-dentin complex, and research in this area gradually subsided.

Clinical Procedures Related to Regenerative Endodontics

Since the report of this original body of research, there have been substantial fundamental advances in our understanding of canal disinfection, coronal microleakage, and the entire field of regenerative medicine. The development of these technologies and a new knowledge base has resulted in a recent surge in the publication of clinical procedures typically applied to immaturely developed (i.e., open apex) permanent teeth with a diagnosis of pulpal necrosis. With these publications has come some controversy regarding the terminology that should be used to describe the clinical procedure and outcomes. There are several procedures designed to treat the incompletely formed root that occur following endodontic procedures. *Apexification* is defined as a method to induce a calcified barrier in a root with an open apex or the continued apical development of an incompletely formed root in teeth with necrotic pulp tissue.[2] This is distinct from revascularization, since apexification does not attempt to regain vital tissue in the canal space. Rather, the outcome of an apexification procedure is establishment of an apical barrier against which an obturating material may be placed. A second term, *apexogenesis,* is defined as a vital pulp therapy procedure performed to encourage continued physiologic development and formation of the root end.[2] An important distinction is that apexogenesis is indicated for teeth in which there has been no loss of vascularity, thus no need to "revascularize" the canal space. Another term used to describe root development is *maturogenesis*.[133] In that the desired outcome of revascularization procedures is to set the stage for *physiologic root development,* the term that best describes revascularization outcomes.

*References 30, 65, 79, 80, 82, 83, and 116.

Revascularization can be broadly defined as the restoration of vascularity to a tissue or organ.[5] The use of this term was recently discussed in a letter to the editor of the *Journal of Endodontics*.[53] The authors stated that the term *revascularization* does not completely address the desired outcomes of regenerative endodontic procedures, because the desired outcome is regeneration of the pulp-dentin complex, rather than just revascularization of the canal space. Further, they suggested that "induced or guided tissue generation and regeneration" would be more appropriate terminology. Others have observed that "Guided tissue regeneration has some merit except that … we do not know what tissue fills the pulp space."[123] Given the lack of clinical and histologic evidence, it is difficult to settle on consistent terminology, and the ongoing development and application of tissue-engineering principles makes this a moving target that is likely to change with development of new clinical procedures. It is important to note that basic research and preclinical studies have identified many of the critical issues for regenerative endodontic procedures, yet case studies published to date continue to use revascularization methods coupled with recent advances in canal disinfection and new materials (e.g., MTA) that minimize the potential for coronal microleakage. Thus, this is a dynamic period of development of this class of procedures.

There have been numerous recent case reports and case series on revascularization of the necrotic pulp space. Investigators and clinicians have used a variety of medicaments to disinfect the canal space. These include a triple antibiotic paste (a 1 : 1 : 1 mixture of ciprofloxacin/metronidazole/minocycline) or variation thereof,[14,58,59,92,121] $Ca(OH)_2$ alone or in combination with antibiotics,[21,22,59] and formocresol.[103]

The triple antibiotic paste—because its use has been reported in a number of case reports and case series and because it is less commonly used in dentistry than $Ca(OH)_2$ and formocresol—deserves additional review. The development of the triple antibiotic paste was led in large part by Hoshino and colleagues.[48,100] 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. Astute practitioners realized that the triple antibiotic paste could be a valuable adjunct for revascularization procedures, since 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.[134] 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 1.7×10^8. After irrigation with 1.25% NaOCl, 10% of the teeth sampled cultured bacteria free. The mean colony-forming unit (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. This study provides strong support for the effectiveness of the triple antibiotic paste in disinfection of immature teeth with apical periodontitis.

Related dog studies sought to determine how best to create a matrix, or scaffold, onto which stem cells could grow in the canal system.[120] Using a protocol similar to that described earlier, immature dog teeth that were infected and confirmed to have developed apical periodontitis were then disinfected with the triple antibiotic paste. At the revascularization step, teeth were randomized into four groups: (1) no further treatment, (2) stimulation of bleeding into the canals, (3) placement of collagen solution in the canals, (4) placement of a mixture of collagen solution and blood. A fifth group of teeth that were not infected were left for natural development. The teeth were radiographically monitored for 3 months, at which time the dogs were sacrificed for histologic examination. In the teeth where nothing was done to create bleeding into the canal, there was evidence of persistent apical radiolucency and a lack of further development in root length and root wall thickness. Histologic sections from these teeth demonstrated persistent apical inflammation and thin root walls. In teeth where a blood clot was formed in the canal system, there was radiographic evidence of resolution of the apical pathosis and an increase in root length and root wall thickness. Histologic sections demonstrated healing of the apical area, including bone and periodontal ligament, ingrowth of tissue into the root canal system, and thickening of the root walls. Findings indicated that there was a trend toward more vital tissue within the canal lumens for roots in which blood clot was formed after disinfection with triple antibiotic paste. In addition, the results suggest the importance of inclusion of a blood clot to improve chances of revascularization. The fibrin polymer of the blood clot likely served as a matrix, or scaffold, for stem cell growth, thus enhancing the likelihood of tissue regeneration. Collectively, these two preclinical studies provide evidence for the efficacy of triple antibiotic paste and suggest the importance of a scaffold in promoting tissue ingrowth.

Prior to reviewing the techniques used for revascularization, it is important to consider the relative advantages and limitations of revascularization compared to other options for treatment of the immature tooth with necrotic pulp (see also Chapter 23). There are several challenges clinicians face when presented with an incompletely formed root in need of endodontic treatment. 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.[25,26,33,45,115] However, a disadvantage of the traditional apexification procedures is that the short-term[98] or long-term[6,7] use of $Ca(OH)_2$ can reduce root strength. 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.[24] The advent of one-step apexification,[23,41] by creation of artificial barriers using materials such as MTA,[102,135] has greatly decreased the number of appointments and time to completion. One-step apexification, however, does not generally result in further root development.[13,31] A primary advantage of revascularization procedures in these cases is the greater likelihood there will be an increase in root length and root wall thickness.

Overview of Clinical Revascularization Cases

To date, only case reports and case series on endodontic revascularization treatment are 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 in actual patients and thus provide a higher level of evidence than preclinical studies. While techniques for endodontic revascularization have varied in published case reports and case series (Table 16-2 for a summary and Figs. 16-4 to 16-8), there have been some consistent features worth noting. Nearly all reported cases involve patients 8 to 18 years old and teeth with immature apices. Age may be an important issue, since some studies suggest that younger patients have a greater healing capacity or stem cell regenerative potential.[3,27,63,69,84] Moreover, the large diameter of the immature (open) apex may foster the ingrowth of tissue into the root canal space and may be indicative of a rich source of mesenchymal stem cells of the apical papilla (SCAP; see Fig. 16-4).[56,113,114] These tissues are likely lacerated during the evoked bleeding step and constitute one likely source of mesenchymal stem cells delivered into the root canal space. 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. In addition, NaOCl, alone or in combination with other irrigants, was used to disinfect the canal space. In a majority of cases, a combination of triple antibiotic or NaOCl 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. In most cases, a blood clot formed in the canal. The formation of a blood clot might serve as a protein scaffold, permitting three-dimensional ingrowth of tissue.

Nearly all of these reports noted continued thickening of the root walls and subsequent apical closure. An example of increased root length and thickening of root walls is seen in Fig. 16-6.[92] Because of the lack of histology in these clinical cases, it should be recognized that radiographic findings of continued root wall thickness does 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 ingrowth of cementum, bone, or a dentin-like material.* It should also be noted in some of the case reports[21,58,59] that although the teeth

*References 61, 80, 82, 96, 105, and 107.

TABLE 16-2

Summary of Case Reports and Case Series on Revascularization of the Necrotic Canal

Patent Age (Years)	Irrigation	Intracanal Medicament	Med. Time	Blood Clot?	Pulp Space Barrier/ Restoration	Recall	Apical Pathosis	Root Lengthened	Root Wall Thickening?	Authors
13	5% NaOCl, 3% H_2O_2	Metro/cipro	3 × 1 wk	Pulp	$Ca(OH)_2$/GI, composite	30 mo	Healed	Yes	Yes	Iwaya et al., 2001[58]
11	5.2% NaOCl, 0.12% CHX	Metro/cipro/ mino	26 d	Clot	MTA/GI	2 y	Healed	Yes	Yes	Banchs & Trope, 2004[14]
9–10	2.5% NaOCl	$Ca(OH)_2$	7–32 mo	Hard-tissue barrier	Amalgam	7 mo-5 y	Healed (4)	Yes (4)	Yes	Chueh & Huang, 2006[21] Chueh et al., 2009[20]
8	5.2% NaOCl, 0.12% CHX	Metro/cipro/ mino	21 d	Clot	MTA/ composite	8 mo	Healed	Yes	Yes	Petrino, 2007[92]
9	5.2% NaOCl, 2% CHX	Metro/cipro/ mino	3 wk	Clot	MTA/GI	12.5 mo	Healed	Yes	Yes	Thibodeau & Trope, 2007[121]
9–14	5% NaOCl	Metro/cipro/ mino E-mycin/ $Ca(OH)_2$, $Ca(OH)_2$	7 d-6 mo	Calcific barrier, pulp, clot, CollaCote	MTA/bonded restoration or amalgam	1–5 y	Healed (8) Reduced (1)	Yes (7) Questionable (2)	Yes (9)	Cotti et al., 2008[22]
9	5.2% NaOCl/3% H_2O_2	$Ca(OH)_2$	2 wk	Clot	MTA/GI/ composite	30 mo	Healed	Yes	Yes	Jung et al., 2008[59]
9–18	2.5% NaOCl/3% H_2O_2	Formocresol	N/A	Clot	GI	6 mo-3.5 y	Healed (4) Reduced (5)	Unable to determine	Unable to determine	Shah et al., 2008[103]

$Ca(OH)_2$, Calcium hydroxide; *CHX,* chlorhexidine; *cipro,* ciprofloxacin; *d,* day; *E-mycin,* erythromycin; *GI,* glass ionomer; *H_2O_2,* hydrogen peroxide; *metro,* metronidazole; *mino,* minocycline; *mo,* month; *MTA,* mineral trioxide aggregate; *NaOCl,* sodium hypochlorite; *wk,* week; *y,* year.

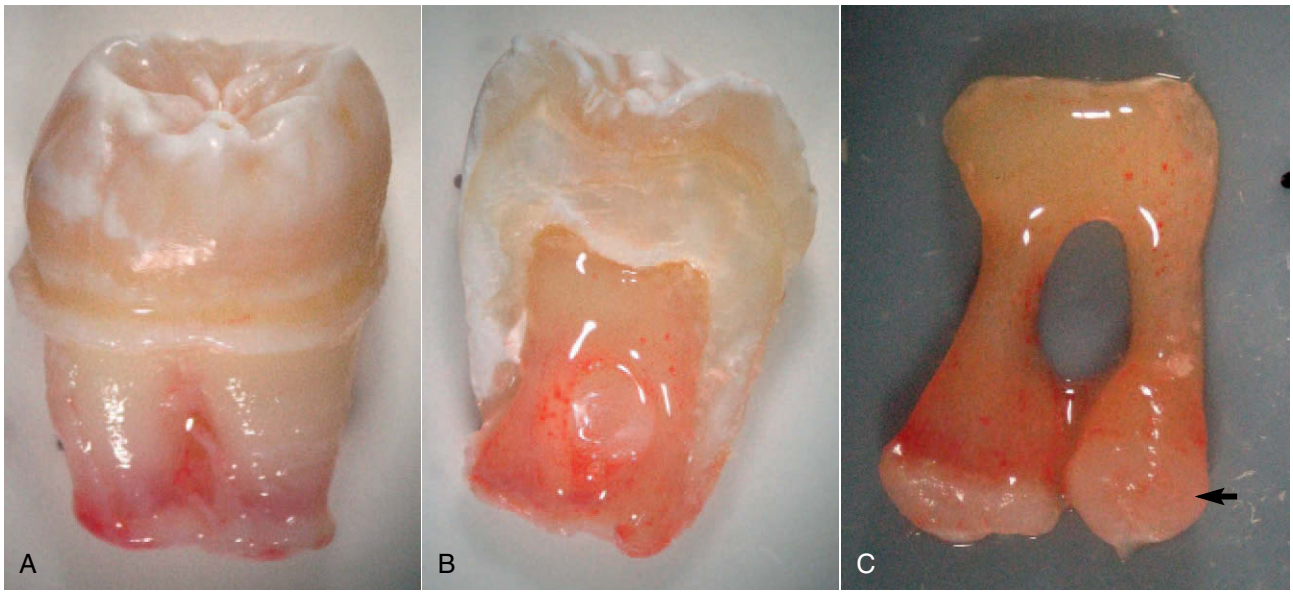


FIG. 16-4 A-C, Dissection of an immature permanent tooth indicating the extent of the apical papilla. Note that this structure is likely lacerated during the evoked bleeding step of revascularization cases and thus cells from this structure, including mesenchymal stem cells of the apical papilla (SCAP), 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.)*

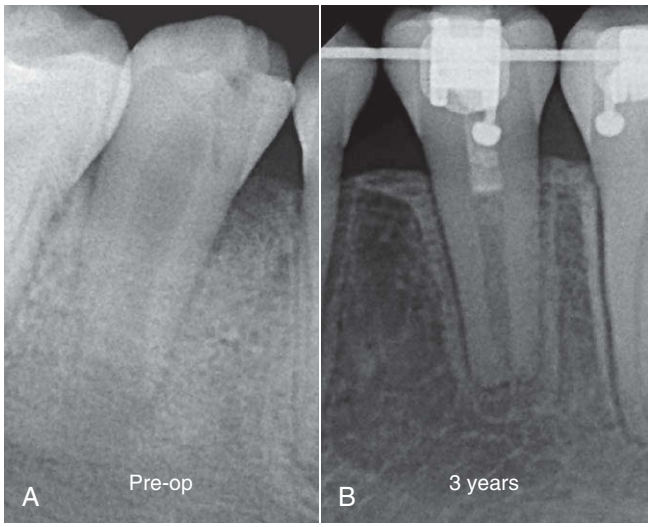


FIG. 16-5 Revascularization case of a 14-year-old female patient with a diagnosis of pulpal necrosis secondary to dens invaginatus. The tooth was isolated, accessed, and irrigated with 2.5% sodium hypochlorite and 2% chlorhexidine, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 96 days. Upon recall, symptoms had subsided, the tooth was isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established, and the root canal system was sealed with white mineral trioxide aggregate and a composite restoration. *(Courtesy Dr. Sandra Madison.)*

were nonresponsive to pulp testing, vital tissue was identified in the apical portion of the canal space. In these cases, necrotic tissue was removed until bleeding was observed, then the canals were disinfected with antibiotic paste[58,59] or $Ca(OH)_2$.[21] 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.[18] 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 percent 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 length (Fig. 16-9) or root width (Fig. 16-10), with the anticipated finding that instrumentation with files of greater taper resulted in a slight but detectable loss of apical root width. The results indicated that revascularization treatment with either the triple antibiotic paste or $Ca(OH)_2$ produced significantly greater increases in root length compared with either the MTA or NSRCT control groups. The formocresol group differed only in comparison to the MTA apexification control group. In terms of changes in root width (see Fig. 16-10), treatment with the triple antibiotic paste produced significantly greater increases in dentin wall thickness compared with the MTA and NSRCT control groups. Treatment

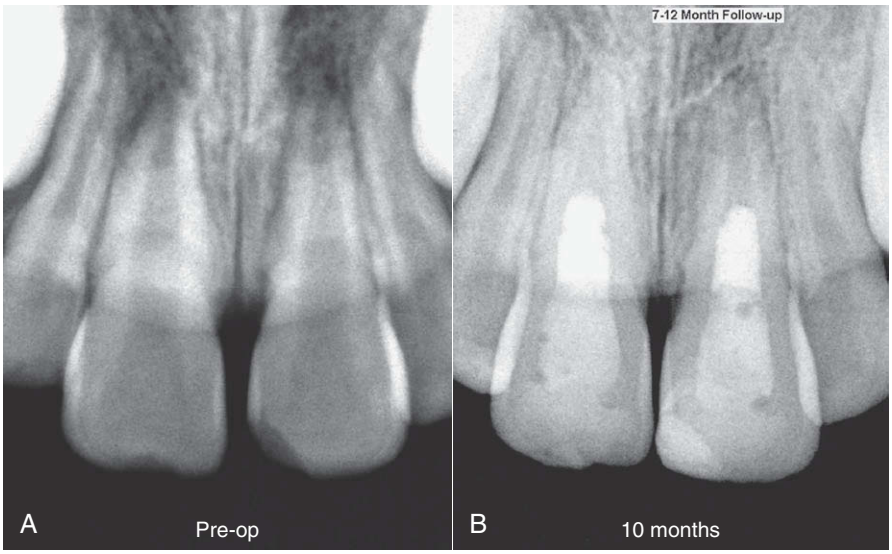


FIG. 16-6 Revascularization case for treating an 8-year-old female patient with a diagnosis of pulpal necrosis secondary to dental trauma (lateral luxation with +2 class fracture of teeth #8 and #9). The teeth were isolated, accessed, and irrigated with 5% sodium hypochlorite and 2% chlorhexidine, followed by placement of a mixture of ciprofloxacin, metronidazole, and minocycline for 21 days. Upon recall, symptoms had subsided, the teeth were isolated, and the triple antibiotic paste was removed by irrigation. Bleeding was established in both teeth, and the root canal systems were sealed with white mineral trioxide aggregate and a composite restoration. *(Courtesy Dr. Alan Law.)*

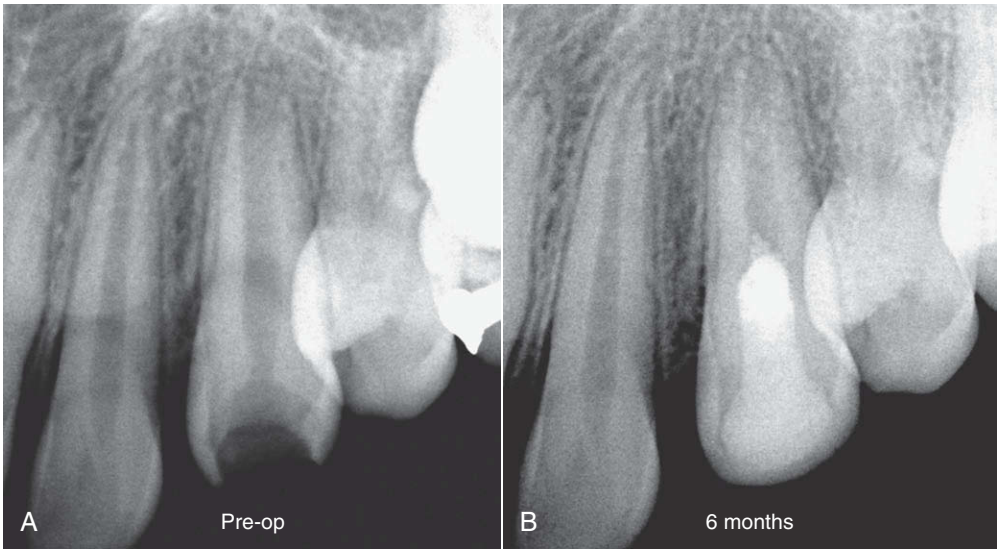


FIG. 16-7 Revascularization case illustrating treatment delivered to a 13-year-old female patient with a diagnosis of pulpal necrosis secondary to caries, with an unspecified prior history of trauma. The tooth was isolated, accessed, and irrigated with 5% sodium hypochlorite and 0.12% chlorhexidine, 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. *(Courtesy Dr. Alan Law.)*

with $Ca(OH)_2$ or formocresol resulted in significantly greater change in dentinal 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 dentinal 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 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. Another secondary analysis indicated that the location of $Ca(OH)_2$ placement appeared to be a strong predictor of radiographic outcome. When $Ca(OH)_2$ placement was restricted to the coronal half of the root canal, the increase in root wall thick-

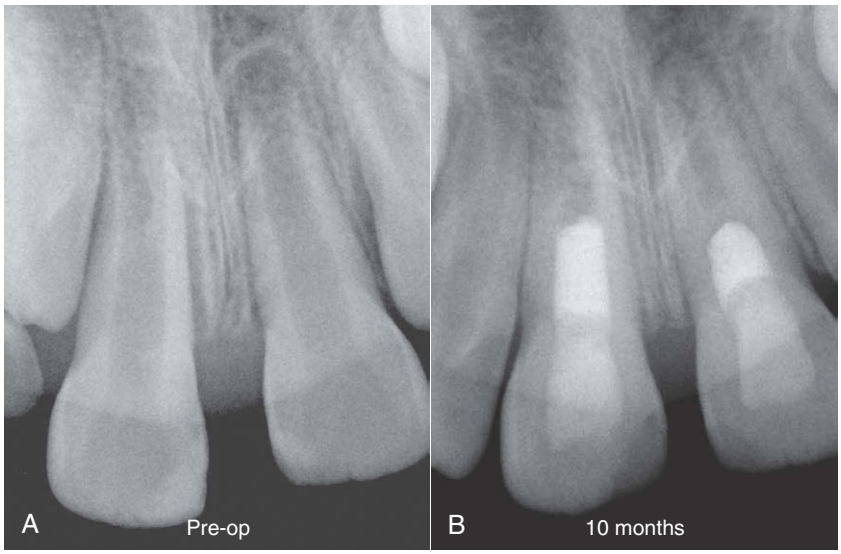


FIG. 16-8 Revascularization case illustrating treatment delivered to a 9-year-old male patient with a diagnosis of pulpal necrosis secondary to trauma, with a class 3 fracture in tooth #8 and a class 2 fracture in tooth #9. The patient reported moderate to severe pain in both teeth. The teeth were isolated, 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. *(Courtesy Dr. Alan Law.)*

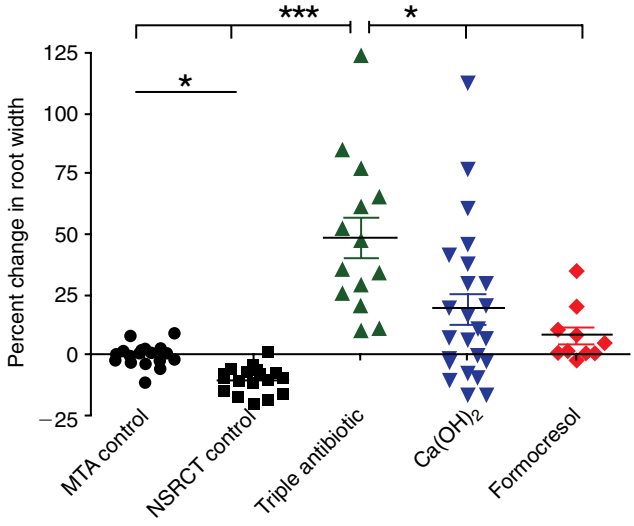


FIG. 16-9 Retrospective analysis of the percentage of change in root length from preoperative image to postoperative image, measured from the cementoenamel junction (CEJ) to the root apex in 40 control patients and in 48 patients following a revascularization procedure. ***$P < 0.001$ versus mineral 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.)*

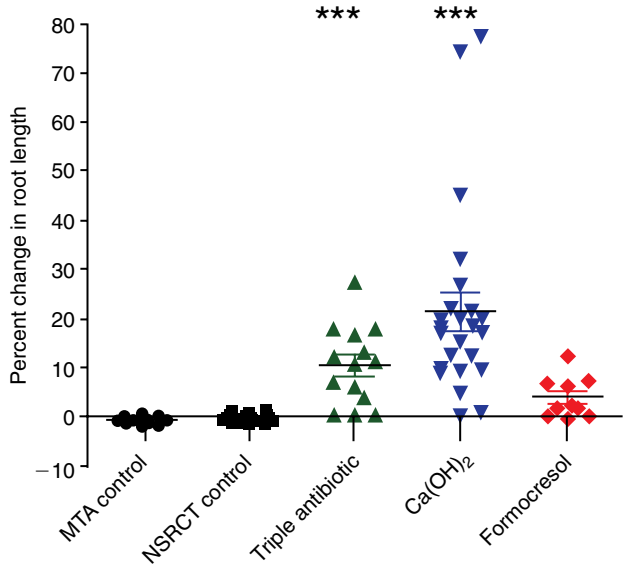


FIG. 16-10 Retrospective analysis of the percentage of change in dentinal wall thickness from preoperative image to postoperative image, measured at the apical third of the root (position of apical third defined in the preoperative image) in 40 control patients and in 48 patients following a revascularization 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.)*

ness was 55%, compared to a 3% increase when it was placed in the apical half of the root canal system. This might be due to residual $Ca(OH)_2$ having a cytotoxic interaction with stem cells. 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 considerations 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 is to develop 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 issues should include the number of appointments (at least two), the possible adverse effects (primarily potential minocycline staining of the crown), the potential lack of response to treatment and alternative treatments, and possible posttreatment symptoms. Clinical staining of the crown and any root structure above the gingival margin appears to be due to the presence of minocycline. This can be minimized by using a delivery system that restricts the drug below the cementoenamel junction (CEJ). When it does occur, it can often be reduced or eliminated by a walking bleach method with sodium perborate. Likely alternative treatments would include MTA apexification, no treatment, or extraction.

At the first appointment (Fig. 16-11, *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 periradicular 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 is copiously and slowly irrigated with 20 ml of NaOCl followed by 20 ml of 0.12% to 2% chlorhexidine (CHX). Since 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 $Ca(OH)_2$. Both medicaments have been shown to be effective (see Figs. 16-9 and 16-10). The triple antibiotic paste has the advantage of being a very effective antibiotic combination against odontogenic microorganisms[48,100]; its efficacy is supported by several case studies.[18] 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. Alternatively, $Ca(OH)_2$ has the advantage of being widely available and is a commonly used medicament, but it may be cytotoxic to stem cells.[14,18,20] 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. 16-11, *F-J*), the patient is evaluated for resolution of any signs or symptoms of an acute infection (e.g., swelling, sinus tract pain, etc.) that may have been present at the first appointment. The antimicrobial treatment is repeated if resolution has not occurred.[20,59] In most cases, the acute signs and symptoms have resolved. Since revascularization-induced bleeding will be evoked at this appointment, the tooth should not be anesthetized 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.[93] Following isolation and reestablishment of coronal access, the tooth should be copiously and slowly irrigated with 20 ml NaOCl, possibly together with gentle agitation with a small hand file to remove the antimicrobial medicament. After drying the canal system with sterile paper points, a file is placed a few mm 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 may be inserted into the root canal system to serve as a resorbable matrix to restrict the positioning of the MTA. About 3 mm of MTA is then placed, followed by a restoration. A 12- to 18-month recall should be considered as the earliest time point to conduct the clinical examination and evaluate continued radiographic improvement in root development.[18]

Clinical Measures of Treatment Outcome

The goal of nonsurgical root canal treatment is to maintain or restore the health of periradicular tissues. The goals of revascularization procedures extend beyond those of nonsurgical root canal treatment in that there is an additional objective of regaining vital, functioning tissue capable of supporting continued root development in cases of the immature permanent tooth with a necrotic root canal system. The measures of success for revascularization are not only radiographic evidence of periradicular health but also radiographic and other clinical evidence of functioning vital tissue in the canal space. Radiographic evidence of functioning pulp (or pulplike) tissue would include continued root growth, both in length and wall thickness. These findings have been shown in many published cases. Other measures of the presence of vital, functioning tissue in the canal space include laser Doppler blood flowmetry[117]; pulp testing involving heat, cold, and electricity[91]; and lack of signs or symptoms. The ideal clinical outcome is an asymptomatic tooth that does not require retreatment, but to validate that regenerative endodontic techniques are truly effective, nonsubjective vitality-assessment methods are essential.

It is worth noting that the published revascularization cases have shown increased root wall thickness that is limited to the

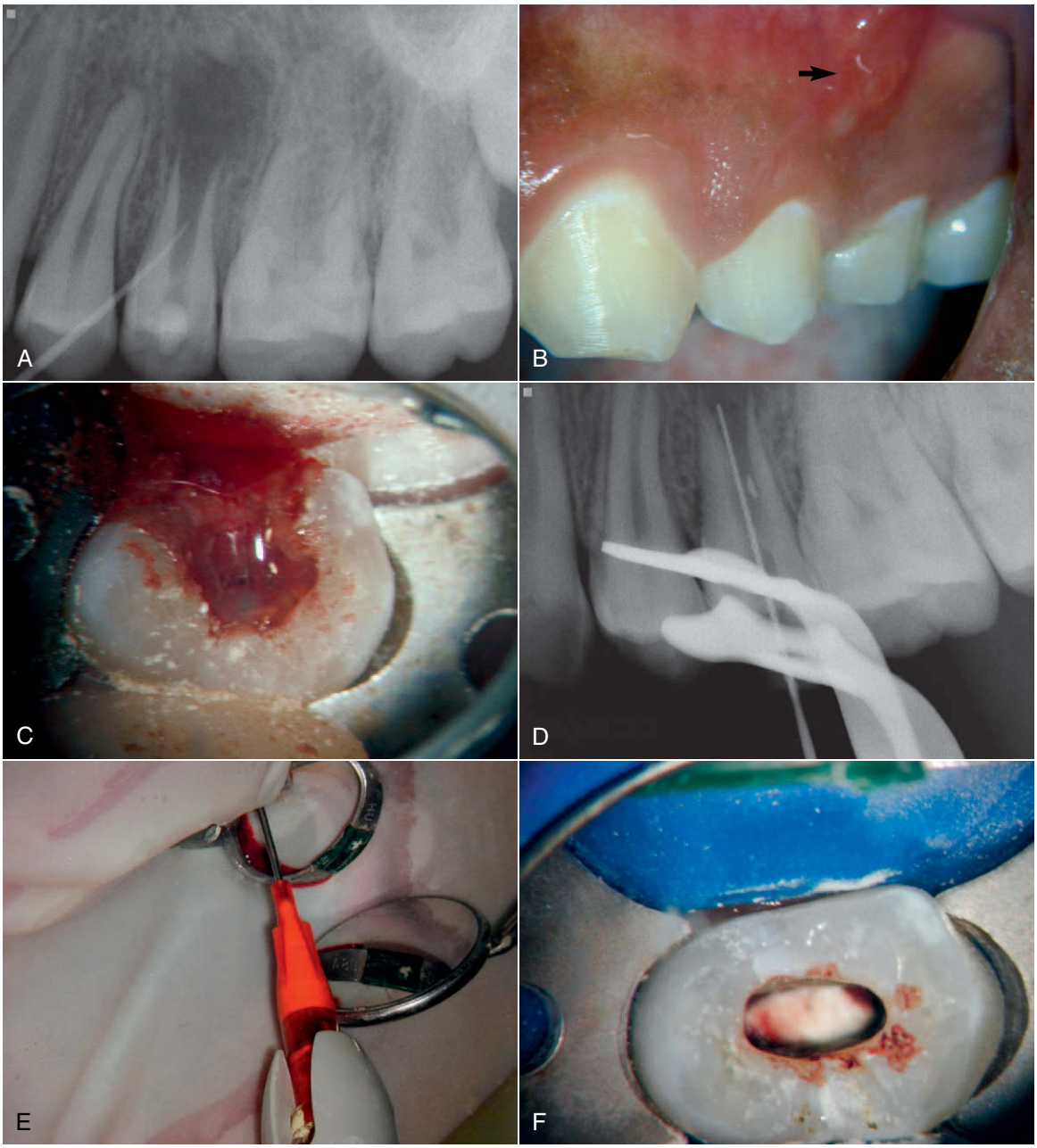


FIG. 16-11 Example of a revascularization protocol. First appointment: Clinical examination revealed a sinus tract **(A),** soft-tissue swelling that required an incision for drainage **(B),** and purulent/hemorrhagic exudates upon access **(C-D).** A working length file was placed into the root canal system, and the tooth was slowly irrigated with 20 ml of 5.25% sodium hypochlorite (NaOCl) and 20 ml of 0.12% chlorhexidine using a Max-I-Probe needle inserted to the apical third. **E,** The canals were dried and medicated by a triple antibiotic paste (a 1:1:1 mixture of ciprofloxacin/metronidazole/minocycline) using a Centrix syringe that facilitates delivery below the cementoenamel junction (CEJ). The patient returned 1 month later. The tooth was isolated, accessed, and the medicament removed by slow irrigation with NaOCl. CollaPlug was placed below the CEJ **(F)** to serve as a matrix to position the mineral trioxide aggregate coronal to the blood clot **(G).**

Continued

midroot and apical root. 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.[24] Future clinical studies should focus on extending revascularization into the cervical area, potentially strengthening this area and decreasing the risk of root fracture.

SUMMARY

The field of regenerative endodontics is rapidly advancing. It is based upon 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.

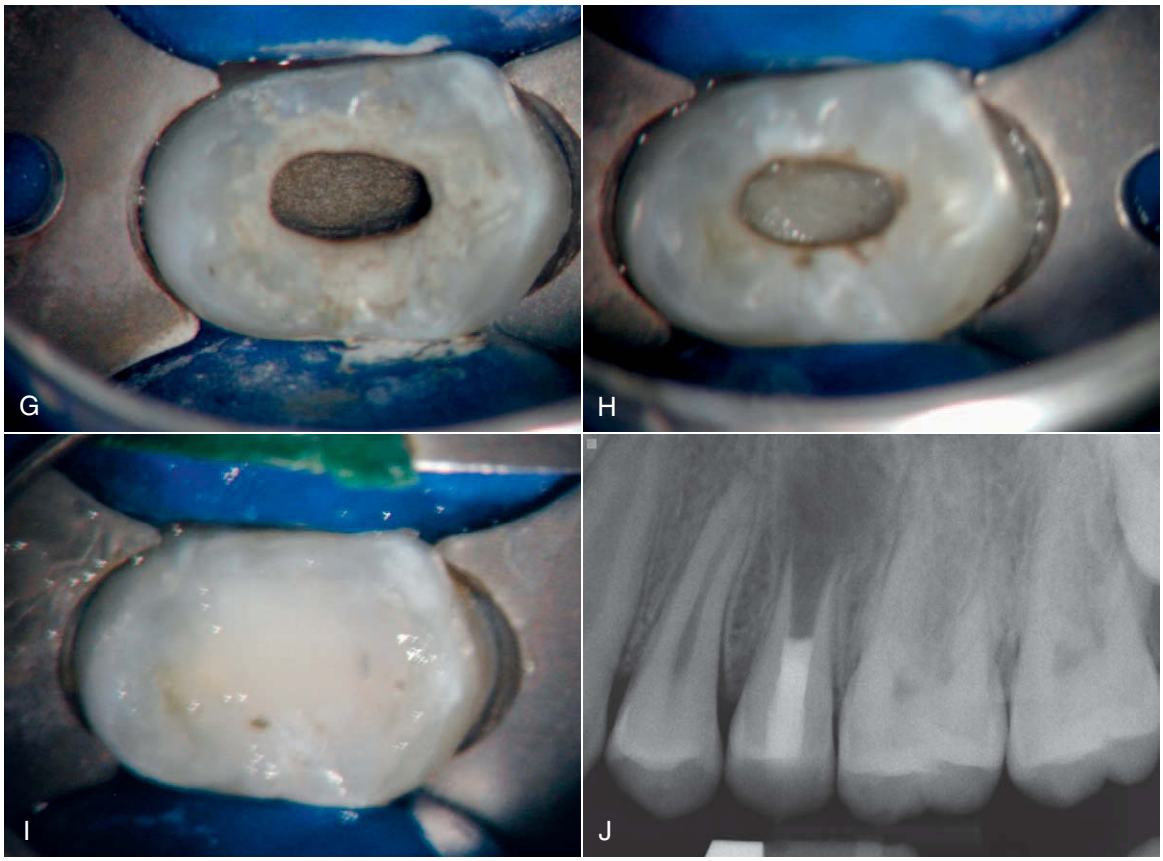


FIG. 16-11, cont'd The tooth was then sealed with a layer of Fuji II **(H)** and a composite **(I-J).** *(Courtesy Dr. Anibal Diogenes.)*

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 an approach using human stem cells implanted into a human root in an immunocompromised mouse provides a useful model for evaluating combinations of stem cells/scaffolds/growth factors for regenerating the pulp-dentin complex. 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.

To date, none of the published clinical studies fully engage the concepts of tissue engineering. Instead, these studies are best described as revascularization procedures that attempt to regenerate biologic tissues within the root canal space, without necessarily replicating the pulp-dentin complex. Discussion about the requirements of an appropriate scaffold and growth factor was begun in the 1960s by Nygaard-Østby,[82] who had no access to our contemporary instruments, materials, and the entire 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, a revascularization procedure provides a very real treatment of high value in cases with an otherwise poor prognosis. Histologic analyses of revascularization procedures conducted in patients[82,83] or animals[78,96,129] suggests that the increase in root dimensions is often due to deposition of cementum-like material, dentin, or bone.

Future clinical research will likely focus on 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 revascularization procedures 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 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 17

The Role of Endodontics After Dental Traumatic Injuries

ASGEIR SIGURDSSON, MARTIN TROPE, and NOAH CHIVIAN

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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 will concentrate 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.[23,132] It occurs primarily in the anterior region of the mouth, affecting the maxillary more than the mandibular jaw.[28] Serious accidents like automobile crashes can affect any tooth and 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.[88,98] 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 there is a sufficient and good margin for the restoration to be possible. If the tooth can be maintained from a periodontal point of view, the pulp is treated as with 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 electric or thermal stimulus. This makes the traumatized tooth vulnerable to false-negative readings from such tests.[112]

Teeth that give a response at the initial examination cannot be assumed to be healthy and 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.[66]

The transition from no response to a 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.[30]

Thermal and electric 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 following 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[64,65] (Fig. 17-1). 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; how cold it is will give much more reliable responses than water ice. The electric pulp test relies on electric impulses directly stimulating the nerves of the pulp. These tests have limited value in young teeth but are useful in cases where 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 electric pulp test becomes important.

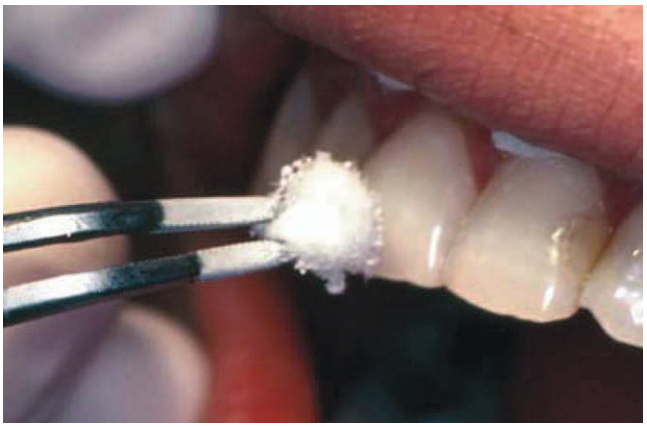


FIG. 17-1 Difluorodichloromethane (−40° F [−40° C]) gas is sprayed on a cotton pellet and then placed on the incisal edge of the maxillary incisor.

Laser Doppler flowmetry (LDF) was introduced in the early 1970s for measurement of blood flow in the retina.[81] The technique has also been used to assess blood flow in other tissue systems such as skin and renal cortex. It utilizes a beam of 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. 17-2).

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



FIG. 17-2 Laser Doppler machine. *(Courtesy Moor Instruments, Devon, United Kingdom.)*

pulp.[103,164,165] 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 which would remain necrotic.[165]

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

Radiographs are essential tools in the thorough examination of traumatized hard tissues. They may reveal root fractures, subgingival crown fractures, tooth displacements, bone fractures, resorptions of the root, and adjacent bone or foreign objects. One radiograph will be insufficient in most dental trauma cases. In their new guidelines for the management of traumatic dental injuries, the International Association of Dental Traumatology (IADT) has recommended taking at least four different radiographs for almost every injury.[58-60] Those four include a direct 90-degree on the axis of the tooth, two with different vertical angulations, and one occlusal film (Fig. 17-3). Multiple radiographs will increase the likelihood of diagnosing root fractures, apical translocations, and other possible injuries.

Early detection of root resorption is extremely difficult on dental radiographs, however. The radiodensity of the root requires that a significant amount of root substance is 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, but 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. One should also carefully evaluate the adjacent bone for resorption[153] (Fig. 17-4), discussed in more detail later.

New radiographic techniques like tuned-aperture computed tomography (TACT) and cross-sectional CT scans have shown promise in improving the clinician's ability to diagnose root resorption (see also Chapter 29).[105]



FIG. 17-3 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.

In instances of soft-tissue laceration, it is advisable to radiograph the injured area prior to 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. 17-5).

In reviewing the films of traumatized teeth, special attention should be directed to the dimension of the root canal space, the degree of apical closure of the root, the proximity of fractures to the pulp, and the relationship of root fractures to the alveolar crest. Whereas conventional periapical films are generally useful, an occlusal or Panorex film can supplement them when the clinician is looking for bone fractures, dislocation of teeth, or the presence of foreign objects.

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 or crack of 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

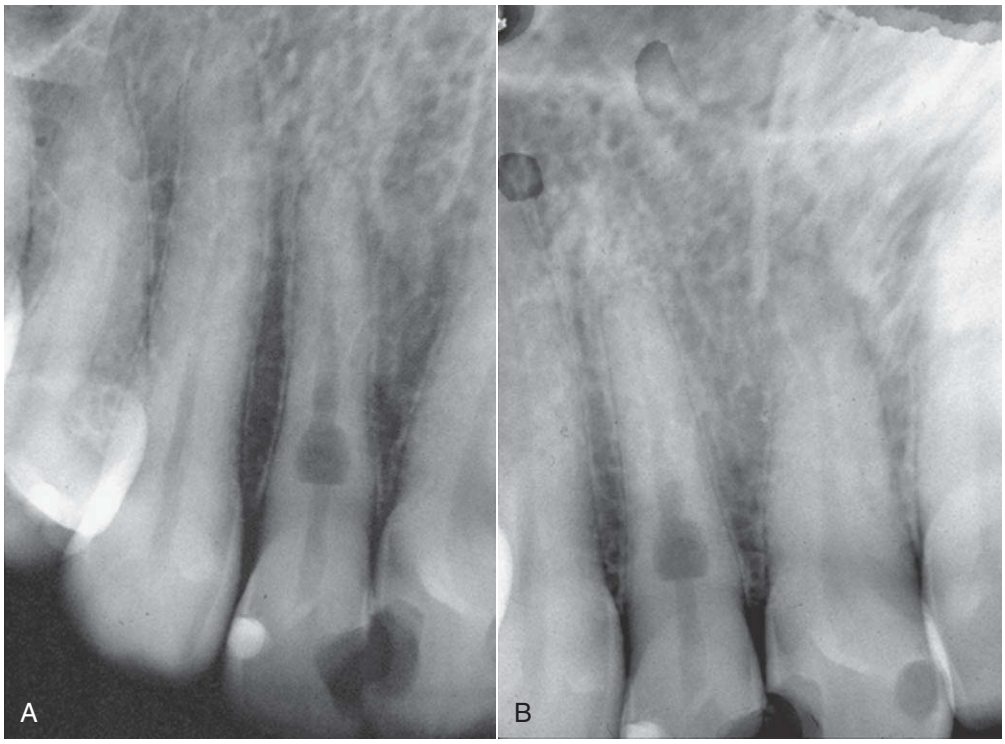


FIG. 17-4 Angled radiographs to show internal resorption. Radiographs from two different horizontal projections depict **(A)** the lesion within the confines of the root canal on both views and **(B)** the adjacent bone intact on both views.

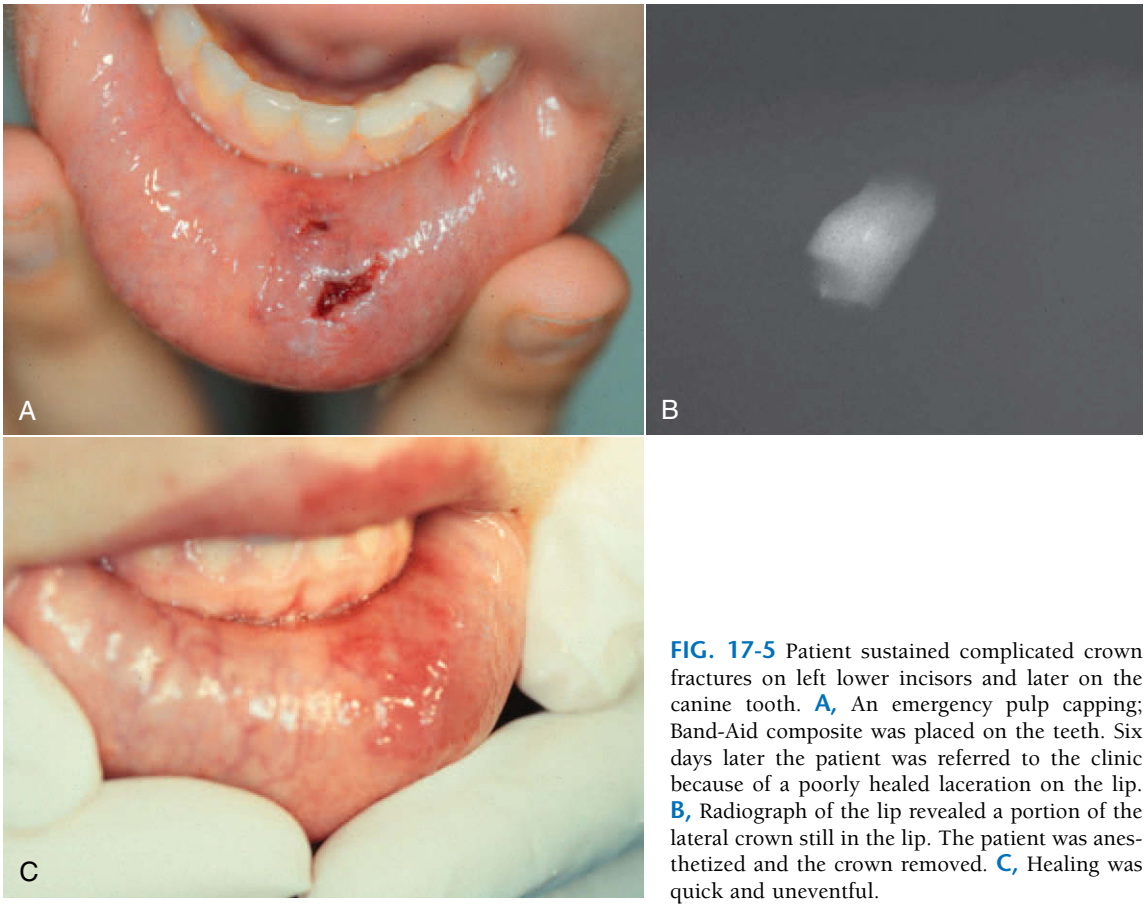


FIG. 17-5 Patient sustained complicated crown fractures on left lower incisors and later on the canine tooth. **A,** An emergency pulp capping; Band-Aid composite was placed on the teeth. Six days later the patient was referred to the clinic because of a poorly healed laceration on the lip. **B,** Radiograph 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.

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 enamel and dentin without pulp exposure.[16]

Incidence

This injury is likely to be the most commonly reported dental injury. It is estimated to account for at least a third to 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 esthetic 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 is to prevent any ingress of bacteria into the tubules as well as to reduce 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.[3,41,54,105,140] If, however, 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, the reactive inflammation of the underling pulp, which is a significantly different reaction compared to the negative reaction to composite bonding systems.[3,41,54,105,140]

Complicated Crown Fracture

A *complicated crown fracture* involves enamel, dentin, and pulp[1] (Fig. 17-6).

Incidence

Complicated crown fractures occur in 0.9% to 13% of all dental injuries.[38,119,142]

Biologic Consequences

A crown fracture involving the pulp, if left untreated, will always result in pulp necrosis.[90] However, the manner and time sequence in which the pulp becomes necrotic allows a great deal of potential for successful intervention to maintain pulp vitality. The first reaction after the injury is hemorrhage and local inflammation (Fig. 17-7).

Subsequent inflammatory changes are usually proliferative but can be destructive over time. A proliferative reaction is favored in traumatic injuries, since 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[43,47,75] (see Fig. 17-7). In time, the bacterial challenge will result in local pulpal necrosis and a slow apical progression of the pulpal inflammation (Fig. 17-8).

Treatment

Treatment options for complicated crown fracture are (1) vital pulp therapy, comprising pulp capping, partial pulpotomy, or full pulpotomy; or (2) pulpectomy. Choice of treatment depends on the stage of development of the tooth, the time between trauma and treatment, concomitant periodontal injury, and 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.[91] 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, since a pulpectomy in a mature tooth has an extremely high success rate.[131] However, it has been



FIG. 17-6 Complicated crown fracture involving enamel, dentin, and pulp.

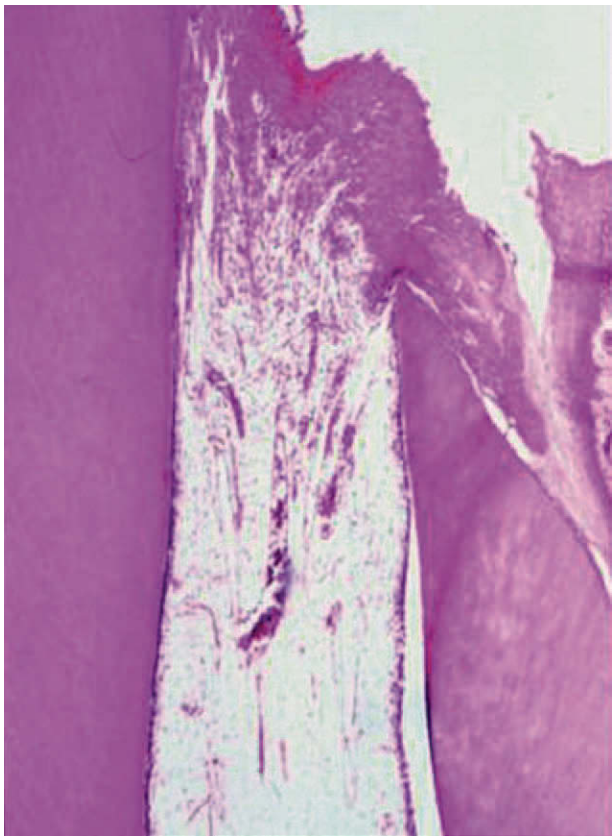


FIG. 17-7 Histologic appearance of the pulp within 24 hours of a traumatic exposure. The pulp proliferated over the exposed dentinal tubules. There is approximately 1.5 mm of inflamed pulp below the surface of the fracture.

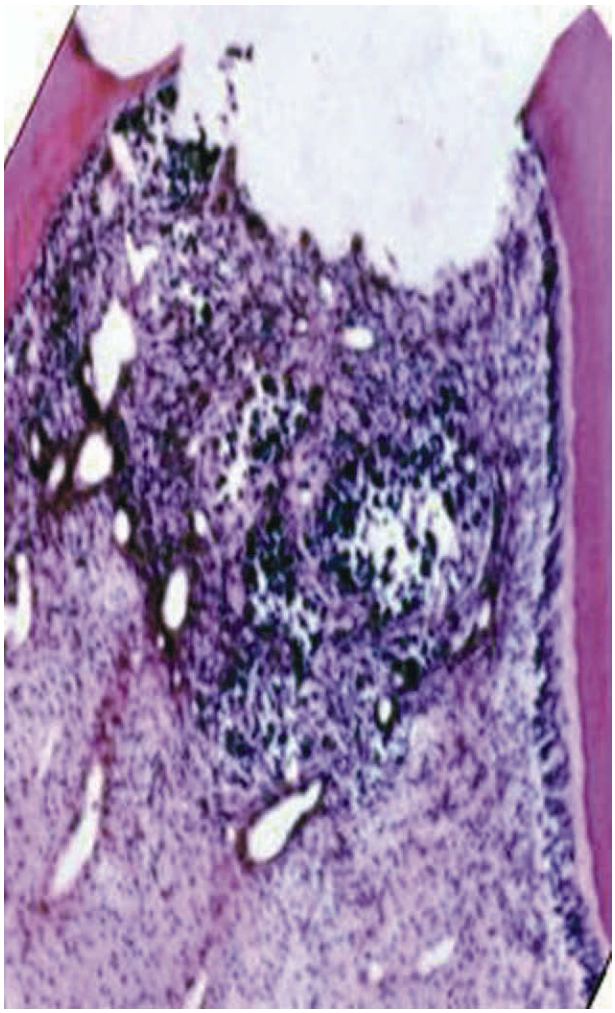


FIG. 17-8 Histologic appearance of the pulp days after a traumatic exposure. Superficial necrosis above a zone of inflamed pulp is seen.

shown that under optimal conditions, vital pulp therapy (rather than removal) on a mature tooth can be carried out successfully.[101,161] Therefore, this form of therapy can be an option under certain circumstances, even though a pulpectomy is the treatment which 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. 17-8). After 48 hours, chances of direct bacterial contamination of the pulp increase, with the zone of inflammation progressing apically[47]; as time passes, the likelihood of successfully maintaining a healthy pulp decreases.

Concomitant Attachment Damage

A periodontal injury will compromise the nutritional supply of the pulp. This fact is particularly important in mature teeth, where the chance of pulp survival is not as good as for immature teeth.[14,53]

Restorative Treatment Plan

Unlike an immature tooth where the benefits of maintaining vitality of the pulp are so great, in a mature tooth, pulpectomy is a viable treatment option. 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 adhered to:

- *Treatment of a noninflamed pulp*. Treatment of a healthy pulp has been shown to be an essential requirement for successful therapy.[141,151] Vital pulp therapy of the inflamed pulp yields an inferior success rate,[141,151] 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.[141] Challenge by bacteria during the healing phase will cause failure (see also Chapter 15).[40] 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.[41,73]
- *Pulp dressing*. Calcium hydroxide is still the most common dressing used for vital pulp therapy. Its main advantage is that it is antibacterial[37,130] and will disinfect the superficial pulp. Pure calcium hydroxide will cause necrosis of about 1.5 mm of pulp tissue, which removes superficial layers of inflamed pulp if present[102] (Fig. 17-9). The high pH (12.5) of calcium hydroxide causes a liquefaction necrosis in the most superficial layers.[123] 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 will initiate an inflammatory response, and in the absence of bacteria,[123] the pulp will heal with a hard-tissue barrier (Fig. 17-10).[122,123] Hard-setting calcium hydroxide will not cause necrosis to the superficial layers of pulp but has also been shown to initiate healing with a hard-tissue barrier.[139,148]

A major disadvantage of calcium hydroxide is that it does not seal the fractured surface. Therefore, an additional material must be used to ensure that the pulp is not challenged by bacteria, particularly during the critical healing phase.

Many materials such as zinc oxide eugenol[25,151] tricalcium phosphate,[77] 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.[54,94,128,140] 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.[94] Application of a calcium hydroxide–based material was characterized by inflammatory cell infiltration, limited tissue necrosis, and partial to complete hard-tissue bridging.[94] In a similar study on human teeth in vivo, it was found that Scotchbond Multi-Purpose Plus caused inflammatory changes when applied directly to exposed pulp tissue, but direct capping with Dycal followed by sealing with Scotchbond Multi-Purpose Plus showed favorable results in pulp tissue.[140]

Mineral trioxide aggregate (MTA) has been shown to be a good pulp capping agent.[55,106,109,163] It has a high pH similar to calcium hydroxide when unset[145] and after setting will create an excellent bacteria-tight seal.[146] It is also hard enough to act as a base for a final restoration.[145] Yet MTA does not enjoy the same popularity as calcium hydroxide as a pulp capping agent in 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 capping agent in anterior teeth.[35] 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.[110] Comparatively few studies have been done comparing the white MTA to the grey one, but most have indicated that the pulp seems to react as well to one as the other.[84,106,120] Further studies are warranted to compare the two formulations

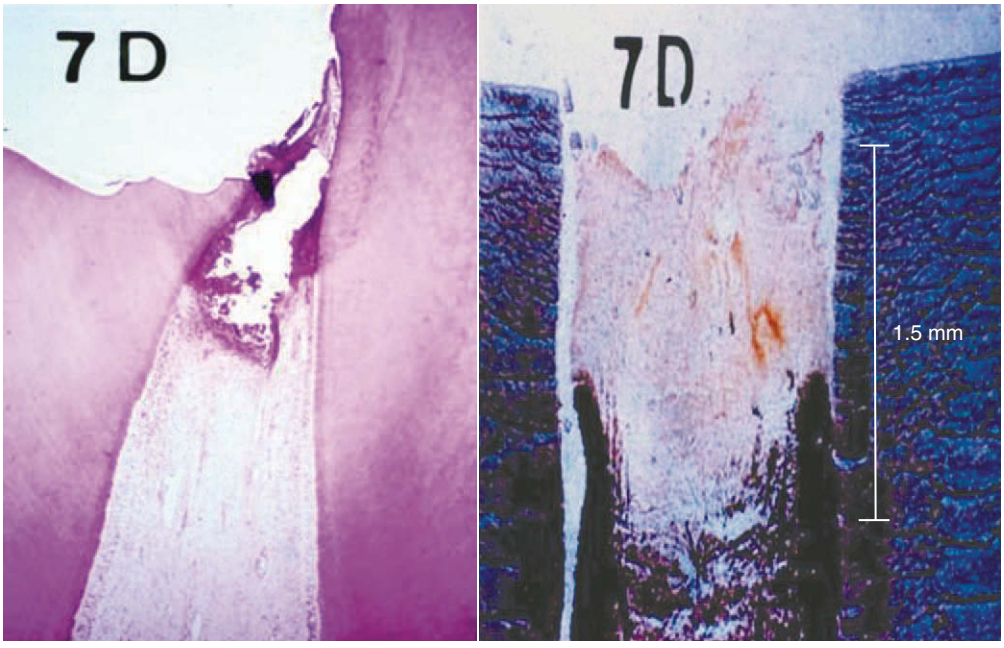


FIG. 17-9 Pulp necrosis of 1.5 mm as a result of the high pH of calcium hydroxide.

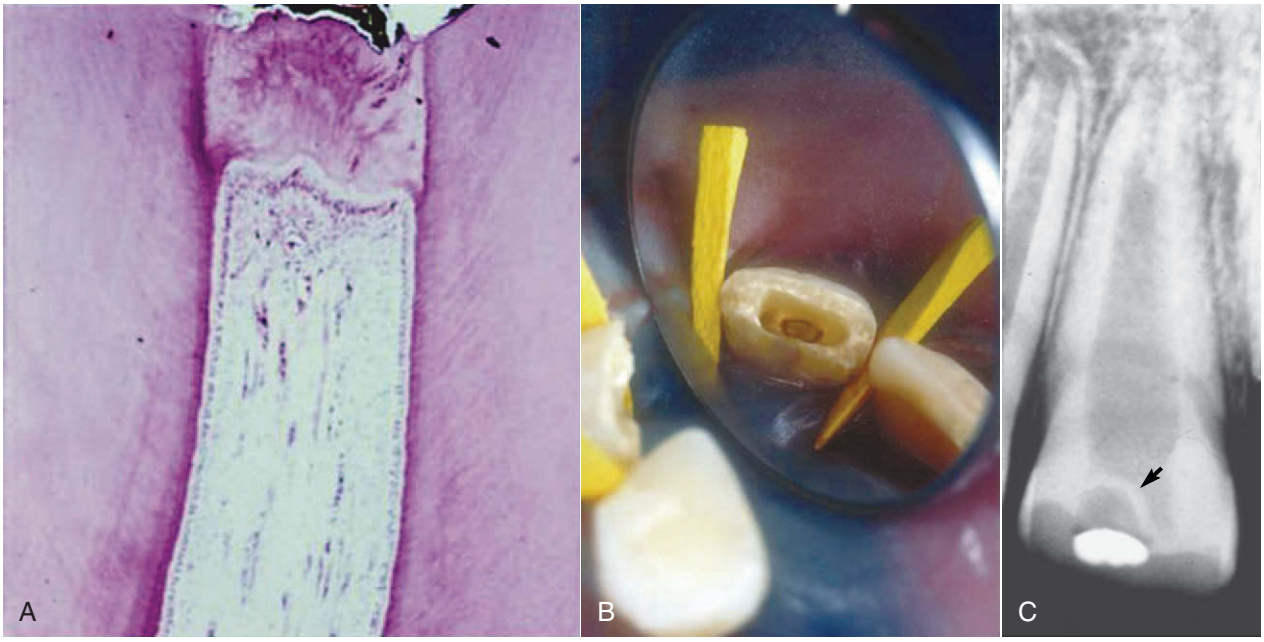


FIG. 17-10 Hard-tissue barrier after calcium hydroxide partial pulpotomy. **A,** Histologic appearance of replacement odontoblasts and a hard-tissue barrier. **B,** Clinical appearance of the barrier on removal of the coronal restoration 3 months after placement of the calcium hydroxide. **C,** Radiographic appearance of the hard-tissue barrier.

of MTA to fully elucidate the concerns raised regarding possible differences.[4] The third—and probably most common—reason why MTA is not as widely used as it could be is its relatively high cost.

Treatment Methods

Pulp Capping

Pulp capping implies placing the dressing directly onto the pulp exposure without any removal of the soft tissue.[24]

INDICATIONS

None; the success rate of this procedure (80%)[62,114] compared to partial pulpotomy (95%)[43] indicates that a superficial pulp cap should not be considered after traumatic pulp exposures. The lower success rate is not difficult to understand, since 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, since there is no depth to the cavity to aid in creating the seal, as there is with a partial pulpotomy.

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 with pulp capping.

TECHNIQUE

Anesthesia, 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 with copious water coolant[70] (Fig. 17-11). 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 dried with a sterile cotton pellet. Use of 5% sodium hypochlorite (NaOCl; bleach) has been recommended to rinse the pulpal wound.[41] The bleach will cause a chemical amputation of the blood coagulum, remove 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; this will compromise the prognosis.[43,122] A thin layer of pure calcium hydroxide is mixed with sterile saline or anesthetic solution to a thick mix and carefully placed onto the pulp stump. If the pulp size does not permit additional loss of pulp tissue, a commercial hard-setting calcium hydroxide can be used.[139] 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 is obtained, the pulp can be capped with MTA, a wet cotton pellet placed on top if it, and the access temporized for the appropriate time. The cotton pellet then needs to be removed as early as possible and the tooth then restored with composite restoration.

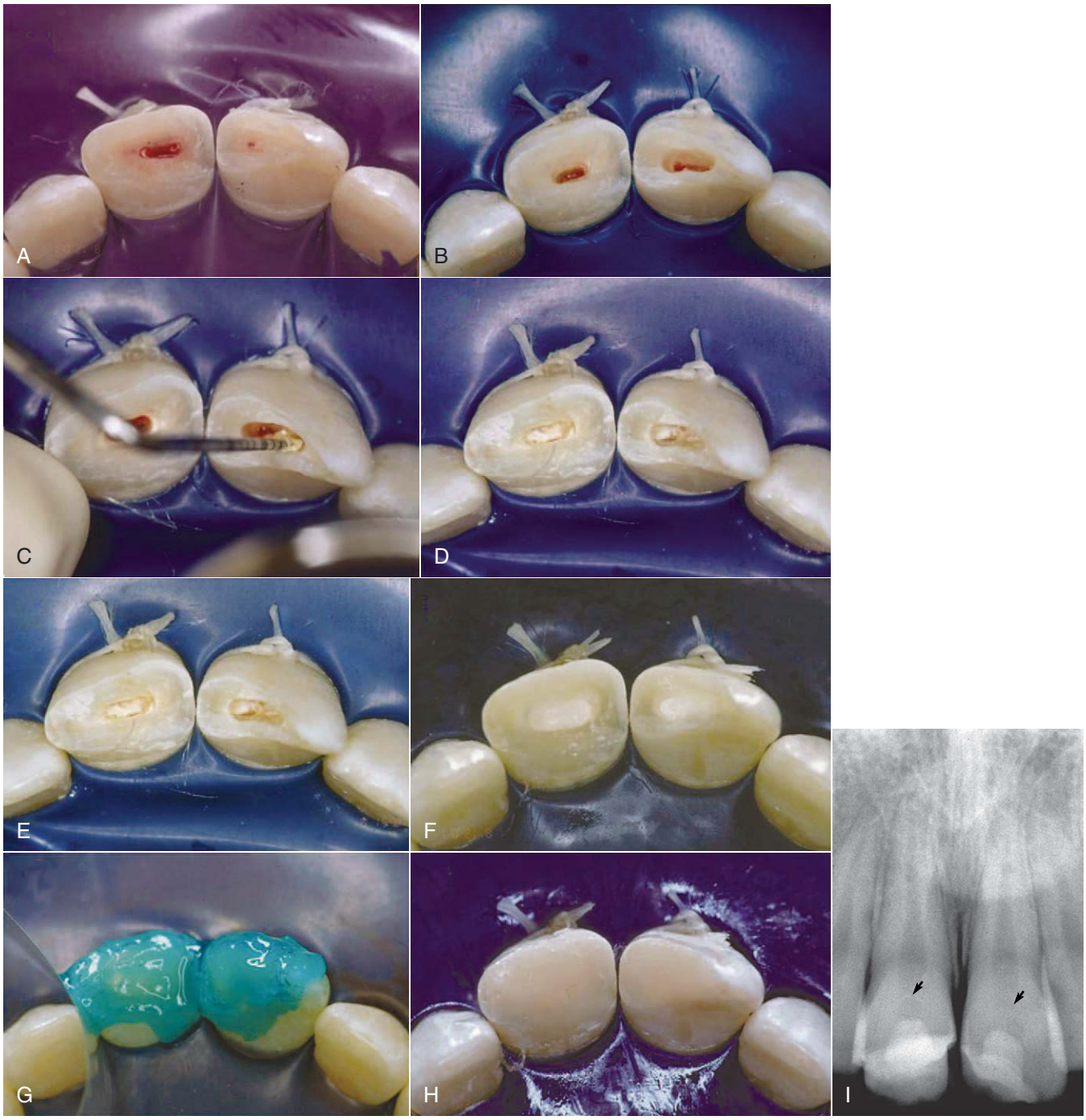


FIG. 17-11 Cvek partial pulpotomy. **A,** The fractured teeth are cleaned and disinfected; a rubber dam is placed. **B,** Cavities are prepared at high speed with a round diamond bur 1 to 2 mm into the pulpal tissue. **C,** Calcium hydroxide on a plugger **(D)** is placed on the soft tissue of the pulp. **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. **G,** Exposed dentin is etched and **(H)** then covered with composite resin. **I,** Radiograph exposed 6 months later shows formation of hard-tissue barriers in both teeth. *(Courtesy Dr. Alessandra Ritter.)*

FOLLOW-UP

As with pulp capping. Emphasis is placed on evidence of maintenance of responses to sensitivity tests and radiographic evidence of continued root development (Fig. 17-12).

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 impor-

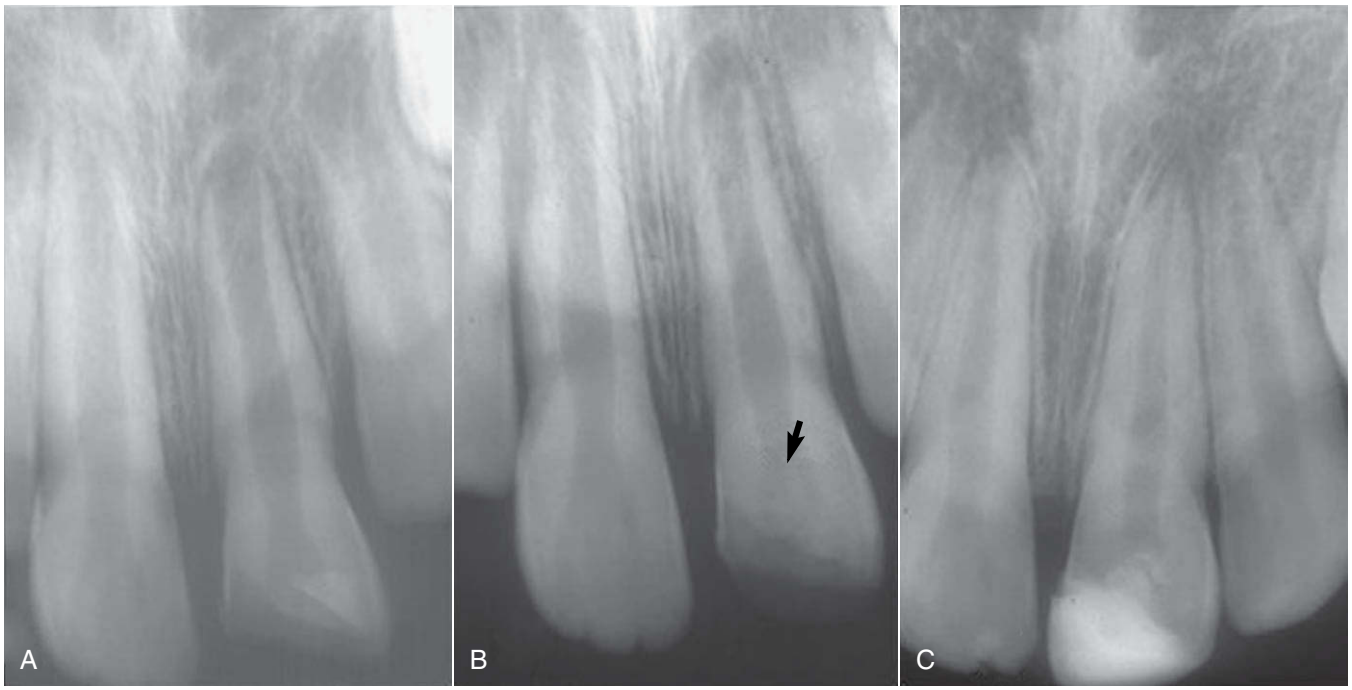


FIG. 17-12 Continued root development after partial pulpotomy. **A,** Radiograph of immature tooth with a complicated crown fracture. **B,** At the time of placement of calcium hydroxide after partial pulpotomy. **C,** Follow-up radiograph confirming that the pulp maintained vitality and the root continued to develop.

tantly, space is provided for a material that can provide 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. Prognosis is extremely good (94% to 96%).[43,63]

Full Pulpotomy

This procedure 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. Since 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 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 or a carious exposure of a young tooth with partially developed apex are two examples of when 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. Benefits only outweigh risks for this treatment form in the immature tooth with incompletely formed apices and thin dentinal walls.

TECHNIQUE

The procedure begins with anesthesia, rubber dam placement, and superficial disinfection as with pulp capping and partial pulpotomy. The coronal pulp is removed as in partial pulpotomy but to the level of the root orifices. Calcium hydroxide dressing, a bacteria-tight seal, and a coronal restoration are carried out as with partial pulpotomy.

FOLLOW-UP

As with pulp capping and partial pulpotomy. A major disadvantage of this treatment method is that 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 ensure the continuation of root formation.

PROGNOSIS

Since the cervical pulpotomy is performed on pulps, which 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 partial pulpotomy.[67] Because of the inability to evaluate pulp status after full pulpotomy, some authors have recommended pulpectomy routinely after the roots have fully formed (Fig. 17-13). 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%.[125,131]

Pulpectomy

Pulpectomy implies removal of the entire pulp to the level of the apical foramen.

INDICATIONS

Pulpectomy is indicated in complicated crown fracture of mature teeth if conditions are not ideal for vital pulp therapy or it is foreseeable that restoring the tooth will require a post placement. This procedure is no different from root canal treatment of a vital nontraumatized tooth.

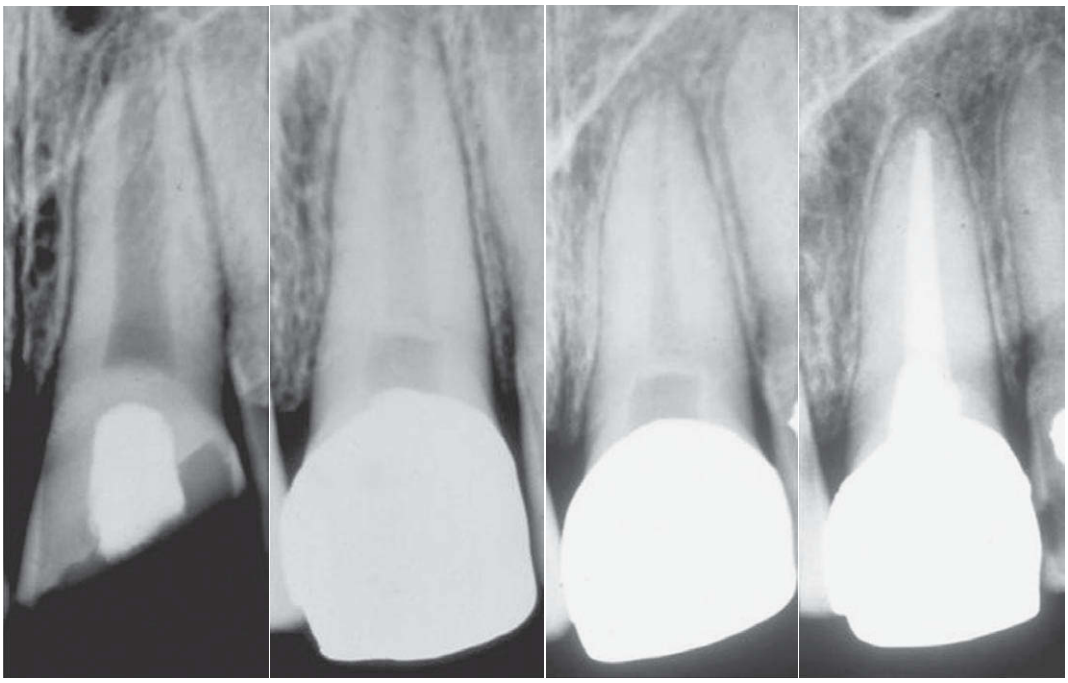


FIG. 17-13 Successful pulpotomy followed by a pulpectomy at 18 months. *(Courtesy Dr. Leif Tronstad.)*

Treatment of the Nonvital Pulp

Immature Tooth: Apexification

INDICATIONS

This procedure 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. Since 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.[44]

These problems are overcome by stimulating the formation of a hard-tissue barrier to allow for optimal filling of the canal and reinforcing the weakened root against fracture, both during and after apexification.[93,143]

TECHNIQUE

Disinfection of the Canal In the vast majority of cases, nonvital teeth are infected,[29,127] so the first phase of treatment is to disinfect the root canal system to ensure periapical healing.[37,48] The canal length is estimated with a parallel preoperative radiograph, and after access to the canals is 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.[49,136] 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. 17-14). Additional disinfecting action of calcium hydroxide is effective after its application for at least 1 week,[130] so the continuation of treatment can take



FIG. 17-14 Creamy mix of calcium hydroxide on a Lentulo spiral instrument ready for placement into the canal.

place any time after 1 week. Further treatment should not be delayed more than 1 month, since 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.[44,76,78] Pure calcium hydroxide powder is mixed with sterile saline (or anesthetic solution) to a thick (powdery) consistency (Fig. 17-15). 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 placed in the access cavity. A radiograph is taken, and the canal should appear to have become calcified, indicating that the entire canal has been filled with the calcium hydroxide (Fig. 17-16). 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 sulphate. These additives do not wash out as readily as calcium hydroxide, so if they are present in the canal, evaluation of washout is impossible.

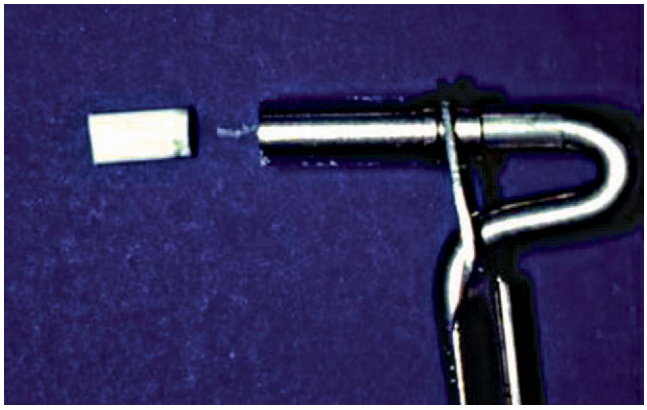


FIG. 17-15 Thick mix of calcium hydroxide.

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, it can be left intact for another 3 months. Excessive calcium hydroxide dressing changes should be avoided if at all possible, since the initial toxicity of the material is thought to delay healing.[95]

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.

MTA BARRIER

The creation of a physiologic hard-tissue barrier, while quite predictable, takes anywhere from 3 to 18 months with calcium hydroxide. The disadvantages of this long time period are that



FIG. 17-16 A-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.*)

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]

MTA has been used to create a hard-tissue barrier quickly after disinfection (see earlier in the section Traditional Method) of the canal (Fig. 17-17). Calcium sulphate is pushed through the apex to provide a resorbable extraradicular barrier against which to pack the MTA. The MTA 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 MTA and left for at least 6 hours. After the MTA is fully set, the entire canal is then filled with a root filling material. Some clinicians have suggested that the filling can be placed immediately, since the tissue fluids of the open apex will probably provide enough moisture to ensure that the MTA will set sufficiently. The cervical canal is then reinforced with composite resin to below the marginal bone level as described later (see Fig. 17-17).

A number of case reports have been published using this MTA apical barrier technique,[68,80,100] and it has steadily gained popularity with clinicians. Presently, no prospective long-term

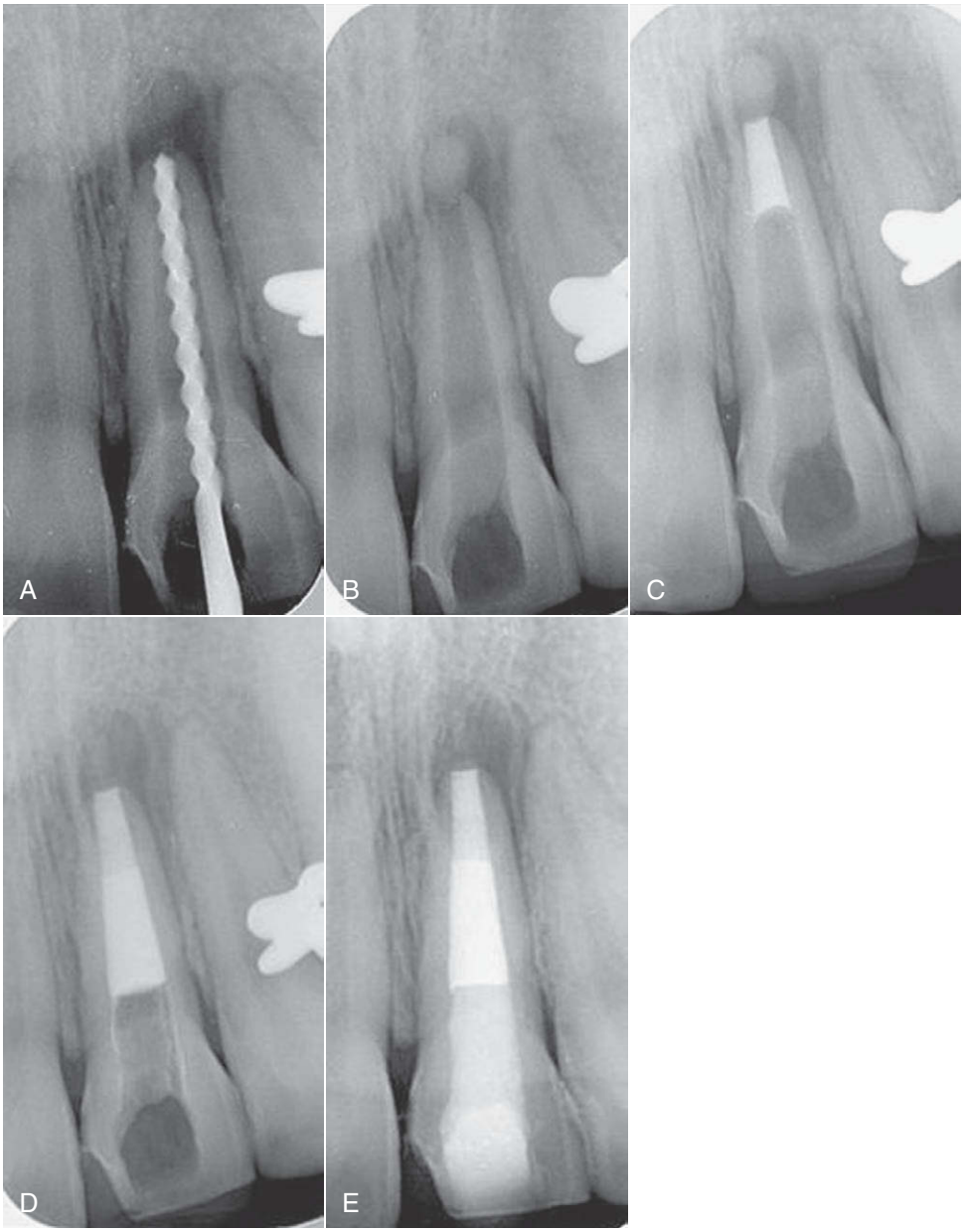


FIG. 17-17 Apexification with mineral trioxide aggregate (MTA). **A,** The canal is disinfected with light instrumentation, copious irrigation, and a creamy mix of calcium hydroxide for 1 month. **B,** Calcium sulfate is placed through the apex as a barrier against which the MTA is placed. **C,** A 4-mm 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.)*

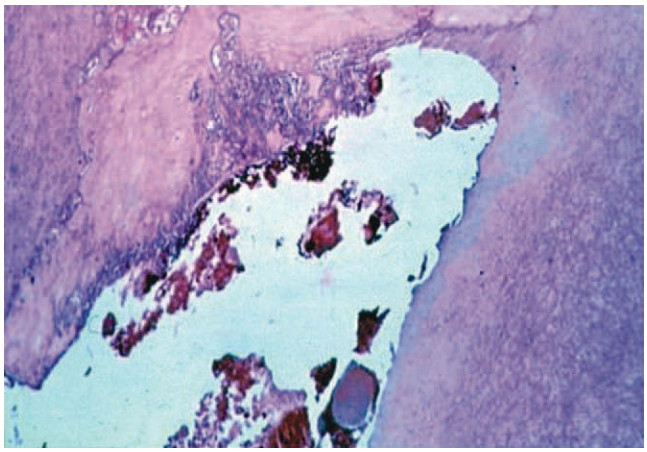


FIG. 17-18 Histologic appearance of hard-tissue barrier after calcium hydroxide apexification. The barrier is composed of cementum and bone with soft-tissue inclusions.

outcome study is available comparing its success rate with the traditional calcium hydroxide technique.

Filling the Root Canal Since 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 10). 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. 17-18). Included also are islands of soft connective tissue, giving the barrier a "Swiss cheese" consistency.[31,45] 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. 17-19). Formation of the hard-tissue barrier might be some distance short of the radiographic apex. This is because it 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.

Reinforcement of Thin Dentinal Walls The apexification procedure has become a predictably successful procedure (see Prognosis),[61] but the 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.[51,147] It has been reported that approximately 30% of these teeth will fracture during or after endodontic treatment.[93] 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.[69,91] Thus after root filling, the material should be removed to below the



FIG. 17-19 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.

marginal bone level and a bonded resin filling placed (see Fig. 17-17).

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 occurs predictably with long-term calcium hydroxide treatment (79% to 96%).[44,93] 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 Revascularization Regeneration of a necrotic pulp has been considered possible only after avulsion of an immature permanent tooth that was reimplanted within 40 minutes (see 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 16). 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 will act 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.[26,107,134] Because in most cases, the crown of the tooth is intact and caries-free, bacterial penetration into the pulp space through cracks[97] 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 has been thought up to now to be impossible.[104] 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. 17-20) describes the treatment of an immature second mandibular premolar with 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, and the patient was prescribed a mixture of antibiotics consisting of equal amounts of ciprofloxacin, metronidazole, and minocycline for 3 weeks.[82]

After rinsing out the antibody mixture after three weeks, 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

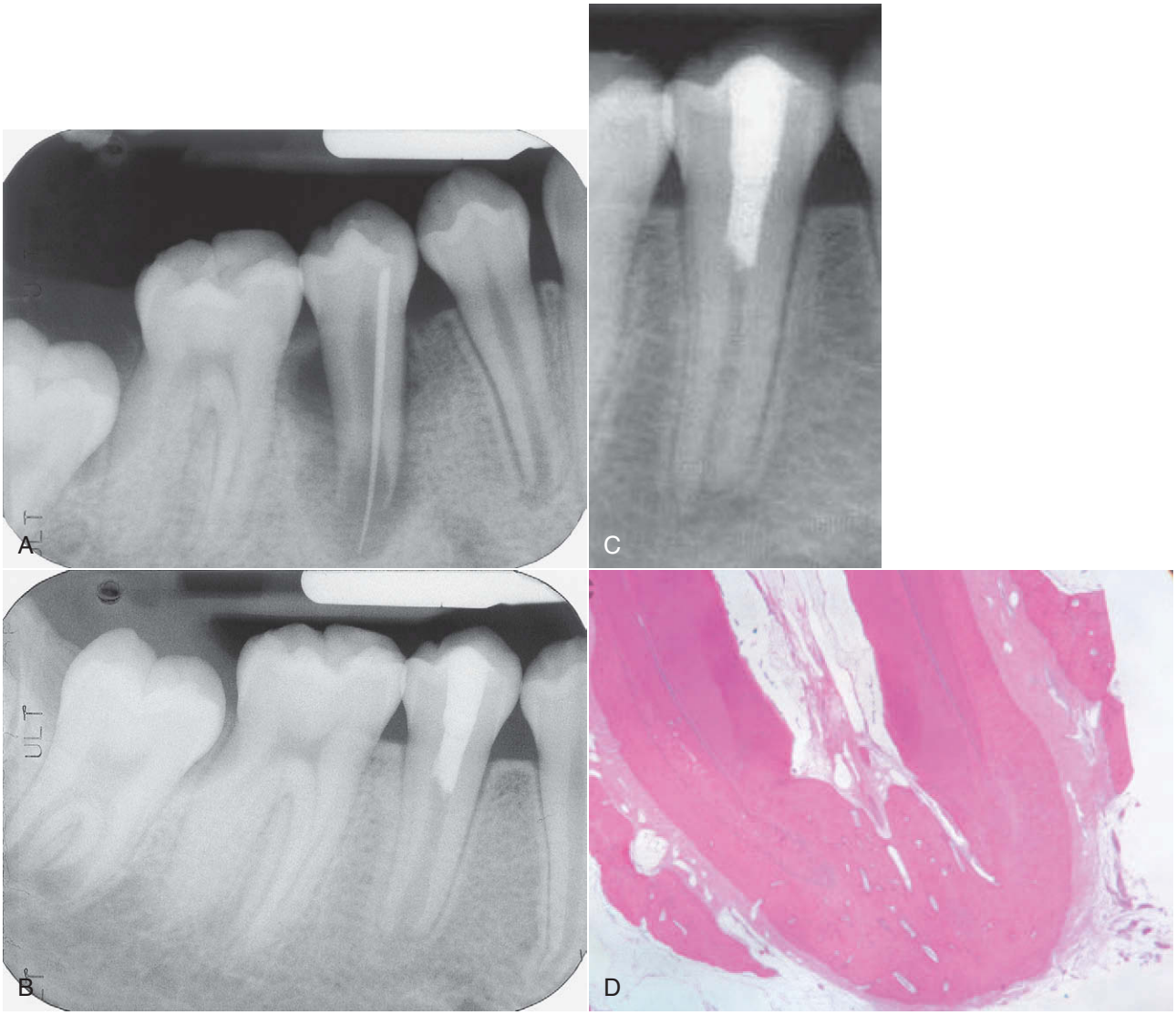


FIG. 17-20 A, Preoperative radiograph showing an immature root with periradicular periodontitis and a traced sinus tract to the apex of the root. **B,** Radiographic checkup 7 months after treatment that included application of a mixture of ciprofloxacin, metronidazole, and minocycline. **C,** At 24-month checkup, showing continued root development both in length and width of the root. **D,** Histologic slide from Thibodeau et al.[144] 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.

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 the adjacent and contralateral teeth. More recently, published cases and case series reports have further supported that it is possible to have ingrowths of tissue followed by new apposition of dentin and continuation of root formation.[89,144] Studies have also confirmed the potent antibacterial properties of the triantibiotic paste used in these cases. Studies are underway to find a synthetic matrix which will act as a more predictable scaffold for new ingrowth of tissue than the blood clot that was used in these examples.[144,162] The procedure described here can be attempted in most cases, and if after 3 months there are no signs of regeneration, then the more traditional treatment methods can be initiated.

Mature Tooth

Conventional endodontic therapy.

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 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.[166] 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.[8,9,86]

Diagnosis and Clinical Presentation

Clinical presentation 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. Since root fractures are usually oblique (facial to palatal) (Fig. 17-21), one periapical radiograph may easily miss its presence. It is imperative to take at least three angled radiographs (45, 90, 110 degrees) so that at least at one angulation, the x-ray beam will pass directly through the fracture line to make it visible on the radiograph (Fig. 17-22).

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 will not be 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 then once it is loose, rotate it back to its original position (Fig. 17-23). The traditionally recommended[113] 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, it will likely not be possible to reposition the segments close to their original position, compromising the long-term prognosis for the tooth.



FIG. 17-21 Extracted root after a root fracture. Note the oblique angle of the fracture.

Healing Patterns

Investigators[20] 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. 17-24, *A*).
2. *Healing with interproximal connective tissue.* Radiographically, the fragments appear separated by a narrow radiolucent line, and the fractured edges appear rounded (see Fig. 17-24, *B*).
3. *Healing with interproximal bone and connective tissue.* Radiographically, the fragments are separated by a distinct bony ridge (see Fig. 17-24, *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 (see Fig. 17-24, *D*).

The first three types of healing patterns are considered successful. The teeth are usually asymptomatic and respond posi-

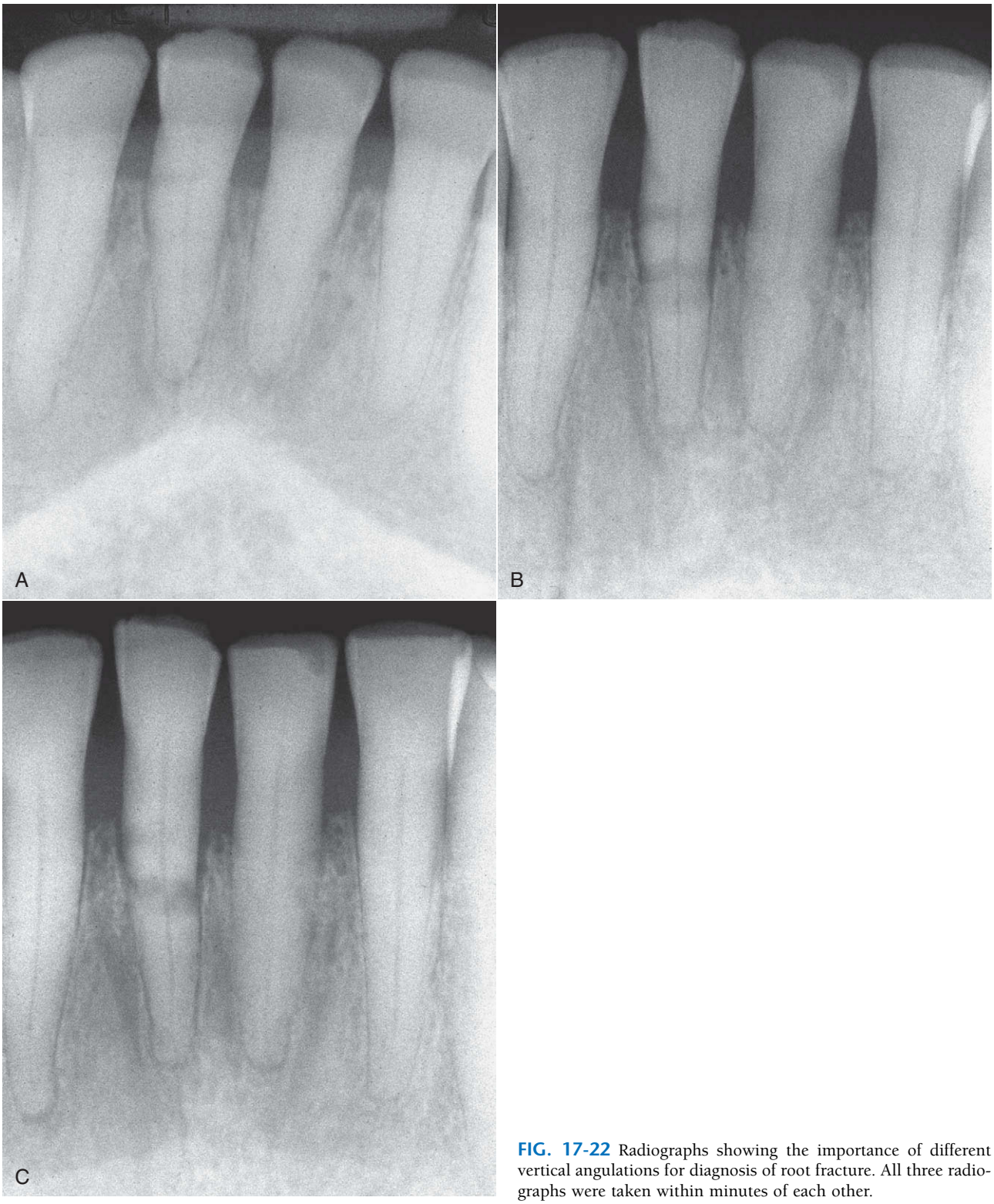


FIG. 17-22 Radiographs showing the importance of different vertical angulations for diagnosis of root fracture. All three radiographs were taken within minutes of each other.

tively to sensitivity testing. Coronal yellowing is possible because calcific metamorphosis of the coronal segment is not unusual[87,166] (see Fig. 17-29).

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[20] (see Fig. 17-24, *D*).

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

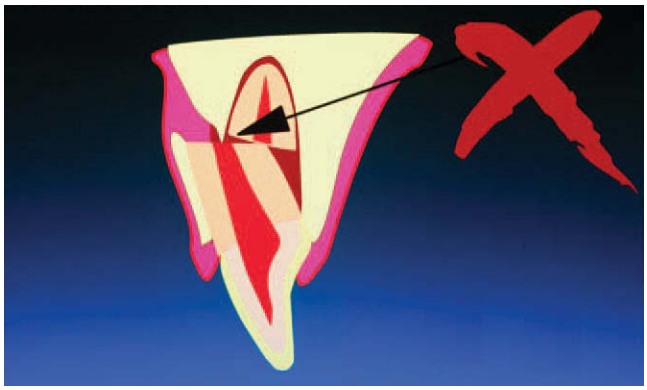


FIG. 17-23 Figure of facial root fracture manipulated back into place.

splinted, chances of healing do not differ from midroot or apical fractures (Fig. 17-25).[166] 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 a restoration to be fabricated.

Midroot and Apical Root Fractures

Permanent pulpal necrosis occurs in 25% of root fractures.[45,86] Initially it is likely that in many cases the pulp in the coronal segment becomes necrotic after the injury, but then because of a very large apical opening in the coronal segment, revascularization is possible if the segments are well reapproximated. In the vast majority of 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 so that long-term calcium hydroxide treatment or an MTA apical plug is indicated (see 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 when 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 detrimental effect on healing of the fracture site. If healing of the fracture has been complete and 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 with all dental traumatic injuries: at 3, 6, and 12 months and yearly thereafter.

Prognosis

Factors that influence repair include:

1. Degree of dislocation and mobility of the coronal fragment are extremely important in determining outcome.[7,17,87,137,166] 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.[17,85]
3. Quality of treatment is vital to successful repair. Prognosis improves with quick treatment, close reduction of the root segments, and semirigid splinting for 2 to 4 weeks.[18]

Complications are pulp necrosis and root canal obliteration. Pulp necrosis can be treated successfully[45,86] 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

Types of luxation injury:

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 reported incidences ranging from 30% to 44%.[50]

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.[133] 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 (see 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 has to first be slightly and gently pulled down to loosen the hold prior to repositioning it in its original position. Current IADT

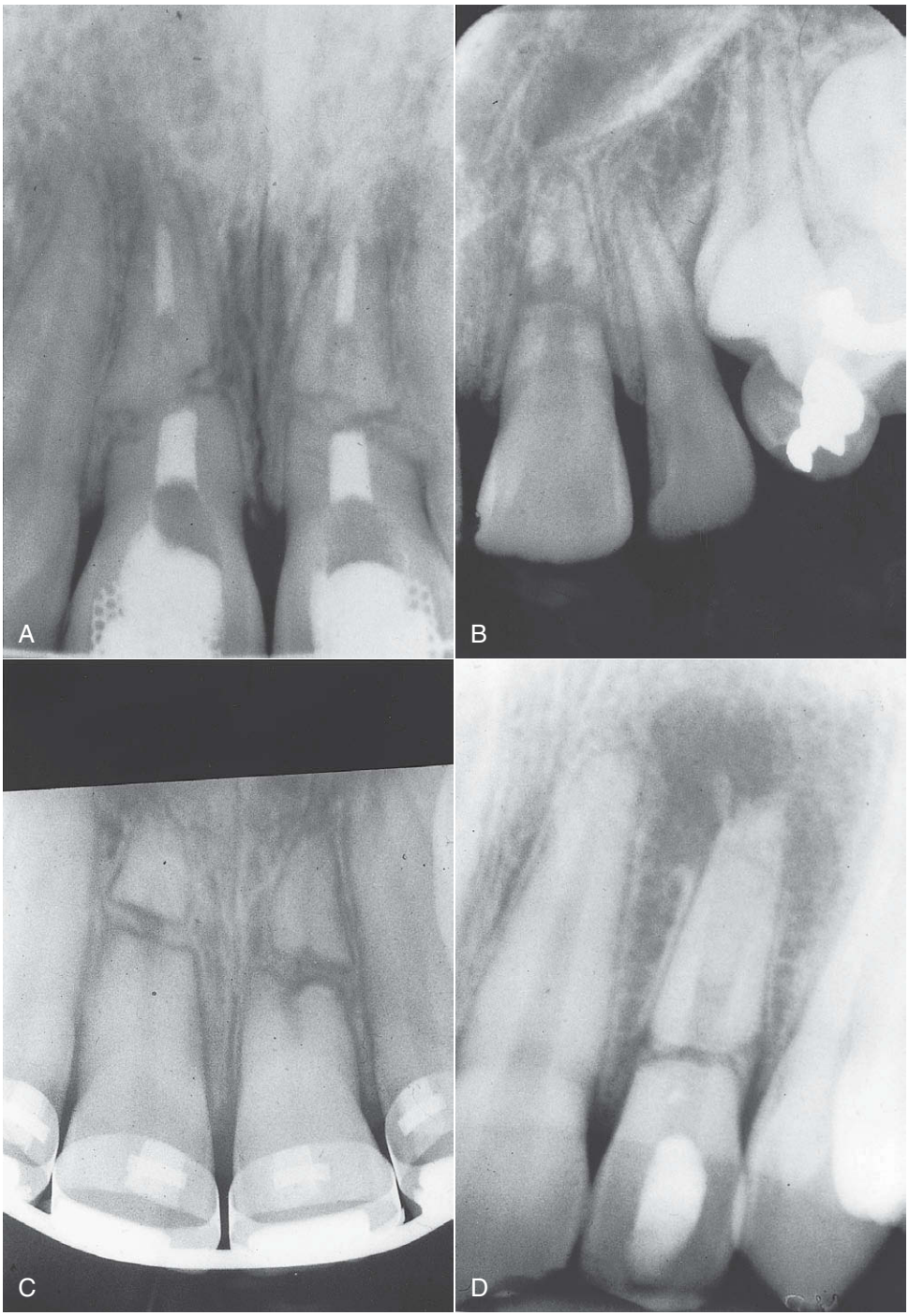


FIG. 17-24 Healing patterns after horizontal root fractures. **A,** Healing with calcified tissue. **B,** Healing with interproximal connective tissue. **C,** Healing with bone and connective tissue. **D,** Interproximal connective tissue without healing.

guidelines call for 2 weeks of physiologic splinting in cases of extrusion luxation and 4 weeks for lateral luxation. Decision on root canal therapy follows the guidelines for avulsion (see 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, so 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.[92] Alternative treatment like orthodontic extrusion or immediate surgical reposition should therefore be considered. If orthodontic extrusion is planned for an intruded tooth, it should be initiated as soon as possible, delaying no more than 2 to 3 weeks post trauma. There are only a few studies that have evaluated the true efficacy of this approach. One study[157] using a dog model indicated that severely intruded teeth showed

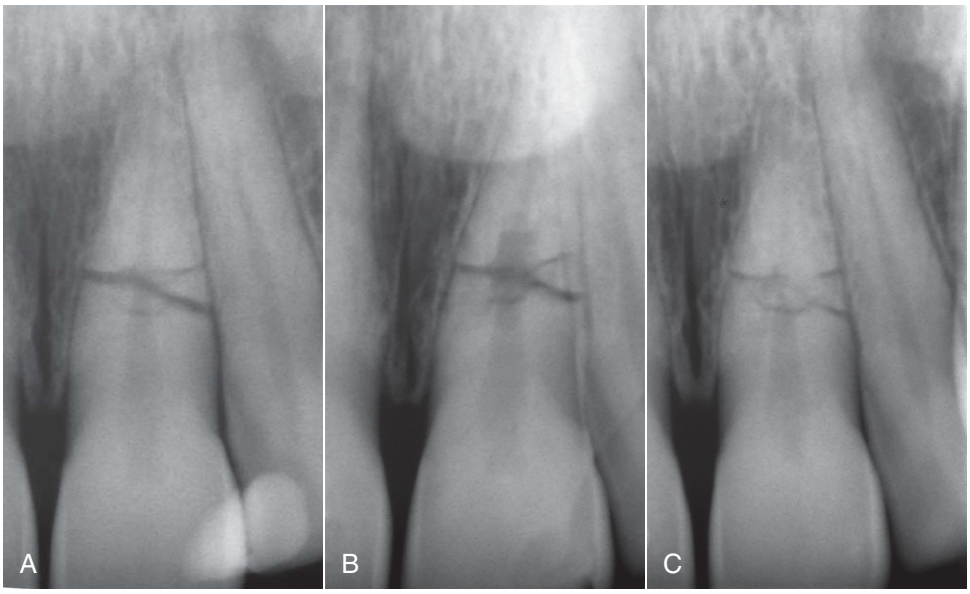


FIG. 17-25 Internal root resorption in the root fracture area; tooth was diagnosed with compound midroot fracture. **A,** Coronal portion was repositioned and then splinted to the lateral incisor with a composite splint. **B,** At 6-month recall, the pulp responded normally to cold, but an internal root-resorptive 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.

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[42] (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,[52] a retrospective study of 58 intruded human teeth, indicated that surgical repositioning resulted in a predictable outcome, with only five of the teeth being 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 is dependent 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 is 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 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 will include root resorption as well as 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 will occur 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 byproducts of this mechanical damage stimulate an inflammatory response. The healing response is dependent 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

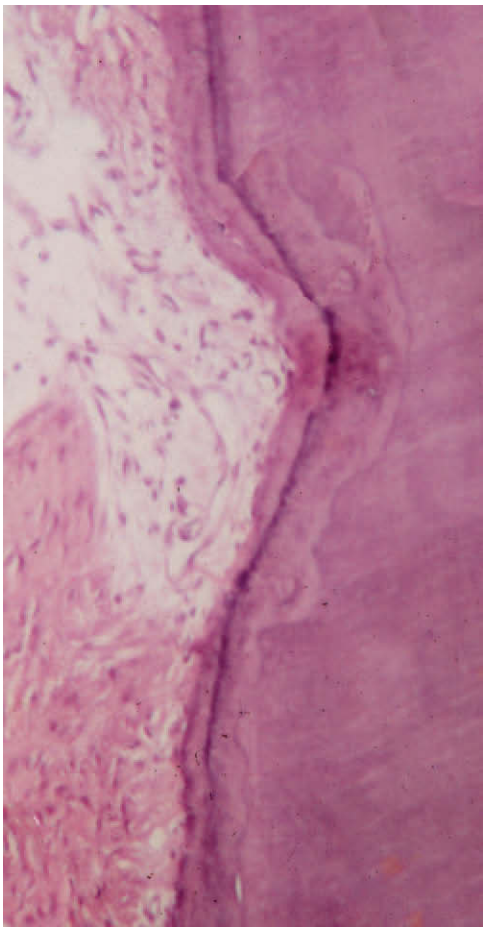


FIG. 17-26 Histologic section showing previous root resorptive defect healed with new cementum and periodontal ligament.

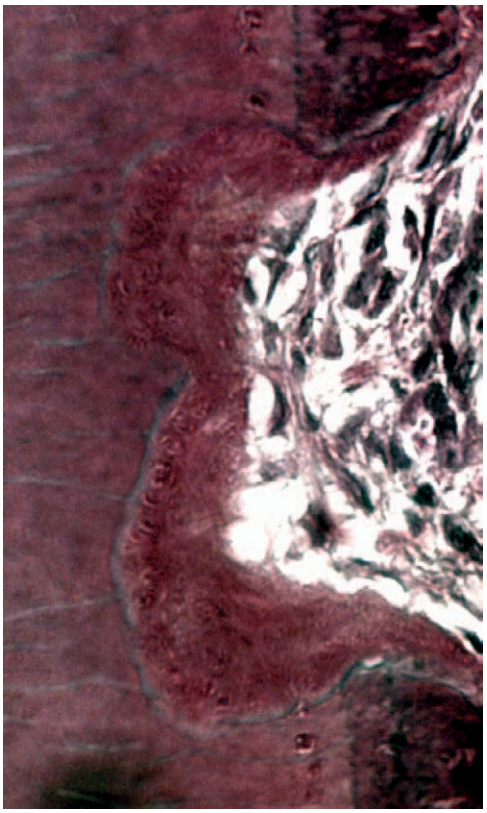


FIG. 17-27 Histologic appearance of osseous replacement. There is direct contact between bone and root structure. Resorptive defects are seen in the bone and root tissue, which is the normal physiologic process of bone turnover. The resorptive defects will be filled by new bone and additional areas resorbed. In this way the entire root will be replaced by bone at a rate dependent on the metabolic rate of the patient.

local inflammatory response and a localized area of root resorption. If no further inflammatory stimulus is present, periodontal healing and root surface repair will occur within 14 days[71] (Fig. 17-26). The resorption is localized to the area of mechanical damage, and treatment is not required, since 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.[96] 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 which are precursors of bone, rather than the slower-moving periodontal ligament cells, will 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*.[96] However, the ankylosis and osseous replacement that follows cannot be reversed and can be considered a physiologic process, since 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*[152] (Fig. 17-27). 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. 17-28).

Treatment Treatment strategies for limiting the effect of the traumatic injury to the periodontal structures are outside the scope of this chapter, but in general this involves minimizing the initial inflammatory response to the injury. The initial inflammation is destructive in nature and will increase the surface area of root to be covered in the healing phase.[21] The smaller the surface area to be covered by new cementum, the higher the chances of favorable repair.

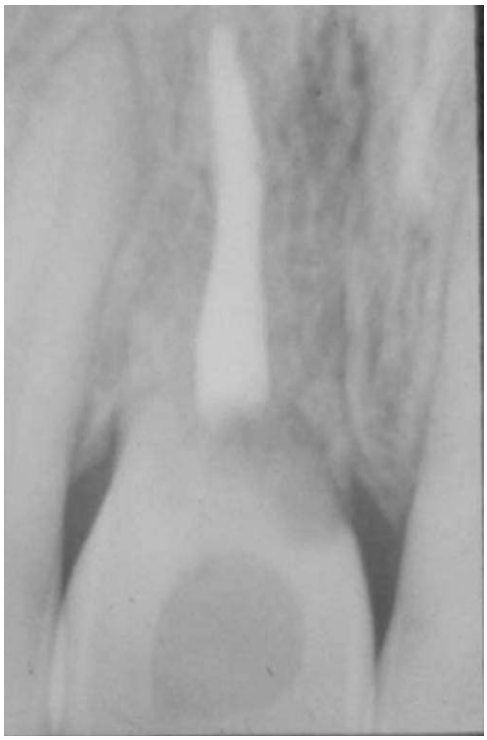


FIG. 17-28 Radiographic appearance of osseous replacement. The root acquires the radiographic appearance of the surrounding bone (without a lamina dura). Note that radiolucencies typical of active inflammation are not present.

A study has indicated that if Ledermix, a drug combining corticosteroid and tetracycline, is placed in the root canal immediately after a severe trauma where osseous replacement was expected, favorable healing will occur at a very high rate.[36] 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.[39] Both 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 STIUMLUS IN THE ROOT CANAL

Recognized inflammatory stimuli that cause root resorption are pressure, pulp space infection, and sulcular infection. This chapter is focused on pulp space infection. Other chapters in this text deal with orthodontic and periodontal issues (see Chapters 18 and 23).

CONSEQUENCES OF APICAL NEUROVASCULAR SUPPLY DAMAGE

Pulp Canal Obliteration (Calcification) *Pulp canal obliteration* is common after luxation injuries (Fig. 17-29). 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 is a nidus for subsequent calcification if the pulp remains vital.[7,15] Pulp canal obliteration can usually be diagnosed within the first year after injury[11] and was found to be more frequent in teeth with open apices (>0.7 mm radiographically), in teeth with extrusive and lateral luxation injuries, and in teeth that have been rigidly splinted.[11]

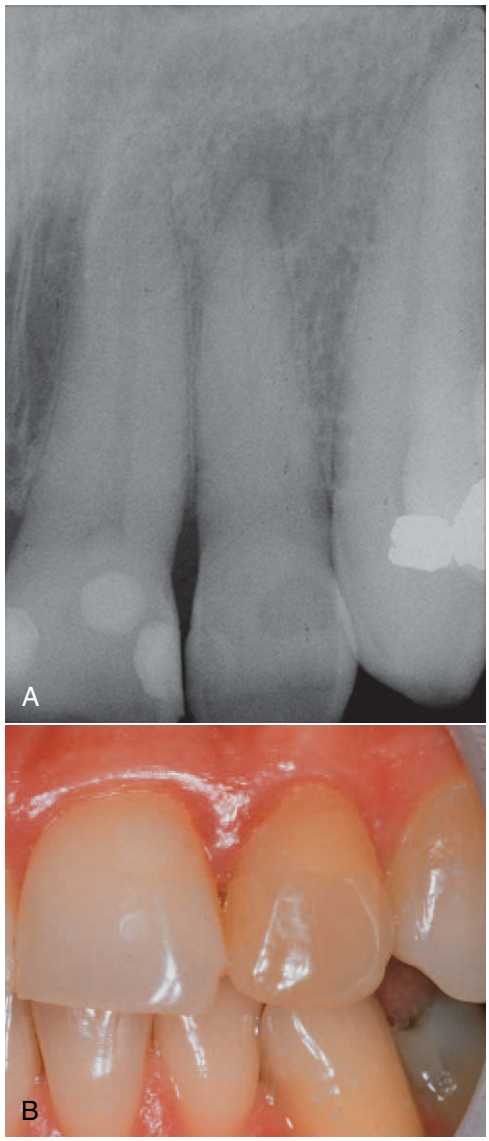


FIG. 17-29 A, Pulp canal calcification after a luxation injury. Radiographic appearance of a calcified maxillary lateral incisor; note that despite calcification, it has now become necrotic and infected, evident by the periapical lesion. **B,** The typical yellow appearance of the tooth caused by thickened dentin in the pulp chamber.

Pulp Necrosis The factors most important for the development of pulp necrosis are type of injury (concussion least, intrusion most) and the stage of root development (mature apex > immature apex).[10] Pulp necrosis will most likely 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



FIG. 17-30 Inflammatory root resorption caused by a pulp space infection. Note the radiolucencies in the root and surrounding bone. *(Courtesy Dr. Fred Barnett.)*

and bone resorption and will continue 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. 17-30).

To have pulp space infection, the pulp must first become necrotic. Necrosis will occur 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 will become infected. For details of the typical bacterial contents of a traumatized necrotic pulp, the reader is referred to Chapter 15 or Bergenholtz.[29] Because a serious injury is required for pulp necrosis, it is usual that areas of cemental covering of the root are also affected, resulting in loss of its protective (insulating) quality. Now bacterial toxins can pass through the dentinal tubules and 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[149] (Fig. 17-31). Radiographically the resorption is observed as progressive radiolucent areas of the root and adjacent bone (see Fig. 17-30).

Treatment Assessing attachment damage due to 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.[155,156] Root canal disinfection removes the stimulus to the periradicular inflammation, thus the resorption will stop.[71,155,156] In most cases a new attachment will form, but if a large area of root is affected, osseous replacement can result

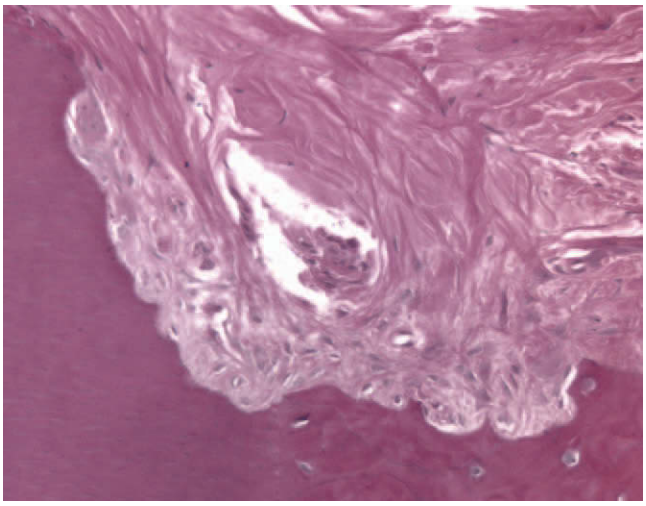


FIG. 17-31 Histologic appearance of multinucleated osteoclasts (dentinoclasts) resorbing the dentin of the root.

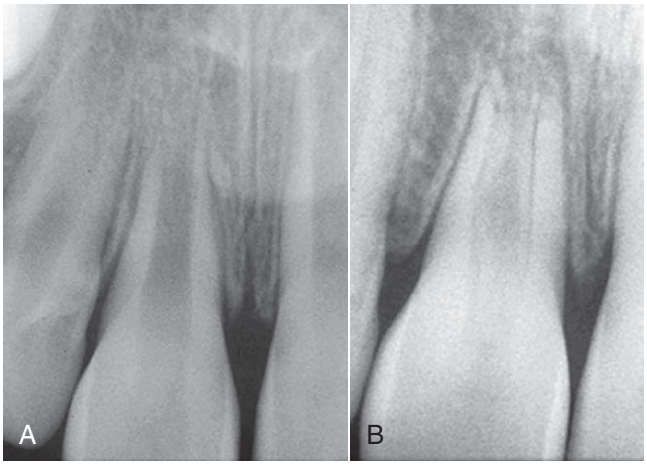


FIG. 17-32 Revascularization of immature root. A tooth with an open apex was replanted soon after the avulsion. The checkup radiograph 12 years later confirms that regrowth has taken place into the pulp chamber. It appears that a bone plug has grown in, and new periodontal ligament has formed, with a lamina dura within the pulp canal. *(Courtesy Dr. Cecilia Bourguignon.)*

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 where 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 replaced in their original position within 60 minutes of the injury[46] (Fig. 17-32). 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.[46,116] Even under the best conditions, however, revascularization will fail 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, since 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 (see Fig. 17-4). These devices appear to detect the presence of vital tissue in the pulp space by 4 weeks after the traumatic injury.[165]

b. *Prevent root canal infection by 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.[155,156] 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 dressing with a creamy mix of calcium hydroxide is placed[155] (see Fig. 17-14). The clinician can now fill the canal at his/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 where the endodontic treatment is started within 10 days of the injury.[155]

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.[156] Calcium hydroxide can effect an alkaline pH in the surrounding dentinal tubules (Fig. 17-33), 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, since the radiodensity of calcium hydroxide in the canal is usually similar to the surrounding dentin (see Fig. 17-16). 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. Since 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. 17-34).

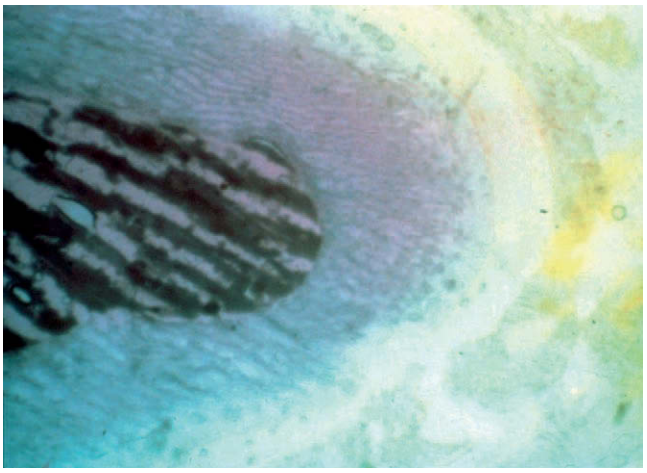


FIG. 17-33 High pH of calcium hydroxide. The root was filled with calcium hydroxide and then cut in cross section. A pH indication shows the high pH in the canal and surrounding root, whereas the surrounding tissue is a neutral pH.

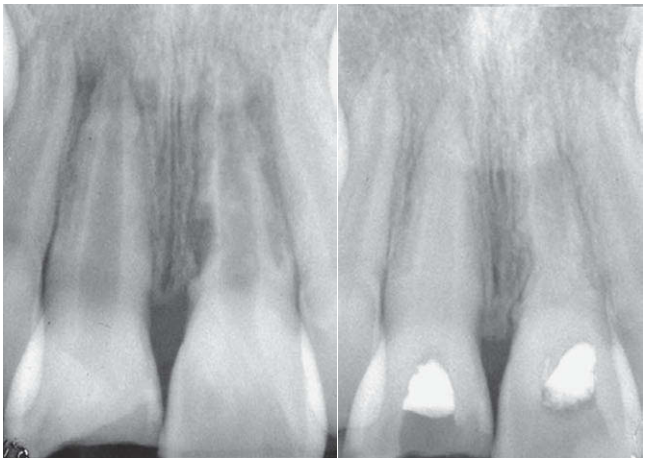


FIG. 17-34 Healing of external inflammatory root resorption after calcium hydroxide treatment. The radiolucencies seen before treatment have disappeared with the reestablishment of the lamina dura. *(Courtesy Dr. Fred Barnett.)*

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

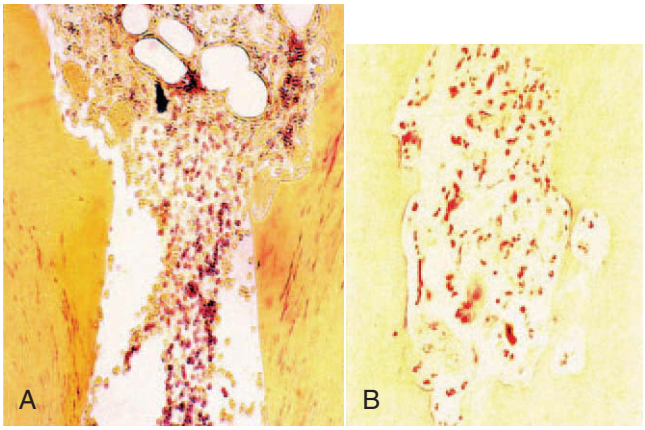


FIG. 17-35 Histologic appearance of internal root resorption. **A,** Section stained with Brown and Brenn. Bacteria are seen (in the dentinal tubules) communicating between the necrotic coronal segment and the apical granulomatous tissue and resorbing cells. **B,** An area of active internal root resorption. (*Courtesy Dr. Leif Tronstad.*)

adjacent to the granulation tissue in the pulp (Fig. 17-35). 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 due to an infected coronal pulp space. Communication between the coronal necrotic tissue and the vital pulp is through appropriately oriented dentinal tubules[159] (see Fig. 17-35). One investigator reports[159] that resorption of the 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[138] 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.[149]

Reasons for the loss of predentin adjacent to the granulation tissue are not obvious. Trauma frequently has been suggested as a cause.[124,160] 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.[159]

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

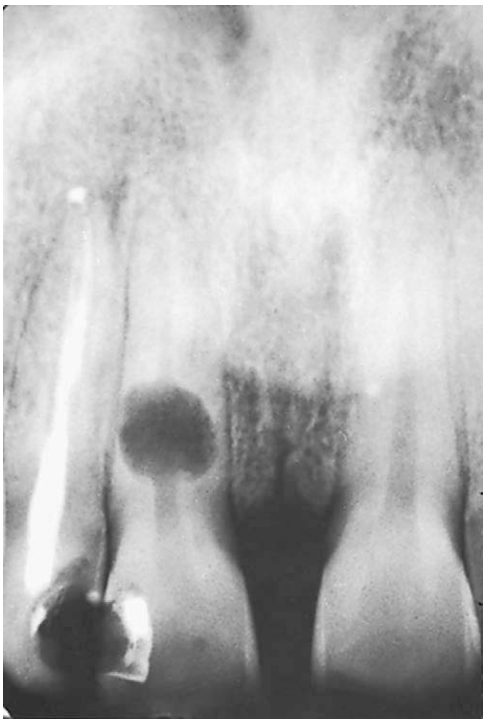


FIG. 17-36 A maxillary incisor with internal root resorption. Uniform enlargement of the pulp space is apparent. Outline of the canal cannot be seen in the resorptive defect.

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. 17-36). 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. 17-35). 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[129,149,156,159] (see Fig. 17-35). Unlike external root resorption, the adjacent bone is not affected with internal root resorption.

TREATMENT

Treatment of internal root resorption is conceptually very easy. Since the resorptive defect is the result of the inflamed pulp, and the blood supply to the tissue is through the apical

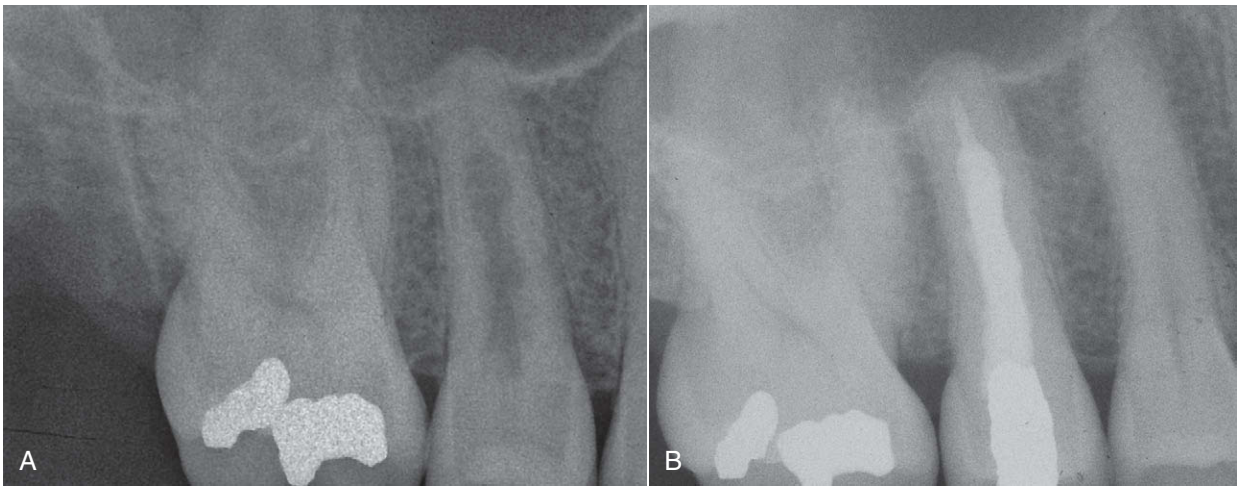


FIG. 17-37 A, Internal root resorption in a maxillary premolar with a history of trauma 7 years prior to the diagnosis (patient's head slammed against side window during an automobile accident). **B,** Three-year follow-up radiograph after endodontic treatment.

foramina, endodontic treatment that effectively removes the blood supply to the resorbing cells is the treatment approach. After adequate anesthesia is 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. 17-37).

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. What follows is 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. 17-36). A defect on the external aspect of the root moves away from the canal as the angulation changes (Fig. 17-38). 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 the radiolucent resorptive defect appear contiguous (see Fig. 17-36). When the defect is external, the root canal outline appears normal and can usually be seen "running through" the radiolucent defect (Fig. 17-39).

External inflammatory root resorption is always accompanied by resorption of the bone in addition to the root (Fig. 17-40); 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. 17-36). On rare occasions if the internal defect perforates the root, the bone adjacent to it is resorbed and appears radiolucent on the radiograph.

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, since 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, since 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. 17-41) 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 due to sulcular infection. History of trauma (often forgotten or its long-

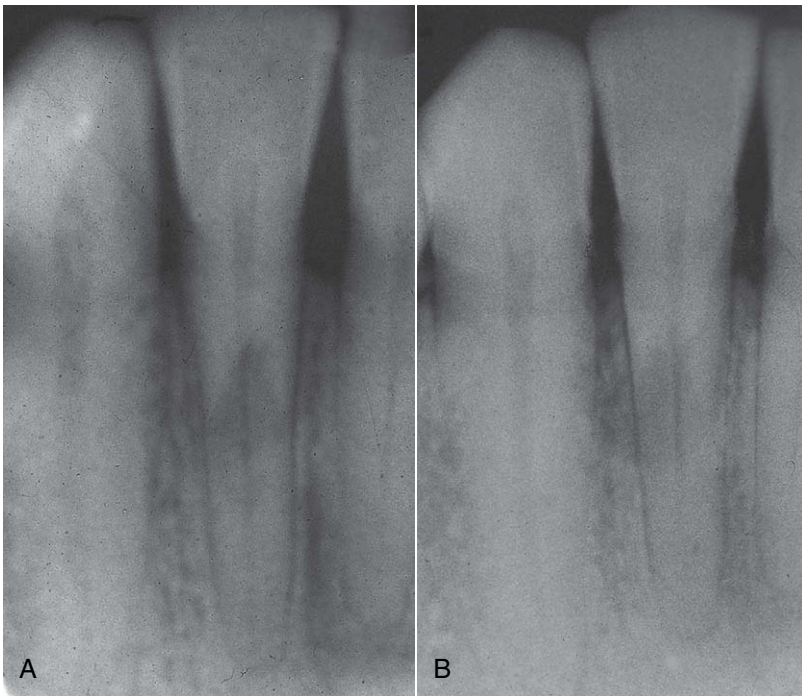


FIG. 17-38 External root resorption. Radiographs from two different horizontal projections depict movement of the lesion to outside the confines of the root canal.



FIG. 17-39 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.



FIG. 17-40 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 infra-bony pocket.

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.

The majority of misdiagnoses of resorptive defects are made between subepithelial external and internal root resorptions. The diagnosis should always be confirmed while treatment is proceeding. 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, since 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

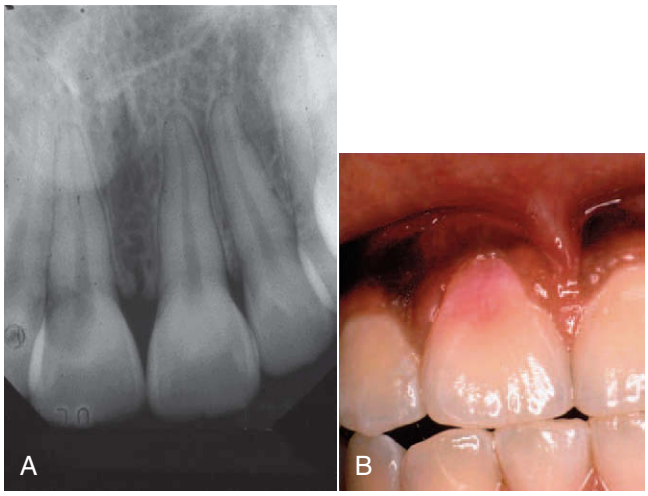


FIG. 17-41 Pink spot of subepithelial external inflammatory root resorption. **A,** Radiographic appearance. **B,** Clinical appearance.

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 is perforating 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 lay 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 usually minimal.[12,135] The hydrated periodontal ligament cells will maintain their viability and repair after replantation, with minimal destructive inflammation as a byproduct. Since 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. 17-26).

If excessive drying occurs before replantation, the damaged periodontal ligament cells will elicit a severe inflammatory response over a diffuse area on the root surface. Unlike the situation described earlier where 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*[21,153] (see Figs. 17-27, 17-28).

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 will inevitably become 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 lead to rapid loss of the tooth[149] (see Fig. 17-30).

The consequences after tooth avulsion appear 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 (cemental) rather than unfavorable (osseous replacement or inflammatory resorption) responses.

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 are made to minimize necrosis of the remaining periodontal ligament while the tooth is out of the mouth. Pulpal sequels 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.[22,135] 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 as an educational offering to, for example, 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.[79] Water is the least desirable storage medium because the hypotonic environment causes rapid cell lysis and increased inflammation on replantation.[33,34]

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.[154] Presently they are considered impractical, since they need to be present at the accident site before the injury occurs, but if we consider that more than 60% of avulsion injuries occur close to home or school,[74] it should 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 where 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.

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 (see 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. Hank's Balanced Salt Solution is presently considered the best medium for this purpose. Milk or physiologic saline is also appropriate for storage purposes.

The medical and accident history is taken and a clinical exam 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 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 is dependent 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,[46] but because the tooth was dry for less than 60 minutes (replanted or placed in appropriate medium), the chance for periodontal healing exists. Most importantly, 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, where periodontal healing would be expected.[12,27,135]

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 as well as continued root development is possible (see Fig. 17-32). Investigators[46] 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.[116,165] A study found that covering the root with minocycline (Arestin, OraPharma, Inc,

Warminster, PA), which attaches to the root for approximately 15 days, further increased the revascularization rate in dogs.[116] 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 will occur in humans as well. As with the tooth with the closed apex, the open-apex tooth is then 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.[22,135] 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 will initiate the inflammatory response on replantation. The tooth should then be soaked in 2% stannous fluoride for 5 minutes and replanted.[32,126] A few years ago, studies were published that indicated that Emdogain (enamel matrix protein, Andover, MA) could be beneficial in teeth with extended extra oral dry times, not only to make the root more resistant to resorption but possibly to stimulate the formation of new periodontal ligament from the socket[57,83] (see Fig. 17-8). 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.[121]

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, where 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.

Since 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, that they recommend 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,[6,56,60] the height, and more importantly the width, of the alveolar bone will 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 within the socket to facilitate replacement of the tooth into the socket.[71] 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 splinting technique that allows physiologic movement of the tooth during healing and is in place for a minimal time period results in a decreased incidence of ankylosis.[3,13,71] Semirigid (physiologic) fixation for 1 to 2 weeks is recommended.[3,5,60] 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 splint. A new titanium trauma splint (TTS) has recently been shown to be particularly effective and easy to use[158] (Fig. 17-42). 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.[152] 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.[152]

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. If these lacerations are sutured, care must be taken to clean the wound thoroughly beforehand because dirt or even minute

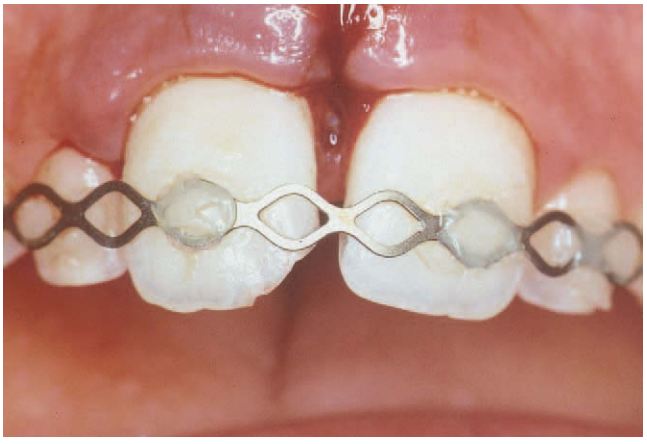


FIG. 17-42 Titanium trauma splint (TTS) in place.

tooth fragments left in the wound affect healing and the esthetic result.

ADJUNCTIVE THERAPY

Systemic antibiotics given at the time of replantation and prior to endodontic treatment are effective in preventing bacterial invasion of the necrotic pulp and therefore subsequent inflammatory resorption.[72] Tetracycline has the additional benefit of decreasing root resorption by affecting the motility of the osteoclasts and reducing the effectiveness of collagenase.[117] The administration of systemic antibiotics is recommended, beginning at the emergency visit and continuing until the splint is removed.[72] 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.[117,118] 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 are 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.[36,39] 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

This 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 this 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. In cases where endodontic treatment is delayed or signs of resorption are present, treat with long-term calcium hydroxide treatment before obturation.

No chance exists for revascularization of these teeth, therefore endodontic treatment should be initiated at the second visit 7 to 10 days later.[14,46] If therapy is initiated at this optimum time, the pulp should be necrotic without (or with minimal) infection.[99,155] Endodontic therapy with an effective interappointment antibacterial agent[155] over a relatively short period (1 to 2 weeks) is sufficient to ensure effective disinfection of the canal.[130] Long-term calcium hydroxide treatment should always be considered when the injury occurred more than 2 weeks before initiation of the endodontic treatment or especially if radiographic evidence of resorption is present.[155]

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. 17-15). The canal is obturated when a radiographically intact periodontal membrane can be demonstrated around the root (see Fig. 17-34). Calcium hydroxide is an effective antibacterial agent[37,130] and favorably influences the local environment at the resorption site, theoretically promoting healing.[150] 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.[150] However, the 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.[95]

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 (DENTSPLY [Australia], Melbourne, Australia), is effective in treating inflammatory root resorption by inhibiting the spread of dentinoclasts[111] without damaging the periodontal ligament; however, its ability to diffuse through human tooth root has been demonstrated,[1] and its release and diffusion is enhanced when used in combination with calcium hydroxide paste.[2]

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 blood supply[46,116,164] (Fig. 17-43). The initiation of endodontic treatment is avoided if at all possible, unless definite signs of pulp necrosis (e.g., periradicular inflammation) are present. 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.[46,164]

Patients are recalled every 3 to 4 weeks for pulp vitality testing. Studies indicate that thermal tests with carbon dioxide snow (−78° C) or difluorodichloromethane (−50° C) placed at the incisal edge or pulp horn are the best methods of sensitivity testing, particularly in young permanent teeth.[64,65,108] One of these two tests must be included in the pulp vitality testing. Recent reports confirm the superiority of the laser Doppler

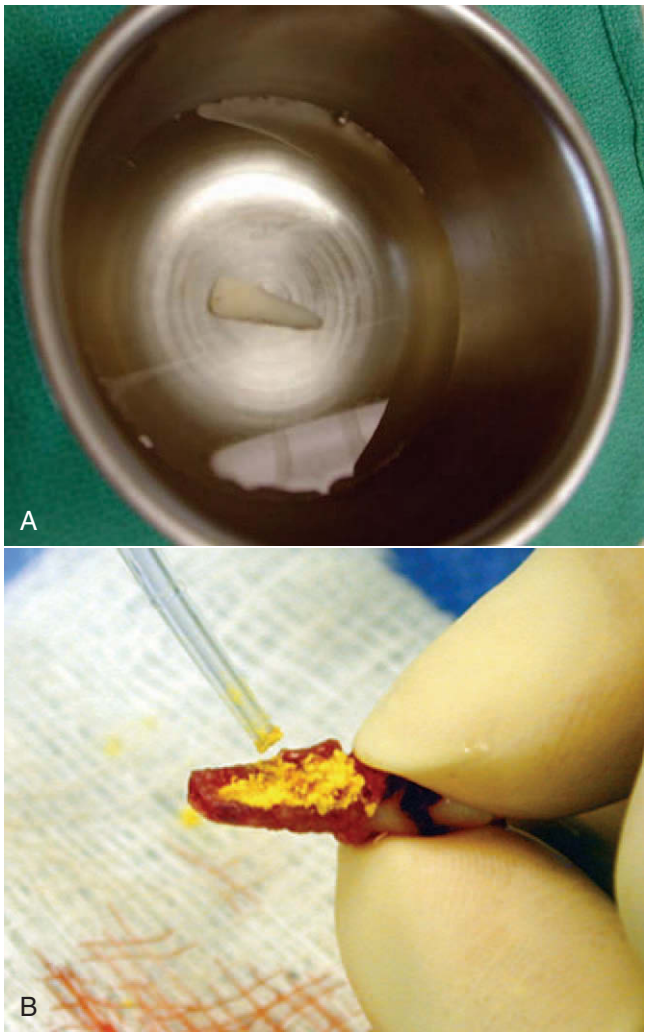


FIG. 17-43 A, Avulsed tooth soaking in doxycycline. **B,** Minocycline powder placed on the root surface before replantation.

flowmeter in the diagnosis of revascularization of traumatized immature teeth[165]; however, the cost of such an instrument precludes its use in the average dental office. Radiographic (apical breakdown and/or signs of lateral root resorption) and clinical (pain to percussion and palpation) signs 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

As with more than 60 minutes dry time.

These teeth are treated endodontically in the same way as those 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,[153,156] 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.

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 first be removed from the walls of the access cavity before placing the temporary restoration, because it is soluble and will wash 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 perform a detailed clinical examination on 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

At the clinician's convenience or following 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. 17-34) 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.[115] 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 esthetics 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. 17-28), a more closely monitored follow-up schedule is indicated. In the case of inflammatory root resorption (see Fig. 17-30), 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 18

Endodontic and Periodontal Interrelationships

DAVID G. KERNS and GERALD N. GLICKMAN

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

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 mortality.[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, by 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.[70,76,77]

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 intercommunications 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.

*References 48, 53, 65, 70, 74, and 76.

†References 7, 18, 45, 52, 67, and 77.

TABLE 18-1

Incidence of Furcation Canals

Investigators	Techniques	Incidence
Rubach and Mitchell (1965)[65]	Sectioned teeth	45%
Lowman et al. (1973)[52]	Dissecting microscope	Maxillary molars 59% Mandibular molars 55%
Burch and Hulen (1974)[13]	Radiopaque dye	Accessory furcal canals 76%
Vertucci and Williams (1974)[81]	Hematoxylin dye	46%
Kirkham (1975)[43]	Radiopaque dye	23%
Gutmann (1978)[33]	Safranin dye	Maxillary molars 28.4% Mandibular molars 27.4%

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 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 potential paths for inflammatory reciprocity.[13,33,45,47,52,65]

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 18-1 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 following induction of pulpal inflammation.[69,70] Serial sectioning of 74 teeth has revealed 45% of accessory canals are present primarily in the apical region.[65] 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. One investigator introduced safranin dye into 102 molar teeth that were placed in a vacuum chamber and found 28% of the teeth had furcation canals, although only 10% of the total group exhibited canals on the lateral root surface.[33]

In addition to the apical foramen and lateral accessory canals, dentinal tubules have also been suggested as another common pathway between 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.[33,85]

Palatogingival grooves are developmental anomalies of the maxillary incisor teeth, with lateral incisors more often affected than central incisors (4.4% versus 0.28%, respectively).[84] These usually begin in the central fossa, cross the cingulum, and extend apically with varying distances. Generally, the incidence of palatogingival grooves ranges from 1.9% to 8.5%.[26,84] Investigators[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.[63] Deep periodontal pocketing and localized destruction of alveolar bone are often related to long-standing 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.[64]

INFLUENCE OF PULPAL PATHOLOGIC CONDITION ON THE PERIODONTIUM

Pulpal pathosis as a cause of periodontal disease has received much attention during the last decade. Pulpal degeneration results in necrotic debris, bacterial byproducts, 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*

*References 7, 18, 45, 52, 67, and 77.

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.[73] This inflammatory process often results in dysfunction of the periodontal ligament and resorption of alveolar bone, cementum, and even dentin. 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 medicaments (e.g., high concentrations of calcium hydroxide, corticosteroids, antibiotics) used for root canal therapy can irritate the periodontal attachment apparatus.[10,11] The nature and extent of periodontal destruction depend on several factors, including virulence of the irritating stimuli present in the root canal system (e.g., microbiota, medications, foreign body reactions), duration of the disease, and host defense mechanisms.[16] The rare prevalence 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.[66] However, in a monkey study, others reported that all tissue of the periodontium had a 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.[61]

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.[57]

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. Investigators proved that pulpitis and pulp necrosis can occur as a result of periodontal inflammation involving accessory and apical canals.[65] In addition, 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 where periodontal breakdown has extended to the apex.[32] One investigator stated that during periodontal therapy, the blood vessels supplying the pulp by way of accessory canals may be damaged.[85] 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, suggesting 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 influence a degenerating or inflamed pulp can have on 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.[65,69,70] Additional studies have failed to confirm a direct correlation between periodontal disease and pulp tissue changes.[19,56,79] 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 medical history review, pulp vitality testing, pocket and furcation probing, tooth mobility

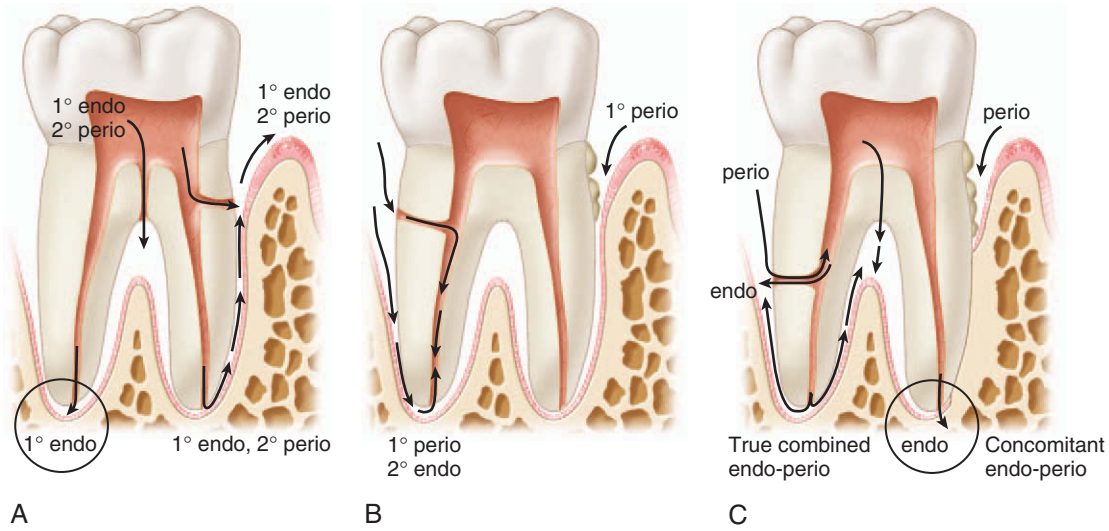


FIG. 18-1 Endodontic and periodontal pathways. **A,** Endodontic lesions. The pathway of inflammation is through the apical foramen, furcation canals, and lateral accessory canals to the periodontium. This results in a primary endodontic lesion, sometimes progressing toward secondary periodontal involvement. **B,** Periodontal lesions. This is the progression of periodontitis by way of lateral canal and apex to induce a secondary endodontic lesion. **C,** True combined endodontic and periodontal lesion and concomitant endodontic and periodontal lesions.

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.[72] Fig. 18-1 lists interrelationships between pulpal and periodontal disease.

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 occurring 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. 18-2) and in furcation areas of two- and three-rooted teeth (Fig. 18-3).

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 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 following nonsurgical endodontic therapy without any periodontal treatment.



FIG. 18-2 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 With Secondary Periodontal Involvement

When a lesion of endodontic origin is not treated, usually pathosis will continue, leading to destruction of the periapical alveolar bone and progression into the interradicular area, causing breakdown of surrounding hard and soft tissues (Fig. 18-4). 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

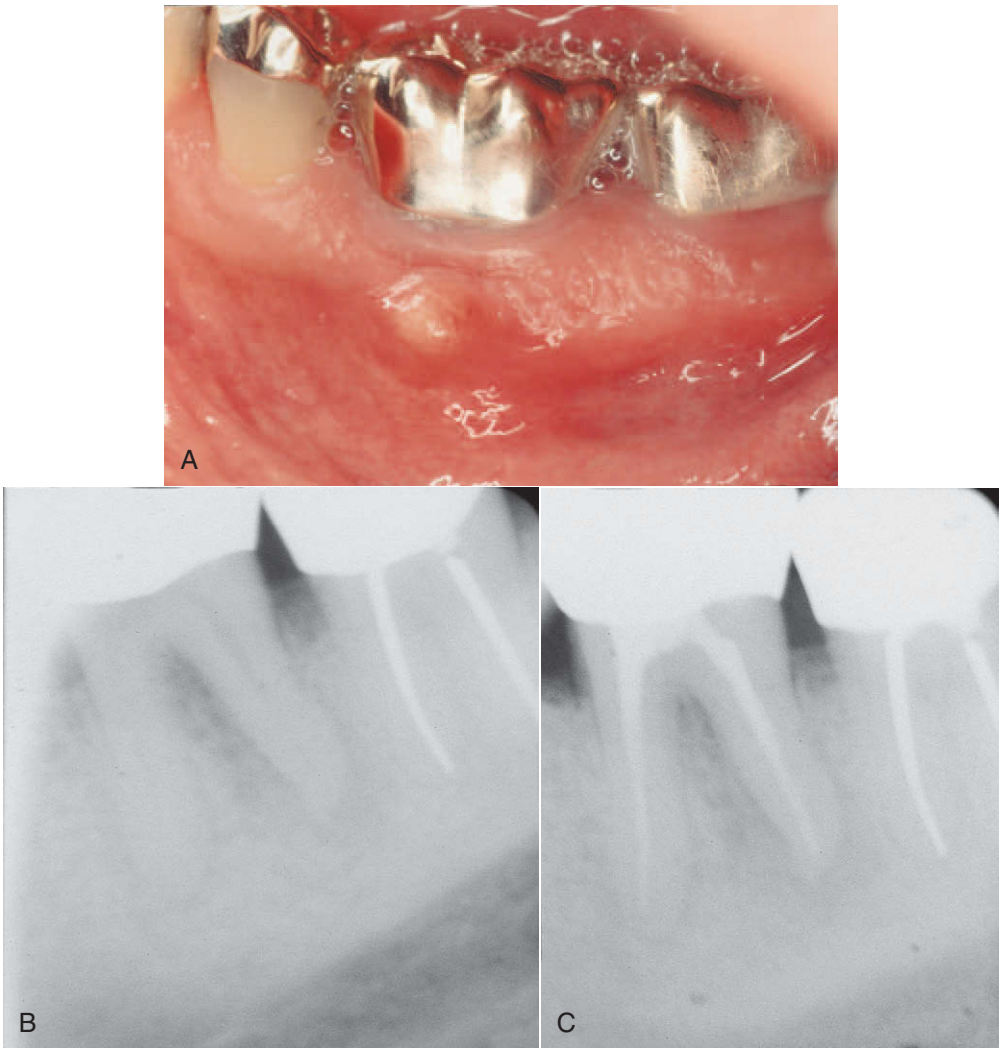


FIG. 18-3 Primary endodontic lesion on mandibular first molar. **A,** Abscess was noted on the buccal surface of the tooth. **B,** Apical radiolucency was evident on the distal root. **C,** Six months after root canal treatment, there is evidence of osseous healing at both the apex and furcation area.

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.

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.[71] 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 will usually reveal 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. 18-5).

The prognosis for those 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.

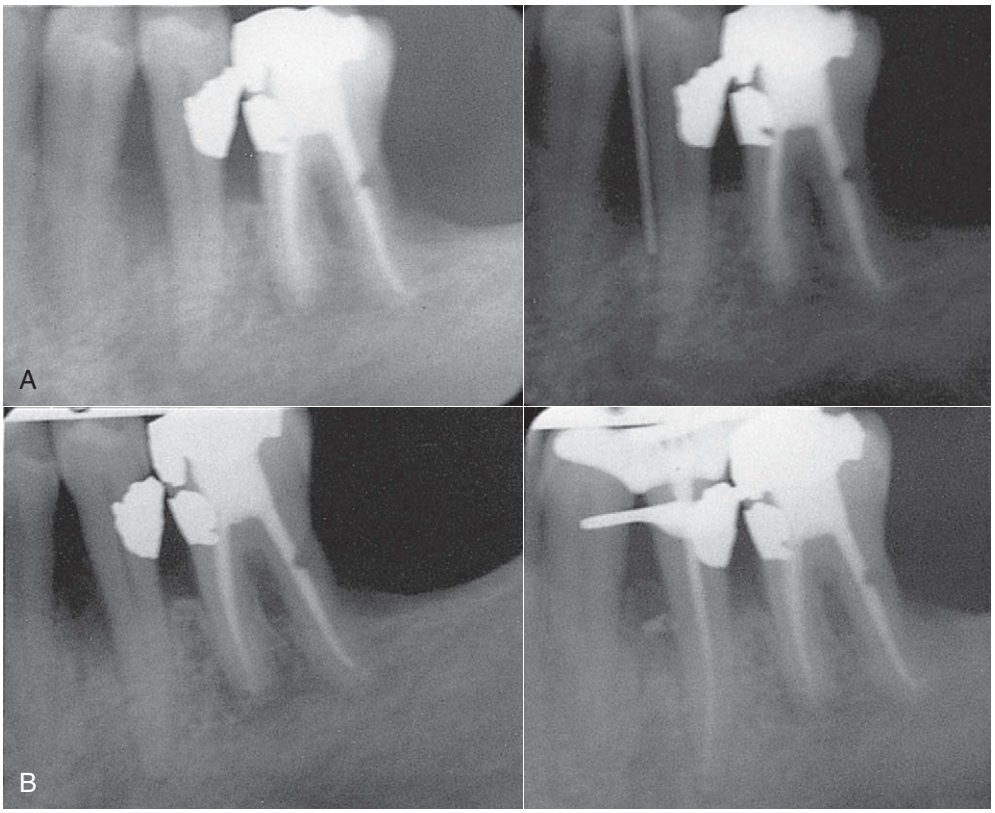


FIG. 18-4 Primary endodontic lesion with secondary periodontal involvement. **A,** Mesial aspect of mandibular second premolar had deep periodontal pocketing (periodontal probe in place demonstrated 6 mm probing depth) even after periodontal therapy. **B,** Pulp tests were performed on the premolar; the tooth was nonresponsive. After endodontic treatment, the periodontal pocket resolved (filling of a lateral accessory canal after obturation is demonstrated).

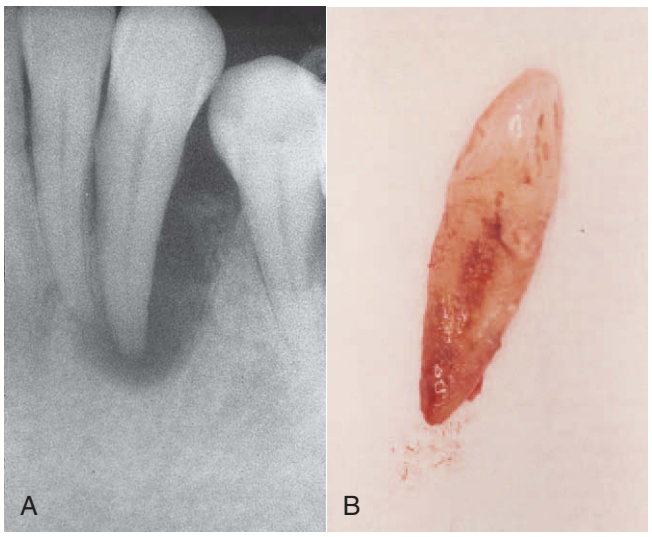


FIG. 18-5 Primary periodontal lesion. **A,** Mandibular cuspid exhibits extensive periodontal destruction; the tooth responded normally to pulp tests. **B,** Extracted tooth depicts extensive calculus accumulation and root concavity.

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. 18-6). The prognosis of multi-rooted teeth with combined pulpal and periodontal



FIG. 18-6 True combined pulpal and periodontal lesions on mandibular second premolar and first molar. Periodontal probing depths were to the apices in both teeth.



FIG. 18-7 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.

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. 18-7). 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 will respond favorably. In actuality, both disease processes must be treated concomitantly, with the prognosis dependent on the removal of the individual etiologic factors and 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, one must resist the temptation to label everything a "combined lesion." Table 18-2 summarizes the differential diagnosis between pulpal and periodontal lesions and highlights a number of common characteristics between these lesions.

Root fractures, especially vertical root fractures, present particular problems in diagnosis (Fig. 18-8). 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.

Sometimes the definitive diagnosis of vertical root fractures has to be confirmed by exploratory surgical exposure of the root for direct visual examination.[82] Vertical root fractures have been associated with root-filled teeth where excessive lateral forces were applied during compaction or possibly with stress induced by a post placement in root-filled teeth.[58] 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] Vertical root fractures that involve the gingival sulcus and periodontal pocket area usually have a hopeless prognosis 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 1 for a 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 18-2

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
HISTOPATHOLOGY		
Junctional epithelium	No apical migration	Apical migration
Granulation tissues	Apical (minimal)	Coronal (larger)
Gingival	Normal	Some recession
THERAPY		
Treatment	Root canal therapy	Periodontal treatment

thought 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.[30,84] It is easy to identify these grooves if one 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. 18-9). These lesions are often confused with the enamel projections in the furcation area of mandibular molars.[55] The prevalence of cervical enamel projections (Fig. 18-10) ranges from 18% to 45%.[36,55] Depending on the apical extent of these cervical grooves, some authors[36,55] 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. 18-11). It may be present as a gingival swelling on the facial aspect that may have 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. Investigators have reported finding unicystic and multicystic (including botryoid) varieties.[3] 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 a lateral periodontal cyst. The histogenesis of lateral periodontal cysts is uncertain, but they favor origin from reduced enamel epithelium. There are cases that 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.*

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

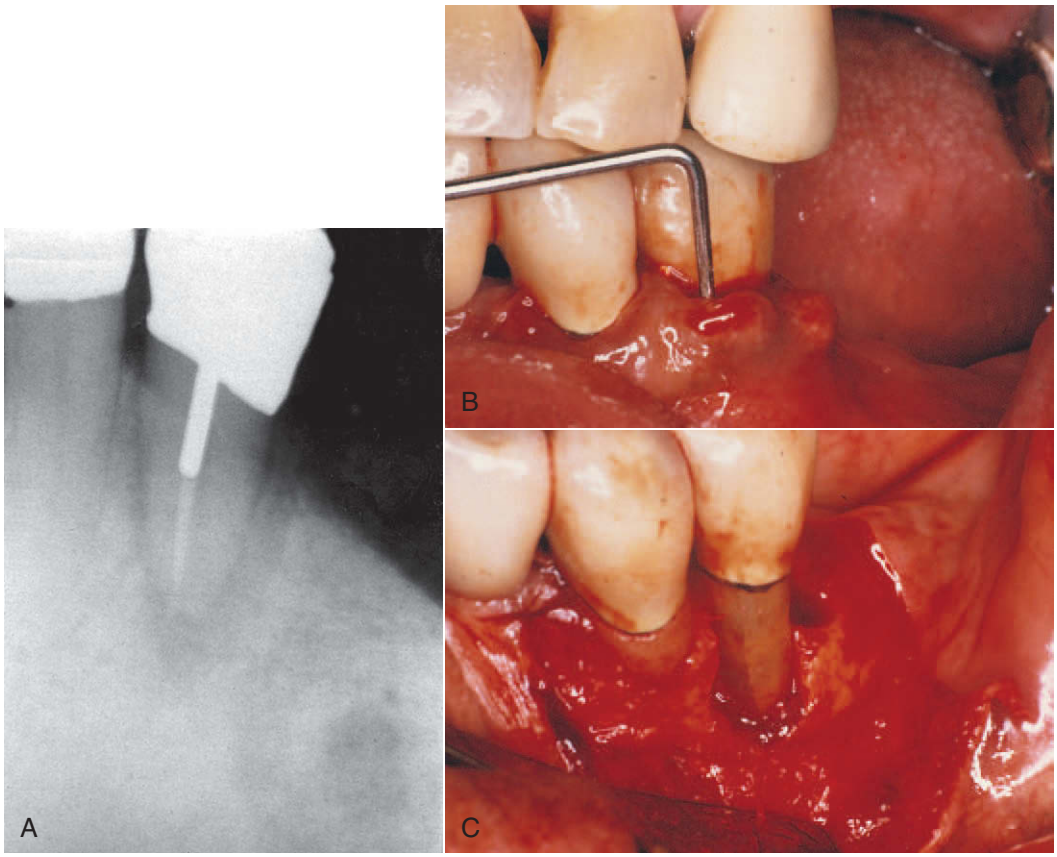


FIG. 18-8 Vertical root fracture. **A,** Radiograph revealed widened periodontal ligament with J-shaped radiolucency around the apex. **B,** Periodontal probe indicated more than 12 mm of probing depth. **C,** Exploratory surgery confirmed vertical root fracture.

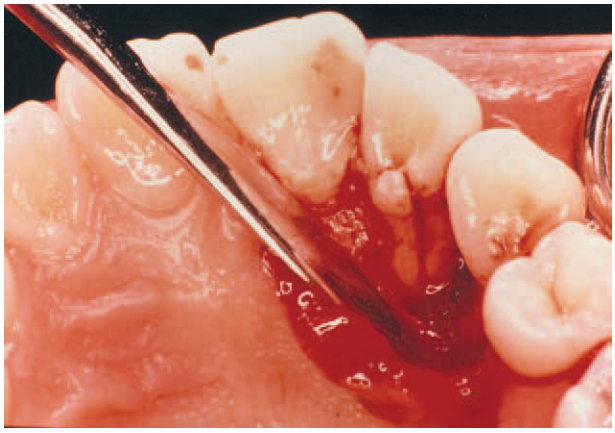


FIG. 18-9 Palatogingival groove on maxillary lateral incisor with periodontal defect.

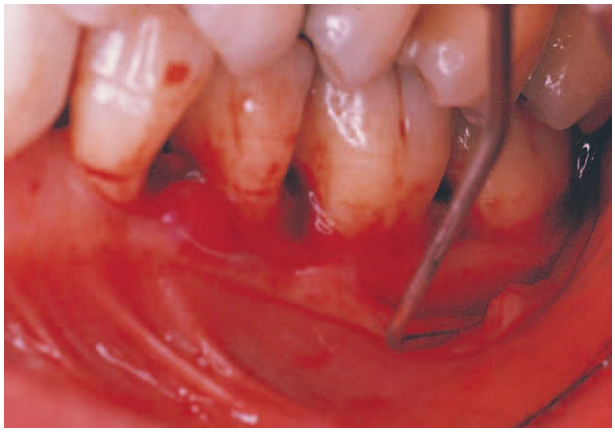


FIG. 18-10 Cervical enamel projection on mandibular molar.

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 III furcation involvement. Root resection is a technique-sensitive procedure (Fig. 18-12) requiring a careful diagnostic process for selection of those teeth that would likely be successful candidates, followed by meticulous interdisciplinary treatment. Factors such as occlusal forces, tooth restorability, and the value of the remaining roots must be examined before treatment. A



FIG. 18-11 Lateral periodontal cyst. **A,** Clinical photograph showing mild gingivitis around tooth #24; 5 mm probing defect on mesial aspect of tooth #24. **B,** Radiograph depicts beginning lesion 2 years earlier. **C,** Radiograph shows extent of lesion at time of referral to periodontist. **D,** Initial flap reflection—lesion present interproximal between teeth #24 and #25.

Continued

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 will depend 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.[83]

Controversy has also existed regarding the benefits and need for endodontic therapy before root resection. Instances develop in which exploratory surgery is necessary; should the

*References 4, 10, 12, 15, 24, 27, and 44.



FIG. 18-11, cont'd E, Lesion was enucleated and submitted for histologic evaluation; diagnosis was 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.

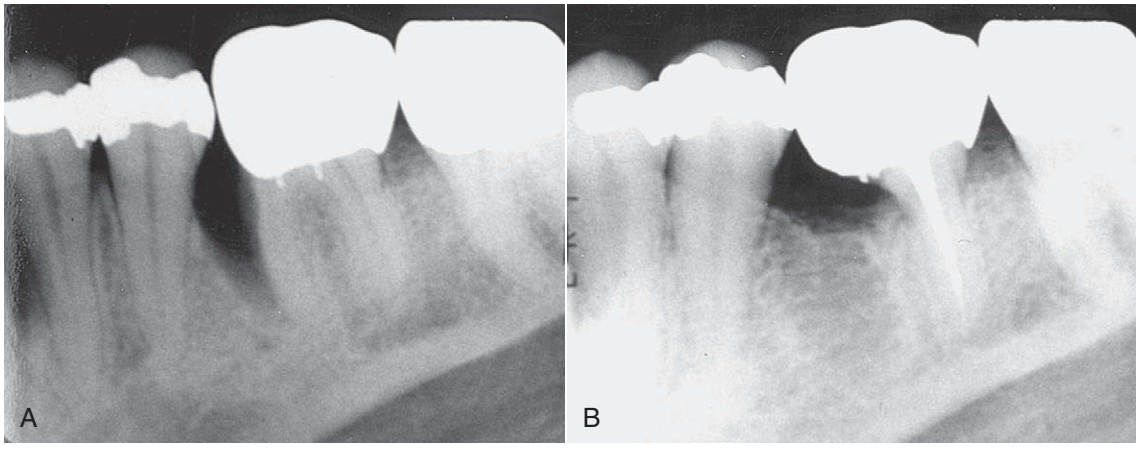


FIG. 18-12 Root resection to correct endodontic and periodontal defect. **A,** Pretreatment radiograph depicts severe periodontal involvement on the mesial. Endodontic treatment was performed before surgical removal of the mesial root. **B,** Five-year follow-up shows the remaining tooth well maintained.

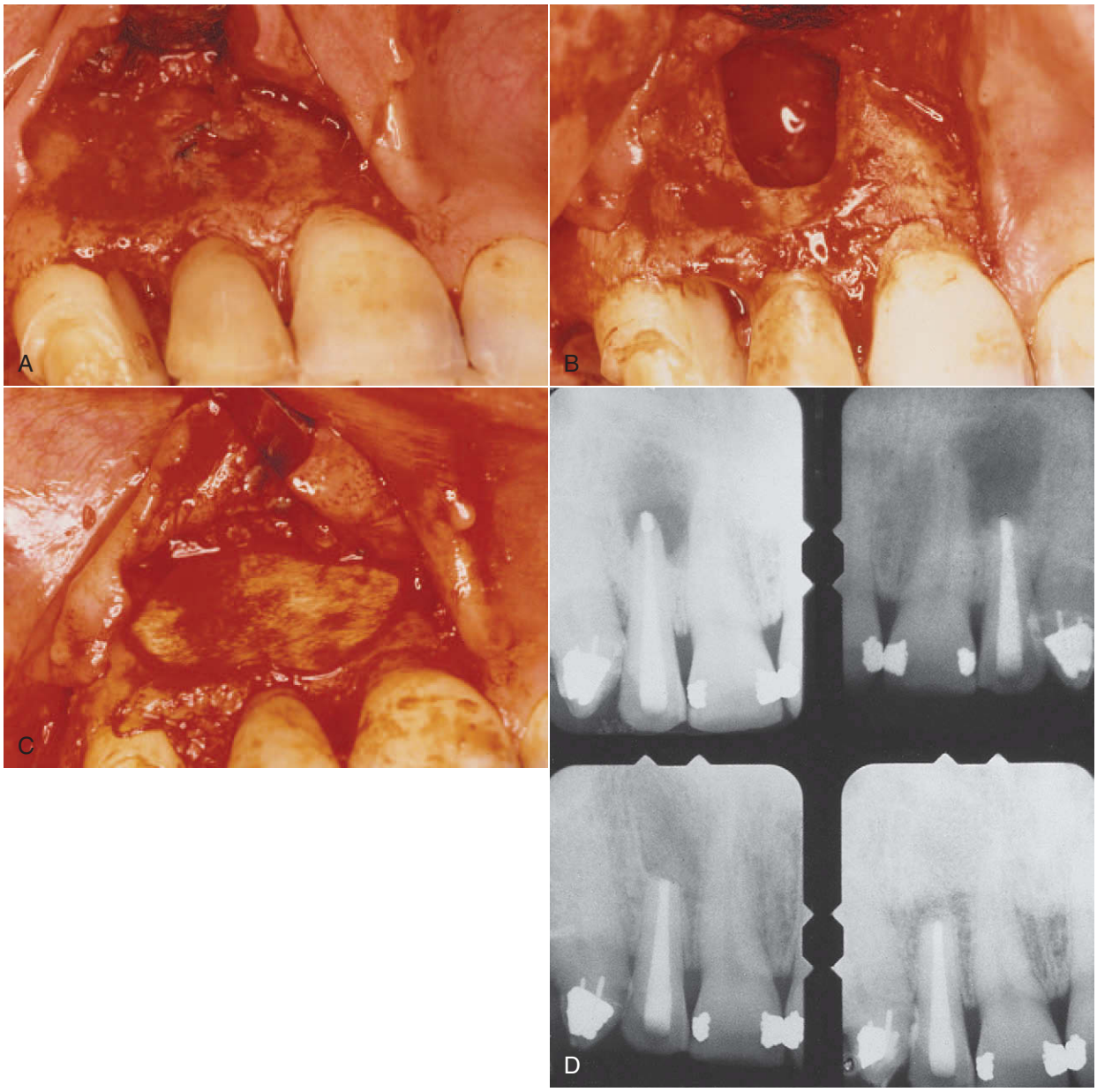


FIG. 18-13 Periradicular endodontic surgery using the combination of bone replacement grafts and collagen GTR (guided tissue regeneration) barrier membranes. **A,** Buccal flap was reflected (note extensive apical lesion). **B,** Osseous window was created with a bur. **C,** The defect was repaired with 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, radiograph reveals complete bone regeneration.

periodontal problem be more extensive than that determined presurgically, the 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.[29,75,78] 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, one investigator reported that vitality of resected teeth can be maintained even after 16 years.[35] 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 such as internal resorption, pulpal inflammation, and necrosis may occur.[2,77]

The concepts of guided tissue regeneration (GTR) or guided bone regeneration (GBR) have been used to promote bone healing after endodontic surgery.[28,60] Fig. 18-13 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.[59,60] 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.[62] 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 also a greater 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.[59]

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.[68] 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 it is exceptionally large in diameter). With the introduction of the GTR concept, a combination of bone replacement graft and GTR membrane (Fig. 18-14) has shown promising results.[1,21,40,62,80] Further studies are required to explore the true benefit of this combination treatment approach in root-end resection procedures.

Forced Eruption or Extruson

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. 18-15). 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 retaining the option for future implant reconstruction if needed. Given the reported success of forced eruption, the technique should be employed 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.[54] 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] ost extruded teeth should be retained in place twice as long as it took to force-erupt them in order 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 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 will 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.

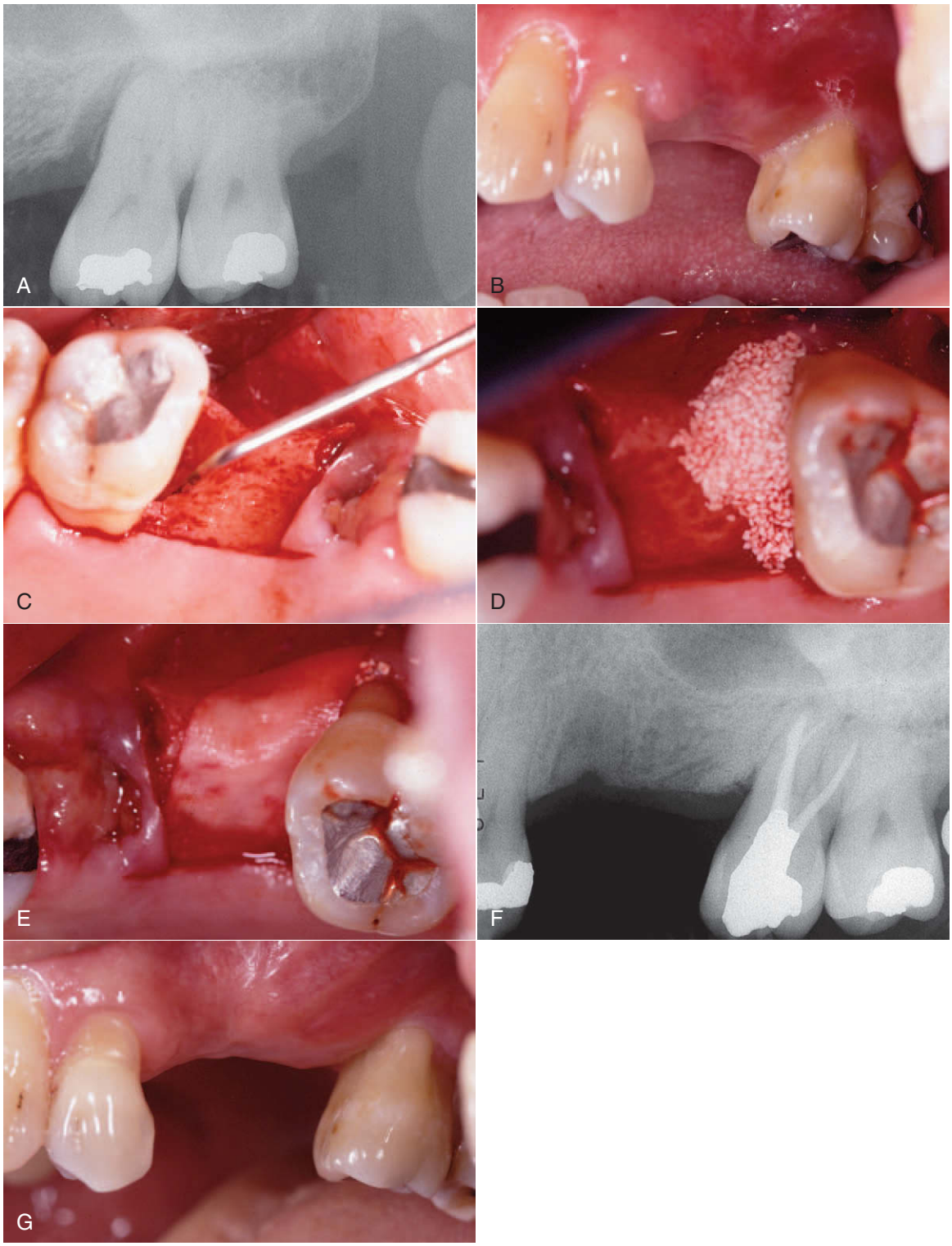


FIG. 18-14 Periodontal surgery using guided bone regeneration. **A,** Pretreatment radiograph demonstrates significant osseous defect on mesial aspect of tooth #14. **B,** Pretreatment clinical photograph of teeth #12 to #16. **C,** Full-thickness mucoperiosteal flap reflected with periodontal probe 7 mm in osseous defect. **D,** Osseous defect filled with BioOss (cancellous bovine bone); patient had root canal treatment started before surgical appointment; RCT was completed 6 weeks after osseous graft placement. **E,** Ossix membrane (collagen) placed over BioOss graft. **F,** One-year posttreatment radiograph with evidence of osseous repair. **G,** One-year posttreatment clinical photograph of area.



FIG. 18-15 Clinical case of forced eruption. **A,** Initial presentation of tooth #29 (buccal view). **B,** Bonded wire to help extrude #29. **C,** Lingual view of #29 after 3 weeks of extrusion. **D,** Lingual view of cemented porcelain crown on tooth #29; 4 months following extrusion and after 1 month of crown retention.

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CHAPTER 19

Endodontic Pharmacology

KARL KEISER and B. ELLEN BYRNE

CHAPTER OUTLINE

THE TRIGEMINAL PAIN SYSTEM
- Detection: The First Step in Pain Perception
- Processing: The Second Step in Pain Perception
- Perception: Thalamus to Cortex

PREDICTORS OF POSTOPERATIVE ENDODONTIC PAIN

NONNARCOTIC 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

Even in the 21st century, the terms *root canal* and *pain* often are considered synonymous. Every clinician who provides endodontic therapy has had to deal with this misperception, and the skill of the clinician is often judged primarily by their success or failure of pain control. Achieving successful endodontic pain control in a predictable and efficient manner requires a working knowledge of the underlying biology of the trigeminal pain system and the mechanisms by which available drugs and therapies provide relief. This chapter presents insights into basic science discoveries regarding acute pain and reviews the best evidence available for successful treatment of pain of pulpal and periradicular origins. It will also include a brief overview of the field of pain pharmacogenomics, which promises the possibility of predicting the effects of an analgesic drug depending on the genetic makeup of the patient. A truly inclusive review of the topic of pain control in endodontics would also include the diagnosis of odontogenic pain (see Chapters 1 and 2), the diagnosis of nonodontogenic pain (see Chapter 3), a discussion of the mediators of pulpal and periradicular inflammation (see Chapters 12 to 14), a review of the pharmacology of local anesthetics (see Chapter 20), and behavioral modification (see Chapter 26).

THE TRIGEMINAL PAIN SYSTEM

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. 19-1). 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 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.[22,23,113,227] It should be noted that this classification system is based purely upon 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[2,127,191,192] and may also influence the activity of peripheral nociceptors (for reviews, see Perl[189] or Hargreaves[91]). Because most pulpal sensory fibers are nociceptive, their terminal branches are free nerve endings, and physiologic

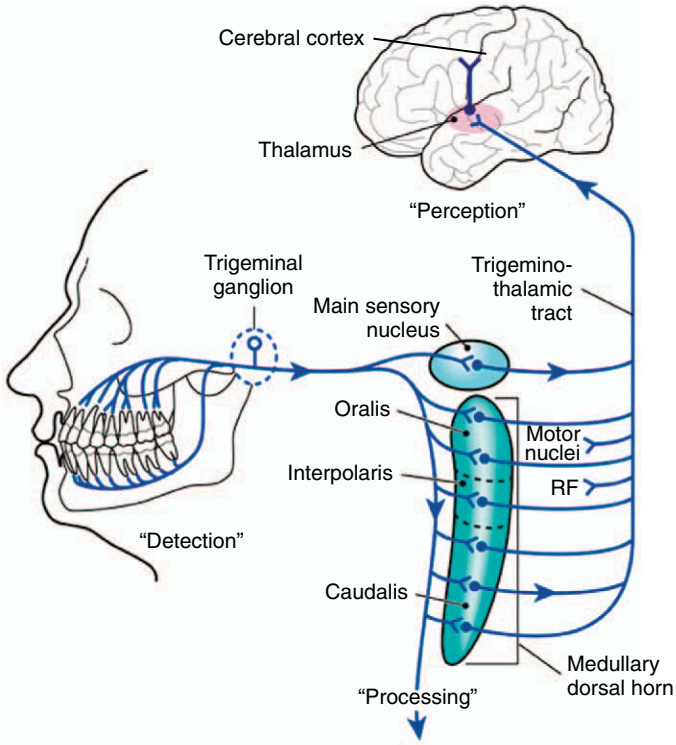


FIG. 19-1 Schematic diagram of the pathway of transmission of nociceptive information from the orofacial region. The trigeminal pain system is a complex multilevel system that begins with the DETECTION of tissue damaging stimuli in the periphery, the PROCESSING of that input at the level of the medullary 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.

TABLE 19-1

Effect of Inflammatory Mediators on Nociceptive Afferent Fibers

Mediator	Effect on Nociceptors	Effect on Human Volunteers
Potassium[115]	Activate	++
Protons[143,212]	Activate	++
Serotonin[15,115]	Activate	++
Bradykinin[15,115,137]	Activate	+++
Histamine[115]	Activate	+
Tumor necrosis factor-α	Activate	?
Prostaglandins[16]	Sensitize	±
Leukotrienes[16,145]	Sensitize	±
Nerve growth factor[139,190]	Sensitize	++
Substance P[86]	Sensitize	±
Interleukin 1[62]	Sensitize (?)	?

Modified from Fields H: *Pain,* New York, 1987, McGraw-Hill.

+, Positive; ++, very positive; +++, extremely positive; ±, equivalent; ?, unknown.

stimulation by any modality (temperature, hyperosmotic fluids) results in the perception of pure pain which can be very 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). Of the nine known voltage-gated sodium channel subtypes ($Na_v1.1$ through $Na_v1.9$), five are believed to exist in the trigeminal system. As will be discussed in Chapter 21, they likely play an important role in the altered sensitivity of pulpal and periradicular nociceptors during inflammation, where even non-noxious stimuli can result in the perception of pain (for review see Henry and Hargreaves[101]).

After generation of an action potential, not only is information sent to the CNS, but in an *antidromic* fashion (i.e., in the reverse direction of the impulse), 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 inflamed pulp and periradicular tissues.

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 19-1). Other mechanisms of peripheral sensitization are listed in Box 19-1.

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

BOX 19-1

Peripheral Mechanisms Contributing to Hyperalgesia and Allodynia

Mechanism

- Composition and concentration of inflammatory mediators[90]
- Changes in afferent fiber: activation and sensitization[131,153,201]
- Changes in afferent fiber: sprouting[21]
- Changes in afferent fiber: proteins[21,78,243]
- Tissue pressure[172,173]
- Tissue temperature[159]
- Sympathetic primary afferent fiber interactions[110,136,188]
- Aβ fiber plasticity[176]

(Modified from Hargreaves KM, Swift JQ, Roszkowski MT, et al: Pharmacology of peripheral neuropeptide and inflammatory mediator release. *Oral Surg Oral Med Oral Pathol* 78:503–510, 1994.)

pulp),[170] 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.[50,80] 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 PG receptors EP2 and EP3 in the trigeminal system[184] and exert their effects by regulating the activity of certain ion channels,[234] including voltage-gated sodium channels (for review see England[56]). 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.[8,79] 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 $Na_v1.8$.[84,85] 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 and, in addition, more profound local anesthesia may be achieved in patients with hyperalgesia of pulpal origin.[95,106,160] 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 later in this chapter).[148,239]

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,[135] 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,[218] as well as other receptors such as TRPV1 and TRPA1.[41,112] 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[41] and interacts with TRPV1.[200] Bradykinin likely increases the excitability of nociceptive neurons through its action on TRPV1 and TRPA1 (for review, see Tominaga et al.[217]).

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.[130] Application of TNF-α rapidly sensitizes TRPV1,[124] 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[64]) and are thought to act at least in part by causing increased release of prostanoids.[214]

Changes in Neuronal Phenotypes

Sensory neurons innervating inflamed peripheral tissues undergo striking phenotypic changes, including changes in transduction mechanisms and plasticity of nociceptor organization. In the dental pulp, it is likely that C fibers predominate in the response to inflammation, because the quality of the pain described by patients with pulpal inflammatory disease (e.g., dull, aching, throbbing sensations) is similar to that reported by patients experiencing experimental C fiber activation.[5,169,174] As described previously, certain combinations of inflammatory mediators causes both activation and sensitization of these fibers. Interestingly, when pulpal and periradicular tissues undergo inflammatory changes, C fibers innervating those tissues are found to contain increased amounts of proinflammatory neuropeptides such as SP and CGRP.[19,24,26] It has also been shown that C fiber terminals in inflamed pulpal tissues undergo a process called *sprouting*, whereby those terminals closest to the inflamed tissues actually grow to form additional terminal endings,[22-24] possibly leading to increased participation in detecting and modulating tissue responses to injury.

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.[175] 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 from patients with a diagnosis of irreversible pulpitis.[235,237] When exposed to PGE_2, neurons isolated from dorsal root ganglia cells have been shown to increase TTX-resistant sodium channel currents within minutes,[79] indicating an increased activation of existing channels, rather than de novo protein synthesis. Interestingly, these sodium channels are relatively resistant to lidocaine,[199] and this may explain the difficulty in achieving profound anesthesia in inflamed tissues (see also Chapter 20).[95]

Signs and Symptoms of Pulpalgia and Acute Periradicular Periodontitis

Again, the hallmarks of pain perception due to acute inflammation are allodynia, hyperalgesia, and spontaneous pain. The peripheral mechanisms for these symptoms include a decrease

TABLE 19-2

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 (EPT 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* 78:503–510, 1994.

EPT, Electric pulp test.

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 19-2). From this perspective, the histology of the pulp would not be expected to correlate with the patient's symptoms. Simply put, inflammatory pain is the result of molecules interacting with nociceptors, an event that cannot be detected by conventional histologic methods.

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.

Analgesic Drugs With the Potential to Act in the Periphery

Pain control can be effected by endogenous mechanisms in the periphery. For example, immune cells in inflamed peripheral tissues may release opioids that act on receptors found on sensory neurons.[213] Several drug classes (discussed later in this chapter) also have the potential for reducing the production of agents that serve to cause pain directly or sensitize peripheral nociceptors. These drugs include NSAIDs,[104,105] corticosteroids,[69] and opiates.[47]

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.[94,141,204] Three distinct subnuclei can be found in this complex. Named for their anatomic position, they are the *subnuclei oralis, interpolaris,* and *caudalis* (see Fig. 19-1). Although the more rostral subnuclei (oralis and interpolaris) receive some nociceptive input from oral tissues,[39] most such input is received at the level of the subnucleus caudalis.[57,146,206] 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[152] cells (for review see Hargreaves[91]).

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 NMDA and 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.[29] In a human clinical trial using an oral surgery model, the AMPA/kainate antagonist, LY293558, was shown to be antihyperalgesic.[75] NK1 antagonists have shown promising results in animal studies, but in general, they have displayed limited analgesic efficacy in humans.[103]

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. 19-2). 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.[206] 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.[208] Subnuclei oralis and interpolaris also receive converging input from orofacial and muscle afferents.[207] 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 3).

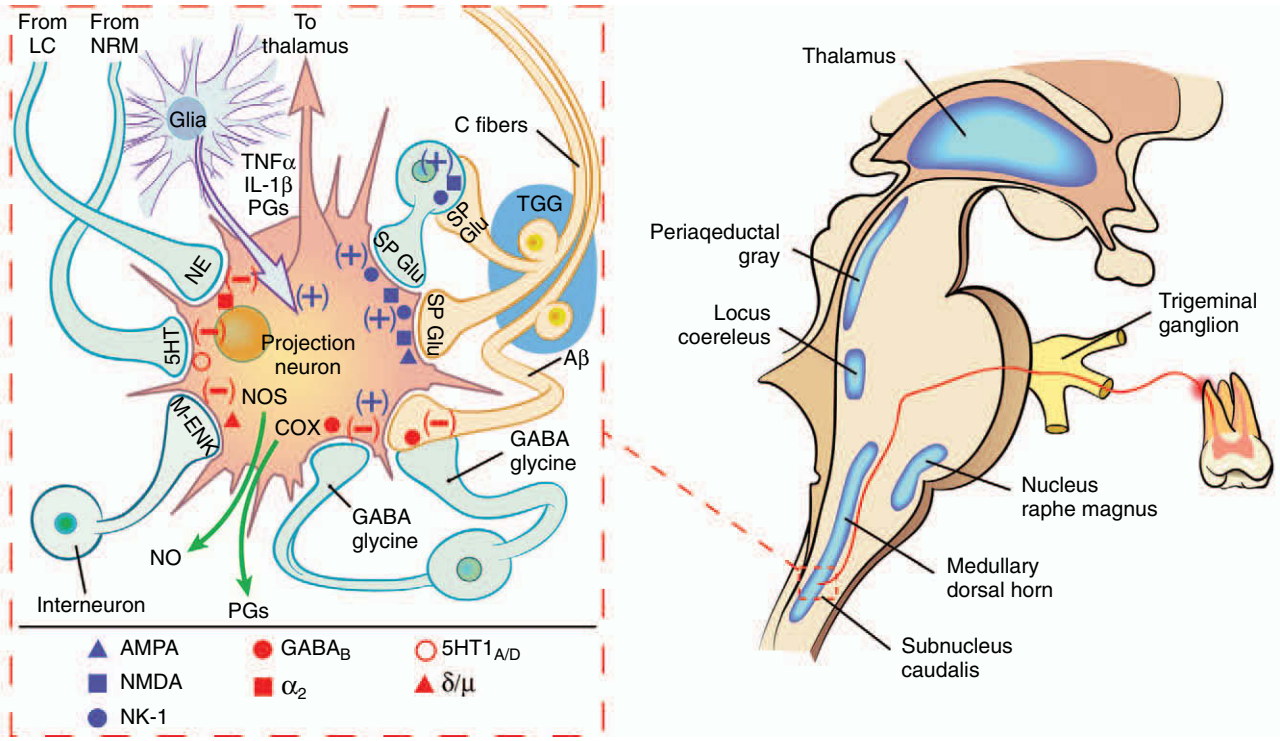


FIG. 19-2 Schematic diagram of the perception and modulation of orofacial pain. Activation of primary afferent fibers (in this example from an inflamed maxillary molar) leads to the entry of a nociceptive signal that is conveyed across a synapse in the subnucleus caudalis of the trigeminal spinal nucleus. 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 metenkephalin (M-ENK) also inhibit transmission of nociceptive information. Projection neurons may have autocrine and/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.

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 be greatly diminished.[180] Likewise, if the source of a perceived toothache were located in a muscle of mastication, palpation of that muscle should aggravate the pain.[247]

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 and/or substance P, whereas inhibitory interneurons release the amino acid, glycine, and/or gamma amino butyric acid (GABA).[141,205]

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.[13] These terminals release a variety of neuroeffective agents, including the endogenous opioid peptides (EOP). 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.[92,138]

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.[193,236] 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,[216] 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.[245] Recent 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).[182] This appears to be due at least in part to central sensitization, since both the ipsilateral (pulpitis) tooth and a contralateral (normal) tooth demonstrated mechanical allodynia to a force transducer.[125] Thus, central sensitization contributes to endodontic pain, and the clinical application of bite force transducers may provide a novel method for diagnosing pain mechanisms.[125,126]

Different studies shed light on the molecular mechanisms involved in central sensitization (for review, see Cousins and Power[36] and Hargreaves[91]), 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 has been cited in several studies as a predictor of postoperative endodontic pain,[108,219,233] 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 shown to provide pain relief far beyond the duration of the peripheral tissue anesthesia.[83,111]

A reduction in the chemical mediators of inflammation at the level of the medullary dorsal horn also should serve to reduce sensitization of second-order neurons. Decreasing the synthesis of proinflammatory PGs, cytokines, nitric oxide, and/or the use of drugs that block the receptors of such agents probably will 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.[29] A similar investigation implicated nitric oxide synthesis at the level of the subnucleus caudalis in the development of tactile hypersensitivity following dental injury.[252] Reduction in nitric oxide synthase levels may also provide protection from central sensitization.[149,157]

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. 19-1). 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.[186]

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 review, see Yaksh[250]). 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[52,241] but also the tendency of the patient to recall the experience as painful or unpleasant even 18 months posttreatment.[72] The clinician should do everything possible to control a patient's anxiety level prior to endodontic treatment (see also Chapter 26). 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.[228] 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[44] but 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.[116] 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.[142] It has been shown that furanocoumarins in grapefruit juice inhibit cytochrome P450 3A4,[183] 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.

PREDICTORS OF POSTOPERATIVE ENDODONTIC PAIN

Although current endodontic treatment can be virtually pain free during the procedure itself, certain patients may still experience some pain after the appointment. Early studies investigating postoperative endodontic pain have reported an incidence of moderate to severe pain in the range of 15% to 25%.[30,97,179] In a prospective clinical study, 57% of patients reported no pain after débridement and shaping of the root canal system, although 21% had slight pain, 15% had moderate pain, and 7% had severe pain.[74] Although some patients may experience moderate to severe pain after endodontic treatment, very few experience what is now commonly referred to as a *flare-up* or a postoperative problem requiring an unscheduled visit with unplanned treatment intervention to manage the patient's symptoms.[231] Patients with a flare-up usually describe severe pain, swelling, or the sensation of pressure within their mandible or maxilla within 1 or 2 days of treatment. The incidence of flare-up varies across studies and ranges from about 2% to 20% of patients, with the higher prevalence generally reported in older studies using classical cleaning and shaping techniques.[12,165,224,225]

Numerous studies have evaluated factors related to postoperative endodontic pain and flare-up to better predict when these conditions are more likely to occur. Table 19-3 summarizes the results of clinical endodontic studies evaluating more than 12,000 patients for predictors of postoperative pain.

TABLE 19-3

Factors Related to Postendodontic Pain

Author and Year	N	Preop Hyperalgesia	Preop Swelling	Necrotic Pulp	Apical Periodontitis	Re-Tx	Tooth Type	Sex	Age	Hx of Allergy	Intracanal Medicament	Apical Patency	One-Step versus Multi-Appt
Seltzer et al., 1961[203]	698	+							+		=		
Frank et al., 1968[67]	585				AP < Pain						=		
Fox et al., 1970[66]	291	+		=	AP < Pain		=	+	=				
Clem, 1970[30]	318			+			+	=				+	
O'Keefe, 1976[179]	147	+		=	=		+	=	+				=
Maddox et al., 1977[144]	252			=				=	=		=		
Soltantoff, 1978[210]	281												Multi is <
Harrison et al., 1979[99]	195				=		+						
Rowe et al., 1980[198]	150	+	+	=	=								
Harrison et al., 1981[96]	245			=	=		=				=		
Pekruhn, 1981[185]	102												=
Mulhern et al., 1982[168]	60	+			=		=	+	=				=
Oliet, 1983[181]	387			=			=	=	+				=
Harrison et al., 1983[97]	229			=			=						
Harrison et al., 1983[98]	229				AP > Pain						+		
Roane et al., 1983[194]	359			=			=						1-Step is <
Balaban et al., 1984[11]	157						+		+				
Creech et al., 1984[37]	49	+											
Jostes & Holland, 1984[114]	58	+											
Marshall & Walton, 1984[151]	50	+		=	=		=	=	=				=
Georgopoulou et al., 1986[74]	245			=	=		=	=	=				
Morse et al., 1987[165]	106				AP > Pain		+	+	+				

Continued

TABLE 19-3

Factors Related to Postendodontic Pain—cont'd

Author and Year	N	Preop Hyperalgesia	Preop Swelling	Necrotic Pulp	Apical Periodontitis	Re-Tx	Tooth Type	Sex	Age	Hx of Allergy	Intracanal Medicament	Apical Patency	One-Step versus Multi-Appt
Flath et al., 1987[61]	116	+		=	=							=	
Genet et al., 1987[73]	443	+		+	AP > Pain		+	+	=				
Torabinejad et al., 1988[223]	2000	+			AP < Pain	+	+	+	+	+	=		
Fava, 1989[57]	60											=	=
Trope, 1990[225]	474				AP > Pain	+					=		
Trope, 1991[224]	226				AP > Pain	+							
Mor, 1992[163]	334			+	AP < Pain		=	=	=				
Walton & Fouad, 1992[234]	946	+	+	+	AP > Pain	=							=
Marshall & Liesinger, 1993[152]	106			=	AP < Pain		=	=	=				
Torabinejad et al., 1994[222]	588	+											
Abbott 1994[4]	100										+		
Imura & Suolo, 1995[107]	1012	+		=	AP > Pain	+	=	=	=				1-Step is <
Eleazer & Eleazer, 1998[55]	402												1-Step is <
Albashaireh, 1998[6]	300												1-Step is <
Glennon et al., 2004[77]	272	+	+				+						
Total	12,572												

Note: A "+" sign indicates that a positive correlation was found between this factor and the presence of pain. AN "=" sign indicates that no correlation was found between this factor and the presence of pain. The absence of either sign indicates that the study did not address this factor. "AP > Pain" indicates that the presence of apical periodontitis was associated with more postoperative pain.

Hx, History; *Tx,* treatment.

Differences in experimental design prevent direct comparison of studies, but interestingly, the presence of *preoperative* pain or mechanical allodynia (reduced mechanical pain threshold or percussion sensitivity) was a positive predictor of postoperative pain in more than 15 studies involving more than 6600 patients.

Other factors were more variable in their predictive value of postoperative pain. In a retrospective study, the dental records of 1000 patients who had received nonsurgical root canal treatment and experienced no flare-ups (i.e., unscheduled return visits) were compared with the records of 1000 patients who experienced flare-ups after the cleansing and shaping of their necrotic root canals.[220] The results showed that factors such as presence of preoperative pain, tooth type, sex, age, history of allergy, and retreatment, were significantly predictive for the incidence of flare-up, although intracanal medicaments, systemic disease, and establishment of patency of the apical foramen had no significant relation to the incidence of flare-ups.[220] Specifically, the highest incidences of flare-ups were associated with mandibular teeth, retreatment procedures, females over the age of 40, and patients with a history of allergies.[220]

In another retrospective study, the flare-up rate was evaluated in patients treated in multiple visits by undergraduate dental students.[163] The incidence in this study was 4.2%, and a positive correlation was found between flare-ups and teeth with necrotic pulp. No correlation was found between the occurrence of a flare-up and the presence or absence of a periradicular radiolucency.

In a prospective study of one-visit nonsurgical root canal treatments, the overall flare-up rate was only 1.8%.[224] However, this same study determined that one-visit endodontic retreatment cases involving teeth with apical periodontitis had almost a 10-fold higher incidence of flare-ups (13.6%), with the recommendation that retreatment of teeth with apical periodontitis should not be completed in one visit. Another prospective clinical study reported an overall flare-up rate of 3.2% of 946 endodontic cases.[233] Patients with severe preoperative pain had a flare-up rate of 19%, although the presence of localized or diffuse swelling related to an incidence of 15%. In this study, pulpal status predicted the flare-up rate, with necrotic teeth having a significantly greater incidence of flare-ups compared with vital teeth (6.5% versus 1.3%). Periradicular status also predicted flare-up rates, with differences noted between chronic apical periodontitis (3.4%), acute apical periodontitis (4.8%), and acute apical abscess (13.1%). No significant difference was seen between single and multiple visits. Finally, no significant increase in flare-ups was seen for teeth undergoing retreatment of a failed root canal, although a subset analysis of retreatment with or without apical periodontitis was not performed.

A recent systematic review of clinical trials evaluating the effectiveness of single versus multiple visits for endodontic therapy concluded that while there was no difference in ultimate outcome of the treatment, those patients treated in one visit were more likely to take analgesic drugs.[132]

In summary, the most consistent factor that predicts postendodontic pain is the presence of preoperative pain or mechanical allodynia. Although no single factor completely predicts the occurrence and magnitude of postoperative pain, an astute clinician should interpret the presence of preoperative pain or mechanical allodynia as a warning sign in selecting the pain control regimen for each patient.

NONNARCOTIC ANALGESICS

Management of endodontic pain is multifactorial and directed at reducing the peripheral and central components of hyperalgesia (see Box 19-1) through combined endodontic procedures and pharmacotherapy. A major class of drugs for managing endodontic pain is nonnarcotic 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.[20,48,89,93] Although these drugs classically are thought to produce analgesia through peripheral mechanisms, the CNS now is believed to be an additional site of action.[147,215] Some researchers have proposed that a splice variant of COX1—COX3—is expressed predominantly in the CNS and is the major site of action of acetaminophen.[28,128,202] However, recent studies indicate that the antipyretic and analgesic effects of acetaminophen do not involve inhibition of COX3[102] but are more likely exerted through effects of an active metabolite on CNS cannabinoid receptors.[7]

Numerous NSAIDs are available for management of pain and inflammation (Table 19-4). Unfortunately, comparatively few studies (particularly for endodontic pain) directly compare one NSAID to one another for analgesia and side-effect

TABLE 19-4

Summary of Selected Nonnarcotic Analgesics

Analgesic	Trade Name	Dose Range (mg)	Daily Dose (mg)
Acetaminophen	Tylenol et al.	325-1000	4000
Aspirin	Many	325-1000	4000
Diflunisal	Dolobid	250-1000	1500
Diclofenac potassium	Cataflam	50-100	150-200
Etodolac	Lodine	200-400	1200
Fenoprofen	Nalfon	200	1200
Flurbiprofen	Ansaid	50-100	200-300
Ibuprofen	Motrin et al.	200-800	2400 (Rx)
Ketorolac*	Toradol	30-60 (oral)	60
Naproxen Na	Anaprox et al.	220-550	1650 (Rx)
Naproxen	Naprosyn	250-500	1500
Ketoprofen	Orudis	25-75	300 (Rx)

Note: COX-2 inhibitors are not included (see text).

Modified from Cooper SA: Treating acute dental pain, *Postgrad Dent* 2:7, 1995.

Rx, Prescription strength.

*A new package insert for ketorolac tablets contains the instructions that the drug should be used only as a transition from injectable ketorolac and for no more than 5 days.

liability, particularly regarding endodontic pain. The lack of comprehensive comparative studies in endodontic models means that only general recommendations can be made, and clinicians are encouraged to be familiar with several of these drugs. Ibuprofen generally is considered the prototype of contemporary NSAIDs and has a well-documented efficacy and safety profile.[45] Other NSAIDs may offer certain advantages over ibuprofen. For example, etodolac (i.e., Lodine) has minimal gastrointestinal (GI) irritation,[9] and ketoprofen (i.e., Orudis) has been shown in some studies to be somewhat more analgesic than ibuprofen.[34] The advantages of NSAIDs include their well-established analgesic efficacy for inflammatory pain. Many of the NSAIDs listed in Table 19-4 have been shown to be more effective than traditional acetaminophen and opioid combinations such as Tylenol with codeine no. 3.[33,45,226]

A 2002 paper represents the first systematic review comparing all endodontic pain studies evaluating oral NSAIDs.[105] 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[49]) 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 19-5 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 developing a postendodontic pain treatment plan.

The introduction of selective inhibitors of COX-2 offered the potential for both analgesic and antiinflammatory benefits and reduced GI irritation.[43,123] Oral surgery pain studies evaluating COX-2 inhibitors have indicated that rofecoxib (i.e., Vioxx) has significant analgesic efficacy in this model.[53] 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.[53] Moreover, COX-2 levels are increased in inflamed human dental pulp,[171] and a COX-2 inhibitor (rofecoxib) is analgesic in patients with endodontic pain. Concern has been raised that the COX-2 inhibitors may also display at least some GI irritation in patients with preexisting GI disease.[230]

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.[17] The debate continued until the demonstration of an increased risk for prothrombic events following long-term administration of rofecoxib, which led to the withdrawal of this drug from the market in 2004.[59] Two meta-analyses have examined the CV safety of traditional NSAIDs and COX-2 inhibitors. Kearney et al. conducted a meta-analysis of 138 randomized trials, and McGettigan and Henry conducted a meta-analysis of 23 controlled observational studies.[118,155] Kearney et al. 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 similar degree of COX-2 selectivity as 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 for 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. 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 U.S. Food and Drug Administration (FDA) has requested changes to the label to better inform consumers regarding the safe use of these products. Given this situation and reasonable alternative NSAIDs, we recommend not considering COX-2 inhibitors for treating routine endodontic pain patients.

Limitations and Drug Interactions

Clinicians should educate themselves not only on the efficacy of the nonnarcotic analgesics but also on their limitations and interactions with other drugs.[25] 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.[31-33,68,248] They also are associated with severe GI complications, and the risk of adverse effects increases with increasing lifetime-accumulated dose of these drugs.[46,240]

The NSAIDs have been reported to interact with a number of other drugs (Table 19-6). Acetaminophen and opioid combination drugs represent alternatives for those patients unable to take NSAIDs.[32] Further information is available on the pharmacology and adverse effects of this important class of drugs.[25,31,46,68,248] 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.

Acetaminophen

Acetaminophen is one of the most commonly used drugs. It is also 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 has become the most common cause of acute liver failure.[134] 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),

TABLE 19-5

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
Acetaminophen (1000 mg) + codeine (60 mg)	197	57	2.2	1.7	2.9
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
Oxycodone IR (10 mg) + acetaminophen (1000 mg) 83		67	2.7	1.7	5.6
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 (1500 mg)	138	65	3.7	2.3	9.5
Acetaminophen (1000 mg)	2759	46	3.8	3.4	4.4
Oxycodone IR (5 mg) + acetaminophen (1000 mg)	78	55	3.8	2.1	20
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.jr2.ox.ac.uk/bandolier/booth/painpag 1/09.

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.

TABLE 19-6

Summary of Selected NSAID Drug Interactions

Drug	Possible Effect
Anticoagulants	Increase in prothrombin time or bleeding with anticoagulants (e.g., coumarins)
ACE inhibitors	Reduced antihypertensive effectiveness of captopril (especially indomethacin)
Beta-blockers	Reduced antihypertensive effects of beta-blockers (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)
Penillamine	Increased bioavailability (especially indomethacin)
Sympathomimetics	Increased blood pressure (especially indomethacin with phenylpropanolamine)
Thiazide diuretics	Reduced antihypertensive effectiveness

Data from 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; Wynn R, Meiller T, Crossley H: *Drug information handbook for dentistry*, Hudson, Ohio, 2000, Lexi-Comp.

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. Healthy adults should not take more than 4 g (4000 mg) of acetaminophen in a 24-hour period. However, the FDA convened an Advisory Panel meeting in June 2009, with discussions on limiting the maximal daily dose of acetaminophen and possibly removing certain medications from the market.[58] Although no action was taken by late 2009, the clinician is encouraged to continue to monitor the FDA website for further information (www.FDA.gov). Opioid pain medications may contain up to 650 to 750 mg of acetaminophen per tablet. Care must be taken when prescribing such combinations to keep the maximum acetaminophen dose below 4000 mg. In an effort to address this, manufacturers have made high-dose opioid/low-dose acetaminophen combinations, such as hydrocodone 10 mg and acetaminophen 325 mg.

TABLE 19-7

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.

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 located at several important sites in the brain (see Fig. 19-2). Activation of this receptor inhibits the transmission of nociceptive signals from the trigeminal nucleus to higher brain regions, and recent studies indicate that opioids also activate peripheral opioid receptors located in dental pulp.[60] Intraligamentary injection of morphine has been shown to significantly reduce pain in endodontic patients and other inflammatory pain states.[47,88] Gender-dependent differences appear to exist in responsiveness to at least the kappa opioid agonists; women have a significantly greater analgesic response to pentazocine compared to men.[71]

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 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 19-7).

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.[32,33,89] In general, patients taking only 30 mg of codeine report about as much analgesia as those taking a placebo.[14,226] Table 19-8 provides comparable doses of other opioids equivalent to 60 mg of codeine.

TABLE 19-8

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.

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, leukotrienes, bradykinin, platelet-activating factor, substance P, etc.) 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.[150]

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.[167] Pretreatment pain ratings were collected, and 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.[27] 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.[225] 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.[54] Intracanal steroids appear to have significant effects for reducing postoperative pain.[195]

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.[151] The results indicated that the steroid significantly reduced the incidence and severity of pain at 4 hours when compared with the placebo. Pain was reduced at 24 hours, but it was not statistically significant, and no difference in incidence or severity was seen at 48 hours.

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.[140] 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 intraligamentary injection of methylprednisolone, mepivacaine, or placebo in preventing posttreatment endodontic pain.[117] 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 following pulpectomy.[156] 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 intraosseous injection of methylprednisolone or placebo to patients with irreversible pulpitis. Highly significant pain reduction in the steroid group was maintained for 7 days after a single injection.[70]

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.[178]

Other studies of systemic administration have evaluated the efficacy 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 dexamethasone or a placebo tablet orally after initial endodontic treatment.[129] 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.[76] Results showed that dexamethasone was effective in reducing posttreatment endodontic pain up to 8 hours after the treatment was completed; no affect on the severity of pain was seen at 24 and 48 hours after treatment.

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.[63] First, overprescribing 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).[251] 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.[119]

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. 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).[166] The results showed that the overall incidence of flare-ups was 2.2%, with no difference between penicillin or erythromycin. Similar results were obtained in a study in which dental students (instead of private practitioners) provided the endodontic treatment.[3] Outcomes found a 2.6% incidence of flare-up, with no statistically significant differences between penicillin or 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, since in general, randomized controlled studies fail to detect any analgesic benefit to antibiotics, whereas open-label or historical control studies often report profound effects.[63]

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.[165,166] The authors concluded that prophylactic administration of antibiotics is preferable to having the patient take antibiotics at the first sign of an infection.

Another study of similar design compared the incidence of flare-up when a cephalosporin or erythromycin was given prophylactically.[164] 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.[221,222] 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.[221] 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.[222] 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[232] 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.[232] 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.[65] 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).[93,120,121,197,209]

Effective management of endodontic pain starts with the "three Ds": *diagnosis*, *definitive* dental treatment, and *drugs* (Box 19-2). Comprehensive reviews on *diagnosis* and *definitive* dental treatment (e.g., incision and drainage, pulpectomy) are provided elsewhere in this text. As described earlier in this chapter, the management of endodontic pain should focus on removal of peripheral mechanisms of hyperalgesia and allodynia (see Box 19-1). 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.[49,100,154,187] However, pharmacotherapy often is required to reduce continued nociceptor input (e.g., NSAIDs, local anesthetics) and suppress central hyperalgesia (e.g., NSAIDs, opioids).

BOX 19-2

Considerations for Effective "Three-D" Pain Control

1. Diagnosis
2. Definitive dental treatment
3. Drugs
   - Pretreat with 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.

Pretreatment

Treatment with an NSAID before a procedure has been shown to have a significant benefit in many studies[45,109] but not all of them.[177] 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.[162] 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.[49,109,162]

Long-Acting Local Anesthetics

A second pharmacologic approach for pain management is the use of long-acting local anesthetics such as bupivacaine and ropivacaine (see Chapter 20 for an extensive review of this topic). Clinical trials indicate that long-acting local anesthetics not only provide anesthesia during the procedure but also significantly delay the onset of posttreatment pain when compared with local anesthetics that contain lidocaine.[38,45,81,82,111] 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,[81,82,111] because an afferent barrage of nociceptors can induce central hyperalgesia.[242-244] 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.[10,161]

Flexible Plan

A third pharmacologic approach is the use of a flexible plan for prescribing analgesics (Fig. 19-3).* 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 nonnarcotic 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.

Studies have shown that treatment combining an NSAID with acetaminophen 1000 mg alone (i.e., no opioid) produces nearly twice the analgesic response as just the NSAID.[18,35,158] Administration of ibuprofen 600 mg with acetaminophen 1000 mg produced significant relief of posttreatment endodontic pain, compared to ibuprofen alone or placebo (Fig. 19-4). Studies have also shown that concurrent administration of an NSAID and an acetaminophen-opioid combination drug provided significantly greater analgesia than an NSAID alone.[18,211] The concurrent administration of acetaminophen and NSAIDs appears to be well tolerated, with no detectable increase in side effects or alterations in pharmacokinetics.[18,133,211,246]

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

*References 1, 33, 87, 89, 93, 120, 122, and 226.

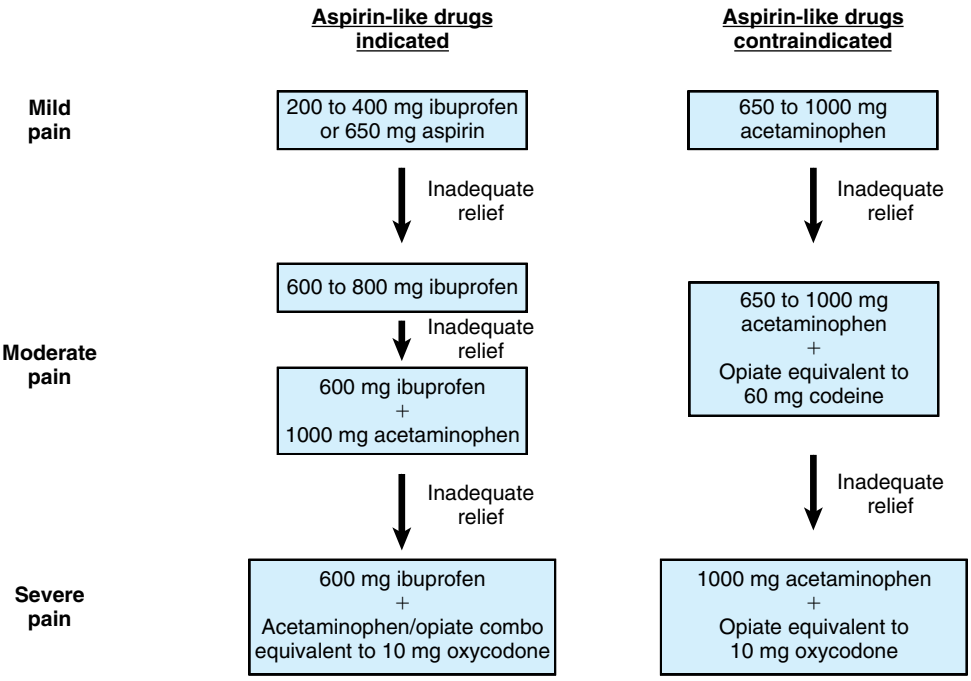


FIG. 19-3 A flexible analgesic strategy.

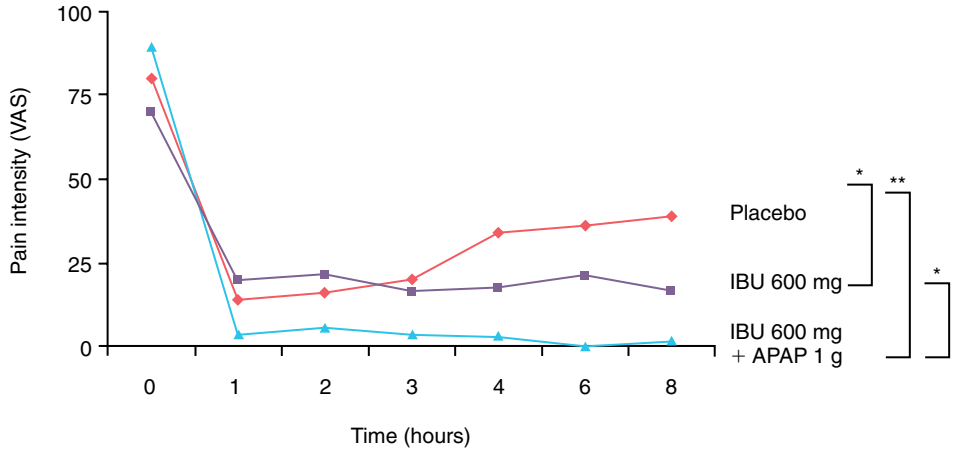


FIG. 19-4 Comparison of ibuprofen 600 mg with acetaminophen 1000 mg to ibuprofen alone or to placebo treatment in postendodontic pain patients. *(From Menhinick KA, Gutmann JL, Regan JD, et al: The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-controlled study.* Int Endod J *37:531–541, 2004. Reprinted with permission.)*

an NSAID followed by an acetaminophen-opioid combination.[1,33] 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.[238] Other opioids can also be added to an NSAID for increased analgesia. Ibuprofen 400 mg with a 10-mg oxycodone tablet produces significantly greater analgesia than ibuprofen alone.[42] A study on posttreatment endodontic pain demonstrated short-term benefits of the combination of flurbiprofen and tramadol.[49] Other NSAID and opioid combinations have also been evaluated.[46] However, the results of clinical trials on the use of NSAIDs alone and combined with acetaminophen (see Fig. 19-4) 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 be 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 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

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 review, see Rollason et al.[196]).

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 morpine.[41] 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—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

20

Local Anesthesia in Endodontics

AL W. READER, JOHN M. NUSSTEIN, and KENNETH M. HARGREAVES

CHAPTER OUTLINE

MECHANISMS OF ACTION FOR ANESTHETICS
CLINICALLY AVAILABLE LOCAL ANESTHETICS
SELECTION OF A LOCAL ANESTHETIC: POSSIBLE ADVERSE EFFECTS, MEDICAL HISTORY, PREOPERATIVE ANXIETY
- Possible Adverse Effects

EFFECTS OF SYSTEMIC DISEASES OR CONDITIONS ON LOCAL ANESTHETICS
CLINICAL ANESTHESIA AND ROUTES OF ADMINISTRATION
IMPORTANT CLINICAL FACTORS IN LOCAL ANESTHESIA
- Traditional Methods of Confirming Anesthesia
- Determining Pulpal Anesthesia in Painless Vital Teeth
- Determining Pulpal Anesthesia in Painful 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 Anesthesia

MANDIBULAR ANESTHESIA WITH 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)
- Long-Acting Anesthetics

ALTERNATE INJECTION SITES
- Gow-Gates and Vazirani-Akinosi Techniques
- Incisive Nerve Block at the Mental Foramen
- Infiltration Injections of a Lidocaine Solution
- Mandibular First Molar Infiltration Injections of an Articaine Solution
- Mandibular First Molar Infiltration Injections of an Articaine Solution Following an Inferior Alveolar Nerve Block

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
- Accuracy of Injection
- Needle Deflection
- Needle Bevel and Success
- Speed of Injection and Success
- Cross-Innervation
- A Theory on Why Failure Occurs With the Inferior Alveolar Nerve Block in Asymptomatic Patients

ENHANCEMENT OF MANDIBULAR ANESTHESIA IN ASYMPTOMATIC PATIENTS
- Supplemental Intraligamentary Injection
- Supplemental Intraosseous Injection
- Use of Mannitol

MAXILLARY ANESTHESIA WITH 2% LIDOCAINE AND 1:100,000 EPINEPHRINE
- Success Rate
- Onset of Anesthesia
- Duration of Anesthesia
- Time Course of Pulpal Anesthesia for the Maxillary First Molar
- Significance of Lip Numbness

ALTERNATIVE ANESTHETIC SOLUTIONS FOR INFILTRATION INJECTIONS
- Plain Solutions: 3% Mepivacaine (Carbocaine, Polocaine, Scandonest) and 4% Prilocaine (Citanest Plain)
- 4% Prilocaine with 1:200,000 Epinephrine (Citanest Forte)
- Articaine with 1:100,000 Epinephrine (Septocaine)
- 0.5% Bupivacaine with 1:200,000 Epinephrine (Marcaine)

EXTENDING THE DURATION OF MAXILLARY INFILTRATIONS
- Increasing the Solution Volume

ALTERNATIVE MAXILLARY INJECTION TECHNIQUES
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
Infiltrations of Articaine Following Maxillary or Mandibular Anesthesia in Patients With Irreversible Pulpitis
Supplemental Buccal Infiltration of Articaine After a Failed Inferior Alveolar Nerve Block in Patients With Irreversible Pulpitis
Intraligamentary Anesthesia
Intraosseous Anesthesia
Intrapulpal Injection
MANAGEMENT OF SPECIFIC ENDODONTIC SITUATIONS
Irreversible Pulpitis
Incision and Drainage
Periradicular Surgery
Interim Treatment for Irreversible Pulpitis Using Pulpotomy
Pain Reduction in Irreversible Pulpitis When Endodontic Treatment Is Impossible
Oral Conscious Sedation
SUMMARY AND FUTURE DIRECTIONS

Effective local anesthesia is the bedrock of pain control in endodontics. Regardless of the clinician's skills, endodontic treatment cannot be delivered 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), the treatment of emergency patients (see Chapter 2), and the development of a comprehensive pain-control plan (see Chapter 19). The authors assume that the reader is familiar with various anesthetic injection techniques; several excellent texts are available for review regarding this point.[116,158]

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 20-1). These channels have a clear clinical relevance.[18,101,143]

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).[273] These channels also are relatively resistant to local anesthetics[216] and are sensitized by prostaglandins.[83] As is explained later in the chapter, the presence of TTX-R sodium channels may explain why local anesthetics are less effective when injected into patients with odontalgia. Moreover, the sensitization of these channels by prostaglandins suggests that rapid-acting nonsteroidal antiinflammatory drugs (NSAIDs) may be useful as a pretreatment to enhance the efficacy of local anesthetics in patients with odontogenic pain[112,179] (see Chapter 19). 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 20-1).

VGSCs consist of an alpha (α) and a beta (β) subunit. The α 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.[101] Interestingly, sensitization of TTX-R channels by prostaglandins lowers the activation threshold and increases the amount of sodium ions that flow through the channel.[83] Put another way, an inflammation-induced elevation in prostaglandin levels sensitizes TTX-R channels, leading to depolarization at lower levels of stimulus strengths. This may explain the increased responsiveness to electrical pulp testing seen in patients with irreversible pulpitis.

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

TABLE 20-1

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

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.[108] 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.[11] 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.[20,100] Prolonged alteration of GPCR function might explain why analgesia obtained with long-acting local anesthetics persists well beyond the period of anesthesia.[40,55,185] 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 (see www.ada.org/prof/resources/topics/color.asp) (Table 20-2). 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.[58] But when it is used for infiltration for anterior teeth, it has a shorter duration of anesthetic action than 2% lidocaine with 1:100,000 epinephrine[43,90] (discussed in more detail later in the chapter).

SELECTION OF A LOCAL ANESTHETIC: POSSIBLE ADVERSE EFFECTS, MEDICAL HISTORY, 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,[114,262] it now is well recognized that even comparatively small amounts of epinephrine can induce measurable tachycardia after nerve block or intraosseous injection.[59,85,220] Several authors have reported increases in heart rate with infiltration injections and nerve blocks using 2% lidocaine with 1:100,000 epinephrine[1,102,140,234,261]; others have reported that no significant changes in heart rate occurred or that the changes were clinically insignificant.[173,260,264] When specific information was given on dosing and heart rate increases, several studies found mean heart rate increases.[1,102,139,261] Two studies found increases on average of about 4 beats/min with approximately 20 µg of epinephrine[102,139]; three studies recorded increases of 10 to 15 beats/min with 45 to 80 µg of epinephrine[1,139,234]; and one study found increases of approximately 21 beats/min using 144 µg of epinephrine.[261] Increasing the amount of epinephrine in an

TABLE 20-2

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

*NOTE: 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 150 pounds (67.5 kg).

†Uniform dental cartridge color codes mandated by the American Dental Association (June 2003).

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 α-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.[61,189] 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 20-2.

Systemic Effects

The acute toxicity of 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 20-1, VGSCs are found in the CNS and the myocardium, the two major areas 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).[48,61] 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.[135,137] An acute hypotensive crisis with respiratory failure also has been interpreted as the result of hypersensitivity to local anesthetics[30]; 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 20-2). Finder and Moore[61] 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.[162,276] 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.[186] 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.[96,162,282] A retrospective study reported that articaine is associated with a fivefold higher incidence of paresthesia compared with lidocaine.[96] A recent study evaluated patients referred with a diagnosis of damage to the inferior alveolar and/or lingual nerve which could only have resulted from an IAN block.[212] Thirty-five percent were caused by a lidocaine formulation, and 30% were caused by an articaine formulation. The conclusion was that there was not a disproportionate nerve involvement from articaine. 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.

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.[239] 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,[228] but case reports of hypersensitivity reactions after administration of local anesthetics have been published.[19,30,187,239] 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[241] concluded that some evidence for exposure to the latex allergen exists, although no causal study has been published.

Reactions to Anesthetic Formulations Containing a Sulfite Antioxidant

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.[6] 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

Several systemic diseases or disorders may require modification of the dosage of local anesthetic. Cardiac patients (e.g., those with unstable angina pectoris, history of myocardial infarction or stroke within the past 6 months, severe hypertension, uncontrolled congestive heart failure, or heart transplant) should not receive a local anesthetic containing a vasoconstrictor and should consult their physicians before undergoing endodontic treatment.[189] A review suggests that patients with Hodgkin's disease or breast cancer who have received radiation treatment to the chest are at risk for radiation-induced coronary artery disease, and this condition also may require medical consultation regarding a reduced dosage of local anesthetic containing a vasoconstrictor.[65]

Alcoholics have been found to be more sensitive to painful stimulation.[254] Alcoholics with a history of depression/unhappiness may also have shallower pulpal anesthesia.[62] In contrast, alcoholics in recovery may not be at increased risk for inadequate pain control with local anesthesia.[62]

Any of the commonly available local anesthetics are safe for use in pregnant or lactating women.[97] 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.[97]

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 20-3). Judicious use of local anesthetic solutions without vasoconstrictors (e.g., 3% mepivacaine) is a reasonable alternative for adult patients.

Authors have found that women try to avoid pain more than men, accept it less, and fear it more.[51,60,147,188] A study found women find postsurgical pain more intense than males, but men are more disturbed than women by low levels of pain that lasts several days.[188] Another study found gender differences in analgesia for postoperative endodontic pain.[233] Anxiety may also modulate differences in pain responses between males and females. We should be aware that women might react differently to pain than men.[60]

CLINICAL ANESTHESIA AND ROUTES OF 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 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.[28,106,169,266] Moreover, from a research perspective, they provide only bimodal responses (e.g., yes or no) and therefore are of little use for detecting parametric differences between anesthetics or routes of injection.

Determining Pulpal Anesthesia in Painless Vital Teeth

Anesthesia in painless vital teeth can be measured more objectively by applying a cold refrigerant (Fig. 20-1) or by using an electric pulp tester (EPT; Fig. 20-2). Application of cold or the electric pulp tester can be used to test the tooth under treatment for pulpal anesthesia before a clinical procedure is started.[26,52,120,154]

TABLE 20-3

Possible Drug Interactions With Vasoconstrictors

Drugs	Possible Adverse Effects	Recommendations
TRICYCLIC ANTIDEPRESSANTS		
Amitriptyline, doxepin	Increased cardiovascular responses	Reduce or eliminate vasoconstrictors
NONSELECTIVE β-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 α-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
THYROID HORMONES		
Levothyroxine	Dysrhythmias (especially with large dosage of vasoconstrictor)	*Euthyroid:* No precaution *Hyperthyroid:* Reduce or eliminate vasoconstrictors
MONOAMINE OXIDASE[35] 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.

Determining Pulpal Anesthesia in Painful Vital Teeth

In painful vital teeth and after administration of local anesthesia, the cold test or electric pulp tester can be used to test for



FIG. 20-1 A cold refrigerant may be used to test for pulpal anesthesia before the start of a clinical procedure. *(Courtesy Coltene Whaledent, Mahwah, NJ.)*



FIG. 20-2 An electric pulp tester also may be used to test for pulpal anesthesia before a clinical procedure is started. *(Courtesy SybronEndo, Orange, CA.)*

pulpal anesthesia before an endodontic procedure is started.[36,52,200,217] If the patient responds to the stimulus, pulpal anesthesia has not been obtained, and supplemental anesthesia should be administered. However, in painful vital teeth (e.g., irreversible pulpitis), the lack of a response to pulp testing may not guarantee pulpal anesthesia.[52,200,217] Therefore, if a patient experiences pain when the endodontic procedure is started, supplemental anesthesia 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.

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.[129] 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 in the individual's mouth. If the answer is yes, supplemental injections should be considered.

Failure to Achieve Anesthesia in Patients With Pain

Obtaining anesthesia often is difficult in patients with endodontic pain who have pulpal pathosis. A number of explanations have been proposed for this.[101] 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. 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.[24,268] Two studies demonstrated that local anesthetics were unable to prevent impulse transmission because of these lowered excitability thresholds.[180,268] Another factor might be the TTX-R sodium channels, which are resistant to the action of local anesthetics[232] and are increased in inflamed dental pulp.[271,273] A related factor is the increased expression of sodium channels in pulps diagnosed with irreversible pulpitis.[250,271,273]

Finally, patients in pain often are apprehensive, which lowers the pain threshold. Therefore practitioners should consider supplemental techniques (e.g., intraosseous injections[197,200,206,217] or periodontal ligament injections[36]) 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.[138,177,178] Although some studies have demonstrated the effectiveness of topical anesthetics,[102,108,192,215] others have shown no significant pain reduction.[82,131,163] 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.[163] The most important aspect of a topical anesthetic may not be its clinical effectiveness but rather its psychologic effect on the patient who believes the practitioner is doing everything possible to prevent pain.

Reversing the Action of Local Anesthesia

Phentolamine mesylate (0.4 mg in a 1.7 ml cartridge, OraVerse, Novalar Pharmaceuticals, San Diego, CA) 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.[104,144] The greatest value of using OraVerse is in the majority of dental procedures where postoperative pain is not of concern. However, some endodontic patients may benefit from the use of a reversal agent when they have speaking engagements, important meetings, or perform in musical or theatrical events. 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.

MANDIBULAR ANESTHESIA WITH 2% LIDOCAINE AND 1:100,000 EPINEPHRINE

Because failure occurs most often with the IAN block,[129] factors that modify mandibular anesthesia must be carefully reviewed. The technique for administering an IAN block can be reviewed in available textbooks.[116,158] This 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.

Anesthetic Success

One way to define anesthetic success is the percentage of subjects who achieve two consecutive 80 readings on electric 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 35% (lateral incisor) to 60% (first premolar and first 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 80 EPT readings at any time during a 60-minute period. Using this criterion, anesthetic failure ranged from 11% (first premolar) to 32% (lateral incisor).†

*References 28, 58, 99, 106, 169, 199, and 266.

†References 28, 58, 99, 106, 169, 199, 243, 266, 267.

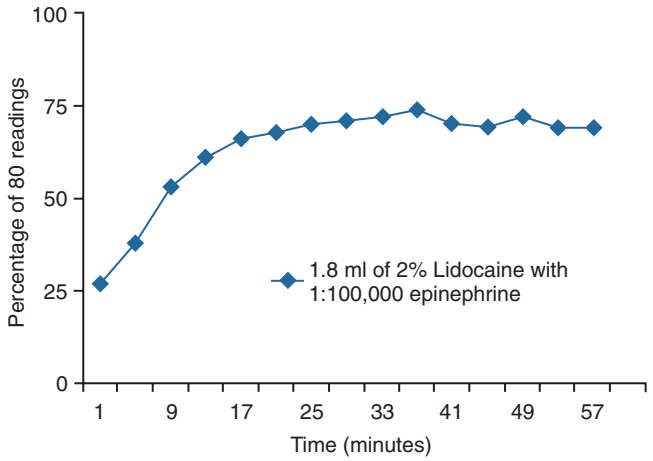


FIG. 20-3 Incidence of first mandibular molar anesthesia as determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes.

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 cases in mandibular teeth.*

Slow Onset

After a conventional IAN block injection, the onset of pulpal anesthesia occurs within 10 to 15 minutes in most cases (Fig. 20-3).† *Slow onset* can be defined as the percentage of subjects who achieved an 80 EPT reading after 15 minutes. In mandibular teeth, slow onset occurs in 19% to 27% 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.[58] Fig. 20-3 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 an 80 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 were as effective as 2% lidocaine with 1:100,000

*References 28, 92, 99, 106, 142, 169, 199, 266, and 290.

†References 28, 58, 99, 106, 169, 199, 243, 266, and 267.

‡References 28, 58, 99, 106, 169, 199, 243, 266, and 267.

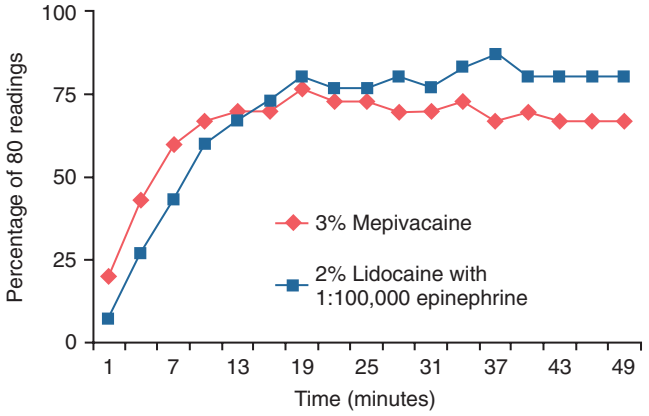


FIG. 20-4 Incidence of first mandibular molar anesthesia: comparison of 3% mepivacaine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 50 minutes. No significant difference between the two solutions was noted.

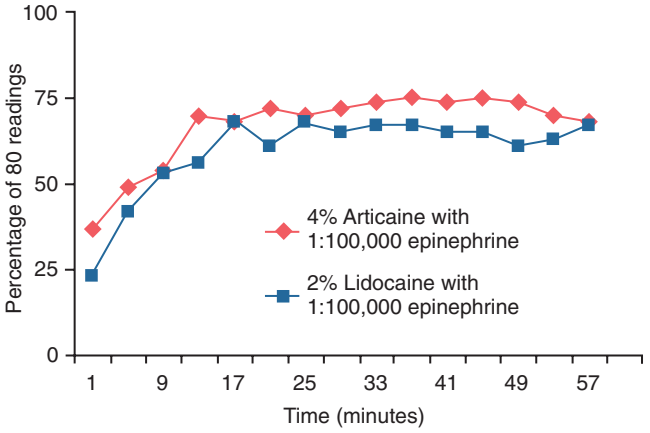


FIG. 20-5 Incidence of first mandibular molar anesthesia: comparison of 4% articaine with 1:100,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two solutions was noted.

(Fig. 20-4).[169] 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.[36] 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 in achieving pulpal anesthesia.[106]

Levonordefrin has 75% α activity and only 25% β activity, making it seemly more attractive than epinephrine (50% α activity and 50% β activity).[158] However, levonordefrin is marketed as a 1:20,000 concentration in dental cartridges.[158] Clinically, the higher concentration of levonordefrin makes it equipotent to epinephrine in clinical and systemic effects,[92,106] so 1:20,000 levonordefrin offers no clinical advantage over 1:100,000 epinephrine.

Articaine With 1:100,000 Epinephrine (Septocaine)

Articaine has been reported to provide very effective local anesthesia.[236] It was approved for use in the United States in April 2000 and is marketed as Septocaine (Septodont, New Castle, DE) and as a 4% solution with either 1:100,000 or 1:50,000 epinephrine.[162,184] 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.[162] 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 20-2).[158]

Clinical Effectiveness of Articaine for Inferior Alveolar Nerve Blocks

The available literature indicates that articaine is equally effective when statistically compared to other local anesthetics.† When 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. 20-5).[176] Two studies found no difference in efficacy between 4% articaine with 1:100,000 and 1:200,000 epinephrine.[184,259] Another study compared the anesthetic efficacy of 4% articaine with 1:100,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine for IAN blocks in patients experiencing irreversible pulpitis, in mandibular posterior teeth.[32] The success rate (none or mild pain upon endodontic access or initial instrumentation) for the IAN block using the articaine solution was 24% and for the lidocaine solution success was 23%. There was no significant difference between the articaine and lidocaine solutions. Neither solution resulted in an acceptable rate of anesthetic success in mandibular posterior teeth. In summary, repeated clinical trials have failed to demonstrate any statistical superiority of articaine over lidocaine for nerve 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."[156] Knowledgeable dentists and educa-

*References 44, 105, 115, 160, 162, 181, 203, 244, and 280.

†References 32, 50, 94, 95, 160, 161, 176, 263, and 281.

tors 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.*[156]

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 done in patients undergoing oral surgery,[46,230] endodontic treatment,[55,185] and periodontic treatment.[40,151] Etidocaine was recently withdrawn from the market by DENTSPLY Pharmaceuticals (York, PA). Bupivacaine provides prolonged analgesia and is indicated when postoperative pain is anticipated, but not all patients want lip numbness for an extended period.[230] Patients should be questioned about their preference. Bupivacaine has a somewhat slower onset than lidocaine, but its duration of pulpal anesthesia in the mandible is almost twice as long (approximately 4 hours; Fig. 20-6).[58]

Ropivacaine (Naropin), a relatively new long-acting local anesthetic, is a structural homolog of bupivacaine.[132] 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.[132] 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.[245] A clinical trial has indicated that levobupivacaine showed significantly better postoperative pain control at 4 and 24 hours after infiltration injection than ropivacaine.[205] Because of their decreased potential for cardiac and CNS toxicity, ropivacaine and levobupivacaine may replace bupivacaine with epinephrine in clinical dental practice.

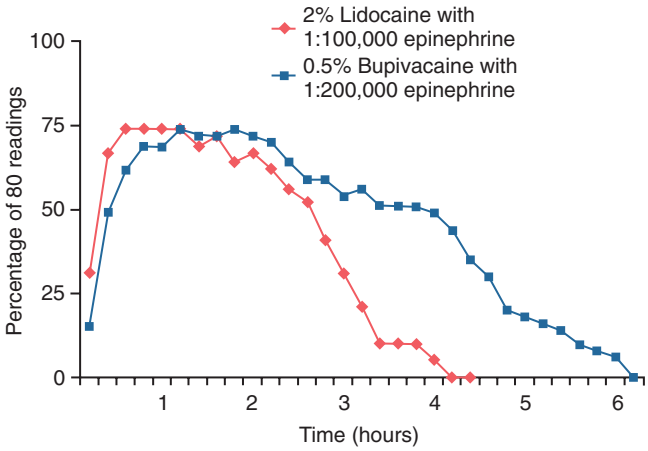


FIG. 20-6 Incidence of first mandibular molar anesthesia: comparison of 0.5% bupivacaine with 1:200,000 epinephrine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 6 hours. The bupivacaine solution showed a longer duration of anesthesia than the lidocaine solution.

ALTERNATIVE INJECTION SITES

Gow-Gates and Vazirani-Akinosi Techniques

Some clinicians have reported that the Gow-Gates technique[88] has a higher success rate than the conventional IAN block injection,[157,158] but controlled experimental studies have failed to show superiority of the Gow-Gates technique.[2,84,182,258] Neither has the Vazirani-Akinosi technique[3,84,158] been found superior to the standard inferior alveolar injection.[84,164,246,258,288] 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.[240] These techniques do not replace the conventional IAN block. The Vazirani-Akinosi technique is indicated for cases involving a limited mandibular opening.

Incisive Nerve Block at the Mental Foramen

The incisive nerve block alone is successful for anesthetizing the premolar teeth,[121,193,275] but it does not anesthetize the central and lateral incisors.[193] A slow injection (60 seconds) did not increase success over a fast injection (15 seconds).[275] The combination of an incisive nerve block and an IAN block increases success of anesthetization of mandibular first molars, but an intraosseous or PDL injection is a better choice for supplemental anesthesia of the first molar if the IAN block fails.[193]

Infiltration Injections of a Lidocaine Solution

Labial or lingual infiltration injections alone are not very effective for obtaining pulpal anesthesia in mandibular teeth.[94,95,286] A combination of labial and lingual infiltration significantly increases success in anterior teeth over either labial or lingual infiltration alone.[172] Adding a labial infiltration (1.8 ml of 2% lidocaine with 1:100,000 epinephrine) to a conventional IAN injection increases the success of pulpal anesthesia in mandibular anterior teeth but not in the mandibular first molar.[33,64]

Mandibular First Molar Infiltration Injections of an Articaine Solution

Four studies showed articaine was significantly better than lidocaine for a primary buccal infiltration of the mandibular first molar.[38,122,124,225] Success rates (two consecutive 80 readings with the electric pulp tester) of 64%, 54%, 64% to 70%, and 87% were recorded for an articaine formulation in these studies. However, the duration of pulpal anesthesia declined over 60 minutes.

Mandibular First Molar Infiltration Injections of an Articaine Solution Following an Inferior Alveolar Nerve Block

A recent study found 4% articaine with 1:100,000 epinephrine resulted in a higher success rate (88%) than 2% lidocaine

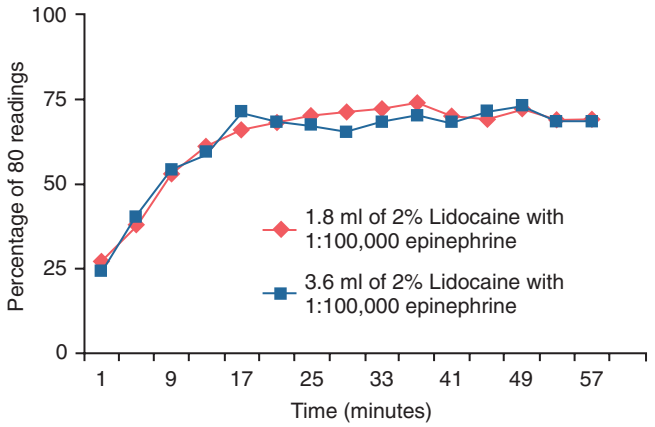


FIG. 20-7 Incidence of first mandibular molar anesthesia: comparison of 3.6 ml and 1.8 ml of 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two volumes was noted.

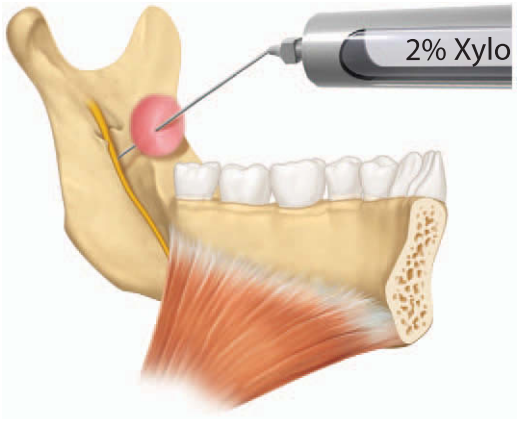


FIG. 20-8 Injection site for the mylohyoid nerve block.

with 1:100,000 epinephrine (71% success rate) for mandibular first molar buccal infiltrations following an IAN block.[98] Success was defined as achieving two consecutive 80 readings within 10 minutes following the IAN block plus infiltration injections, and the 80 reading was continuously sustained through the 60th minute. The finding is important to dentists and patients because we now have a way to help anesthetize the mandibular first molar when the IAN block fails in asymptomatic patients.

ATTEMPTS TO INCREASE SUCCESS OF THE INFERIOR ALVEOLAR NERVE BLOCK

Increasing the Volume of Anesthetic

A possible method for increasing anesthetic success is 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. 20-7).

Increasing the Epinephrine Concentration

A second approach for increasing the success of the IAN block is 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) of epinephrine.[42,267]

Addition of Hyaluronidase

Hyaluronidase reduces the viscosity of the injected tissue, permitting a wider spread of injected fluids.[7] 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.[136,155] 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.[222] In addition, hyaluronidase increased the occurrence of adverse effects (i.e., increased pain and trismus).[222]

Carbonated Anesthetic Solutions

Experimentally, carbonated anesthetic solutions are more effective because the anesthetic is trapped in the nerve.[28] In addition, carbon dioxide (CO_2) has a synergistic relationship with local anesthetics and a direct depressant action on nerves.[28] However, a controlled clinical study was unable to demonstrate a superior effect of lidocaine hydrocarbonate in IAN blocks.[28]

Diphenhydramine as a Local Anesthetic Agent

Diphenhydramine (Benadryl) has been advocated for patients who are allergic to commonly used local anesthetics. Two studies found diphenhydramine was less effective than lidocaine for extractions.[174,272] Another study found the combination of lidocaine/diphenhydramine with epinephrine and diphenhydramine with epinephrine were significantly less effective for pulpal anesthesia than lidocaine with epinephrine for IAN blocks.[277] They also found that the diphenhydramine solutions were more painful upon injection and had a high incidence of moderate postoperative pain.

Addition of Meperidine to Lidocaine

Two studies found the addition of meperidine (Demerol) to a lidocaine formulation did not increase the success of the IAN block.[16,87]

FACTORS IN FAILURE OF THE INFERIOR ALVEOLAR NERVE BLOCK

Accessory Innervation

The mylohyoid nerve is the accessory nerve most often cited as a cause of failure of mandibular anesthesia.[70,278] A controlled clinical trial compared the IAN block alone to a combination of the IAN block and mylohyoid nerve block using 2% lidocaine with 1:100,000 epinephrine (Fig. 20-8), which was aided by the use of a peripheral nerve stimulator.[34] The investigators found that the mylohyoid injection did not significantly enhance pulpal anesthesia of the IAN block (Fig. 20-9), so the study does not support the hypothesis that the mylohyoid nerve is a major factor in failure of the IAN block.

*References 34, 84, 199, 266, 283, and 287.

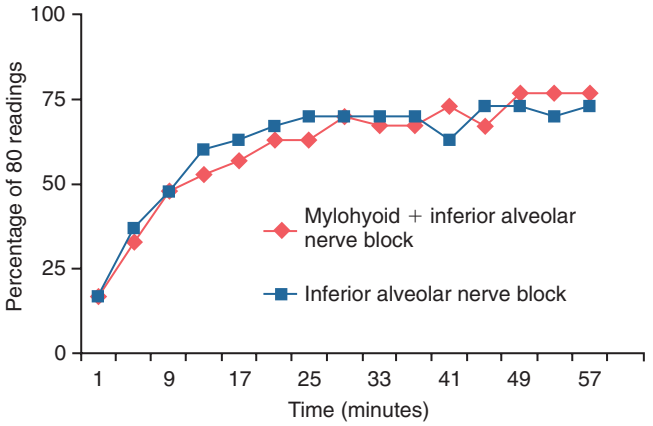


FIG. 20-9 Incidence of first mandibular molar anesthesia: comparison of the combination mylohyoid infiltration plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. No significant difference between the two techniques was noted.

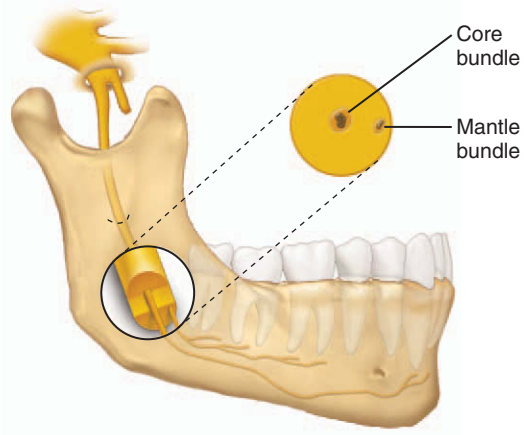


FIG. 20-10 Central core theory. The axons in the mantle bundle supply the molar teeth, and those in the core bundle supply the anterior teeth. The extraneural local anesthetic solution diffuses from the mantle to the core. (*Modified from De Jong RH: Local anesthetics, St Louis, 1994, Mosby.*)

Accuracy of Injection

It has been theorized that an inaccurate injection contributes to inadequate mandibular anesthesia, but a number of studies determined that use of a medical ultrasound unit or radiographs to guide needle placement for IAN blocks did not result in more successful pulpal anesthesia.[14,74,99] 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.[37,45,107a] 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-assisted Wand (CompuDent, Milestone Scientific, Deerfield, IL) has been proposed in which the Wand handpiece assembly and needle are rotated in a fashion similar to the rotation of an endodontic hand file.[107a] 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.[133] 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]) and when the bidirectional Wand needle rotation technique was used (56% success).[133] Neither technique resulted in an acceptable rate of anesthetic success in patients with irreversible pulpitis.

*References 4, 37, 45, 107a, 118, and 226.

Needle Bevel and Success

In asymptomatic subjects, the orientation of the needle bevel away or toward the mandibular ramus for an IAN block did not affect anesthetic success or failure.[252] Therefore, the use of commercial needles with markers to indicate needle bevel is not necessary.

Speed of Injection and Success

A slow IAN block injection (60 seconds) resulted in higher success rates (electric pulp testing) than a rapid injection (15 seconds).[123]

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[227,286] but plays a very small role in failure with IAN block.

A Theory on Why Failure Occurs With the Inferior Alveolar Nerve Block in Asymptomatic Patients

The central core theory may be the best explanation of why failure occurs with the IAN block.[47,255] 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. 20-10). 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 28, 58, 99, 106, 169, 199, 243, 266, and 267.

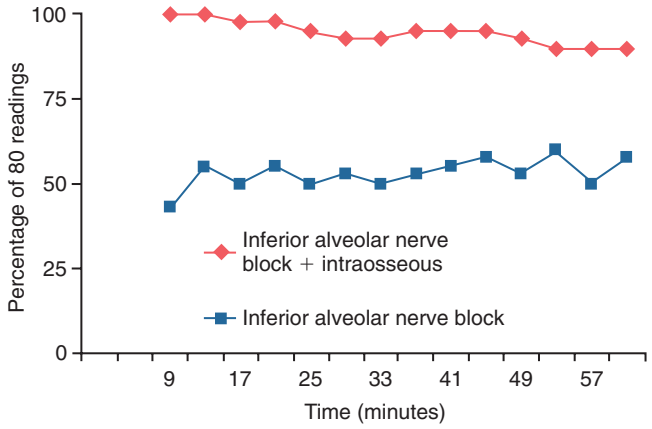


FIG. 20-11 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection of 2% lidocaine with 1:100,000 epinephrine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique was significantly better at all postinjection times.

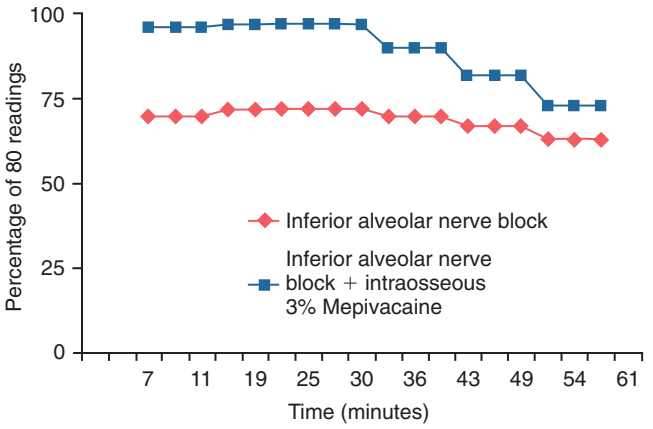


FIG. 20-12 Incidence of first mandibular molar anesthesia: comparison of the combination intraosseous injection with 3% mepivacaine plus the inferior alveolar nerve block to the inferior alveolar nerve block alone. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The combination technique proved significantly better for approximately 30 minutes.

ENHANCEMENT OF MANDIBULAR ANESTHESIA IN ASYMPTOMATIC PATIENTS

Supplemental Intraligamentary Injection

Experimental studies in volunteers without dental pathosis have shown that the addition of an intraligamentary injection of 2% lidocaine with 1:100,000 epinephrine (delivered with a high-pressure syringe) to an IAN block significantly increased the success of pulpal anesthesia for 23 minutes.[29] The short incidence of anesthesia was related to the small amount of anesthetic solution administered.

Supplemental Intraosseous Injection

Several studies have shown that supplemental Stabident intraosseous injection, when added to an IAN block and using local anesthetics with vasoconstrictors, significantly increased the success of pulpal anesthesia for 60 minutes in the first molar (Fig. 20-11).[54,92,251] The intraosseous injection was more successful than the periodontal ligament injection because more anesthetic solution was delivered with the intraosseous injection. The addition of a supplemental intraosseous injection reduced the incidence of slow onset of pulpal anesthesia to zero compared with the IAN block alone (18% incidence).[54]

Another clinical study has shown that the use of 3% mepivacaine as a supplemental intraosseous injection after an IAN block significantly increased the success of pulpal anesthesia for 30 minutes (Fig. 20-12).[77] The shorter duration of the 3% mepivacaine, compared with 2% lidocaine with 1:100,000 epinephrine, was related to the lack of a vasoconstrictor.

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.[8] These researchers found that the use of mannitol in combination with lidocaine increased anesthetic success in IAN blocks about 15% to 20%. The drug combination may be introduced sometime in the future.

MAXILLARY ANESTHESIA WITH 2% LIDOCAINE AND 1:100,000 EPINEPHRINE

Descriptions of conventional techniques for maxillary anesthesia are available for review in numerous articles and textbooks.[116,158]

Clinically, maxillary anesthesia is more easily obtained than mandibular anesthesia.[129] As a frame of reference, the most commonly used injection for anesthetization of maxillary teeth is infiltration with 1.8 ml of 2% lidocaine (1:100,000 epinephrine).

Success Rate

In anterior and posterior teeth, infiltration anesthesia results in a high incidence (90% to 95%) of successful pulpal anesthesia (obtaining an 80 reading on an EPT that ranges from 0 to 80 in intensity).* Failure (never achieving an 80 reading) occurs about 5% to 10% of the time.† Maxillary infiltration anesthesia, therefore, is more successful than an IAN block. The technique does not achieve 100% pulpal anesthesia because of individual variations in response to the drug used and variations in anatomy and tooth position.

Onset of Anesthesia

The onset of pulpal anesthesia usually occurs within 5 to 7 minutes,[90,126,166,175] but slow onset of anesthesia (defined in these cases as achieving an 80 EPT reading after 7 minutes) occurs about 20% of the time in first molars.[90,126,166,175]

*References 31, 90, 95, 126, 166, 175, 210, 213, 263.

†References 31, 90, 126, 166, 175, 210, and 213.

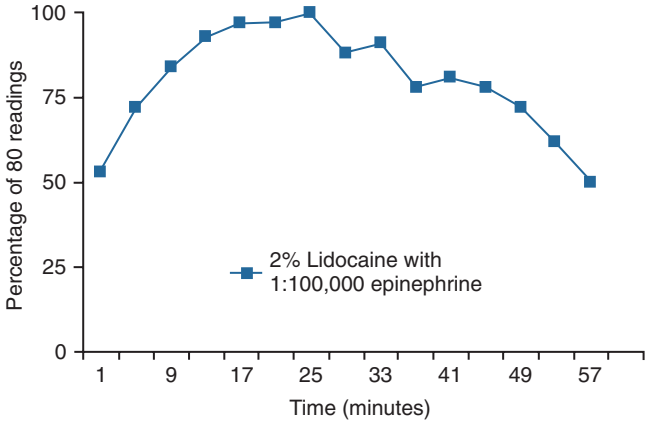


FIG. 20-13 Incidence of first maxillary molar anesthesia, as determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes.

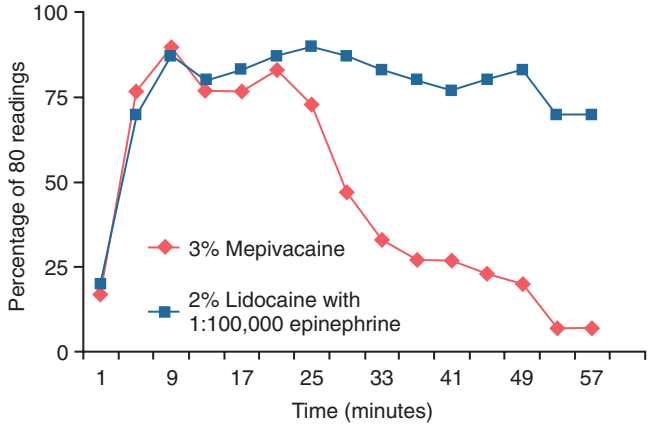


FIG. 20-14 Incidence of first maxillary molar anesthesia: comparison of 3% mepivacaine to 2% lidocaine with 1:100,000 epinephrine. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The 3% mepivacaine showed a shorter duration of anesthesia than the lidocaine solution.

Duration of Anesthesia

The potential problem with infiltration anesthesia in the maxilla is duration. The incidence of short duration of anesthesia (achieving an 80 reading and then losing this reading) ranges from about 60% in lateral incisors to 38% in first molars.[90,126,166,175] Pulpal anesthesia in anterior teeth starts to decline after about 20 to 30 minutes; in molar teeth, anesthesia declines after about 30 to 45 minutes. These time courses have clinical implications. In an emergency endodontic procedure on a first molar, the pulp usually can be removed within 20 to 30 minutes, so the duration of anesthesia is of little consequence. However, if the practitioner is preparing to obturate the tooth and this procedure takes an hour, the patient might experience pain. Therefore additional local anesthetic should be administered.

Time Course of Pulpal Anesthesia for the Maxillary First Molar

Fig. 20-13 shows the time course for complete pulpal anesthesia for an asymptomatic first molar, as defined by the percentage of patients who do not respond to an 80 EPT stimulus across time. Some patients (20%) had a slow onset of anesthesia until around 11 minutes. The overall success rate (no response at the 80 reading) is 95% to 100%, with peak effects observed at around 30 minutes after injection.

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[90,166,175]

ALTERNATIVE ANESTHETIC SOLUTIONS FOR INFILTRATION INJECTIONS

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

Solutions of 3% mepivacaine plain and 4% prilocaine plain provide a short duration of pulpal anesthesia, averaging about 15 to 20 minutes (Fig. 20-14),[126,166] and are indicated for procedures of short duration. 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.[158]

4% Prilocaine With 1:200,000 Epinephrine (Citanest Forte)

The effect of 4% prilocaine (1:200,000 epinephrine) is similar to that of an infiltration injection with 2% lidocaine (1:100,000 epinephrine).[126]

Articaine With 1:100,000 Epinephrine (Septocaine)

Several studies have shown that articaine with epinephrine was similar in action to prilocaine with epinephrine and lidocaine with epinephrine for maxillary infiltration injections.[50,94,95,263] A recent study found maxillary infiltration of 4% articaine with 1:100,000 epinephrine statistically improved anesthetic success, when compared to 2% lidocaine with 1:100,000 epinephrine, in the lateral incisor but not in the first molar. [57]

0.5% Bupivacaine With 1:200,000 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.[43,90,132,257] Although bupivacaine provides long-term anesthesia in the mandible, it *does not* provide prolonged pulpal anesthesia with maxillary infiltration injection.[43,90,132] In the lateral incisor, bupivacaine has a shorter duration of pulpal anesthesia than lidocaine.[43,90] In the first molar, bupivacaine's duration of pulpal anesthesia is equivalent to that of lidocaine.[90]

EXTENDING THE DURATION OF MAXILLARY INFILTRATIONS

Increasing the Solution Volume

For maxillary infiltrations, increasing the volume of 2% lidocaine with 1:100,000 epinephrine from 1.8 ml to 3.6 ml

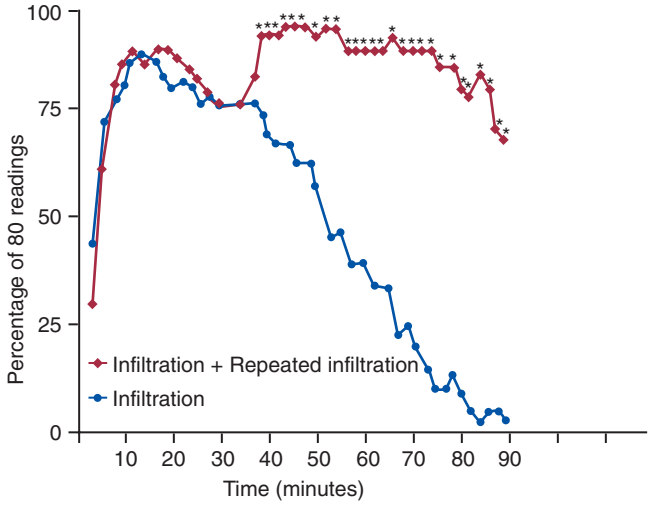


FIG. 20-15 Incidence of maxillary lateral incisor pulpal anesthesia using an initial infiltration and a repeated infiltration 30 minutes later (both infiltrations used 1.8 ml of 2% lidocaine with 1:100,000 epinephrine). Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings). The repeated infiltration injection significantly prolonged the duration of pulpal anesthesia.

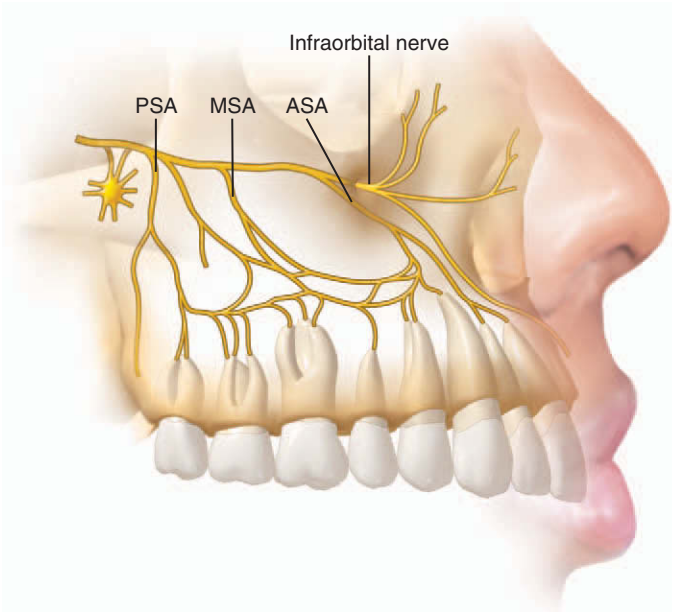


FIG. 20-16 Distribution of the maxillary division of the trigeminal nerve, showing the anterior superior alveolar (ASA) nerve, the middle superior alveolar (MSA) nerve, and the posterior superior alveolar (PSA) nerve.

provided a statistically longer duration of pulpal anesthesia.[175] A repeated infiltration of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine given 30 minutes following an initial infiltration of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine significantly improved the duration of pulpal anesthesia (from 37 minutes through 90 minutes) in the maxillary lateral incisor[238] (Fig. 20-15).

ALTERNATIVE MAXILLARY INJECTION TECHNIQUES

Posterior Superior Alveolar (PSA) Nerve Block

The PSA nerve block provides pulpal anesthesia for the second and third molars and in some first molars.[154] Generally, with the first molar, an additional buccal infiltration injection may be needed after the PSA block to ensure patient comfort. The PSA injection often is indicated when all the molar teeth require anesthesia. If the procedure involves the first molar, an infiltration injection is a better choice because it is more predictable for pulpal anesthesia than the PSA injection.

Infraorbital Nerve Block

The infraorbital nerve block produces profound lip numbness.[12,125] It successfully anesthetizes the first and second premolars, but the duration of pulpal anesthesia is less than 60 minutes.[12,125] The infraorbital block does not successfully anesthetize the pulps of the central or lateral incisors.[12,125] Essentially, this injection technique is the same as an infiltration injection over the premolar teeth for pulpal anesthesia.

Second Division Nerve Block

The second division nerve block successfully anesthetizes the pulps of molar teeth and about 50% of the second premolars.[22,165] It does not routinely anesthetize the pulps of anterior teeth.[22,165] The high tuberosity approach is preferred over the greater palatine technique because the success rate is similar and the procedure is less painful.[22]

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 90s the P-ASA injection, a site-specific injection for maxillary anterior teeth, was introduced.[68,69] The P-ASA injection involves a palatal injection into the incisive canal and derives its name from the injection's supposed ability to anesthetize both the right and left anterior superior alveolar nerves (Fig. 20-16). A P-ASA injection of 0.9 to 1.4 ml of anesthetic solution has been reported to produce anesthesia of the maxillary incisors and usually the canines, with an expected duration of approximately 60 minutes.[68] One study compared the anesthetic efficacy of 2% lidocaine with 1:100,000 epinephrine and 3% mepivacaine using the computer-assisted Wand Plus injection system and the P-ASA injection route.[23] Neither anesthetic produced greater than 58% successful pulpal anesthesia and therefore would not clinically ensure predictable pulpal anesthesia for the four maxillary incisors and canines.

Moreover, 30% to 43% of patients report moderate to severe pain from the needle insertion for the P-ASA injection.[195] For needle placement into the incisive canal, 54% to 58% of subjects reported moderate to severe pain. For deposition of the anesthetic solution, 8% to 12% of subjects reported moderate pain. Postoperatively, approximately 12% to 18% of the subjects experienced temporary numbness or paresthesia of the incisive papilla, and 20% to 28% had swelling or soreness of the incisive papilla. The P-ASA injection, therefore, can be a painful injection, and it has a low to modest success rate.



FIG. 20-17 Palatal injection site for the anterior middle superior alveolar (AMSA) injection.

Anterior Middle Superior Alveolar (AMSA) Nerve Block

The AMSA injection is another new technique for anesthetizing maxillary teeth.[66,67,69] 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. 20-17). The AMSA injection supposedly can anesthetize both the anterior and middle superior alveolar nerves[66,67,69] (see Fig. 20-16). Injection of 0.6 to 1.4 ml of anesthetic solution has been reported to produce pulpal anesthesia of the maxillary central and lateral incisors, canines, and first and second premolars, with an expected duration of 45 to 60 minutes.[66,67,69] A bilateral AMSA injection supposedly anesthetizes 10 maxillary teeth, extending from the second premolar on one side to the second premolar on the opposite side.[67]

Two studies of the AMSA technique found rather modest to low success rates, slow onset, and declining duration of pulpal anesthesia over 60 minutes.[73,146] The AMSA injection would not ensure predictable pulpal anesthesia from the second premolar to the central incisor.[73,146] Moreover, the AMSA injection, whether made with a computer-assisted injection system or a conventional syringe, produced moderate pain on needle insertion in 32% to 38% of study subjects and moderate pain upon solution deposition in 25% to 40% of subjects.[198]

When the Wand Plus was used for the AMSA injection, statistically lower pain ratings were recorded for deposition of the anesthetic solution. However, regardless of whether the Wand Plus or a conventional syringe is used, the AMSA injection can be a painful injection, and it has a low to modest success rate.

SUPPLEMENTAL ANESTHESIA

Supplemental injections are essential when, as frequently occurs, anesthesia from conventional injections is inadequate and the pain is too severe for the practitioner to proceed. Three such supplemental techniques can be used: the intraligamentary injection, the intraosseous injection, or the intrapulpal injection.

If the patient has *profound lip numbness and experiences pain upon endodontic access,* repeating the IAN block does not help the problem. 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.

Infiltrations of Articaine Following Maxillary or Mandibular Anesthesia in Patients With Irreversible Pulpitis

Using a visual analog scale to evaluate success, no significant differences were found between an articaine solution and a lidocaine solution when used as a supplemental infiltration after IAN blocks or maxillary infiltrations in patients with irreversible pulpitis.[229] In a small study of 19 patients, no difference was found between lidocaine (9 patients) and articaine (10 patients) formulations in maxillary infiltrations for patients with irreversible pulpitis.[240]

Supplemental Buccal Infiltration of Articaine After a Failed Inferior Alveolar Nerve Block in Patients With Irreversible Pulpitis

A recent study found that when IAN block failed, there was only a 58% success rate for a supplemental buccal infiltration of 1.8 ml of 4% articaine with 1:100,000 epinephrine in mandibular posterior teeth in patients with irreversible pulpitis.[167] This success rate was much lower than a supplemental intraosseous injection.

Intraligamentary Anesthesia

The technique for intraligamentary injection of anesthesia is reviewed in a number of published papers and textbooks.

Success of Intraligamentary Injection

The success of supplemental intraligamentary injections in achieving pulpal anesthesia for endodontic procedures has been reported to be 50% to 96%.[36,159,249,269] Intraligamentary injection produced a 50% to 79% success rate in endodontic treatment when used as a primary anesthetic technique.[127,159] If the first intraligamentary injection failed, reinjection was successful in 71% of patients, for an overall success rate of 92%.[269] Similar results have been reported by other investigators.[36,249] Intraligamentary injection is not successful in mandibular anterior teeth.[172,274]

Mechanism of Action

An intraligamentary injection forces anesthetic solutions through the cribriform plate into the marrow spaces around the tooth.[53,72,215,248,270] The primary route is *not* via the periodontal ligament, and unlike the intrapulpal injection,[17,265] the mechanism of action is not a pressure anesthesia.[56,183] The intraligamentary injection should be considered an intraosseous injection.

Back-Pressure

Studies have shown that the most important factor for anesthetic success with an intraligamentary injection is injection under *strong back-pressure*.[248,269] Pressure is necessary to force the solution into the marrow spaces.

Anesthetic Solutions

A vasoconstrictor significantly increases the efficacy of an intraligamentary injection.[89,128,134,170,237] Injection of a vasoconstrictor alone (1:100,000 epinephrine) does not produce pulpal anesthesia.[237] Anesthetic solutions with reduced vasoconstrictor concentrations (bupivacaine or etidocaine with 1:200,000 epinephrine) are not very effective with this technique.[89,119,128]

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 an intraligamentary injection is given as a primary injection, needle insertion and injection of the anesthetic solution are only mildly discomforting in posterior teeth[41,159,237,274]; however, in maxillary lateral incisors, an intraligamentary injection can be painful.[274]

When the intraligamentary injection is given as a supplemental injection to anesthetize symptomatic vital teeth (i.e., in irreversible pulpitis), the patient may have moderate pain.[52] Patients should be informed of this possibility.

Onset of Anesthesia

The onset of anesthesia is immediate with an intraligamentary injection,* which means no waiting period is required for the anesthesia to take effect.

Duration

Experimental studies with the EPT have shown that when the intraligamentary injection is given as a primary injection, the duration of profound pulpal anesthesia is approximately 10 to 20 minutes.[183,237,274] 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.[29] Therefore, when the intraligamentary 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.

Postoperative Discomfort

Most patients have postoperative discomfort (mostly mild pain) when an intraligamentary injection is used as a primary technique.[41,237,274] Most of this discomfort occurs the first day after injection, and the duration of discomfort averages 14 hours to 3 days.[41,237,274] Postinjection discomfort is related to damage from insertion of the needle and not from the pressure of solution deposition.[41] About 40% of patients report that the tooth feels high in occlusion.[237,274]

When an intraligamentary injection is used as a supplemental technique for endodontic therapy, pain from the intraligamentary injection probably occurs in addition to any postoperative pain from the endodontic treatment.

*References 41, 183, 237, 249, 269, and 274.

Avulsion

In a letter to the editor of the *Journal of the American Dental Association*, Nelson[191] reported on the avulsion of a tooth following intraligamentary injections. However, *no* clinical or experimental study has reported avulsion or loosening of teeth with this technique.[183,237,274] Avulsion should not be a concern with intraligamentary injections.

Selective Anesthesia

Although some have reported that the intraligamentary injection can be used in the differential diagnosis of pulpally involved teeth,[153,242] experimental studies have shown that adjacent teeth may also become anesthetized with intraligamentary injection for a single tooth.[183,237,274] Therefore the intraligamentary injection should *not* be used for differential diagnosis.

Systemic Effects

When a high-pressure syringe was used in dogs, intraligamentary injection of solutions containing epinephrine caused cardiovascular responses similar to those seen with IV injections.[247] Clinical studies using a high-pressure syringe in human beings found that intraligamentary injections of such solutions did not significantly change the heart rate, rhythm, amplitude, or the blood pressure.[25,194] These studies support the conclusion that intraligamentary injections do not cause significant changes in heart rate in human beings.

Needle Gauge

Different needle gauges (25, 27, or 30) have been shown to be equally effective.[159,269]

Ligamental Syringes

Special ligamental syringes have not proved any more effective than a standard syringe.[41,249,269]

Safety to the Periodontium

Clinical and animal studies have demonstrated the relative safety of the intraligamentary injection technique.* Minor transient damage occurs only at the site of needle penetration, and the tissue subsequently undergoes repair. In very rare cases, periodontal abscesses and deep pocket formation have occurred after intraligamentary injections.[29,274] A very 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 intraligamentary periodontal ligament injections have also been reported.[207,223]

Safety to the Pulp

Clinical and animal studies have shown that intraligamentary injections have no permanent effect on the pulp.† However, intraligamentary injection of a solution with epinephrine produces a rapid, prolonged decrease in blood flow.[134] 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

*References 21, 71, 72, 75, 152, 183, 208, 237, 269, and 274.

†References 149, 183, 209, 223, 237, and 274.

reduced blood flow.[134] This hypothesis was directly tested, and intraligamentary 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).[211] Rather, the depth of the cavity preparation was the most important factor dictating pulpal responses. Intraligamentary injections are therefore unlikely to cause pulpal necrosis.

Safety in Primary Teeth

One study has shown that intraligamentary injection of primary teeth may cause enamel hypoplasia of the developing permanent teeth.[21] 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 intraligamentary injections be used with great care on primary teeth close to developing permanent teeth[21] may not be correct.

Safety in Periodontally Involved Sites

Intraligamentary injections have been shown to be safe in cases of mild to moderate gingival inflammation or incipient periodontitis.[39]

Precautions

No research as yet has shown how painful an intraligamentary injection would be or if it would result in anesthesia in teeth with cellulitis or abscess formation (symptomatic necrotic teeth with periradicular radiolucencies).

New Technology for Intraligamentary Injections: The Wand

A computer-assisted local anesthetic delivery system was introduced by Milestone Scientific (Livingstone, NJ) that can be used to administer an intraligamentary injection. The Wand (also called *CompuDent* or *CCLAD*) accommodates a standard local anesthetic cartridge that is linked by sterile microtubing to a disposable, penlike handpiece with a Luer-Lok needle (Fig. 20-18). The device is activated by a foot control, which automates the infusion of local anesthetic solution at a controlled rate. Two flow rates, slow or fast, may be initiated and maintained by a foot pedal control. 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 intraligamentary injection.

Anesthetic Success of the Intraligamentary Injection, Using the Wand in Asymptomatic Vital Teeth

An experimental study recently compared the anesthetic efficacy of primary intraligamentary 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 computer-controlled local anesthetic delivery system in the mandibular first molar.[13]Successful pulpal anesthesia (two consecutive 80 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.[274] Therefore the computer-controlled local anesthetic delivery system offers the advantage of increasing the duration of pulpal anesthesia; however, the anesthesia slowly decreases over 60 minutes.



FIG. 20-18 Wand computer-assisted local anesthetic delivery unit. The handpiece assembly and microtubing are also shown. *(Courtesy CompuDent/Milestone Scientific, Livingston, NJ.)*

Anesthetic Success of the Intraligamentary Injection Using the Wand in Symptomatic Vital Teeth

Supplemental intraligamentary injection, administered with a computer-controlled anesthetic delivery system in mandibular posterior teeth diagnosed with irreversible pulpitis after failure of an IAN injection, produced a 56% anesthesia success rate.[196] These results were somewhat disappointing because the computer-controlled anesthetic delivery system should have been capable of delivering approximately 1.4 ml of anesthetic solution with the intraligamentary injection by consistent maintenance of a precise flow rate.

Intraosseous Anesthesia

Stabident and X-Tip Systems

The intraosseous route delivers a local anesthetic solution directly into the cancellous bone adjacent to the tooth to be anesthetized. Infiltration injections of lidocaine formulations are not effective for anesthesia of the mandibular molar teeth because of the thickness of the cortical plate; therefore dentists do not attempt infiltration anesthesia in the posterior mandible. The intraosseous injection overcomes this problem by allowing direct access to the cancellous bone. One clinical study directly compared the infiltration and intraosseous injection techniques, using 1.8 ml of 2% lidocaine with 1:100,000 epinephrine in the maxillary lateral incisor.[201] The two techniques produced similar results, except that the intraosseous technique had a quicker onset and a shorter duration of anesthesia (Fig. 20-19).

Two intraosseous systems have been studied clinically: the Stabident system (Fairfax Dental Inc, Miami, FL) and the X-tip

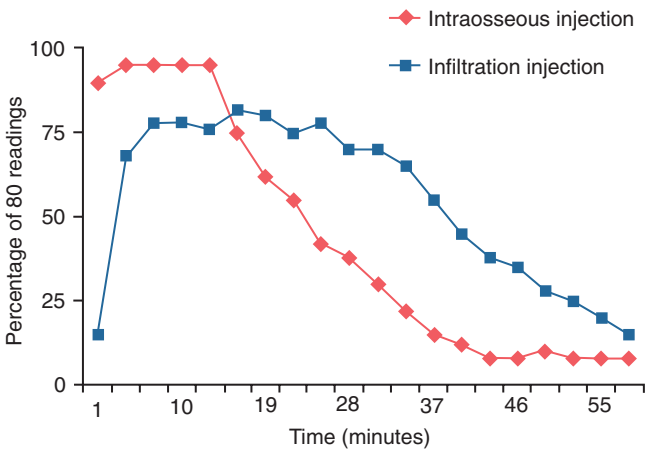


FIG. 20-19 Incidence of anesthesia for intraosseous and infiltration injections. Results were determined by lack of response to electrical pulp testing at the maximum setting (percentage of 80 readings) across time for 60 minutes. The intraosseous injection showed a quicker onset and a shorter duration of anesthesia.

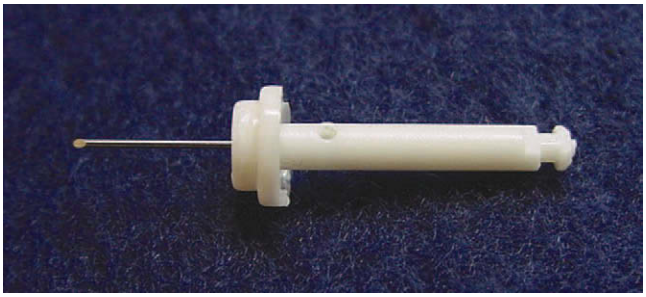


FIG. 20-20 Stabident perforator, a solid 27-gauge wire with a beveled end that is placed in a slow-speed handpiece.



FIG. 20-21 The anesthetic solution is delivered to the cancellous bone through the needle placed into the hole made by the perforator.

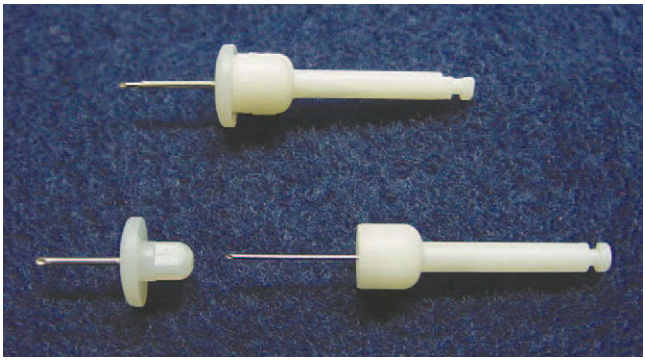


FIG. 20-22 The X-tip anesthesia delivery system consists of an X-tip (*top*) that separates into two parts: the drill (a special hollow needle) and the guide sleeve component (*bottom*).

system (DENTSPLY, Tulsa, OK). Two other anesthetic systems have been introduced: the IntraFlow (Pro-Dex Inc, Irvine, CA) and the Comfort Control Syringe (DENTSPLY International, York, PA). The IntraFlow system combines a slow-speed handpiece with an anesthetic cartridge dispenser system and a rotating needle/drill. The anesthetic solution is delivered after the cortical bone is perforated. When the IntraFlow system was used as a primary technique in a small group of 15 patients with irreversible pulpitis, an 87% success rate was reported (two consecutive 80 readings with a pulp tester).[216] While encouraging, more research on the IntraFlow system is needed. The Comfort Control Syringe is an electronic delivery system and has five preprogrammed injection rates. No published controlled studies have evaluated the Comfort Control Syringe system in clinical dentistry.

The Stabident system is composed of a slow-speed, handpiece-driven perforator, a solid 27-gauge wire with a beveled end that drills a small hole through the cortical plate (Fig. 20-20). The anesthetic solution is delivered to cancellous bone through the 27-gauge, ultrashort injector needle placed into the hole made by the perforator (Fig. 20-21).

The X-tip anesthesia delivery system consists of an X-tip that separates into two parts, the drill and the guide sleeve (Fig. 20-22). The drill, a special hollow needle, leads the guide sleeve through the cortical plate, whereupon it is separated and withdrawn. The remaining guide sleeve is designed to accept a 27-gauge needle for injection of the anesthetic solution (Fig. 20-23). The guide sleeve is removed after the intraosseous injection is complete.

The technique for intraosseous injection of anesthesia using the Stabident or X-tip system can be reviewed in the systems' instruction manuals or in published papers.[35,78,218-220]

PERFORATION PAIN AND SOLUTION DEPOSITION WITH IRREVERSIBLE PULPITIS

Generally the Stabident system produces a very low incidence of moderate pain from perforation and solution deposition in asymptomatic patients.[35,78,218-220] A higher incidence was reported when the system was used to make intraosseous injections in mandibular posterior teeth with 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.[200,217]

With the X-tip system, 48% of patients with irreversible pulpitis had moderate to severe pain with perforation, and 27% had moderate pain with solution deposition.[197] Patients with

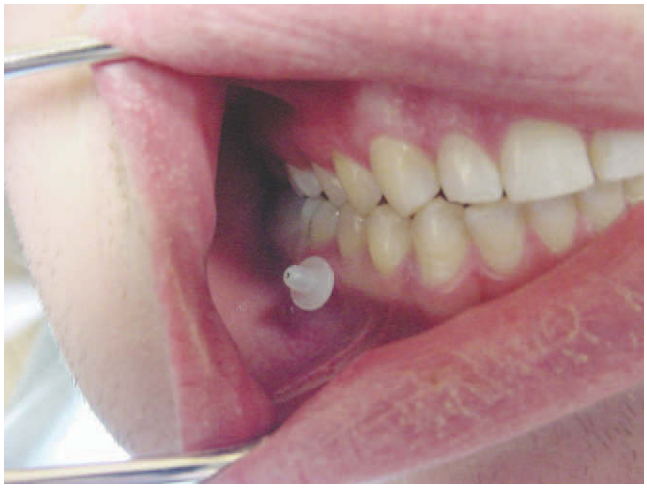


FIG. 20-23 Anesthetic solution is injected through the X-tip guide sleeve.

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 hyperalgesia, which leads to increased pain responsiveness and preoperative anxiety.

PERFORATOR BREAKAGE

In about 1% of cases, the metal perforator separates from the plastic shank during use.[35,54,79,197,218-220] 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 studies.[35,54,79,197,218-220]

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 intraosseous systems instruct the user to locate the perforation site in attached gingiva. The 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.[79,197] 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 for delivering the anesthetic solution is almost impossible to find. The clinician may want to consider using the X-tip 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.

*References 35, 54, 79, 92, 218-220, and 251.

Success of the Inferior Alveolar Nerve Block in Patients With Irreversible Pulpitis

Clinical studies of endodontics in patients with irreversible pulpitis have found that success (mild or no pain upon endodontic access or initial instrumentation) with the IAN block occurred between 19% and 56% of the time.* These studies would indicate that anesthesia is often difficult to achieve in irreversible pulpitis with only the IAN block.

Success of Intraosseous Anesthesia With Irreversible Pulpitis

STABIDENT SYSTEM

In a clinical trial on patients with posterior teeth with irreversible pulpitis, a supplemental mandibular intraosseous Stabident injection (1.8 ml of 2% lidocaine with 1:100,000 epinephrine) was 91% successful in achieving total pulpal anesthesia.[200] A second clinical trial evaluated the Stabident intraosseous injection in patients with irreversible pulpitis when conventional local anesthetic techniques failed. These researchers found that an initial supplemental intraosseous injection (0.45 to 0.9 ml of 2% lidocaine with 1:100,000 epinephrine) was successful in 79% of posterior mandibular teeth.[206] A second intraosseous 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. A third clinical trial in patients with mandibular teeth with irreversible pulpitis showed that supplemental intraosseous injection of 1.8 ml of 3% mepivacaine increased the success rate to 80% compared with the IAN block alone (25% success).[217] A repeat intraosseous injection of 1.8 ml of 3% mepivacaine increased the success rate to 98%. Therefore one cartridge of 3% mepivacaine plain is not as effective as one cartridge of 2% lidocaine with 1:100,000 epinephrine. However, injection of 3% mepivacaine does not produce the tachycardia observed with solutions that contain epinephrine.

X-TIP SYSTEM

One clinical trial evaluated a supplemental X-tip intraosseous injection (1.8 ml of 2% lidocaine with 1:100,000 epinephrine) in patients with irreversible pulpitis when a conventional IAN block failed.[197] The X-tip injection site was 3 to 7 mm apical to the mucogingival junction of the mandibular molar or premolar tooth. The results showed an 82% success rate for

*References 15, 32, 36, 197, 200, and 217.

anesthesia; it also showed an 18% failure rate caused by backflow of the anesthetic solution into the oral cavity. These researchers concluded that when an IAN block fails to provide profound pulpal anesthesia, the X-tip system, when used in an apical location without backflow of the anesthetic solution into the oral cavity, successfully achieves pulpal anesthesia in mandibular posterior teeth with irreversible pulpitis.

Intraosseous Success With an Articaine Solution in Patients With Irreversible Pulpitis

For posterior teeth diagnosed with irreversible pulpitis, the supplemental intraosseous injection of 1.8 ml of 4% articaine with 1:100,000 epinephrine was 86% successful when the IAN block failed.[15] Therefore, the success rate of the articaine solution was similar to a solution of lidocaine.

Repeating the Intraosseous Injection

Repeating the intraosseous injection, using 1.4 ml of 2% lidocaine with 1:100,000 epinephrine 30 minutes after the initial intraosseous injection, provided an additional 15 to 20 minutes of pulpal anesthesia—similar to the duration of the initial intraosseous injection.[117]

Key to Successful Intraosseous Injection

The key to a successful intraosseous injection is flow of the anesthetic into the cancellous space. If anesthetic solution leaks out of the perforation site into the oral cavity, no anesthetic effect is achieved. 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.

Duration

In patients with irreversible pulpitis, supplemental intraosseous injection using either the Stabident or X-tip system provided anesthesia for the entire débridement appointment.[197,200,217]

Timing of Intraosseous Injection

Considering the high failure rate of the IAN block in patients in pain, prudent practice would be to give all patients with irreversible pulpitis a supplemental intraosseous injection after an IAN block—that is, once the signs of lip numbness have developed, an intraosseous injection should be given. 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[5]; nevertheless, up to 96% of baby boys currently 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.

Anesthetic Success in Painful Teeth With Totally Necrotic Pulps and Radiolucent Areas

No study has investigated the use of intraosseous anesthesia in patients with painful 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 intraosseous 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).

Systemic Effects of Intraosseous Injection

CARDIOVASCULAR EFFECTS

A transient increase in heart rate has been reported in 46% to 93% of cases involving Stabident and X-tip intraosseous 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 intraosseous injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine, 2% mepivacaine with 1:20,000 levonordefrin, or 1.8 ml of 1.5% etidocaine with 1:200,000 epinephrine.[27,92,221,251] Another clinical trial reported transient tachycardia after intraosseous injection, but not with infiltration injection, of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine in the maxillary anterior region.[279] 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 systems, results in a transient tachycardia. No significant change in diastolic, systolic, or mean arterial blood pressure has been observed with intraosseous injection of 2% lidocaine with 1:100,000 epinephrine.[27,221]

An intraosseous injection of 1.4 ml of 2% lidocaine with 1:100,000 epinephrine with the Wand (CompuDent, CCLAD, Milestone Scientific, Livingston, NJ) at a fast rate resulted in a significantly higher heart rate when compared to a slow solution deposition using either the Wand or traditional syringe.[256]

Clinical Significance of Increased Heart Rate. Although the patient is likely to notice the transient tachycardia that occurs after Stabident or X-tip intraosseous injection of 2% lidocaine with 1:100,000 epinephrine, it generally is not clinically significant in healthy patients.[221] The clinical significance, cardiovascular effects, and contraindications to the use of vasoconstrictors in intraosseous injections have been reviewed.[221]

*References 35, 54, 79, 92, 197, 200, 217-220, and 251.

*References 35, 54, 79, 92, 197, 200, 218-220, and 251.

Lack of Heart Rate Effect Of 3% Mepivacaine in Intraosseous Anesthesia. No significant tachycardia occurs when 3% mepivacaine is used for intraosseous anesthesia.[77,221] Clinicians should keep in mind that this anesthetic is an alternative for intraosseous injection in patients whose medical condition or drug therapy suggests caution in the use of solutions containing epinephrine or levonordefrin.[77,221]

CAUTIOUS USE OF LONG-ACTING ANESTHETICS FOR INTRAOSSEOUS ANESTHESIA

In an attempt to increase the duration of pulpal anesthesia with intraosseous injections, some clinicians may consider using long-acting anesthetics. Bupivacaine (Marcaine) and etidocaine (Duranest) are long-acting anesthetics but only for IAN blocks. Long-acting anesthetics do not show an extended duration of anesthesia when injected by the intraosseous or maxillary infiltration routes.[43,90,109,251] Bupivacaine and etidocaine have cardiotoxic effects[10] and for intraosseous anesthesia basically are equivalent to 2% lidocaine with epinephrine in terms of efficacy, duration, and heart rate effects. Therefore bupivacaine and etidocaine offer no advantage clinically and should not be used for intraosseous anesthesia.

Plasma Levels of Lidocaine After Intraosseous Injection

Some authors have cautioned that administration of an overly large volume of local anesthetic with an intraosseous injection could lead to overdose reactions.[113] 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 intraosseous and infiltration injections (Fig. 20-24).[279]Although systemic concentrations of vasoconstrictors have a short-lived effect on the heart rate, the plasma concentration of lidocaine delivered with intraosseous injection is no more than that delivered with infiltration, so the intraosseous 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 intraosseous techniques.* Therefore the precautions for the maximum amount of lidocaine for an infiltration injection would seem to apply to an intraosseous injection.

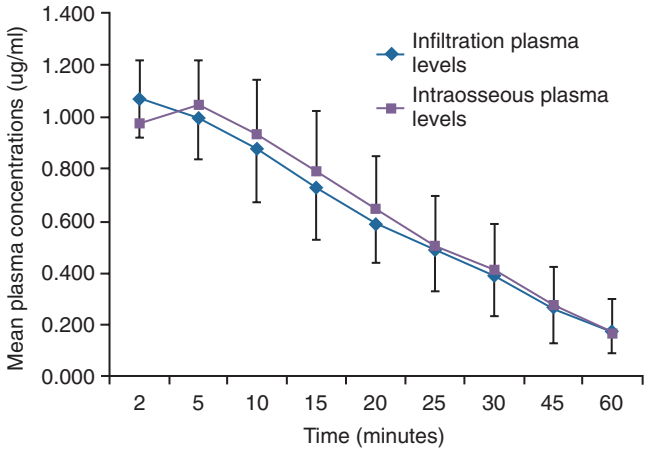


FIG. 20-24 Mean plasma concentrations of lidocaine for the intraosseous and infiltration injection techniques. No statistical differences were seen between the two techniques at any time period.

Postoperative Discomfort

When intraosseous 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.[35,92,218-220] Less postoperative discomfort is reported for the Stabident intraosseous injection than for periodontal ligament injection.[237]

One study found that significantly more men experienced postoperative pain with the X-tip system than with the Stabident system.[78] 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 that the X-tip system generates more frictional heat during perforation. In patients with irreversible pulpitis, the postoperative pain of the endodontic procedure likely adds to any postoperative pain from the intraosseous injection.

Postoperative Problems

With the Stabident system, fewer than 5% of patients develop swelling or exudate at the site of perforation.[35,92,218-220] The X-tip system may show a higher incidence of postoperative swelling clinically.[78] With both systems, swelling or exudate (or both) may persist for weeks after the injection, but this resolves with time.[35,78,92,218-220] 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.[35,78,92,218-220] 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 for the intraosseous injection is lower than that reported for the periodontal ligament injection (36% to 49%).[41,237]

INTRAPULPAL INJECTION

In about 5% to 10% of mandibular posterior teeth with irreversible pulpitis, supplemental injections, even when repeated, do not produce profound anesthesia; pain persists when the pulp is entered. This is an indication for an intrapulpal injection.

The major drawback of the intrapulpal injection technique is that the needle is placed and the injection is made directly into a vital and very sensitive pulp; this injection is often moderately to severely painful.[200] Because endodontics currently offers more successful methods of supplemental anesthesia, the intrapulpal 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 reoccurrence of pain during instrumentation. Yet another disadvantage is that the pulp obviously must be exposed to allow direct

*References 35, 54, 79, 92, 197, 200, 217, and 218-220.

injection. Anesthetic problems frequently occur before exposure while the clinician is still working in dentin.[196,197,200,217]

The advantage of the intrapulpal injection is that it produces profound anesthesia if given under back-pressure.[17,265] 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 intrapulpal anesthesia.[17,265] Depositing the anesthetic solution passively into the chamber is insufficient because the solution will not diffuse throughout the pulp.

MANAGEMENT OF SPECIFIC ENDODONTIC SITUATIONS

Irreversible Pulpitis

When 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. Intraosseous injections are helpful in these cases, and an intrapulpal injection may be used. However, irreversible pulpitis must be differentiated from a symptomatic necrotic tooth with apical pathosis. In the latter condition, it is unknown whether intraosseous injections are effective, and bacteria may be forced into the periradicular tissues by an intrapulpal injection.

Mandibular Teeth

Up until the 1980s, before supplemental intraligamentary and intraosseous injections became popular, clinicians would administer conventional anesthesia. 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.

Integration of the results of many recent clinical trials has changed the paradigm for anesthesia in these cases. Now, after administering conventional anesthesia and observing signs of soft-tissue anesthesia (which is required for a successful supplemental injection), the clinician administers an intraosseous injection. The patient should be informed that the tooth is not as numb 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. For mandibular teeth with irreversible pulpitis, the supplemental intraosseous injection has evolved into a routine second injection, which is made before the clinician places a rubber dam and begins the access opening preparation.

The authors recommend using 1.8 ml of 3% mepivacaine plain (e.g., 3% Carbocaine) for the intraosseous 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.[77,221] 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 intraosseous anesthesia. Individual 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 given, 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 administered, which should be successful.[217] Again, the clinician should make sure that lip numbness 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 intrapulpal injection is given. Prudent practitioners often modify the access preparation under these conditions by creating a straight-line shaft 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 intrapulpal injection with high back-pressure if this technique is indicated.

Maxillary Molar and Premolar Teeth

The initial anesthetic dose of 2% lidocaine with 1:100,000 epinephrine is doubled (to 3.6 ml) for the buccal infiltration.[175] A small amount of anesthetic solution usually is 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.[200] Administration of an intraosseous injection before proceeding with access may prove helpful in anesthetizing these teeth. As an alternative, the clinician can administer the anesthetic and then test the pulp with an EPT or cold refrigerant. If the response is negative, access preparation may proceed. If the response is positive, an intraosseous injection should be given. The clinician must keep in mind that pulp testing may not guarantee pulpal anesthesia in teeth with irreversible pulpitis.[52,200,217] Therefore, if a patient experiences pain despite a negative result on pulp testing, an intraosseous injection should be given.

Infiltration anesthesia does not last as long in maxillary teeth as in mandibular teeth. If the patient experiences pain during the later stages of instrumentation, an additional infiltration injection is necessary. 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.[91]

Maxillary Anterior Teeth

Anesthetic can be administered initially as a labial infiltration. Articaine with 1:100,000 epinephrine may be used.[57] Palatal

anesthesia may be necessary for the rubber dam clamp. Although supplemental anesthesia is not often necessary, the intraosseous injection should be successful when given. 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.

Symptomatic Teeth With Total Pulp Necrosis and Periradicular Radiolucencies

Symptomatic teeth with total pulp necrosis and periradicular radiolucencies are an indicator of pain in the periradicular 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) are given in all situations. For maxillary teeth with no swelling, anesthesia is administered with conventional infiltration or a block. If soft-tissue swelling is present (i.e., cellulitis or abscess), infiltration should be done on either side of the swelling, or a block should 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 treating this region, anecdotal experience suggests that careful consideration of a supplemental injection consisting of an additional palatal infiltration injection may be helpful.

Asymptomatic Teeth With Total Pulp Necrosis and Periradicular Radiolucencies

Asymptomatic teeth with pulp necrosis are the easiest to anesthetize; patient comfort usually is attained without difficulty. Although it may be tempting to proceed without anesthesia, the patient may have pain during instrumentation.

The conventional injections are administered: IAN block and long buccal injection for mandibular teeth, 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 intraosseous injection. An intrapulpal injection should not be given, because bacteria and debris may be forced from the canal into the periradicular tissue. Additional infiltration may be necessary in the maxilla if anesthesia begins to wear off.

Incision and 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 used 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. Except in a few studies,[9,86,235] the computer-assisted injection system caused less pain than the conventional syringe technique for AMSA, palatal injections, and IAN block injections.*

Profound anesthesia usually is difficult to achieve in these cases, 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. (See Chapters 2 and 15 for additional information on incision and drainage procedures.)

Periradicular Surgery

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 is 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 when compared to anesthesia for nonsurgical procedures, because when reflecting a flap and opening into bone, the anesthetic solution is diluted by bleeding and removed by irrigation.[284]

As a prophylactic measure, an intraosseous injection using an anesthetic agent with 1:100,000 epinephrine may be administered at the site—after the above injections and before the surgery. This seems to enhance depth of anesthesia and may provide better hemostasis.

Use of a long-acting anesthetic has been advocated for surgery.[46,230] 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.[40,284]

After periradicular surgery, administration of a long-acting anesthetic has been suggested.[158] However, postsurgical pain is usually not severe and can be managed by analgesics.[171]

*References 81, 107, 192, 195, 214, 272, and 285.

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 IRM or IRM base/glass ionomer core.[161] They found pain was present in 10% of patients at 6 months and in 22% at 12 months. While not ideal, the third option of pulpotomy and temporary restoration may allow the patient time to find the means to finance complete endodontic treatment.[49,130,168]

Pain Reduction in Irreversible Pulpitis When Endodontic Treatment Is Impossible

Endodontic débridement (pulpectomy or pulpotomy) most predictably relieves the pain of irreversible pulpitis.[204] When débridement is not possible, clinicians may prescribe strong analgesics and penicillin in an attempt to relieve the pain. Unfortunately, the pain persists, and penicillin has no effect on the pain of untreated irreversible pulpitis.[190] One clinical trial evaluated pain reduction in untreated irreversible pulpitis by giving an intraosseous injection of Depo-Medrol (a long-acting methylprednisolone).[76] 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

Oral conscious sedation is beneficial in reducing patient's anxiety, but it is not a way to reduce pain during dental treatment.[150] Profound local anesthesia is still required to control the patient's pain.

SUMMARY AND FUTURE DIRECTIONS

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. In contrast, little evidence is available when decisions are based on clinical anecdotes or advertisements. This 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 the combination local anesthetics, possibly 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.[80,289] 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.

The use of adjuvants to shorten the onset or increase the duration of local anesthetics also has been evaluated. The addition of hyaluronic acid significantly increases bupivacaine anesthesia in rats.[253] Tramadol has both a unique central mechanism of action and peripheral anesthetic properties. For patients in pain, 30 to 200 mg of tramadol significantly enhanced postoperative analgesia when combined with 1.5% mepivacaine.[224] Other studies have shown that the addition of 1 mg of morphine to a local anesthetic solution significantly increases postoperative analgesia in human beings and animals.[63,148] Clonidine directly inhibits pulpal pain neurons[99] and enhances anesthesia when combined with a local anesthetic.[111,141] Also, it does not increase the risk of cardiotoxicity seen with local anesthetics.[93] In a clinical model of postsurgical pain, the combination of lidocaine with bupivacaine produced better long-term pain control than lidocaine alone.[145]

Considerable ongoing research is directed at the development of new local anesthetic formulations that allow clinicians to better treat patients in pain.

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CHAPTER 21

Periradicular Surgery

BRADFORD R. JOHNSON, MOHAMED I. FAYAD, and DAVID E. WITHERSPOON

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Nonsurgical endodontic treatment is a highly predictable treatment option in most cases, but surgery may be indicated for teeth with persistent periradicular pathosis unresponsive to nonsurgical approaches. Surgical root canal therapy, including root-end resection, has been practiced since at least the mid-1800s.[199] In 1906, Schamberg[455] described using radiographs to assist diagnosis and using 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.

Over the past 2 decades, 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.[2,72,355] A recent study found that endodontists perform almost 78% of surgical root canal treatments; general dentists and other specialists perform 15.5% and 6.6%, respectively.[229] 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 feel 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.[325,405,420] 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.[146,171]

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.[471] Nonsurgical retreatment, when possible, often is the first choice for attempting to correct obvious deficiencies in the previous treatment. However, microorganisms can survive even in apparently well-treated teeth in dentinal tubules, canal irregularities, deltas, and isthmus areas.[261,290,537] 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.[485] Unlike primary endodontic infections, which predominately are associated with mixed anaerobic microbiota, it has been commonly accepted that treatment failures are more frequently associated with one or two microorganisms (see also Chapter 15).[471] However, recent 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.[446] Fungi and viruses recently have also emerged as potential causes of root canal failure and may play either a primary or secondary role in persistent periradicular pathosis.[350,380,444,545]

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.[469] 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.[157,206,447,474,486] Although the presence of extraradicular microbial colonies has been somewhat controversial, recent studies using DNA-DNA hybridization techniques have confirmed the persistence of microorganisms in the periradicular tissues of some root canal–treated teeth.[178,484]

Overextended filling materials may contribute to treatment failure, presumably as a result of a chronic inflammatory response.[350] Although this is possible and even likely with certain toxic materials (e.g., pastes containing formaldehyde[357]), 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.[69] Some have suggested that overextension of filling materials may contribute to endodontic failure because certain dental materials may induce periodontal ligament (PDL) cell apoptosis.[449] 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.[345] Although relatively uncommon, true periradicular cysts (completely enclosed, epithelium-lined cavities) may not be expected to resolve after nonsurgical treatment.[346] 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.[86] 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 and is discussed in greater detail later in this chapter. Although several promising approaches to the management of vertical root fractures have been proposed,[214,254,481] the prognosis generally is believed to

be poor. Extraction usually is the treatment of choice, especially if appropriate tooth replacement options are available. 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.[75,166] 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.[187,387]

Rationale for Surgical Treatment

Although nonsurgical retreatment generally is believed to be the preferred first approach in the management of persistent apical periodontitis,[74,471,485] periradicular surgery is indicated when nonsurgical retreatment is impractical or unlikely to improve on the previous result (see Chapter 25). 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, suspected vertical root fracture, or when a biopsy is indicated (Figs. 21-1 to 21-4). 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.[217] 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. Case-specific variables and clinical judgment are key elements in the decision-making process; current evidence supports the contention that the prognosis for surgical treatment is approximately the same as that for nonsurgical retreatment.*

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.[255] In a prognosis study of single-tooth dental implants, investigators[183] reported that factors considered significant by health care professionals may not be important to patients.

*References 121, 122, 128, 277, 450, and 547.



FIG. 21-1 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.

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.[55,325,420]

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 (see also Chapter 11).[46,170] 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.[249,367,368] 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.[170] That is, the risks, 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, clinician'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 defini-

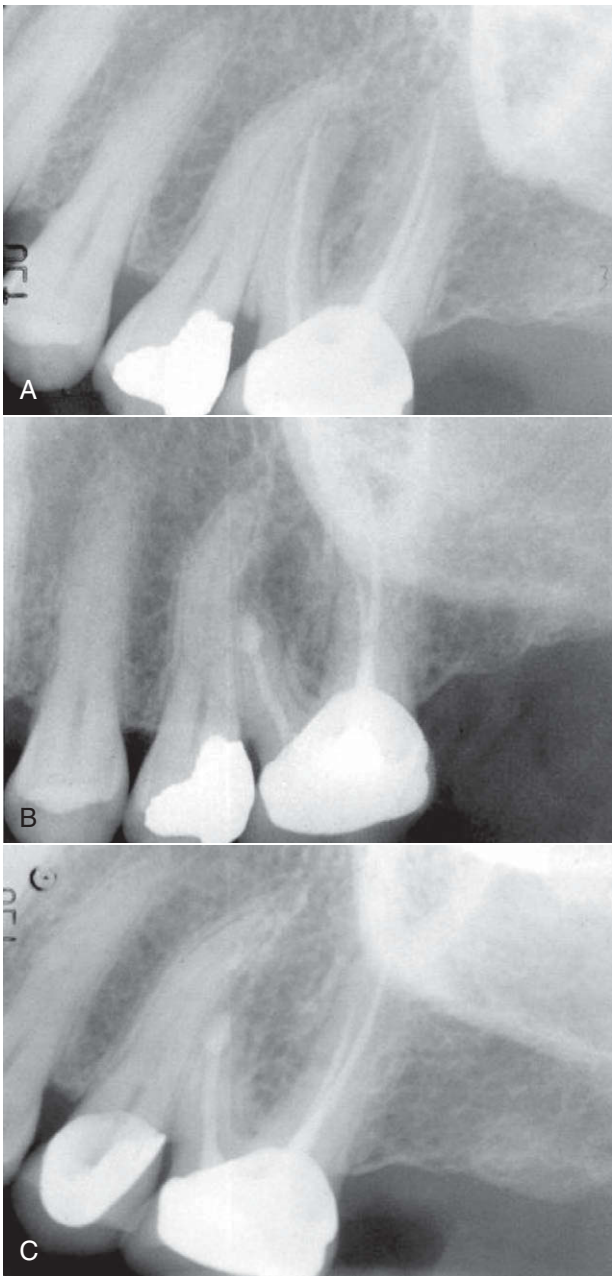


FIG. 21-2 A, Previously treated maxillary first molar with persistent periradicular disease and obvious transportation of the main mesiobuccal (MB-1) canal. Nonsurgical retreatment is unlikely to correct this iatrogenic condition; surgery is the preferred choice. **B,** Immediate postsurgery radiograph. 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 attempt to locate an MB-2 canal.

tion of success varies considerably and is inconsistent from study to study.

One study[65] 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.[285,292] Surgical root canal treatment often is reported to have a much lower success rate than implant placement.[217,521] However, most of these studies would be considered weak by today's evidence-based standards and, more importantly, 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 authors have reported success rates over 90% for surgical root canal treatment.[308,430,521,578]

In broad terms and under ideal conditions, the prognoses for nonsurgical retreatment, surgical treatment, and implant placement would 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.[201]

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 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, but all wounds progress through three broad, overlapping phases in the process of healing: the inflammatory phase, the proliferative phase, and the maturation phase.[227,539] Although these phases can be identified in healing tissue, none of them has a clear beginning or end. In a wound such as an endodontic surgical site that 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 very similar for all tissues.[517] This phase can be broken down further into clot formation, early inflammation, and late inflammation.

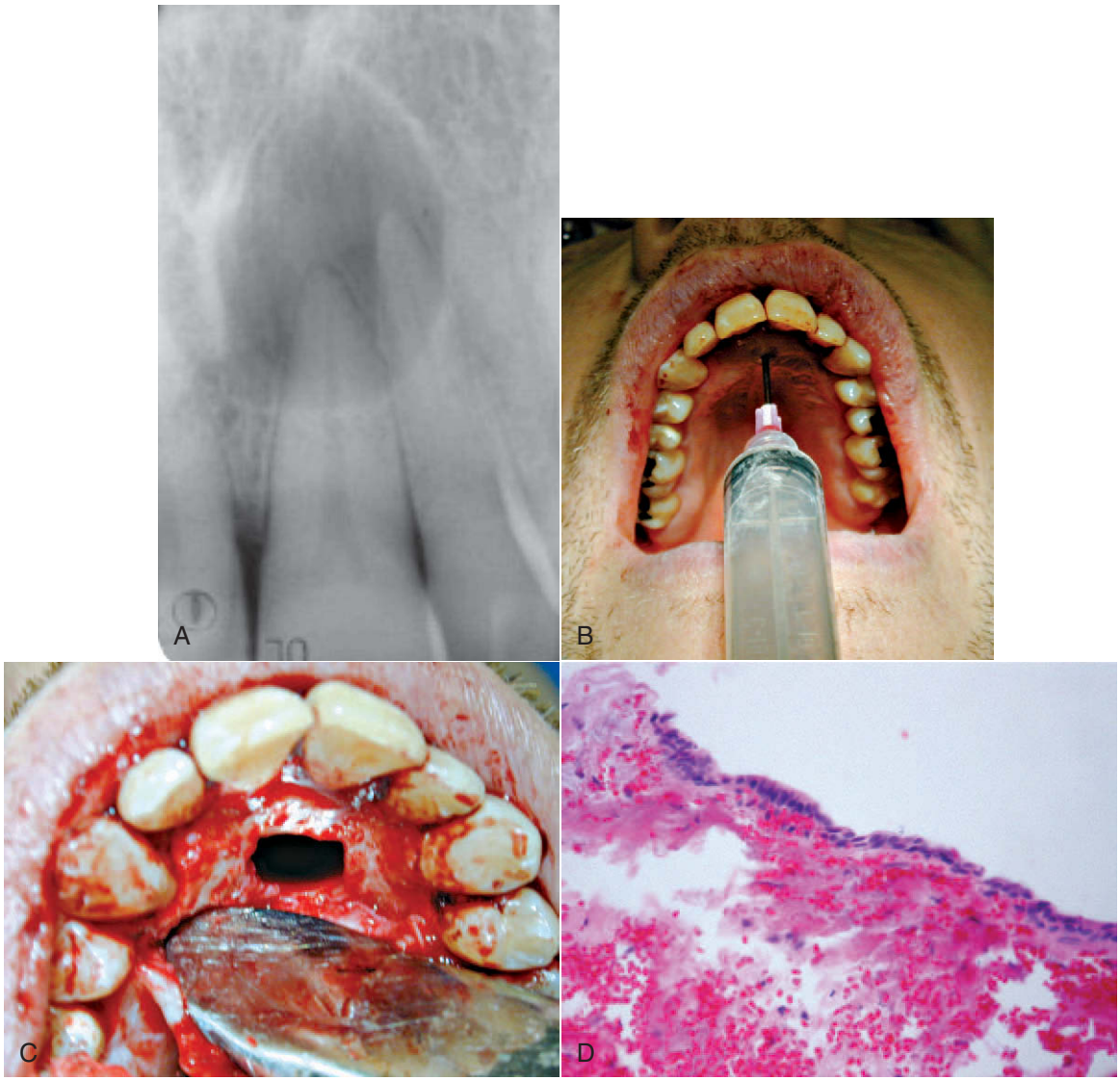


FIG. 21-3 A, Indication for surgery (biopsy): large radiolucent lesion in the area of the maxillary left central and lateral incisors was detected on routine radiographic examination. All anterior teeth responded within normal limits to pulp vitality testing. **B,** After administration of a local anesthetic 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 (×400); the lesion was diagnosed as a nasopalatine duct cyst. *(Courtesy Dr. Vince Penesis.)*

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.[34,234,495] 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 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: *pavementing,* in which red

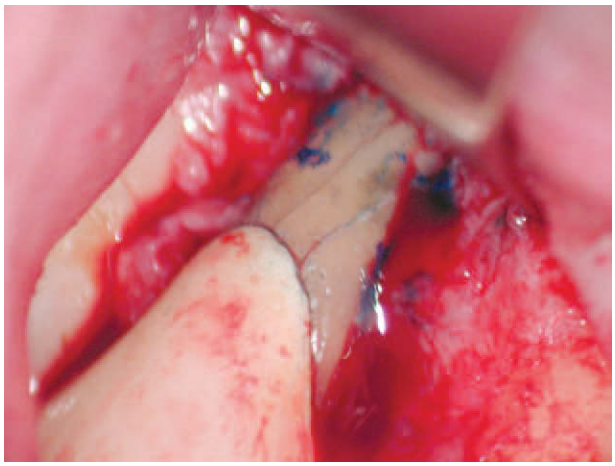


FIG. 21-4 Exploratory surgery was indicated to rule out or confirm a root fracture. Magnification and staining with methylene blue dye confirmed the presence of multiple root fractures. The tooth was subsequently extracted. *(Courtesy Dr. Martin Rogers.)*

blood cells undertake intravascular agglutination, allowing PMNs to adhere to the endothelial cells; *emigration*, in which PMNs actively pass through the vascular wall; and *migration*, in which PMNs use amoeboid motion, under the influence of various chemotactic mediators, to move into the injured tissues.[201]

The principal role of PMNs is wound decontamination by means of phagocytosis of bacteria. The high number of PMNs in the wound site is relatively short lived, dropping 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 chemoattractants in the wound site. Monocytes subsequently evolve into macrophages. Macrophages have a much longer lifespan 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.[105,212] Reduction in the number of macrophages in the wound site delays healing, because the wound does not progress to the next phase. For example, age-related reduction in healing potential appears to be partly due to 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, and two key cell types, fibroblasts and endothelial cells, have a primary role. 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 ectomesenchymal 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]) 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 (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.[104,269,394,490]

A focused type of fibroblast known as a *myofibroblast* plays a significant role in wound contraction, particularly in incisional-type wounds.[299,498] 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.[130,131]

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,[188,272] 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 factors alpha and beta (TGF-α, TGF-β), epidermal growth factor (EGF), interleukin 1 (IL-1), tumor necrosis factor alpha (TNF-α),[138] and

lactic acid.[235,245,479] All of these have been shown to stimulate new vessel development.

Epithelium

The first step in epithelial healing is 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.[211]

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. Degradation of collagen is controlled by a variety of collagenase enzymes, and remodeling results in a gradual reduction in cellularity and vascularity of 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.[131,234]

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.[211]

HARD-TISSUE HEALING: EXCISIONAL DENTOALVEOLAR WOUND

The inflammatory and proliferative phases of hard-tissue healing 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 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 an organizational unit 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.[210] 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 different 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.[210]

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 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.

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.[226,227] 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.[20,21] Some hypothesize[67,68,526,527] 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.[67,68,526,527]

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: TGF-β, bone morphogenetic protein (BMP), PDGF, FGF, and IGF.[94,181,458,488]

About 3 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 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, but the cortical plate has not yet totally reformed. Maturation and remodeling of the osseous tissue continues for several more months.[210]

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.[207]

Cementoblasts: Cementogenesis

During regeneration of the periradicular tissues, cementum forms over the surface of surgically resected root ends.[110] The exact spatial and temporal sequences of events leading to this formation of new cementum remain 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.[25] 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.[133,198] 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.[201,301,302]

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.[314,333] The current preferred term for this condition is *bisphosphonate-associated osteonecrosis* (BON).[8] BON can occur spontaneously but is more commonly associated with dental procedures that involve bone trauma. Patient variables that increase the risk for BON include age (older than 65 years), chronic use of corticosteroids, use of bisphosphonates for more than 2 years, smoking, diabetes, and obesity.[264,316,322,552] The risk for BON 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.[8] The estimated incidence of BON in patients taking oral bisphosphonates ranges from 0 to 1 in 2260 cases, although dental extractions may quadruple the risk of developing BON.[8,322] The majority of cases have been reported in patients taking IV bisphosphonates (e.g., zoledronic acid and pamidronate). Approximately 20% of patients taking IV bisphosphonates may develop BON.[8,322]

Conservative and timely management of apical periodontitis is essential to reduce the risk of BON in patients taking bisphosphonates. Since periapical pathosis may exacerbate or increase risk,[8] the no-treatment option is not a viable choice. Treatment options and the potential risk for BON should be thoroughly discussed with all patients who are taking bisphosphonates. 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 managing persistent periradicular inflammation are surgical root canal therapy or extraction, the question of which option is more or less likely to lead to BON 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.[8] Prophylactic antibiotics are generally not indicated unless the patient is at risk for developing an infection independent of bisphosphonate use.[8] Since bisphosphonates have a substantial half-life when incorporated into bone, their discontinuation prior to dental treatment is unlikely to provide any measurable benefit and may place the patient at greater risk for complications the drug is intended to prevent.

Clinical observations[315] have suggested the possibility of predicting which patients may be at greater risk for BON, based on serum levels of C-terminal cross-linking telopeptide of type I collagen (CTX). This observation may provide a valuable tool for future risk assessment but requires additional validation prior to widespread acceptance.[8] The most current treatment recommendations for patients taking bisphosphonates can be accessed online at various dental and medical specialty websites, including: www.ada.org/prof/resources/topics/osteonecrosis.asp.

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 lifespan 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.[258] 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.[428,550,551]

Nonsteroidal Antiinflammatory Drugs

Bone homeostasis is regulated by many factors, including prostaglandins (PGs).[253] PGs are important to both normal and pathologic bone turnover and modulate osteoblast proliferation and differentiated functions.[407,408] 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.[258] Nonsteroidal antiinflammatory drugs (NSAIDs) inhibit the enzyme, cyclooxygenase (COX), which is involved in the synthesis of PGs. This is the same mechanism by which NSAIDs control pain. By inhibiting the COX enzymes and the subsequent production of prostaglandins, NSAIDs accomplish the desired antiinflammatory 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.[222] These drugs also have been shown to delay fracture healing and adversely affect bone formation in animals and humans.[16,182] In one study, the use of NSAIDs reduced the amount of bony ingrowth into an orthopedic implant.[208]

In other studies, NSAIDs reduced bone resorption associated with experimentally induced periapical lesions,[19,370] and systemic NSAIDs may play a positive role in maintaining bone height around endosseous titanium dental implants.[244] Additional research is needed to clarify the specific influence of NSAIDs on healing after periradicular surgery.

Cyclooxygenase-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.[572] Additional studies have shown a persistent delay in healing with COX-2 NSAIDs[192,193]; however, this difference in effect was not apparent clinically in a study examining the healing of spinal fusion wounds.[173]

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 4 and 25). 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 but 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 alone is not a reliable predictor of operative risk and should only be used as a general guide.[191] Even experienced anesthesiologists exhibit differences of opinion when using the ASA system to classify patients.[215,376] The patient's psychologic status and anticipated procedural stress should be considered in addition to 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.

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 19 and 20). The use of local anesthetics with vasoconstrictors was addressed in the recently revised seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7).[218] Most experts feel that 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 all patients except those with severe cardiovascular disease or other specific risk factors.[38,218,563] 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.[294] 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.[557] 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 potential benefits.[557] Patients with prosthetic joints may also be at increased risk for developing a hematogenous joint infection after oral surgery. Although the need for prophylactic antibiotics in this group of patients is somewhat controversial, consultation with the patient's orthopedic surgeon is advised; current recommendations include antibiotic coverage for many of these patients.[1] Selection and dosage of the antibiotic may be different from the commonly accepted regimen for preventing bacterial endocarditis, since different microorganisms are more commonly associated with late joint infections.

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.[9,85,242,460] 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, investigators[443] 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 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, so 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.[84,538] In a review prepared for the American Dental Association Council on Scientific Affairs and Division of Science, Jeske and Suchko[247] 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. 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.

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.[29] At low-dose therapeutic levels (less than 100 mg per day), aspirin may increase bleeding time and complicate surgical procedures. However, one study[29] concluded that low-dose aspirin therapy (less than 100 mg per day) 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.[366] In a recent survey of surgical patients, approximately a third reported using a nonprescription medication that might inhibit coagulation or interact with anesthetics.[365] In particular, ginkgo biloba, ginger, garlic, ginseng, feverfew, and vitamin E inhibit platelet aggregation and can increase the risk of bleeding.[93] 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 U.S. 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 between the mandibular canal, the mental foramen, and the root apices of mandibular teeth has been well studied, but 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 access to the mandibular posterior teeth.[288] 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[113,337]; however, this varies considerably, and the practitioner must examine each patient carefully to determine its location (Figs. 21-5 and 21-6). Vertical location of the mental foramen may vary even more than horizontal location. One investigator[337] 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

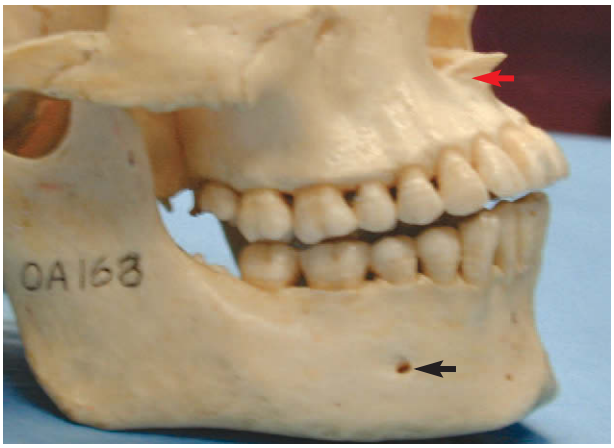


FIG. 21-5 Lateral view of the skull showing anterior nasal spine and proximity of the maxillary anterior root apices to the floor of the nose *(red arrow)*, as well as the typical location of the mental foramen *(black arrow)*.



FIG. 21-6 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.

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.[336] 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.

Periapical radiographs taken from two vertical angulations, 0 degrees (parallel technique) and −20 degrees, may help determine the buccolingual position of the canal in the same way horizontally angled radiographs may differentiate between buccal and lingual roots.[295] That is, if the apparent distance between the root apices and the mandibular canal is greater on the radiograph taken at the −20-degree angulation, the mandibular canal is positioned in the lingual half of the mandible. This is a useful rule to understand but may have limited practical application because visualizing the mandibular canal is difficult in angled radiographs. 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.[528]

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.[129] 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.[129,295] 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. Perforation of the sinus during surgery is fairly common, with a reported incidence of about 10% to 50% of cases.[149,168,438] 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.[213] 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.[213] In a report of 146 cases of sinus exposure during periradicular surgery, researchers[548] 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.[50,149,548] 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, and 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 to prevent inadvertent displacement into the sinus. A suture can be placed through the packing material to prevent displacement and aid retrieval. Researchers[246] suggest 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. One surgeon[541] 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 × 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

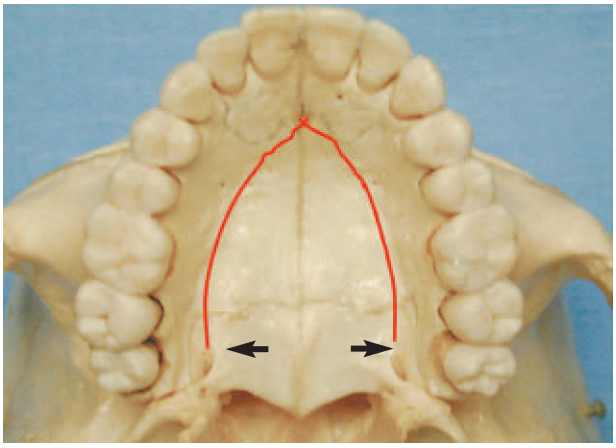


FIG. 21-7 Palatal view showing the position of the greater palatine foramen *(arrows)*. The approximate location of the anterior palatine artery is marked in red.

palatal root end without direct invasion of the sinus.[17] 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.

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. 21-7). 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 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 Fig. 21-5, 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. The average maxillary canine is about 26 mm long and usually presents no difficulty for surgical access, but the combination of a shallow vestibule and 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 VOLUMETRIC TOMOGRAPHY

Radiographic examination is an essential component in all aspects of endodontic treatment, from diagnosis and treatment planning to assessing outcome. Information gained from conventional films and digital periapical radiographs is limited by the fact that the three-dimensional (3D) anatomy of the area is compressed into a two-dimensional (2D) image. As a result of superimposition, periapical radiographs reveal limited aspects of the 3D anatomy. In addition, there may also be geometric distortion of the anatomic structures imaged. These problems may be overcome using small-volume cone-beam volumetric tomography (CBVT) imaging techniques that can produce 3D images of individual teeth and the surrounding tissues. CBVT may be particularly useful in diagnosis and treatment planning for periradicular surgery[108,155,236,300] (Fig. 21-8). The term *cone-beam computed tomography* (CBCT) is often used interchangeably with CBVT.

Differences Between CT and CBVT Imaging

The benefits of 3D medical computed 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.[482] The slices of data are then "stacked" and can be reformatted to obtain 3D 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 recent cone-beam innovations in CBVT technology.

In 2000, the FDA approved the first CBVT 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 CBVT 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, enabling objects within the volume to be accurately measured in different directions. Unlike the CBVT voxel, a medical CT voxel is not a perfect cube, and measurements made in multiple planes are not accurate. In addition to increased accuracy

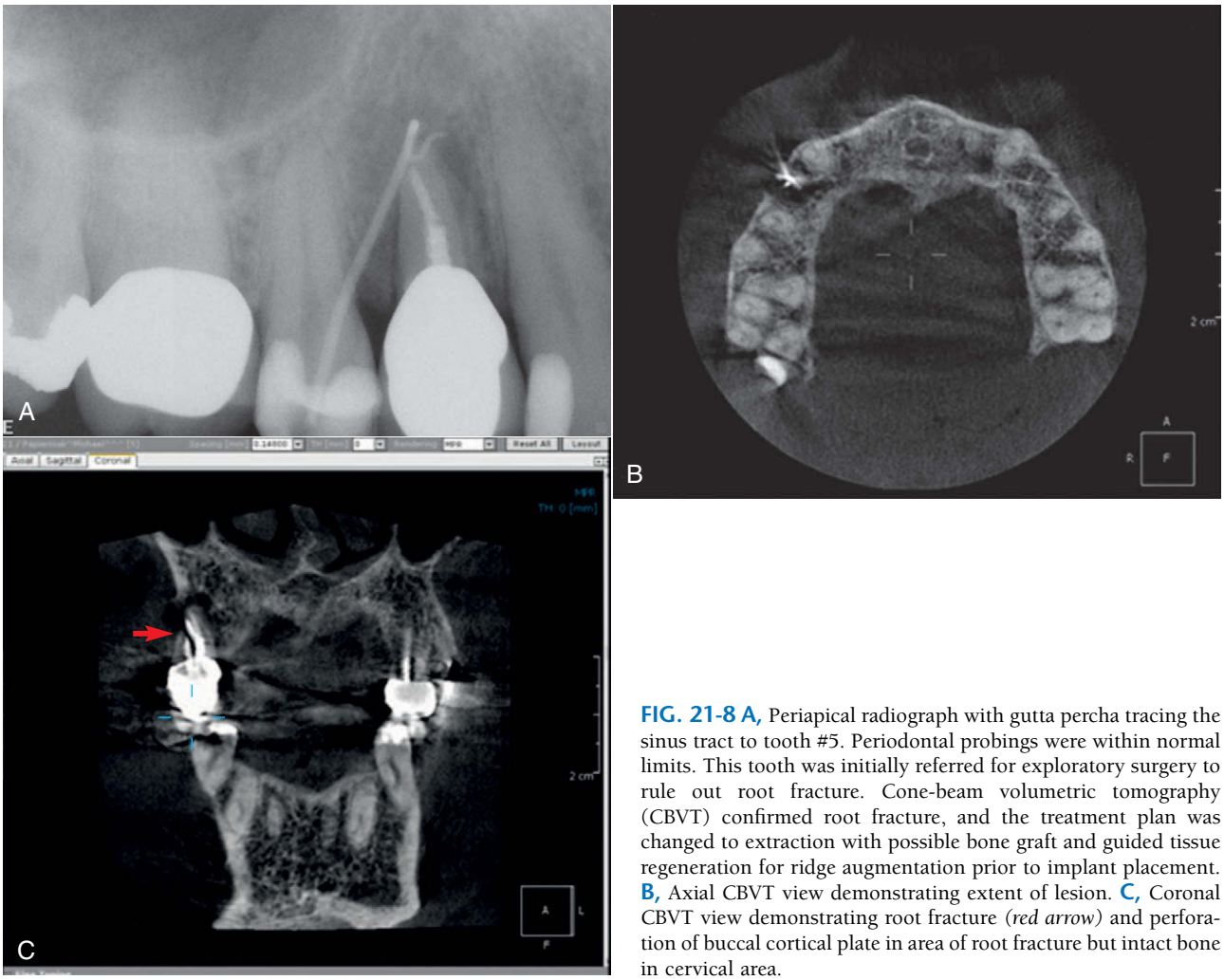


FIG. 21-8 A, Periapical radiograph with gutta percha tracing the sinus tract to tooth #5. Periodontal probings were within normal limits. This tooth was initially referred for exploratory surgery to rule out root fracture. Cone-beam volumetric tomography (CBVT) confirmed root fracture, and the treatment plan was changed to extraction with possible bone graft and guided tissue regeneration for ridge augmentation prior to implant placement. **B,** Axial CBVT view demonstrating extent of lesion. **C,** Coronal CBVT view demonstrating root fracture *(red arrow)* and perforation of buccal cortical plate in area of root fracture but intact bone in cervical area.

and higher resolution, CBVT offers significant scan-time reduction, radiation dose reduction, and reduced cost for the patient.[454,564,575] CBVT systems can be classified into two categories: limited (dental or regional) CBVT or full (ortho or facial) CBVT. The field of view (FOV) of limited CBVT ranges in diameter from 40 to 100 mm, whereas the FOV of full CBVT ranges from 100 to 200 mm.[108] Another difference between limited and full CBVT is that a voxel is generally smaller for the limited version (0.1 to 0.2 mm versus 0.3 to 0.4 mm), so limited CBVT systems offer higher resolution and are better suited for endodontic applications. Limited volume CBVT scanners capture small volumes of data that can include just two or three individual teeth. For example, the 3D Accuitomo (J. Morita Corporation, Osaka, Japan) captures a 30 mm high × 40 mm diameter cylindrical volume of data, similar in overall height and width to a periapical radiograph. Recently, the Planmeca Promax 3D (Planmeca, Helsinki, Finland) has been introduced to the dental market. This CBVT scanner, like the 3D Accuitomo, can also produce images of small specific areas of interest, with the smallest FOV being a 50 mm high × 30 mm diameter cylinder.

The most important and clinically useful feature of CBVT technology is the highly sophisticated software that allows the huge volume of data collected to be reconstructed. Tomographic slices as thin as one voxel thick may be displayed in a number of different 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 superimposition of the surrounding structures makes CBVT superior to conventional periapical radiography.[297]

Scan times are typically 10 to 40 seconds, although the actual exposure time is significantly less (2 to 5 seconds) because scans involve a number (up to 360) of separate small, individual exposures rather than one continuous exposure. With medical CT scanners, scanning and exposure times for the skull can be significantly longer. Most CBVT 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 2.0 mm thick. Comparing the radiation dose of different CBVT 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 CBVT 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.[379] The higher effective dose from particular models of CBVT scanners is in part due to the larger size of the FOV used and the type of image receptor employed. The small volume scanners, such as the 3D Accuitomo and Planmeca Promax 3D, capture information from a small region of the jaw, approximately the same area as a periapical radiograph. The effective dose of the 3D Accuitomo has been reported to be in the same range as two to three 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.[118,184] If multiple teeth in different quadrants require endodontic evaluation or treatment, a large-volume CBVT scan with a large FOV may be more appropriate. If endodontic information is required from multiple teeth in one jaw, a large-volume CBVT scan with the FOV 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 CBVT scan by up to 65%.[303]

Potential Applications of CBVT in the Management of Endodontic Posttreatment Disease

Clinical radiographic examinations are usually limited to 2D views captured using radiographic film or digital sensors. Crucial information related to the true 3D 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 CBVT is the accuracy of measurements in all dimensions.[313] 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. 21-9). When compared to conventional periapical radiographs, CBVT enables clinicians to detect changes in apical bone density at an earlier stage and therefore has the potential to detect previously undiagnosed periradicular pathoses.[155,297,352] CBVT may also prove to be a useful tool for noninvasive differentiation of periapical cysts and granulomas,[468] 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. 21-10). Investigators 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.[528] It is likely that similar results could be achieved with CBCT using considerably less radiation. Researchers concluded that CBVT may play an important role in periapical microsurgery of palatal roots of maxillary first molars.[423] The distance between the cortical plate and the palatal root apex could be measured using CBVT, 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.[352] Root morphology (shape, size, curvature, number of canals) can be visualized in three dimensions. Unidentified (and untreated) canals in root-filled teeth can be routinely visualized in axial slices. Recently CBVT has been used to determine the location and extent of invasive external root resorptive defects.[379]

One of the most useful potential applications of CBVT may be the assessment of endodontic treatment outcomes, both nonsurgical and surgical. When compared to 2D film and digital images, CBVT is dimensionally precise and can detect smaller changes in bone density.[313]

It is worth remembering that CBVT 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 CBVT use are developed. CBVT 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 11 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, since 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 medicolegal standpoint.

Premedication: NSAIDs, Antibiotics, Chlorhexidine, and Conscious Sedation

Administration of an NSAID, either before or up to 30 minutes after surgery, enhances postoperative analgesia.[473] NSAIDs

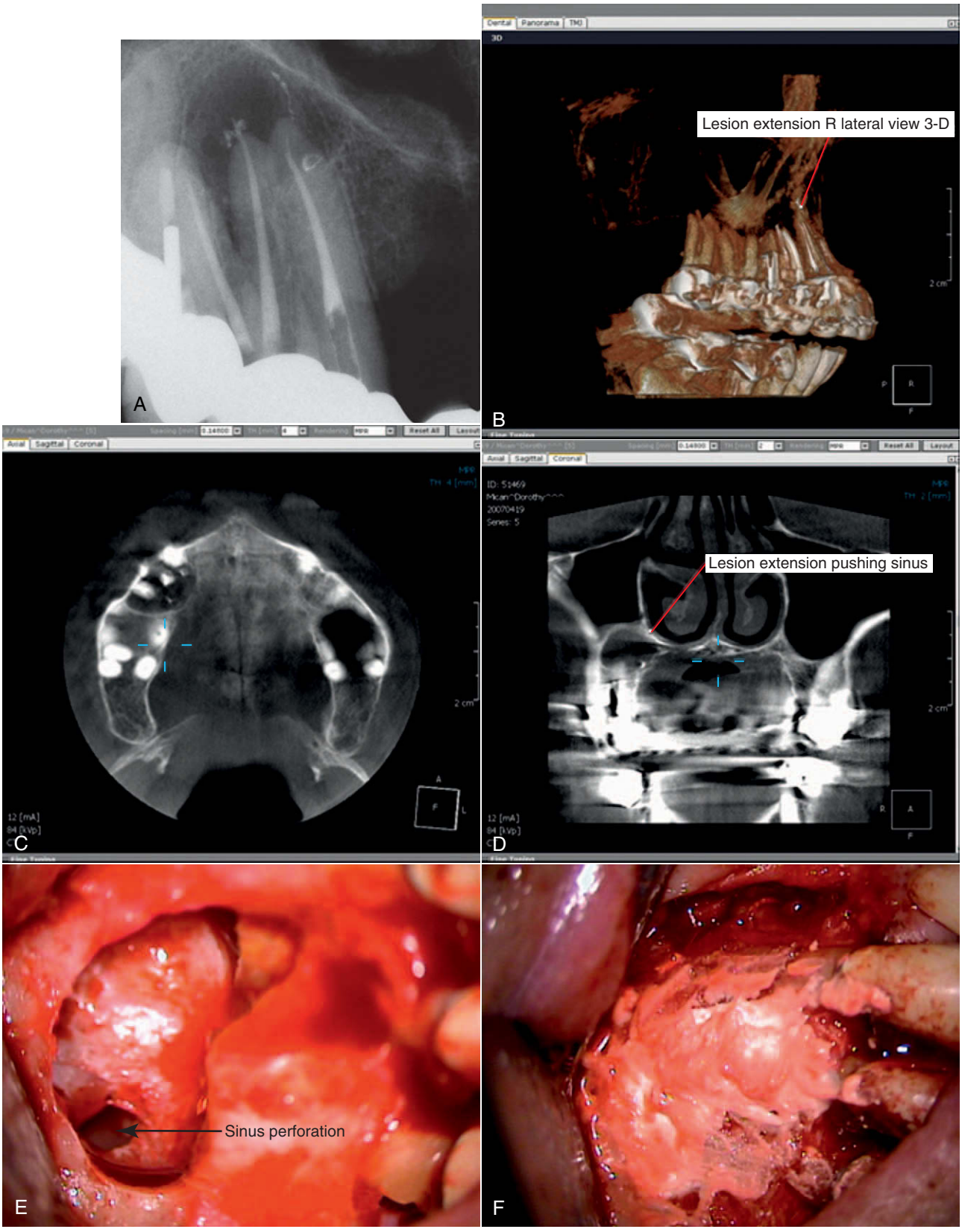


FIG. 21-9 A, Preoperative periapical radiograph showing large radiolucency associated with teeth #4, #5, and #6. **B,** Cone-beam volumetric tomography (CBVT) reconstruction (lateral view). Rotation of the 3D volume on a computer monitor demonstrated that intact bone was present around the apex of tooth #6, and the lesion was associated only with #4 and #5 (could not be detected with multiple-angled periapical radiographs). **C,** Axial CBVT view demonstrating facial-palatal extent of the lesion. **D,** Coronal CBVT view showing displacement of the sinus membrane (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.

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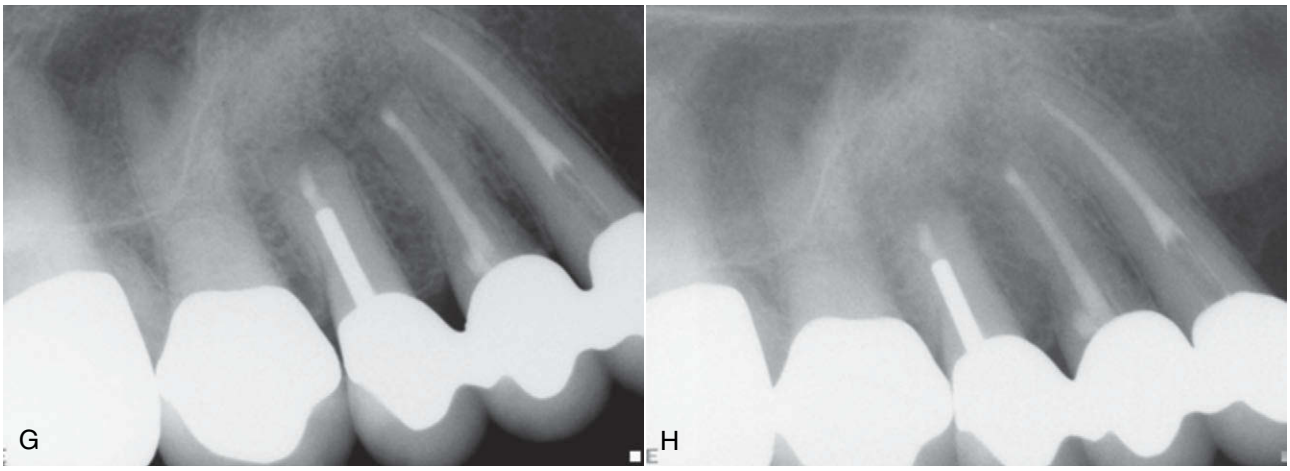


FIG. 21-9, cont'd G, Immediate postoperative radiograph. **H,** Two-year follow-up radiograph demonstrating good healing.

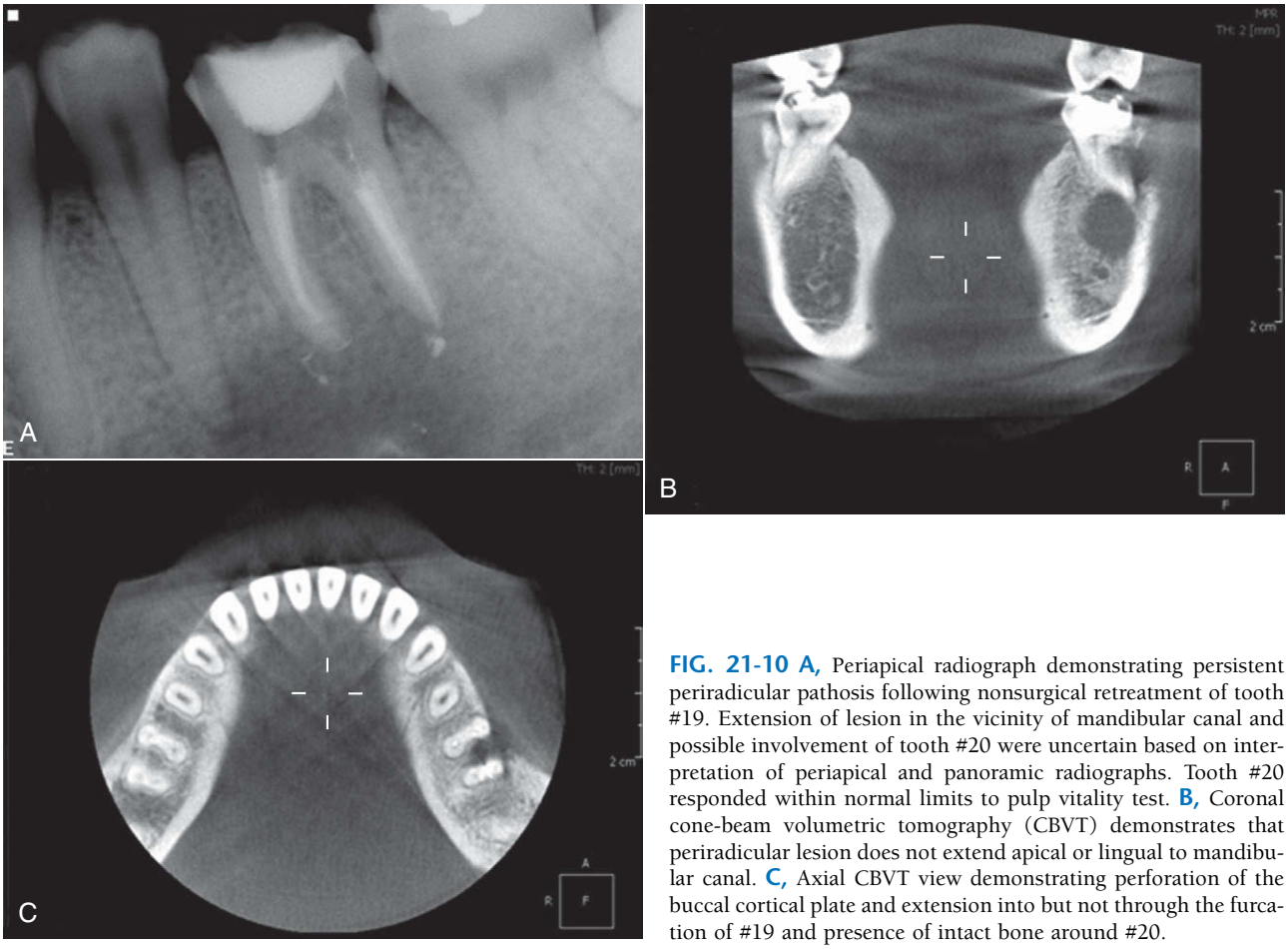


FIG. 21-10 A, Periapical radiograph demonstrating persistent periradicular pathosis following nonsurgical retreatment of tooth #19. Extension of lesion in the vicinity of mandibular canal and possible involvement of tooth #20 were uncertain based on interpretation of periapical and panoramic radiographs. Tooth #20 responded within normal limits to pulp vitality test. **B,** Coronal cone-beam volumetric tomography (CBVT) demonstrates that periradicular lesion does not extend apical or lingual to mandibular canal. **C,** Axial CBVT 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.

generally have proved more effective in the management of postoperative oral surgery pain than placebo or acetaminophen and codeine combinations.[10,41,136] The combination of preoperative administration of an NSAID and use of a long-acting local anesthetic may be particularly helpful for reducing postoperative pain.[137] 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.[328] 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 19 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.[12] For most patients, the risks of indiscriminate antibiotic therapy are believed to be greater than the potential benefits.[500] The incidence of infection after oral surgery in healthy patients is very low. One investigator[358] reported that only 1% of patients developed infections after third molar extractions. 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.[154] However, the use of prophylactic antibiotics for more invasive procedures, such as orthognathic surgery, has significantly reduced the risk of postoperative infection and complications.[139] 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,[75,166] 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 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.[8,265,520] 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,[66,567] although the evidence in this area is conflicting. Postoperative use of chlorhexidine mouth rinse can reduce bacterial growth on sutures and wound margins[374] but may interfere with fibroblast reattachment to the root surface.[15] 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.[80] As with all orally administered drugs, the dosage cannot be closely titrated, so 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.

INSTRUMENTS AND OPERATORY SETUP

The development of microsurgical techniques and new materials has changed the typical surgical instrument tray dramatically over the past 15 years. 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. Fig. 21-11 shows a typical basic surgery tray arrangement. This setup is not a definitive guide to the 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. 21-12 to 21-23) to achieve excellent results.

Periradicular surgery can be performed without the benefit of enhanced magnification and illumination, but those who use microscopes, endoscopes, and orascopes report dramatically improved visualization and control of the surgical site.[39,89,266,332] The authors are unaware of any randomized controlled clinical trials comparing surgical outcomes with and without the use of magnification and illumination. However, current 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. Surgeons accustomed to using microscopes, endoscopes, orascopes, and microsurgical instruments find it extremely difficult to go back to "dark field" surgery.

LOCAL ANESTHESIA FOR SURGERY

Local anesthesia for surgical root canal procedures differs from that for nonsurgical root canal treatment, primarily in the need

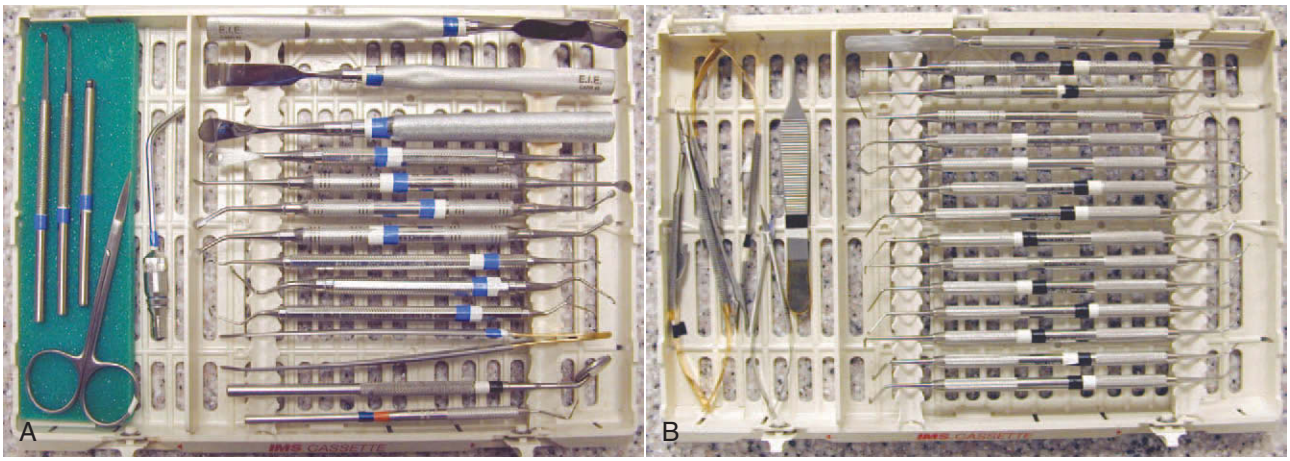


FIG. 21-11 A, Basic tray setup for initial surgical access. Surgical instruments shown are distributed by Hu-Friedy ([HF] Chicago), CK Dental Specialties ([CKDS] Orange, CA), EIE ([EIE]), and G. Hartzell & Son ([GHS]). *Left to right (left section of tray):* Small round micromirror (CKDS); medium oval 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 SuperEBA Placing & Plugging instrument (MRFR [HF]); left SuperEBA 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).

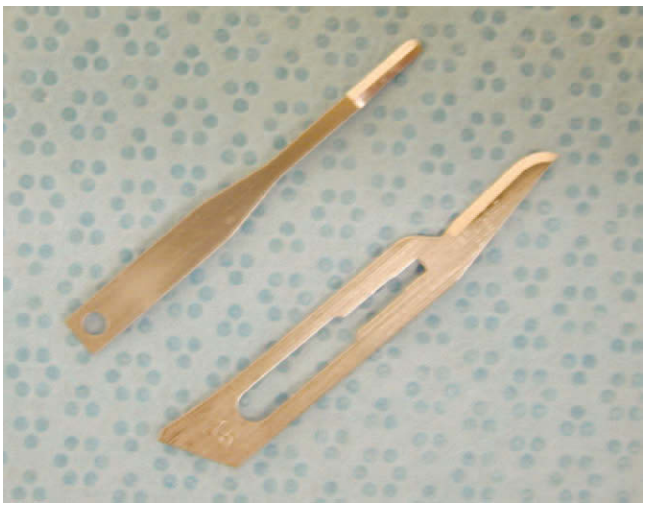


FIG. 21-12 Comparison of microsurgical scalpel *(top)* to #15C surgical blade. Microsurgical scalpels are particularly useful for the intrasulcular incision and for delicate dissection of the interproximal papillae.

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 25) 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.[266] The local anesthetic is first slowly deposited in the buccal root apex 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,[119,194] but use of a local anesthetic with 1:200,000 epinephrine may result in greater blood loss during surgery.[221,472] 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

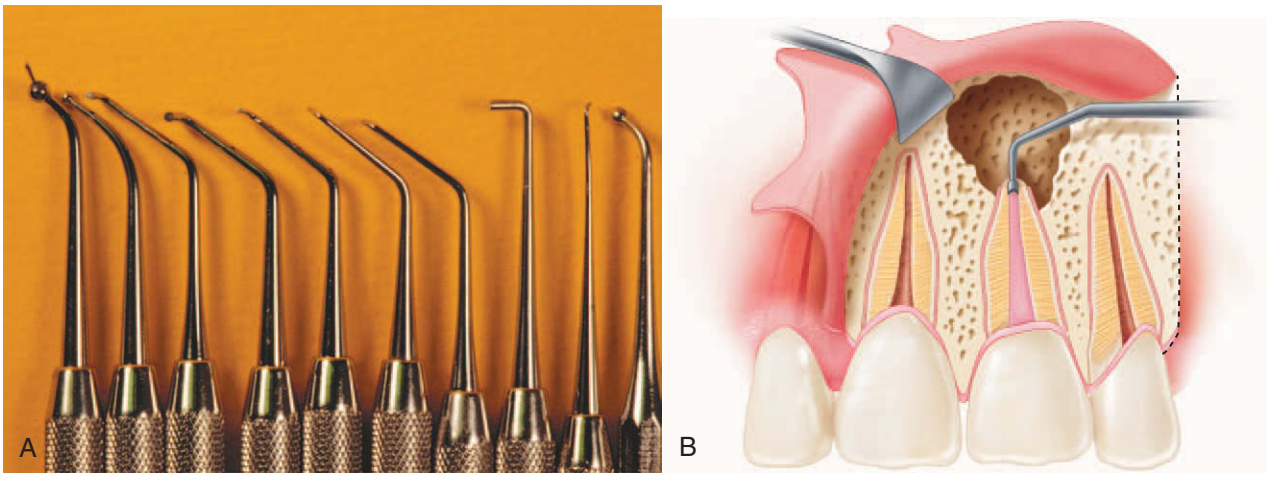


FIG. 21-13 A, Microcondensers in assorted shapes and sizes for root-end filling. **B,** The microcondenser should be selected to fit the root-end preparation.



FIG. 21-14 Comparison of standard #5 mouth mirror *(top)* to diamond-coated micromirrors (CK Dental Specialties).

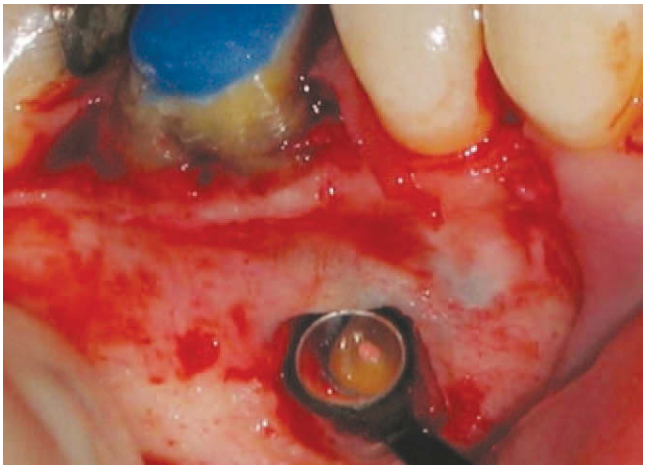


FIG. 21-15 Micromirror used to inspect resected mesial root of a mandibular first molar.



FIG. 21-16 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).

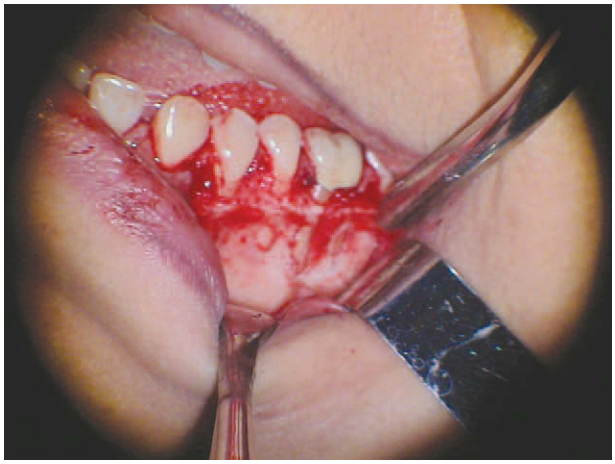


FIG. 21-17 Retractors positioned to expose the surgical site and protect adjacent soft tissues from injury. Care must be taken to rest the retractors only on bone, not on the reflected soft-tissue flap or on the neurovascular bundle as it exits the mental foramen.



FIG. 21-18 Teflon sleeve and plugger specially designed for placement of MTA (DENTSPLY Tulsa Dental).

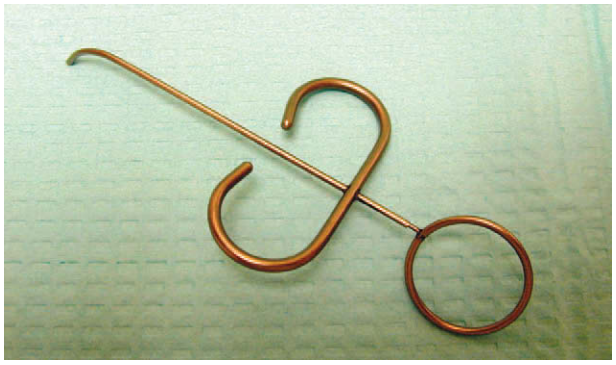


FIG. 21-19 Messing gun–type syringe (CK Dental Specialties) can be used for placement of various root-end filling materials.

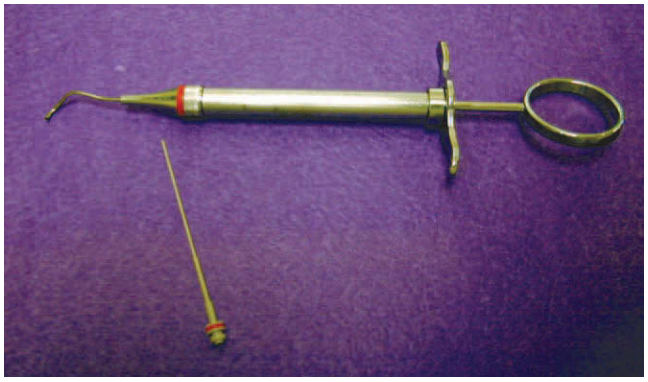


FIG. 21-20 Another delivery system designed specifically for MTA placement (Roydent). Kit includes a variety of tips for use in different areas of the mouth and a single-use Teflon plunger.

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

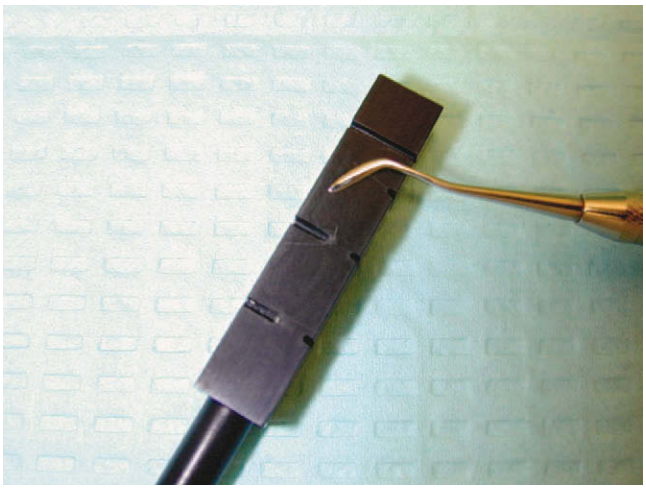


FIG. 21-21 Hard plastic block with notches of varying shapes and sizes (G. Hartzell & Son). MTA is mixed on a glass slab to the consistency of wet sand and then packed into a notch. The applicator instrument is used to transfer the preformed plug of MTA from the block to the root end.

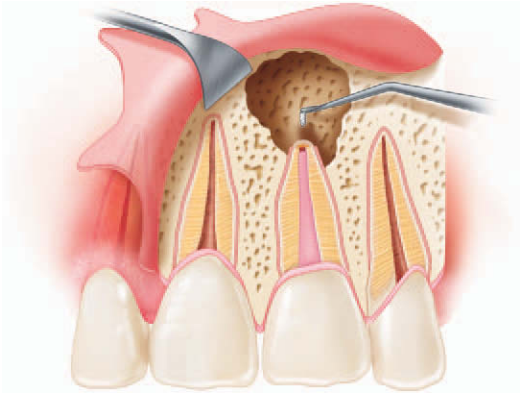


FIG. 21-22 Placement of a root-end filling.

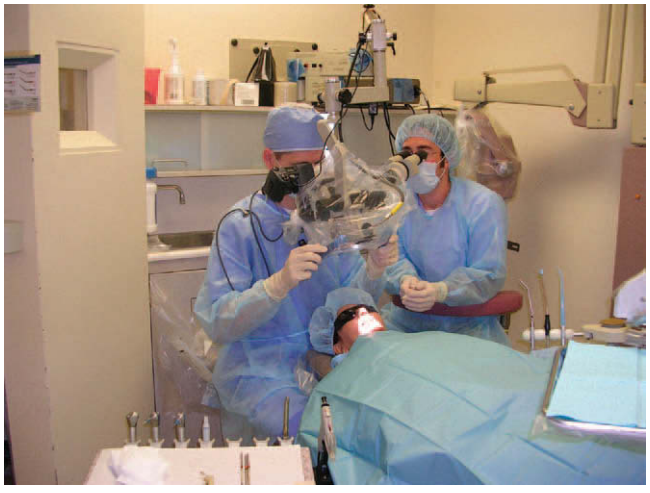


FIG. 21-23 Surgeon, assistant, and patient positioned for initiation of surgery. The patient should be given tinted goggles or some other form of eye protection before the procedure is begun.

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 endpoint (i.e., replacement of the reflected tissues) is essential for designing each phase of the surgery. Visualization of a 3D 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 the 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 3D 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.[163] A vertical (rather than an angled) releasing incision severs fewer vessels,[306] reducing the possibility of hemorrhage. Also, the blood supply to the tissue coronal to the incision is not compromised,[459] 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.



FIG. 21-24 Intrasulcular incision with two vertical releasing incisions (rectangular flap).

Vertical Incision

The general principles for placement of a vertical relieving incision are:

1. The incision should be made parallel to the supraperiosteal vessels in the attached gingiva and submucosa (Fig. 21-24).
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.
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. The entire dental papilla is completely mobilized.
- An intrasulcular incision that excludes the dental papilla (papillary-based incision, Fig. 21-25). 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).[304] With this technique, at least 2 mm of attached gingiva must be retained to prevent mucogingival degeneration.[279] 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.[11,189,323,534] It is widest over the central and lateral incisors, narrows over the canine and the first premolar,



FIG. 21-25 Papillary-based incision with one vertical releasing incision.



FIG. 21-26 Intrasulcular incision with one vertical releasing incision (triangular flap).

and then widens over the second premolar and the first molar. These variances were similar for both the maxilla and mandible.[491] Overall, this incision technique has a narrow margin of safety. It generally is recommended for use in the maxilla, especially where the esthetics 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.[494,495] These researchers found that the papillary-based incision resulted in rapid recession-free healing. In contrast, complete mobilization of the papilla led to a marked loss of papillary height. The authors suggested that use of the papillary-based incision in aesthetically sensitive regions could help prevent papillary recession and surgical cleft, or double papilla.

Flap Design

Combinations of vertical and horizontal incisions are used to achieve various flap designs. 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 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. 21-26 and 21-27)
   - 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. 21-28 and 21-29)
   - 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 of the submucosa and the attached gingiva (Figs. 21-30 and 21-31). 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 downgrowth of epithelium and loss of soft-tissue attachment.[211] 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.[200]



FIG. 21-27 Reflection of the triangular flap to expose the root-end area.

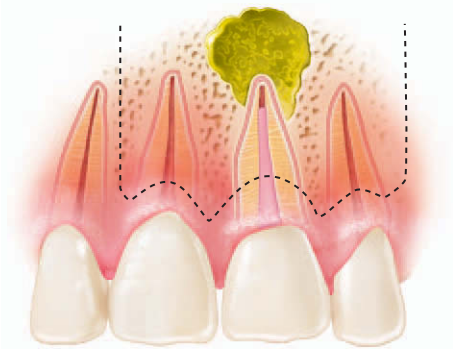


FIG. 21-28 Submarginal (Ochsenbein-Luebke) flap.

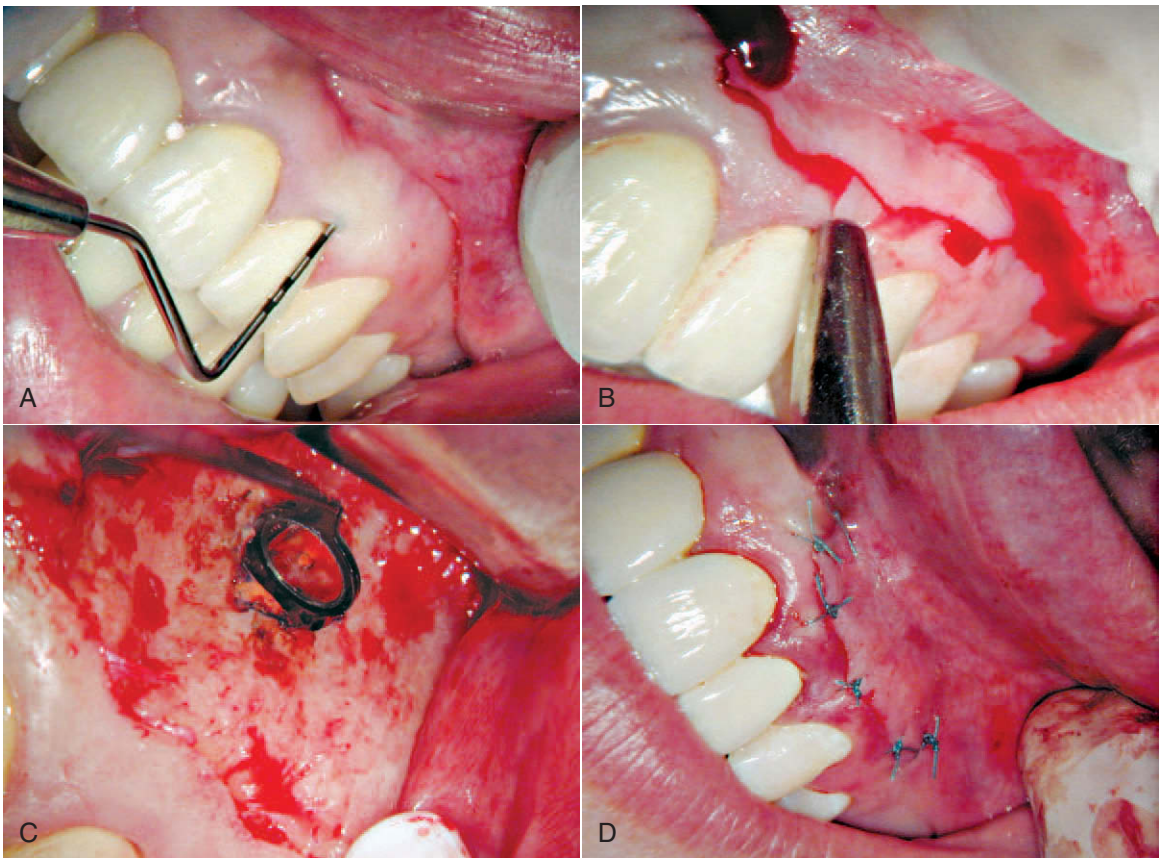


FIG. 21-29 Clinical case involving a submarginal incision and flap. **A,** Periodontal probing of entire area was performed before this type of flap was selected and incision started. A submarginal incision often is used in esthetic anterior areas of the mouth where postoperative gingival recession might expose crown margins. **B,** Incision should be at least 2 mm apical to depth of sulcus. **C,** With flap reflected, osteotomy and root-end resection were performed. Methylene blue dye was placed to mark outline of root end and 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.)*

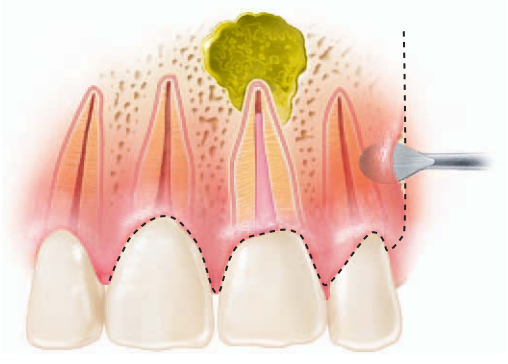


FIG. 21-30 Elevator placed in the vertical incision for the first step in undermining flap reflection.

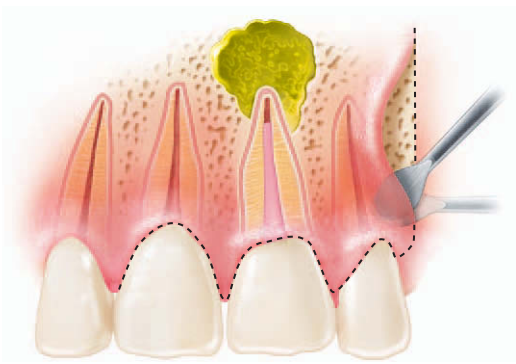


FIG. 21-31 Continuation of reflection of full-thickness flap.

The underlying bone of the cortical plate is irregular, and it is critical to avoid damaging the fragile tissues during elevation.

The clinician should take great care not to slip during the tissue reflection process; this can be done 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.[210,211]

The general principles of retraction are (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; 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: healthy hard tissue must be preserved, and heat generation during the process must be minimized.

Temperature increases above normal body temperature in osseous tissues are detrimental. Heating osseous tissue to 117° F to 122° F (47° C to 50° C) for 1 minute significantly reduces bone formation and is associated with irreversible cellular damage and fatty cell infiltration.[153,320] Two critical factors determine the degree of injury: how high the temperature is increased 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.[403,404] 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.[150-153] 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.[150-153]

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.[493] 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.[81,107,204,320,341] 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.



FIG. 21-32 Surgical handpiece with 45-degree angle head and rear air exhaust (Impact Air 45).

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.[81,555]

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[260]; histologically, healing can be delayed up to 3 weeks.[160] 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[493]; hence the recommendation for a gentle brushstroke technique.[200] 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. 21-32).

PERIRADICULAR CURETTAGE AND BIOPSY

Most periradicular lesions originate in the pulp and can be classified histopathologically as granulomas or cysts.* Histologically, such lesions consist mainly of granulation tissue associated with angiogenesis, fibroblasts, connective tissue fibers, and inflammatory cells. Foreign material, cholesterol clefts,[345,351] and stimulated strands of epithelium also may be present. The stimulated epithelium can form into a stratified, squamous epithelium–lined cystic cavity.[347] These periradicular lesions (granulomas and cysts) are inflammatory lesions that develop in response to irritation caused by intraradicular

*References 52, 57, 275, 278, 280, 340, 348, 363, and 467.

and extraradicular microorganisms associated with the root canal system[344,349] or by foreign materials forced into the periradicular tissues.[569]

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.[289] 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 histopathologic assessment of all tissues removed from the body cannot be understated. 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.[13,116,176,364,390]

The technical aspect of removing soft tissue from the bony crypt varies among surgeons and clinical settings. A large array of bone and periodontal curettes is 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.[200] 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, 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. Further investigation is warranted 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.[148] Anxiety and stress can be alleviated with planning, sedation, and profound local anesthesia.

Local Hemostatic Agents

Collagen-Based Materials

A wide variety of 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,[256,257] activation of factor XII (Hageman factor),[317,318] 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.[203] It does not increase the incidence of infection and only slightly delays early bone repair.[233] Osseous regeneration in the presence of collagen typically proceeds uneventfully, without a foreign body reaction.[159]

Collagen-based materials can be difficult to apply to the bony crypt because they adhere to wet surfaces, particularly instruments and gloves.[448] Several collagen-based products are commercially available. They include CollaCote (Integra Life Sciences, Plainsboro, NJ) (Fig. 21-33, *A*), CollaStat (American Medical Products Corp, Freehold, NJ), Hemocollagene (Septodont, Kent, UK), and Instat (Ethicon, Piscataway, NJ). These materials act in essentially similar ways, and the surgical area undergoes a similar healing pattern.[464,480] Overall, studies of wound healing with collagen-based hemostatic agents have shown favorable results.

Surgicel

Surgicel (Ethicon, Somerville, NJ) 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.[389] 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,[354] and



FIG. 21-33 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.

healing is retarded, with little evidence of resorption of the material at 120 days.[56] Use of Surgicel in extraction sockets resulted in greater postoperative pain compared with a control in a split mouth–designed study.[389]

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.[156] 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.[71] However, these effects were transitory and did not impair long-term bone healing.[372]

Bone Wax

Historically, bone wax has been advocated for controlling both hemostasis and debris in the bony crypt during periradicular surgery.[461] 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[112,359] by producing a chronic inflammatory foreign-body reaction[35] and impairing the clearance of bacteria.[250] The use of bone wax can no longer be recommended, because it impairs healing and several good alternatives are available.[559]

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. Two studies using a rabbit model[243,284] reported hemostatic control for 5 minutes, near-normal healing, and 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.[281] 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,[124] differences in the degree of epidermal maturation, and tattoo formation.[510]

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.[544] It is gradually removed from the site of implantation regardless of whether new bone has formed.[106] Use of calcium sulfate during periradicular surgery does not significantly affect healing, and deposition of cementum and osseous healing proceed normally.[28] 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.[268] 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.[79,268] All granulation tissue should be removed from the root apex area prior to placement of the epinephrine pellet to assure direct contact with bone.[267] Vasoconstrictive amines exert their effects by binding to and interacting with adrenergic receptors in various body tissues. When epinephrine is bound to α_1- and α_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. 21-30, *B*) provide very good localized hemostasis in periradicular surgery.[529]

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.[200,252] A recent study[529] 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, so 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.[536] 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.[511] 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.[362] 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 healing that may be less than ideal.

Determining 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,* intraradicular or extraradicular chemical substances, or extraradicular physical factors,[351,473,476] but etiology cannot always be determined with complete certainty. Frequently a number of factors are involved.[95] Most cases involve some form of bacterial participation (e.g., bacteria within apical ramifications), and 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 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.

*References 241, 270, 348, 470, 514, 515, 530, and 549.

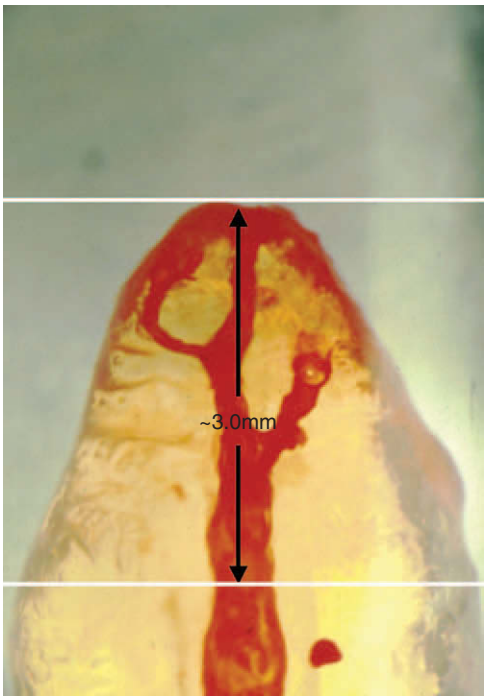


FIG. 21-34 Cleared section of a typical single-canal root that was injected with dye to demonstrate apical accessory canals. Most of the apical ramifications can be eliminated with a 3 mm resection.

Root-End Resection

Two main principles dictate the extent of the root-end resection. First and foremost, 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 7). 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 irregularities (e.g., accessory or lateral canals) in the apical 3 mm of the tooth.[125,462] An apical resection of approximately 3 mm should include most accessory and lateral canals and thus eliminate most residual microorganisms and irritants (Fig. 21-34). 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. 21-35).

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.[69] 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 closet 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 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.* 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.[336,391-393] The surgeon should position the resection of the root to avoid possible damage to these structures.

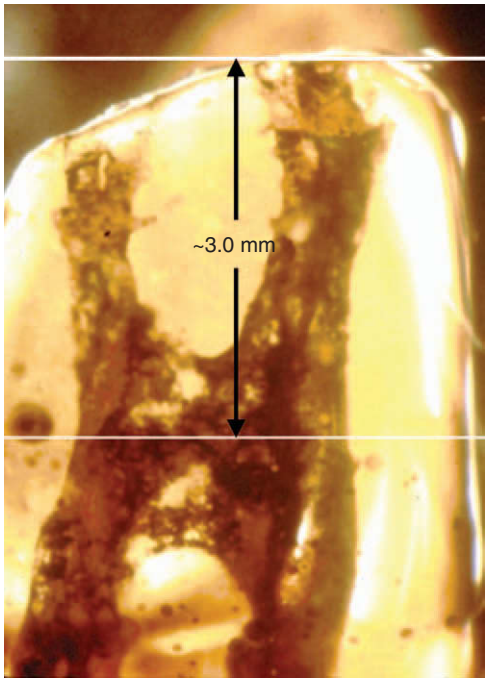


FIG. 21-35 Cleared section of a mesiobuccal (MB) root in a maxillary first molar. Root resection at the recommended 3 mm level exposes isthmus tissue connecting the MB-1 and MB-2 canals.

Angle of Resection

Enhanced magnification and illumination techniques have eliminated the need to create a beveled root surface in most cases.[91,422] From a biologic perspective, the most appropriate angle of root-end resection is perpendicular to the long axis of the tooth (Figs. 21-36 and 21-37). 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.[321] 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. The probability that irritants from within the canal system will gain access to the healing tissues also rises as the resection angle increases.[172,497] 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.[185] 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.[452]

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

*References 40, 87, 90, 216, 266, 422, 429, and 465.

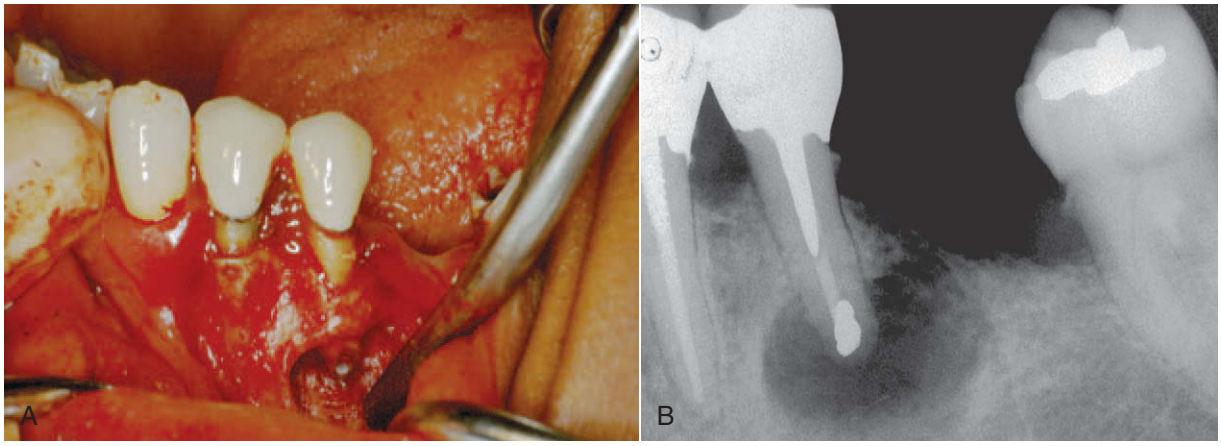


FIG. 21-36 Periradicular surgery circa 1990. **A,** The root end was prepared with a 45-degree bevel and rotary bur microhandpiece. Amalgam was a commonly used root-end filling material at this time. **B,** Immediate postoperative radiograph of a mandibular second premolar with amalgam root-end filling. Although many teeth treated this way healed successfully, newer materials and techniques described in this chapter are currently recommended.

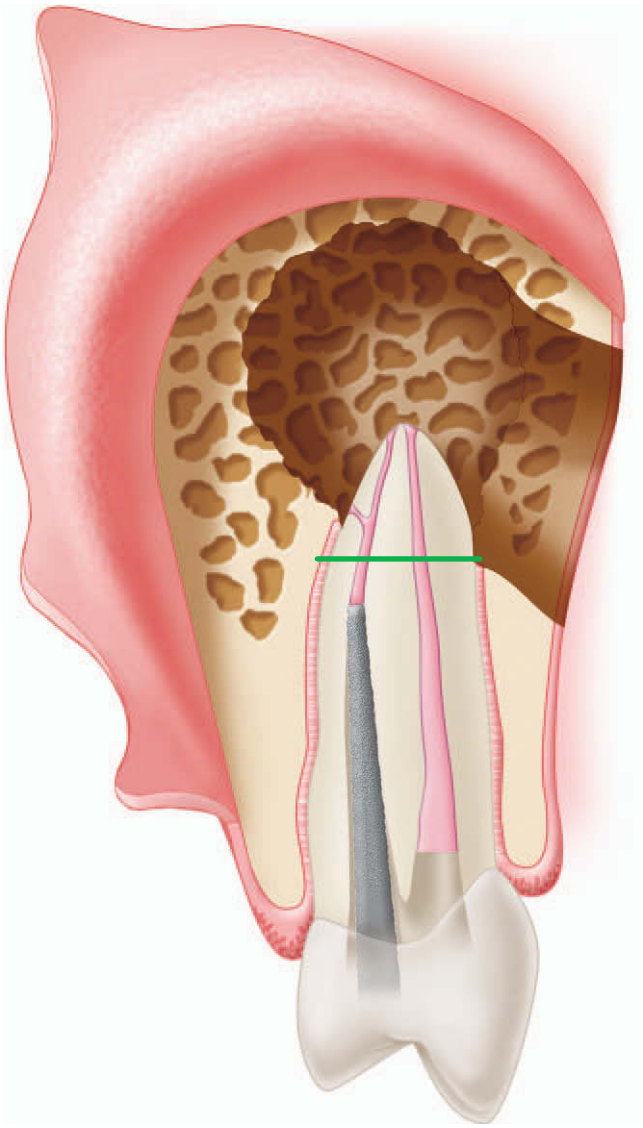


FIG. 21-37 Perpendicular or near-perpendicular root-end resection *(green line)* can be achieved with the use of microsurgical instruments and enhanced magnification and illumination.

of cementum and subsequent regeneration of the PDL across the resected root end. Two important aspects of this process are the surface topography and chemical treatment of the resected root end. Healthy cementum on the root end is required for successful regeneration of periodontal tissues.[23] 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.[51,453,533]

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.[553] However, with a smooth resected root-end surface, the surgeon is better able to detect surface cracks and anatomic variations.[339] 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.[201] Several studies have compared the root-end surface after resection.[339,358,554] Generally, crosscut fissure burs in both high-speed and low-speed handpieces produced the roughest and most irregular surfaces. Researchers[339] 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, Savannah, GA), and the Multi-Purpose bur (DENTSPLY Maillefer, Milford, DE), then finished with either a multifluted carbide finishing bur (Brasseler) 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.[554] 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.[42,43,228,524,566] 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.[415-419] One study[110] examined the effect of citric acid demineralization of resected root ends on periradicular healing. A 1- or 2-minute application 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 recently 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.[59,61]

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.[63] Unlike the lower-pH solution, EDTA does not adversely affect the surrounding tissues.[62]

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.[58,60,63] 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.[309] 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.[14] 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] but EDTA has been shown to be more favorable to human PDL cell attachment.[570]

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.[312] 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 {personal 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.

ROOT-END CAVITY PREPARATION

Root-end cavity preparation is a crucial step in establishing 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. 21-38 and 21-39). 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.[169,431] Traditionally, a microhandpiece with a rotating bur has been used, but with the advent of ultrasonic tips designed specifically for this purpose (Fig. 21-40), root-end preparations now are most often performed with the ultrasonic technique.[87] Clinical evidence is emerging

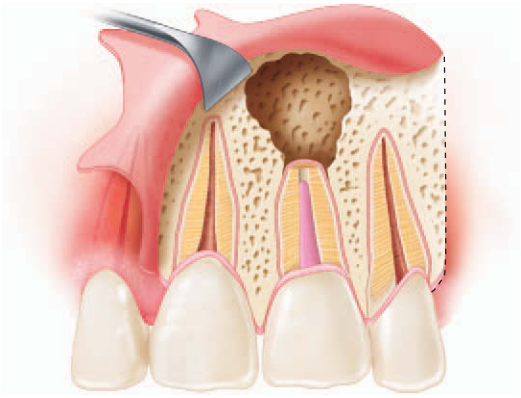


FIG. 21-38 Diagram of a perpendicular root-end preparation and 3-mm-deep cavity preparation along the long axis of the root.



FIG. 21-39 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.

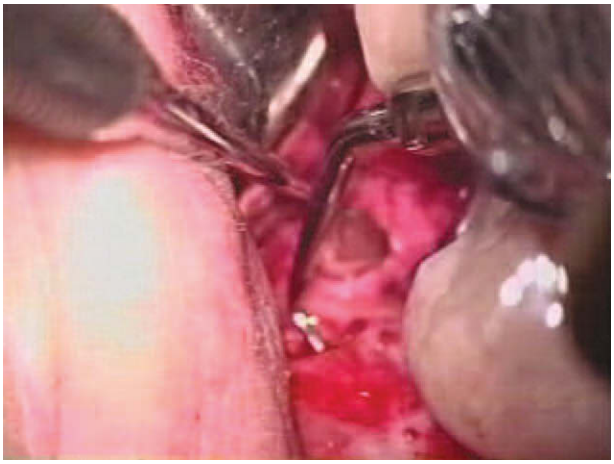


FIG. 21-40 Ultrasonic tip in use. Preparation along the long axis of the root is possible using tips designed for each area of the mouth. In this case, the tip is properly positioned for root-end cavity preparation of a maxillary first premolar but is dangerously close to the lip because of improper retraction. Heat generated by an ultrasonic tip can cause a thermal burn, which may result in scar-tissue formation.

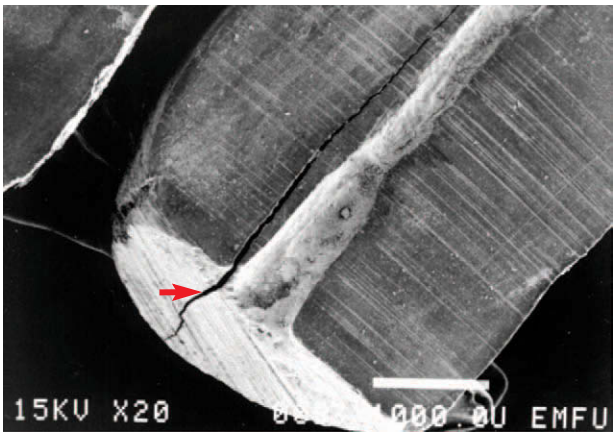


FIG. 21-41 Scanning electron microscope (SEM) image of a root end prepared in vitro with an ultrasonic device at high-power setting. A distinct fracture line can be seen *(red arrow)*. Root-end preparation with ultrasonic devices should be done at low power and with water coolant.



FIG. 21-42 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.

to support the benefit of ultrasonic root-end preparation when compared to traditional bur preparation, especially in molar surgery.[126]

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.[92,287,329,561] Ultrasonic apical preparation generates significantly less smear layer compared with burs alone[195]; root-end preparation with a bur produces a heavy smear layer at all levels of preparation.[202]

The major concern with ultrasonic root-end preparation is the potential for creating root fractures as a result of the ultrasonic vibration (Fig. 21-41).

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).[406] This classification system is not universally used in studies of root-end cavity preparation, so 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.[48,286,356,406] 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.[3,175,282,335,451] 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[338] and cadaver-based studies[82,196] 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 reasons may account for the differences observed. 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[167,282] and therefore is recommended for clinical use.

Significance of Ultrasonic Tip Design

Several different types of ultrasonic tips are available for root-end preparation (Figs. 21-42 and 21-43), 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.[543] 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.[190,386] 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.[240] 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 preparation.[73,190,240,356,406] The cavity wall 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.[73,579] Overall, the quality of coated-tip preparation has been suggested to be superior.[386]

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[273]; such an increase can injure pulpal and periodontal tissues.[361]



FIG. 21-43 Ultrasonic tip with diamond coating and irrigation port (DENTSPLY Tulsa Dental).

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.[27,435] An ultrasonic preparation can be made into the root 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. 21-44).

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 byproducts, 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

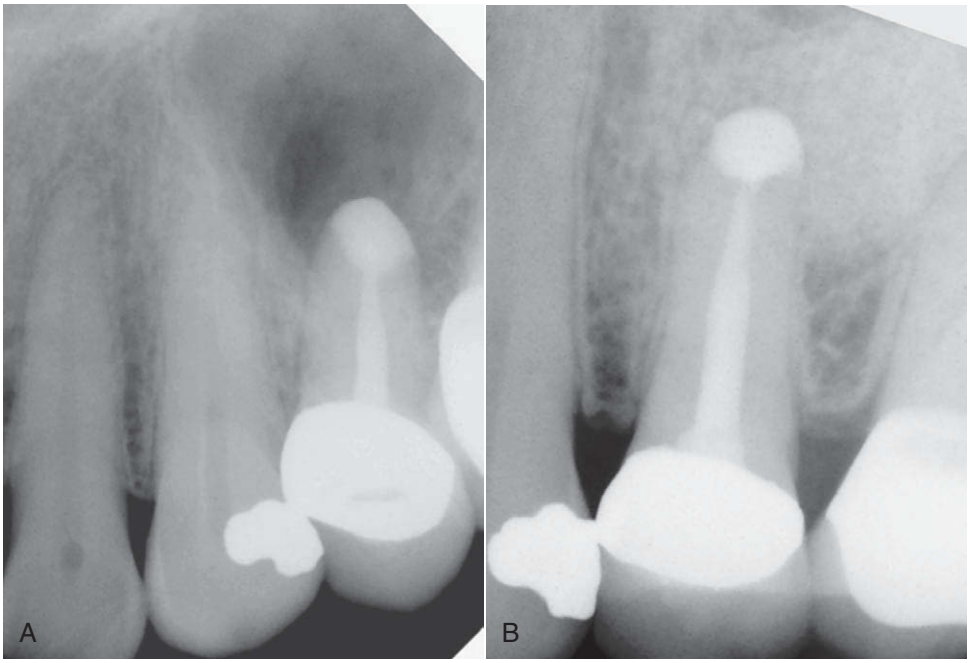


FIG. 21-44 A, Immediate postoperative radiograph of a bonded root-end filling in a maxillary first premolar. **B,** Follow-up radiograph at 20 months showing good periradicular healing.

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 SuperEBA), glass ionomer cement, Diaket, composite resins (Retroplast), resin–glass ionomer hybrids (Geristore), and mineral trioxide aggregate (ProRoot-MTA).

Zinc Oxide Eugenol Cements

Zinc oxide powder and eugenol liquid (ZOE) 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.[230] 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.[230-232] 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.[132,331,532] 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 (DENTSPLY/Caulk, Milford, DE) and SuperEBA (Bosworth Company, Skokie, IL).

Intermediate Restorative Material

Intermediate restorative material (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.[111,388] 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.[209,311,398-401] In vitro, IRM prevents adherence of enamel matrix proteins.[445]

SuperEBA

SuperEBA 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*-ethoxybenzoic acid. SuperEBA 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.[562] SuperEBA has radiopacity[463] and sealing effects similar to those of IRM and is less leaky than amalgam.[219,369] The leakage pattern of SuperEBA does not appear to be affected by root-end conditioning or finishing techniques.[165,451] When SuperEBA 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.[161] The environment of the periradicular wound may affect the long-term stability of SuperEBA, which has been shown to disintegrate over time in an acid pH environment.[31]

Biologically, SuperEBA is well tolerated in the periradicular tissues when used as a root-end filling, but it has no capacity to regenerate cementum. Bone healing has been demonstrated at 12 weeks, with some fibrous tissue persisting. SuperEBA root-end fillings show a basophilic-stained line adjacent to the filling material, which may indicate hard-tissue formation.[385,399,401,516] Collagen fibers appear to grow into the cracks of the material,[377] but the significance of this is unknown. SuperEBA has limited antibacterial effect.[98] The cytotoxicity of SuperEBA is similar to that of amalgam and IRM.[99,574] The incidence of persistent disease after endodontic surgery in which SuperEBA 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 SuperEBA always resulted in less persistent disease. The follow-up period for these studies ranged from 0.5 to 4.6 years.*

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.[44,396,397]

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.† Long-term surface changes can occur in silver-GIC that may affect its stability in the periradicular tissues.[54] Glass ionomer cements are susceptible to attack by moisture during the initial setting period, resulting in increased solubility and decreased bond strength.[179,483,565] 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.[571] The cytotoxicity of chemical- and light-cured GIC does not differ significantly from that of SuperEBA or amalgam.[99,375] The tissue response to GIC is considerably more favorable than to amalgam and similar to that with ZOE-based materials.[97,101,127,397] In a comparative clinical study using amalgam or GIC for root-end filling, healing was evaluated clinically and radiographically after 1 and 5 years.[248,571] 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.[248,571]

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.[556] It is a powder consisting of approximately 98% zinc oxide and 2% bismuth phosphate

*References 140, 307, 378, 429, 492, and 535.

†References 102, 103, 239, 425, 494, and 560.

mixed with a liquid consisting of 2.2-dihydroxy-5.5 dichlorodiphenyl methane, propionylacetophenone, triethanolamine, caproic acid copolymers of vinyl acetate, and vinyl chloride vinyl isobutyl ether. Leakage studies comparing Diaket to other commonly used root-end filling materials have shown it to have superior sealing ability,[180,251,296,540] but 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[262] and generated long-term chronic inflammation in osseous[478] and subcutaneous tissues.[373] However, when mixed at the thicker consistency advocated for use as a root-end filling material, Diaket has shown good biocompatibility with osseous tissues.[360,556] 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.[556] In animal studies, Diaket has shown a better healing response than resected gutta-percha in uninfected teeth[558] and a healing response similar to that with MTA, but no cementum formation was evident.[414] 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 in vitro studies. Composite resins also tend to leak less than amalgam, SuperEBA, IRM, and GICs.[120,326,327,494] However, blood contamination during the bonding process reduces bond strength and increases leakage.[334,531] Marginal adaptation varies, depending on conditions and bonding agents.[18] 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.[77,205,409,410,489] Studies have shown that once the composite resin sets, cells can grow on its surface.[310,343,383,573] The healing response of the periradicular tissues to composite resins in general appears to be very diverse, ranging from poor to good[26,516]; 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-hydroxyethyl)-*p*-toluidine, and 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.* 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.[23,435,436] 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.[26] 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.[435] 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%.[442] A 10-year follow-up of 34 of these cases showed complete healing in 33 of the cases.[440]

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[88] 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.[174,353,457,522] 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.[4,5,47,421,466] 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.[487] In vitro leakage assessment of Geristore and Dyract indicates that the materials leak less than root-end fillings made of IRM, amalgam, or SuperEBA.[64,197] Geristore has a leakage pattern similar to that of MTA.[456] An acid pH significantly reduces dye leakage of Geristore.[427] These materials are less sensitive to moisture than conventional glass-ionomer cement; however, dry environments produce stronger bonds.[96] The effect of blood contamination during the bonding phase in a clinical scenario is unknown. Geristore appears to have the potential

*References 23, 26, 342, 433-437, and 439-442.

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.[83] 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.[143,144,457] However, the healing response in the periradicular region is best described as unpredictable. In a recent 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, but 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.[298]

Mineral Trioxide Aggregate

MTA (ProRoot MTA; DENTSPLY, Tulsa Dental, Tulsa, OK), a material developed specifically as a root-end filling,[503] has undergone numerous in vitro and in vivo investigations comparing its various properties to SuperEBA, 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.* When various in vitro leakage models were used, MTA prevented leakage as well as composite resin and GIC,[7,123,162,560] but the setting and subsequent leakage of MTA are not affected by the presence of blood.[501] Torabinejad et al.[509] 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.[503] In a study comparing the setting time, compressive strength, radiopacity, and solubility of MTA to those of amalgam, SuperEBA, and IRM, MTA was found to be less radiopaque than amalgam but more radiopaque than SuperEBA and IRM.[503] 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 SuperEBA. Initially MTA has a pH of 10.2, which rises to 12.5 at 3 hours after mixing.[503] The pH has been reported to be approximately 9.5 at 168 hours after mixing.[145] MTA is less cytotoxic than amalgam, SuperEBA, or IRM root-end fillings.[505] Endodontic surgery studies in dogs and monkeys have reported less periradicular inflammation and cementum deposition immediately adjacent to the root-end filling material.[28,164,223,502,506] Other investigators[223,224] 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.[293] 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.[23] 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. Recently, 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.[496] 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 antiinflammatory 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.[100] 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 recent 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.[450]

In 2002, a variation of the original formula of gray MTA was 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.[225] One study compared the tissue reaction evoked by the two materials when used as root-end fillings in canines.[298] The only statistically significant difference observed was in the presence of macrophages and/or multinucleated giant cells adjacent to the material. Gray MTA had more samples with mild to moderate infiltration of macrophages and/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.

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, appear to have a clear advantage over the other available materials. 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-

*References 263, 283, 501, 504, 505, and 507-509.

end cavity than most other materials. Several devices have been modified or developed specifically for use with MTA (see Figs. 21-18 to 21-22 and Fig. 21-45). Typical clinical cases showing the surgical procedures described in the previous sections are presented in Figs. 21-46 to 21-48.

CLOSURE AND SUTURING

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 is then gently curetted and irrigated with sterile saline or water to remove any remnants of hemostatic agents and packing 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 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 is then 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.[271] 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 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. 21-49). 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.

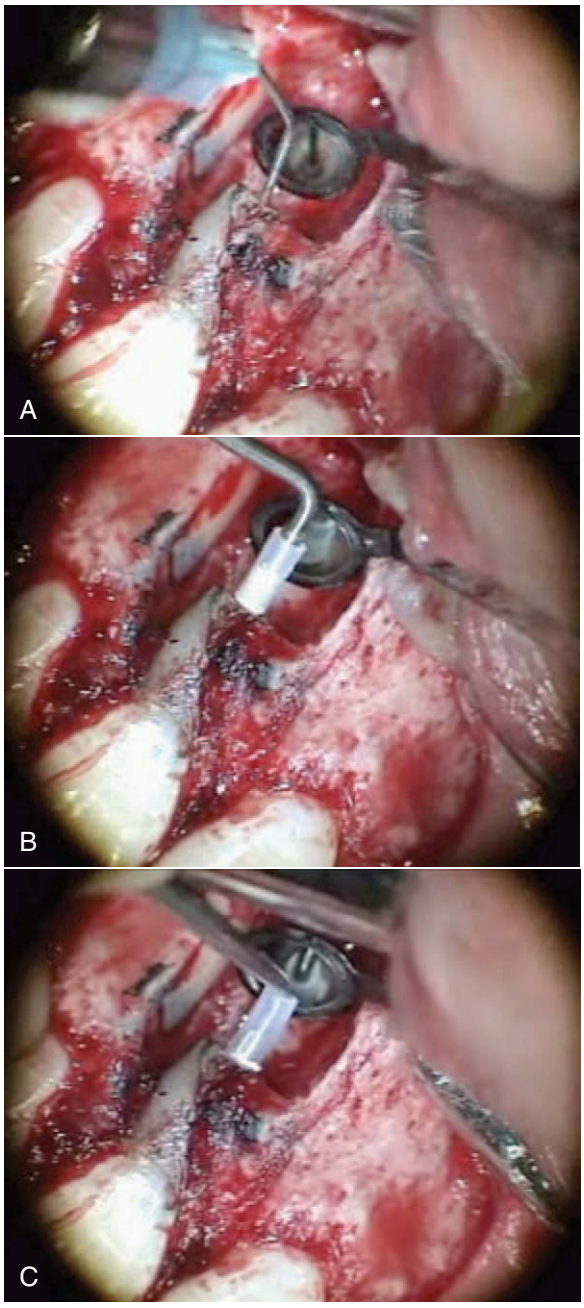


FIG. 21-45 A, Stropko syringe used to dry a root-end preparation before placement of the root-end filling material. **B,** Clinical use of an MTA delivery system (DENTSPLY Tulsa Dental). The device is loaded with MTA and placed over the root-end preparation. **C,** Pressing the plugger into the sleeve 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.

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

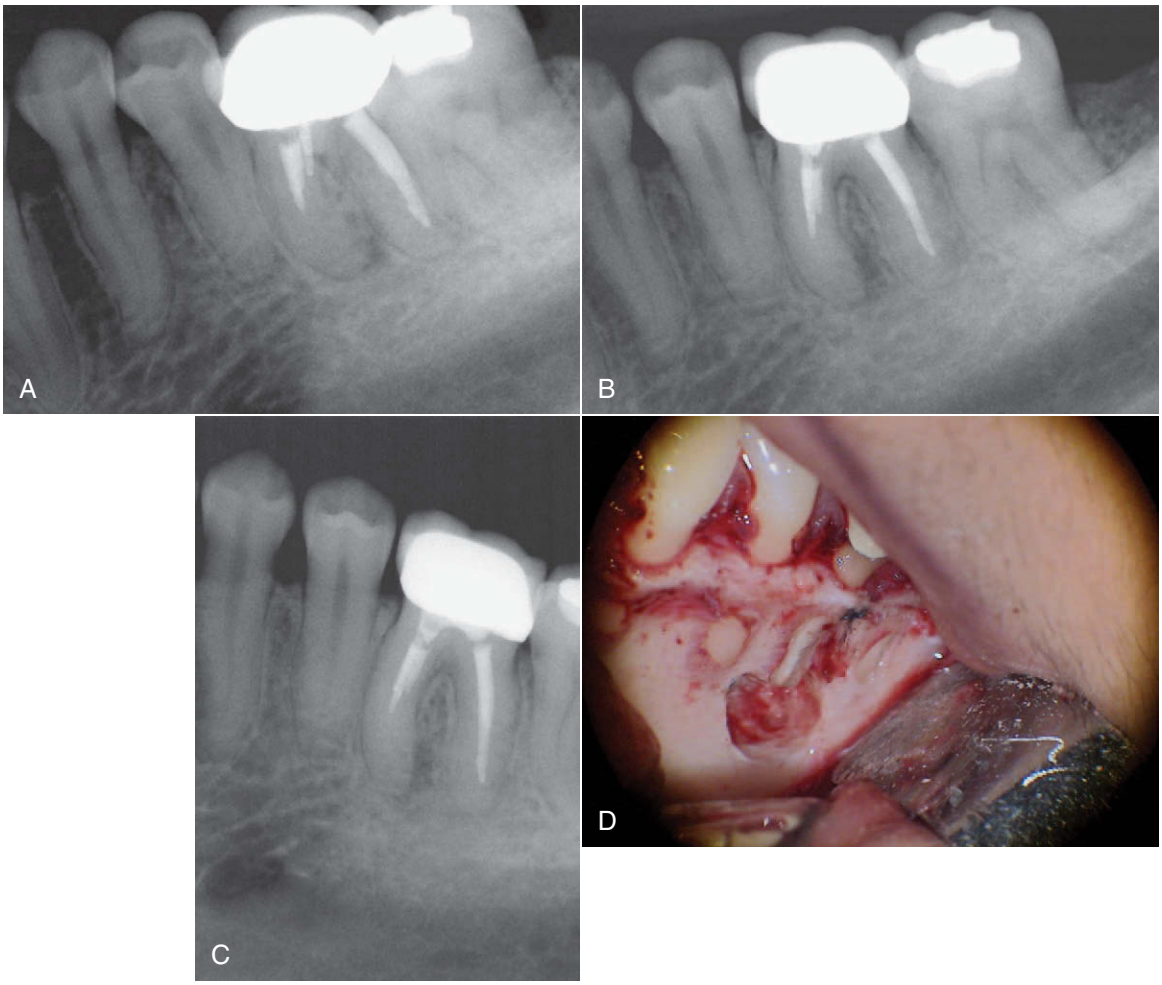


FIG. 21-46 A, Preoperative radiograph (mesial angle) of a left mandibular first molar. Periradicular disease and symptoms had persisted after nonsurgical retreatment by an endodontic resident. The mesial canals were completely obstructed at the midroot level. The preoperative evaluation included three 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. *Continued*

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 wound margins. Silk suture material has been commonly used in dental surgery for decades and is both inexpensive and easy to handle, but it tends to support bacterial growth and allows for a wicking effect around the sutures. For these reasons, other materials are preferable to silk.[89]

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 surgery) or if the suture will be used in areas of the mouth where access is very 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.[411]

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.[109,186,384,568] 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.

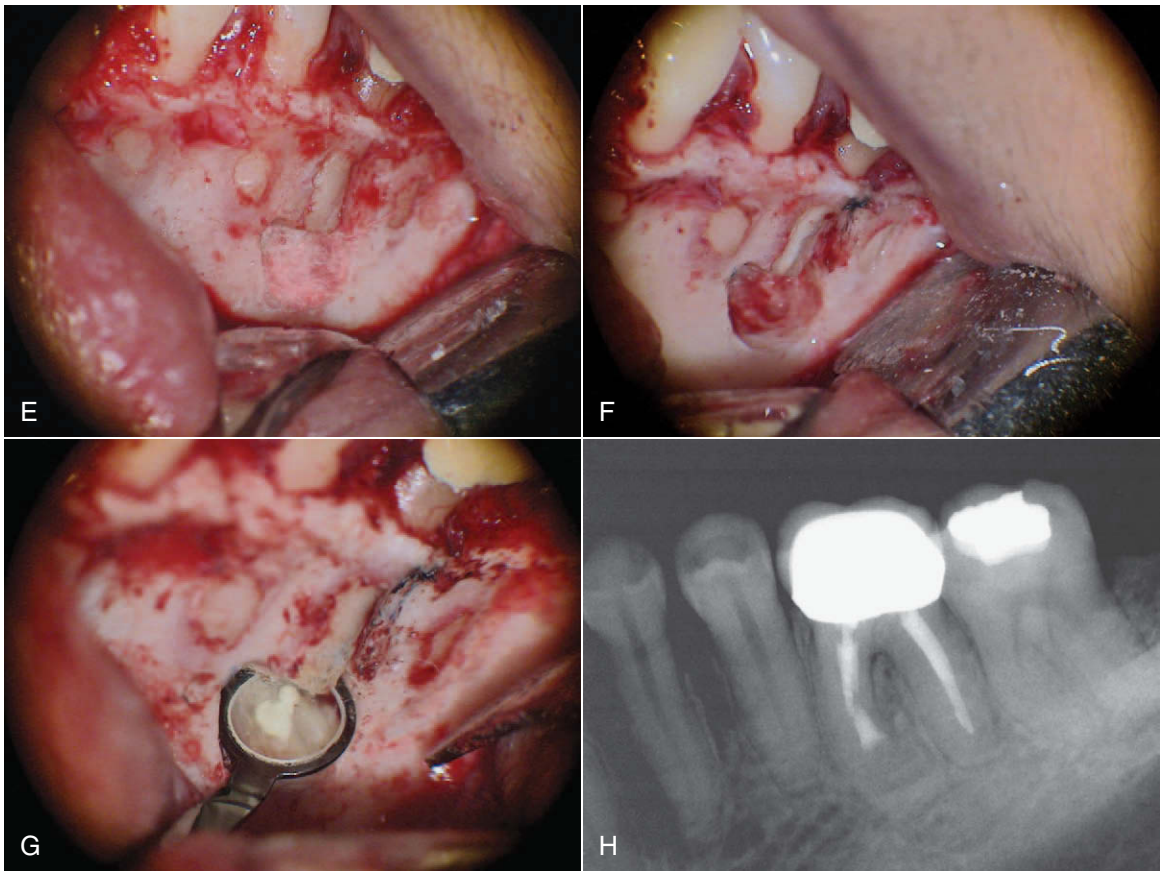


FIG. 21-46, cont'd E, Racellets were packed into the bony crypt to establish hemostasis. **F,** Racellets were removed (one usually is left in deepest part of crypt during root-end preparation and filling), and root end was beveled perpendicular to long axis of root. Methylene blue dye can be useful for identifying root outline and locating any cracks. Ultrasonic root-end preparation was completed to a depth of 3 mm, connecting MB and ML canals. **G,** MTA root-end filling was placed and inspected. Bony crypt was then gently curetted to initiate bleeding and remove any remnants of hemostatic materials. Flap was repositioned and radiograph taken. **H,** Immediate postoperative radiograph confirmed depth and density of root-end filling and absence of any foreign objects. (NOTE: Calcium sulfate and bone grafting material placed in osseous defect and over root because of large buccal dehiscence, although this is not routinely required in these cases.) *(Courtesy Dr. Vince Penesis.)*

GUIDED TISSUE REGENERATION AND ENDODONTIC SURGERY

The amount and location of bone adjacent to the root structures affect the prognosis of periradicular surgery. In one study[267] the authors 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. 21-50 and 21-51 are examples of cases that required guided tissue regeneration (GTR).

An apicomarginal defect[134] or a localized bony defect distinguished by a total deficiency of alveolar bone over the entire root length has a significant adverse affect on the outcome, reducing the rate of complete healing to 27% to 37%.[220,477] The presence of a periradicular lesion 15 mm or greater in diameter also has been linked to a poorer prognosis.[220] Advanced periodontitis with deep pocket formation has been associated with chronic periradicular inflammation after endodontic surgery and subsequent failure of the root-end surgery.[432] The cause of failure has been identified as ingrowth of nonosteogenic tissues into the periradicular surgical site and downgrowth 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.

The basic principle of guided tissue and bone regeneration is that different types of cells repopulate a wound at different rates during healing. Soft-tissue cells are considerably more motile than 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

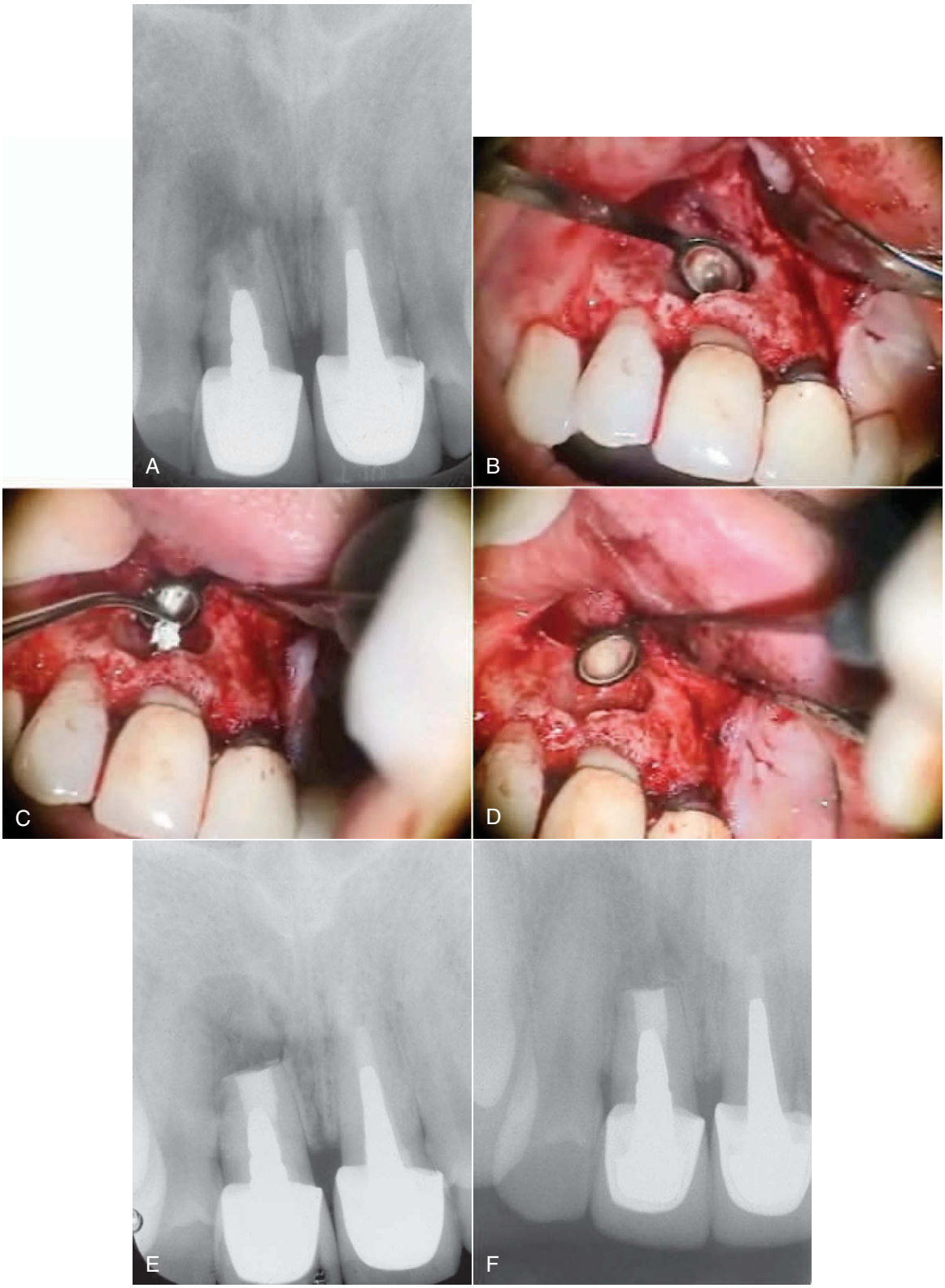


FIG. 21-47 A, Preoperative radiograph of a maxillary left central incisor showing evidence of previous surgery but no apparent root-end filling. Because of short root length and inadequate band of keratinized gingiva, an intrasulcular incision and full-thickness triangular mucoperiosteal flap design were selected. **B,** Root was minimally resected, and root-end cavity was prepared ultrasonically. **C,** MTA was placed and condensed into root-end cavity preparation. **D,** Root-end filling was inspected before flap was repositioned and sutured. **E,** Immediate postoperative radiograph. **F,** Six-month follow-up radiograph showed good initial periradicular healing. *(Courtesy Dr. Shawn Velez.)*

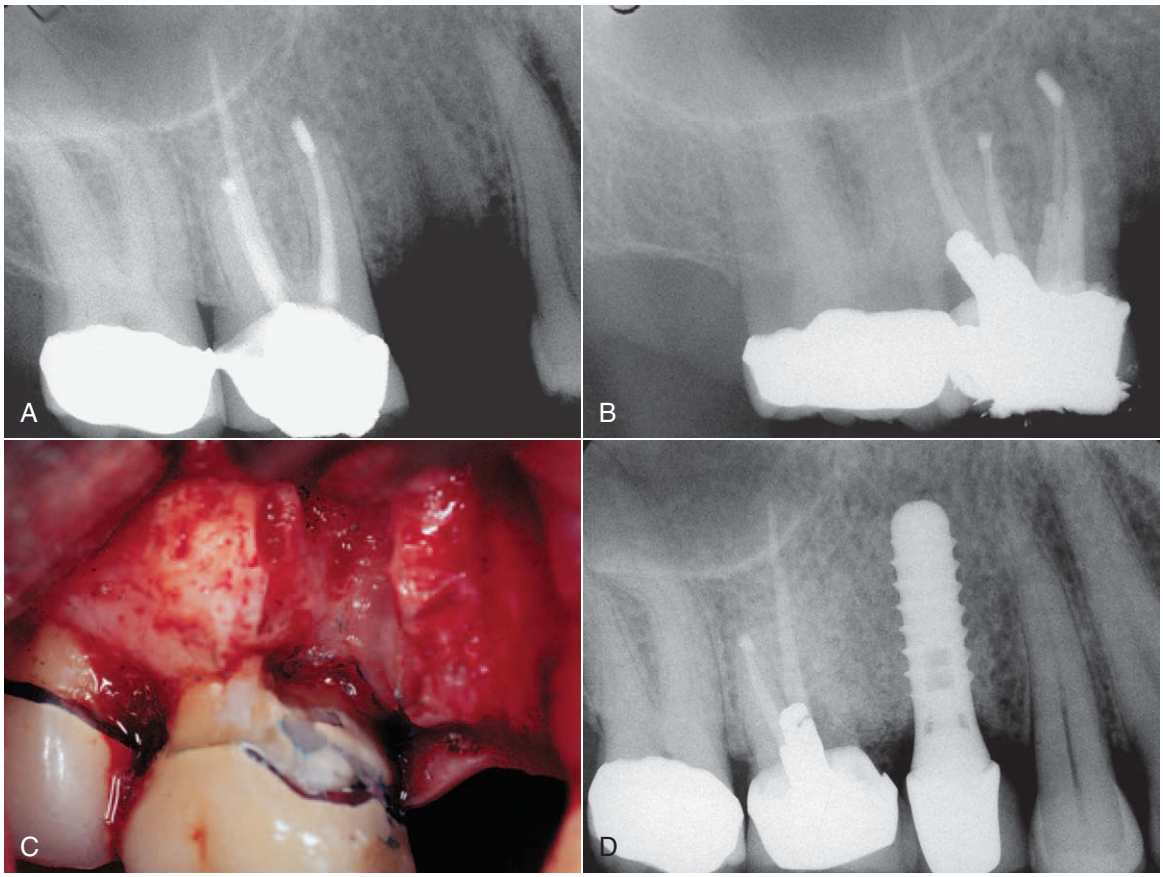


FIG. 21-48 A, A maxillary right first molar that was sensitive to percussion and tender to palpation over the MB root. Periodontal probing revealed a deep, narrow bony defect on the facial aspect of the MB root. Presence of a vertical root fracture was confirmed visually using magnification and methylene blue dye. 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.

other cells with osteogenic potential to repopulate the defect, resulting in new connective tissue attachment and bone formation. Researchers[114,115] 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.[142] 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.* 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.

Researchers[381] compared the healing of 20 large periradicular defects (>10 mm diameter) with and without the use of resorbable membrane. They reported that at 12 months after surgery, the sites in which membranes had been used had healed more quickly, and the quality and quantity of the regenerated bone was superior. A study evaluated periradicular and periodontal healing in cases involving apicomarginal defects when guided tissue regeneration (Bio-Oss and Bio-Gide membrane; Osteohealth, 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.[135] 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.[177]

Several different types of membranes are available. They can be grouped into two broad categories: nonresorbable and resorbable (Table 21-1). 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

*References 6, 78, 117, 147, 259, 319, 395, 402, 413, 518, 523, and 576.

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 to 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. 21-49 Example of postoperative instructions. Written instructions provide an essential reference for the patient; verbal instructions often are difficult to remember after surgery. Instructions may be modified as needed to provide instructions the patient can understand. Readability of these instructions is at approximately eighth-grade level using the Flesch-Kincaid Grade Level scale.

functions: to act as a mechanical substructure that supports the membrane and overlying soft tissues and to serve as a biologic component that enhances bone formation. Bone graft materials (Table 21-2) 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 osteoconductive 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; it is not always possible to predict before surgery when grafting materials may be needed.

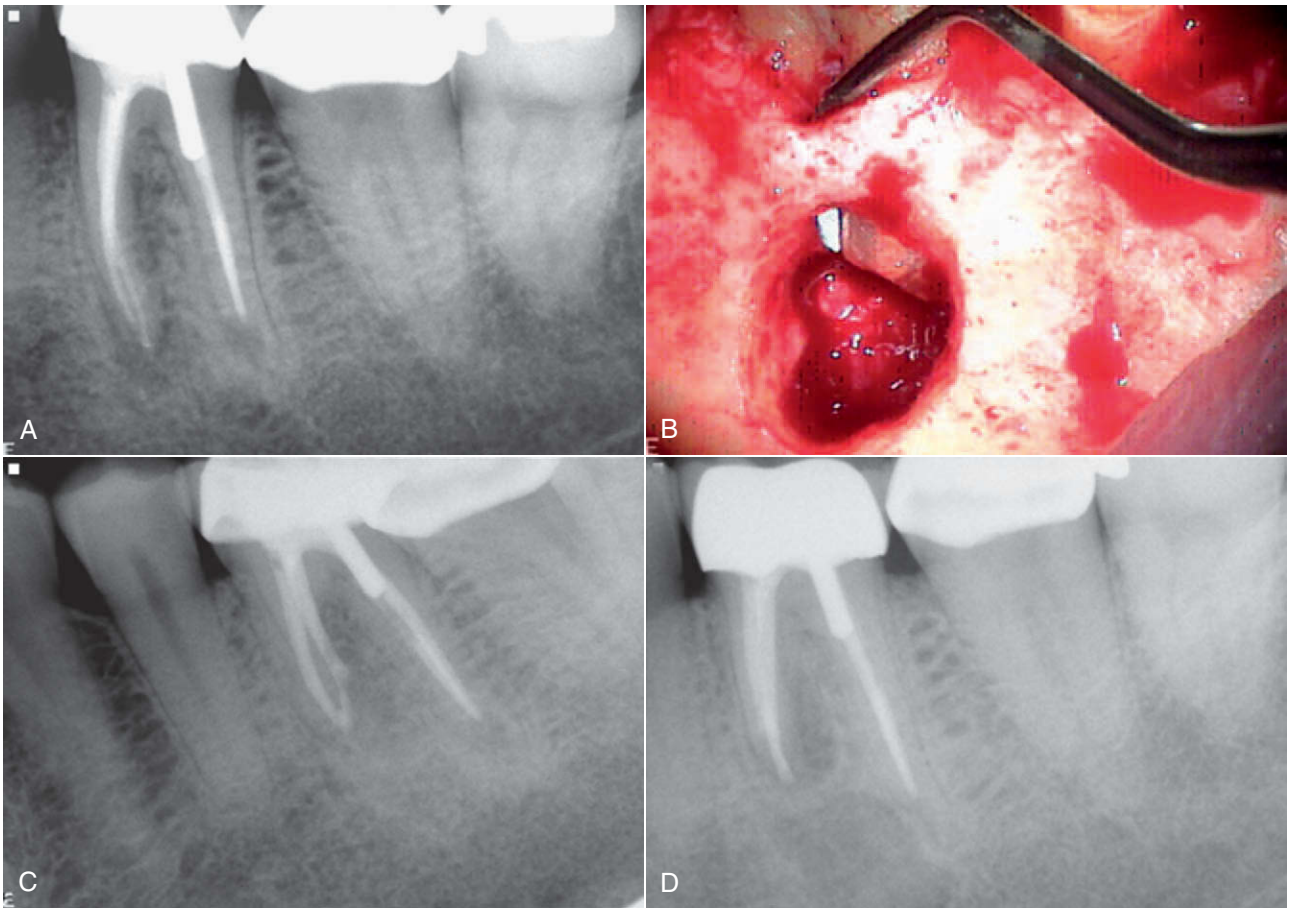


FIG. 21-50 A, Preoperative periapical radiograph of tooth #19. **B,** Clinical image demonstrating periodontal defect along facial aspect of the mesial root. **C,** Immediate postsurgical radiograph. The mesial root end was prepared with ultrasonics and filled with MTA. A mixture of DFDBA and Capset was placed for guided tissue regeneration. **D,** One-year follow-up radiograph demonstrating good healing. Periodontal probings within normal limits.

If GTR techniques are to be used during periradicular surgery, a resorbable membrane should be chosen, and a protocol should be followed (Figs. 21-52 and 21-53):

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 adversely affect the outcome.*

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.[291] 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 to 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.[32,238,324,577] An atraumatic extraction technique is desirable, since 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.

*References 70, 305, 426, 499, 512, and 513.

INTENTIONAL REPLANTATION

Intentional replantation may be an option when surgical access is very 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. 21-54). However, any tooth that can be atraumatically removed in one piece is a potential candidate

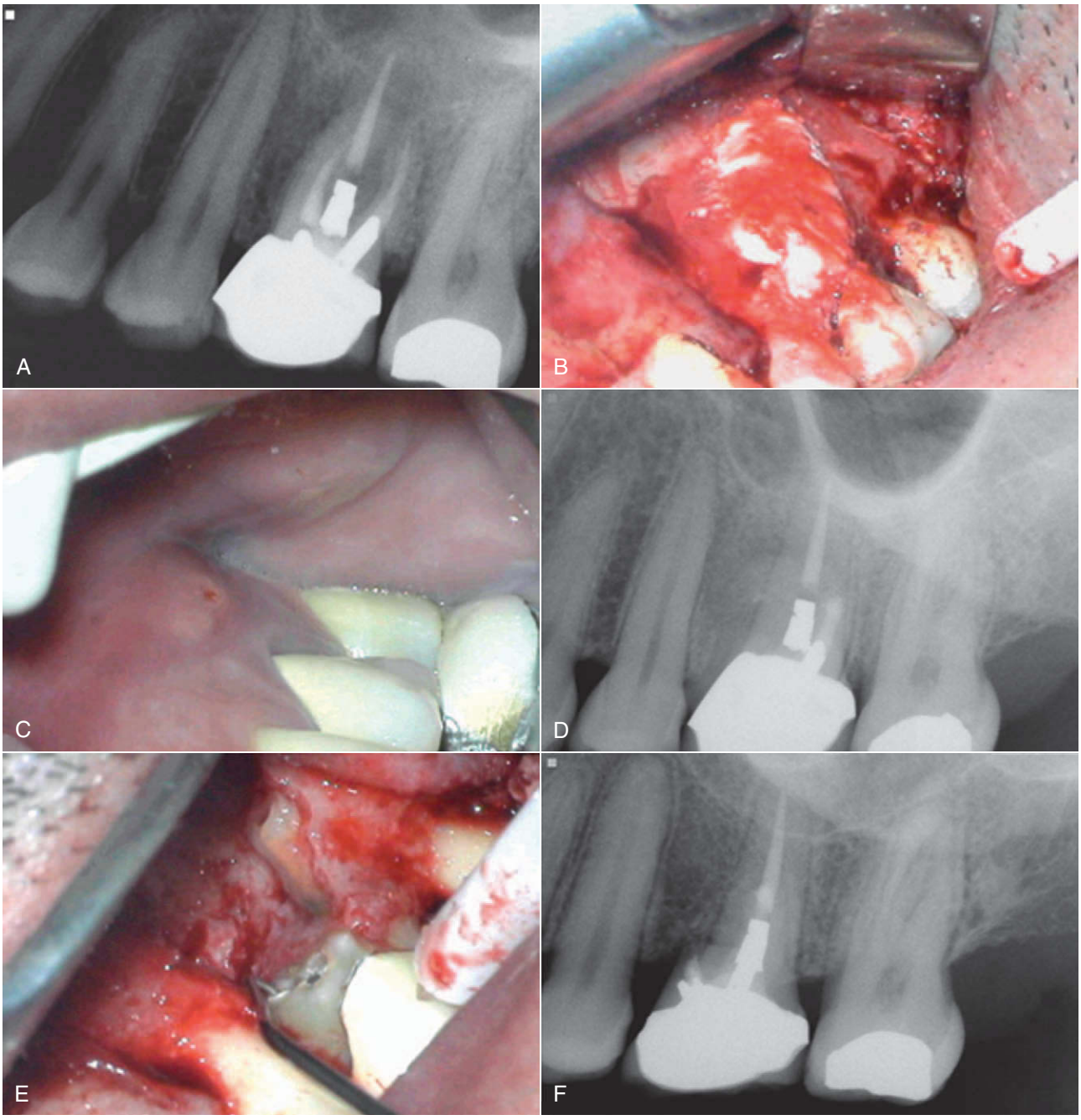


FIG. 21-51 A, Preoperative periapical radiograph of tooth #14. **B,** Clinical image. **C,** The MB root was removed owing to root fracture. Ultrasonic root-end preparation with MTA fill was performed on the DB root and remnant of the MB root. A significant amount of buccal bone is now missing and GTR is indicated. **D,** A mixture of DFDBA and Capset was placed in the bony defect. **E,** Immediate postoperative radiograph. **F,** Two-year follow-up radiograph.

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,[382] although some investigators recently demonstrated moderate success using a dentin-bonded resin and intentional replantation for the treatment of teeth with root fractures.[214,254,481] 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.[214]

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 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

TABLE 21-1

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 21-2

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)

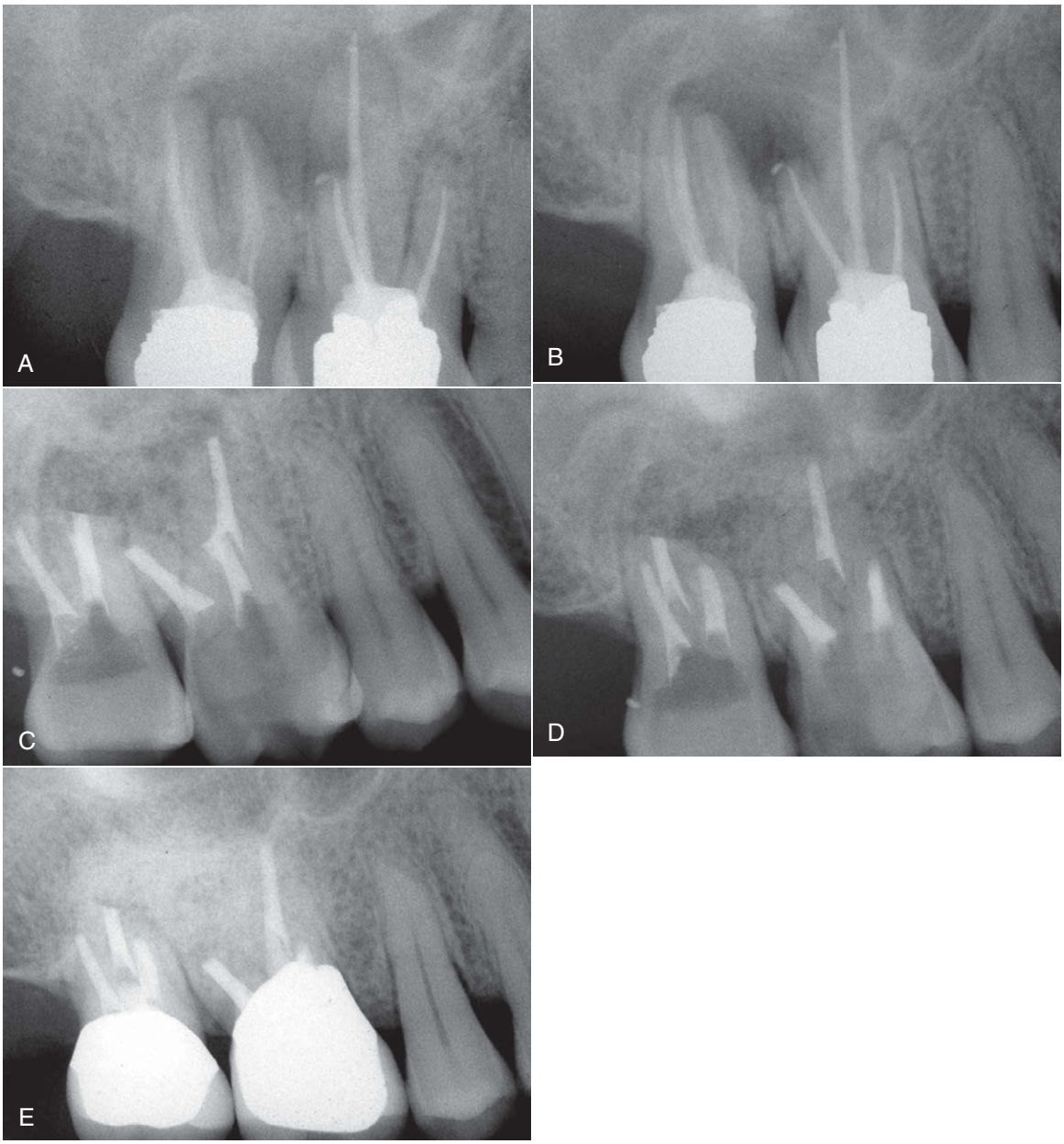


FIG. 21-52 A, Preoperative angled radiograph of maxillary right first and second molars. Both teeth had been treated previously, and patient reported a history of pain in the area for the past 5 years. Treatment plan included nonsurgical retreatment followed by periradicular surgery with bone grafting 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. Root ends were prepared with ultrasonics, conditioned with 17% ethylenediamine tetra-acetic acid (EDTA), filled with Diaket, and smoothed with a superfine diamond finishing bur. Crypt was packed with BioOss xenograft material, and a Guidor resorbable membrane was placed. **D,** Immediate postoperative radiograph (straight-on view). **E,** Four-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.

soaked in a physiologic solution such as Hank's Balanced Salt Solution. After root-end preparation and filling (described previously), 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.[22,24,371]

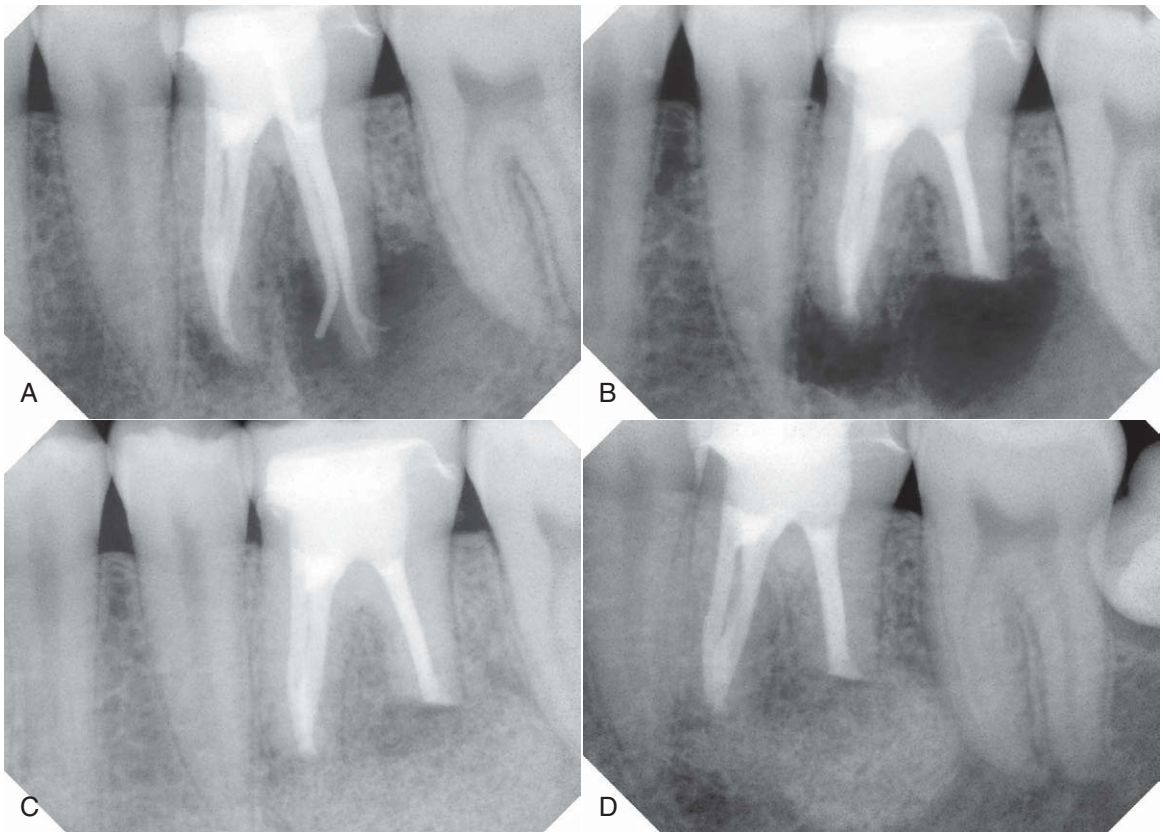


FIG. 21-53 A, Preoperative radiograph of a mandibular left first molar. Gutta-percha was inserted into the buccal sulcus and traced to the apex of the distal root. Nonsurgical root canal treatment had been performed 12 months earlier. **B,** Root-end resection and MTA root-end fillings (M and D roots). **C,** Immediate postoperative radiograph. BioOss xenograft material was placed. **D,** The 19-month follow-up radiograph showed good periradicular healing.

POSTOPERATIVE CARE

As previously noted, NSAIDs generally are the preferred class of drugs for managing postoperative pain (also see Chapter 19).[10,41,53,136] 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.[141] 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.[237,330] 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.[89,200] 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.[76] 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. 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 should

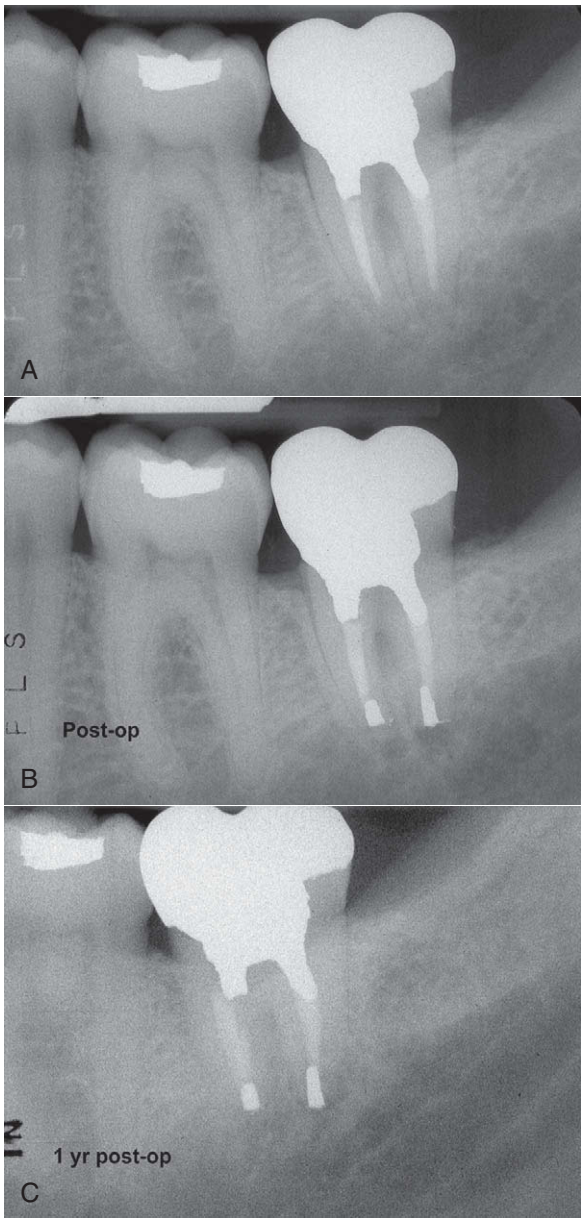


FIG. 21-54 Intentional replantation. **A**, Preoperative radiograph of a mandibular left second molar. The tooth was persistently sensitive to percussion and biting after nonsurgical retreatment. **B,** Radiograph of the tooth immediately after extraction, root-end preparation and filling, and replantation. **C,** At the 1-year follow-up visit, the tooth was asymptomatic and showed good periradicular healing. *(Courtesy Dr. Matt Davis.)*

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, investigators[519] 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



FIG. 21-55 Postoperative ecchymosis can be alarming to the patient but resolves spontaneously within 7 to 14 days.

surgery.[276] 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. 21-55) 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.[265] 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,[17,30,265,542] but this practice is controversial, and no evidence supports the routine use of antibiotics and decongestants in these cases. An academician[546] 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.[274,438,548] 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.

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%[424]; 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.[525] 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.[158,412] Robinson and Williams[424] 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 or, alternatively, a pinprick or sharp instrument can be applied. 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 just 15 years ago. Enhanced magnification and illumination, microsurgical instruments, ultrasonics, new materials for hemostasis and root-end filling, 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

22

Restoration of the Endodontically Treated Tooth

DIDIER DIETSCHI, SERGE BOUILLAGUET, and AVISHAI SADAN

CHAPTER OUTLINE

SPECIAL FEATURES OF ENDODONTICALLY TREATED TEETH
- Compositional Changes in Nonvital Teeth and Influence of Endodontic Therapy
- Dentin Structure and Properties of Endodontically Treated Teeth
- Fracture Resistance and Tooth Stiffness of Endodontically Treated Teeth
- Esthetic 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 Restorations for Full Crowns
- Crown Preparation and Temporary Restoration

SPECIAL FEATURES OF ENDODONTICALLY TREATED TEETH

Endodontically treated teeth are structurally different from vital teeth; major changes include altered tissue physical characteristics, loss of tooth structure, and possibly also discoloration. Tissue modifications must therefore be analyzed at different levels, including tooth composition, dentin microstructure and tooth macrostructrure. It is fundamental to understand the implication of such features on tooth biomechanics, since they will largely influence restorative approach and means (Table 22-1). Numerous in vitro studies dealing with the complexity of the nonvital tooth substrate are reported in the literature; ultimately, in vivo trials describe the global effect of tissue qualitative and quantitative changes on restoration, long-term behavior, and survival.

Compositional Changes in Nonvital Teeth and 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 bonded water.[69,73] This alteration was associated with a slight change in values for the Young modulus and proportional limit.[79] However, no decrease in compressive and tensile strength values was associated with this change in water content.[79] Only one study showed no difference in moisture content between vital and nonvital teeth.[129] No difference in collagen cross-linkage was found between vital and nonvital dentin.[147] There is no other evidence of chemical alteration due to the removal of pulpal tissue.

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, either with mineral content (chelators) or organic substrate (sodium hypochlorite).[82,115,122] Chelators mainly deplete calcium by complex formation and also affect noncollagenous proteins (NCP), leading to dentin erosion and softening.[82,88,151] Sodium hypochlorite supposedly demonstrates a proteolytic action by extensive fragmentation of long peptide chains such as collagen.[72] These alterations are likely to fragilize dentin and root structure and reduce adhesion to this substrate.

Dentin Structure and Properties in Nonvital and Endodontically Treated Teeth

It is important to know about the normal variations in dentin physical properties, which must be differentiated from alterations related to loss of vitality or endodontic treatment. For instance, dentin microhardness and elasticity actually varies

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

between peritubular and intertubular dentin and depends 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 root surface).[74,91,107] Most if not all the decrease in hardness on approaching the pulp can be attributed to changes in hardness of the intertubular dentin.[90,91] Overall dentin elasticity modulus is considered to be in the range of 16.5 to 18.5 GPa[16,33,52] even though variations can be observed due to the measuring methods.[92,127,145]

The changes in mineral density due to the variation in the number and diameter of tubules within the tooth may also explain variations in the properties of dentin. It was shown that dentin hardness values are inversely related to dentin tubule density.[130] Ultra microindentation measurements also demonstrated significantly higher values for hardness and elasticity modulus when forces were parallel to the tubules rather than perpendicular.[139] Differences in maximum strength and compressive strength were also found to vary according to tubule orientation.[127] 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.[99] No difference was found in the Young modulus of aged, transparent dentin (also called *sclerotic*) and normal dentin,[20,93,175] 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% while the fatigue lifetime is deleteriously affected.[93]

However, no or only minor differences in microhardness values were found between vital and nonvital dentin of contralateral teeth after periods varying from 0.2 to 10 years.[100,161] The literature does not support a widely held belief that attributes particular weakness or brittleness to nonvital dentin. It is also believed that the age-related reduction of pulp volume, progressively replaced by secondary or tertiary dentin, could account for a reduced fracture resistance of aged, nonvital teeth; this assumption is evaluated in the literature. The only impact of age-related tissue changes are the aforementioned reduction in fracture toughness and fatigue lifetime attributed to dentin sclerosis.[93]

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,[68,164] as well as microhardness.[34,81,153] 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.[118]

In conclusion, 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

The major changes in tooth biomechanics are attributed to the loss of tissue following decay, fracture, or cavity preparation, including the access cavity prior to endodontic therapy.

The loss of tooth structure following a conservative access cavity preparation affects tooth stiffness by only 5%.[177] The influence of subsequent canal instrumentation and obturation lead only to a slight reduction in the resistance to fracture[177] and ultimately have little effect on tooth biomechanics.[144,177] Practically, 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 additional preparation, especially the loss of marginal ridges. The literature reports 20% to 63% and 14% to 44% reduction in tooth stiffness following occlusal and MOD cavity preparations, respectively.[45,98,144] 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).[78,89,102,128]

The presence of residual tissue in the cervical area (which comprises the *ferrule* for restorations) and a larger amount of residual 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.[7,24,169] Crown preparations with as little as 1 mm coronal extension of dentin above the margin doubles the fracture resistance of preparations, compared to those where the core terminates on a flat surface immediately above the margin[109,169]; therefore, a minimal 1-mm ferrule is considered necessary to stabilize the restoration.[169] However, the widths of preparation shoulder and crown margin do not appear to influence fracture strength.[2]

It is critical to understand that the most important part of the restored tooth is the tooth itself, and no restorative material or combination of materials will perfectly substitute for tooth structure.



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.



FIG. 22-2 Severe discoloration can significantly disturb esthetics, 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.

Esthetic Changes in Nonvital and Endodontically Treated Teeth

Color change and darkening of nonvital teeth is a common clinical observation (Fig. 22-2), and improper endodontic techniques can contribute to discoloration. For instance, inadequate cleaning and shaping can leave necrotic tissue in coronal pulp horns, resulting in tooth darkening. As well, root canal filling materials (gutta-percha and root canal cements) retained in the coronal aspect of anterior teeth can detract from the esthetic 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.[35,71,137]

Thin gingival tissue or in general, thin biotype is considered a negative factor for esthetic outcome of restorative and prosthetic treatment of discolored teeth.[116,117,124]

Endodontic treatment and subsequent restoration of teeth in the esthetic zone require careful control of procedures and materials to retain a translucent, natural appearance. It is therefore strongly recommended to avoid the use of potentially staining endodontic cements and to clean all material residues in the pulpal chamber and access cavity.

RESTORATIVE MATERIALS AND OPTIONS

Endodontic treatment 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 will 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.[140]

When properly cured, resin composites are highly esthetic, 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 which 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.[37,55] 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.[183] 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.[61,142]

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,[142] it was reported that the resistance to fracture of endodontically treated teeth is reduced by 69% in cases where MOD cavities are present.[143] 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, a recent clinical report indicates that direct in vitro fiber-reinforced composite restorations may represent a valuable alternative to conventional restorations of endodontically treated teeth.[38] 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.[167]

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.[94] 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.[62] 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 esthetic 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 are either variations of feldspathic porcelains (e.g., In-Ceram, Cerec, IPS Empress) or may be fabricated from other ceramic systems, including alumina, zirconia, or silica.[3,39] 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.[46,77] 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.[10] 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.[62,63] A recent 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.[29] Other investigators reported that composite resin MZ100 increased the fatigue resistance of overlay-type restorations in endodontically treated molars when compared to porcelain MKII.[103] Another recent 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 Core Materials heading). More frequently, the cementation of a post inside the root canal is necessary to provide retention for the core material and the crown.[172] 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 esthetics 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.[155] The post's ability to anchor the core is also an important factor for successful reconstruction, since 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.[112]

The Foundation Restoration: General Considerations

Although there are many materials and techniques 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.[6,85,106,134] 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.[101,181] A longer ferrule increases fracture resistance significantly.[86] 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. Within the same root, the shape of the canal

will vary between the cervical level and the apical foramen.[66] 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 which exhibit deep concavities on the furcal surface of their mesial root.[17,95] 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.[99] 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 is significantly increased 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 esthetics, 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 non-stiff 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 high-modulus posts.[105,126,150]

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 are all likely to occur.[160] 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.[48] 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.

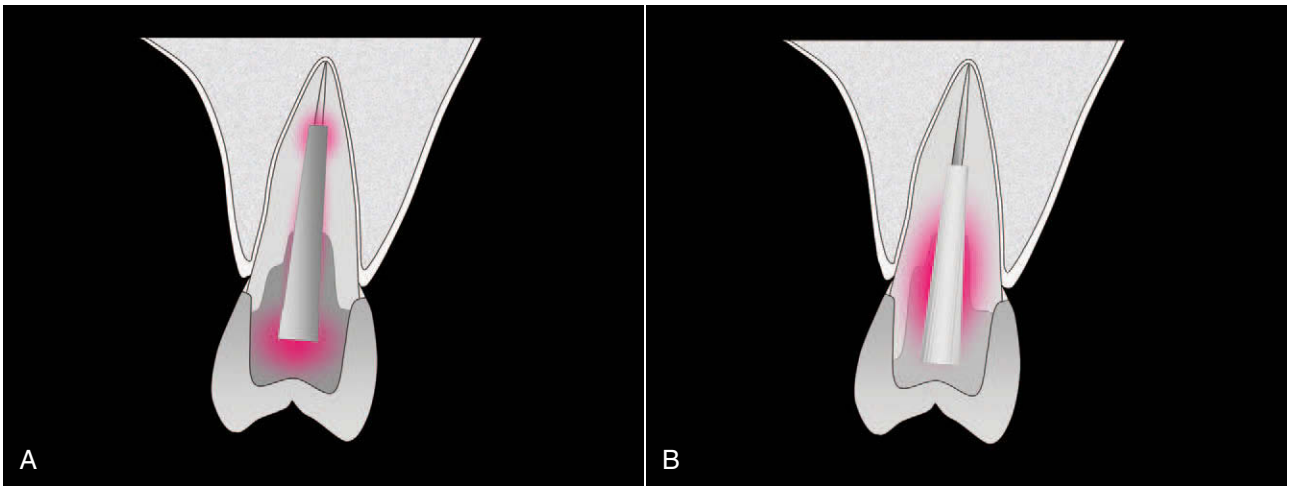


FIG. 22-3 A, Stress distribution within a metallic post and core foundation and residual tooth structure, according to photoelastic and FEM studies. The post is cemented and usually penetrates the apical portion of the root. Functional stresses accumulate inside the foundation, slightly around the post 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, 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 *38:733–743, 2007.*)

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 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.[28] 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 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.[96] A recent study indicates that the flexural strength of stainless steel posts is about 1430 MPa and that flexural modulus approximates 110 GPa.[136] 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.[171] 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 (see later).[123] 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.[146] 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.[83,158,170] Unfortunately, modern techniques for root canal preparation use tapered nickel-titanium (NiTi) rotary shaping files, which results in a very wide tapered and unretentive canal exhibiting a significant divergence from apical to coronal.[152] 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.[84] Posts designed with mechanical locking features in the heads and roughened surface texture can show better retention of the core.[30]

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.[67] Fibers are commonly 7 to 20 μm in diameter and are used in a number of different 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, esthetic qualities, and their ability to chemically bond to the polymer matrix.[51] A recent study indicates that the flexural strength of glass-, silica-, or quartz-fiber posts approximates 1000 MPa and that flexural modulus is about 23 GPa.[36] 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.[148,182] 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. Recent 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.[13,19,58] 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 which evaluated three types of bonded fiber posts, investigators reported 3.2% failure of 1306 fiber posts in recalls of 1 to 6 years.[57] 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.[163]

Zirconia Posts Zirconia posts are composed of zirconium dioxide (ZrO_2) partially stabilized with yttrium oxide and exhibit a high flexural strength. Zirconia posts are esthetic, partially adhesive, very 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.[12] 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.[1,133] 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.[41,44]

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.[140] Composite core materials have been shown to exhibit slightly better mechanical values than conventional materials, but improvements are negligible.[184] However, they appear to be superior to silver-glass-ionomer cement and amalgam.[31] 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.[27] However, no adhesive has been shown to completely eliminate microleakage at the margins of the restoration.[18] 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.[135] A recent 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.[56] 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.[8]

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. Thirty years ago, investigators described the amalcore.[119] 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.[87]

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.[108] Others have suggested the use of adhesive resins to bond amalgam to coronal tissue.[162]

Significant disadvantages of amalgam cores are the "non-adhesive 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.[180]

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.[165] 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, 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.[32]

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.[47,166]

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.[104] 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.[168]

One 6-year retrospective study reported a success rate higher than 90% using a cast post and core as a foundation restoration.[9] 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 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.[50] Contemporary resin-based luting cements have been shown to exhibit compressive strengths around 200 MPa and elastic moduli between 4 and 10 GPa.[25] 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.[11] However, bonding to root canal dentin may be compromised by the use of endodontic irrigants such as sodium hypochlorite, hydrogen peroxide, or their combination.[121] 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.[159] 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, Plymath Meeting, PA).[113] Other reports indicate that the contamination of the dentin walls by eugenol diffusing from endodontic sealers may also affect the retention of bonded posts.[70,176] 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.[110,179] 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.[111]

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.[54] 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.[53,174]

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.[49,65] 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.[15] It is generally accepted that bonding to dentin of the pulp chamber is

more reliable than to root canal dentin, especially at the apical level.[132] 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.[138]

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.[80,157]

A recent study indicates that bond strength to radicular dentin might be maximized by adopting procedures that compensate for polymerization stresses.[14] 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 will reduce 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, there are other reports indicating that resin-dentin bonds degrade over time.[23,60] 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 was recently 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) have been shown to produce more durable resin-dentin bonds made with single-step etch-and-rinse adhesives.[156]

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).[22,131] The release of MMPs such as collagenases has been evidenced in both coronal and root dentin of fully developed teeth of young patients.[154] 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.[21]

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.[114] 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.[141] Adhesion performance to dentin was found comparable to multistep luting cements, but bonding to enamel without prior phosphoric acid etching is not recommended.[76] 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 esthetic 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.[125]

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[120,173,178] (see Fig. 22-1; 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 canine and anterior guidance are maintained; 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 are likely to be reduced or lost and posterior teeth then submitted to more lateral stresses, generating higher demand for restorative materials.

The literature reports average biting forces varying 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.[59,75] 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 achieve stabilization of the restoration for improved short- and long-term service. However, in certain conditions like reduced tooth support, conventional materials are not obsolete.

Esthetic Evaluation and Requirements

Anterior teeth, premolars, and often the maxillary first molar, along with the surrounding gingiva, compose the esthetic zone of the mouth. Changes in the color or translucency of the visible tooth structure, along with thin soft tissues or biotype, diminish the chance for a successful esthetic treatment outcome.

Potential esthetic 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 esthetic 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 esthetic 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 to 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 form together the *foundation restoration* to support the coronal restoration of endodontically treated teeth. The recent 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 Fig. 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 restorative procedure does not necessarily lead to extreme biomechanical involvement and therefore allow 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 then 1.5 mm thickness). The three additional clinical factors which 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 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. In less favorable biomechanical conditions (group guidance, steep occlusal anatomy, bruxism, clenching, etc.), 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 which 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, since a well-bonded post can help absorb stresses more evenly throughout the remaining tooth structure. Benefit and increased risk of fissure and 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 esthetics. Metal or carbon post color can be masked with resin opaquer and gold post and core ceramized to enhance esthetic integration. Such procedures can help to approach a more ideal restoration biomechanical behavior through the fabrication of rigid but more esthetic foundations. Upper lateral and lower incisors, together with extremely thin biotype, are probably the only real esthetic 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 altogether a 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

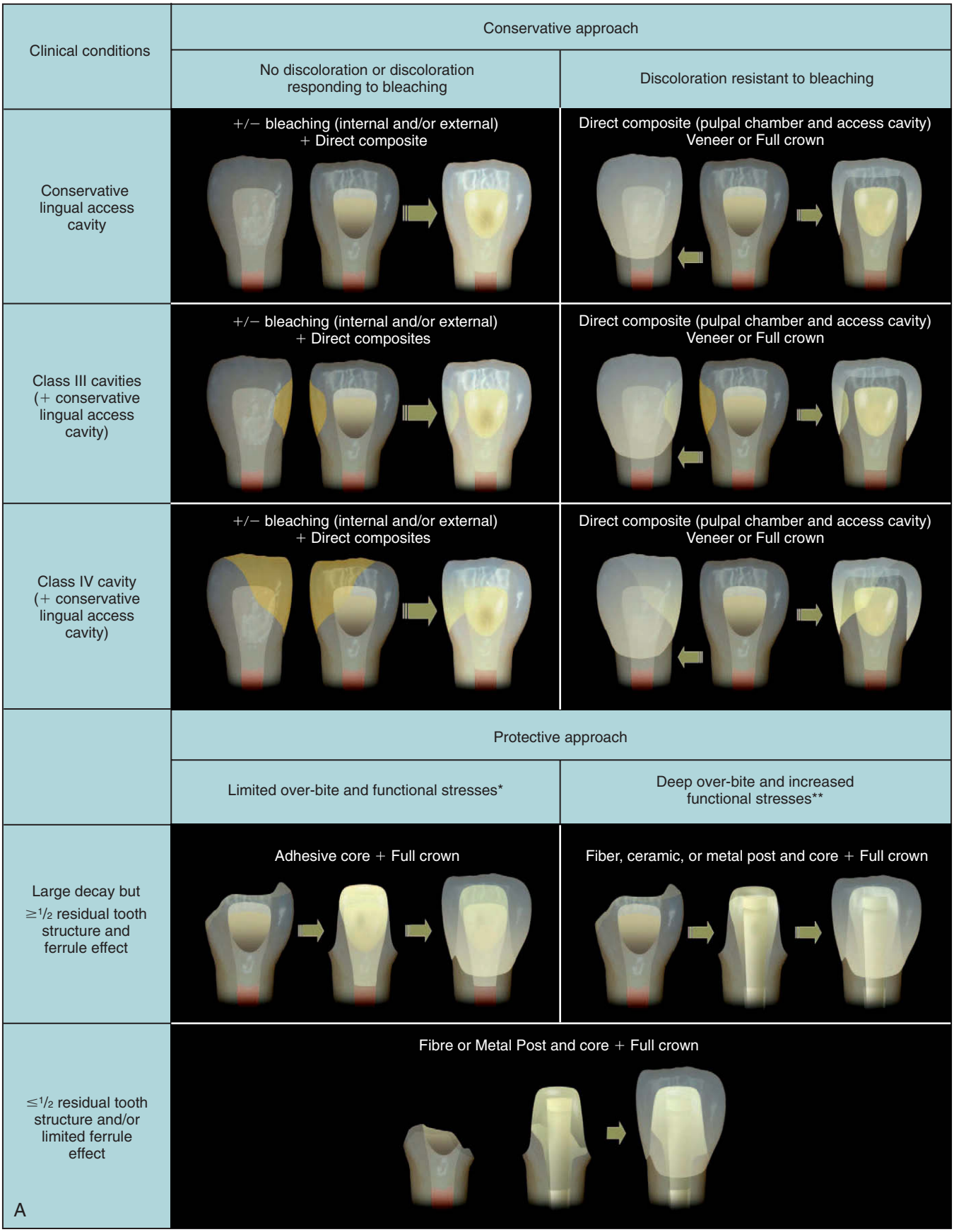


FIG. 22-5 A, Current recommendations for the treatment of nonvital anterior teeth. *Normal function and anterior guidance; **moderate to severe parafunctions and abnormal occlusion/anterior guidance.

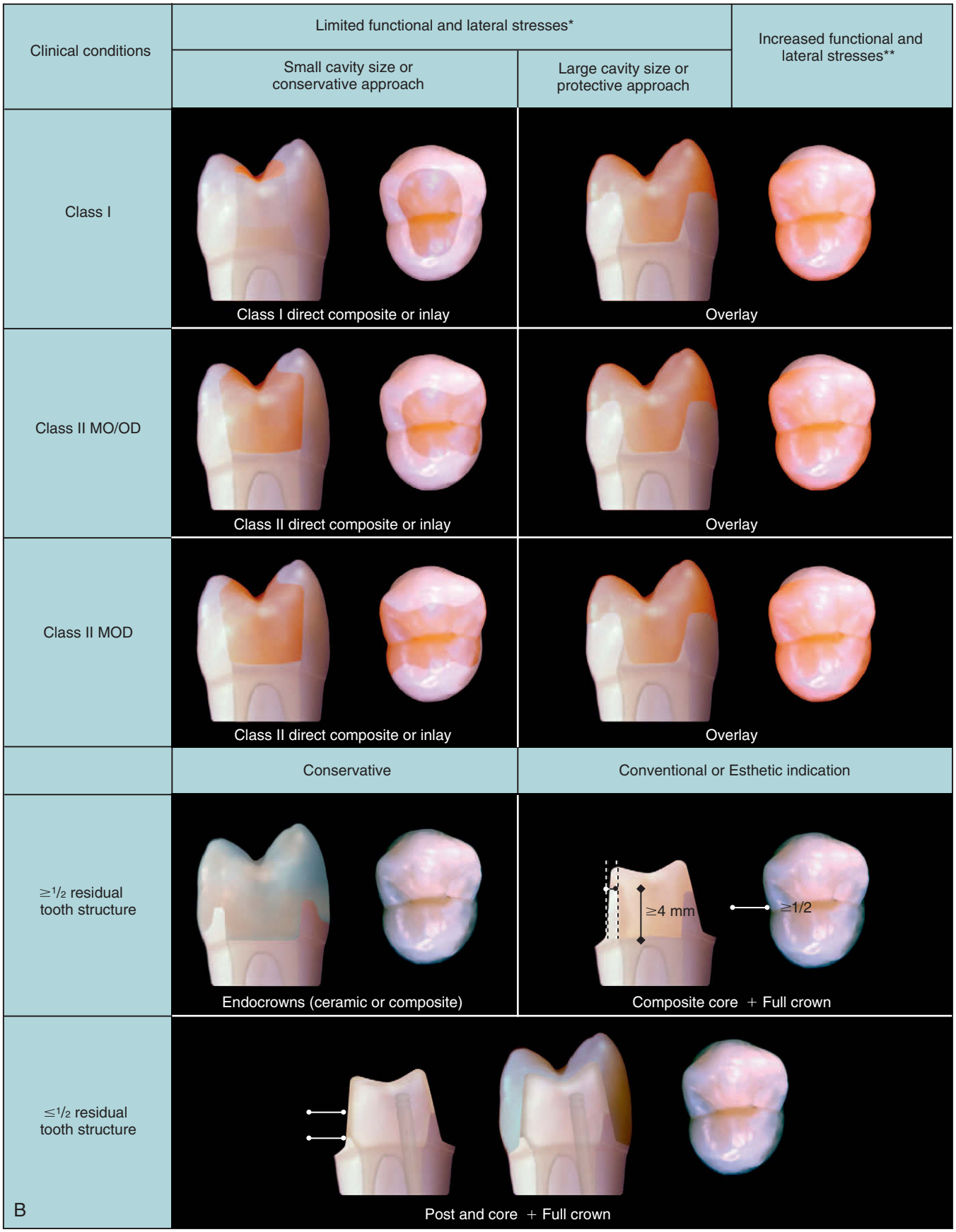


FIG. 22-5, cont'd B, Current recommendations for the treatment of nonvital posterior teeth. *Relatively flat anatomy and canine guidance, normal function; **group guidance, steep occlusal anatomy, parafunctions.

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 and/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 and/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; *Dual,* dual curing; *LC,* light curing; *PFM,* porcelain fused to metal; *SA,* self-adhesive.

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[43] (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 esthetic zone, the post should not detract from the esthetics of the coronal tooth structure, ceramic crown, or gingiva. Current restorative procedures allow fabrication of highly esthetic ceramic coronal restorations that have no metal substructure. When such restorations with remarkable lifelike color and vitality are selected, it usually implies the use of nonmetal esthetic 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 pulpal chamber floor) and 1.5 to 2 mm thickness, core and restoration stability are granted through macromechanical retention and/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 esthetic 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

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 3/4 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; *CP,* composite; *DBA,* dentin bonding agent; *Dual,* dual curing; *LC,* light curing; *PFM,* porcelain fused to metal; *SA,* self-adhesive.

roots or conical anatomy are contraindications to orthodontic extrusion. Once again, when a long-lasting, functional restoration cannot be predictably created, tooth extraction might be better than to pursue heroic efforts to restore extremely weak teeth, 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 Figs. 22-6 through 22-14.

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 the 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 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:

1. In case of nonadhesive cementation (metal posts only)[64]:
   - 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)[42,142]:
   - 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 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.[40] Figs. 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. Endocrown is a modification of the aforementioned concept, with extension of the restoration into the pulpal chamber as additional retentive and stabilization structure. Figs. 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 from:

- 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



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 help of a silicone index. **E-F,** Postoperative views. The two conservative restorations are shown after finishing and polishing, with rubber dam still in place **(E)** and after several weeks **(F)**. Note that esthetic integration can not 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 and/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,** Postoperative view. 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 esthetic 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 esthetic 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 esthetics. This approach is sometimes suitable to meet patient's high esthetic demands.

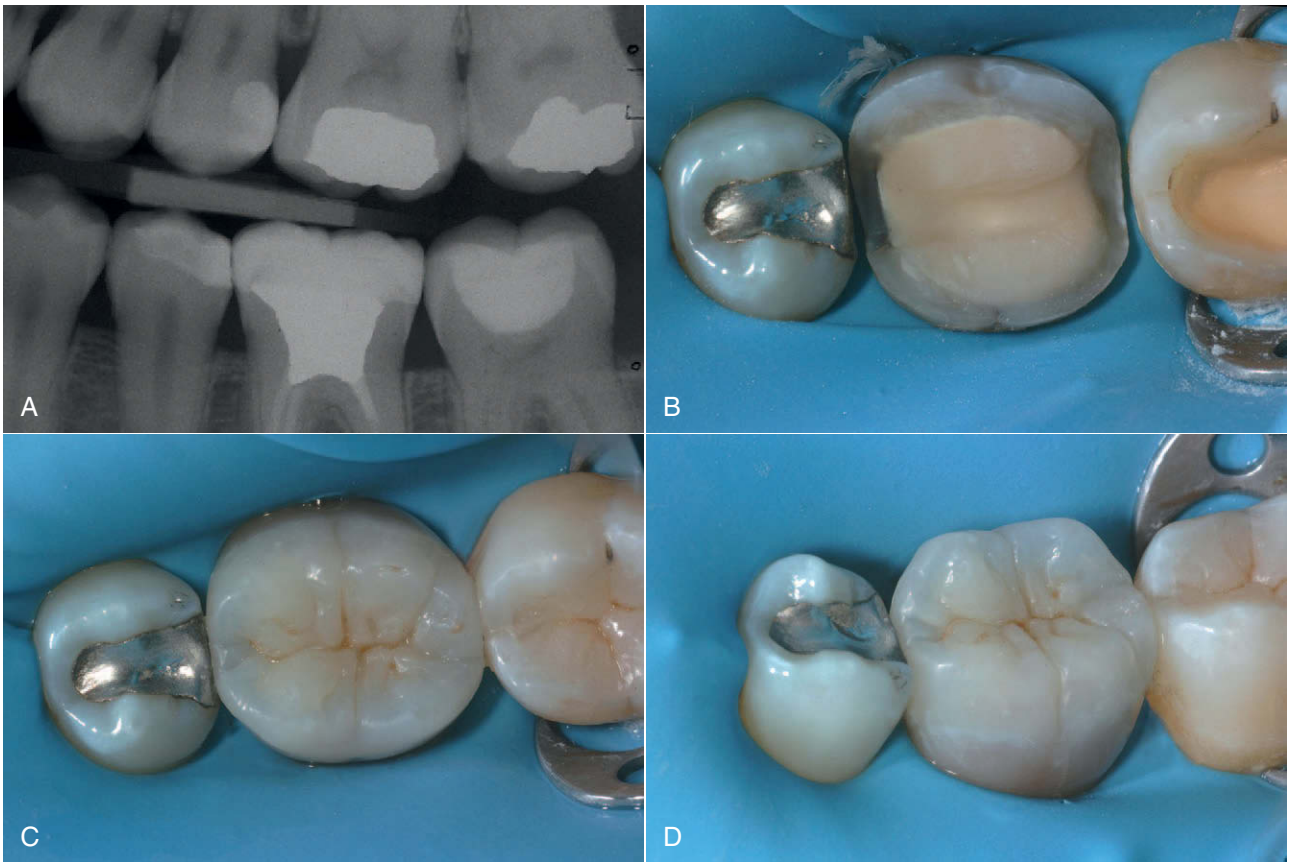


FIG. 22-9 Onlay/Overlay. Nonvital posterior tooth with sufficient amount of residual tissue to justify partial restoration. Preparation involves both proximal surfaces or only two surfaces, but in a less favorable occluso-functional environment (thin walls). No significant discoloration of visible buccal 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, to relocate cervical margins occlusally. **C-D,** After impression and model fabrication, the restoration is generated in the laboratory to optimize restoration anatomy, function, and esthetics. 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 esthetics and is a good alternative to full crowns.

Amalgam Core

A coronal-radicular amalgam foundation may be realized with or without post (Figs. 22-11 through 22-14). The amalgam coronal-radicular core without 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, homogenous 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 palatinal 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.



FIG. 22-10 Endocrown. Nonvital posterior tooth with reduced amount of residual cervical tissue but sufficient 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, to 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 esthetics. 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 *37:259–272, 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 restoring the appropriate coronal volume. Preparation of cores is made to manage optimal space for PFM crowns. **F,** Posttreatment result. This treatment approach is feasible for teeth already restored with amalgam cores and when PFM restorations are mandated. Satisfactory esthetics can be attained with intrasulcular restoration margins.

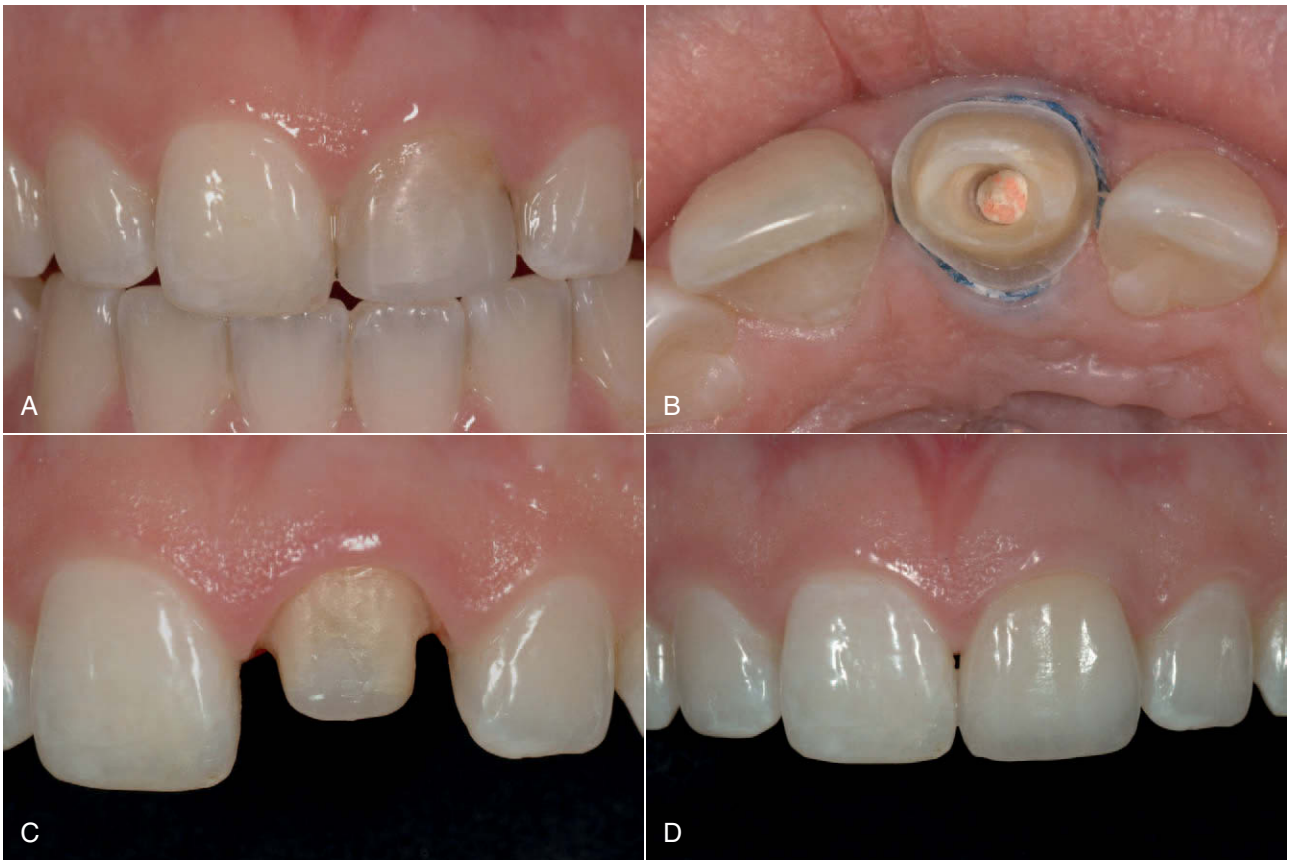


FIG. 22-12 Composite core (without post). Nonvital tooth with more than half the coronal structure left and sufficient wall thickness. Core stability is granted by adhesion, remaining pulpal chamber, and residual wall height. **A-B,** Preparation. Existing restorative material and all remnants of endodontic 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 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 esthetics. It is considered a good restorative solution for highly esthetic ceramic crowns placed on teeth with limited tissue loss and discoloration.

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 only for a limited time in the mouth 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 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



FIG. 22-13 Composite core with white post (resin reinforced fiber or zirconia post). Nonvital tooth with less than half the coronal structure left and/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, since 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 esthetic outcome. It is considered a good restorative solution for highly esthetic 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 and/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 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, since 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 esthetics. 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.



FIG. 22-15 Nonvital teeth with limited coronal structure and/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 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, since 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.

not different from 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 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 result 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 decisions about the restorative options available to restore endodontically treated teeth with predictable clinical results.

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CHAPTER

23

Pediatric Endodontics: Endodontic Treatment for the Primary and Young Permanent Dentition

PAULA J. WATERHOUSE, JOHN M. WHITWORTH, JOE H. CAMP, and ANNA B. FUKS

CHAPTER OUTLINE

THE PULP-DENTIN COMPLEX IN PRIMARY AND YOUNG PERMANENT TEETH
- Fundamentals of Dentin/Pulp Histology
- Dentinogenic Response to Injury
- Reactions to Dental Caries
- Reactions to Restorative Procedures
- Reactions to Dental Materials

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 Following 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, since they may present a risk to developing permanent successors and will in any case

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 primary teeth are to safeguard esthetics 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.[242]

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 thus focus on preserving pulp vitality, at least until dental development is complete (see also Chapter 16).

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 will be 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 (see also Chapter 12).

Fundamentals of Dentin/Pulp Histology

The pulps of primary and permanent teeth are histologically and functionally similar.[248,249] Odontoblasts are the dentin-forming cells that line the periphery of the pulp chamber and extend their cytoplasmic processes into its tubular structure. They become increasingly crowded as they retreat centrally, and tubular density therefore increases in deep dentin (Fig. 23-1). A cell-free zone is located just below the odontoblastic layer and contains an extensive plexus of nerves (Raschkow's plexus) and blood capillaries that may support and regulate odontoblast activity and provide sensory innervation to the pulp. Raschkow's plexus does not fully mature until the late stages of root formation, and pulp sensitivity tests may therefore be inconclusive in immature permanent teeth. Beneath the cell-free zone is the cell-rich zone of Höhl, a band of tissue containing uncommitted cells capable of differentiation into odontoblast-like cells should primary odontoblasts be lost.[110] The core of the dental pulp contains larger blood vessels and nerves which are surrounded by loose connective tissue.[204] The volume of perfusion through the incomplete apices of immature teeth is considerable and underpins the enormous reparative capacity of young pulps in the face of injury.

Chapter 12 provides a more detailed review of tissue structure and functions within the dentin-pulp complex, and Box 23-1 provides relevant key points.

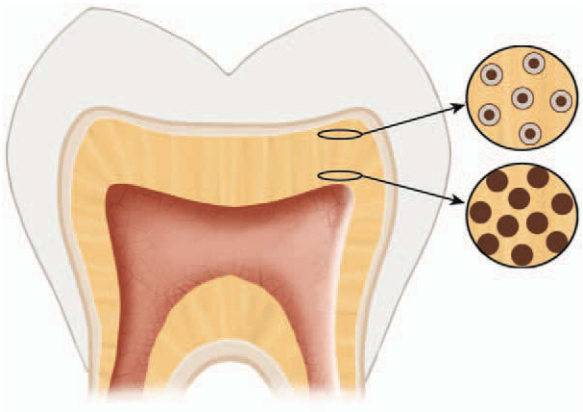


FIG. 23-1 Odontoblasts move closer as they retreat centrally on an ever-diminishing pulp circumference. Deep dentin contains wider and more closely arranged dentinal tubules, making it more porous than superficial dentin.

BOX 23-1

The Pulp-Dentin Complex—Key Points of Clinical Relevance

- Deep dentin is more porous than superficial dentin, and in primary and young permanent teeth, the dentin is thinner and more porous than in their mature permanent counterparts. The pulps of primary and immature permanent teeth are therefore at special risk from deep carious lesions, deep cavities, and traumatic injuries.
- Sensory innervation to the pulp does not mature until the late stages of root formation; pulp testing may therefore be inconclusive in immature teeth.
- Young, well-perfused pulps have enormous reparative capacity in the face of injury.

Dentinogenic Response to Injury

Without dentin, the pulp would be a highly sensitive and vulnerable organ, and in the absence of a pulp, the defensive capacity of dentin would be limited. The tissues are therefore inseparable and work together as a functional complex.

Key responses of the dentin-pulp to injury include the deposition of tertiary dentin, which increases the distance between the injury and the pulp, and a reduction in dentin permeability by the formation of sclerotic dentin.[309] Chapter 13 further discusses pulpal responses to injury.

The nature and quality of the tertiary dentin depend on its tubular structure and influence local dentin permeability. In the case of a *mild injury,* the odontoblasts responsible for primary odontogenesis can frequently survive the challenge and are stimulated to secrete tertiary, *reactionary dentin* (Fig. 23-2) beneath the site of injury.[275] Because the original odontoblasts are responsible for this matrix secretion, tubular continuity and communication with the primary dentin matrix will be present [see Fig. 23-2, *B*; Fig. 23-3, A].[184]

When the injury is *severe,* as in pulp exposure, the affected odontoblasts may die. Since odontoblasts are postmitotic cells with no capacity for renewal, their replacement must come from elsewhere. If suitable conditions exist in the pulp, a new

generation of odontoblast-like cells may differentiate, probably from the cell-rich zone, and secrete a tertiary, *reparative* dentin matrix (Fig. 23-4). Because this dentin is formed by a new generation of cells, discontinuity in the tubular structure will occur, with a subsequent reduction in tissue permeability (Fig. 23-3, *B*).[31]

A critical question that arises is "What are the factors responsible for triggering the stimulation of odontoblastic activity?" Although much still has to be learned about the molecular control of cell activity in general and odontoblastic activity in particular, one family of growth factors, the transforming growth factor superfamily (specifically TGF-βs)[110] has been reported to have extensive effects on the mesenchymal cells of many connective tissues.[180]

During tooth development, the odontoblasts secrete TGF-βs, and some of them remain sequestered within the dentin matrix. These TGF-βs may be released during any process leading to tissue dissolution, such as dental caries or the use of acid etching.[309] Dentin matrix should not be considered an inert dental hard tissue but rather a potential tissue store of a cocktail of bioactive molecules (particularly growth factors) waiting to be released if appropriate tissue conditions prevail.[309]

In contrast to reactionary responses, reparative dentinogenesis represents a more complex sequence of biologic processes. Migration and differentiation of pulpal progenitor cells must take place, creating a new generation of odontoblast-like cells before matrix secretion. A series of stereotypic wound-healing reactions will occur in the pulpal connective tissue, including vascular and cellular inflammatory reactions.

In vitro and in vivo experiments on reparative odontogenesis demonstrate that the noninflamed pulp constitutes an appropriate environment where competent pulp cells (potential preodontoblasts) can differentiate into new odontoblast-like cells, forming reparative dentin.[171,203,212]

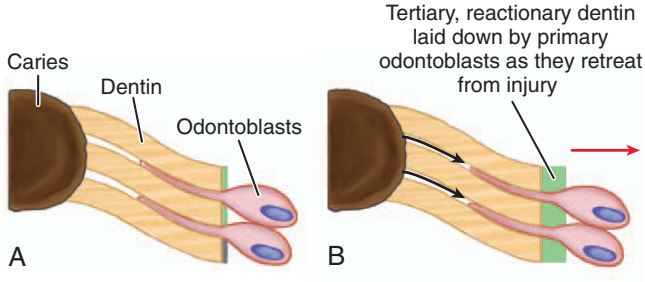


FIG. 23-2 Schematic representation of tertiary reactionary dentin. **A,** A mild insult to dentin, such as dental caries that does not extend deeply to threaten survival of primary odontoblasts. **B,** Liberation of dentin matrix components such as TGF-β leads to upregulation of odontoblast activity, with deposition of a reactionary dentin matrix whose tubular pattern is continuous with underlying tissue.

Reactions to Dental Caries

When the carious process advances from enamel into dentin, sclerotic dentin is formed by the apposition of minerals into

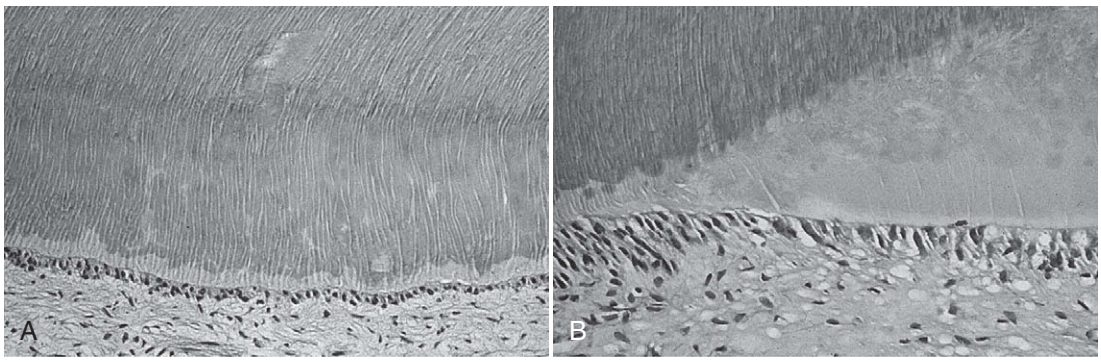


FIG. 23-3 Histologic sections of tertiary reactionary and reparative dentin. **A,** Reactionary dentin: histologic section showing tubular continuity. **B,** Reparative dentin: histologic section showing lack of continuity. *(From Pinkham JR, Casamassimo PS, McTigue DJ, et al:* Pediatric dentistry: infancy through adolescence, *ed 4, St Louis, 2005, Saunders; Courtesy Carlos Alberto S. Costa, DMD, PhD.)*

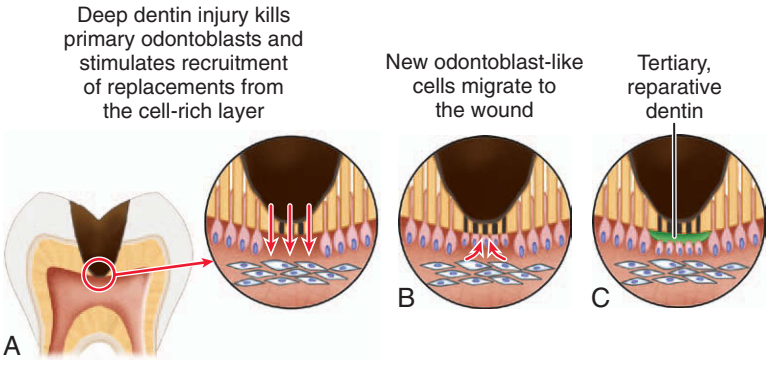


FIG. 23-4 Schematic representation of tertiary reparative dentin. **A,** A significant insult, such as deep caries, which kills primary odontoblasts in a localized area of pulp. **B,** New odontoblast-like cells differentiate from cell-rich zone. **C,** Secretion of a tertiary reparative dentin matrix commences. Tubular structure is irregular and has no continuity with underlying tissue.

and between the dentinal tubules (intratubular and intertubular dentin), and reactionary tertiary dentin is secreted. Sclerotic dentin can be seen in radiographs as a radiopaque area as the aforementioned apposition of minerals increases the radiopacity of this dentin.

The quality and amount of tertiary dentin depend on the depth and rate of progression of the carious lesion. The faster the lesion progresses, the poorer and more irregular the reactionary dentin. In addition, if the noxious irritant is too intense, the cytoplasmic processes of the odontoblasts degenerate, and "dead tracts" are formed.

When the carious process advances more rapidly than the elaboration of reactionary dentin, blood vessels of the pulp dilate, and scattered inflammatory cells become evident, particularly subjacent to the area of the involved dentinal tubules. If the carious lesion remains untreated, a frank exposure will eventually occur. The pulp will react, resulting in an acute inflammatory infiltrate, and the previously chronic pulpitis will become acute. A small abscess may develop under the region of the exposure, and cells of the chronic inflammatory series may accumulate farther away from the central area of irritation. The remainder of the pulp may be uninflamed (chronic partial pulpitis with acute exacerbation). As the exposure progresses, the pulp may undergo partial necrosis, followed in some instances by total necrosis.

Drainage is apparently the factor determining whether partial or total necrosis occurs. If the pulp is open and drainage can occur, the apical tissue may remain uninflamed or chronically inflamed. If drainage is impeded by food packing or a restoration, the entire pulp may become necrotic.[110]

Reactions to Restorative Procedures

The principal factors affecting the dentin-pulp complex during restorative procedures (e.g., cavity and crown preparation) are the cutting of the dentin, the generation of heat, and the desiccation of the tissue. When uninvolved dentin is operated on, as in extension for prevention or in crown preparation, tubules unprotected by reactionary dentin are cut. The tissue reaction that occurs is similar to that occurring with caries: intratubular and intertubular mineralization takes place, resulting in sclerotic dentin, followed by the formation of tertiary dentin. Remaining dentin thickness (RDT) is apparently the most significant factor determining pulp survival and the secretion of tertiary reactionary dentin. Maximal reactionary dentin was observed in a study wherein the cavity RDT was between 0.5 and 0.25 mm. The reduced reactionary dentin beneath cavities with an RDT below 0.25 mm (described in the same study) appeared to be related to reduced odontoblastic survival.[188] In these deep cavities, little more than 50% of the odontoblasts survived, whereas in shallow cavities, odontoblastic survival was about 85% or greater, and despite the cutting of the odontoblastic process, the cells responded by secreting reactionary dentin. The same authors demonstrated that the choice of restorative material influenced the secretion of reactionary dentin to a lesser extent than the RDT. The effect of cutting dentin can be observed histologically as a calciotraumatic band, which represents an interruption of the apposition between the secondary and tertiary dentin.

Pulp reactions to restorative procedures can be mild or severe, depending on the technique used. When the technique is gentle, the reaction will be mild, and minor alterations in the odontoblastic layer can be observed as a result of fluid accumulation. In a severe reaction, the nuclei of the odontoblasts may be aspirated into the dentinal tubules, hemorrhage may be present, and inflammation is extensive, sometimes resulting in cell necrosis. A gentle technique implies using appropriate cooling and minimal pressure. Cutting a cavity without using water cooling might lead to irreversible changes in the pulp because of the heat generated at the tip of the bur. The application of pressure increases the damage. Prolonged air blasts are also deleterious to the pulp. To prevent the generation of heat and damage to the pulp, the following measures should be taken: (1) The cavity preparation should be as shallow as possible, respecting the principles of cavity preparation and embracing contemporary partial and serial caries excavation techniques (see later); (2) suitably sized, sharp burs should be used, balancing the need for conservatism with the risks of localized overcutting with small burs; (3) appropriate cooling should be employed and minimum pressure exerted; and (4) excessive drying of dentin by air syringe should be avoided.[226]

Reactions to Dental Materials

There is substantial evidence that adverse responses within the dentin-pulp complex to restorative materials are more closely linked with microbial agents than with irritant or toxic components of the materials themselves.[22]

The routine lining of cavities has long been advocated as a means of pulp protection. Both calcium hydroxide ($Ca[OH]_2$) and zinc oxide eugenol (ZOE) cements have proven track records for promoting pulp health by destroying residual microorganisms in the depths of cavity preparations.[14,69,147,153] The antimicrobial and remineralizing potential of glass ionomer cements has been recognized during the last 2 decades,[166] and the importance of marginal seal has been supported by evidence on favorable pulp outcomes following bonded composite restorations.[42-44,145,277,292]

Researchers[201] have begun to use the term *artificial enamel* to describe their aspirations of hybrid-layer formation[202] and impervious, pulp-protecting barriers formed by resin-based materials. The response of the pulp to direct contact with dental materials is of special importance in the primary and young permanent teeth of children where pulp capping, pulpotomy, and apexification procedures are important modalities of treatment.

A variety of materials, medicaments, antiseptics, anti-inflammatory agents, antibiotics, and enzymes have been used as pulp-capping agents, but $Ca(OH)_2$ has been the standard by which all others were judged.[9,58,110] Before 1930, when Hermann[115] introduced $Ca(OH)_2$ as a successful pulp-capping agent, pulp therapy consisted of devitalization with arsenic and other fixative agents. Hermann demonstrated the formation of "secondary dentin" (now termed *tertiary dentin*) over the amputation sites of vital pulps capped with $Ca(OH)_2$.

In 1938, Teuscher and Zander[296] introduced $Ca(OH)_2$ in the United States, confirming complete dentinal bridging with healthy radicular pulp under $Ca(OH)_2$ dressings. Further reports firmly established $Ca(OH)_2$ as the pulp-capping agent of choice. After these early works, many studies have reported on various forms of $Ca(OH)_2$, with success rates ranging from 30% to 98%.[82] The dissimilar results have been attributed to variables in study design, including the model (human or

animal), tooth selection, area of the pulp involved (coronal or cervical), type of $Ca(OH)_2$ employed, outcome criteria, and study duration.

When $Ca(OH)_2$ is applied directly to pulp tissue, necrosis of the adjacent pulp tissue and inflammation of the contiguous tissue occur. Dentin bridge formation occurs at the junction of the necrotic tissue and the vital inflamed tissue. Although $Ca(OH)_2$ works effectively, the exact mechanisms are not fully understood. Compounds of similar alkalinity (pH 11) cause liquefaction necrosis when applied to pulp tissue, but $Ca(OH)_2$ maintains a local state of alkalinity necessary for bone or dentin formation. Beneath the region of coagulation necrosis, cells of the underlying pulp tissue differentiate into odontoblast-like cells and elaborate dentin matrix.[110]

Occasionally, in spite of successful bridge formation, the pulp remains chronically inflamed or becomes necrotic. Internal resorption may occur after pulp exposure and capping with $Ca(OH)_2$. In other cases, complete mineralization of the remaining pulp tissue occludes the canals and greatly complicates any future attempts at root canal treatment.

It was postulated[330] that calcium diffused from a $Ca(OH)_2$ dressing into the pulp to participate in reparative dentin formation. Experiments with radioactive ions, however, have shown that calcium ions from the circulation, not from the $Ca(OH)_2$ pulp cap are involved in bridge formation.[16,227,260] Calcium hydroxide promotes bridge formation by creating low-grade irritation in the underlying pulpal tissue, a phenomenon observed even after short-term application and removal of the material.[54]

Different forms of $Ca(OH)_2$ have produced markedly different responses when applied as a pulp cap.[291,305] The reactions to Dycal (DENTSPLY/Caulk, Milford, DE), Prisma VLC Dycal (DENTSPLY/Caulk), Life, and Nu-Cap (GC America, Alsip, IL) have been shown to be similar.[279,280,291] The chemically altered tissue created by application of these compounds is resorbed first, and the bridge is then formed in contact with the capping material.[302,305] With $Ca(OH)_2$ powder and nonsetting products such as Pulpdent Paste (Pulpdent Corp, Watertown, MA) the bridge forms at the junction of the chemically altered tissue and the remaining subjacent vital pulp tissue. The altered tissue degenerates and disappears, leaving a void between the capping material and the dentin bridge. For this reason, a bridge can be visualized better on radiographs with products such as Pulpdent Paste than with the other commercial compounds. The quality of the dentin bridging was equally good with either material. Investigators have, however, highlighted the frequent occurrence of porosities and tunnel defects in $Ca(OH)_2$-generated dentin bridges.[48] A related study[47] further demonstrated long-term evidence that $Ca(OH)_2$ becomes softened and allows leakage, resulting in recurrent pulpal inflammation and necrosis after 1 to 2 years. Despite its widespread use, the body of good evidence on $Ca(OH)_2$ pulp capping is surprisingly limited,[214] and efforts to improve the body of evidence or develop well-documented alternatives are justified.

Direct Bonding of Pulp Exposures

Although direct bonding of pulp exposures is controversial, it has been advocated by numerous researchers.[42,213] This may be explained by investigators[45,46] who have demonstrated that immediate and long-term biologic seal are more important to the healing of pulp exposures than the stimulatory effects of any particular wound dressing material. This was revealed graphically in surface-sealing experiments[44,45] where severe pulp inflammation followed direct pulp capping with composite, silicate cement, zinc phosphate cement, and amalgam. When similar pulp caps were surface sealed with ZOE to prevent microbial ingress, cell reorganization and dentin bridge formation occurred under the acid cements and composite. Although healing occurred beneath the surface-sealed amalgam, there was no evidence of bridge formation. The conclusion is that in the absence of microbial contamination, the pulp has an inherent healing capacity, with low-grade irritation promoting hard-tissue repair. These findings agree with experiments[142,222] on gnotobiotic animals that showed pulpal healing in the absence of bacterial contamination, whereas the introduction of bacteria prevented healing.

Numerous bonding materials and composite resin systems have been tested as pulp-capping agents, with reported healing of the pulp and deposition of hard tissue at the exposure site.[4,43-45,106,213,323] Unanimity exists among these researchers that healing does not depend on the material but on its capacity to provide a biologic seal.

Although direct bonding of pulp exposures has gained widespread popularity, it should be noted that no long-range histologic studies have been published. Also, unfavorable effects with direct bonding have been reported. In a comparison of acid-etched, bonded, composite pulp caps, and Dycal, Pameijer and Stanley[218] found 45% versus 7% pulpal necrosis and 25% versus 82% dentin bridging, respectively. Another report[112] comparing All Bond 2 (Bisco, Schaumburg, IL) with $Ca(OH)_2$ showed persistent inflammatory reactions and hyaline alteration of extracellular matrix inhibiting complete pulpal repair or bridge formation in the bonded specimens. Conversely, the $Ca(OH)_2$ specimens produced complete dentin bridge formation. These results led the authors to conclude that direct bonding is not recommended for human pulp, and vital pulp therapy using acidic agents and adhesive resins is contraindicated.

This remains a controversial area with no universally-adopted conclusions. However, the advent of new materials such as mineral trioxide aggregate (MTA) offer much promise in pulp capping and may make the debates over bonded pulp caps academic.

Mineral Trioxide Aggregate

Favorable pulp responses have been reported following the use of MTA.[1,140,198,200,228] When compared with $Ca(OH)_2$, MTA produced significantly more dentinal bridging in a shorter period of time with significantly less pulpal inflammation. Dentin deposition also began earlier with MTA.

MTA is a biocompatible material with an antibacterial effect similar to that of $Ca(OH)_2$ and the property of providing a biologically active substrate for cell attachment. This feature makes it effective in preventing microleakage and improving the prognosis of treatment. MTA has been shown to promote pulp healing with dentin bridge formation and minimal inflammatory reaction in exposed pulps in monkeys[228] and humans,[200] and its dentinogenic effect in short-term capping experiments was demonstrated in dogs.[71] The material was approved for use by the United States Food and Drug Association (FDA). Following promising preliminary studies,[2] it has been shown to provide impressive results for pulp capping (Fig. 23-5) and

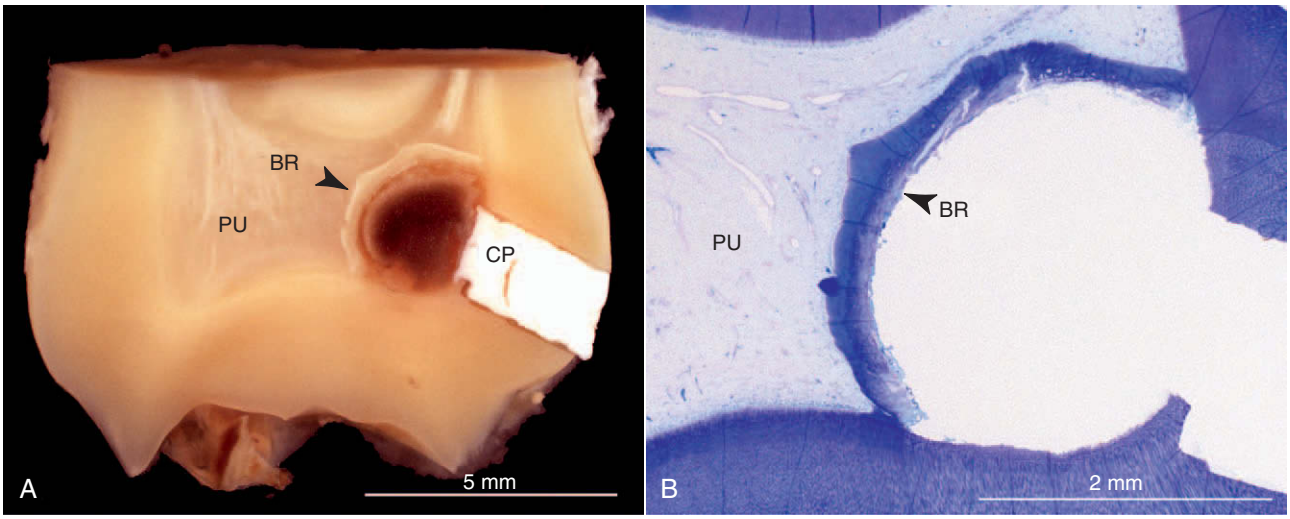


FIG. 23-5 Pulpal response to mineral trioxide aggregate (MTA) capping after 3 months observation. Distal macrophotographic view of the mesial half of a maxillary left third molar (tooth 28 [FDI]) shows remnants of restorative and capping material (CP) and a distinct hard-tissue bridge (BR) across the exposed pulp (PU). The photomicrograph **(B)** is part of a histologic section of the specimen in **(A)**. Note mineralized hard-tissue barrier (BR) stretching across full length of exposed pulp **(B)**. *(Reproduced from Nair PNR, Duncan HF, Pitt Ford TR, Luder HU: Histological, ultrastructural and quantitative investigations on the response of healthy human pulps to experimental capping with mineral trioxide aggregate: a randomised controlled trial.* Int Endod J *41:128, 2008.)*

pulpotomy treatments in young permanent teeth[27,326] and for pulpotomies in the primary dentition.[3,63]

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[78] and Nelson and Ash,[205] there are 12 basic differences between primary and permanent teeth (Fig. 23-6):

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 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 permanent teeth.
6. The facial and lingual surfaces of primary molars converge occlusally so that the occlusal surface is much narrower in the faciolingual 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 nearer the cervical area and more at the apex than do the roots of permanent molars.



FIG. 23-6 Cross section of primary and permanent molars. Divergence of the primary molar roots allows space for the developing permanent premolar.

9. The enamel is thinner (approximately 1 mm) on primary teeth than on permanent teeth, and it has a more consistent depth.
10. The thickness of the dentin between the pulp chamber and enamel in primary teeth is less than that 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.

Root Formation

According to Orban,[215] 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

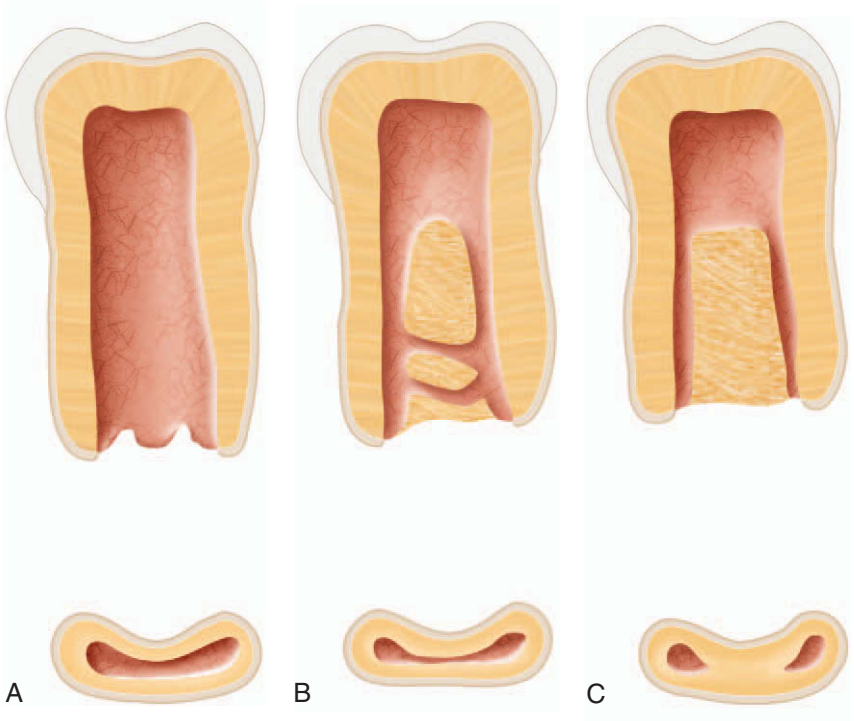


FIG. 23-7 Faciolingual cross section of the mesial root of a mandibular primary molar. **A,** Formation of the root at the time the root length is completed; only one canal is present. **B,** Differentiation of the root into separate canals by the continued deposition of dentin (*shaded areas*). Small fins and branches are present between the two canals. **C,** Canals are completely divided; root resorption has begun.

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. 23-7, *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 there is formation of dentin islands within 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. 23-7, *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. 23-7, *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 of time than in the permanent tooth because of the shorter length of the primary roots.

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. 23-6).[204]

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. Figs. 23-8 and 23-9 show silicone models of root canals, and Fig. 23-10 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 computerized tomography[221] 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.[205] To understand some of the variations in the primary root canal systems requires an understanding of root formation (see previous discussion and Box 23-2).

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. 23-8 and 23-9). The permanent tooth

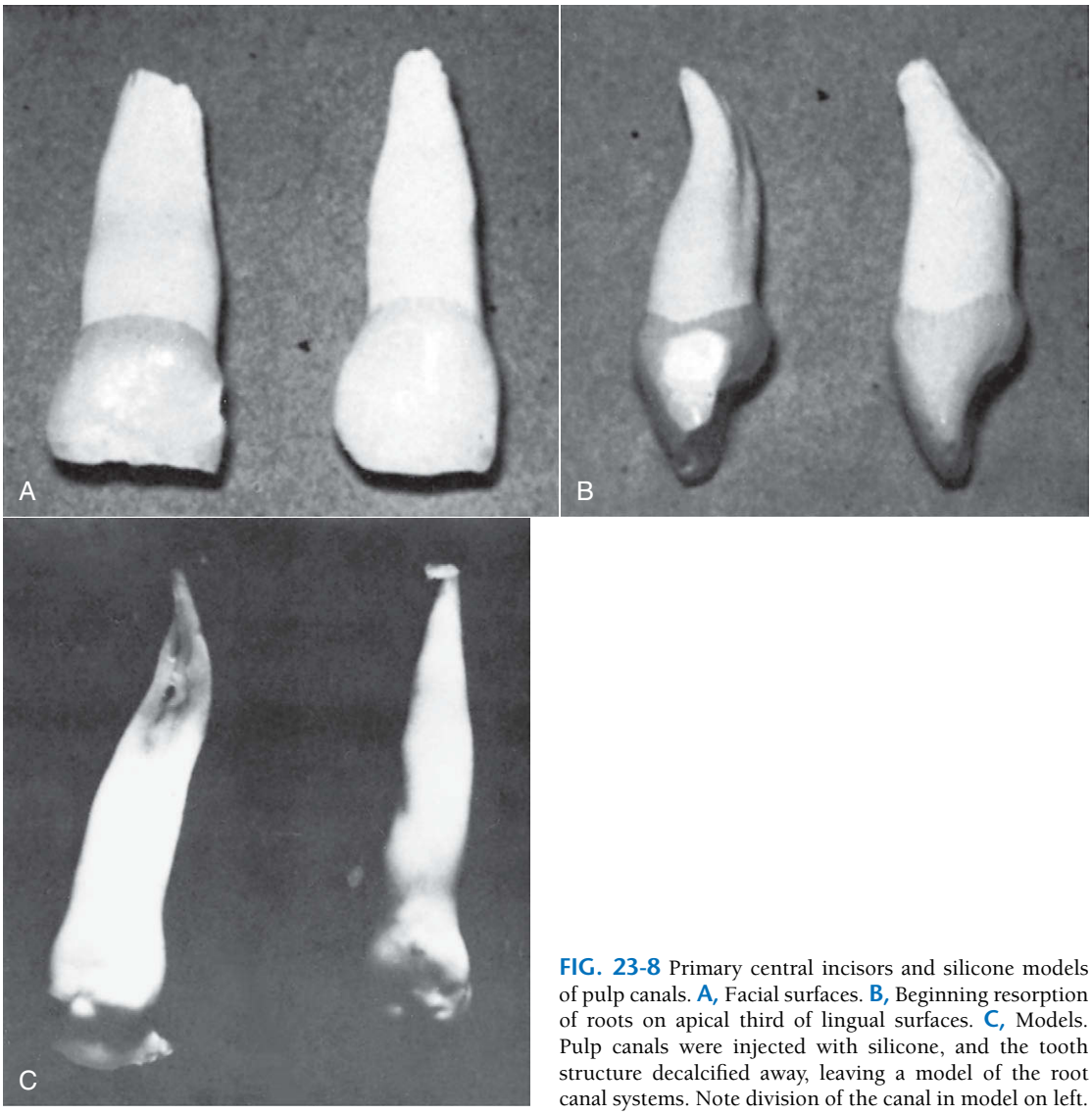


FIG. 23-8 Primary central incisors and silicone models of pulp canals. **A,** Facial surfaces. **B,** Beginning resorption of roots on apical third of lingual surfaces. **C,** Models. Pulp canals were injected with silicone, and the tooth structure decalcified away, leaving a model of the root canal systems. Note division of the canal in model on left.

BOX 23-2

Root Canal Anatomy 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.

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. 23-8, *A*).

Maxillary Incisors

The root canals of maxillary primary incisors are almost round in cross section but somewhat compressed in a faciolingual direction. Commonly these teeth have one canal without bifurcations. Apical ramifications and accessory and lateral canals are rare but may occur (see Fig. 23-8).[331]

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.[331]

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. 23-9).[331]

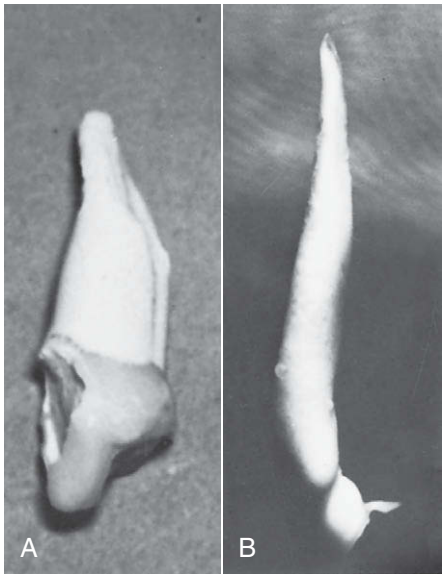


FIG. 23-9 Maxillary primary canine and silicone model of the root canal. **A,** Mesial surface. **B,** Model of the root canal.

Primary Molars

Often, primary molars have the same number and position of roots as the corresponding permanent molars (see Fig. 23-10). 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. 23-7, *B* and Fig. 23-10).

In primary teeth, the deposition of secondary dentin has been reported to occur after root-length completion.[24,117,134] 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.[117]

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

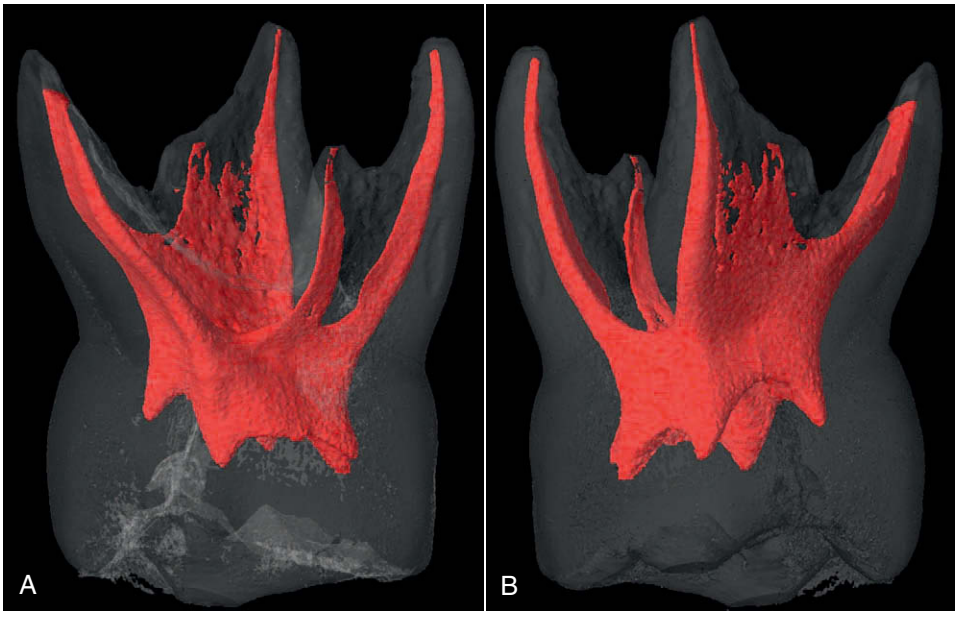


FIG. 23-10 Three-dimensional tomographic images of a maxillary second primary molar. **A,** Mesial view of primary molar with four root canals. Note the fins of pulp tissue between distofacial and palatal canals. **B,** Same tooth from the distal view. Note wide area of pulp tissue connection between distofacial and palatal canals. *(Courtesy Paul Dummer and Sue Bryant, Cardiff University, UK.)*

network between the facial and lingual aspects of the root canals (see Fig. 23-10).

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%.[117,331]

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.[204,331]

Fusion of the palatal and distofacial roots occurs in approximately a third of maxillary primary first 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.[117,331]

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.[117,331]

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.[117,331]

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 demand emphasis for 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 empirical treatment and anecdotal case reports. Assumptions regarding pulpal status prior to 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.[58,176,282]

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. Distinction should, wherever possible, be made between provoked and spontaneous pain.

Provoked pain is usually triggered by a thermal or 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. 23-11), 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 wake the child from sleep. In both primary and young permanent teeth, spontaneous pain and provoked pain which continues long after the causative factor is withdrawn are usually associated with extensive, irreversible pulpal inflammation extending into the root canals.[105] 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. Since 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 regenera-

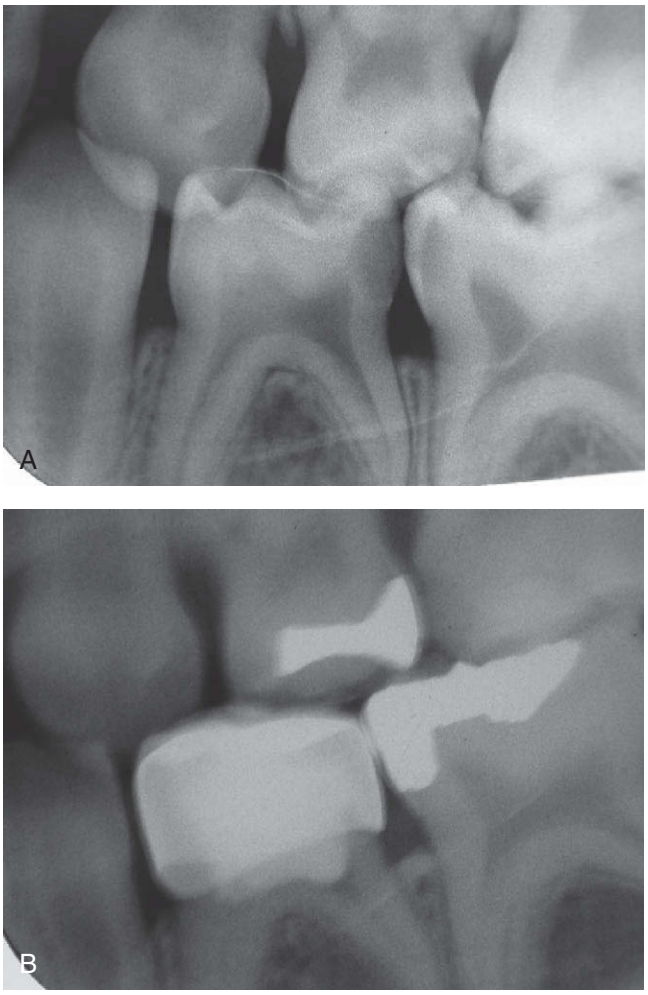


FIG. 23-11 Pain and bone loss caused by food impaction in interproximal space between primary molars. **A,** First and second mandibular primary molars with caries and marginal bone loss caused by food impaction. **B,** Healthy bone after restoration of teeth.

tive 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, however, neglect to conduct a proper clinical examination, since other conditions such as papillitis caused by interproximal food impaction may mimic pulpitic pain.

Equally, the absence of pain should not encourage clinical complacency, since varying degrees of pulp degeneration or even complete necrosis can be encountered without any report of pain. Children may thus present without any complaint, despite extensive carious lesions and draining parules. Those who have developed early childhood caries (e.g., nursing bottle caries), may equally have no experience of their teeth feeling any other way and have no special pain history to report.[226]

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 draining parules may strongly suggest pulpal pathoses. In addition, attention should be paid to teeth with extensive fractured and missing restorations or restorations with carious marginal breakdown. 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 before exteriorization. Bone destruction following a chronic dentoalveolar abscess can also be detected by palpation.

Though 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 ipsilateral 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.

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; one author[226] has recommended the 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.[9] Although these tests may indicate the presence of some vital responsive tissue, they will 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. Equally, young patients may give unreliable responses to such provocation tests because of apprehension, fear, or general management problems.

The unreliability of electronic methods in immature permanent teeth has been reported in several studies.[91,92,94,148] One author[148] 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 ascertaining 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.[91,92,94,148] Heat is unreliable as a diagnostic test in young children.[92]

Laser Doppler flowmetry has been reported[68,272,328] to be reliable for diagnosing pulpal vitality in immature permanent teeth, but the equipment has not been perfected for clinical use[68] and is cost prohibitive. It is also affected by blood pigmentation in discolored crowns.

Radiographic Examination

Clinical examination should be supplemented where appropriate with high-quality radiographs. Interradicular 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, exposed on a small film by a paralleling technique. The integrity of the lamina dura of the affected tooth should be compared with that of adjacent or contralateral teeth.[226]

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 antimere 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. What may appear as an intact barrier of secondary or tertiary dentin overlying the pulp may actually be a perforated mass of irregularly calcified and carious dentin overlying a pulp with extensive inflammation.[58]

The presence of calcified masses within the pulp is an important diagnostic sign (Fig. 23-12). Mild, chronic irritation to the pulp stimulates tertiary reactionary dentin formation. When the irritation is acute and of rapid 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.[58] 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. 23-12). Pathologic bone and root resorption indicates advanced pulpal degeneration that has 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,[105] 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 has become advanced enough to be seen on radiographs, there is usually a perforation of the root by the resorption (Fig. 23-13). In some instances, however, the process is reversible and self-correcting, and the resorbed area becomes filled with mineralized tissue.[110,219] 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).[110,219]

In immature permanent teeth, it is often difficult to assess the extent of apical closure. Plain-film images are frequently misleading, since under normal conditions, they show the mesiodistal plane of the tooth but give little information in 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 the mesiodistal. 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 which are apically diverging in the faciolingual view may be parallel or converging in the mesiodistal view. Again, contemporary three-dimensional imaging techniques are beginning to improve understanding, both in the research laboratory (Fig. 23-14) and in the clinic.[221]

In summary, radiographs add to the diagnostic process by visualizing the presence or absence of:

1. Deep caries with possible or definite pulp involvement.
2. Deep restorations close to a pulp horn.



FIG. 23-12 Calcified mass in the pulp chamber. There is internal and external root resorption. Calcified mass *(arrow)* is an attempt to block a massive carious lesion. Because of resorption, this tooth should be extracted. Note bone loss in the bifurcation area.

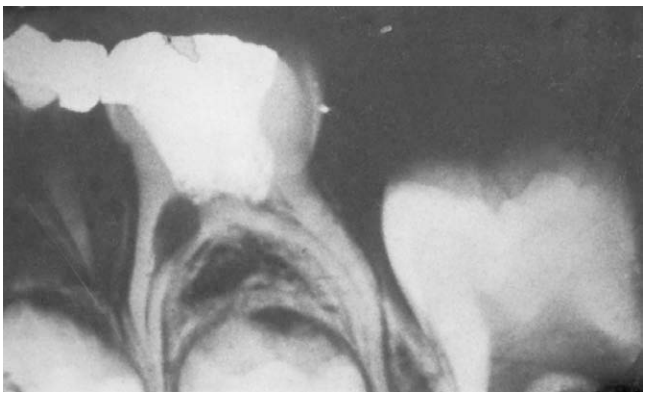


FIG. 23-13 Pulpotomy failure. Note internal resorption that has perforated the pulp chamber floor and resulted in a furcal radiolucency.

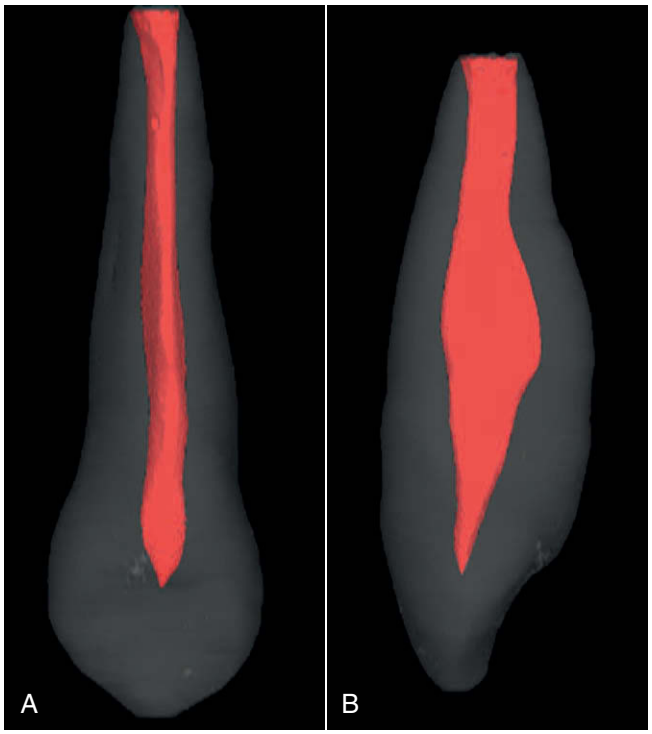


FIG. 23-14 Three-dimensional tomographic images of an immature maxillary canine. **A,** Faciolingual view. **B,** Mesiodistal view. Such imaging techniques are bringing new anatomic awareness to both the research laboratory and the clinic. *(Courtesy Paul Dummer and Sue Bryant, Cardiff University, UK.)*

3. 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 Chapter 17.
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.[226]

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[58,226,282] or deep purple colored[226] 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),[58,282] 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. 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 nature of 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.[226]

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 MTA pulp cap may be a better candidate for a pulpotomy, apexification, or pulp regeneration (see later section on immature permanent teeth).

Investigators[105] 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, including percussion, electrical pulp test, thermal tests, mobility, and history of pain, was obtained. 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 following vital pulpotomy in extensively carious primary molars has been reported.[319] Thirty-nine primary molars with no history of spontaneous pain had blood samples harvested from the radicular pulp stumps immediately following 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 following 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 empirical clinical findings to diagnose the inflammatory status of the pulp.

Diagnosis Following Traumatic Injuries in Children

The details of trauma management are considered in Chapter 17. Injuries to the primary dentition are common, occurring in 1 in 3 children by the age of 5 years.[11,99] Diagnosis following traumatic injuries requires consideration of other factors in addition to those previously discussed. The most frequent injury in the primary dentition is tooth displacement due to less dense bone and shorter roots. Healing varies from normal without sequela to canal calcification or pulpal necrosis. Canal calcification may vary from an amorphous material resembling osteodentin to a partial or complete closure of the canal.[121,247]

Pulp Diagnosis and Treatment Planning After Trauma

Treatment guidelines are almost nonexistent concerning healing and complications following trauma in primary teeth. The literature is devoid of histologic or microbiologic studies in this area. Borum and Andreasen[29] in a study of 545 traumatized primary maxillary incisors 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 calcific obliteration of the root canal than those which had been luxated.

When making a decision upon treatment, the proximity of the primary tooth to the permanent successor is an important consideration. Treatment least likely to damage the permanent tooth should be chosen.[9,29] Studies present conflicting data on the merits of treating or extracting traumatized primary teeth. Some have suggested no relationship,[11,12] others have shown more extensive developmental disturbances if the primary tooth is treated and preserved.[266]

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.[29,125]

Pulpal necrosis ranging from 50% to 82% is reported[29,120,125,263] in traumatized primary incisors with dark gray discoloration, compared to 25% in those with no discoloration. One author[49] points out that color change in the absence of other clinical findings is unreliable. 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.[130,136] 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[8,38] and transient apical breakdown up to 4 months' duration[38] have 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 which 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 a deep carious lesion in the absence of signs or symptoms of irreversible pulp pathosis.[265] 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 clinical and radiographic examination, direct evaluation of the cavity preparation, and a good knowledge of tooth anatomy and the caries process.[14]

The aims of IPT are[250] to arrest the carious process, provide conditions conducive to the formation of reactionary dentin, and promote remineralization of the altered dentin that was

left. This in turn is expected to promote pulpal healing and preserve/maintain the vitality of the pulp.

Technique

In outline, the clinical steps involved may be divided into stages involving partial caries removal, placement of an antibacterial agent, and restoration of the crown in a way that provides optimum coronal seal.

Some studies using IPT in primary teeth advocated a two-stage approach.[70,305] After initial partial caries removal without local anesthetic, the cavity was restored for 1 to 3 months, using a reinforced ZOE cement or glass ionomer restoration. Following this, further caries removal and definitive restoration was undertaken using local anesthetic. 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.[72] 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.[278]

The following technique is based upon a recommended approach[250]:

1. Local anesthetic
2. Isolation with 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 only be used to remove caries at the dentin-enamel junction and angled outward at the dentin-enamel junction, taking care not to produce a pulp exposure.
5. Placement of an appropriate lining material such as glass ionomer cement, hard-setting $Ca(OH)_2$, ZOE, or a directly bonded restoration
6. Definitive restoration providing optimum coronal seal, such as an adhesive restoration or preformed metal (stainless steel) crown

Unfortunately, there is presently insufficient evidence to promote a definitive choice for the lining material placed over the residual stained dentin.[250]

Although additional prospective clinical evaluation of IPT is required, studies involving the primary dentition show good success rates of over 90% at 3 years.[6,70,72,316] However, it has been suggested that successful outcome is highly dependent upon optimum coronal seal to eradicate the nutrient supply to residual cariogenic microorganisms. When intracoronal amalgam restorations were compared with extracoronal preformed metal (stainless steel) crowns post IPT, failure was 7.7 times more likely to occur with the use of amalgam. Similarly, adhesive restorations were also reported to provide optimum coronal seal after pulp therapy.[108] Therefore, to secure the best possible outcome following IPT, or indeed any pulp therapy for the primary tooth, definitive restoration should involve a bonded restoration and/or preformed metal (stainless steel) crown.

The Hall Technique

This emerging technique of managing dental caries in primary molars is noteworthy within this section of indirect pulp therapy. The technique itself was introduced by 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 from 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 Dr Hall's records indicate that her technique may have similar results to more conventional approaches in the primary care setting.[132]

Subsequently, a randomized, controlled clinical trial was undertaken comparing the Hall technique with conventional restorations in carious primary molars in primary care.[131] Interestingly, the Hall technique was preferred to conventional restorations by the majority of children, guardians, and clinicians. After a review period of 2 years, comparing the teeth managed using Hall preformed metal (stainless steel) crowns 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 already ongoing debate related to whether restorative treatment provided by general clinicians in primary care is an effective way of managing caries in the primary dentition.[74,183]

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 nutrient supply and so stops or slows the lesion's progress.[173] Notwithstanding this, the importance of an effective and complete coronal seal should be of utmost importance.

Direct Pulp Capping in Primary Teeth

The lifespan 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. Further, 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.[110]

Carious pulpal exposures in primary teeth should not be direct pulp capped. *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.*[7,250] Under these circumstances, it is presumed that the conditions for a favorable pulpal response are optimal.

Pulpotomy in Primary Teeth

The AAPD guidelines[7] for pulp therapy for primary and young permanent teeth describe the pulpotomy procedure in primary

BOX 23-3

Evidence of Successful Pulp Therapy

- Vitality of the majority 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

BOX 23-4

Contraindications for Pulpotomy in a Primary Tooth

- A history of spontaneous toothache (not caused by papillitis resulting from food impaction)
- A nonrestorable tooth where postpulpotomy coronal seal would be inadequate
- A tooth near to exfoliation or if no bone overlies the crown of the permanent successor tooth
- Evidence of periapical or furcal pathosis
- Evidence of pathologic root resorption
- A pulp that does not bleed (necrotic)
- Inability to control radicular pulp hemorrhage following a coronal pulp amputation
- A pulp with serous or purulent drainage
- Presence of a sinus tract

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. Evidence of successful pulp therapy includes those features listed in Box 23-3.

For all pulpotomy procedures for the cariously exposed vital pulp of primary teeth, the procedure common to all is the complete amputation of coronal pulp tissue. Following this, and once hemostasis has been achieved, a decision is made on the wound dressing or technique to apply to the pulp stumps.

Some of the options available are listed below, and the success of all 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. Yet despite these, formocresol and in particular one of its constituents, formaldehyde (FAD), has come under close 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 over the safety of these materials make it clear that additional research on the use of these and other pharmacotherapeutic agents is required.

Nonpharmacologic hemostatic techniques have been recommended, including electrosurgery[169,253,269,270] and laser therapy.[64] Research involving human clinical trials on both these techniques is sparse; nevertheless, electrosurgical pulpotomy is currently being 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.[236,278,318]

Indications and Contraindications for Pulpotomy

The pulpotomy is indicated for *pulp exposure on primary teeth in which the inflammation or infection is judged to be confined to the coronal pulp.*[146,295] 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 for pulpotomy on a primary tooth are outlined in Box 23-4.

Technique

Pulpotomy is used for primary teeth with radicular pulp tissue judged to be free from 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:

- Effective control of infection.
- Complete removal of inflamed coronal pulp tissue.
- Appropriate wound dressing.
- 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 points that follow highlight the diagnostic aids available:

- An accurate pain history is helpful in deciding the possible stage/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/chamber. They also provide information relating to periradicular pathosis.
- Clinically, pulpal involvement can be assessed immediately following caries removal by looking very carefully for evidence of a pulp exposure.

Technique of 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. Following successful coronal amputation and hemostasis (Fig. 23-15, *A-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

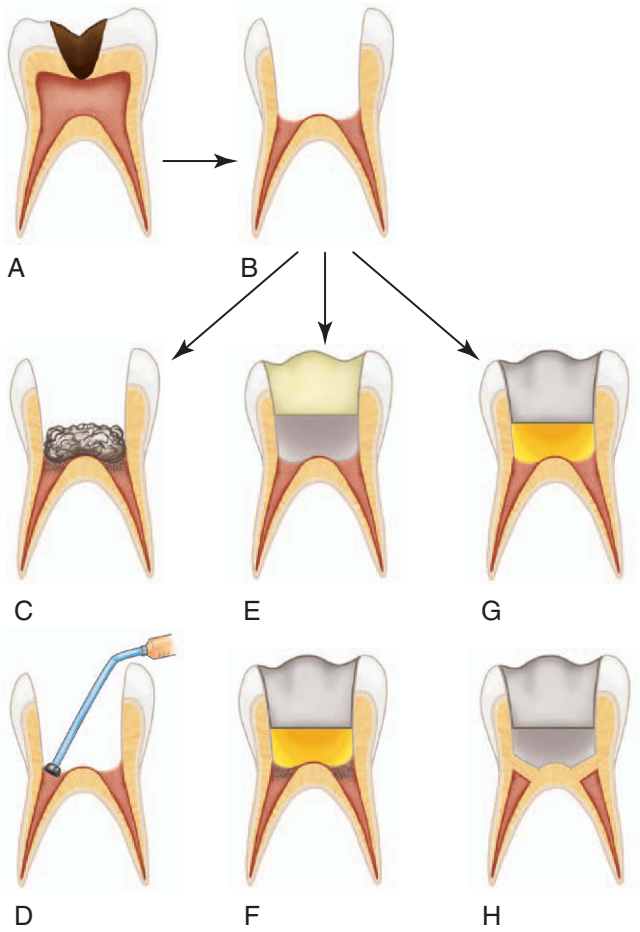


FIG. 23-15 Illustration to show three different approaches to the radicular pulp following coronal pulp amputation during pulpotomy in an extensively carious primary molar. **A,** Primary molar with caries extending to the pulp. Note inflammatory response in coronal pulp. **B,** Tooth has undergone removal of the coronal pulp. Radicular pulp tissue is healthy and has stopped bleeding. **C,** A 1:5 dilute solution of formocresol may be applied to 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 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 within the root canals. **G,** Mineral trioxide aggregate (MTA) is placed directly over the radicular pulp stumps, and the tooth is provided with an intracoronal restoration as before. **H,** The radicular pulp tissue remains vital beneath the MTA; a calcific bridge may form over time beneath the MTA.

might be subjected to electrosurgery or laser application. Fig. 23-15, *C-H* provides a schematic overview of the options and expected outcomes.

Stages in the Technique of Coronal Pulp Amputation

1. Following the initial diagnosis of probable vital pulpal involvement, the primary tooth is anesthetized and isolated with a rubber dam.
2. All caries are removed, and the observation of bleeding from exposure sites indicates vital (if inflamed) coronal pulp tissue (Fig. 23-16, *A-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. 23-16, *C-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 removed will cause hemorrhage. 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 filaments 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, it can be reentered with a small round bur to amputate the suspected inflamed tissue; the canal is then rinsed again, and cotton pellet pressure is applied.
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. 23-15):

- Application of dilute formocresol solution for 5 minutes
- Application of 15% ferric sulfate solution for 15 seconds (Fig. 23-16, *E-F*)
- Permanent placement of MTA
- Electrosurgical manipulation of the wound surfaces
- Laser manipulation of the wound surfaces

Formocresol Pulpotomy

Technique (see Fig. 23-15, C-D*)*

1. Following coronal pulp amputation and once hemostasis has been achieved, *one-fifth dilution formocresol solution* (Box 23-5) on a cotton pellet 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



FIG. 23-16 Clinical stages of a vital pulpotomy using ferric sulfate solution. **A,** Extensive dental caries affecting a mandibular first primary molar. Note proximity of 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 bleeding coronal pulp prior to amputation. **D,** Roof of pulp chamber removed completely. **E,** A 15.5% solution of ferric sulfate is applied to radicular pulp stumps using a dento-infuser tip supplied by the manufacturer. **F,** Hemostasis is evident at 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 of Vidya Srinivasan, Edinburgh Dental Institute, UK.)*

BOX 23-5

Formulation of a One-Fifth Dilution of Formocresol Solution

1 part Buckley's formocresol solution is mixed with:
- 1 part distilled water and 3 parts glycerin.

BOX 23-6

Formulation of Full-Strength Buckley's Formocresol

Formaldehyde	19%
Tricresol	35%
Glycerin	15%
Water	31%

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 (FAD), 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.[225]

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.[93,193]

Although animal studies have identified radioisotope-labeled formocresol or FAD in major organs following systemic injection or multiple pulpotomies, researchers concluded that the doses of formocresol were far in excess of those used in normal clinical practice. It has therefore been suggested that the findings should not be extrapolated to clinical use in humans.[192,196,197] 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.[182] Notwithstanding this, the amount of formocresol absorbed systemically by way of the pulpotomy route is small and may not contraindicate the use of the drug.[220]

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 non-presensitized animals,[273] and presensitized animals showed only a weak allergic potential.[314] The demonstration of an immune response to formocresol-fixed, autologous tissue implanted in connective tissues or injected into root canals has, however, been reported.[26,298]

Is Formocresol Carcinogenic?

In respect of mutagenicity and carcinogenicity of FAD, it is generally accepted that FAD is genotoxic in vitro, inducing mutations and DNA damage in cells from a variety of organisms, including humans.[101,208,217,288,324] The possible link between FAD and carcinogenesis has been investigated within 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.[133] 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.[317] In the dental context, the amount of FAD within a diluted solution of formocresol is small, but there are no data relating to 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.[109]

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)[17] showed a majority of 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, there has been a general trend away from using formocresol, driven by several factors: difficulty obtaining the medicament, concerns related to its safety, and promising clinical results of newer, nonaldehyde techniques.[318] However, the recent U.K. Clinical Guideline, *Pulp Therapy for Primary Molars,* still includes the formocresol pulpotomy as an option,[250] as does the AAPD guidance.[7] Although formocresol pulpotomy is still widely taught in predoctoral pediatric dentistry programs in the United States,[231] there is a lack of consensus over its use for vital pulp therapy in primary teeth.

The current formocresol pulpotomy technique is a modification of that reported by Sweet in 1930.[287] 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 length of time of application, concentration used, the method of application, or a combination of all these factors.[66,96,172,177]

A one-fifth dilution of formocresol solution has been widely advocated based upon the outcomes of both in vitro and in vivo studies comparing dilute formocresol (see Box 23-5) with the undiluted solution (Box 23-6). 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.[164,165,286] Clinical studies have shown diluted formocresol to be as successful as full-strength formocresol.[85,86,185,186]

There is enough evidence today to conclude that ***if formocresol is to be used at all,*** *the one-fifth concentration should be preferred*

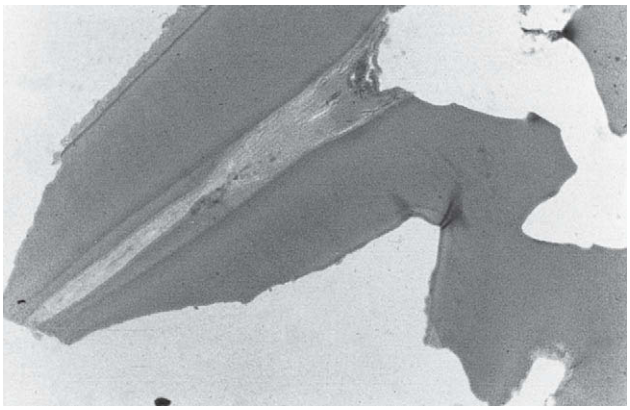


FIG. 23-17 Narrowing of the apical region of the root canal following formocresol pulpotomy. Histologic section of tooth showing deposition of new dentin along the walls of the apical region of the root canal.

for pulpotomy procedures because it is as effective as and less damaging than the traditional preparation.

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[187,251] to an increase in defects and positional alterations of the underlying permanent tooth.[181] It should be pointed out that studies of this nature are follow-up studies long after treatment, without 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, with some studies finding no consistent effect[187,312] and some reporting early exfoliation.[119,185]

Unlike the tissue response to $Ca(OH)_2$, no dentin bridge should be anticipated after applying formocresol to exposed pulp tissue (see Fig. 23-15, *C-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. 23-17).

Criteria for Success

Failure of a formocresol pulpotomy is usually detected on radiographs (see Fig. 23-13). 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 the deposition of calcified tissue. In the primary molars, radiolucency develops in the bifurcation or trifurcation area. In the anterior teeth, a radiolucency 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 be 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.[32,259] An amorphous, eosinophilic material shown to contain phenolic groupings similar to those present in medicaments was found in the lesions. Other researchers[194] have observed furcal lesions in untreated, pulpally involved primary molars containing granulomatous tissue with stratified squamous epithelium, suggesting the potential for cyst formation. In a subsequent study involving failed, pulpotomized primary molars, most specimens were diagnosed as furcation cysts.[195] These findings emphasize the importance of periodic follow-up to endodontic treatment on primary teeth. Fig. 23-18 shows a favorable long-term outcome following a formocresol pulpotomy in a mandibular primary molar.

ALTERNATIVES TO FORMOCRESOL PULPOTOMY

A 2006 comprehensive review is suggested for readers requiring a detailed look at the numerous alternative techniques available.[278]

Glutaraldehyde Pulpotomy

Glutaraldehyde has been a suggested 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.[290,322] Fixed pulp tissue may be replaced with dense collagenous tissue over time.[150] Studies have also demonstrated less systemic distribution than was thought to occur with formocresol.[57,159,196,220,321]

Glutaraldehyde has been shown to be rapidly metabolized with little toxic effect.[118,137,143,196,240] It has also been demonstrated that although glutaraldehyde has antigenic action similar to formocresol, it is of a lower potential.[237,239] However, there are perceived problems with the use of glutaraldehyde as a pulpotomy agent:

- Glutaraldehyde solutions are reported to be unstable.[235]
- Neither the optimum concentration nor time of application of glutaraldehyde solution has been established conclusively.[97,162,238]
- Clinical studies reported increasing failure rates with increasing time post pulpotomy,[89] with lower levels of clinical success than formocresol.[5]

Despite the perceived enhanced safety of glutaraldehyde with respect to formocresol, it did not gain favor over formocresol for pulpotomy in the primary dentition.[17,76]

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. The manufacturers also supply a special dento-infuser tip for this purpose (Astrigident, Ultradent Products Inc, Salt Lake City, UT) (see Fig. 23-15, *E;* Fig. 23-16, *E*).

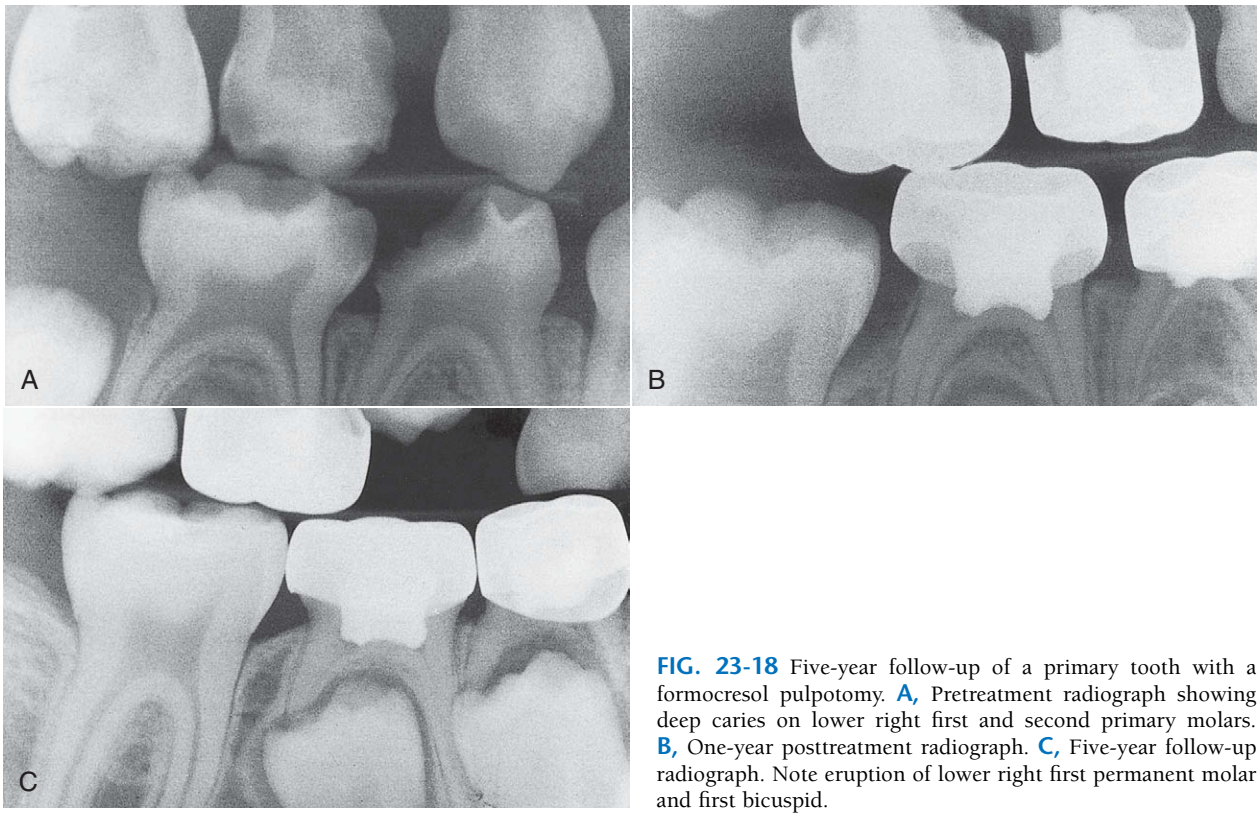


FIG. 23-18 Five-year follow-up of a primary tooth with a formocresol pulpotomy. **A,** Pretreatment radiograph showing deep caries on lower right first and second primary molars. **B,** One-year posttreatment radiograph. **C,** Five-year follow-up radiograph. Note eruption of lower right first permanent molar and first bicuspid.

3. Upon removal of the cotton pellet, the wounds appear brown, and no bleeding should be evident. If a small amount of residual bleeding does occur, 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. 23-15, *F*).

Ferric sulfate has been used as a hemostatic agent for crown and bridge impressions.[79] 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.[160] Initially the use of ferric sulfate was recommended as a hemostatic agent during the $Ca(OH)_2$ 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[262]—considered by some to be important factors contributing to the failure of pulpotomies using $Ca(OH)_2$. Its first documented use in this way was in primate teeth.[152]

The use of ferric sulphate itself as a hemostatic pulpotomy agent was investigated by researchers using animal models (rat[40] and baboon).[88] From the relatively few clinical studies that have been published using ferric sulfate as a pulpotomy medicament, four are prospective comparative,[75,88,129,219] and two are retrospective.[30,276]

In all the comparative studies, formocresol was used 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 one study,[219] half of the teeth were restored with composite resin and the remaining half with preformed metal (stainless steel) crowns.

Prospective Studies

In another study,[75] 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, there was a statistically significant difference 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 the study by Fuks et al.,[88] 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 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 and 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,[129] 34 teeth were used in each group, and identical results were obtained 24 months post treatment for both ferric sulfate and formocresol: a 97% total success rate was recorded. In each group, only one tooth presented internal resorption and was therefore considered a failure.

In 2002, results of a larger comparative study were published[219] with a sample comprising 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 author feels that this finding could be due to three reasons: the larger sample size and the very good recall rate, which increased the possibility to observe and locate more failures, and 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, with only those cases of internal resorption that were either extensive in size or progressing with time 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,[276] 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.[30] 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; that the criteria for selection of cases were not strictly defined; and that 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[75] 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.[219]

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.[219] 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.[276] This proposal may be accepted only in cases of minimal or unchanged internal resorption but 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 rest 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 recent meta-analysis[163] that for human carious primary molars with reversible coronal pulpitis, both ferric sulfate and formocresol pulpotomies similarly give 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.[199]

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. 23-15, *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 $Ca(OH)_2$.[228] Similar results were found when human intact third molars were used to compare the effect of pulp capping with MTA and $Ca(OH)_2$.[2] MTA was found to maintain pulp integrity after pulp capping and pulpotomy in animal studies[59,255] and to have a dentinogenic effect on the pulp expressed by the induction of dentin bridge formation (see Fig. 23-15, *H*) where it touches the pulp tissue.[255,257,310]

Clinical Studies

One of the first preliminary studies published comparing MTA with formocresol in humans[63] 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 post 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.[245] 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 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[122] in a more extensive study that included part of the material presented by Eidelman et al.[63] and a longer follow-up time. These authors 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. Following 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 by 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 between 4 and 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 presented 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.[107] 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 post 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.

MTA is commercially available in the United States as ProRoot MTA (DENTSPLY Tulsa Dental, Tulsa, OK), and Angelus MTA (Angelus, 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-gram packets. The composition of MTA is similar to cement used in the building industry, and such material should be kept dry during storage, because contact of the powder with moist air will lead to "air-setting," affecting its physical properties. The same could be applied to the "clinical-grade" cement, and the manufacturer of ProRoot MTA recommends the 1-gram sachet as a 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 watertight, air-tight container such as an Eppendorf tube (Eppendorf UK Ltd, Cambridge, UK), reducing its cost.[283] Alternatively, 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 gram 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, Irvine, CA) 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 away from the pulpal stump and tooth structure as possible while still allowing electric arcing to occur.
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[80] 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,[157] it was a decade later that Mack[168] became the first U.S. clinician to routinely perform electrosurgical pulpotomies. Oringer[216] 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 following 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.[278]

Several clinical studies[253,270] have produced results comparable to those found with the use of formocresol. Conflicting results have been reported from histologic studies, ranging from comparable results to formocresol pulpotomy[269] to pathologic root resorption with periapical and furcal involvement.[271] A retrospective human study[169] in 1993 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.

Laser Pulpotomy

Several reports have appeared in the literature on the use of the carbon dioxide laser for performing vital pulpotomies on primary teeth.[64,161] Investigators[64] 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. They 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 should 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.

Other researchers[161] 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. 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.[84,250,278]

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 impact upon 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 23-7).

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. 23-19). 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.[244] Because of these factors, use of an apex locator is not reliable to establish canal length.

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, canal length is usually established by measurement of the diagnostic radiograph, ensuring the working length of endodontic instruments are 2 or 3 mm short of the radiographic apex. The 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 is not achieved, 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[126] that minor hypoplasia is increased in permanent successor teeth after root canal treatment of the primary precursors. Others[39] have reported no such increased effect and concluded that defects result from the infection existing before the pulpectomy and not the procedure itself. It

BOX 23-7

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 interradicular septum.

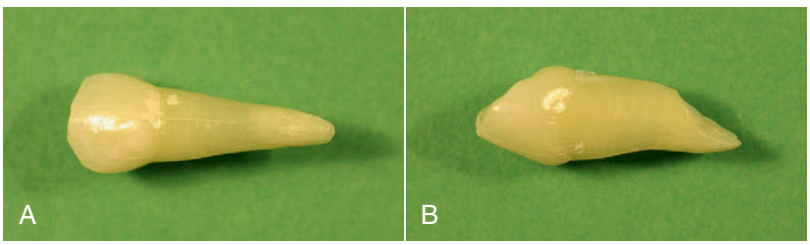


FIG. 23-19 Primary maxillary canine. **A,** Facial view, where apical foramen appears to be at the apex of the root. **B,** Physiologic root resorption has occurred at the palatal aspect of the root; apical foramen is positioned more coronally and will not be coincident with the perceived radiographic apex of the tooth.

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.

Success of endodontic treatment on primary teeth is judged by the same criteria that are 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 96% have been reported.[13,123,155,327] The usual means of studying root canal filling on primary teeth have been clinical and radiographic. There exists a great need for histologic study in this area.

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.[62,98,138,281]

Rabinowitch[234] published the first well-documented scientific report of 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.[13] (See Bennett[21] for a review of the techniques of partial and total pulpectomy.)

In a well-controlled clinical study of primary root canals using Oxpara paste as the filling material,[155] 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 for 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 23-8 lists the categories of teeth that are not good candidates for pulpectomy.

BOX 23-8

Contraindications for Pulpectomy in the Primary Dentition

- An 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

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, there is invariably a perforation of the root by the resorption (see Fig. 23-13). 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, and at least 4 mm of root length is necessary for the primary tooth to be treatable.[244]

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, in which it is recommended that the clinician use a facial approach followed by an acid-etched composite restoration to improve aesthetics (Fig. 23-20).[170]

Posterior Primary Teeth

Access openings into the posterior primary root canals are essentially the same as those 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. 23-21).

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.

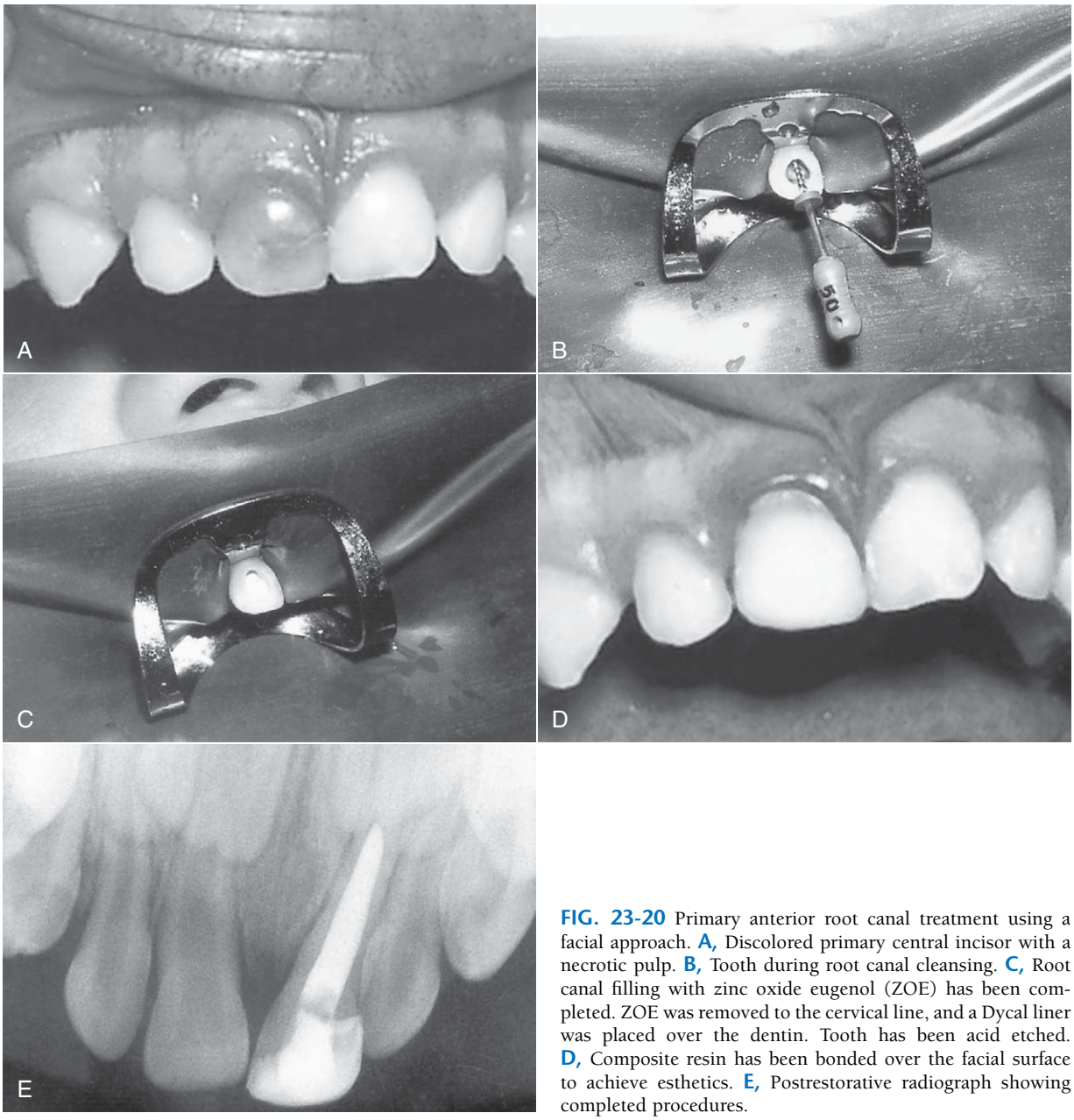


FIG. 23-20 Primary anterior root canal treatment using a facial approach. **A,** Discolored primary central incisor with a necrotic pulp. **B,** Tooth during root canal cleansing. **C,** Root canal filling with zinc oxide eugenol (ZOE) has been completed. ZOE was removed to the cervical line, and a Dycal liner was placed over the dentin. Tooth has been acid etched. **D,** Composite resin has been bonded over the facial surface to achieve esthetics. **E,** Postrestorative radiograph showing completed procedures.

Technique

As in permanent endodontic therapy, the main objective of the chemical and mechanical preparation of the primary tooth is débridement 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. Fig. 23-22 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. The 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 unreliable because root resorption may create lateral openings into the periodontal tissues at any level.[244]
5. To prevent overextension through the apical foramen, it is advisable that the working length be shortened to 2 to 3 mm short of the radiographic length, especially in teeth exhibiting signs of apical root resorption (see Fig. 23-22, *B*).
6. After establishment of the working length, the canal is cleaned and gently shaped (as described in Chapter 9). *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 Corp, Watertown, MA) is contraindicated because of the danger of perforation or stripping of the roots.*



FIG. 23-21 Illustration to show the safe removal of the roof of a pulp chamber in a primary molar. A non-end cutting bur ensures that the relatively thin floor to the pulp chamber is not perforated inadvertently by rotary cutting instruments.

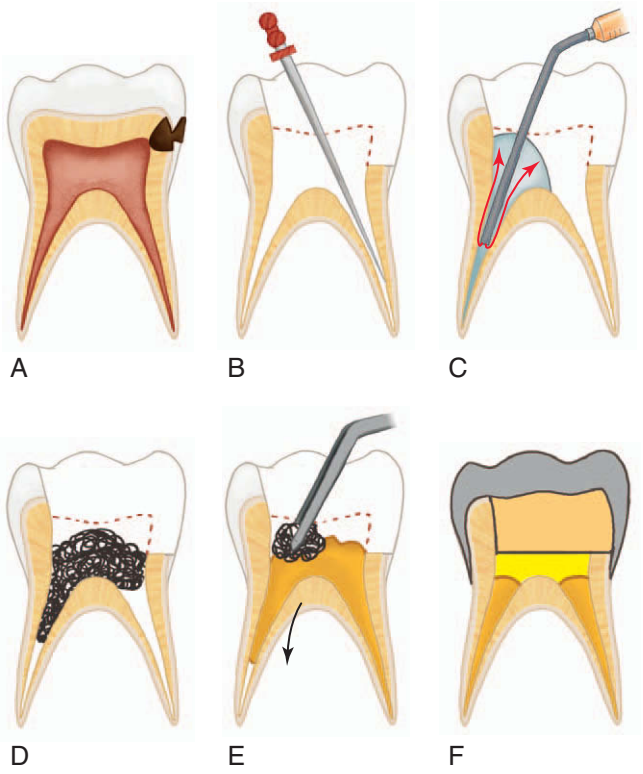


FIG. 23-22 Illustration to show the stages of pulpectomy and root canal filling in a mandibular second primary molar. **A,** Extensive approximal caries. Note the irreversible inflammation present in coronal and radicular pulp. **B,** Following caries removal and unroofing of the pulp chamber, the coronal pulp is amputated. Irreversibly inflamed tissue will bleed profusely. A premeasured hand file is placed approximately 2 mm from the radiographic apex; canals are gently cleaned with minimal shaping. **C,** Irrigation with sodium hypochlorite or chlorhexidine digluconate solution should be undertaken during the cleaning phase. **D,** If root canals are not to be obturated at the same visit, they may be dressed with nonsetting calcium hydroxide, or canals can be left empty and the tooth restored with a small cotton wool 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; shown here is the ZOE being tamped down the canal by the piston action of a cotton pledget held in tweezers. **F,** Following root canal filling, the tooth is restored definitively using a preformed metal (stainless steel) crown.

7. The more flexible nickel-titanium (NiTi) instruments are recommended rather than stainless steel (SS). Hand or rotary techniques are ideal for primary teeth. If SS 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 9). Débridement of the primary root canal is more often accomplished by chemical means than by mechanical means.[130] This statement should not be misinterpreted as a de-emphasis on the importance of thorough débridement and disinfection of the canal. Initially, RC-Prep (Premier Dental Products, Norristown, PA) may be used as the canals are negotiated. It will digest and emulsify pulp tissue and is an effective lubricant. Once a working length has been established, the use of NaOCl solution to dissolve organic debris can play an important part in removal of tissue from inaccessible areas of the root canal system (see Fig. 23-22, *C*).

The Nonvital Discolored Primary Incisor

If a primary incisor is intrinsically discolored following loss of pulp vitality, there may be an esthetic need to improve the tooth's color. Bleaching is not advised in the primary dentition. The anatomy of the maxillary primary incisors is such that access may successfully be made 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.

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 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. 23-20, *C-D*).

The Decision to Obturate

After canal débridement, the canals are again copiously flushed with NaOCl and are then dried with sterile premeasured paper

points. If the canals are dry and there is no exudate, obturation is performed in the same session. If the obturation cannot be done in 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:

- 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.[167]

No material currently available meets all these criteria. The filling materials most commonly used for primary pulp canals are ZOE paste, iodoform paste, and $Ca(OH)_2$. These will be discussed briefly in the following section.

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 iodoform-containing pastes.[124,151] The antibacterial activity of ZOE has been shown to be greater than that of an iodoform-containing paste (KRI paste, Pharmachemic AG, Zurich, Switzerland), whereas its cytotoxicity in direct and indirect contact with cells is equal to and less than (respectively) than 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.[243] It resorbs rapidly and has no undesirable effects on successor teeth when used as a pulp canal medicament in abscessed primary teeth. Further, KRI paste that extrudes into periapical tissue is rapidly replaced with normal tissue.[124] 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.[174,289] 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 Co, Tokyo) have been published.[83,207] 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, one researcher[167] considers the calcium hydroxide–iodoform mixture to be a nearly ideal primary tooth root canal obtundant. Another preparation with similar composition, Endoflas, is available in the United States (Sanlor Laboratories, A.A. 7523 Cali, Colombia, South America). The results of root canal treatments using Endoflas in a students' clinic were similar to those observed with KRI paste.[87]

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. It is, however, sometimes 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 employed and accessibility of the canal to relevant instruments.

If using ZOE, 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 the Lentulo spiral. A cotton pellet held in cotton pliers and acting as a piston within the pulp chambers is quite effective in forcing the ZOE into the canals (see Fig. 23-22, *E*). The endodontic pressure syringe[23,103] is also effective for placing the ZOE in root canals. 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.[56]

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 injected into the canal with disposable plastic needles. This technique is particularly easy to use for primary incisors but less practical for the narrow canals of primary molars.[210]

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.[39,123] The adequacy of the obturation is checked by radiographs (see Fig. 23-20, *E*; Fig. 23-23, *A*; Fig. 23-24, *C*).

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 are 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. 23-22, *F*).

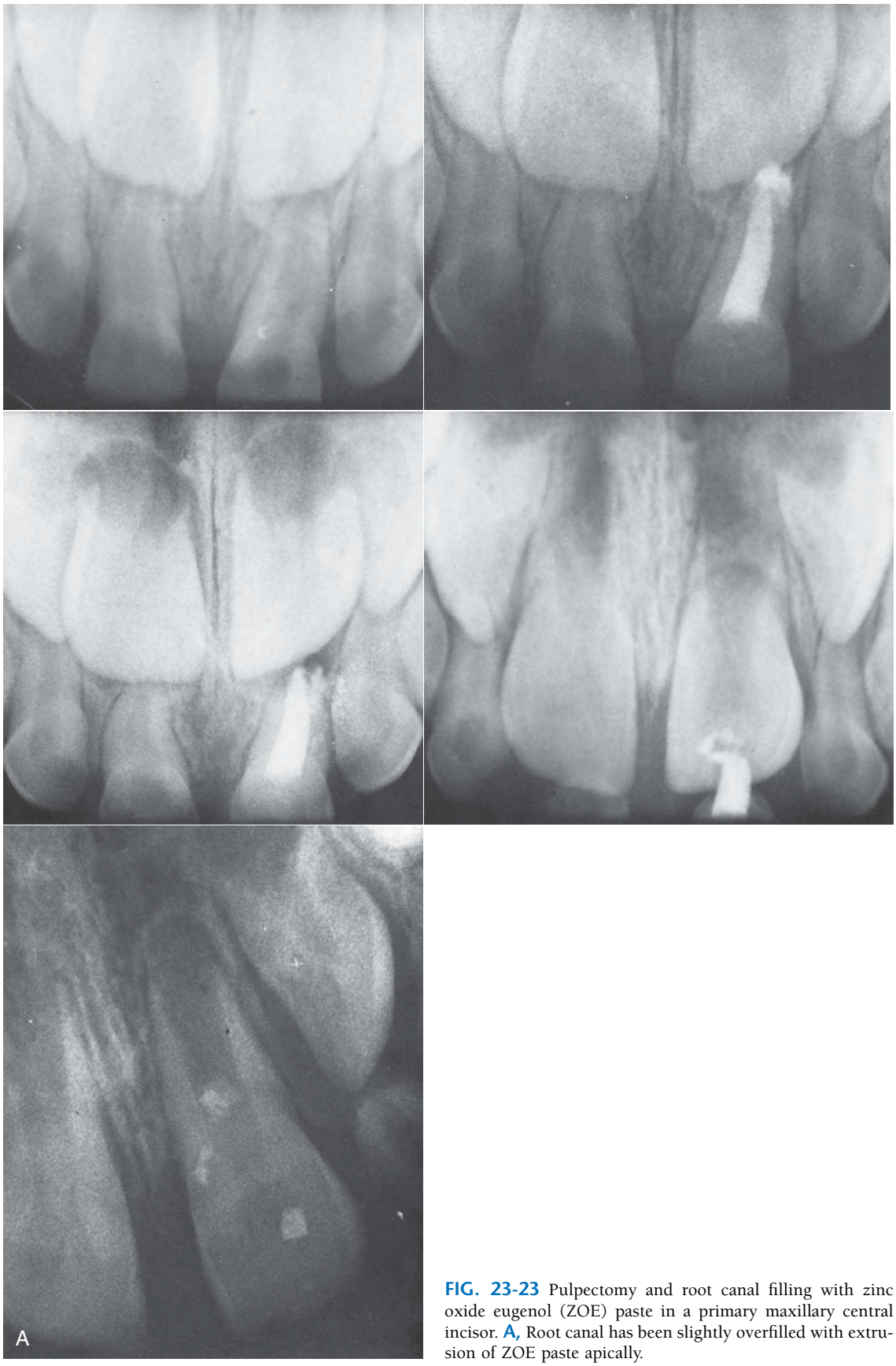


FIG. 23-23 Pulpectomy and root canal filling with zinc oxide eugenol (ZOE) paste in a primary maxillary central incisor. **A,** Root canal has been slightly overfilled with extrusion of ZOE paste apically.

Continued

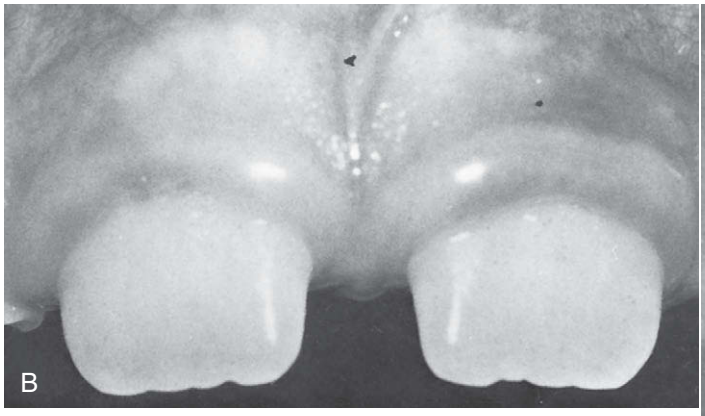




FIG. 23-23, cont'd B, Same patient showing newly erupted permanent incisors. Note there are no enamel defects present on the crown, despite overfill of the root canal of the predecessor. **C,** Radiograph almost 5 years after pulpectomy and root canal filling of predecessor. Note normal apical development and almost total absorption of ZOE remnants.

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. 23-25).

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[39,283] that pulpectomized primary teeth may show delayed exfoliation. 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 filler, and in time, most showed complete absorption or reducing amounts. Retention of filler was not related to success and caused no pathosis.[254] Therefore no attempt is made to remove retained filler from the tissues (see Fig. 23-23, *A*; Fig. 23-24, *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 treatments were considered successful when no pathologic resorption associated with bone rarefaction was present.[95,124] If evidence of pathosis is detected, extraction and conventional space maintenance are recommended.

Investigators[223] 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 there is an acute problem, 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[87]; they 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/root ratio, thin dentin walls, and a wide and often apically diverging root that presents significant endodontic and restorative challenges (Fig. 23-26). A central responsibility of all clinicians is therefore to safeguard pulp survival 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 will be given to the root canal treatment and restoration of nonvital, immature permanent teeth. The emerging potential of pulp regeneration and bioroot engineering will also be considered.

Indirect Pulp Therapy: Avoiding Pulp Exposure

The pulps of young permanent teeth are at risk of breakdown following traumatic injuries (considered fully in Chapter 17), dental caries, and restorative dentistry. There is good evidence that residual dentin thickness is a key determinant of pulp survival after cavity preparation,[190] and avoiding pulp exposure

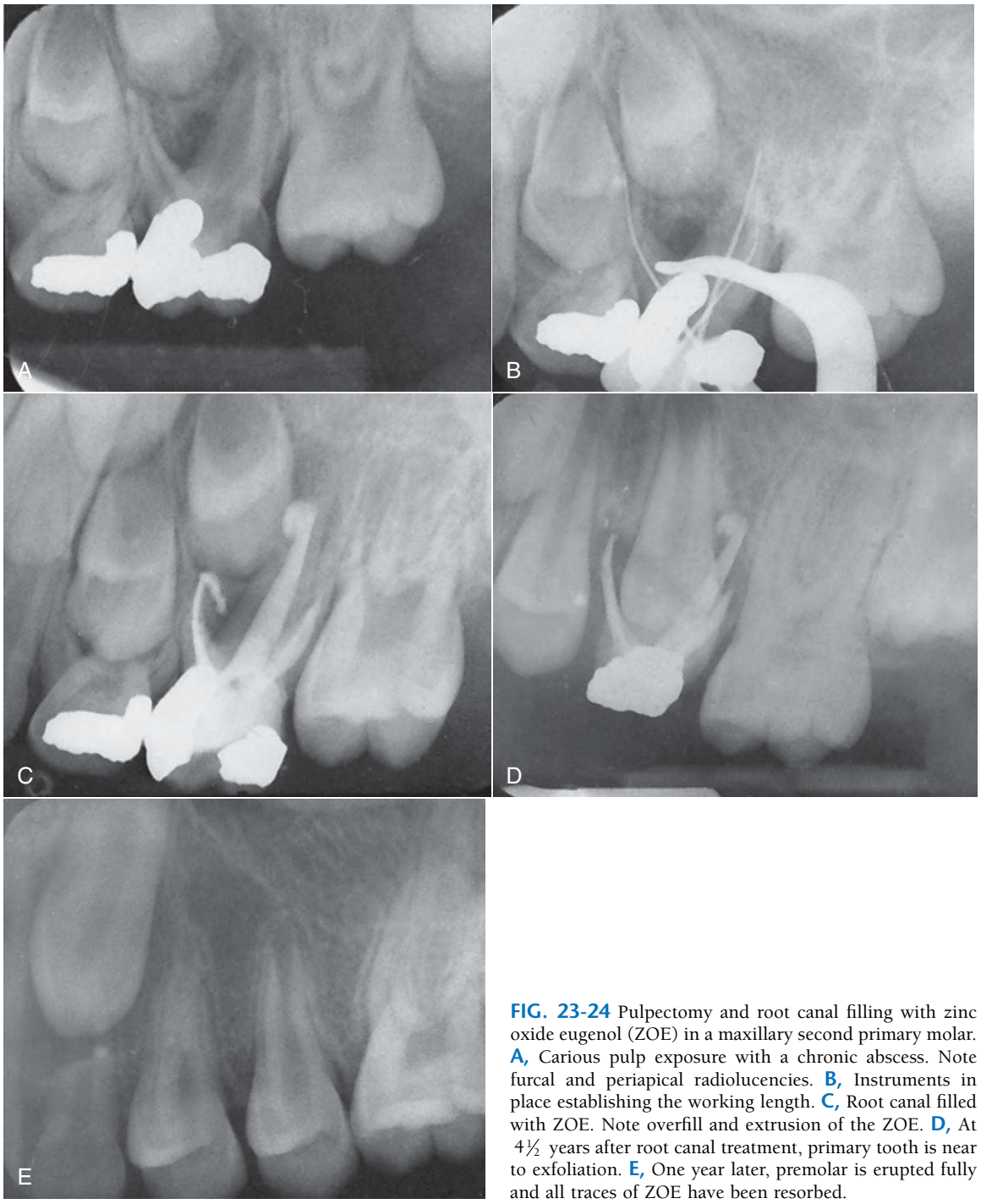


FIG. 23-24 Pulpectomy and root canal filling with zinc oxide eugenol (ZOE) in a maxillary second primary molar. **A,** Carious pulp exposure with a chronic abscess. Note furcal and periapical radiolucencies. **B,** Instruments in place establishing the working length. **C,** Root canal filled with ZOE. Note overfill and extrusion of the ZOE. **D,** At 4½ years after root canal treatment, primary tooth is near to exfoliation. **E,** One year later, premolar is erupted fully and all traces of ZOE have been resorbed.

has been considered advantageous. The management of deep caries by partial and 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.[25] In the case of serial excavation, the need for a secondary excavation has recently been brought into question, while acknowledging the need for a tightly-sealing coronal restoration.[241] Researchers continue to investigate the role of antimicrobial treatments, including ozone fumigation,[229] photo-activated disinfection (PAD), and antimicrobial resins in sterilizing deep layers of affected dentin[307] and creating the conditions for arrest and remineralization. Considerable interest has also focused on the active upregulation of reactionary dentinogenesis by applying bioactive agents such as the TGF-β family of molecules to the depths of cavity preparations.[306] 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.[232] More basic and clinical

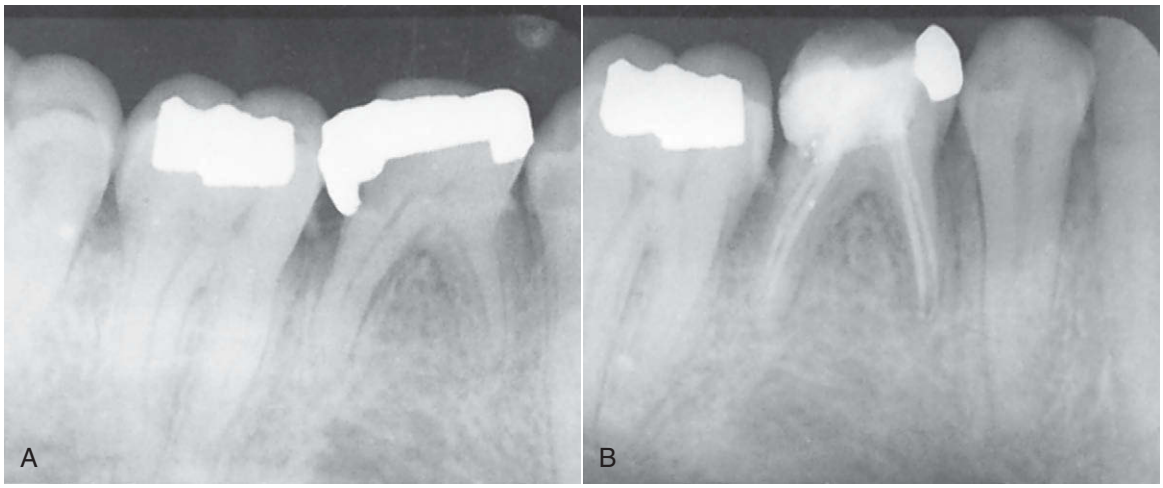


FIG. 23-25 Pulpectomy and root canal filling with gutta-percha in a retained mandibular primary second molar with no succedaneous permanent tooth. **A,** Carious exposure of the pulp. **B,** Because the permanent premolar is absent, root canals were filled with gutta-percha and sealer rather than just zinc oxide eugenol.



FIG. 23-26 Premature loss of vital pulp functions in an immature maxillary permanent incisor. Development is arrested, leaving a fragile tooth with a compromised crown-root ratio, thin dentin walls, and a wide-open root end which presents endodontic and restorative challenges.

research is needed before such agents can be widely adopted in practice.

There is, however, a substantial body of evidence to support the view that complete excavation is not necessary for the successful management of deep caries,[299] and that indirect pulp therapy may be the most effective means of securing pulp health in asymptomatic teeth.

Managing Pulp Exposure

Even with the greatest clinical judgment and restorative skill, pulps are sometimes exposed during deep caries excavation. A recent survey has also shown 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.[211] 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.[20] Attitudes may be changing, especially for the management of immature permanent teeth, where pulp diagnosis is notoriously difficult and the stakes for pulp preservation are high. Though much of the evidence is relatively short term and involves previously uninflamed pulps, MTA has established a strong track record for promoting reparative tertiary dentin bridge formation after direct pulp capping[200] (see Fig. 23-5).

In a radically different approach to the management of deep caries, one of the authors (JC) has advocated complete caries excavation and direct capping of the exposure in the immature teeth of children (Fig. 23-27). 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 the 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 lavaging 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.[3,43] The solution may be left in contact with the exposed pulp tissue for 10 to 15 minutes, refreshing with new solution every 3 to 4 minutes. Care must be taken in aspirating the excess NaOCl to prevent further hemorrhage, the aspirator tip being placed lateral to and never directly above the crown.

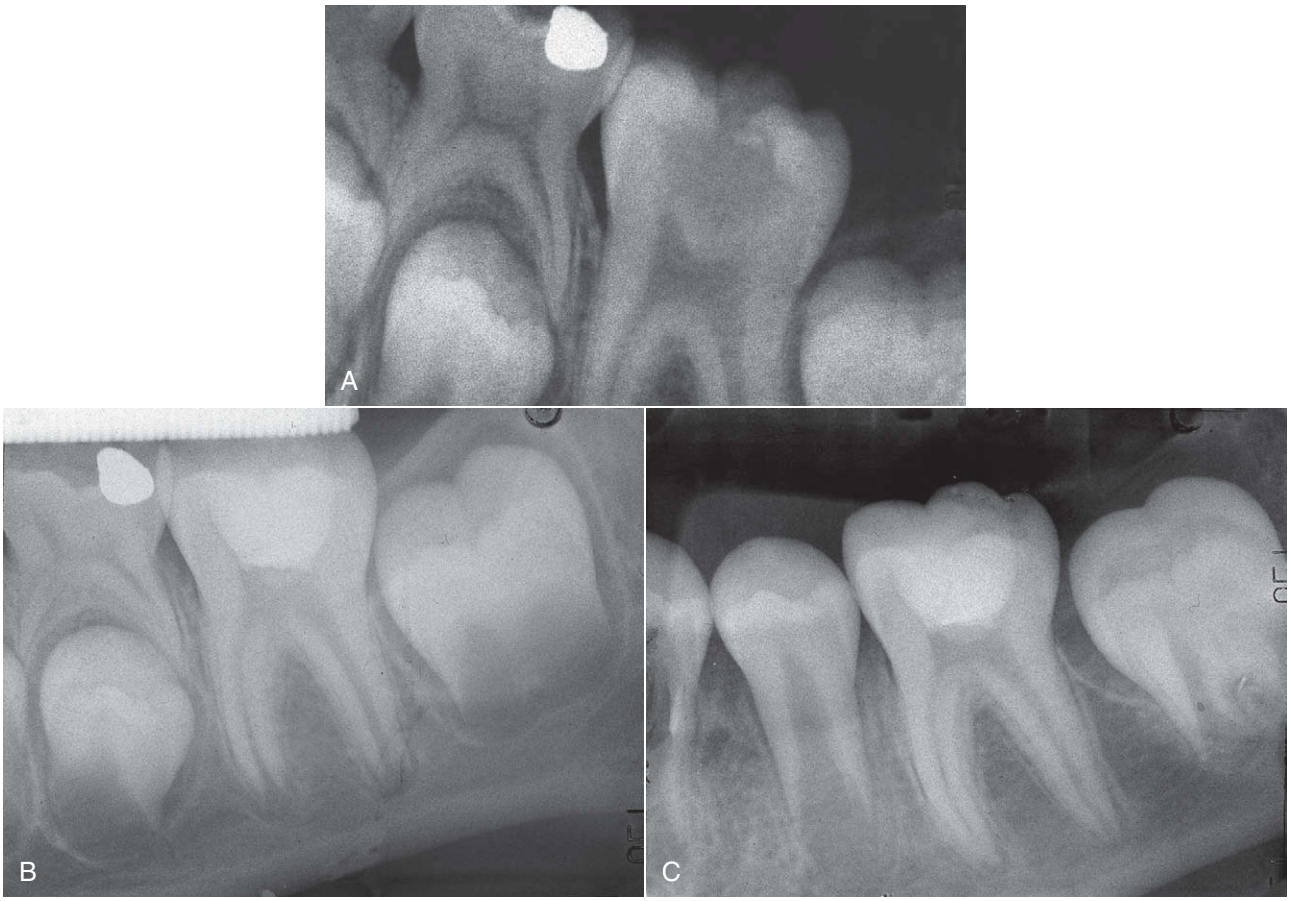


FIG. 23-27 Direct pulp capping in a symptomatic young permanent mandibular molar with an open apex. **A,** Extensive carious exposure in an immature molar with a history of spontaneous pain. **B,** After complete caries excavation and hemostasis, the pulp is overlaid with mineral trioxide aggregate and the tooth sealed with composite resin. **C,** Three-year recall shows completed root formation. Note lack of calcification within pulp chamber and canals.

Once hemorrhage is controlled, the tooth structure is cleansed with cotton pellets moistened with NaOCl, once 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 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. Following removal of the Cavit and cotton, the MTA is examined to ensure that it is set hard, and the tooth is restored with a bonded composite restoration. If the MTA has failed to harden, the author has advocated washing out the uncured material and repeating the procedure after removing pulp tissue to canal orifice level. This approach has received further support from two studies. In the first,[73] 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. (2008)[27] 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. 23-28, *A-B*), often resulting in multiple, large pulp exposures. Hemostasis was secured by bathing the pulp with 5.25% to 6% NaOCl for between 1 and 10 minutes (Fig. 23-28, *B-D*). One pulp which continued to bleed after this time was considered unsuitable for direct capping. After the application of 1.5 to 3 mm gray or white MTA (Fig. 23-28, *E*) and permanent bonded restorations at 5 to 10 days, teeth were reviewed for between 1 and 9 years (mean 3.94 years). The recall rate was 92.5%, and 97.96% of teeth were found to have favorable outcomes on the basis of radiographic appearances, subjective symptoms, and cold testing (Fig. 23-28, *F*). Of the 15 teeth which were immature at the time of treatment, 100% went on to complete root formation.[27]

At the time of writing, a conservative general consensus probably holds with partial and serial caries excavation, avoiding pulp exposure and attempting to create an environment which will upregulate reactionary tertiary dentin responses.[25,241] But evidence is growing that in correctly chosen cases with no indication of irreversible pulpitis or periapical change and where radical caries excavation is followed by confirmation of hemostasis, direct capping with MTA can enjoy remarkable levels of success in teeth with incomplete apices. Although

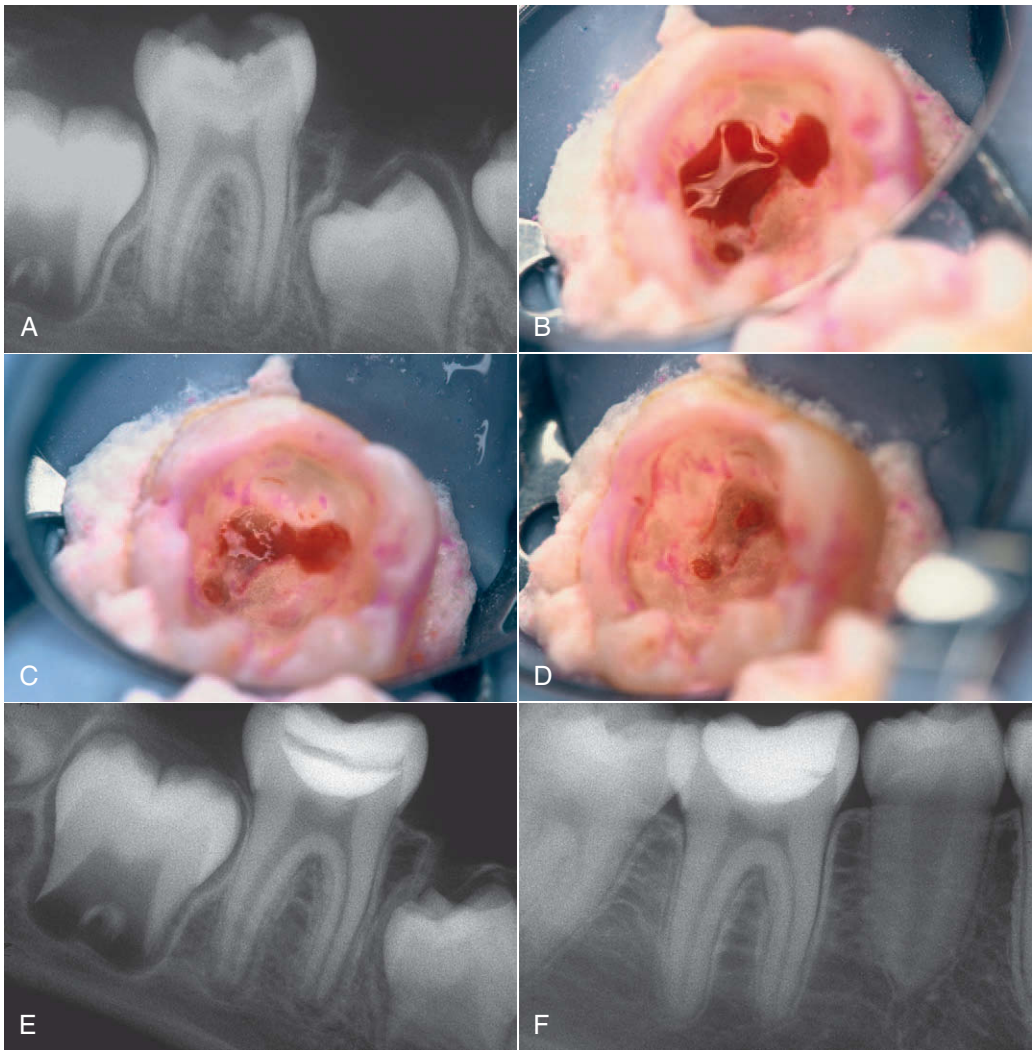


FIG. 23-28 Radiographic and clinical sequence of mineral trioxide aggregate (MTA) direct pulp capping of a mandibular right molar in a 9-year-old female. **A,** Pretreatment radiograph showing initial deep caries and immature apices. **B-D,** Five-minute application of 5.25% sodium hypochlorite hemostasis, on two 1.5- to 2.0-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 showing permanent restoration and evidence of complete root formation. The tooth exhibited 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 *139:305-315, 2008. Copyright © 2008 American Dental Association. All rights reserved. Reprinted with permission.)*

generally restricted to asymptomatic teeth, one of the authors (JC) has noted success in both asymptomatic and symptomatic teeth, with resolution of pain and continued root development. This procedure may in effect be considered a very superficial form of mechanical and chemical pulpotomy. *Because the loss of vital pulp function is so devastating in teeth with immature apices, it seems advisable to attempt conservative pulp-preserving procedures in deeply carious permanent teeth. If failure occurs, 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.[15] This is then covered with a wound-dressing agent in an effort to promote healing at the amputation site. 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.

The Cvek Pulpotomy on Immature Permanent Teeth

Pulpotomy is an established technique for preserving vital pulp functions in immature teeth which have been subject to pulp-exposing trauma. A full account of the Cvek pulpotomy is

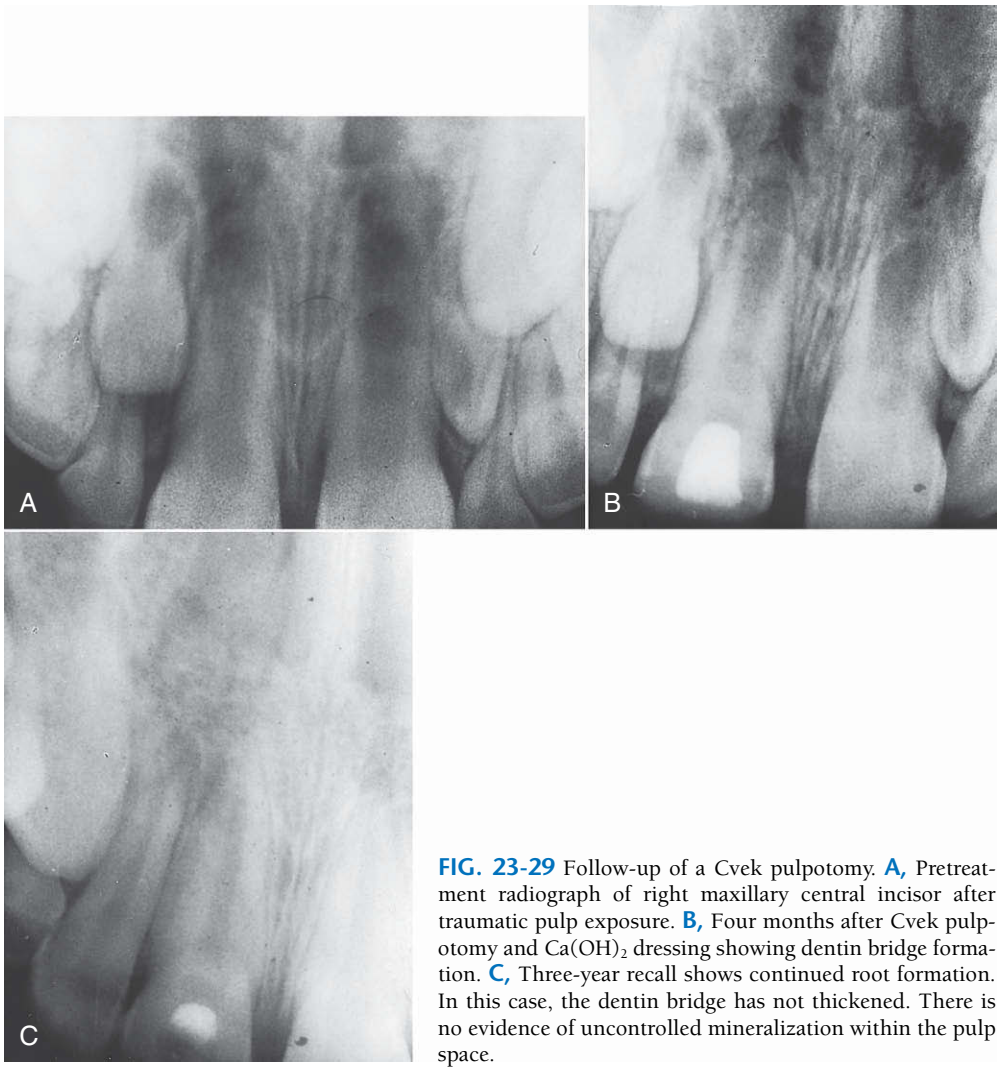


FIG. 23-29 Follow-up of a Cvek pulpotomy. **A,** Pretreatment radiograph of right maxillary central incisor after traumatic pulp exposure. **B,** Four months after Cvek pulpotomy and $Ca(OH)_2$ dressing showing dentin bridge formation. **C,** Three-year recall shows continued root formation. In this case, the dentin bridge has not thickened. There is no evidence of uncontrolled mineralization within the pulp space.

provided in Chapter 17 (see Fig. 17-11). Briefly, studies[55,113,114] have shown that inflammation is confined to the surface 2 to 3 mm of the pulp when traumatically exposed and left untreated for up to 168 hours. In experimental animals, the results were the same whether the crowns were fractured or ground off.[55] Direct invasion of vital pulp tissue by bacteria did not occur, although the pulps were left exposed to saliva.

In their classic report, Cvek[50] described a pulpotomy technique where only the superficial 2 to 3 mm of hyperplastic inflamed tissue was removed with a water-cooled high-speed diamond bur[102] 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 which 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 Apexogenesis).

Following hemostasis with saline and a pulp cap of calcium hydroxide overlaid with a sealing coronal restoration, Cvek reported success in an impressive 94% to 96% of cases (Fig. 23-29). However, histologically better results have been shown more recently with MTA as the pulp-capping agent.[1,140,198,200,228] MTA is thus recommended as the pulp-capping agent of choice in cases that do not extend deeply into the roots, where subsequent retrieval may present considerable challenge. Preference should be given to nonstaining white MTA products.

The question then is whether such techniques can be applied to posterior teeth, and especially to those with symptomatic cariously-affected pulps. The answer has historically 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.[55,113] The extent of inflammation in the pulp of a cariously exposed pulp is therefore difficult to judge, with the expectation that pulpotomy may be less predictable than posttraumatic exposure.

Partial Pulpotomy on Asymptomatic Young Permanent Posterior Teeth

A technique termed *partial pulpotomy for the management of vital carious pulp exposure on young permanent molars* has been reported by several researchers.[175,176,179,209] 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 a 1- to 3-mm cutback of pulp tissue judged to be inflamed beneath the exposure site to reach underlying healthy tissue. After hemostasis, the exposure site has traditionally been covered with $Ca(OH)_2$ and the tooth sealed with ZOE and a permanent restoration. Mejàre and Cvek[179] followed 31 initially symptom-free teeth for a mean of 56 months (range 24 to 140 months) and found 93.5% healing. Others[175] reported 91.4% success in 35 cases followed between 12 and 48 months. Exposures exceeding 2 mm 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 again met with success in the hands of the authors and others.[261]

Pulpotomy on Symptomatic Young Permanent Teeth

Although some success has been reported,[179] vital pulp therapy in permanent teeth with a history of pain has generally been contraindicated. Reports do, however, support this pulp-preserving approach even to symptomatic teeth.[261] Of the six teeth with temporary pain, widened periodontal ligament space, and/or condensing osteitis managed in Mejàre and Cvek's study,[179] by $Ca(OH)_2$ pulpotomy, 66.7% healed. More recently, one study[33] 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 from clinical symptoms, responded to sensitivity testing, 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[233] 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 $Ca(OH)_2$ (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, though four pulps had undergone calcific metamorphosis.[65] In a 2006 clinical report,[326] investigators 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 between 6 and 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.[326]

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 promoted. 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. The correlation of clinical symptoms with histologic state is, however, poor.[60]

Follow-Up After Pulp Capping and Pulpotomy

After pulp capping and pulpotomy, the patient should be seen periodically for 2 to 4 years to determine success. Although normal vitality tests (e.g., electrical and thermal sensitivity tests) are reliable after pulp capping, they are not usually helpful in the pulpotomy-treated tooth. Since 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. Some researchers[110,154] believe that pulp capping and pulpotomy procedures invariably lead to progressive calcification of the root canals. After successful root development, they advocate pulpectomy and root canal treatment before canals become obliterated and challenging to manage in the event of future endodontic infection. However, it has been the experience of the authors and others[53,90,114] 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. 23-29, *C*; Fig. 23-30).

In a follow-up study of clinically successful pulpotomies,[50] researchers removed the pulps 1 to 5 years later for restorative reasons and found the tissue to be histologically normal.[53] They concluded that changes seen in the pulps 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 necessity for a retentive post. Nevertheless, calcific obliteration, internal resorption, and pulp necrosis are potential sequelae of pulp-capping and pulpotomy procedures which should be screened for during recall appointments. Although unlikely, they are possible, and the patient should be clearly informed.

In posterior teeth, where surgical endodontics is difficult, the observation of continued calcification after root closure may justify reentry of the tooth for root canal treatment. In anterior teeth, if calcification of the canal has made conventional endodontics impossible, surgical endodontics may be performed with relative ease. Therefore in anterior teeth, routine endodontic therapy after completion of root development is contraindicated unless clinical signs and symptoms of pathosis are present or unless such therapy is necessary for restorative procedures (e.g., placement of a post for retention of a crown because of missing tooth structure).



FIG. 23-30 Deep pulpotomy for apexogenesis. **A,** Immature maxillary central incisor 10 weeks after traumatic pulp exposure and a deep pulpotomy dressed with $Ca(OH)_2$. Note early formation of a dentin bridge. **B,** Three-year recall showing the dentin bridge to have thickened and root formation to be complete; tooth remained asymptomatic and required no further treatment at that time. Note absence of uncontrolled mineralization within apical pulp space.

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,[77,191,256,315] and several authors have reported better results with the diluted rather than the full-strength form. However, they reported a high incidence of internal resorption, which increased in severity with longer periods of time.[86,224]

The formocresol procedure had appeal because there was a lower risk of pulp calcification, a recognized complication of $Ca(OH)_2$ pulpotomy. After root completion, the tooth could easily be reentered, the pulp extirpated, and routine endodontic therapy performed. Contrary to these findings, one group of researchers[14] has shown calcification of the canals by continuous apposition of dentin on the lateral walls, with equal frequency whether using $Ca(OH)_2$ or formocresol. The only common denominator to this reaction was the presence of dentin chips that had accidentally been pushed into the radicular pulp tissue.

Although this treatment has been reported to be partly successful, it cannot be routinely recommended in an era of effective alternatives such as MTA.

Formocresol pulpotomy procedures can only be recommended as temporary treatments on permanent teeth with necrotic pulps, where clinical success after 3 years has been reported.[301] The formocresol pulpotomy has particularly been performed in preference to extraction when routine endodontic treatment was not economically viable. This procedure may buy symptom-free time until funds become available to complete root canal treatment.

APEXOGENESIS

Apexogenesis is treatment designed to preserve vital pulp tissue in the apical part of a root canal in order to complete formation of the root apex (Fig. 23-31).[111]

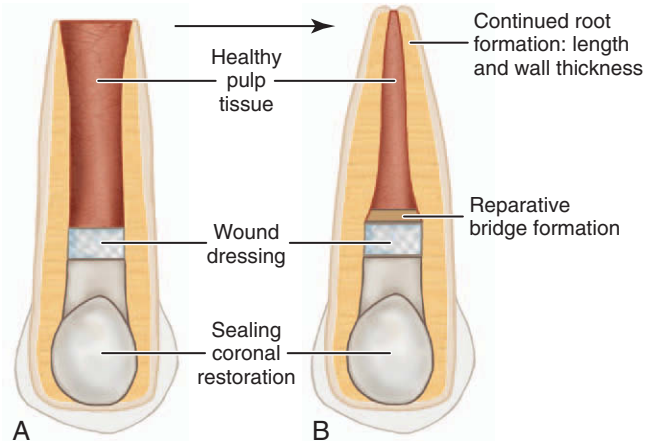


FIG. 23-31 Schematic representation of apexogenesis, a treatment designed to preserve at least the apical portion of pulp tissue in a healthy condition in order to complete root formation. **A,** Following a deep pulpotomy and hemostasis, the radicular pulp is dressed and a sealing coronal restoration 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.

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. Examples include carious exposures and some trauma cases in which treatment of the exposed pulp is delayed, and it becomes necessary to extend further 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. 23-30). In posterior teeth, the use of endodontic files or reamers may be necessary if tissue is being amputated within the canals. Extension of the amputation site into the root canals is undertaken

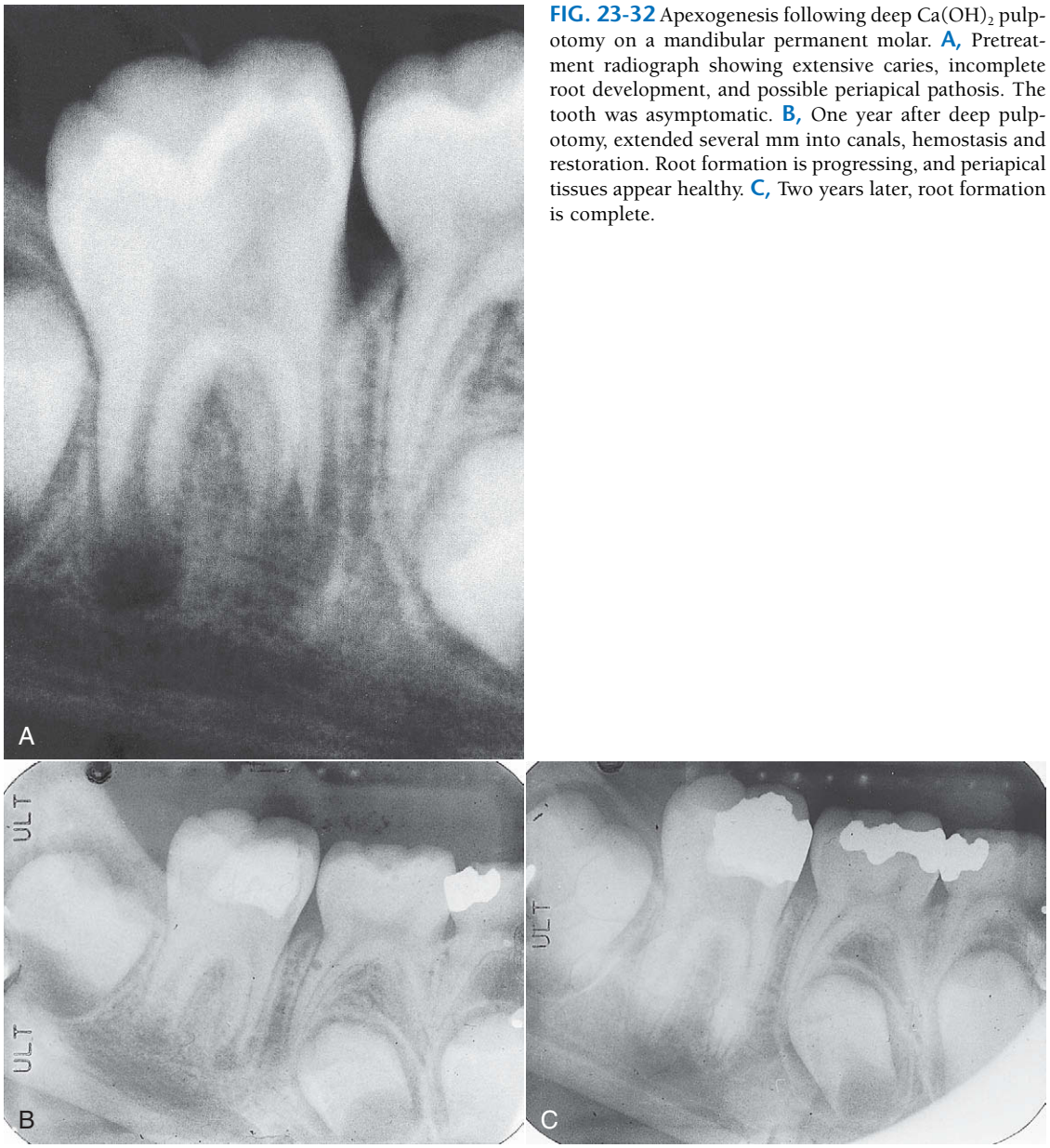


FIG. 23-32 Apexogenesis following deep $Ca(OH)_2$ pulpotomy on a mandibular permanent molar. **A,** Pretreatment radiograph showing extensive caries, incomplete root development, and possible periapical pathosis. The tooth was asymptomatic. **B,** One year after deep pulpotomy, extended several mm into canals, hemostasis and restoration. Root formation is progressing, and periapical tissues appear healthy. **C,** Two years later, root formation is complete.

only when there is little confidence that more superficial pulp capping or pulpotomy will be successful and where there is a desperate desire to preserve pulp functions in teeth with wide-open, blunderbuss apices (Fig. 23-32).

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 securely restoring the crown. It is challenging to determine the status of pulp tissue residing 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 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 if these concerns hold true in an era of microscopy and ultrasonic instrumentation.

Calcium hydroxide powder has usually been preferred over hard-setting products, carried into the canal with an amalgam carrier or gun system employed 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 $Ca(OH)_2$.[308]

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 applying such products, and it is recommended that they be overlaid with a hard-setting calcium hydroxide or glass ionomer cement[52] before meticulous cleaning of the cavity walls and restoration with a bonded composite resin restoration. Though little has been published on this topic, MTA probably does represent a viable alternative to calcium hydroxide, provided the challenges in its retrieval are recognized.

APEXIFICATION

Apexification, or root-end closure, is the process whereby a nonvital, immature, permanent tooth which has lost the capacity for further root development is induced to form a calcified barrier at the root terminus (Fig. 23-33). 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 will at best result in closure of the root end and cannot be expected to cause further root development in terms of length or wall thickness. Apexification is thus regarded as a treatment of last resort in immature teeth which 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 history archives in the years to come.

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 will 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 worsening the crown/root ratio if further root reduction was required to achieve a seal, 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. 23-33, *A-B*; Fig. 23-34).[284]

Diagnosis of pulpal necrosis in a tooth with an incompletely formed apex is often difficult, with the electronic pulp tester rarely providing meaningful data,[95] and thermal tests often giving 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.

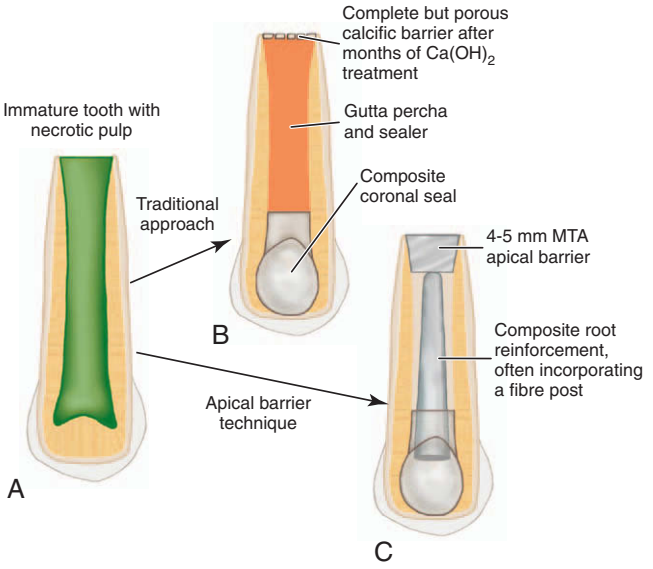


FIG. 23-33 Schematic representation of apexification, a treatment to develop a hard barrier at an open root end. **A,** Immature permanent tooth with a nonvital pulp. **B,** Traditional approach, with the formation of a "calcified barrier" at the root end following repeated dressing over many months with $Ca(OH)_2$. Canal is subsequently filled with gutta-percha and sealer before coronal restoration. **C,** Artificial apical barrier technique, where a 4- to 5-mm plug of mineral trioxide aggregate is placed at the root end. Canal space is subsequently restored with dual-curing composite resin, often accompanied by a fiber post to provide mechanical support.

If any doubt persists, it is usually wise to adopt a watch-and-wait approach before entering the immature tooth endodontically. Only when there is convincing evidence of pulp breakdown should the tooth be entered, and exposed dentin should in the interim 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 conformation in mesiodistal and faciolingual dimensions (see Fig. 23-14). In practical terms, the precise limit of apical opening that can be filled by conventional means is unclear.

Many materials have been reported to successfully stimulate apexification. The use of nonsetting $Ca(OH)_2$ was first reported by Kaiser in 1964.[141] The technique was popularized by the work of Frank,[81] and since that time, $Ca(OH)_2$ alone or in combination with other drugs became the most widely accepted material to promote apexification until the

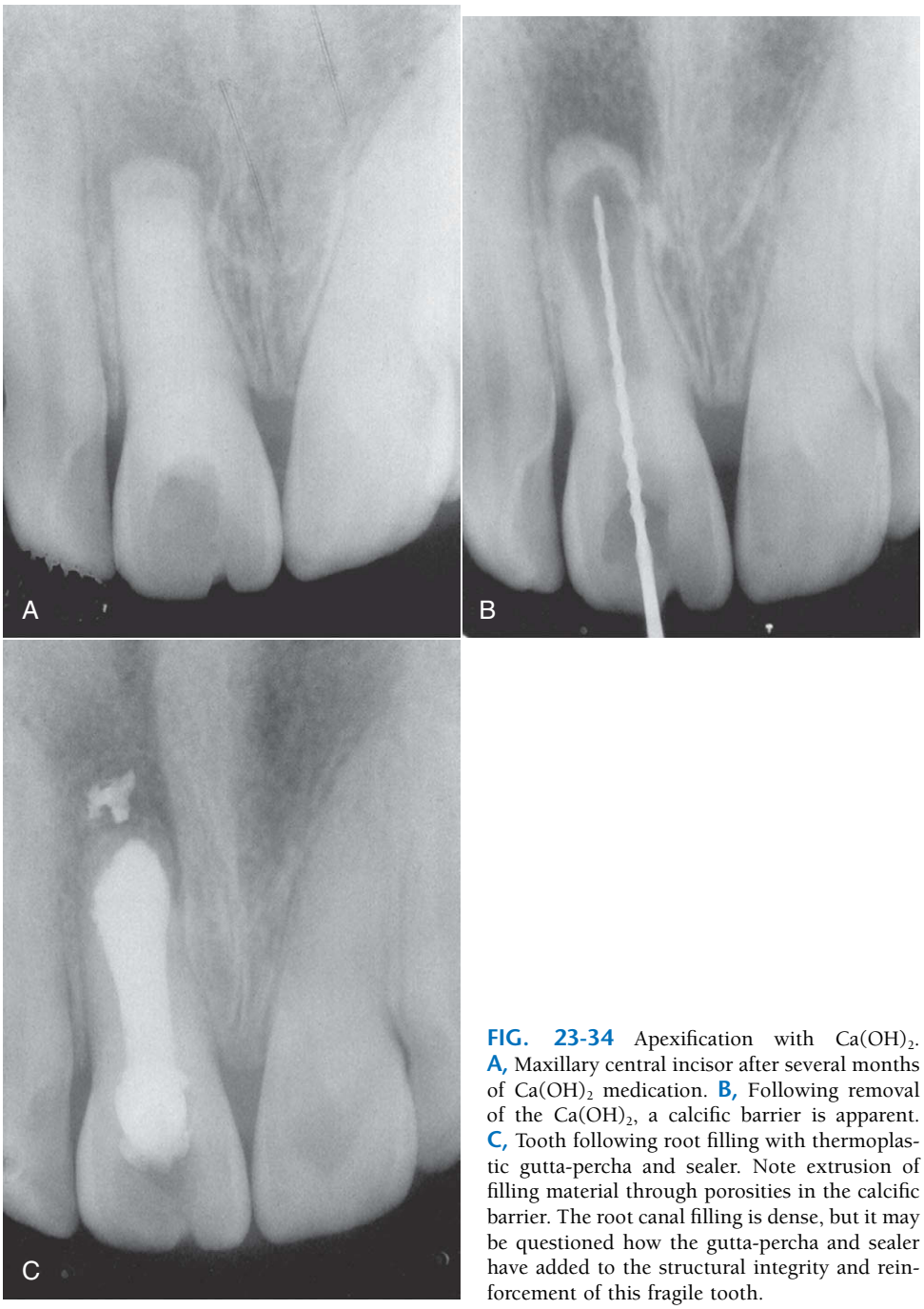


FIG. 23-34 Apexification with $Ca(OH)_2$. **A,** Maxillary central incisor after several months of $Ca(OH)_2$ medication. **B,** Following removal of the $Ca(OH)_2$, a calcific barrier is apparent. **C,** Tooth following root filling with thermoplastic gutta-percha and sealer. Note extrusion of filling material through porosities in the calcific barrier. The root canal filling is dense, but it may be questioned how the gutta-percha and sealer have added to the structural integrity and reinforcement of this fragile tooth.

development of MTA and the potential for rapid, artificial barrier formation.

For historical perspective, $Ca(OH)_2$ powder has been mixed with CMCP, metacresyl acetate, Cresanol (i.e., a mixture of CMCP and metacresyl acetate), physiologic saline, Ringer's solution, distilled water, and anesthetic solution. All have been reported to stimulate apexification, but most reports in the U.S. literature[34,108,285] have advocated mixing $Ca(OH)_2$ with CMCP or Cresanol, whereas reports from other parts of the world[52,178] 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.[320] Comparable outcomes have been noted in humans and animals with tricalcium phosphate,[41,149,246] collagen calcium phosphate,[206] osteogenic protein-1,[267] bone growth factors,[300] and a number of other materials.[320]

The most important factors in achieving apexification seem to be thorough débridement 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[67]). *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 cortical plates.*

Apexification Technique

In the apexification technique, the canal is cleaned and disinfected in line with the principles defined in Chapter 9. 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, especially in the anterior teeth, to accommodate the larger instruments necessary to clean the root canals, but care should be taken not to heavily instrument the already thin and relatively fragile walls of the root. Neither should operators deceive themselves that they are able to steer instruments against all walls of the canal system for complete débridement, particularly in apically diverging canals.

The length of the canal is established by radiographs, since the absence of an apical constriction may make electronic methods unreliable.[128] A constant drying point, determined with paper points, may provide helpful additional information on length.[18] Irrigation is central to the débridement 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,[313] 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 débridement, 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-monthly intervals to wash out the $Ca(OH)_2$ paste and inspect clinically (with the aid of a gutta-percha or paper point or by direct visual inspection through the microscope) and radiographically for the development of a calcified barrier (see Fig. 23-34, *B*). Treatment typically extends over 9 to 24 months, with obvious demands on patient and parent compliance, while risking tooth embrittlement and cervical root fracture following long-term medication with calcium hydroxide.[10,51,252]

Histologic studies consistently report the absence of Hertwig's epithelial root sheath, and normal root formation should never be anticipated. Instead, there appears to be a differentiation of adjacent connective tissue cells into specialized cells; there is also deposition of calcified tissue adjacent to the filling material. The calcified material that formed over the apical foramen was histologically identified as an osteoid (i.e., bonelike) or cementoid (i.e., cementum-like) material (Fig. 23-35).[34,108,284] 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.[61] The closure of the apex may be partial or complete but consistently has minute communications with the periapical tissues (see Fig. 23-33, *B*; Fig. 23-34, *C*; Fig. 23-35). For this reason, apexification stimulated by pastes must always be followed by

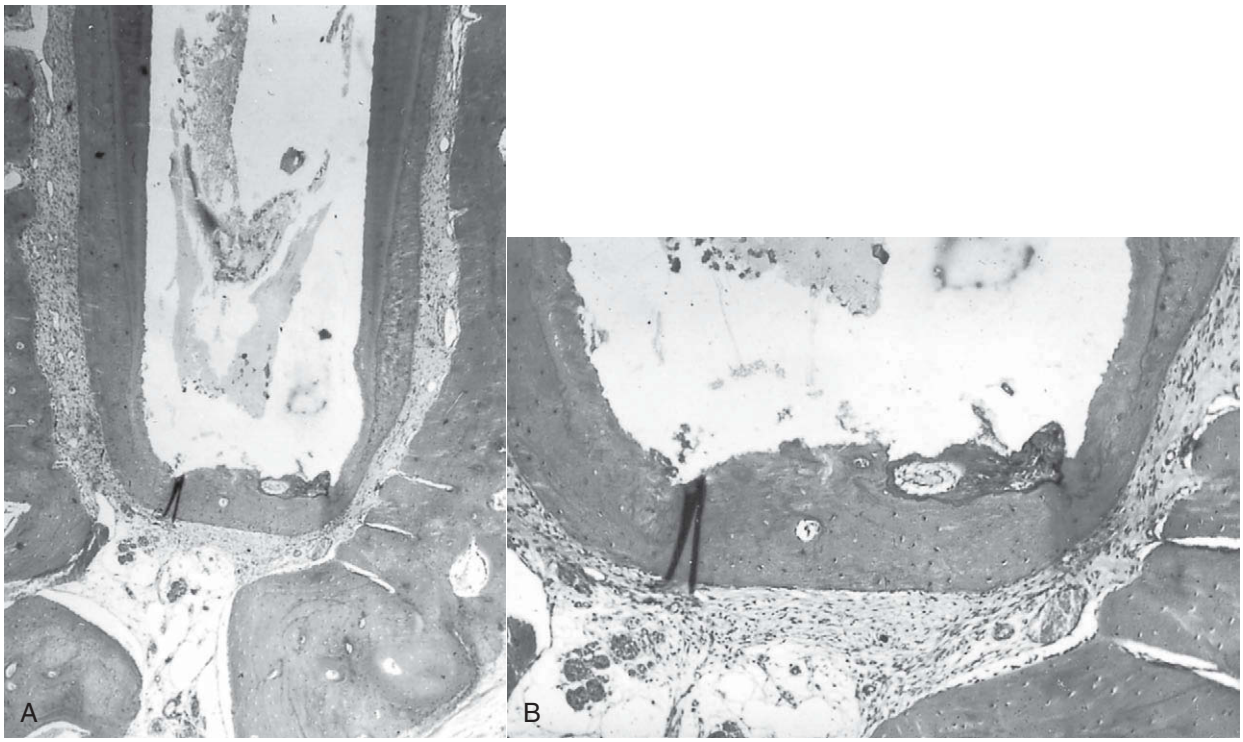


FIG. 23-35 Histologic section of a dog's tooth after $Ca(OH)_2$ apexification. **A,** Cementum-like mineralized tissue is closing the wide-open root end. Debris is apparent within the canal because of inadequate débridement before filling. **B,** Higher magnification showing cellular detail; periodontal ligament is free of inflammation. Root canal filling material was lost in histologic preparation. Note presence of tissue communication through the apical barrier (stain H&E).

obturation of the canal with a permanent root canal filling, traditionally of thermoplastic gutta-percha and sealer, though 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. 23-33, C)

Investigators[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.[264]

The use of MTA as an apical barrier was first reported in 1996,[300] and subsequent clinical investigations in animals[267] and humans[230,258,274] 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 $Ca(OH)_2$.[230] Simon et al. (2007)[274] 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.[258] 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. The risk of tooth fracture after long-term $Ca(OH)_2$ medication[10,252] is also eliminated.

MTA Barrier Technique (see Figs 23-33, 23-36 and 23-37)

In the apexification technique, the canal is cleaned and disinfected as described for $Ca(OH)_2$ apexification. Research[158] has suggested that tissue pH may affect the hydration reaction and final physical properties of MTA, and it has become standard

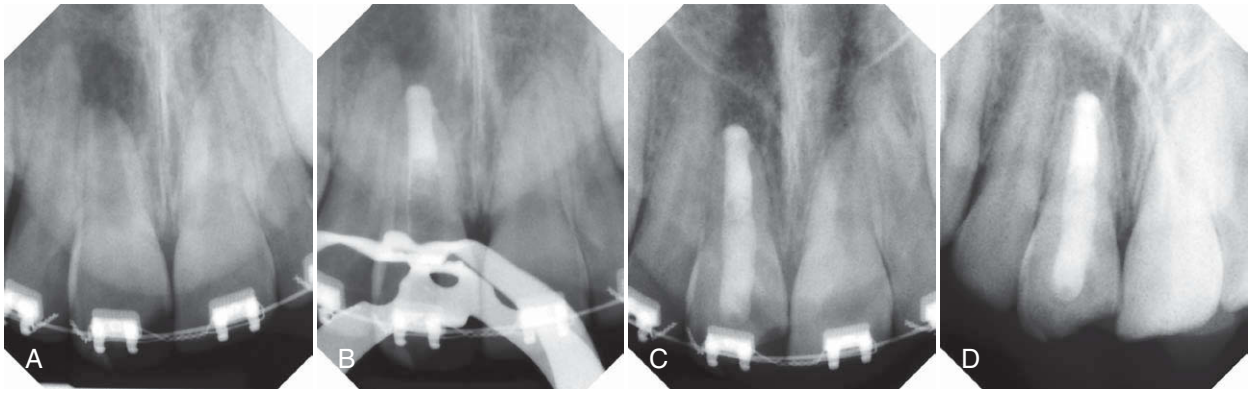


FIG. 23-36 Mineral trioxide aggregate (MTA) apical barrier apexification procedure and restoration with bonded composite. **A,** Maxillary right central incisor with a large periapical lesion. Patient is undergoing orthodontic treatment. **B,** Following placement of MTA in the apical 4 mm of the canal. **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.

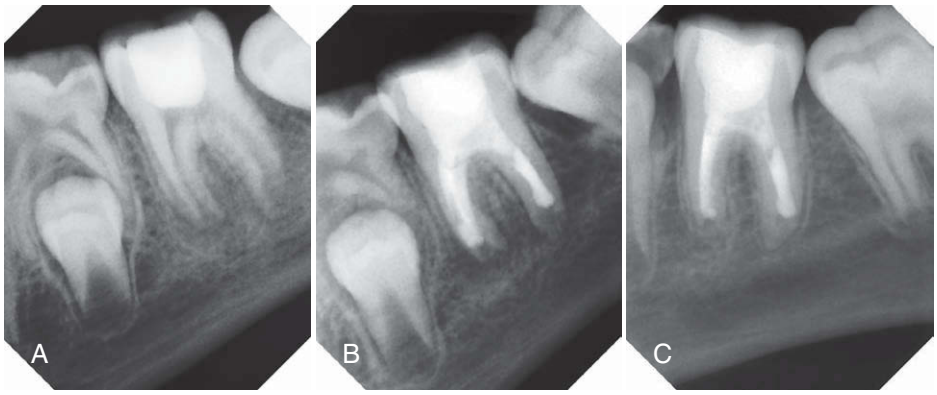


FIG. 23-37 Apexification with mineral trioxide aggregate (MTA) barriers in a mandibular permanent molar. **A,** Periapical lesion reveals open apices in this pulpless molar. **B,** Apical plugs of MTA and remainder of the pulp space restored with bonded composite resin. **C,** Two years later, showing closure of the root ends with cementum.

practice to medicate canals for at least 1 week with $Ca(OH)_2$ to raise the acidic pH of the inflamed periapical tissues before permanently sealing.

When the tooth is free of signs and symptoms of infection, it is reisolated with a rubber dam and the $Ca(OH)_2$ washed free, often with the help of ultrasonics and small brushes. After drying the canal with large, sometimes inverted, premeasured paper points, the canal is filled incrementally with MTA, 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 and set some 1 to 3 mm short of the root end, often with the help of sonic or ultrasonic energy to settle the material.[329] Apical matrices have not generally been considered necessary to limit the flow of MTA, though impressive-looking results may be obtained (see Fig. 17-17) by packing the apical region with calcium sulphate prior to MTA application.[304] An apical plug of 4- to 5-mm thickness is usually considered optimal (see Fig. 23-36, *B*).[311] 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 will become impregnated into the material. Excess water in the access preparation is dried with cotton pellets and the opening 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, and particularly those which are pulpless and have undergone apexification, are at high risk of fracture. Within 3 years of long-term $Ca(OH)_2$ medication and root filling with gutta-percha, it has been reported[51] that 28% to 77% of immature teeth suffered a cervical root fracture. 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 appears 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.[104,116,144] A 2004 study[156] demonstrated significantly greater resistance to root fracture after placement of a 4-mm thick apical plug of MTA followed by an intracanal composite resin when compared with MTA followed by gutta-percha and sealer. Root reinforcement has also been reported to improve by the cementation of a metal post within the channel created by removal of a light-transmitting composite curing post.[36] The potential of fiber-reinforced posts would also appear great,[28] though little clinical evidence has yet been published on the treatment of immature teeth.

Alternative materials which have been suggested to bond and reinforce fragile roots include resin-modified glass ionomer cements[100] and most recently, Resilon.[294] The evidence on Resilon however, remains highly contentious,[325] and the ideal of predictable bonding and tooth reinforcement throughout the length of the root canal system remains some way off.[293]

Bonding 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. 23-33, *C*; Fig. 23-36, *C*). Care should be taken to etch and bond the canal according to the manufacturer's instructions, with proper wash-out 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 to counter some of these adverse events.[35]

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, a light-curing composite resin is placed 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 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 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. 23-33, *C*). 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, there have been few treatment options 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 number of ground-breaking reports in which nonvital immature teeth with clear evidence of pulp breakdown and periapical suppuration were encouraged to grow new pulps and complete root formation (length and wall thickness; see also Chapter 16).[19,37,135,139,297] Success is dependent on the activity of a newly identified population of stem cells, the so-called stem cells from apical papilla (SCAP) (Fig. 23-38, *A*), a hidden treasure with enormous potential for tissue regeneration and bioroot engineering.[127]

All immature teeth with open apices may be considered candidates for regenerative treatment, even if they have frank pulp-space infection, a discharging sinus, or have been previously root canal treated.

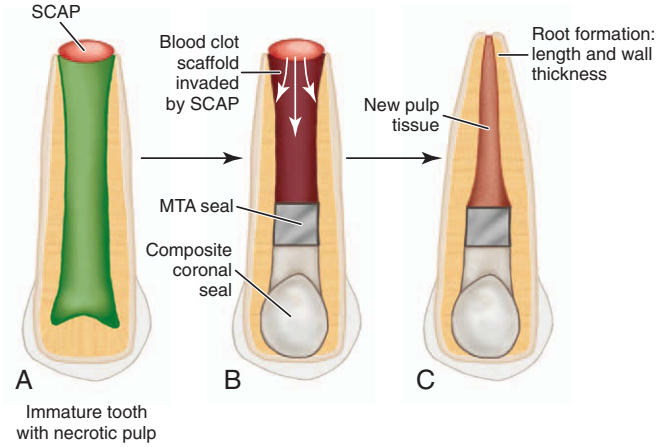


FIG. 23-38 Schematic representation of pulp regeneration. **A,** Immature, nonvital permanent tooth, showing the location of the apical papilla with its rich collection of stem cells. **B,** Following medication of the canal with triantibiotic paste, the canal is overinstrumented to encourage bleeding up to the cervical level. Subsequent blood clot is overlaid with MTA and a sealing restoration and forms a scaffold for invasion by SCAP cells. **C,** Pulp regeneration is expected to allow continued root formation in a previously pulpless tooth. *SCAP,* Stem cells from apical papilla.

The currently recommended protocol (see Fig. 23-38)[139,303] involves disinfecting the isolated canal system with little or no mechanical instrumentation and copious irrigation for 10 to 15 minutes with 5.25% NaOCl. The carefully dried canal is then medicated with a freshly mixed paste of ciprofloxacin, metronidazole, and minocycline in a macrogol ointment/propylene glycol base, which is sealed in place with a well-sealing provisional restoration. After 1 week, the tooth is carefully reopened under strict asepsis, and after washing out the antibiotic paste with NaOCl, the periapical tissues are gently instrumented to encourage bleeding into the canal. A blood clot is produced to the cemento-enamel junction, which will form a scaffold for pulp regeneration. The tooth is then sealed with several millimeters thickness of MTA before closing the crown with a provisional and later a resin-bonded composite restoration (see Fig. 23-38, *B-C*). Details of protocol vary from report to report, with some researchers demonstrating success with simpler protocols involving formocresol rather than antibiotic disinfection, and closure of the crown with glass ionomer cement.[268] Procedures are likely to evolve rapidly in this most exciting area of practice. Teeth are kept under periodic clinical and radiographic review, with the suggestion[303] that more traditional treatment may be considered if there are no signs of regeneration within 3 months. Clinical reports to date offer much promise, since there is little to be lost in attempting a treatment that could transform the prospects of a tooth which would otherwise require apexification. It is conceivable that $Ca(OH)_2$ and even MTA apexification techniques may in the foreseeable future be consigned to the history books. Readers are cross-referred to Chapter 16, which discusses broader aspects of regenerative endodontics, and to the clinical Figures 16-5 to 16-8 which illustrate immature teeth managed in this way.

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.[189] Our call for action has never been louder as we seek effective, biologically based treatments for our pediatric patients.

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CHAPTER 24

Effects of Age and Systemic Health on Endodontics

CARL W. NEWTON and 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[17]

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.[75] Between 1980 and 1995, this same population grew by 28% to a historic high of 33.5 million people. 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 persons 85 years and 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. Soon these people will enter the 65-years-and-older category. As the baby boomers begin to age, the United States will see an unparalleled increase in the absolute number of older persons.[12] Although one in eight Americans was 65 years of age or older in 1994, in a little more than 30 years, about one in five is expected to be in this group (Fig. 24-1).[3]

Information available 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 Midwestern states currently have the highest proportions of older adults. Florida was the only state where the population of older adults was greater than 16% of its population in 1993; however, it is predicted that 32 states will fall in this category by 2020.[51]

Increased longevity of the dentition with expanding fields of advanced restorative procedures and periodontology lends support that dentistry will see a substantial growth in the number of older patients.[16] Until 1983, persons aged 65 years and older made an average of 1.5 dental visits annually, a lower utilization rate than that of any other age group.[78] Between 1983 and 1986, a 29% increase in visits by those 65 years and older was noted by the National Health Survey.[77] 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 65 years of age or older exceeded the percentage of the population in that age group.[44] In the future, clinicians will routinely serve a growing number of older persons, who will account for one third to two thirds of their workload.[42,43,45]

The growth of the population aged 65 years and older has affected every aspect of U.S. society, presenting challenges as well as opportunities to policymakers, families, businesses, and health care providers.[51] Future dental services (including root

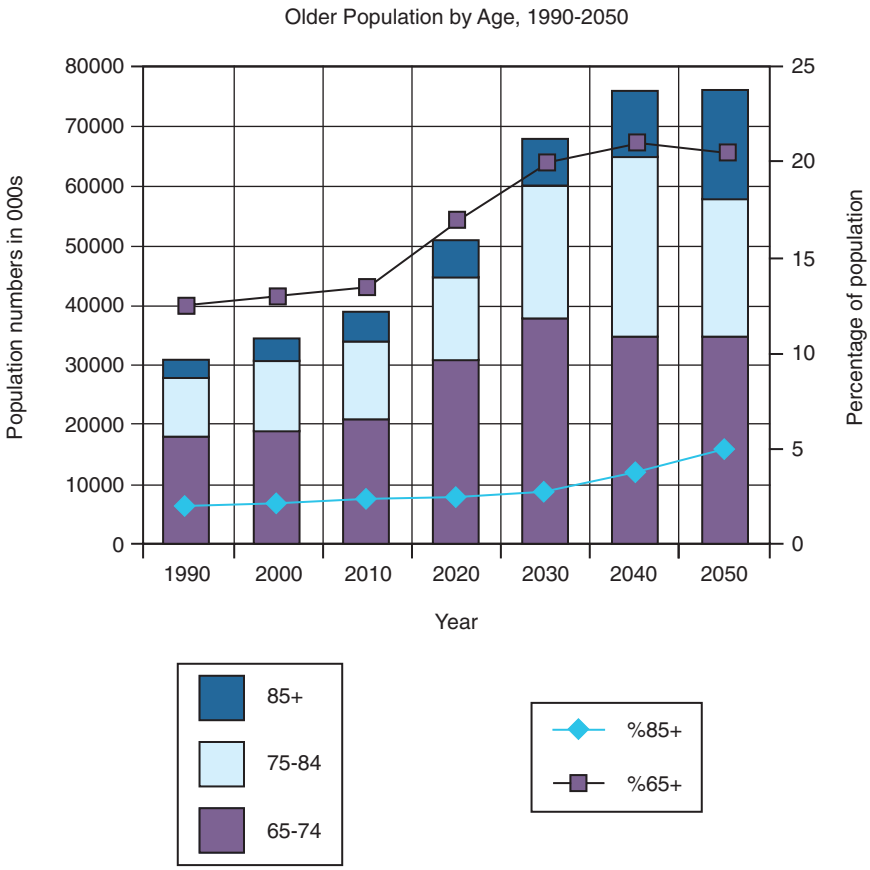


FIG. 24-1 Projected percentage growth rates of U.S. adults age 65+ years and 85+ years from 1990 to 2050. *(From U.S. Bureau of the Census, Population Division Population Projections Program. Available at: http://www.census.gov/ Accessed August 2009.)*

canal procedures) for older patients are anticipated 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[49]

The latter 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 curriculum, research, and publications on aging. The National Institute on Aging stated that all dental professionals should receive education concerning treatment of older adults as part of basic professional education.[14,49]

This chapter discusses the effect of aging on the diagnosis of pulpal and periapical disease and successful root canal treatment. The quality of life for older patients can be significantly improved by saving teeth through endodontic treatment and can have a large and impressive value to 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.[30] 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 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.[7]

Most older patients have had 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.[21] 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 psychologic and social life history and with 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.[58] Clinicians' perception of the

effects of aging on diagnosis and treatment of pulp and periradicular disease is also improving along with the rate at which older patients need and seek specialty care.[27]

Because the primary function of teeth is mastication, it is presumed that loss of teeth leads to detrimental food changes and reduction in health.[31] 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 a 93-year-old man, one clinician was told, "I can't, Doctor. You see, I go dancing every Saturday night, and I need that tooth. I can't look like that." The majority of people older than 60 years who still have their own natural anterior teeth would not wish for a change in appearance if they needed prosthodontic reconstruction.[28]

Clinicians should not presume that they know what is best for senior patients or what they can afford without the patient's full awareness. 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.[5,69,71] Obtaining informed consent also requires that root canal treatment be offered as a favorable alternative to the trauma of extraction and the cost of replacement. The distribution of specialists 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.[80] 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.[82]

This chapter compares the typical geriatric patient's endodontic needs with those of the general population. (Pulp changes attributed 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 those factors that will 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 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, aging causes dramatic changes to the cardiovascular, respiratory, and central nervous systems that result in most drug therapy needs. However, the decline in renal and liver function in older patients should also be considered when predicting behavior and interaction of drugs (e.g., anesthetics, analgesics, antibiotics) that may be used in dental treatment.

The review of the patient's medical history is the first opportunity for the clinician to talk with the patient. The time and consideration taken at the outset will 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 in filling out the forms and 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 fill out 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, the clinician is ultimately responsible for the treatment.

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.[29] It is very important that the clinician take a careful history and update it at every visit.[46] *The Physicians' Desk Reference* should be consulted and any precaution or side effect of medication noted. It is available online at http://www.pdr.net/ and several other websites have been developed specifically for drug interactions.

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 conditions 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 lead to an emergency. Management of medical emergencies in the dental office is best directed toward prevention rather than treatment. Oral and pharyngeal cancers are primarily diagnosed in the elderly, and the prognosis is poor.[76]

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.

Chief Complaint

Most patients who are 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).

The clinician should, without leading, 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 handicaps may become evident at this time.

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 exhibit 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 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 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 pains 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 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.[33] 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 and restoration, or it may be as subtle as a routine crown preparation 15 or 20 years ago. 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. 24-2).

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.



FIG. 24-2 Multiple restorations on this 74-year-old patient suggest repeated episodes of caries and restorative procedures with pulpal effects that accumulated in the form of subclinical inflammation and calcification.

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. (Chapter 1 gives 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. 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 need for treatment can be established and can provide a focus for the examination.

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 centering on the patient's chief complaint, 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.[63] Furthermore, many systemic diseases may initially manifest with prodromal oral signs or symptoms.[14]

Missing teeth contribute to reduced functional ability (Fig. 24-3). The resultant loss of chewing efficiency leads to a higher carbohydrate diet of softer, more cariogenic foods. Increased sugar intake to compensate for loss of taste[23,48] and xerostomia[6] are also factors in the renewed susceptibility to decay. Saliva plays a significant role in the maintenance of oral and general health. 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 is most commonly associated with dental caries and oral fungal infections.[50,65]



FIG. 24-3 Missing teeth and the subsequent tilt, rotation, and supraeruption of adjacent and opposing teeth contribute to reduced functional ability and increased susceptibility to caries and periodontal disease.

Gingival recession, which creates sensitivity and is hard to control, exposes cementum and dentin that are less resistant to decay than enamel. A clinical study of 600 patients older than age 60 years showed that 70% had root caries and 100% had some degree of gingival recession.[81] The removal of root caries is irritating to the pulp and often results in pulp exposures or reparative dentin formation that affects the negotiation of the canal if root canal treatment is later needed (Fig. 24-4).[20] Asymptomatic pulp exposures on 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. 24-5).

Interproximal root caries is difficult to restore, and restoration failure as a result of continued decay is common (Fig. 24-6). 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.[74]

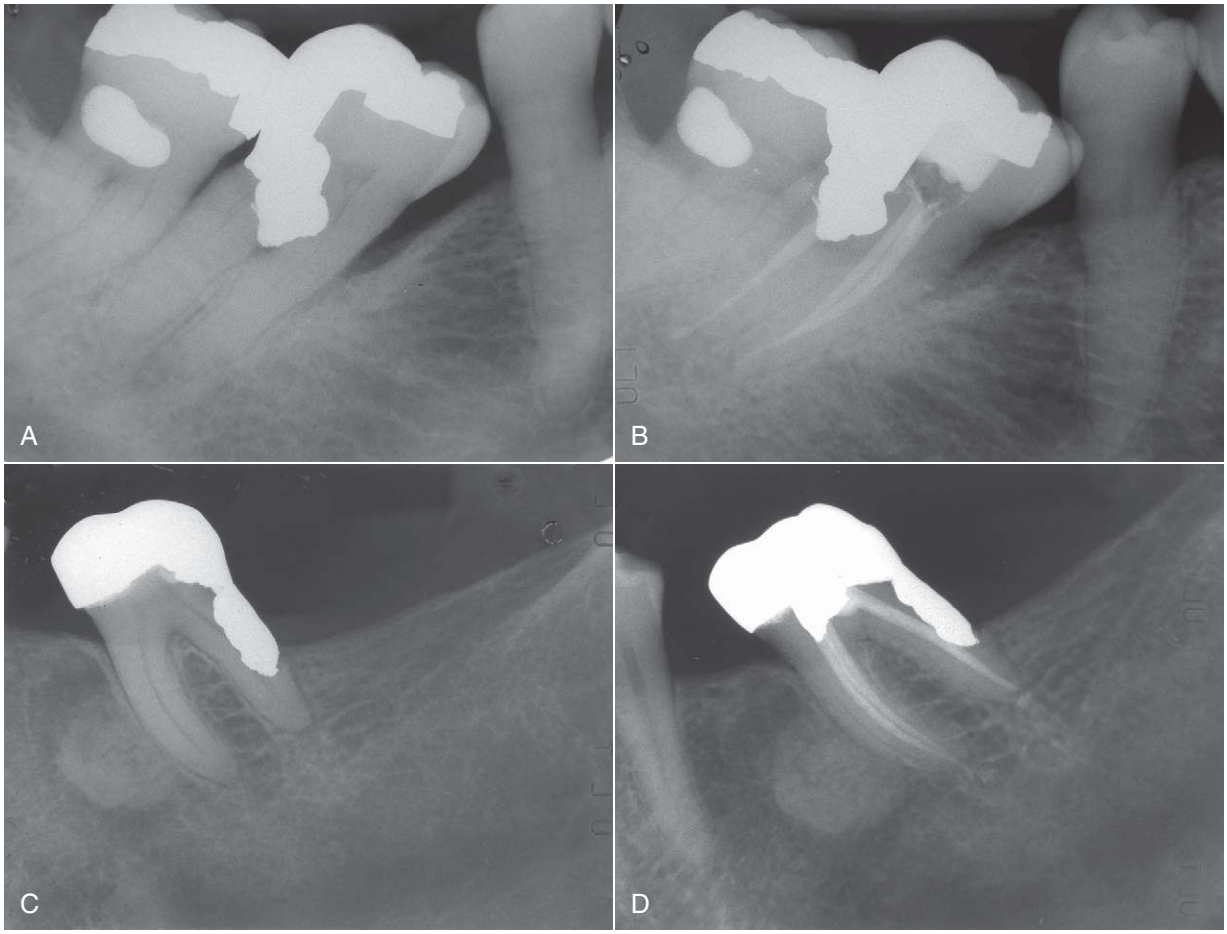


FIG. 24-4 Gingival recession exposes cementum and dentin, which are less resistant to caries. **A-B,** Root caries often results in pulp exposures **(C-D)** that require endodontic treatment.

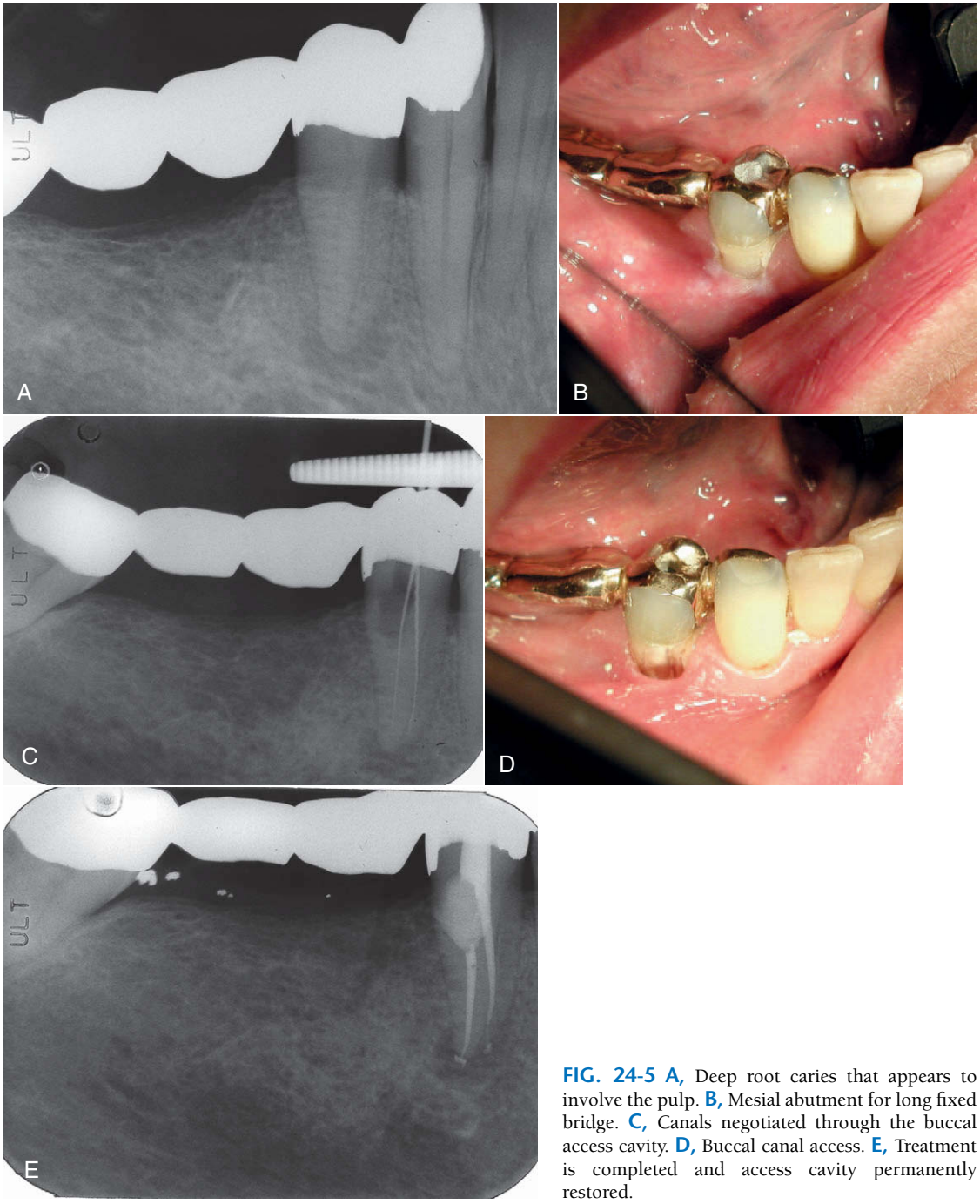


FIG. 24-5 A, Deep root caries that appears to involve the pulp. **B,** Mesial abutment for long fixed bridge. **C,** Canals negotiated through the buccal access cavity. **D,** Buccal canal access. **E,** Treatment is completed and access cavity permanently restored.

Attrition (see Fig. 24-6), abrasion, and erosion (Fig. 24-7) also expose dentin through a slower process that allows the pulp to respond with dentinal sclerosis and reparative dentin.[67] 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[55]; in molar teeth the greatest deposition occurs on the floor of the chamber.[10] Although this pulp may appear to recede, small pulpal remnants can remain or leave a less calcific 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. 24-8). 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. 24-9).[37] The thickness of young apical cementum[60] is 100 to 200 μm and increases with age to two or three times that thickness.[86]

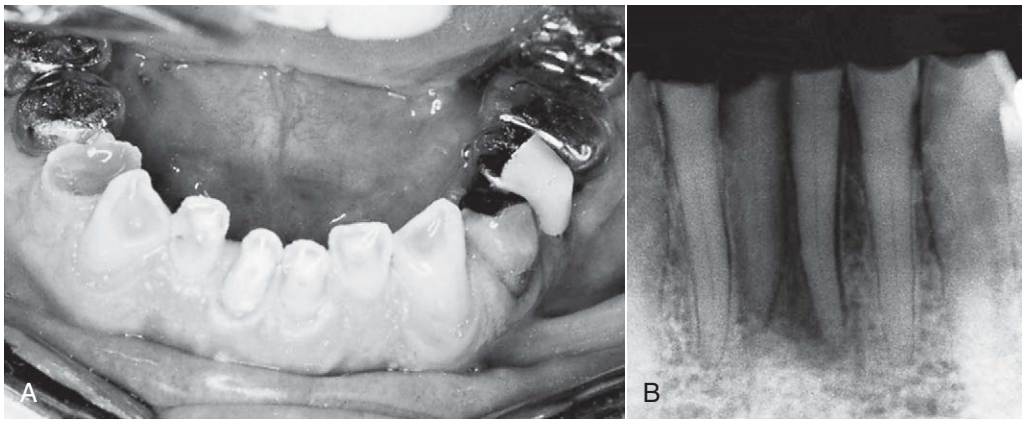


FIG. 24-6 A, Attrition exposes dentin through a slow process that allows the pulp to respond with reparative dentin, but pulp exposure eventually becomes clinically evident **(B)**.

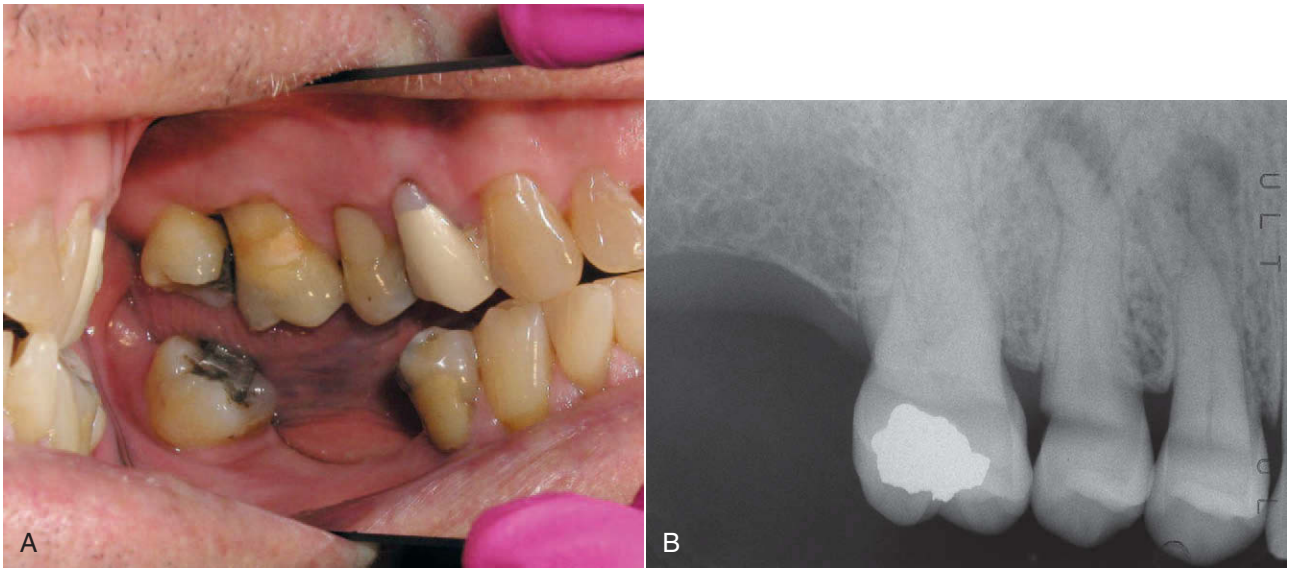


FIG. 24-7 Gingival recession also exposes cementum and dentin that are less resistant to abrasion and erosion, which can expose pulp or require restorative procedures that could result in pulp irritation.

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. 24-10). Dentinal tubules become more occluded with advancing age, decreasing tubular permeability.[47] 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.[62] 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.[11] 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. 24-7) may be evident as a result of staining, but they do not indicate dentin penetration or pulp exposure.[13,32] 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 as well as a periapical one. If incomplete cracks are not detected early, the prognosis for cracked teeth in older patients is questionable (Fig. 24-11).

Periodontal disease may be the principal problem for dentate seniors.[18] The relationship between pulpal and periodontal disease can be expected to be more significant with

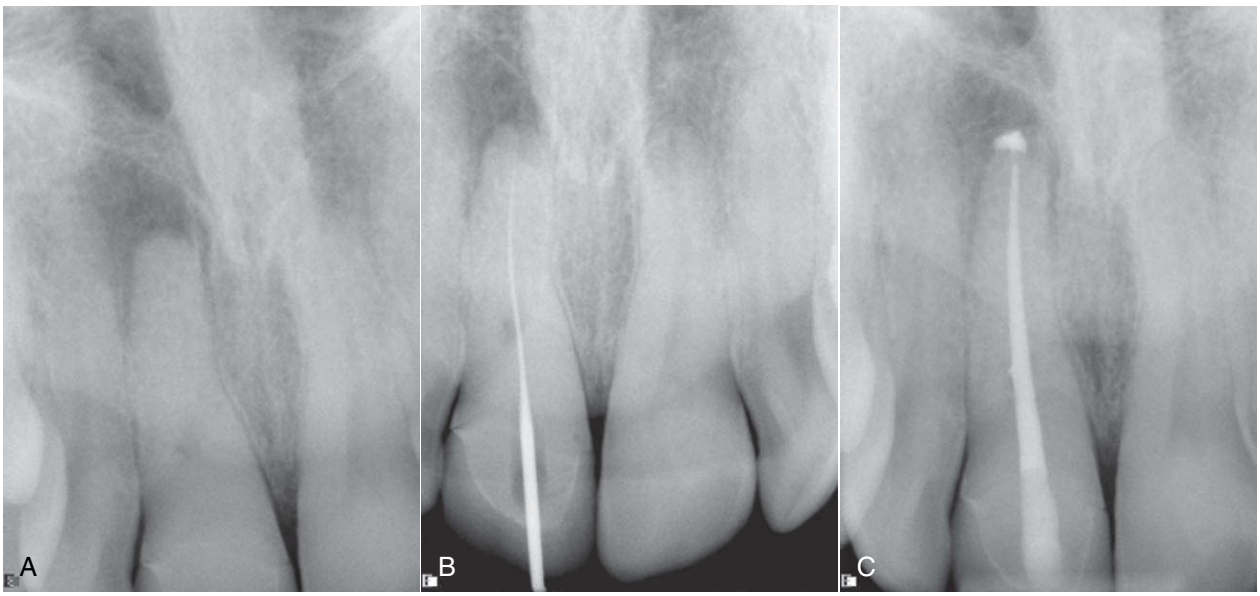


FIG. 24-8 A, In general, canal chamber volume is inversely proportional to age. As age increases, canal size decreases. The upper right central incisor exhibits a reduced canal and chamber volume. **B,** Negotiation using small hand files. **C,** Treatment completed for this calcified canal.



FIG. 24-9 Histologic image of the cementodentinal junction (CDJ) in an older tooth illustrates increased cementum deposition with age and increased distance from the apex. *(Courtesy Dr. Thomas Stein.)*



FIG. 24-10 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.

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.[41] The periodontal tissues must be considered a pathway for sinus tracts (Fig. 24-12). 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.[22]

Periodontal treatment can produce root sensitivity, disease, and pulp death.[40] In developing a successful treatment plan, it is important to determine the effects of periodontal disease and its treatment on the pulp (Fig. 24-13). Age was not a factor in response to periodontal therapy for advanced periodontal tissue breakdown.[39] The mere increase in incidence and



FIG. 24-11 A split tooth should always be considered when a nonvital pulp and chronic apical periodontitis are evident on a nonrestored tooth. The crack usually extends into the periodontium, and the prognosis is generally poor.

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 as pathways of communication,[59] as is the permeability of dentinal tubules.

Examination of sinus tracts should include tracing with gutta-percha cones to establish the tracts' origins (Fig. 24-14). 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 débridement 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. Slow and gentle testing should be done to determine

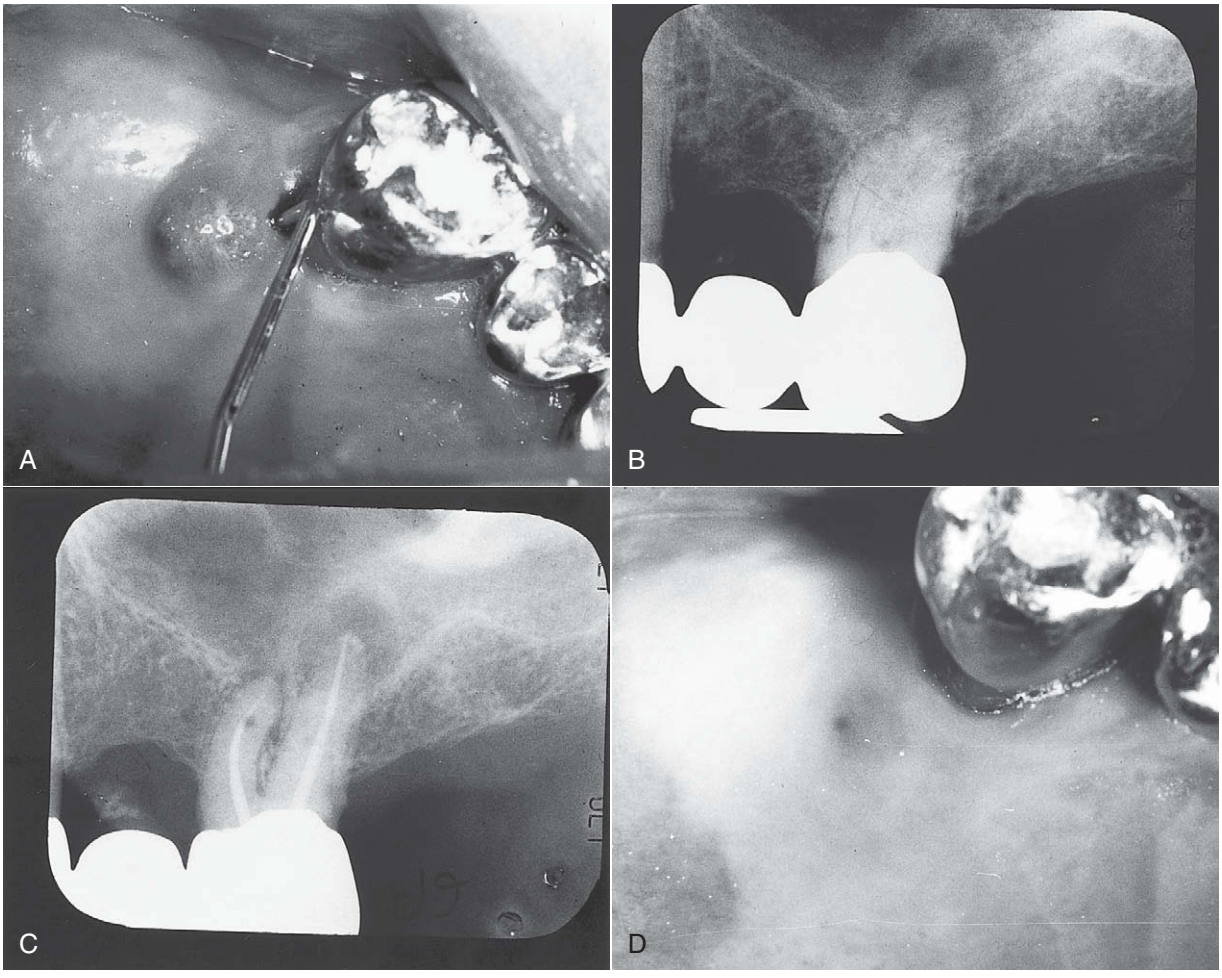


FIG. 24-12 A, A periodontal pocket is probed, and a sinus tract is evident on this maxillary molar abutment. **B,** This could indicate pulpal or periodontal disease or both. **C,** Root canal treatment results in healing **(D)** that confirms the pulpal cause of this periradicular lesion.

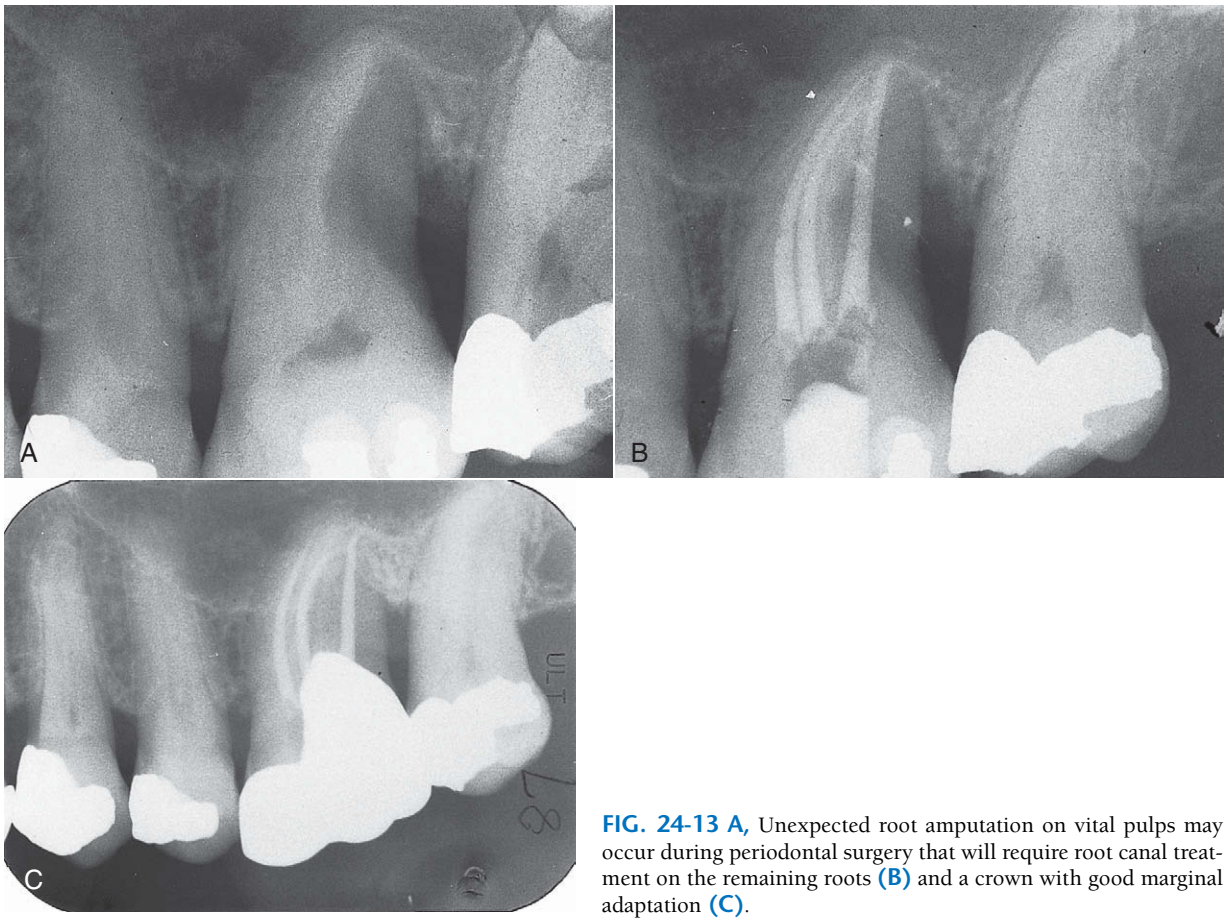


FIG. 24-13 A, Unexpected root amputation on vital pulps may occur during periodontal surgery that will require root canal treatment on the remaining roots **(B)** and a crown with good marginal adaptation **(C)**.

pulp and periapical status and whether palliative or definitive therapy is indicated. Vitality responses must correlate with clinical and radiographic findings and be interpreted as a supplement in developing clinical judgment. (Techniques for clinical pulp-testing procedures are discussed in Chapter 1.)

Transilluminating[56] 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 when pulpal or periapical disease is observed and little or no cause for pulpal irritation can be observed clinically or on radiographs. The high magnification available with microscopes during access opening and canal exploration permits visualization of the extent of cracks in determining prognosis. Cracks that are detected while the pulp is still vital can offer a reasonable prognosis if immediately restored with full cuspal coverage. The chronic nature of any periapical pathologic condition caused by vertically cracked teeth indicates that it is longstanding, and the prognosis is questionable (even when pocket depths appear normal). Periodontal pockets associated with cracks indicate a poor prognosis.

The reduced neural and vascular components of aged pulps,[9] the overall reduced pulp volume, and the change in character of the ground substance[66] create an environment that responds differently to both stimuli and irritants than that of younger pulps (Fig. 24-15). Arteriosclerosis, a common condition in older people, has not been shown to occur in the pulp.[36]

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. 24-16).[8] 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. 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.[26] Attachments that reduce the amount of surface contact necessary to conduct the electric stimulus are available (SybronEndo, Orange, CA), and bridging the tip to a small area of tooth structure with an explorer has been suggested.[53] Use of even this small electric stimulus in patients with pacemakers[83] is not recommended; any such risk would outweigh the benefit. The same caution holds true for electrosurgical units.

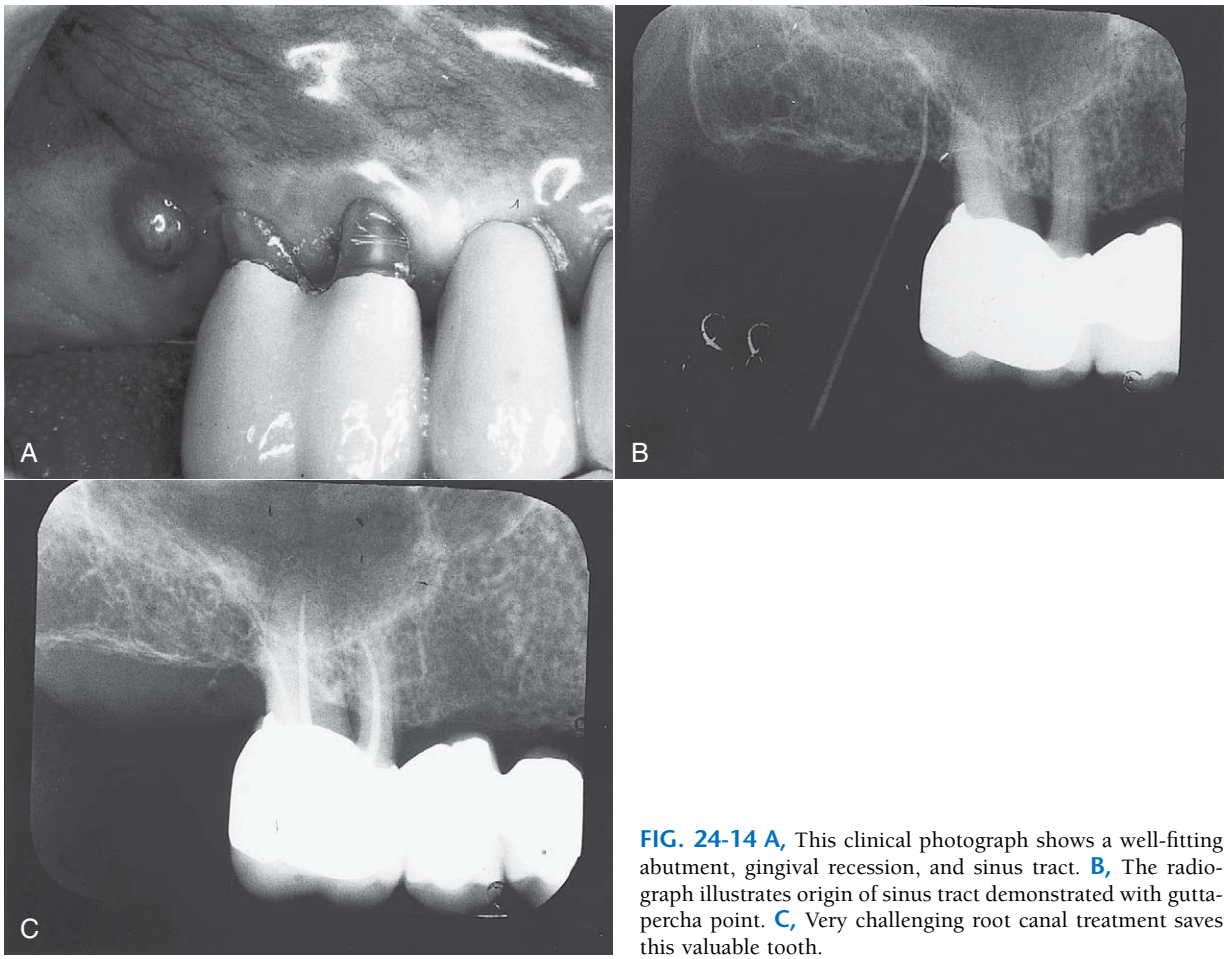


FIG. 24-14 A, This clinical photograph shows a well-fitting abutment, gingival recession, and sinus tract. **B,** The radiograph illustrates origin of sinus tract demonstrated with gutta-percha point. **C,** Very challenging root canal treatment saves this valuable tooth.



FIG. 24-15 The cellularity of older pulp tissue gradually decreases, and the odontoblasts decrease in size and number and may disappear altogether in certain areas, particularly on the pulpal floor in multirooted teeth.

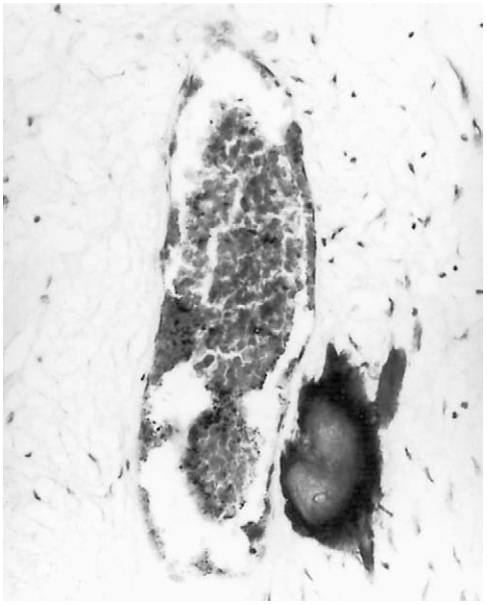


FIG. 24-16 Fibrosis appears to occur along the pathways of degenerated vessels and may serve as foci for pulpal calcification.

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. 24-17); 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.

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.

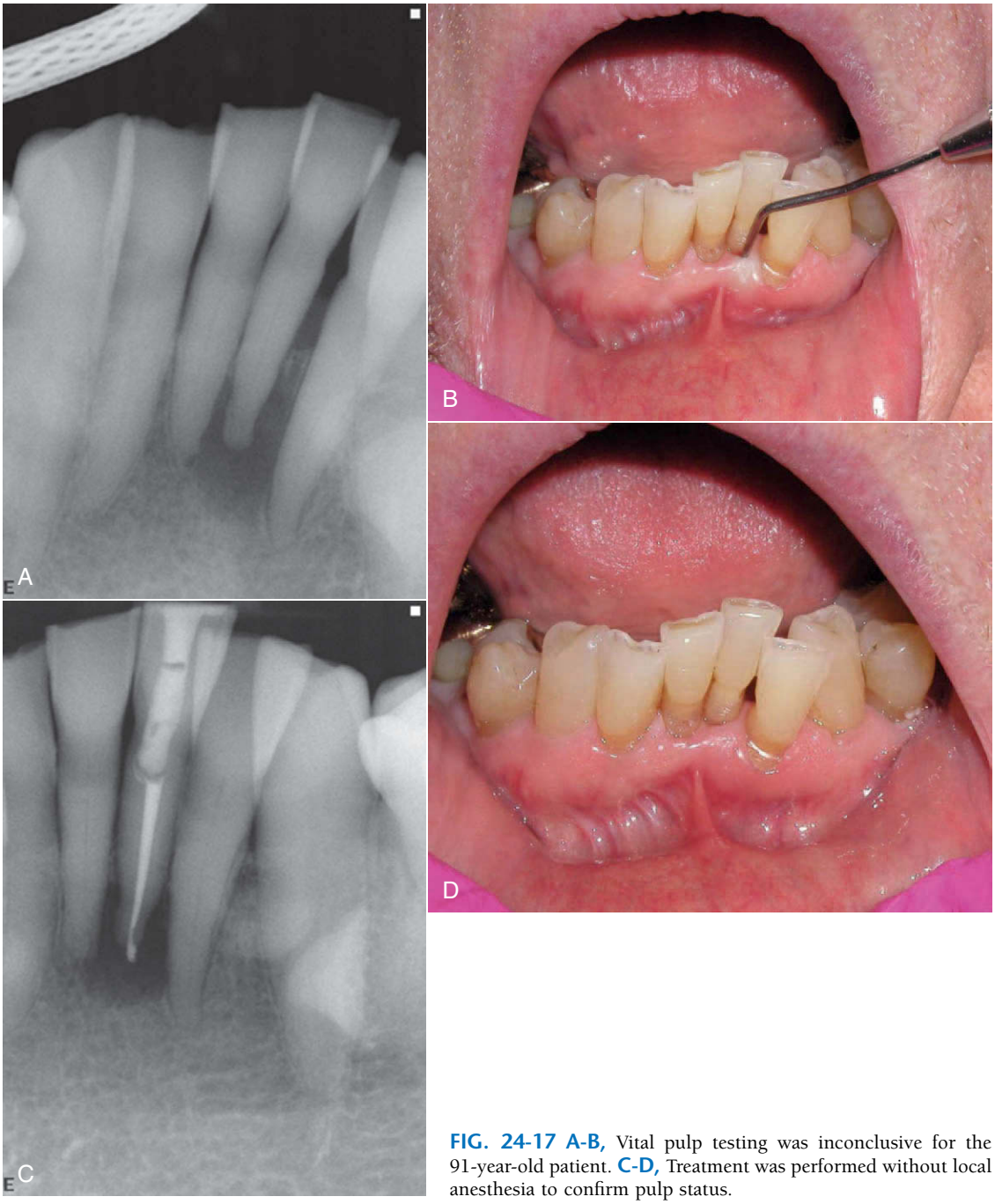


FIG. 24-17 A-B, Vital pulp testing was inconclusive for the 91-year-old patient. **C-D,** Treatment was performed without local anesthesia to confirm pulp status.

Radiographs

Indications for and techniques of taking radiographs do not differ much among adult age groups. However, several physiologic, 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. 24-18) 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 after the original diagnostic radiograph suggests that more information is needed for diagnosis or to determine the degree of difficulty of treatment. Digital radiography may be more useful than conventional radiography in detecting early bone changes.[85]

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.[38] Comparisons with adjacent teeth should be made. Small canals are the rule in older patients. A 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]

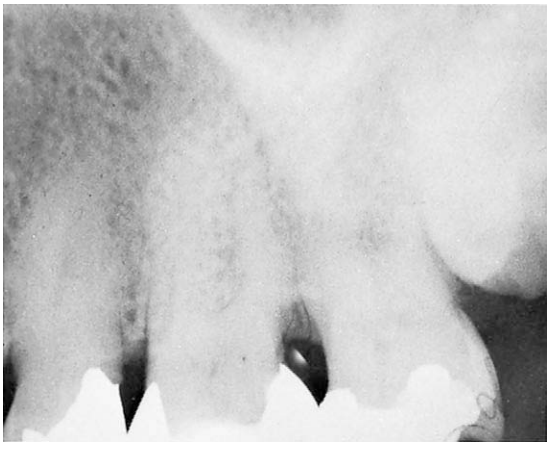


FIG. 24-18 Dense bone may indicate the need for increased exposure times to improve contrast needed to see the canal and root anatomy.

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. 24-19).[15] 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. 24-20) 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 cleansing, shaping, and filling are clearly understood and consistently met.[24] 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. 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 attachments, guide planes for partial abutments, rest seats, or overdentures require significant tooth reduction (Fig. 24-21). Predicting the need for future root canal treatment and a clinician's ability to perform treatment later is 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. 24-22). 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

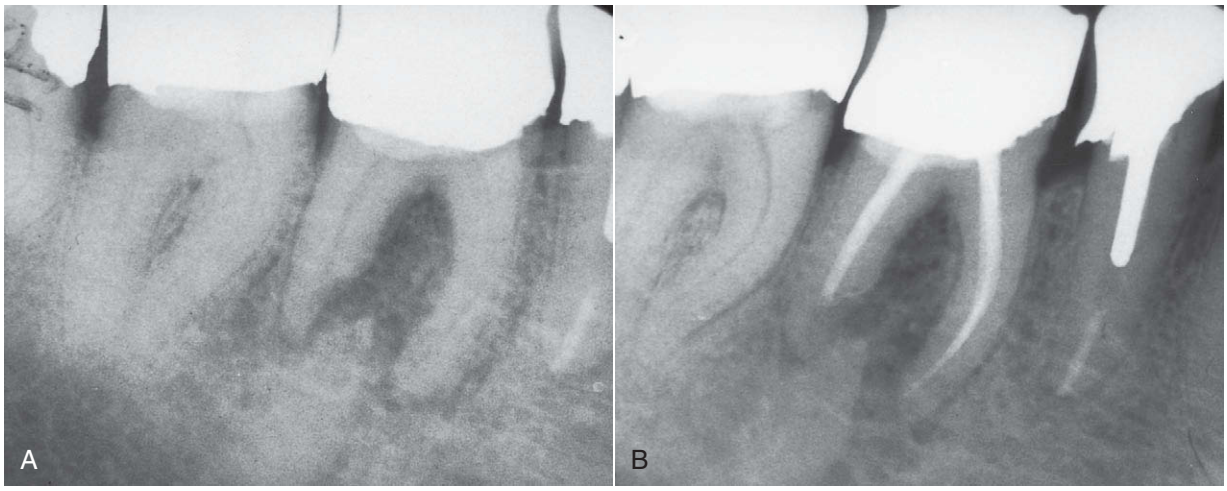


FIG. 24-19 A, Resorption associated with chronic apical periodontitis may alter the shape and position of the foramen through osteoclastic activity. **B,** The narrowest point in the canal is now positioned farther from the radiographic apex.

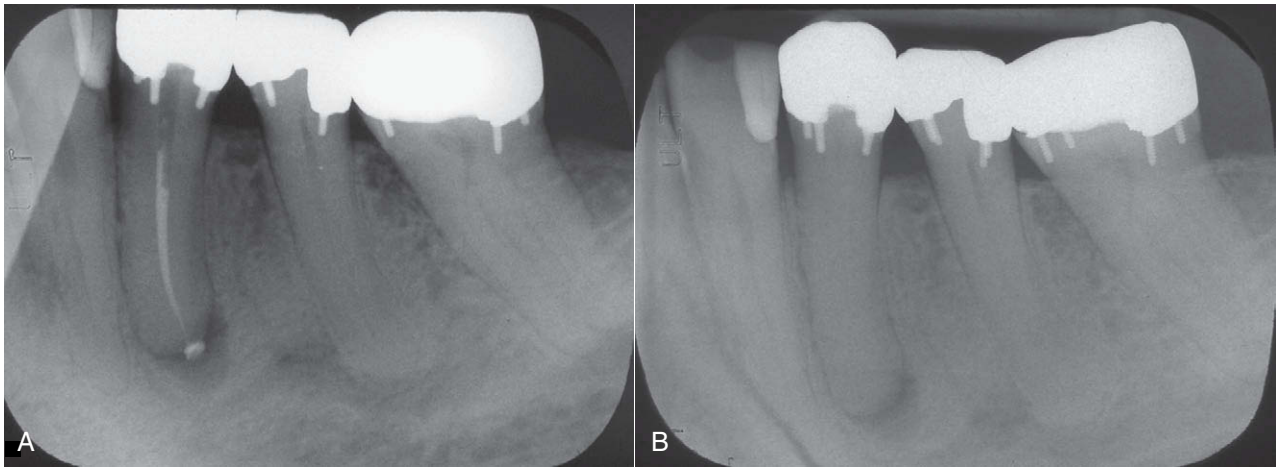


FIG. 24-20 A, Hypercementosis may completely obscure the apical anatomy and result in a constriction farther from the radiographic apex **(B)**.

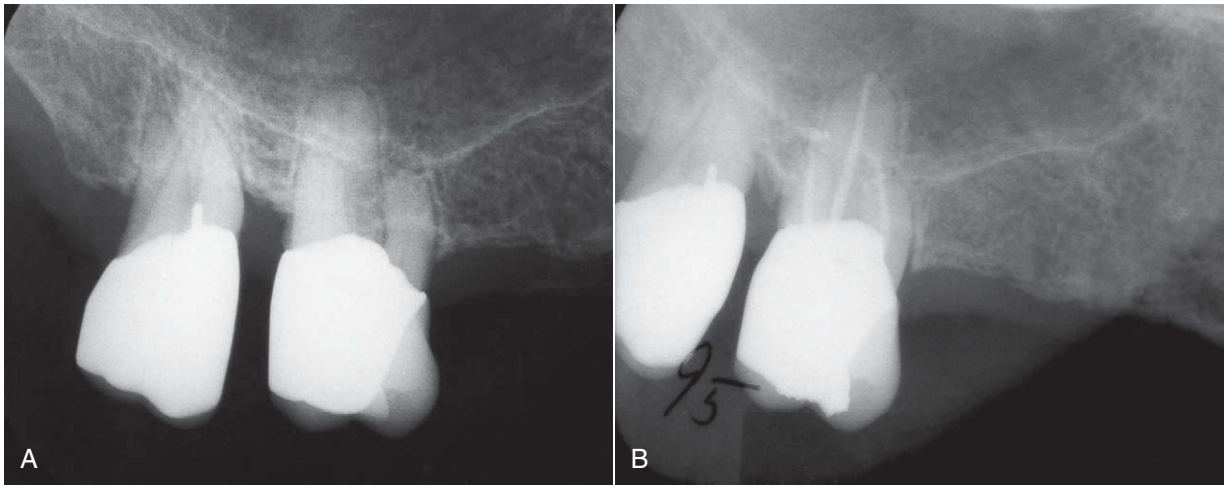


FIG. 24-21 A, Valuable maxillary first- and second-molar abutments in an 85-year-old patient. **B,** Later need for endodontic therapy was made very challenging because of access difficulties.

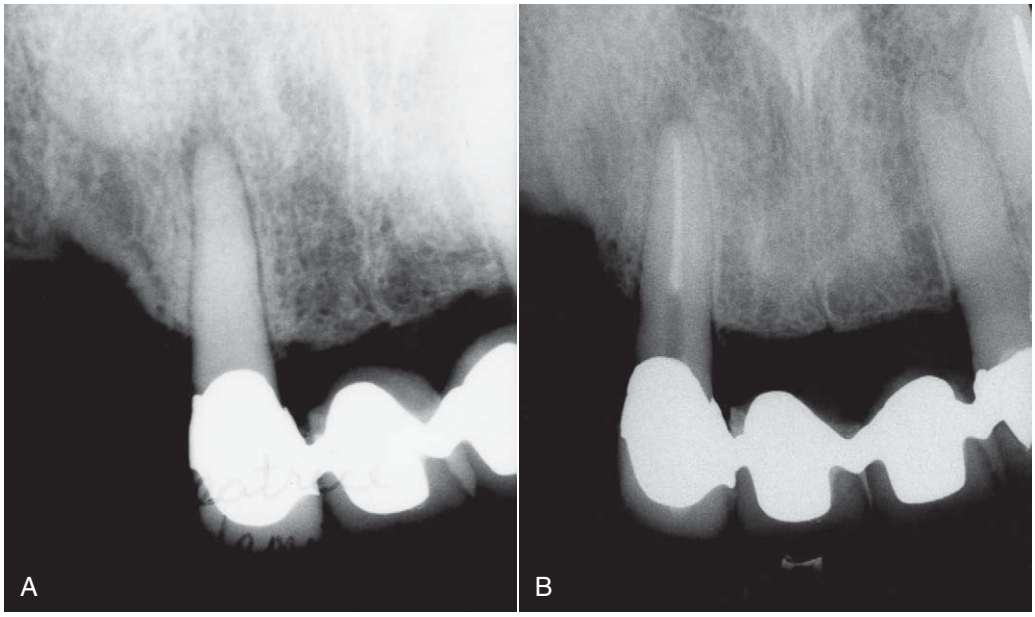


FIG. 24-22 A, Intracoronal attachments, guide planes, and rest seats for partial abutments require significant tooth reduction and a bulk of metal that makes endodontic treatment more likely and difficult. **B,** Successful treatment and preservation of the restoration when the canal is small are challenges to the most skilled clinician.

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. 24-23).

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. Obtaining signed consent to outlined treatment is encouraged and may be especially useful if the patient is forgetful.

Determining the patient's desires is as important as determining his or her needs, and it is required in obtaining informed consent (Fig. 24-24). Priorities in treating pain and infection to properly and esthetically restore teeth to health and function should be unaffected by age (Fig. 24-25). 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. 24-26). Patients deserve a 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, he said, "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 psychologic 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.

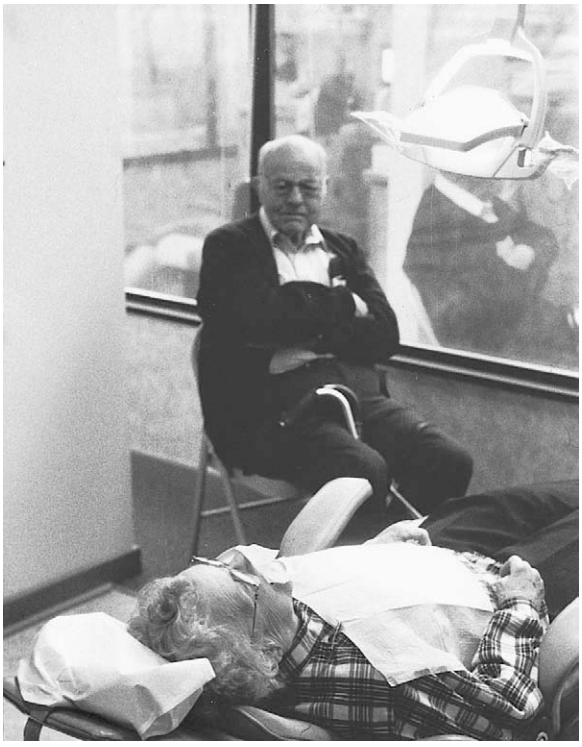


FIG. 24-23 Some patients may request or require that a family member be present during consultation or treatment.

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 there are specialists for performing root canals but will trust and do what their clinician recommends. In advance of the scheduled visit, referring clinicians 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, clinicians should consider making the referral appointment for the patient while he or she is in the dental office.

Obtaining informed consent before dental procedures usually is not a problem. However, medically compromised or cognitively impaired patients may make it 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 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

The vast majority of 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 (Fig. 24-27). Such access is a benefit required in most 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



FIG. 24-24 Many geriatric patients benefit from the use of a pillow for neck support.



FIG. 24-25 This 84-year-old patient was pleased to find that all these 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.

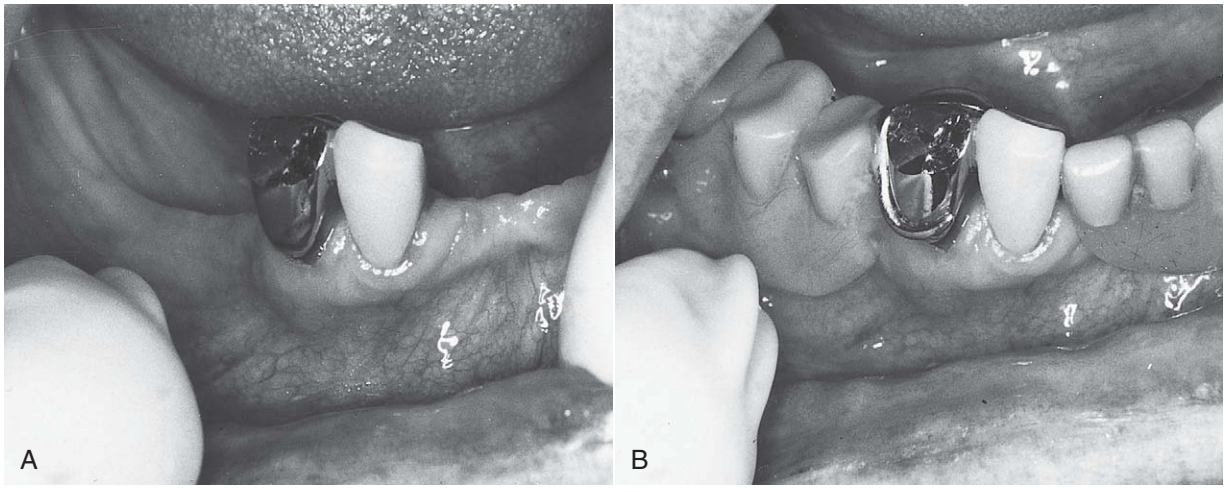


FIG. 24-26 A, These two remaining abutments were the only teeth this 99-year-old patient needed to retain a removable prosthesis **(B)**.



FIG. 24-27 A, Office design should consider access for wheelchairs and walkers. **B,** Staff should be experienced in assisting patients into and out of the dental chair and operatory.

office building, including both the interior design and the exterior approach, must be able to accommodate people with special needs.[52] 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 rest room. Dental units can be designed to accommodate patients while in their own wheelchairs so that they do not require any transfer.[72]

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, as well as 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.[19]

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 well as 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 fatigue is evident, the procedure should be terminated as soon as possible. Bite blocks are useful in comfortably maintaining freeway space and reducing jaw fatigue.

Retired persons 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 to 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.

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 they do not extend as far into the dentin. In addition, the dentinal tubules are more calcified.[9,66] A painful response may not be encountered until actual pulp exposure has occurred.

Anatomic landmarks that are 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[35] 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.[64] 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. Smaller amounts of anesthetic should be deposited during intraosseous injections, and the depth of anesthesia should be checked before repeating the procedure. Like intrapulpal anesthesia, intraosseous anesthesia is not prolonged; the pulp tissue must be removed within 20 minutes. The majority of patients receiving an intraosseous injection of 2% lidocaine with 1:100,000 epinephrine (correct ratio) solution 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 suggests caution, 3% mepivacaine is a good alternative for intraosseous injections.[57] Chapter 20 gives further information.

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

Either single-tooth or multitooth rubber dam isolation can be used. 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. 24-28). A three-holed rubber dam with the tooth in question in the middle allows for better maneuverability, especially when using the dental operating microscope. A petroleum-based lubricant for the lips and gingiva reduces chafing from saliva or perspiration beneath the rubber dam. Reduction in salivary flow and gag reflex reduces the need for a saliva ejector. Artificial saliva is available and should be used just before isolation, because it is difficult to apply after the dam is in place.



FIG. 24-28 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.

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 isolation cannot be compromised when sodium hypochlorite is used as an irrigant. Difficult-to-isolate defects produced by root decay present a good indication, in initial preparation, for the use of sonic handpieces that use flow-through water as an irrigant.

The many merits of single-visit root canal procedures is generally recommended when isolation is compromised (see Chapters 4 and 5). The few minor benefits of multiple-visit treatment are further reduced if an interappointment seal is difficult to obtain.

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. Canal position, root curvature, and axial inclinations of roots and crowns should be considered during the examination (Fig. 24-29). The effects of access on existing restorations and the possible need for actual removal of the restoration should be discussed with the patient before initiating treatment (Fig. 24-30). Coronal tooth structure or restorations should be sacrificed when they compromise access for preparation or filling (Fig. 24-31). Perhaps all restorations should be removed before endodontic treatment in order to remove the common factors (caries, marginal breakdown, cracks) that may have caused the pulp and periradicular disease and to assess the tooth's prognosis and future treatment needs. One study found that almost all teeth (93%) had these factors; only 56% were detectable clinically or on radiographs.[1] (See Chapter 7 for more details on proper access openings.)

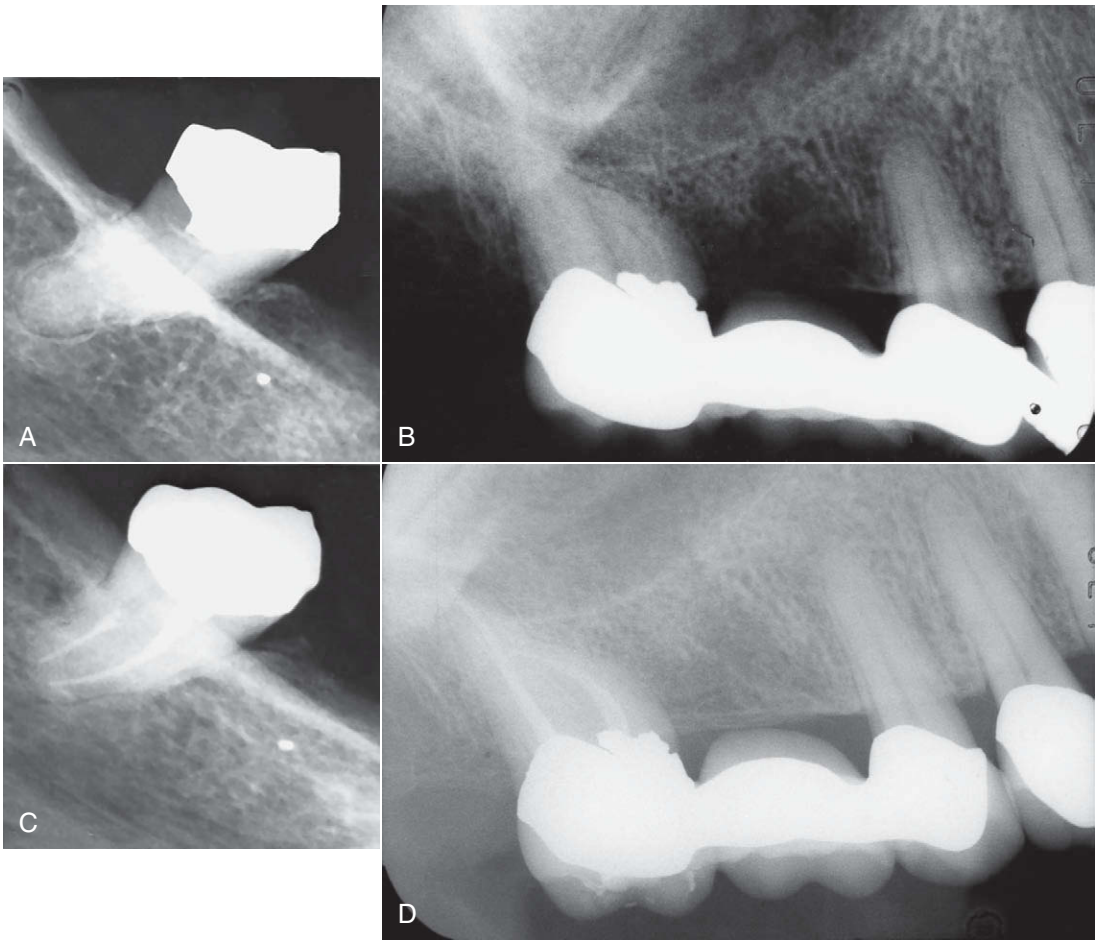


FIG. 24-29 A, This mandibular third molar in a 78-year-old patient has tilted mesially and has been restored with a full crown, upright to allow a path of insertion for a removable partial denture. **B,** Successful root canal treatment is the primary objective. Retaining the crown when possible also represents significant savings to the patient. This crown will be replaced; a post space has been prepared. **C,** This maxillary third molar on the same patient is tilted mesially and rotated, making access **(D)** the most difficult part of this treatment.

Magnification in the range of 2.5× to 4.5× (e.g., Designs for Vision, Ronkonkoma, NY) 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[34] offer clear magnification ranging up to 25× or greater and have obvious advantages in treating smaller and narrower geriatric canals (see Chapter 6, Fig. 6-4).

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 will not stick in solid dentin, but it will resist 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 contraindicated 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 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. 24-32). 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. 24-33). Supraerupted teeth can be easily perforated (Fig. 24-34) 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. 24-35).

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 chronic apical periodontitis will usually have patent canals (Fig. 24-36). Surgical access (Fig. 24-37) may be preferred if the risk of deviation from the long axis exists

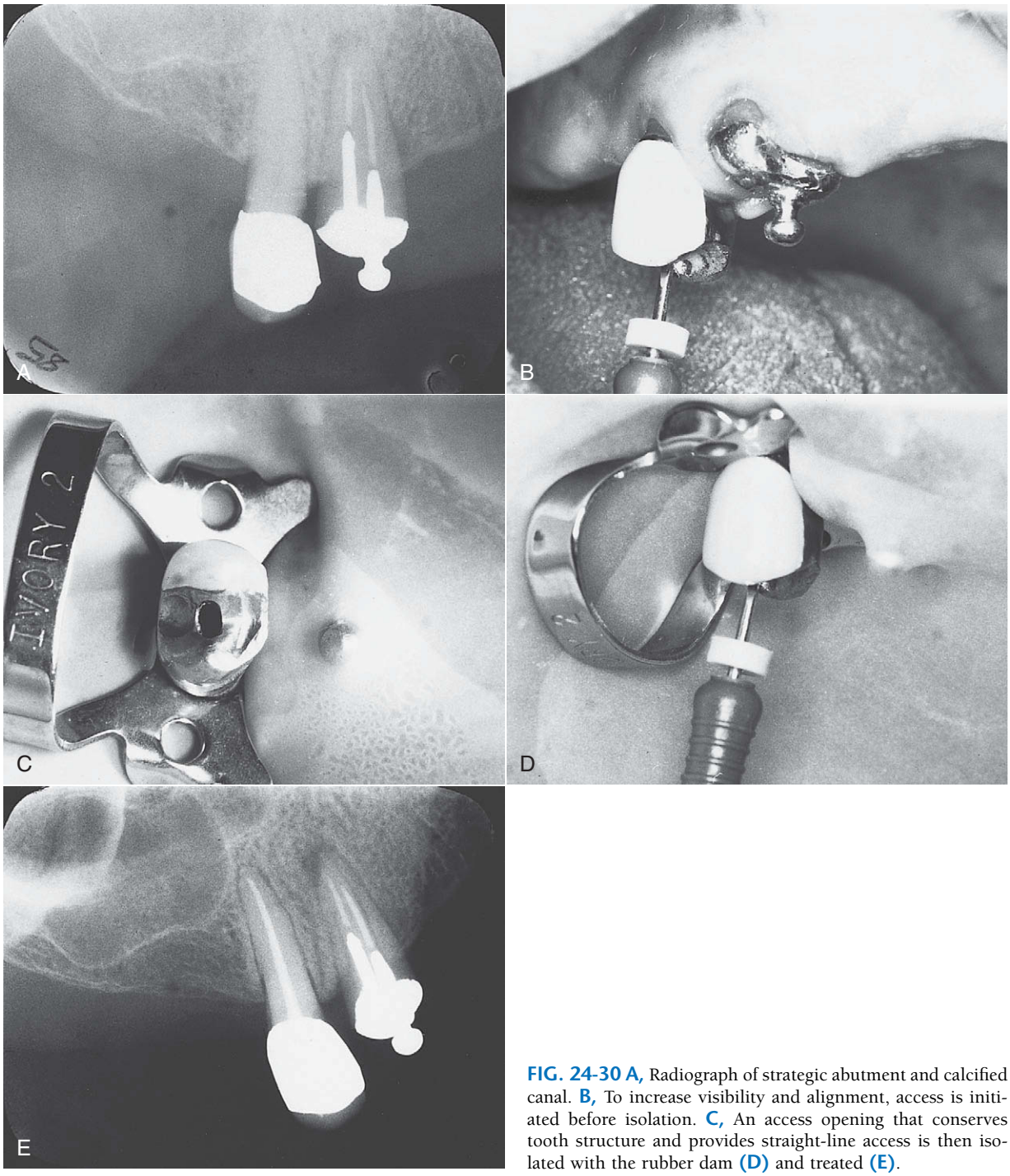


FIG. 24-30 A, Radiograph of strategic abutment and calcified canal. **B,** To increase visibility and alignment, access is initiated before isolation. **C,** An access opening that conserves tooth structure and provides straight-line access is then isolated with the rubber dam **(D)** and treated **(E)**.

when canals are calcified and the tooth is heavily restored (Fig. 24-38).

Very few canals of older teeth, even maxillary anterior teeth, have adequate diameter to allow the safe and effective use of broaches. Older pulps may give a clinical appearance that reflects their calcified, atrophic state with 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/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

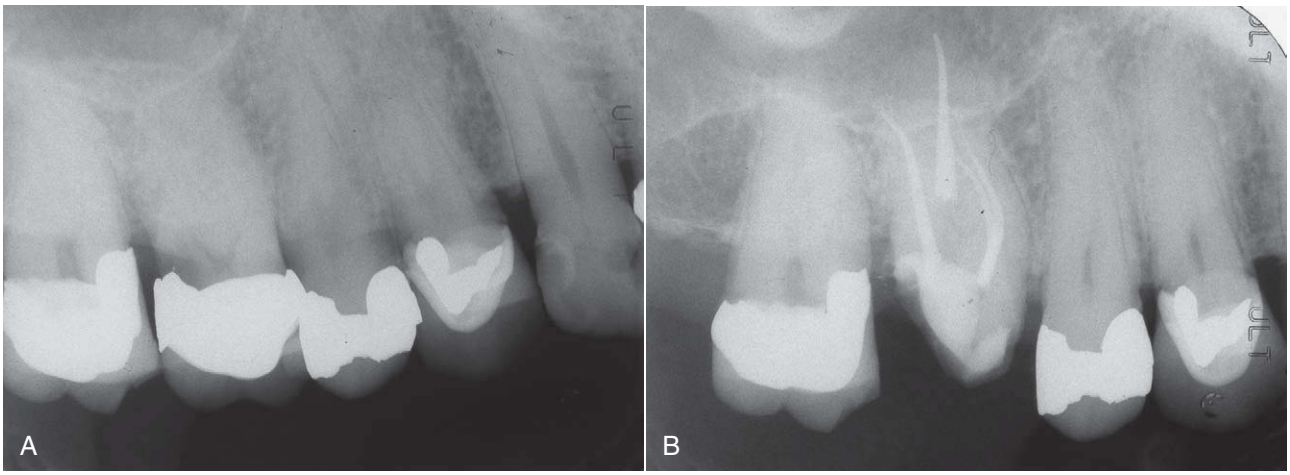


FIG. 24-31 A, Caries control and isolation of this tooth necessitated crown removal. **B,** Endodontic treatment was completed in one visit.

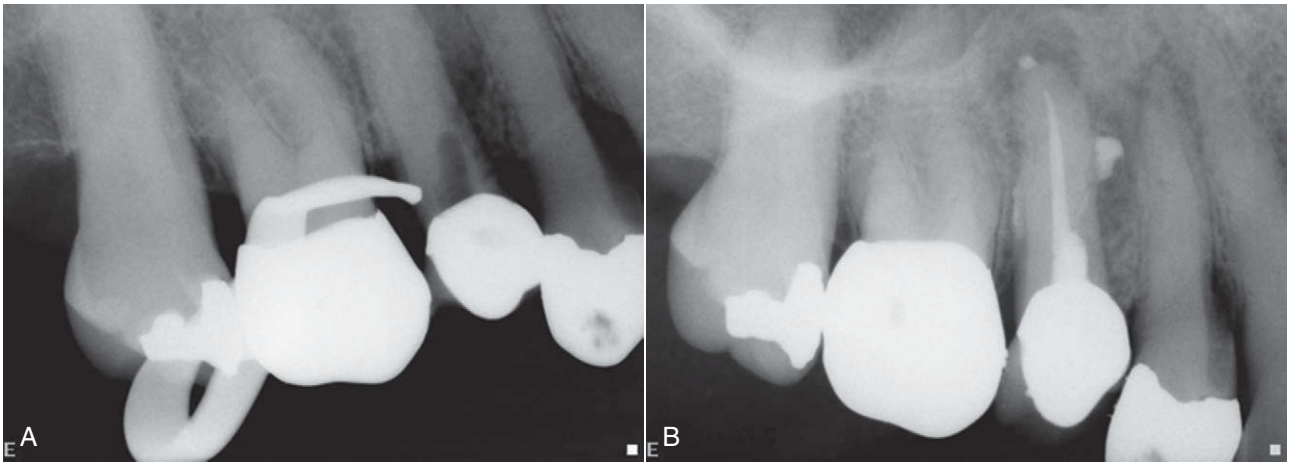


FIG. 24-32 A, Pain, bleeding, or disorientation of the probing instrument may indicate a perforation, which should be immediately investigated with a radiograph. **B,** Repair has the best success when the perforation opening is pinpoint and periodontal tissues are normal.

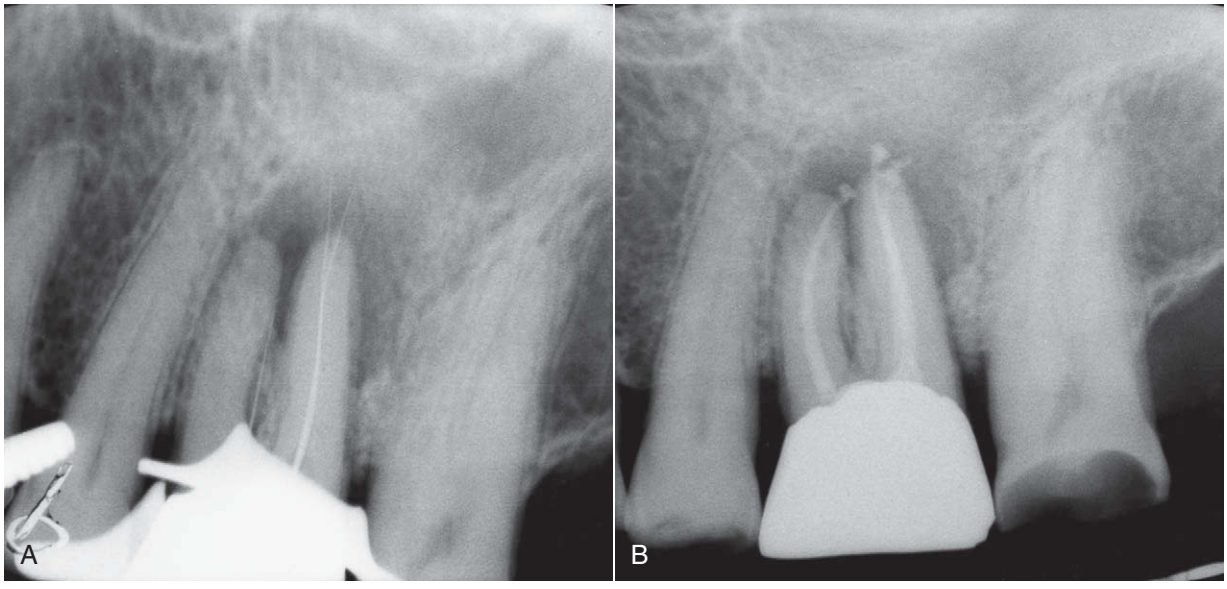


FIG. 24-33 A, Perforation occurred during access of these calcified canals. **B,** Endodontic treatment was immediately completed after the perforation repair.

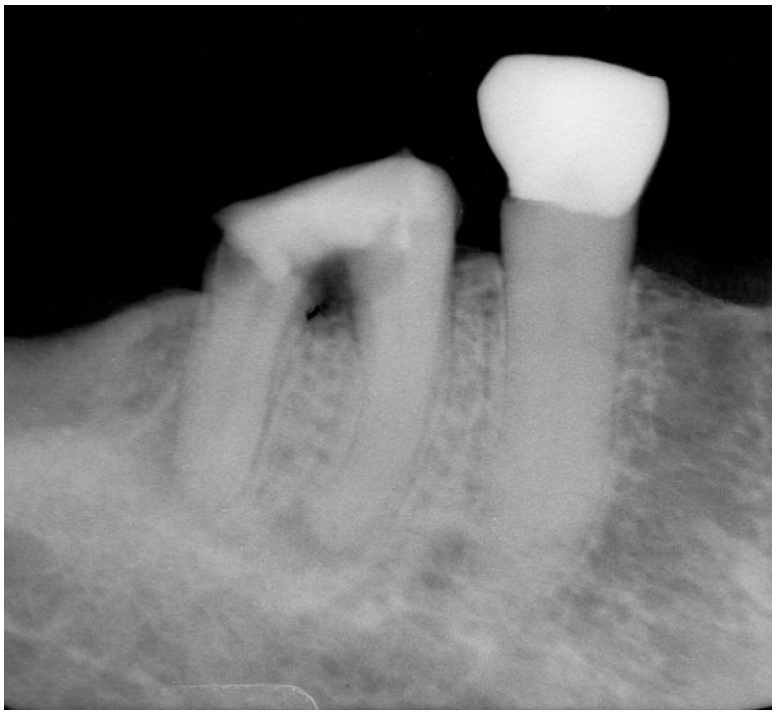


FIG. 24-34 Supraerupted mandibular first molar that was perforated during access.

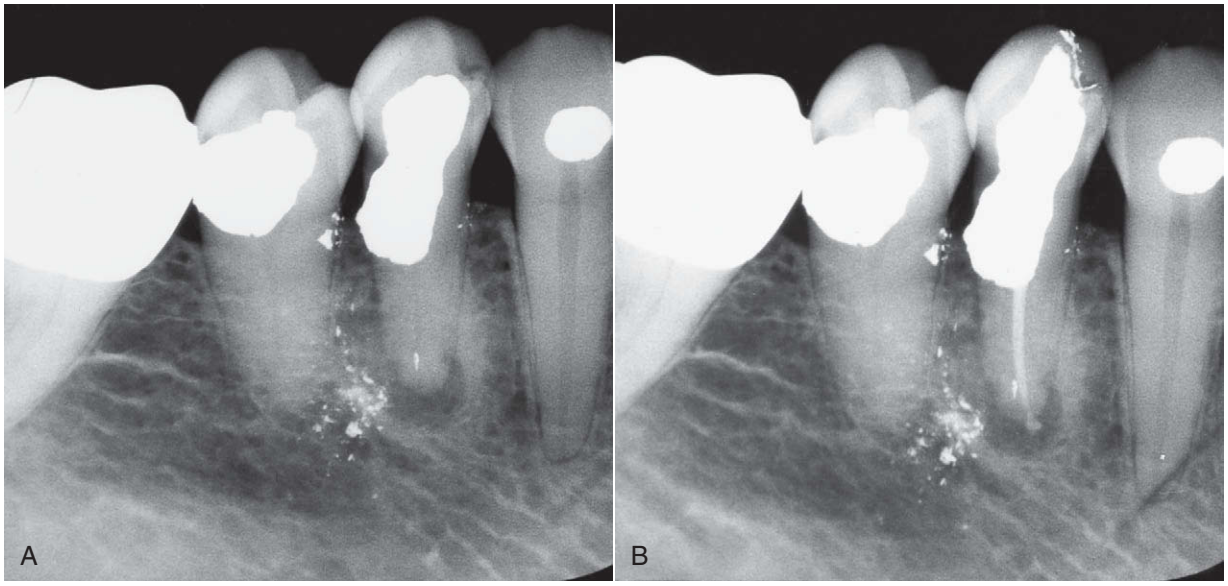


FIG. 24-35 A, Midtreatment referral of a mandibular first premolar only after perforation and repair with amalgam. **B,** Canal was still detectable with enhanced vision technique, and treatment was completed.

are found. An older tooth is more likely to have a history of earlier treatments, with a combination of calcifications present. (Chapter 9 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.[37] 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.[68]

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 with greater force than the fracture resistance of small files (when used with a reaming action) and result in instrument fatigue and fracture. The benefits of instruments with no rake angle and a crown-down technique are recommended.

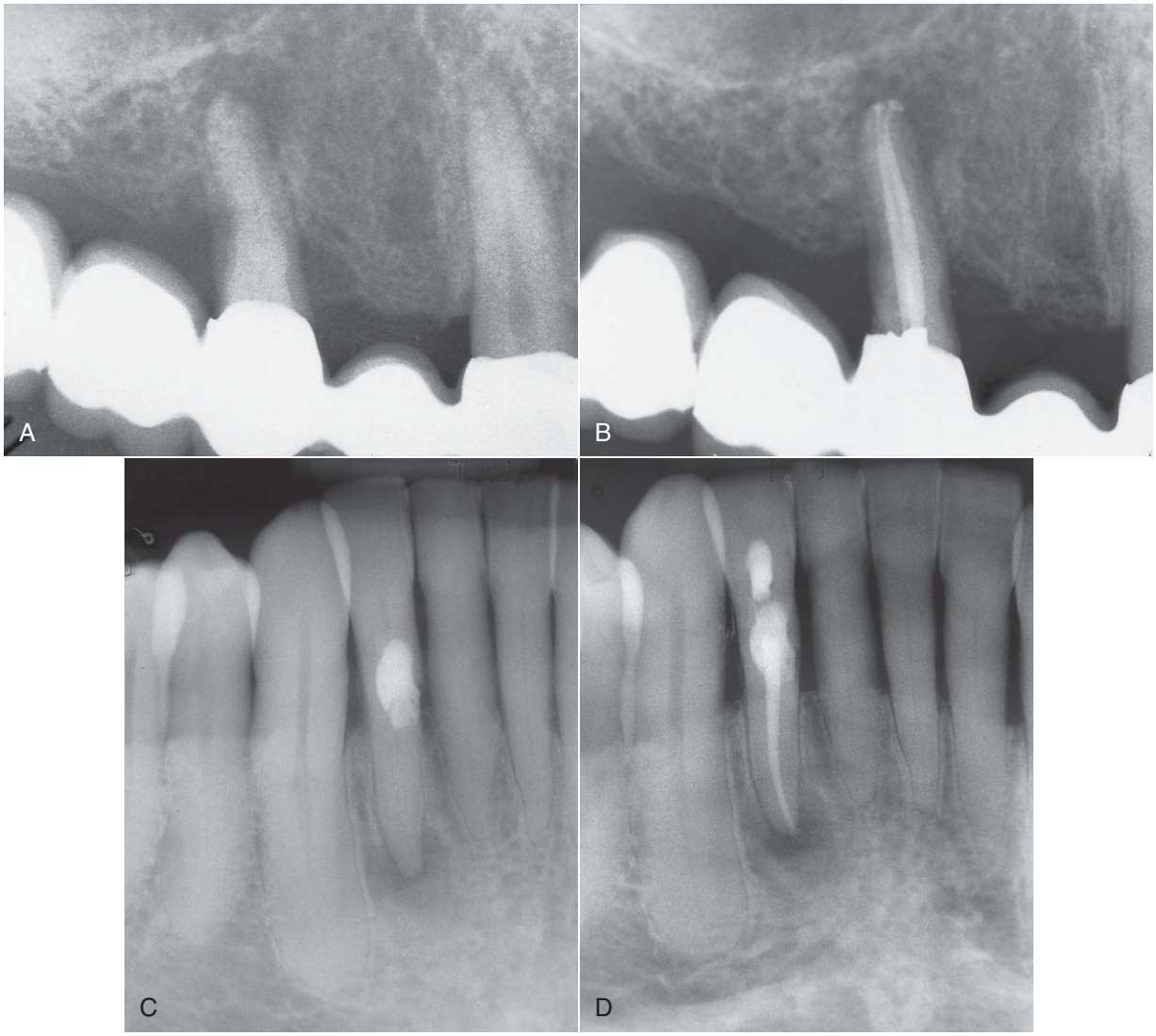


FIG. 24-36 A, Strategic middle abutment for a long fixed bridge presents a challenge to the most skilled clinician to perform predictable root canal treatment. **B,** Poor crown/root ratio makes root-end surgery a poor option. Root canal treatment should have been considered before using this lone-standing premolar 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 on 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.

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 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. 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 sometimes limited in heavily restored teeth when contact with metal can bleed off the current.

The frequency and intensity of discomfort 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.[71] 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 preparations[84] and further reduces the risk of overinstrumentation or the forcing of debris into the periapical tissues,[54] which could cause an acute apical periodontitis or

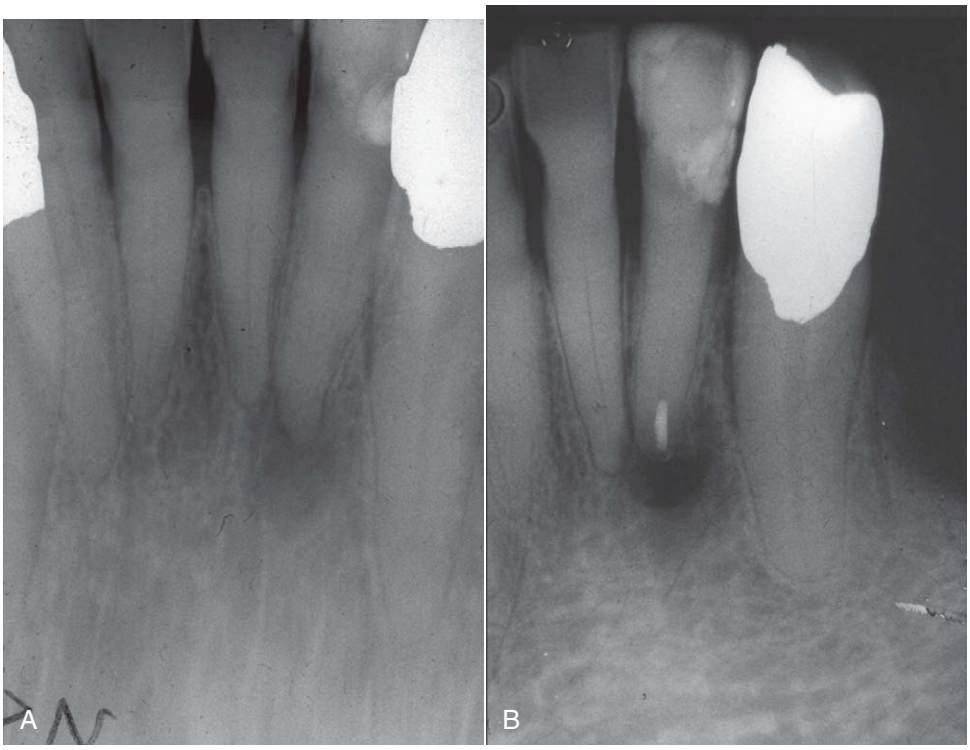


FIG. 24-37 A, Extremely calcified canals and thin roots make the risk of perforation high. **B,** Surgical access and root-end filling was performed rather than weakening the tooth trying to locate the root canal system via an orthograde approach.

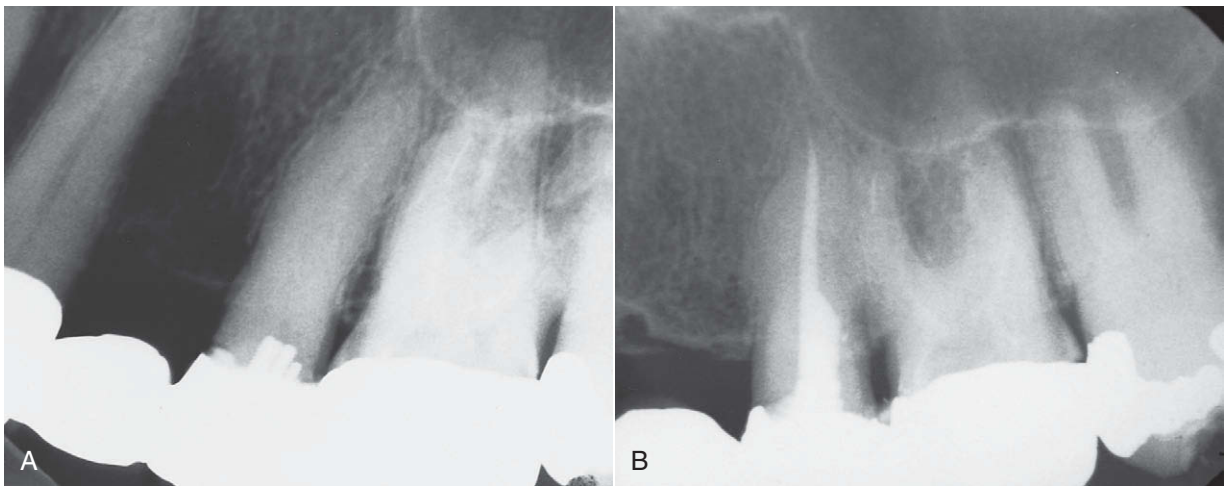


FIG. 24-38 A, Multiple pins were used to restore this middle abutment on a fixed bridge. **B,** Extreme care and exceptional skill are required to maintain proper alignment to find and treat the canal with a deep access opening.

abscess. Further access to periapical tissues through the canal is likewise limited.

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. 24-39). (See Chapter 10 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.[70] Even a root-filled tooth should not have its canals exposed to the oral environment. A thermoplastic synthetic polymer-based root-filling material (Resilon) may significantly reduce the coronal leakage that can result from root caries after root canal treatment, as well as increase resistance to root fracture.[61,73] Permanent restorative procedures should be scheduled as soon as possible, and intermediate

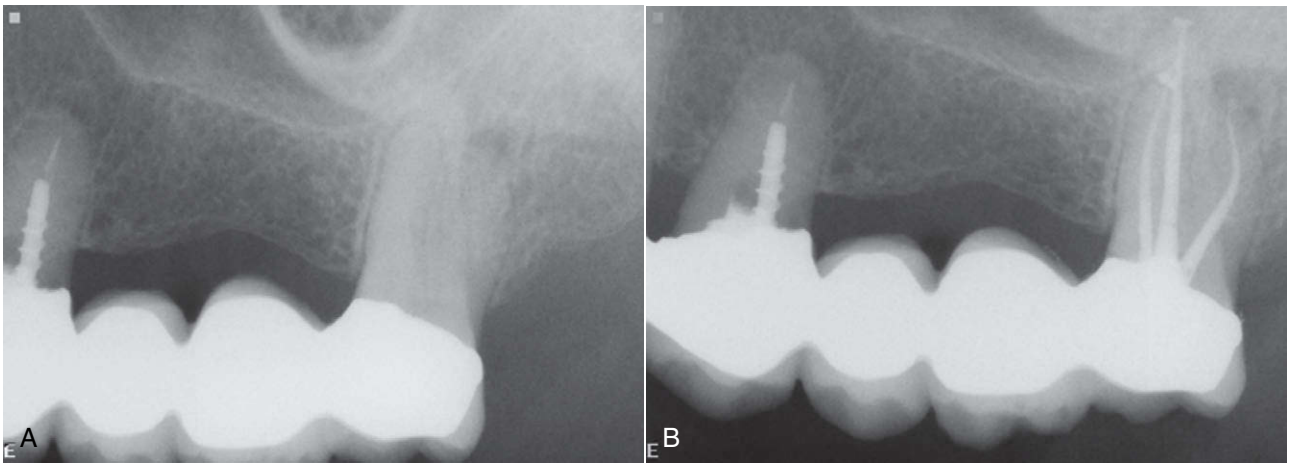


FIG. 24-39 A, Third molar abutment for 7-unit bridge in an 84-year-old patient with limited opening. **B,** Instrumentation of this molar without excessive removal of coronal or midroot dentin.

restorative materials should be selected and properly placed to maintain a seal until that time. When 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. Infected nonvital pulps with periapical pathosis must have this process 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. With aging, arteriosclerotic changes of 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 will heal as readily in elderly as in young patients,[71] a 6-month recall period to evaluate repair by means of radiographs may not be adequate (Fig. 24-40).[25]

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. 24-41), 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. (For further information on assessing failures and possible retreatment, see Chapter 25.)

ENDODONTIC SURGERY

Generally, considerations and indications for endodontic surgery are not affected by age. The need for establishing drainage and relieving pain are not common indications for surgery. Anatomic complications of the root canal system, such as small (Fig. 24-42) 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. 24-43). 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.[4] 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.[79] 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 treatment of older patients include an increase in the incidence of fenestrated or dehisced roots and exostoses (Fig. 24-44). 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. Ability to gain such access varies with the skill of the surgeon; however, some areas are unreachable by even the most experienced clinicians.

The position of anatomic features—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.

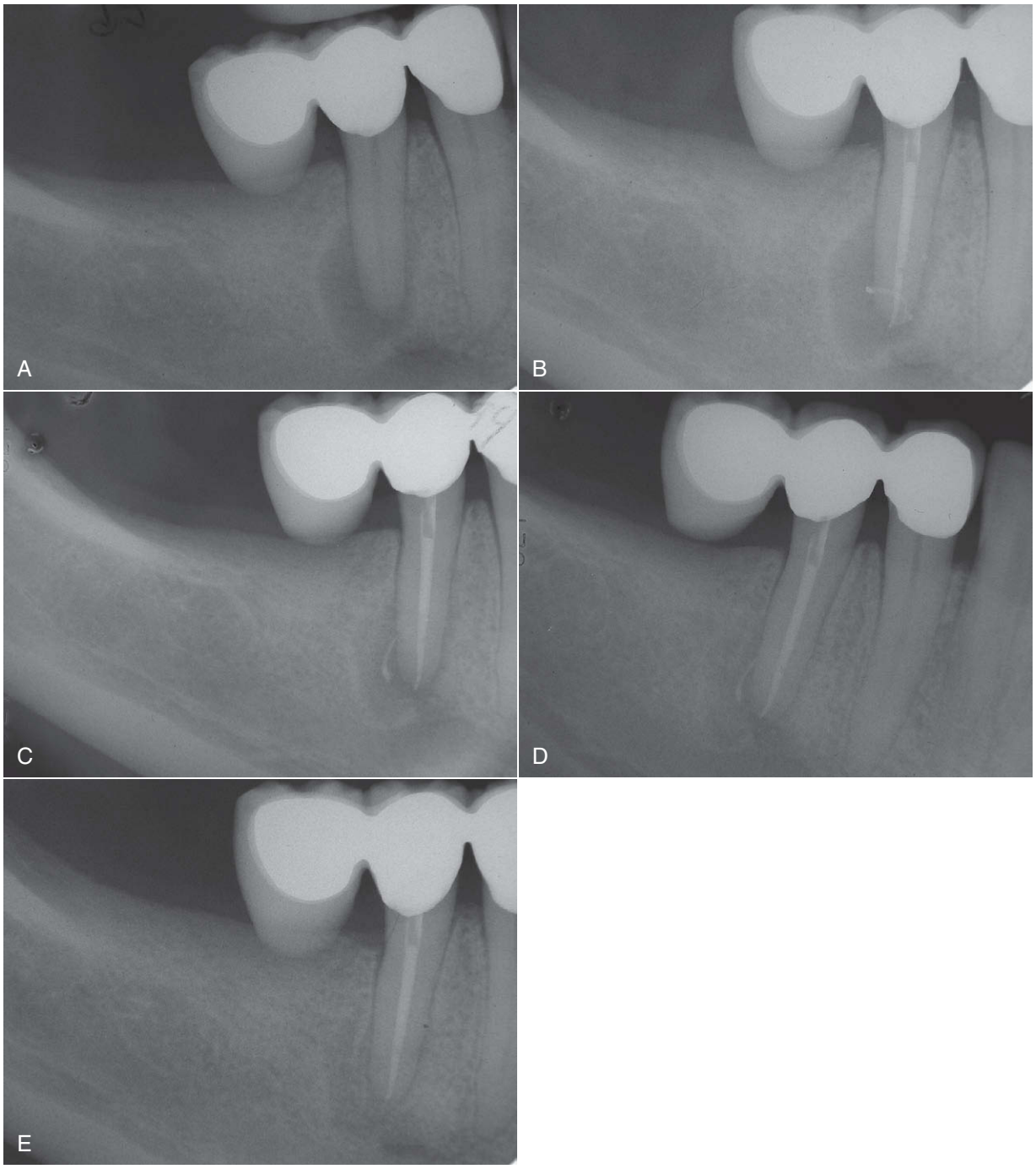


FIG. 24-40 A, Mandibular premolar in an older patient exhibits apical periodontitis. Following endodontic treatment **(B)**, healing was incomplete at 6 months **(C)** and after 1 year **(D)**. Complete healing of the apical periodontitis was seen 5 years postoperatively **(E)**.

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. 24-45). This factor is especially important when the tooth will be used as an abutment (Fig. 24-46). (Detailed surgical procedures are presented in Chapter 21.)

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 will change to brown and yellow before it disappears. Immediate application of an ice pack after surgery reduces bleeding and initiates coagulation to reduce the extent



FIG. 24-41 The incidence of root-end fillings during periradicular surgery increases with age as calcified canals become more difficult to detect and negotiate as a cause of failure of nonsurgical treatment.

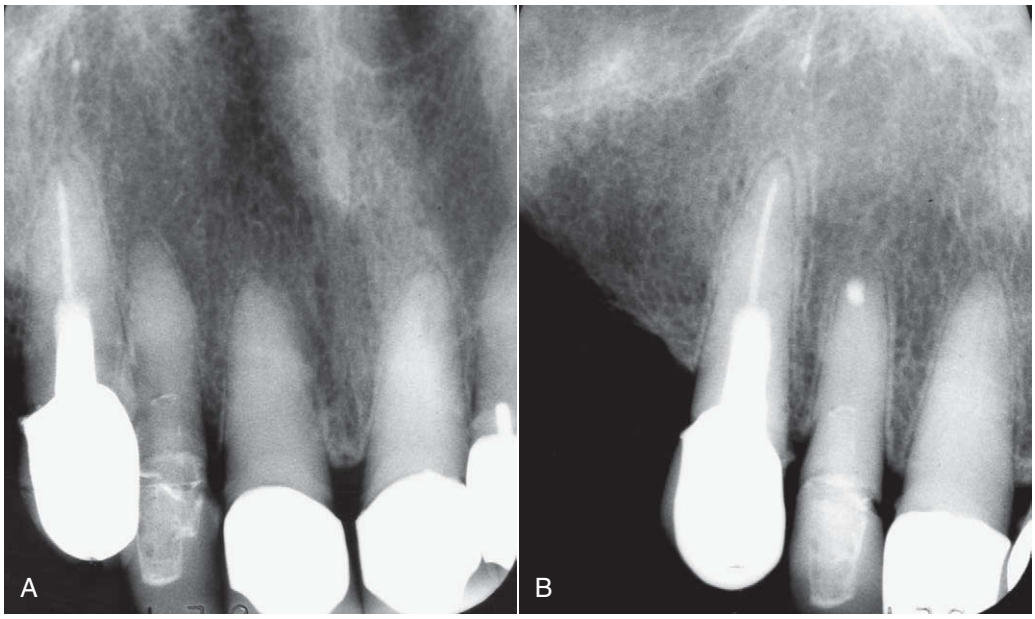


FIG. 24-42 A, Even though a small canal is detectable on the radiograph, it appears that an earlier unsuccessful attempt has been made to find it. **B,** It was decided that the risk of damage to a satisfactory restoration justified surgical treatment for this patient.

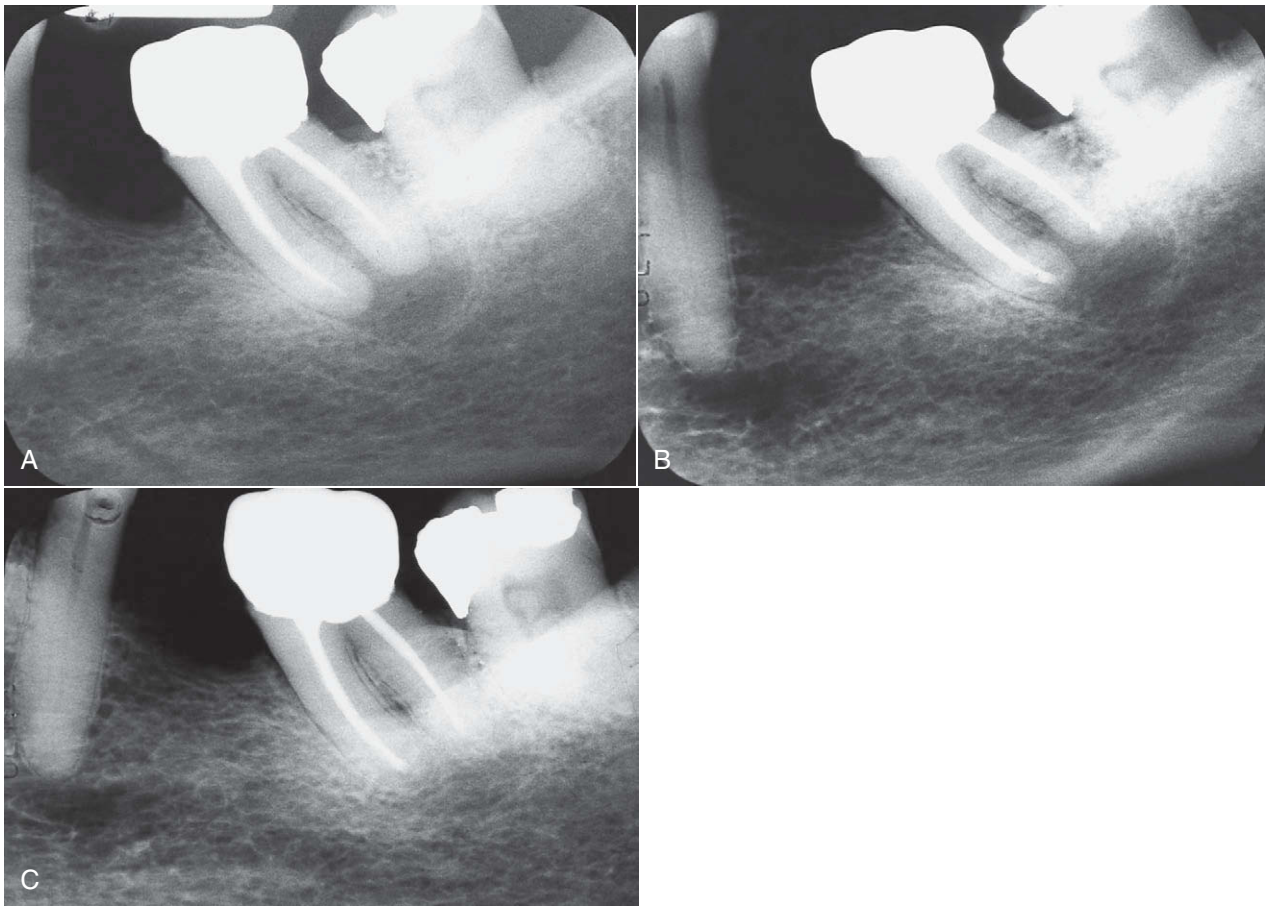


FIG. 24-43 A, Unsuccessful and symptomatic treatment on a mandibular second molar of a 78-year-old patient was surgically treated **(B)**, with less trauma than the extraction alternative. **C,** Complete healing at 1 year, apparently unaffected by age.

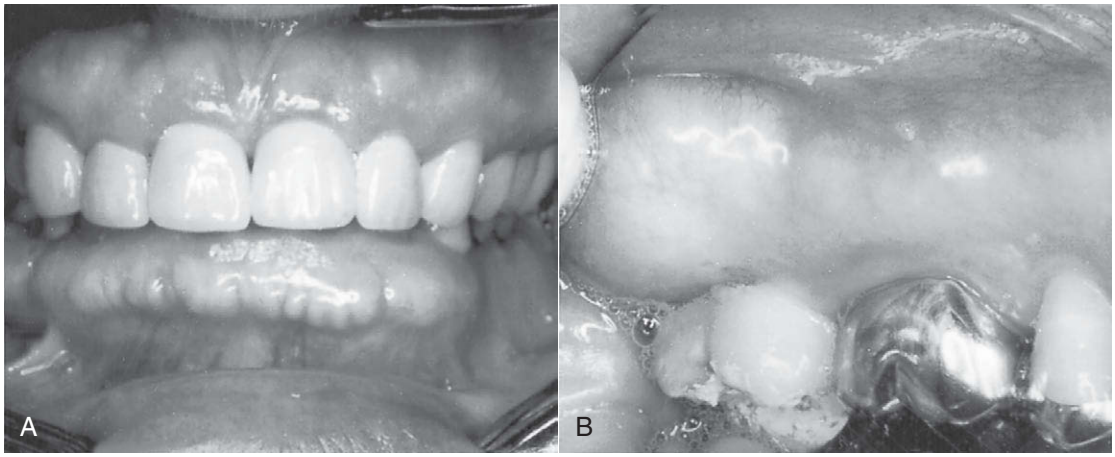


FIG. 24-44 Exostoses, as illustrated in this mandibular anterior tooth **(A)** and maxillary molar **(B)**, covered with thin tissue that can easily be torn during flap reflection, as well as the more obvious heavy bone and its effect on surgical access.

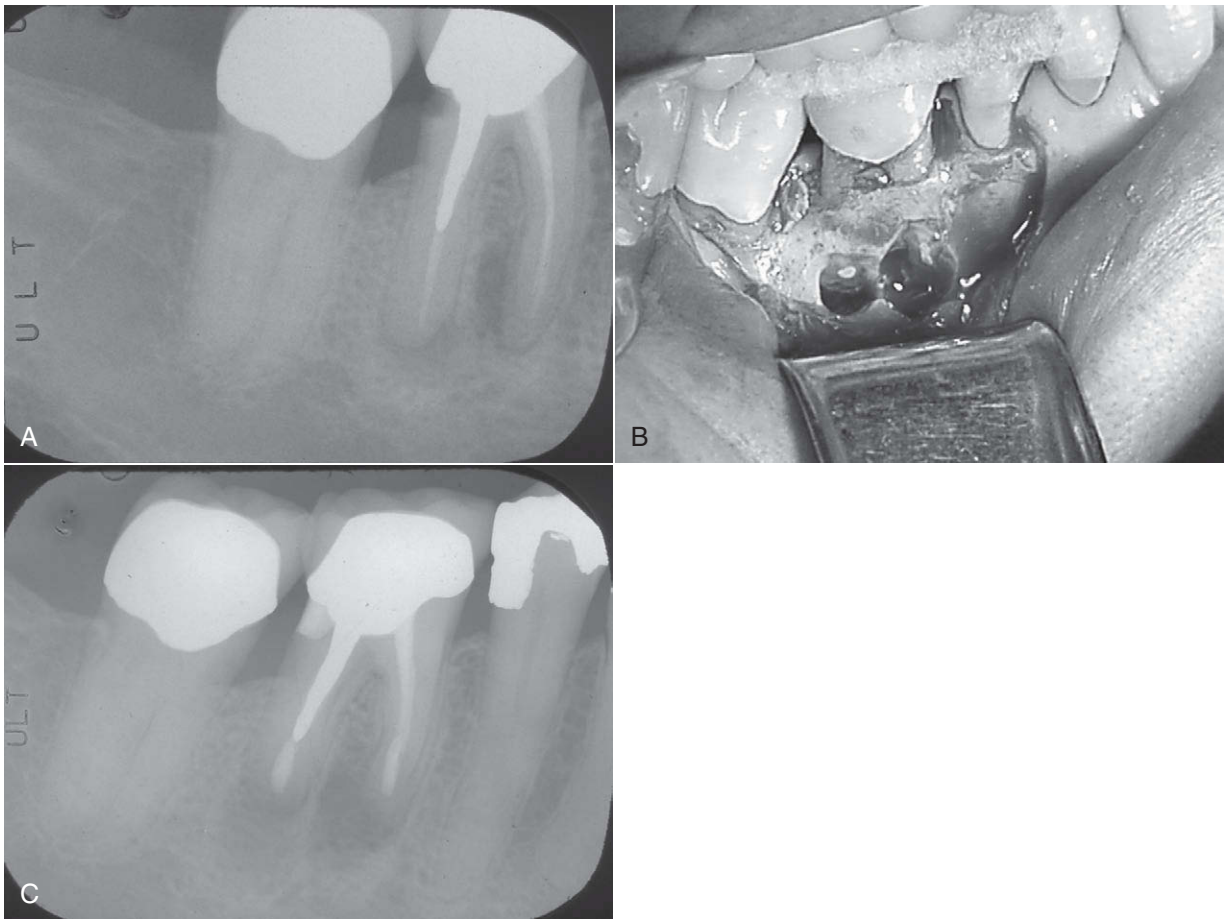


FIG. 24-45 A, Periradicular periodontitis and distal root caries evident in this 78-year-old patient. **B,** Apicoectomy, retrocavity preparation, and retrofilling, and **C,** cervical caries restoration were done for this lower molar tooth.



FIG. 24-46 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.



FIG. 24-47 This fracture of a cast post presents a very difficult challenge to remove.

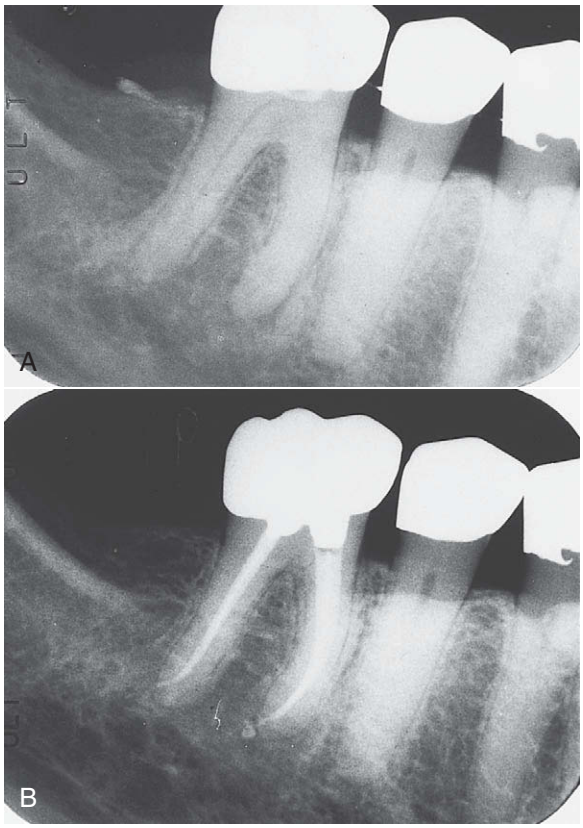


FIG. 24-48 A, Intact margins permitted the continued use of this crown. **B,** The access was restored with an amalgam-bond core.

of ecchymosis. Later, application of heat helps to 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. 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. 24-47). Posts are not usually needed when root canal treatment is performed through an existing crown that will continue to be used (Fig. 24-48). (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. 24-49 and 24-50). There can also be insufficient vertical and horizontal space when opposing or adjacent teeth are missing. Patient desires to save appliances can sometimes be fulfilled with creative attempts that may outlive them (Fig. 24-51).

In conclusion, 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 a part of its destiny.

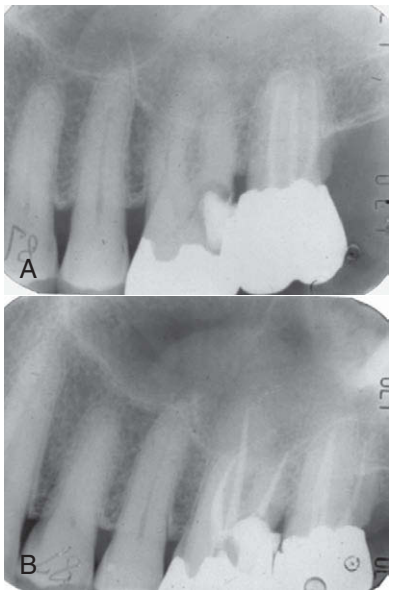


FIG. 24-49 A, This older woman insisted on saving her tooth in spite of extensive root caries on the distobuccal root. **B,** Root canal treatment was performed, and the root caries was removed and restored with Geristore.

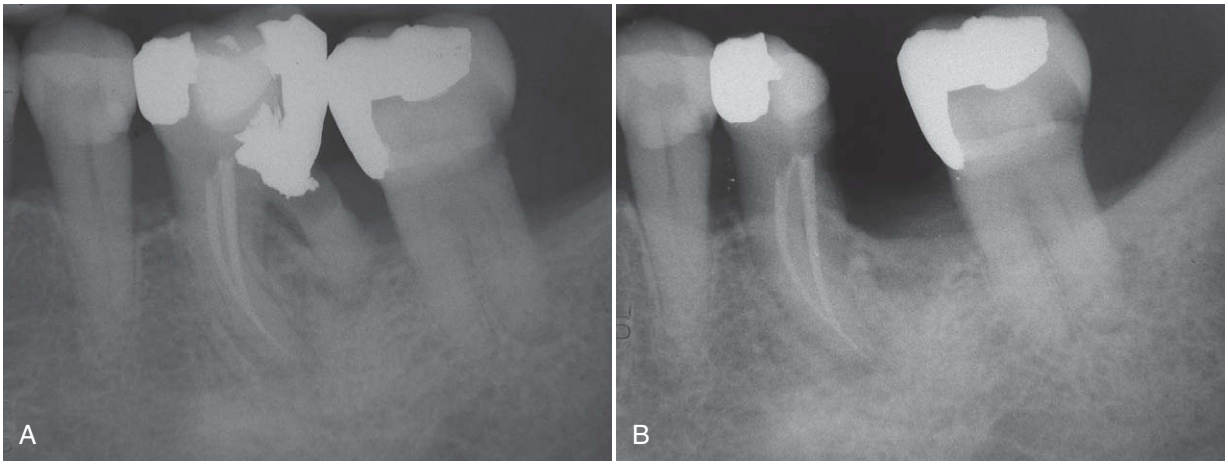


FIG. 24-50 A, Root caries and periodontal involvement of this mandibular molar tooth necessitated hemisection to retain the mesial root for restoration **(B)**.

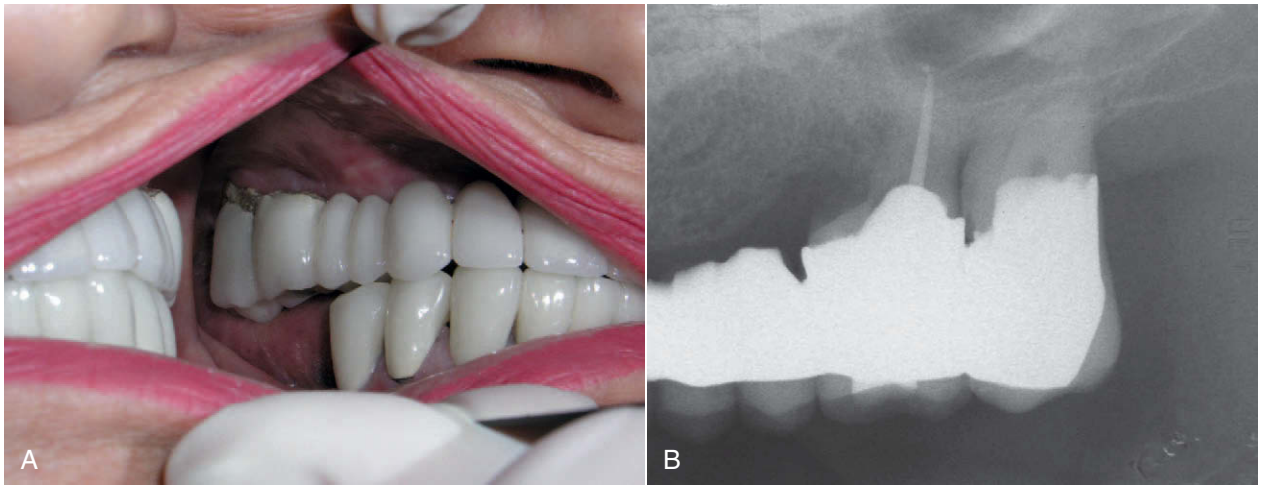


FIG. 24-51 A, Unrestorable caries contraindicated endodontic treatment on this molar abutment. **B,** Root amputation of buccal roots was performed to increase the lifespan of this bridge.

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CHAPTER
25

Nonsurgical Retreatment

ROBERT S. RODA and 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

Nonsurgical root canal therapy has become a routine procedure in modern dentistry. Recent technical and scientific advances in endodontics have resulted in the retention of millions of teeth that would otherwise be lost. Even as recent 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. Clinicians should be able to diagnose persistent endodontic disease and be aware of the options for treatment. If clinicians wish to approach treating these teeth, they should have the appropriate armamentarium and be capable of performing these very specialized techniques at the highest level (Fig. 25-1). Also, clinicians must always have a scientifically sound, evidence-based rationale for every treatment decision that is made so 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 where treatments performed at the highest level of clinical competence result in an undesirable outcome, and there are other instances where a procedure is performed well below a scientifically acceptable standard and yet provides long-term success.[179,180] We must begin to dissect the science from emotion and ego, and this separation may start with nomenclature. Friedman states 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."[218] 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[26]; with success rates varying between 86% and 98%,[54,55] it has proven to be a very reliable treatment option. Conversely, the incidence of posttreatment disease, although small, translates into a very 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. 25-2). These include iatrogenic procedural errors such as poor access cavity design, untreated canals (both major and accessory),[235] canals that are poorly cleaned and obturated,[34,96] complications of instrumentation (ledges, perforations, or separated instruments),[148] and overextensions of root filling materials.[141] Coronal leakage[114,127,172,182,207,219] has also been blamed for posttreatment disease, as has persistent intracanal and extracanal infection[143,191,203] and radicular cysts.[139] 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 discernable or



FIG. 25-1 Some of the armamentarium needed to perform retreatment at the highest level.

may ultimately never be known. The most important causative factors for the clinician, however, are those related to treatment planning and determining prognosis. To effectively plan treatment, the clinician may place the etiologic factors into four groups[148] (Fig. 25-3):

1. Persistent or reintroduced intraradicular microorganisms
2. Extraradicular infection
3. Foreign body reaction
4. True cysts

*1. **Persistent or reintroduced intraradicular microorganisms*** When the root canal space and dentinal tubules are contaminated with microorganisms or their byproducts, 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

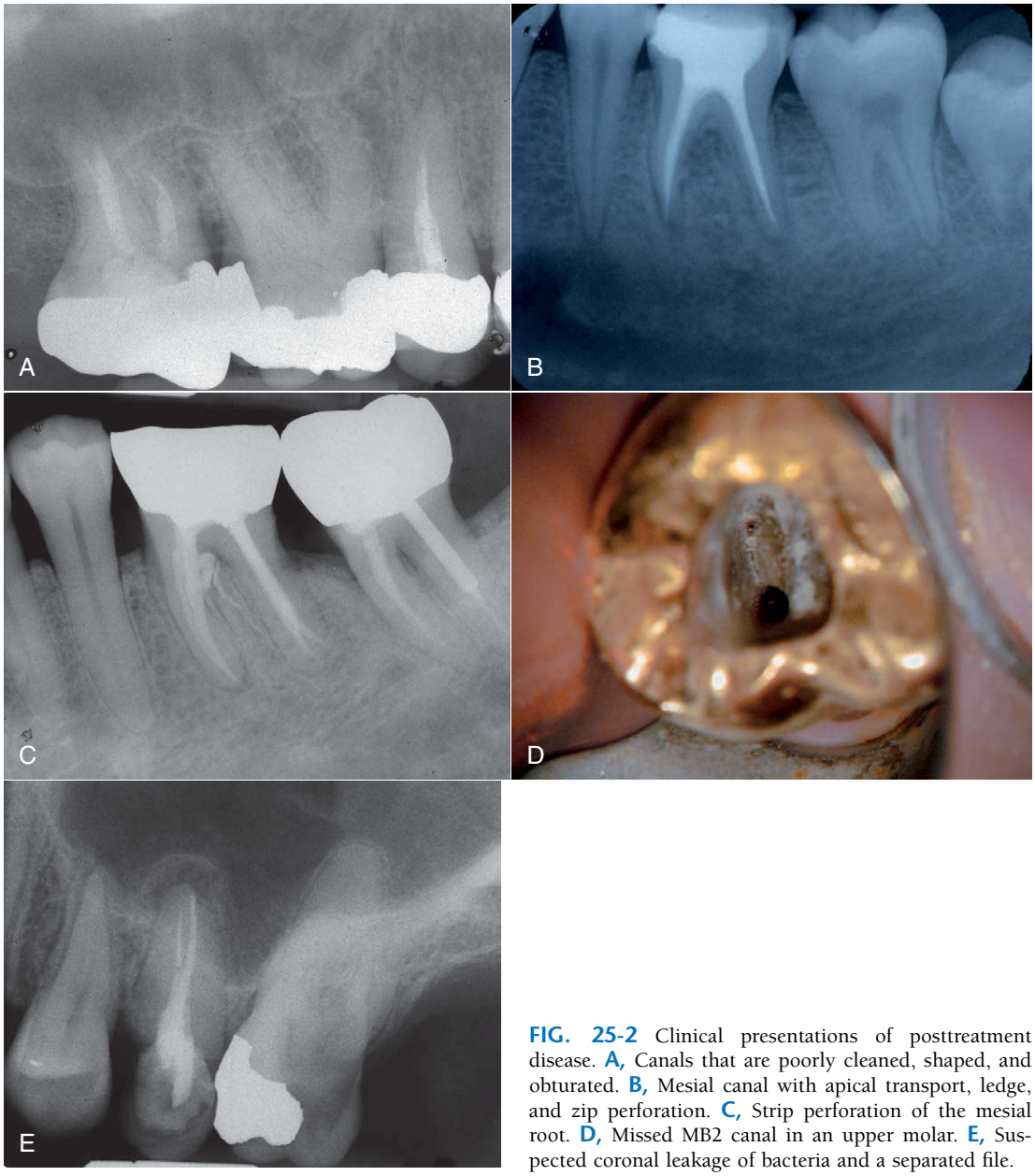


FIG. 25-2 Clinical presentations of posttreatment disease. **A,** Canals that are poorly cleaned, shaped, and obturated. **B,** Mesial canal with apical transport, ledge, and zip perforation. **C,** Strip perforation of the mesial root. **D,** Missed MB2 canal in an upper molar. **E,** Suspected coronal leakage of bacteria and a separated file.

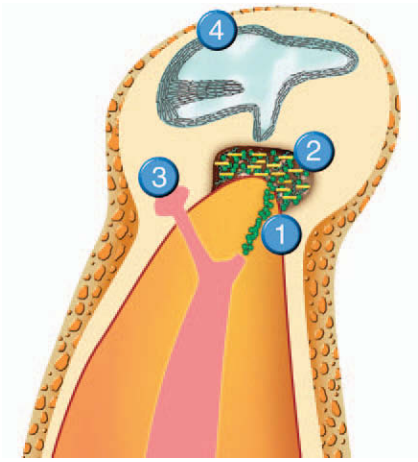


FIG. 25-3 The causes of posttreatment disease. *1,* Intraradicular microorganisms. *2,* Extraradicular infection. *3,* Foreign body reaction. *4,* True cysts. *(Modified from Sundqvist G, Figdor D: In Orstavik and Pitt-Ford* Essential Endodontology, *New York, 1998, Blackwell; diagram courtesy of DENTSPLY Tulsa Dental, Tulsa, OK.)*

endodontically diseased tooth can lead to posttreatment disease. If initial endodontic therapy does not render the canal 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, 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.[140] 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 that results in persistent disease but the inability to remove or entomb the microorganisms present that creates the pathologic state. Although infected root canals of endodontically untreated teeth generally contain a polymicrobial, predominantly anaerobic flora,[202] cultures of infected, previously root-filled teeth produce very few or even one single species. The infecting flora are predominantly gram positive and not anaerobic. A very commonly isolated species is *Enterococcus faecalis*,[60,158] which has been shown to be very resistant to canal disinfection regimes.[14,30] 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.[148] Though posttreatment disease has been primarily blamed on bacteria in the root canal system, fungi, notably *Candida albicans,* are found frequently in persistent endodontic infections and may be responsible for the recalcitrant lesion.[189]

2. 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,[187] extrusion of infected dentin chips,[84] or by contamination with overextended, infected endodontic instruments.[226] 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.[220] It has also been shown[143,191,203] that two species of microorganisms, *Actinomyces israelii* and *Propionibacterium propionicum* can exist in the periapical tissues and may prevent healing after root canal therapy.

3. Foreign body reaction Occasionally, persistent endodontic disease occurs in the absence of discernable 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[186] and cellulose fibers from paper points.[108] 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.[148,190] 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, healing in the presence of overextended filling materials can still occur.[59,119,148]

4. True cysts Cysts form in the periradicular tissues when retained embryonic epithelium begins to proliferate due to the presence of chronic inflammation. The epithelial cell rests of Malassez are the source of the epithelium, and cyst formation may be an attempt to help separate the inflammatory stimulus from the surrounding bone.[157] The incidence of periapical cysts has been reported to be 15% to 42% of all periapical lesions,[139,196] and determining whether periapical radiolucency is a cyst or the more common periapical granuloma cannot be done radiographically.[20] 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. With pocket cysts, the lumen is open to the root canal of the affected tooth. True cysts, owing to their self-sustaining nature, probably do not heal following nonsurgical endodontic therapy[96,142] and usually require surgical enucleation (Fig. 25-4).

When a patient presents with posttreatment disease, clinical decision making depends upon determining the cause of the persistent disease and then making an assessment of how best to treat the pathologic condition. In the following section, the reader will be presented with a rationale and methods for performing endodontic diagnosis that will allow for 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".[7] The proper diagnosis is probably the most important portion of any endodontic procedure. This is not as bold 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. 25-5). To make a correct diagnosis, clinicians must rule out non-odontogenic etiology, perform all appropriate tests, properly interpret the patient's responses to

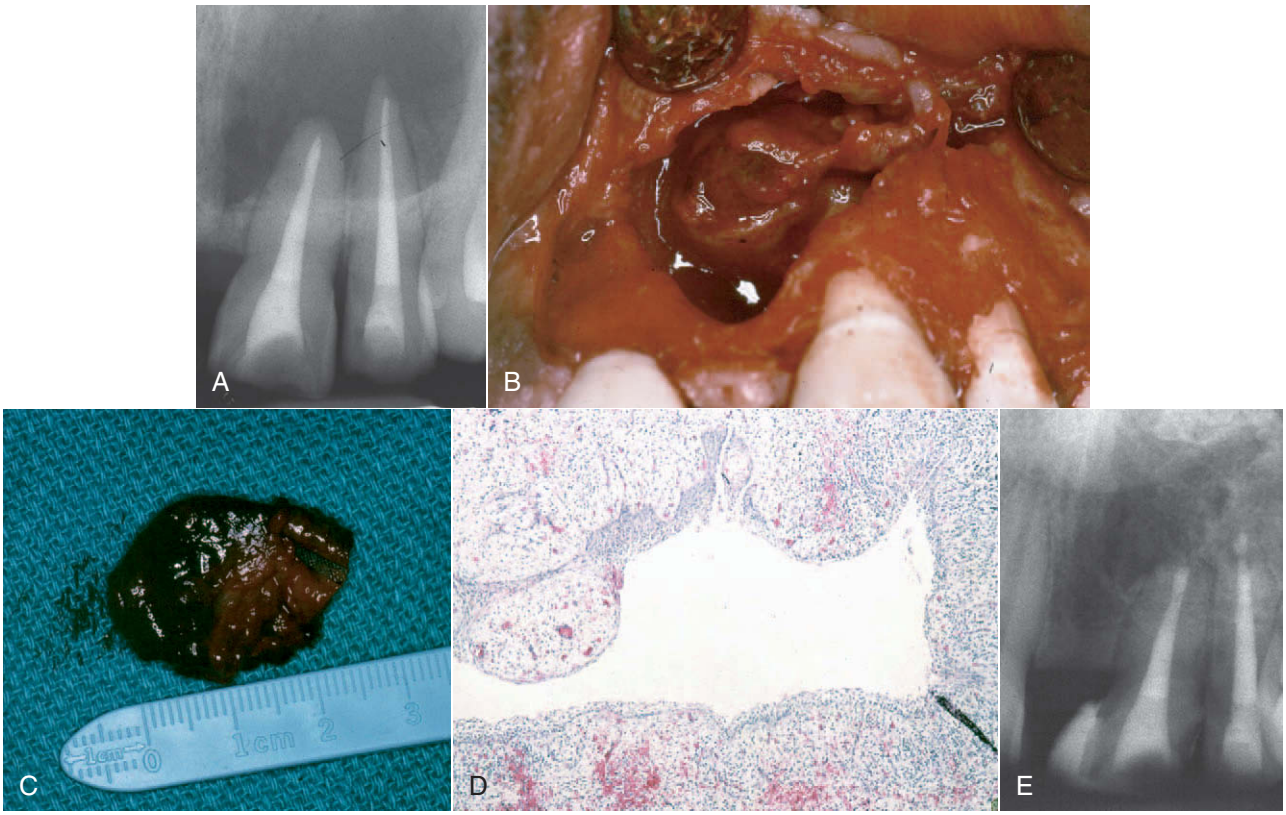


FIG. 25-4 A, Apparently good nonsurgical retreatment with large persistent lesion. **B,** Surgical exposure of apical lesion in situ. **C,** Large lesion removed in toto. **D,** Histopathologic section confirming cystic nature of the lesion. **E,** Four-year postoperative film showing apical scar formation due to the large size of the lesion. The teeth were asymptomatic and in function.

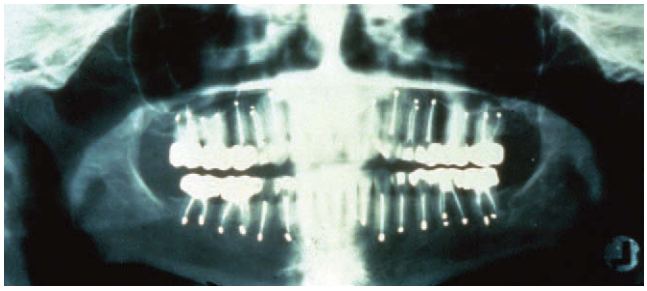


FIG. 25-5 This patient was misdiagnosed for years and underwent unnecessary endodontic therapy. The actual cause of the patient's complaint was nondental pain. *(Courtesy Dr. Ramesh Kuba.)*

these tests, derive 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 diagnosis are necessary. In cases of persistent disease, the diagnosis may not be straightforward; the clinician may be dealing with partially treated pulp canals, missed canals, or many other types of problems associated with the previous treatment. These must be included in the diagnostic description for each case.

Endodontic diagnosis has been 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. The canals can be assumed to be contaminated regardless of how esthetically 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 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 their 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."

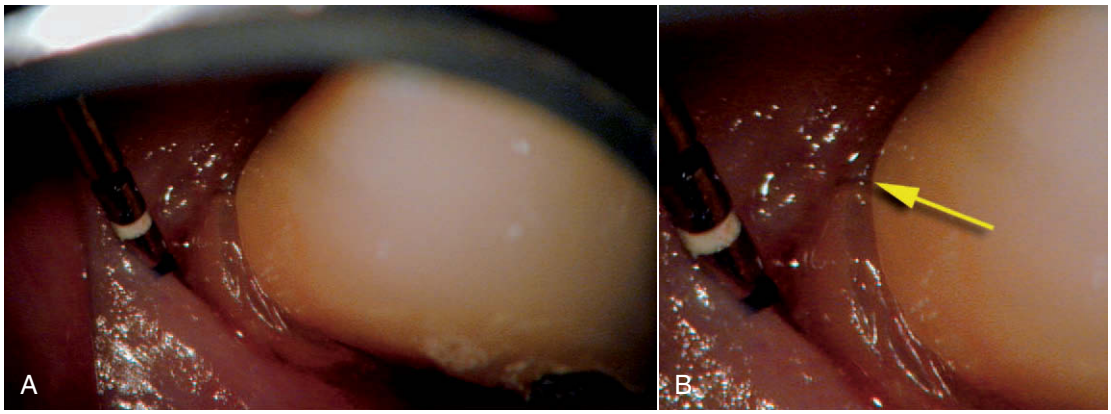


FIG. 25-6 A, Buccal aspect of a premolar with posttreatment disease. **B,** Higher magnification reveals a vertical fracture *(Courtesy Dr. Jay Rosenthal.)*

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, including clinical and radiographic examinations. 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 very fine fractures on root surfaces (Fig. 25-6). Exposed dentin from recession and narrow-based probing defects may be the result of an endodontic infection draining through the sulcus; however, they are sometimes indicative of vertical root fracture (for further information, please refer to Chapter 1). 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[75]; this condition has been associated with posttreatment disease.[97]

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.[49] In cases with previous endodontic therapy, radiographs are very 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,[209] resorption, and canal anatomy. If films are used, 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 rule" (Fig. 25-7). Bitewing radiographs are useful in determining periodontal bone height and to look for caries or fractures. All sinus tracts should be traced with a cone of gutta-percha followed by a radiograph to localize their origin.[96]

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.[218] These will allow the diagnostician to begin developing a sense of the status of the periradicular tissues. Such tests are of great importance any time an endodontic diagnosis is needed. However, they are of even greater importance when evaluating teeth that have been previously treated with endodontic therapy, owing 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,[75] as can periodontal disease.[218]

Pulp vitality tests are often of little value when examining teeth with previous endodontic therapy, but if the patient's chief complaint reveals the need for these tests, they must be performed. 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.[75] 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, but a positive response usually means there is responsive pulp tissue remaining in the tooth.[75] Care is always warranted in interpreting pulp test results, however, since false-positive and false-negative results may occur.[180] 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 less 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.

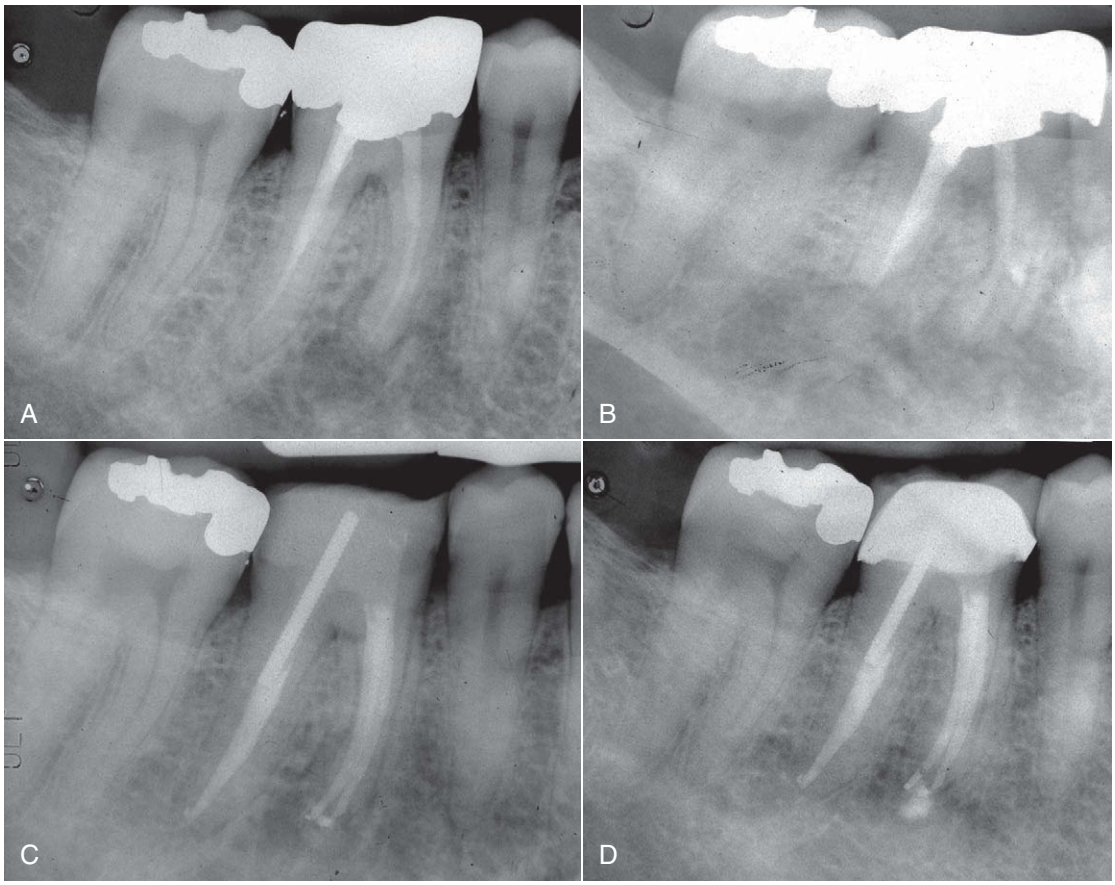


FIG. 25-7 A, Posttreatment disease. Previous endodontic therapy performed 3 years previously. **B,** Distal angle radiograph reveals asymmetry, indicating the presence of an untreated mesiobuccal canal. **C,** Immediate postobturation film showing treated MB canal. **D,** Fourteen-month postoperative view. The patient was asymptomatic.

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 upon his or her own perceptions of the options, not by the clinician's opinion about what is "best" for the patient. The reader is reminded, however, that if the cause of the posttreatment condition remains unknown despite a thorough diagnostic workup, 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. Because of the interdisciplinary nature of modern dentistry, consultation with other clinicians who are treating the patient often 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:

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. 25-8). This approach is sometimes a useful, short-term option if the etiology of the 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 less than desirable as a 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 them from the practice is one that each clinician must make based upon the clinician's experience, judgment, and knowledge of the patient.

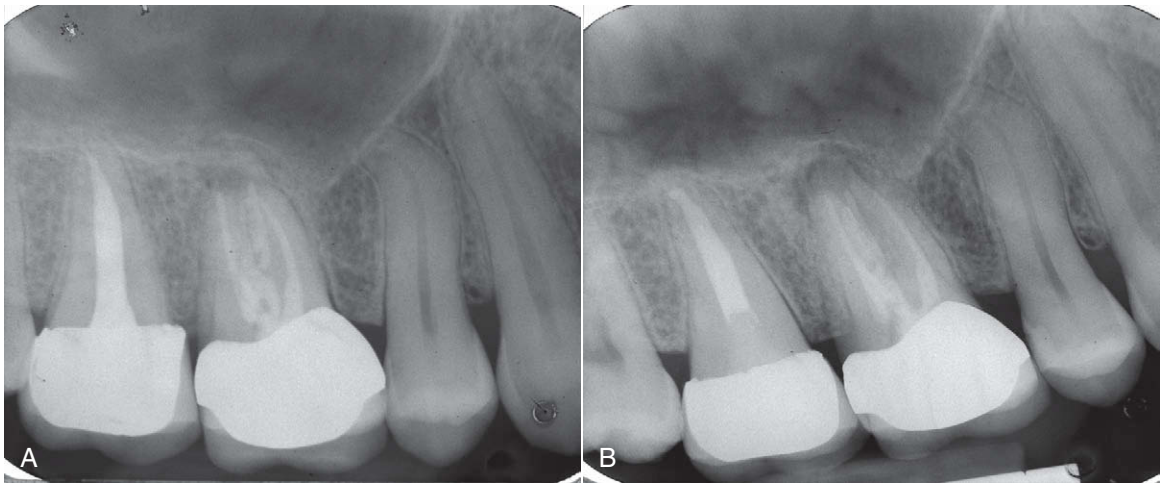


FIG. 25-8 A, Radiograph indicating presence of asymptomatic persistent apical periodontitis 7 years after initial treatment. The patient elected no treatment at that time. **B,** Six-year follow-up. Lesion has enlarged, and the tooth has become symptomatic.

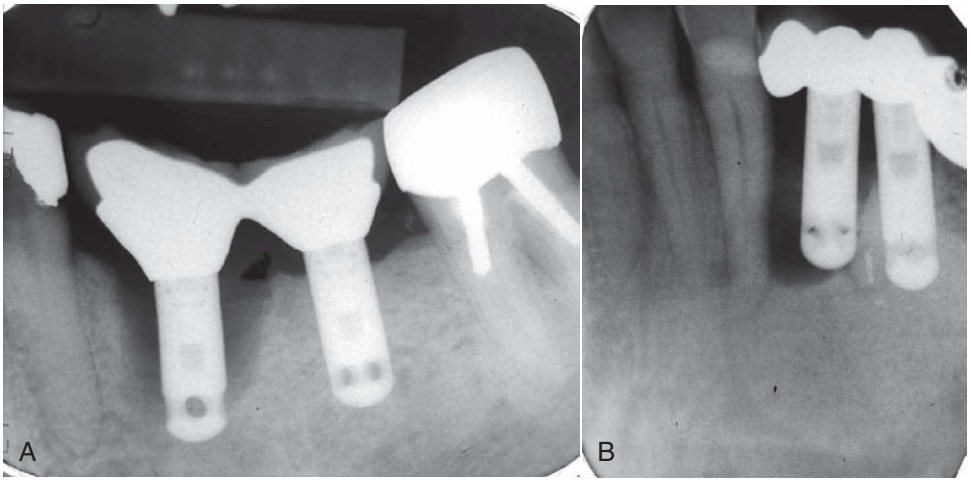


FIG. 25-9 A, Classic periimplantitis. The implant needed removal. **B,** Another periimplantitis. Note the endodontically treated root tip apical to the implant that may have contributed to the persistent disease. Perhaps apicoectomy should have been performed.

Extraction of the tooth is usually considered a viable option. Recent 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 site preparation prior to the implant, which can add months more. Despite published long-term success rates for dental implants,[2] post-implant disease does occur[2,71,72] (Fig. 25-9) and can leave the patient with very 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 esthetics can be inferior to that of natural teeth in the esthetic zone of the mouth, and there are patients that are just not candidates for implant procedures.[2] Fixed partial dentures are another replacement alternative with a very long history of successful use, but there are negative outcomes possible also. Most concerning to the endodontist is the likelihood that retainer fabrication procedures will result in endodontic disease of the abutment teeth[79] that may potentially occur at a rate of up to 10%[130,224] (Fig. 25-10). 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 (teeth, gingiva, and mucosa) if not meticulously cleaned. Owing 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

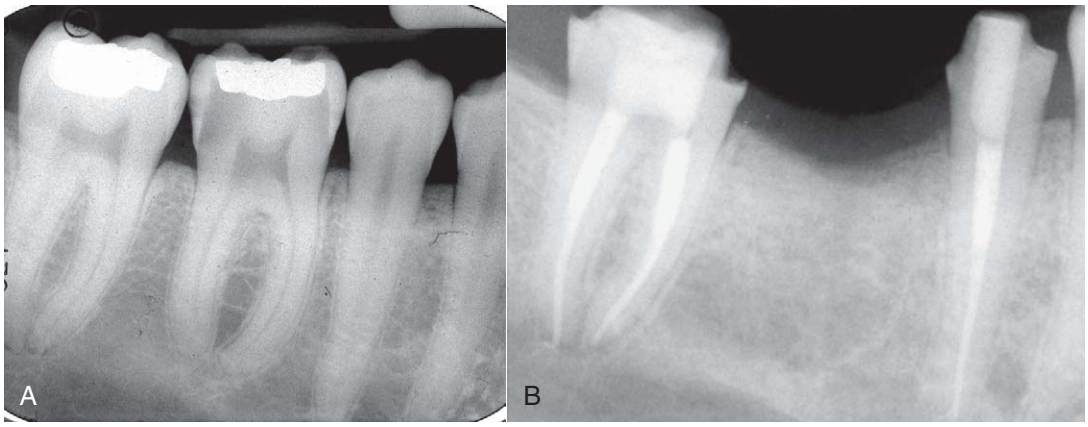


FIG. 25-10 A, Preoperative film showing deep caries approaching the pulp. The patient's holistic dentist advised extraction and replacement rather than endodontic therapy to retain the tooth. **B,** Fixed partial denture fabrication procedures resulted in irreversible pulpitis on both abutments, requiring endodontic therapy.

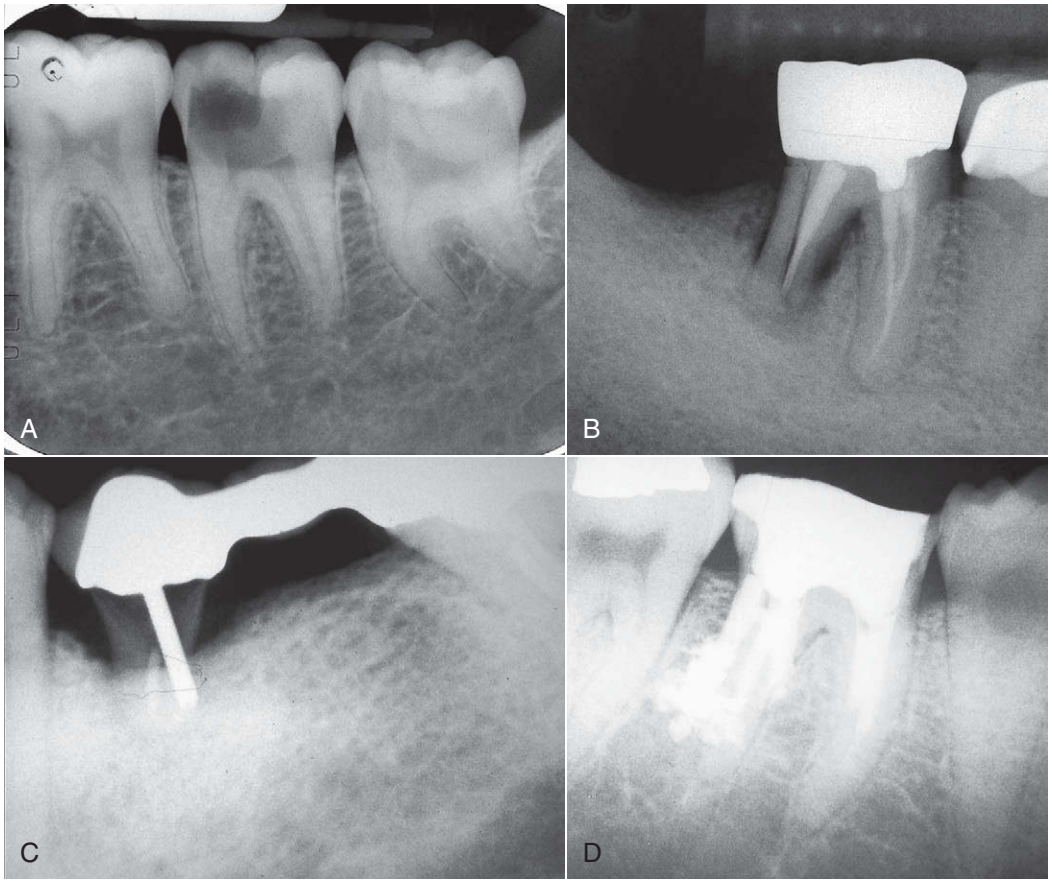


FIG. 25-11 A, Deep caries approaching the furcation and the biologic width. Necessary crown-lengthening surgery would open the furcation to bacterial invasion and persistent periodontal disease. **B,** Distal root vertical fracture resulting in a split root. **C,** Severe caries and post perforation. Inadequate root structure remaining to restore. **D,** Multiple distal root perforations so weaken the root as to make it unrestorable. NOTE: Cases A, B, and D could have resective endodontic surgery such as hemisection, but long-term prognosis is poorer than for extraction and replacement.[28,111]

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 unrestorable, retaining the tooth with endodontic procedures is better for the patient.

Various situations may render a tooth unrestorable (Fig. 25-11), but the line of demarcation between restorable and unrestorable is a movable one, depending on who is evaluating the tooth. There are several widely agreed upon situations that render a tooth unrestorable. 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 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 an irretrievable 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. 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).[76,147] A situation sometimes 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.[5] 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, several choices are available. If the cause of the posttreatment disease is persistent extraradicular infection, foreign body reaction, or the presence of a true cyst, nonsurgical root canal therapy has little likelihood of allowing healing to occur, and surgical methods should be employed.[148] 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.

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% and 98%,[55] but with apical surgery alone, only 59% heal completely.[148] When apical surgery is preceded by orthograde retreatment, however, the incidence of complete healing rises to 80%.[148] In general, nonsurgical retreatment will be the preferred choice, since 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: 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.[124,218]

There are many factors to consider when deciding whether to retreat surgically or nonsurgically. 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 they do not, then apical surgery alone may be indicated, although the patient must be made aware of the potentially compromised nature of the treatment. The clinician must be armed with the best equipment and knowledge available, and critical self-evaluation should allow the experienced clinician 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.[55,148] If there has been a previous procedural complication, such as a ledge that cannot be bypassed or a separated instrument that cannot be removed, surgery may become a better option. Most times, it is still prudent to attempt the retreatment, since 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 earlier. The clinician must be careful not to worsen the situation by overly vigorous attempts to treat the previous complication; 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, since many surgical failures are due to poorly cleaned and filled canal systems[163] (Fig. 25-12). In many instances, performing the surgery a second time can be avoided altogether. If there is evidence of root fracture (narrow-based probing defect and/or a J-shaped radiolucency encompassing the root apex and progressing in a coronal direction,[208] (Fig. 25-13) nonsurgical retreatment would be unlikely to improve that situation. Apical exploratory surgery may be necessary that could result in root resection or even extraction of the tooth.

Each case should be approached as a unique set of considerations that must be reviewed and interpreted prior to selecting a treatment method. Once the selected option is undertaken, the prudent clinician is always watchful because additional pieces of information can be discovered during treatment that may modify previous decisions.

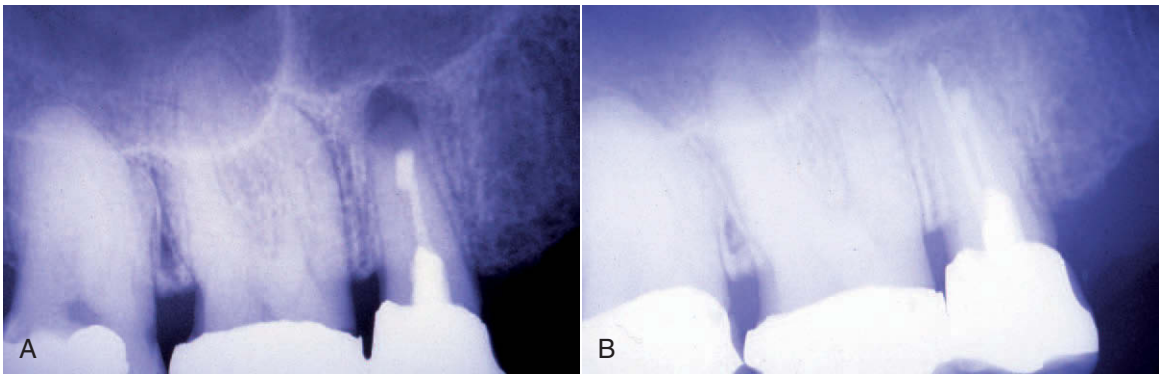


FIG. 25-12 A, Posttreatment disease following apical surgery. Off-center positioning of the root filling indicates the presence of a second untreated canal. **B,** One year following nonsurgical retreatment showing complete healing.

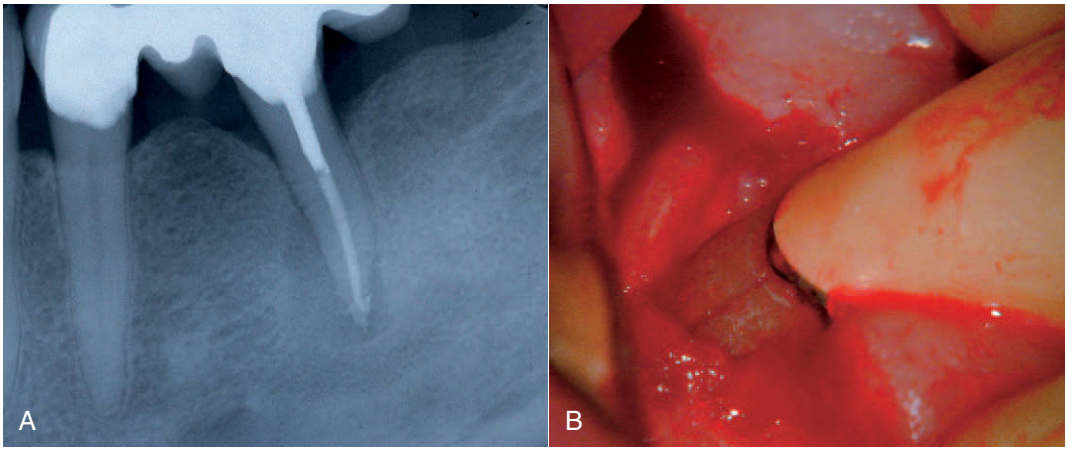


FIG. 25-13 A, The J-shaped radiolucency possibly indicates root fracture. **B,** Exploratory surgery confirms presence of vertical root fracture.

NONSURGICAL ENDODONTIC RETREATMENT

The primary difference between nonsurgical management of primary endodontic disease versus 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 and shaping procedures be instituted that will allow for effective obturation and case completion. The remainder of this chapter will be devoted to these topics in the order they generally present themselves to the clinician treating the previously root-filled tooth.

Coronal Access Cavity Preparation

Retreatment access has been called *coronal disassembly*[163,164] 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 to 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, since 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 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. 25-14). 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 esthetics will be minimally changed. Even if the crown requires replacement, the clinician may elect to retain it during the endodontic retreatment to

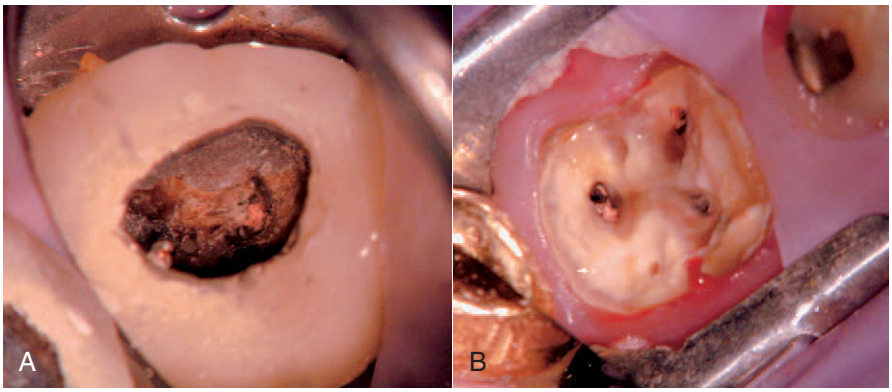


FIG. 25-14 A, Limited visibility and access with crown present. **B,** Enhanced visibility and access with crown off. Note the isolation achieved using a Silker-Glickman clamp and sealing putty.

allow for better isolation with the rubber dam. Unfortunately, retreatment may be more difficult with the crown in place, because 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.[137] 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 (PFM) 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, Tulsa, OK) or the Great White bur (SS White, Burs Inc, Lakewood, 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 owing 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.[80] Copious coolant water spray and the use of diamond burs are recommended during access through porcelain to minimize occurrence of this event.[206] In a novel approach, researchers[165] recently showed that compared to 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, the clinician can take the easiest road and simply cut it off. If the crown is to be preserved, a more conservative approach must be used. Two considerations that 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 is even more of a concern lately because of the increasing popularity of tooth-colored restorations, mainly different types of porcelain or PFM restorations, which are being bonded to the tooth. These restorations are less likely to withstand the stresses of removal than those composed completely of metal, and restorations that are bonded are much more difficult to remove because of 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.

There have been many devices 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. 25-15), which uses 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 (Miltex, York, PA), the Trial Crown Remover (Hu-Friedy, Chicago, IL), and the Trident Crown Placer/Remover (CK Dental Industries, 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 Dental Products, Johnson City, TN) works in this fashion and can be effective

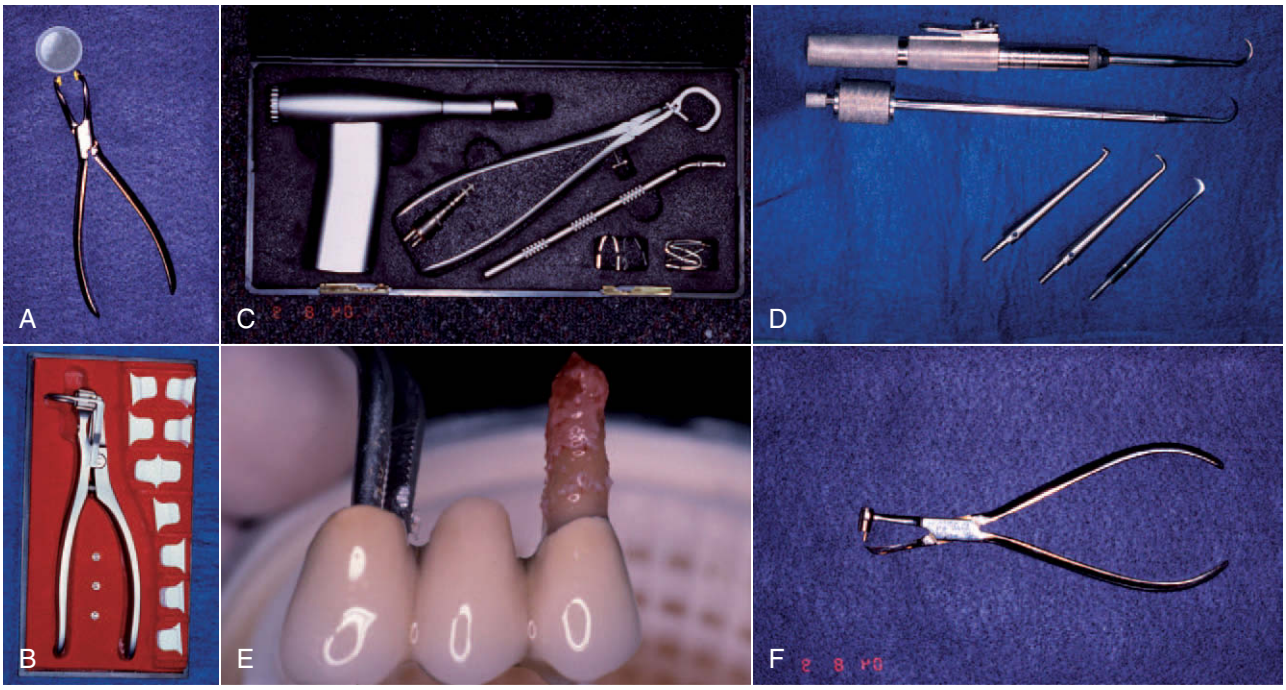


FIG. 25-15 A, KY Pliers (GC America, Alsip, IL) and supplied emery powder. **B,** Roydent Bridge Remover (Roydent Dental Products, Johnson City, TN). **C,** CoronaFlex Kit (KaVo Dental Corp, Lake Zurich, IL). **D,** *Top,* Crown-A-Matic (Peerless International, N. Easton, MA); *Bottom,* Morrell Crown Remover (Henry Schein Inc, Melville, NY) with interchangeable tips. **E,** Tooth inadvertently extracted using a crown/bridge remover. Endodontic therapy was performed, and the tooth was replanted, a procedure known as *unintentional replantation*. **F,** Kline Crown Remover (Brasseler USA, Savannah, GA).

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, S. Easton, MA) delivers a shock impulse to loosen the crown. As mentioned before, crown margin damage may result as can inadvertent extraction of the tooth if the periodontium is compromised[159] (see Fig. 25-15, *E*). A different approach to conservative crown removal involves drilling a small hole through the crown in order 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, so their use on anterior teeth and 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. 25-16). 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 the mouth, generating enough force to pull the crown off. Care must be taken by the clinician to avoid using this technique when the opposing tooth is extensively restored, since 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.

Post Removal

Once the access is prepared, it is very common to encounter a post, inasmuch as posts are frequently used in the restoration of endodontically treated teeth. There are many different types of posts the clinician may encounter during retreatment (Fig. 25-17). These can be classified into two categories: prefabricated posts and custom-cast posts. Historically, cast posts were more commonly used than prefabricated posts, but over the past 2 decades, cast posts have become much less popular.[177] 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

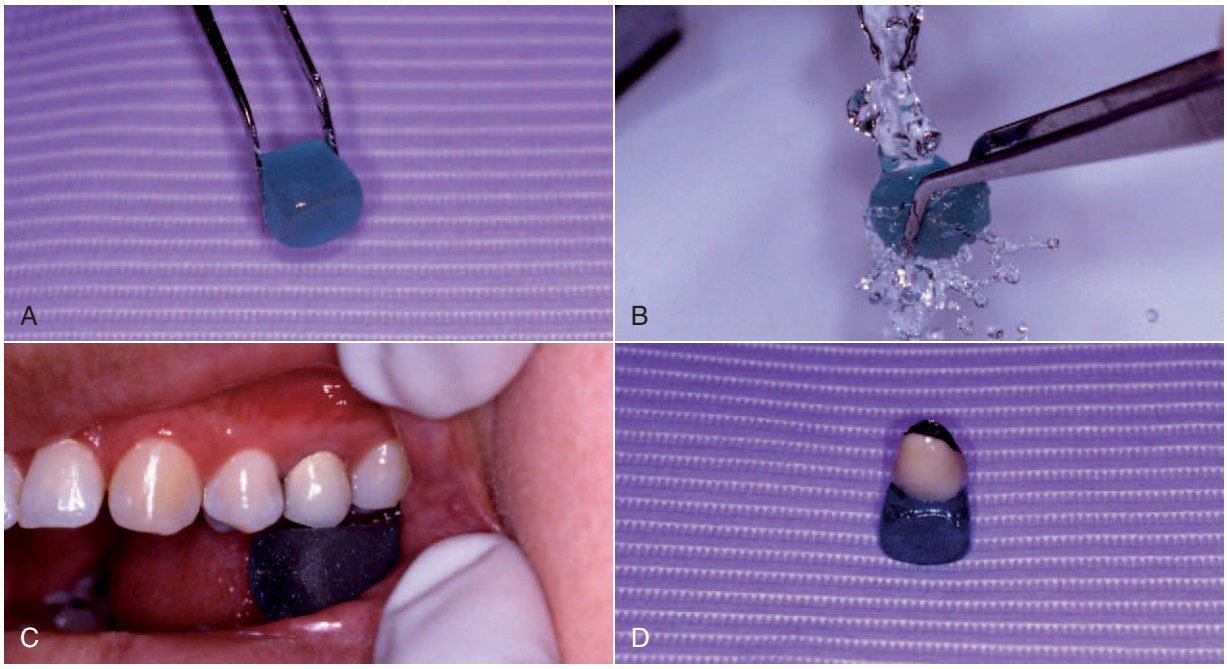


FIG. 25-16 A, Richwil Crown and Bridge Remover (Almore International Inc, Portland, OR). **B,** Using hot water to soften the material. **C,** Remover is placed on the restoration to be removed, and patient bites into the material. **D,** The removed crown adhering to the material.

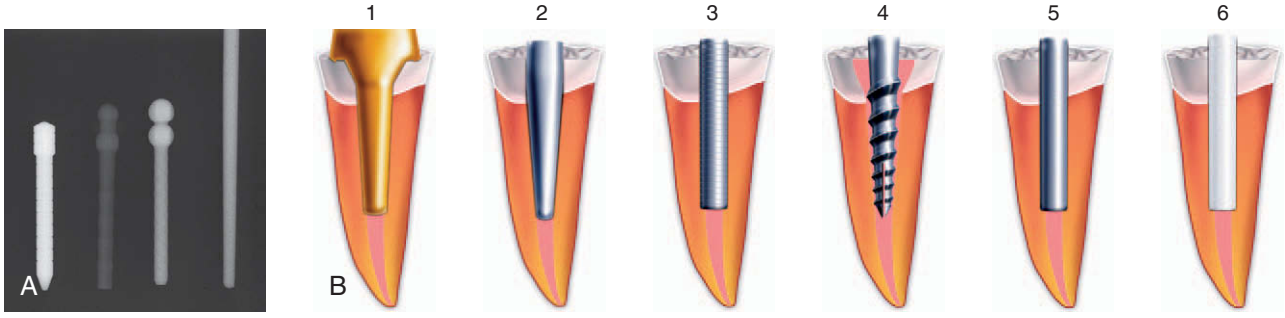


FIG. 25-17 A, Relative radiopacities of post materials *(left to right)*: stainless steel, fiber post, titanium post, gutta-percha. **B,** Diagrammatic representation of post types: *1,* custom cast; *2,* tapered; *3,* parallel; *4,* active; *5,* passive/metal; *6,* passive/nonmetal. *(Diagrams Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

acid-etched groups. There are also many materials that have been used to fabricate posts: 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 non-precious metal alloys. These posts will also come in a variety of shapes and configurations, since they are custom manufactured for each root in which they are placed. Most of these will 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 Tulsa Dental, Tulsa, OK), use acid-etched metal posts that are bonded into the canal with cements such as Panavia (Kuraray America, Houston, TX) or C&B Metabond (Parkell Inc, Edgewood, Farmingdale, NY). Removal of these posts is extremely difficult and occasionally impossible regardless of which technique is used.[70] A study[62] has shown that heat generation with ultrasonic vibration may help to decrease retention of resin-cemented posts, but concern for heat-generated periodontal ligament damage may preclude this technique.[177]

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, since the clinician will have more techniques and instruments available to use.[1] Also, the more anterior the tooth is, the less the opposing occlusion will interfere with post removal.

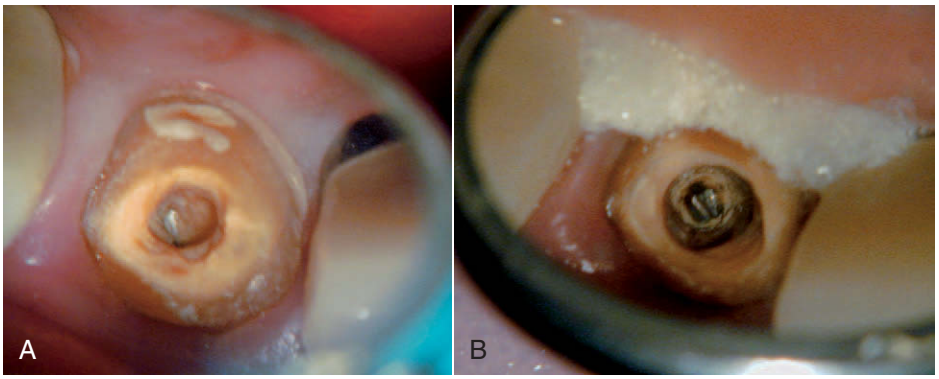


FIG. 25-18 A, Broken post (incisal view before excavation). **B,** Root has been so thinned and weakened by excavation procedures, restorability is questionable.

Post Removal Techniques

After the initial access and the post to be removed has been located, the clinician is faced with the decision of how to remove it. There have been many techniques 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. 25-18).

The first step in post removal is to expose it properly by removal of 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 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 helped 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[16,29,103]—although the effects of ultrasonic vibration may be minimal in reducing retention of well-fitted, long, large-diameter titanium posts.[17] 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, a study[83] failed to duplicate this effect. Nonetheless, care should be taken not to push the ultrasonic tip against the post with too much force, because 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 and will aid in the reduction of post retention without unduly weakening the root. If the root is thin 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, they are also more prone to breakage. At this point, the ultrasonic handpiece should be used with copious air/water spray as a coolant. Owing 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 to clean not only the area of debris but also reduce the temperature produced that could potentially cause damage to the periradicular tissues.[177] 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; this will help dissolve the cement around the post. Using a solvent in conjunction with removal of cemented obstructions may prove beneficial, since the ultrasonic energy produced will set up shock waves in the solvent and make it penetrate deeper into the canal space, exerting a faster solvent action on the cement.[67]

Using an ultrasonic instrument in this fashion is not simply helpful in reducing post retention, it 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. 25-19). 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. Many of the instruments to be discussed involve using a trephine bur to shape the coronal end of the post, and ultrasonic exposure will facilitate this process. Another instrument to consider for exposing and loosening a post is the Roto-Pro bur

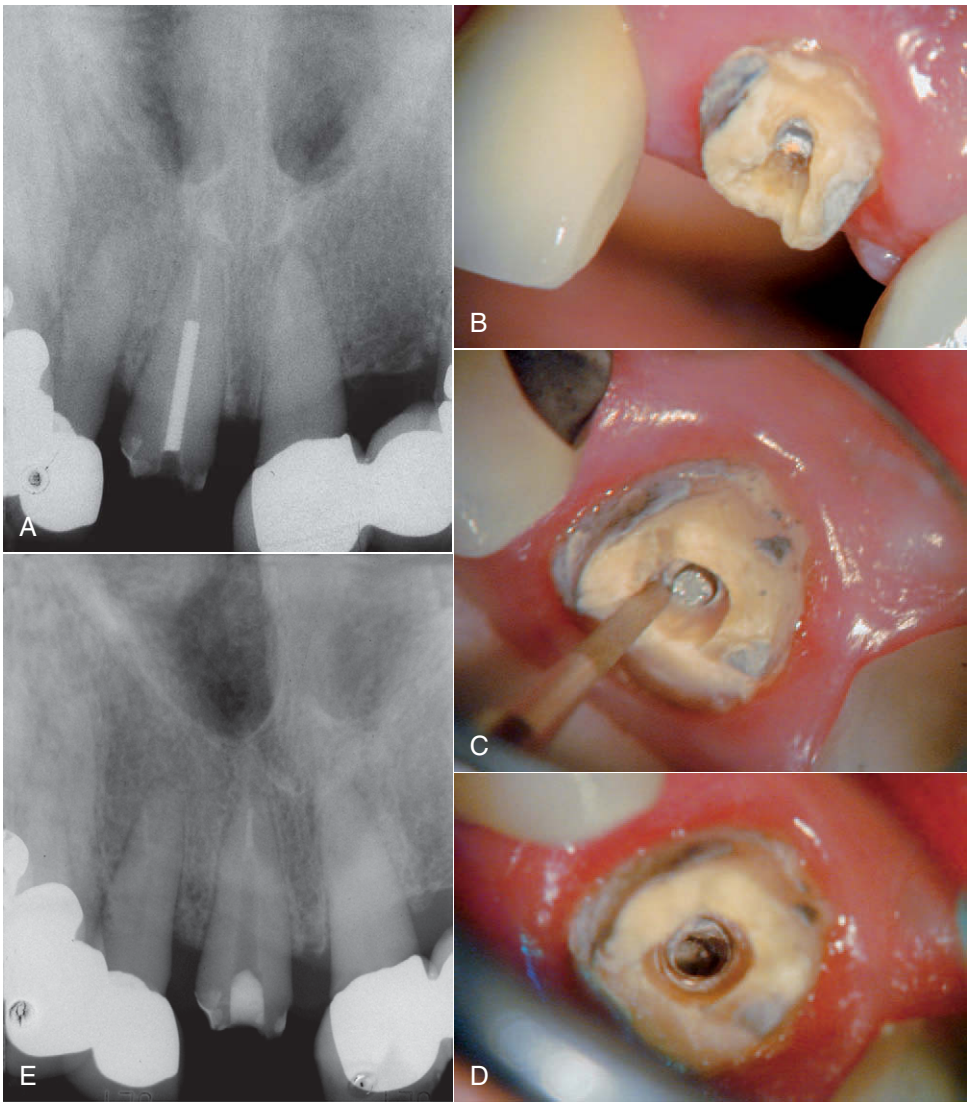


FIG. 25-19 A, Radiograph of fractured post. **B,** Fractured post, labial view. **C,** Ultrasonic troughing. **D,** Post removed by ultrasonic alone. **E,** Check film confirming complete post removal.

(Ellman International, Oceanside, NY) (Fig. 25-20). There are three shapes available, all of which are six-sided, non-cutting tapered burs that are used in a high-speed handpiece around the circumference of the post. The vibrations created when the non-cutting 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) that is a very effective instrument for removing parallel or tapered, nonactive preformed posts.[125,167] 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. 25-21) 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.[32]

The Thomas Screw Post Removal Kit (Thomas extracteur de pivots, FFDM-Pneumat) (Fig. 25-22) 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, but the extraction mandrels

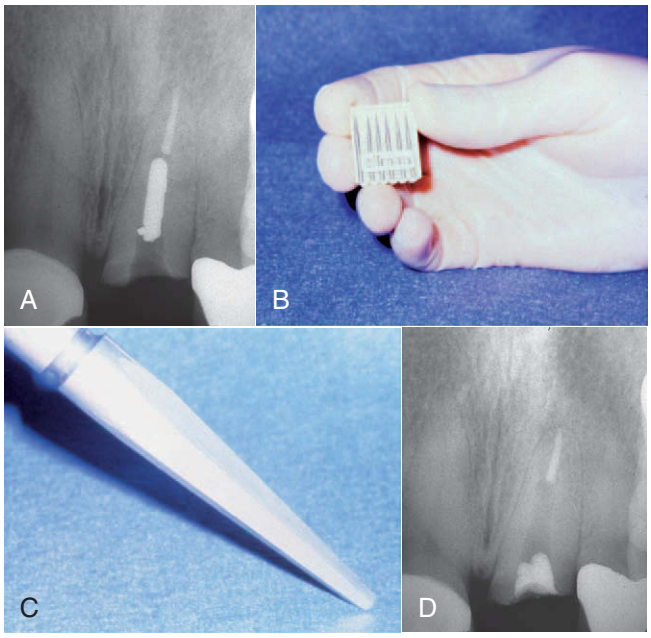


FIG. 25-20 A, Radiograph of fractured post. **B,** Roto-Pro Kit (Ellman International, Oceanside, NY). **C,** Roto-Pro Bur. **D,** Post removed by vibration of the instrument alone.

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.

The Ruddle Post Removal System (Sybron SybronEndo, Orange, CA)[163] (Fig. 25-23) 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 not only useful in the removal of parallel or tapered passive types of posts but also in 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. This is the smallest of the kits that work using a pulling action, which may help with cases where access is difficult. However, this kit does have one disadvantage: a smaller variety of trephine burs and extraction mandrels than 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)[200] (Fig. 25-24). This device works in a similar manner as some of the others, except 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. Since 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.[200] Care must be taken to align the pulling forces of this instrument with the long axis of the root to prevent fracture[32]; 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.[31] However, in a survey of the American Association of Endodontists, it was one of the least used techniques.[201] Clearly, techniques that are common in one country are not always that common in another.

The recent increased popularity of cosmetic dentistry has created an impetus towards 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. The use of a high-speed bur to channel down through the post may result in a high rate of root perforation.[154,177] The use of the Largo Bur (DENTSPLY Limited, Surrey, UK)[64] and the Peeso drill[154] to remove these posts has been advocated, and most of the post manufacturers have removal burs in the kit.[40] These manufacturers' removal kits have been shown to be more efficient at removing their own fiber posts than the use of diamond burrs and ultrasonics.[120] In addition, a new bur, the GyroTip (MTI Precision Products, Lakewood, NJ), has been designed for the specific purpose of removing fiber-reinforced composite posts (Fig. 25-25). 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 and/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.[177]

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. 25-21 Gonon Post Removal Technique. **A,** Fractured post in a lower incisor. **B,** Tooth isolated with a rubber dam. **C,** Gonon Kit (Dent Corp, White Plains, NY). **D,** Ultrasonic exposure of the post. **E,** Domer bur creating a shape 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.

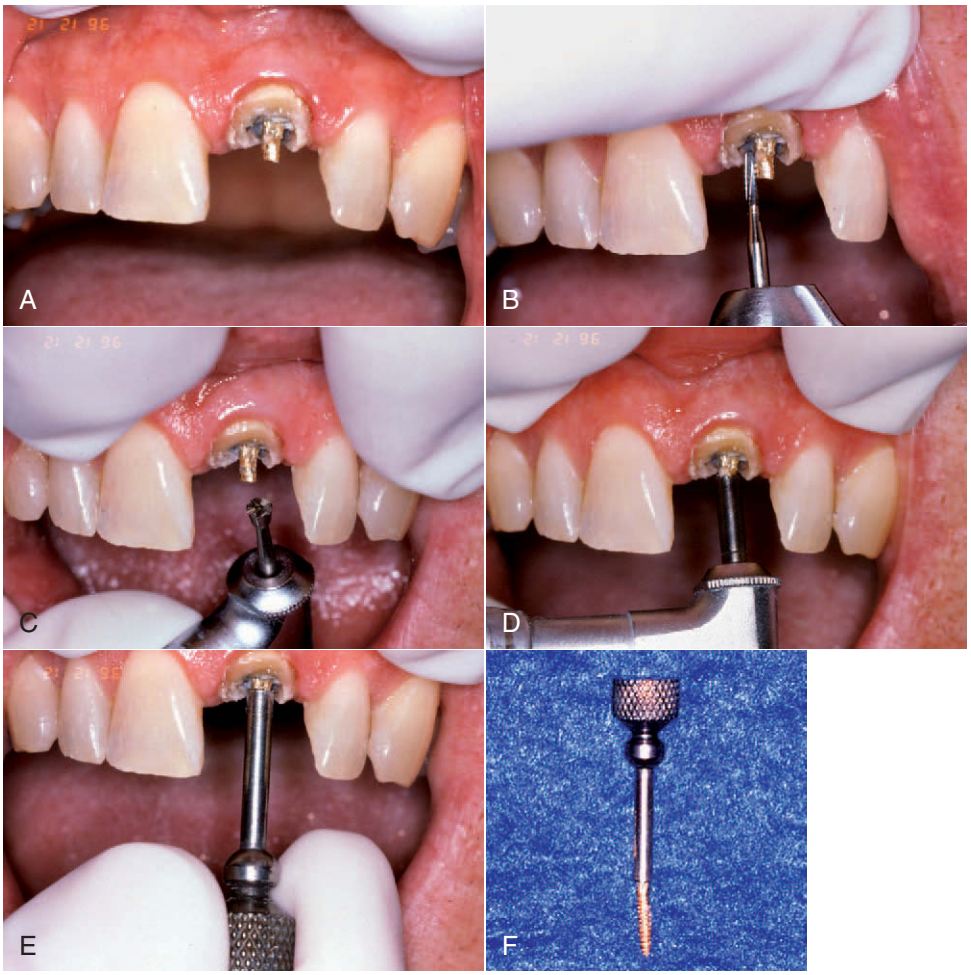


FIG. 25-22 Thomas screw post removal technique. **A,** Broken screw post. **B,** Head of post being contoured to a roughly cylindrical shape. **C-D,** Thomas Post Removal Kit. **E,** Domer bur creating a shape the trephine bur can engage. **F,** Trephine bur milling the post. **G,** Application of counterclockwise rotational force using the wrench. **H,** Post removed.

Potential Complications of Post Removal

As with many dental procedures, post removal has risks. These risks include fracture of the tooth, leaving the tooth unrestorable, root perforation, post breakage, and inability to remove the post.[201] An additional concern is ultrasonically generated heat damage to the periodontium.[177]

Even though there may still be some who feel posts strengthen teeth, it is widely accepted that they do not.[177] Actually, it has been shown that post preparation alone weakens teeth.[221] It seems obvious that any additional work that may require removal of further tooth structure will 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 using ultrasonics, but there was no significant difference between these two groups and teeth with posts that were not removed.[4] The authors speculated that the potential for vertical root fracture might be increased, but the clinical significance of this 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 very predictable procedure.[1] But if 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.[154] If perforation occurs, the clinician should repair it immediately; the prognosis will worsen as the time between perforation and repair lengthens.[21,102,182] Once a perforation occurs, the clinician must reconsider the prognosis and determine whether or not 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 and/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 recent development of mineral trioxide aggregate (Pro-Root MTA, DENTSPLY Tulsa Dental), perforations can

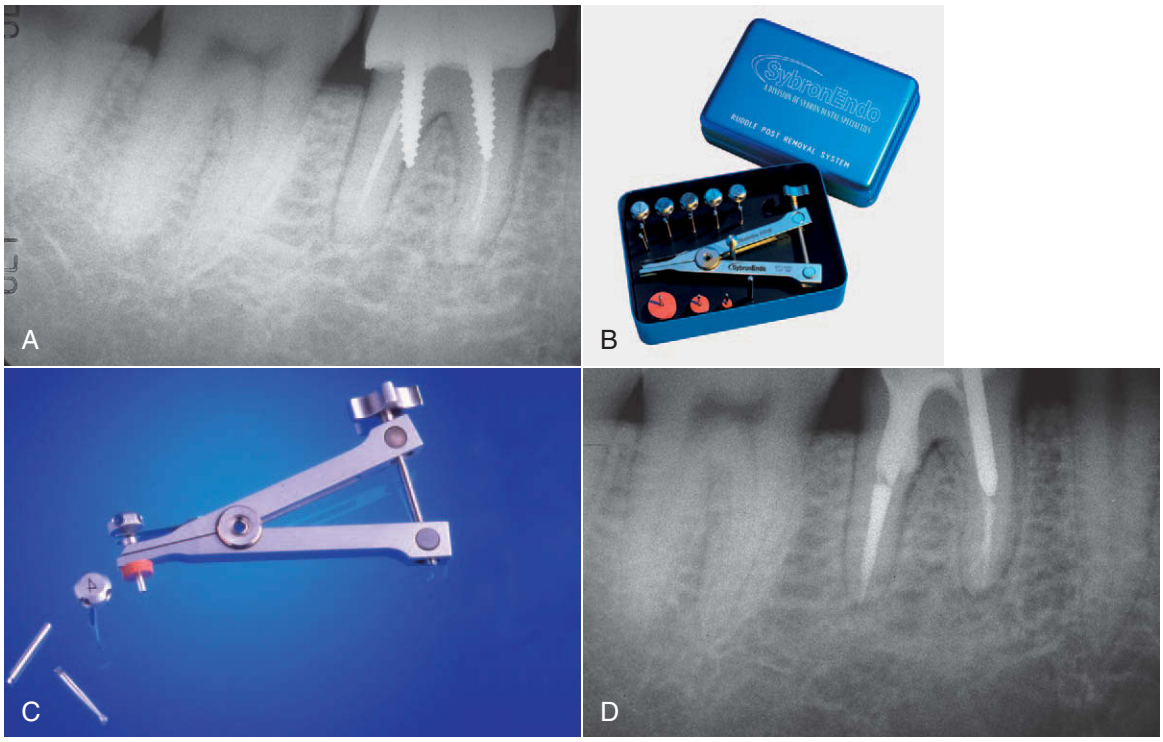


FIG. 25-23 A, Perforated post requiring removal. **B-C,** Ruddle post removal kit. **D,** Post removed and perforation repaired. *(**B** Courtesy SybronEndo, Orange, CA.)*

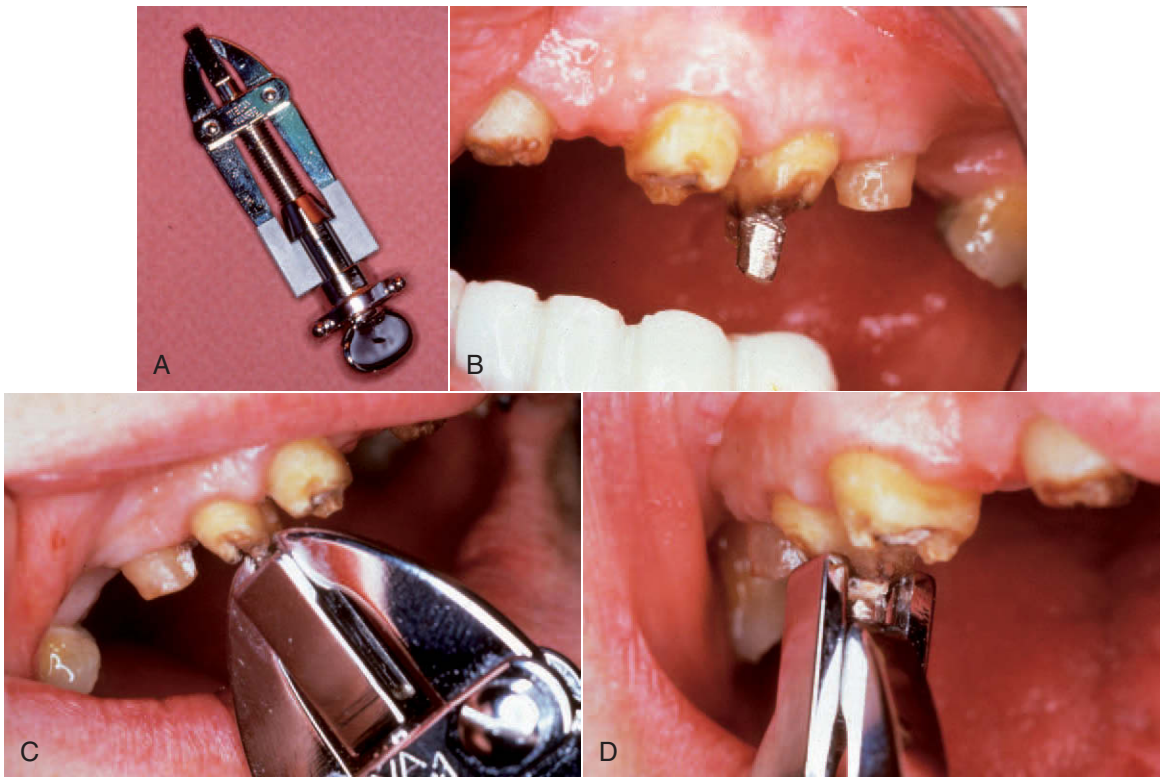


FIG. 25-24 A, Eggler Post Remover (Automaton-Vertriebs-Gesellschaft, Germany). **B,** Post has been contoured with a high-speed bur. **C,** Eggler Post Remover grasping the post. **D,** Elevating the post. *(From Stamos DE, Gutmann JL: Revisiting the post puller.* J Endod *17:466, 1991.)*

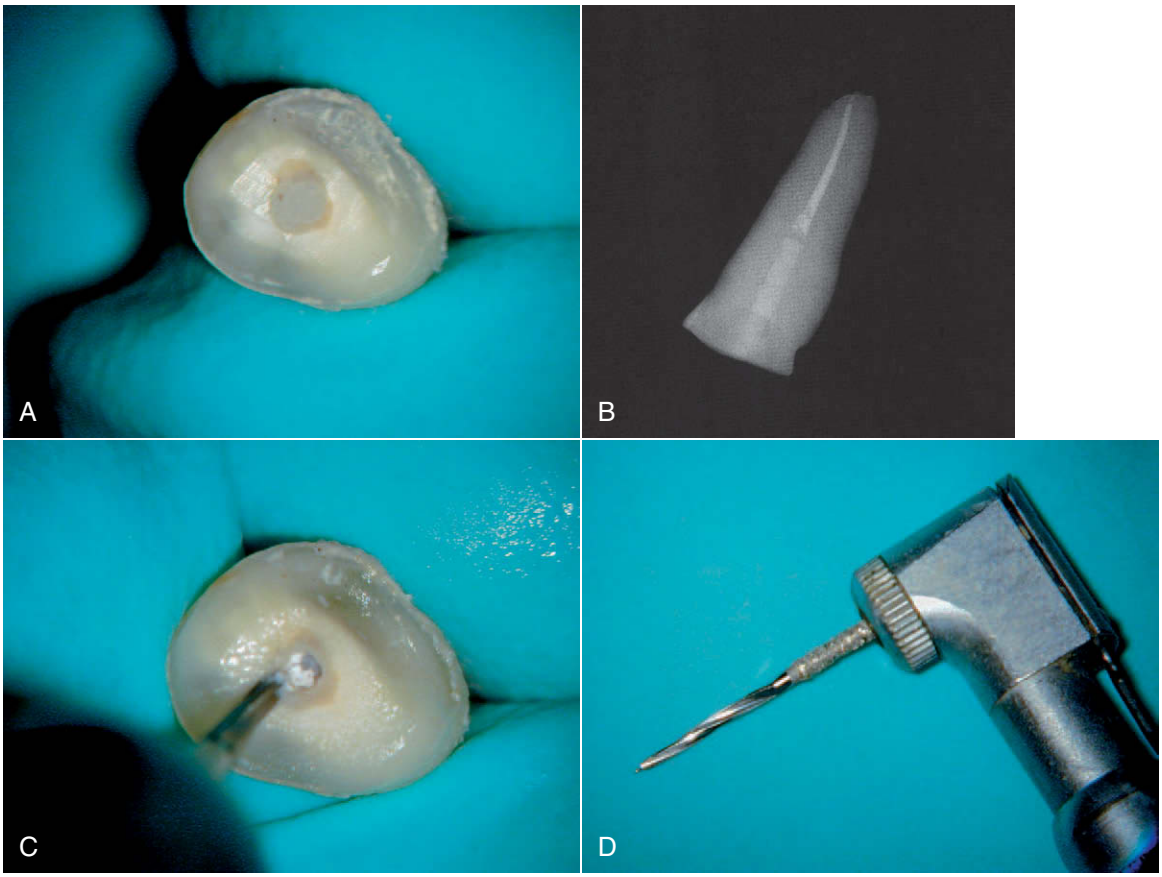


FIG. 25-25 GyroTip technique (MTI Precision Products, Lakewood, NJ). **A,** Broken fiber post in an extracted tooth. **B,** Radiograph of test tooth with post in place. **C,** Creating a pilot hole. **D,** GyroTip instrument.
Continued

be repaired with a favorable prognosis.[160] 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 and 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.[177]

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.[67,177] This damage may be as serious as both tooth and permanent bone loss (Fig. 25-26). 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, extraction and replacement with an implant and/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, the clinician must regain access to the apical area by removing the previous root-filling materials (Fig. 25-27). 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, but many other materials have been and are still being used. Silver points were very 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, Madison, CT), 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 very 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

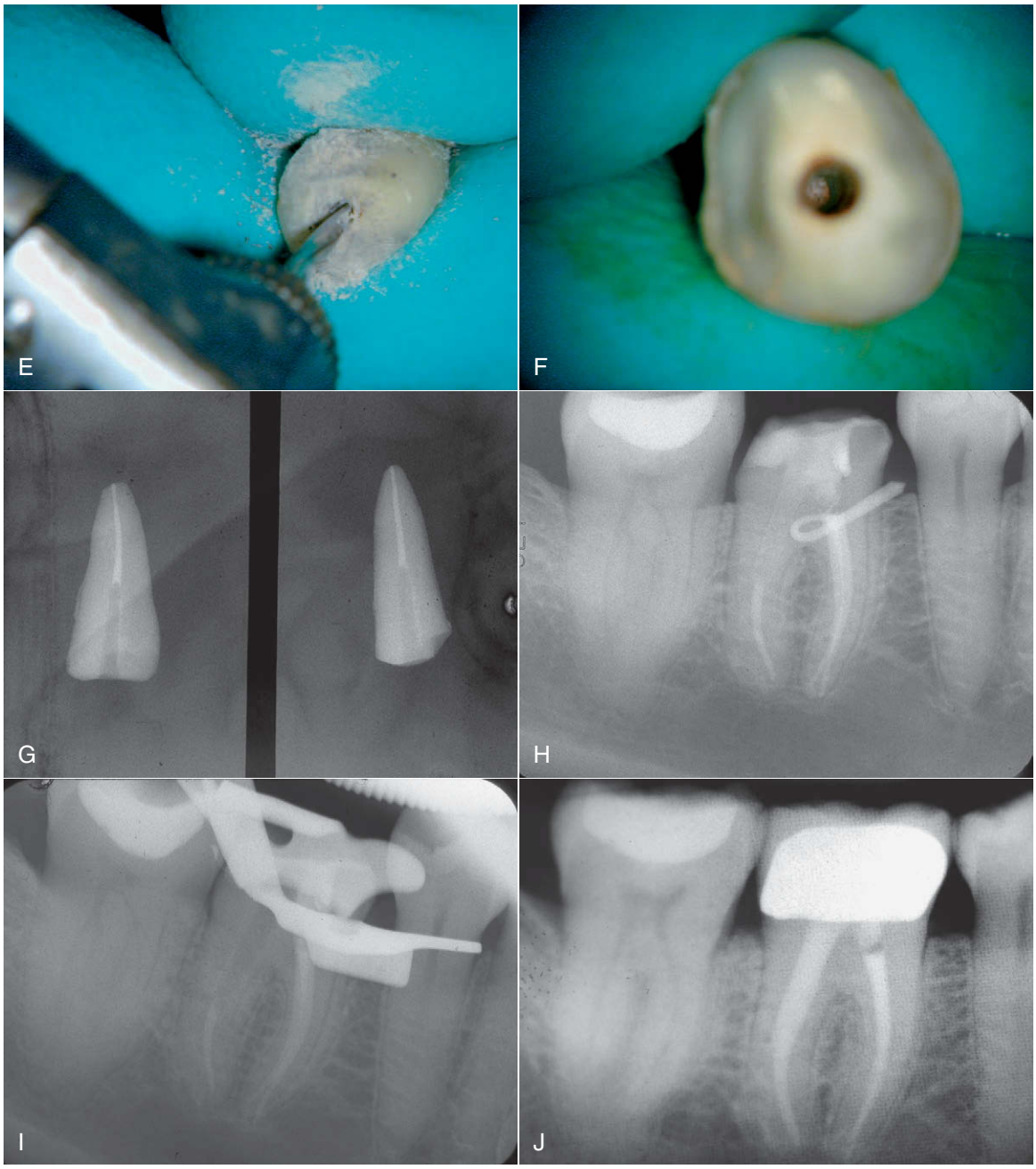


FIG. 25-25, cont'd E, GyroTip cutting through the fiber post. Note alignment with long axis of post. **F-G,** Post removed. **H,** Clinical case showing fiber post perforation into furcation area. **I,** Post removed with the GyroTip. **J,** One-year follow-up of MTA repair.

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.[58,163] 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 other heat sources—such as the Touch 'n Heat (SybronEndo) or DownPak (Hu-Friedy, Chicago, IL) (Fig. 25-28, *A*)—to provide constant, consistent heat application to soften the gutta-percha in the

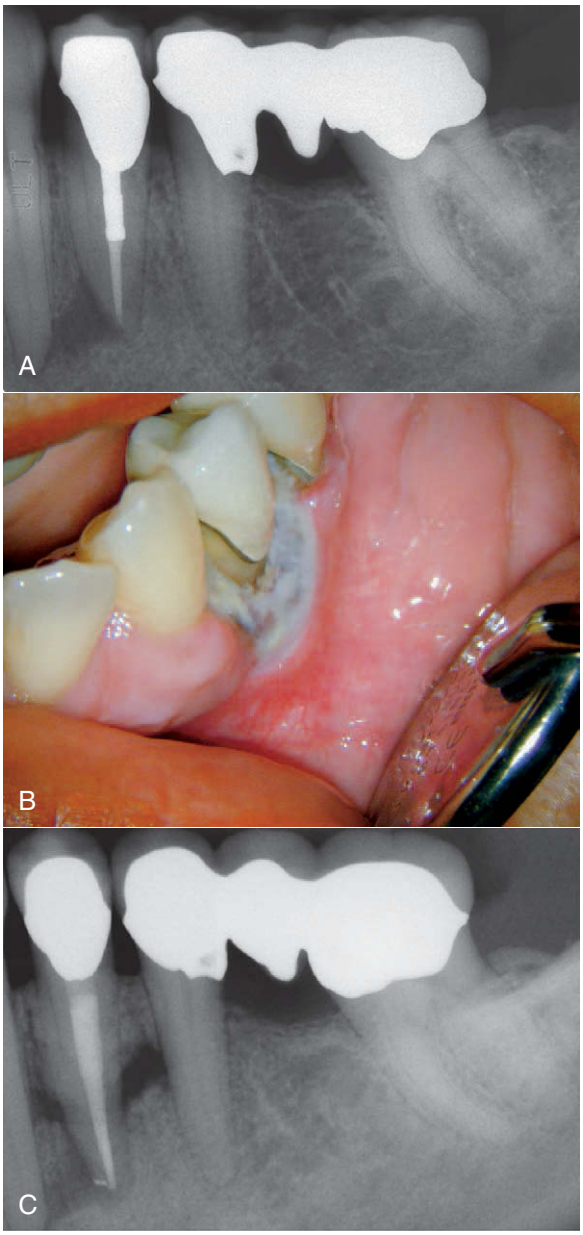


FIG. 25-26 Tissue damage from heat generated by ultrasonic application to a post during removal. The ultrasonic tip was applied to the post for no more than 5 minutes at high power, with the assistant applying a constant water spray. **A,** Preoperative radiograph. **B-C,** These images were taken 1 month after retreatment. Note sloughing bone visible on **C.** The tooth was lost 1 month later. *(From Schwartz RS, Robbins JW: Post placement and restoration of endodontically treated teeth: a literature review.* J Endod *30:289, 2004.)*

coronal portion of the canal.[75] Care must be exercised to not overheat the root, which can cause damage to the periodontal ligament.[115,169,170] 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 (Figure 25-28, *B*). After removing as much gutta-percha as possible with the heated instrument, then remove any remaining coronal material with



FIG. 25-27 Accumulation of materials removed from retreated teeth in a 3-month period.

small Gates-Glidden drills, taking care not to overenlarge the cervical portion of the canal. However, since 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 third of the canal and create a reservoir for potential solvent use.[129] 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.[199] If that is not possible, 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. 25-29), including chloroform,[132] methylchloroform,[228] eucalyptol,[242] halothane,[90,110] rectified turpentine,[104] and xylene.[78] All of the solvents have some level of toxicity,[12,35] so their use should be avoided if possible; however, a solvent is usually needed to remove well-condensed gutta-percha. The most popular solvent is chloroform, since 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 after a report of suspected carcinogenicity.[222] There was no associated ban on its use in dentistry,[132] but the report did result in the search for alternatives, some of which are listed. When used carefully, chloroform is regarded as a safe and effective endodontic solvent.[35,132] 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.[238] Rectified turpentine has a higher level of toxicity than chloroform[12] and produces a very pungent odor in the operatory. Halothane has been shown to be as effective a solvent as chloroform in several studies,[90,110] but a more recent study indicated that the time for removal of the root filling was longer than using chloroform.[231] 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.[35] Although methylchloroform is less toxic than chloroform, it is also less effective as a solvent for gutta-percha.[228] Both halothane and chloroform have been

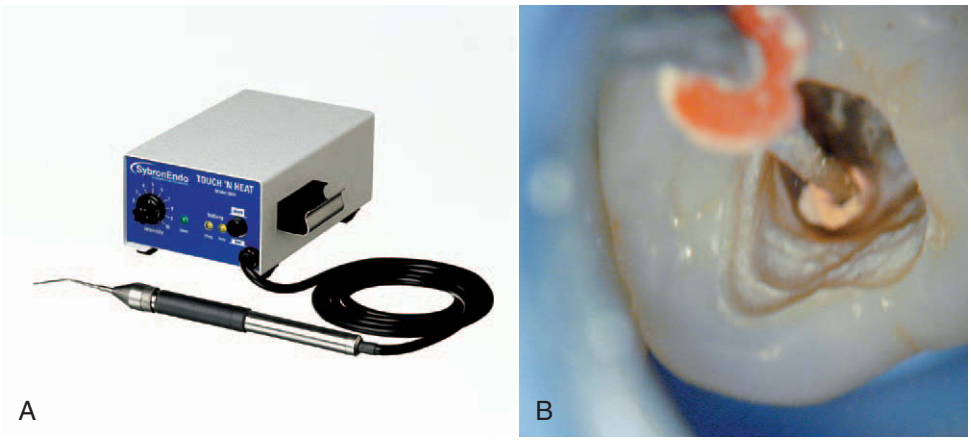


FIG. 25-28 A, Touch 'n Heat instrument. **B,** Gutta-percha adhering to the Touch 'n Heat tip as it cools. (*A Courtesy SybronEndo, Orange, CA.*)

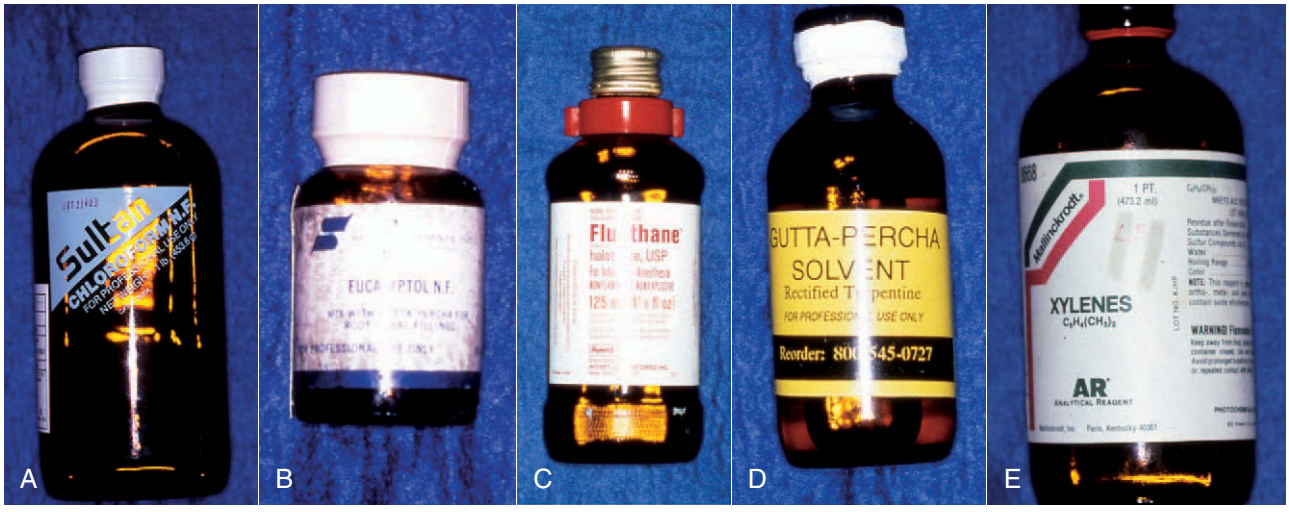


FIG. 25-29 A, Chloroform. **B,** Eucalyptol. **C,** Halothane. **D,** Rectified turpentine. **E,** Xylenes.

shown to affect the chemical composition of dentin[43,105] and may affect bonding strengths of adhesive cements to the altered dentin.[44] The clinical significance of these effects remains unknown. The evidence for the carcinogenicity of chloroform in humans is suspect,[132] but with careful use, its toxicity may be eliminated as a risk factor to both the patient[35] and the personnel in the operatory.[3] 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, Johnson City, TN) (Fig. 25-30), 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-



FIG. 25-30 C+ files. These rigid instruments remove gutta-percha more efficiently than more flexible types of K-files.

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.[75] Unfortunately, electronic apex locators, which in non-retreatment situations are very accurate,[106] seem to frequently misread the working length when gutta-percha is initially being removed. This clinical observation may be a result of 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,[227] but 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.[223] To avoid overextending root filling materials into the periodontium, it is recommended that a radiograph be made to gain a preliminary measurement when the estimated length is approached. 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). Frequent replenishment of the solvent should be used, 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[163] will remove much of the remaining film of gutta-percha and sealer that remains adherent to the canal walls and in the irregularities of the canal system.[234] Cleanliness of canals after gutta-percha removal is not improved by using a microscope[10]; 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.[75]

It should be noted that there exists a glass ionomer–based endodontic sealer (Ketac-Endo, 3M, Pymble, Australia) that is used in conjunction with gutta-percha.[156] This sealer is virtually insoluble in both chloroform and halothane[230] and must be retreated by removing the gutta-percha and then using ultrasonics to débride 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.[56,136]

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. The file is then slowly and firmly withdrawn with no rotation, removing the overextended material[133] (Fig. 25-31). 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 apical fragment should not be softened with solvent, because this application can decrease the likelihood of the Hedstrom file getting a solid purchase, hindering retrieval.[199]

Using rotary systems to remove gutta-percha in the canals has been advocated for their enhanced efficiency and effectiveness in removing gutta-percha from treated root canals.[163] This has generally been borne out in the literature. There are several

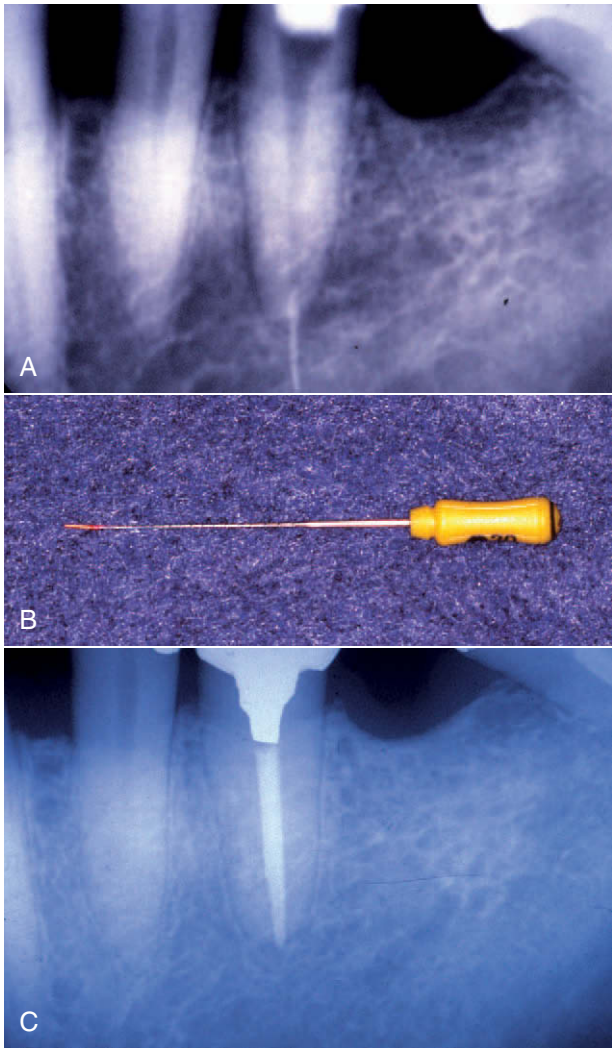


FIG. 25-31 Removal of overextended gutta-percha. **A,** Preoperative radiograph showing overextended filling material. **B,** A small Hedstrom file pierces the overextended material and retrieves it. **C,** Eighteen-month reevaluation. The tooth is asymptomatic.

types of mechanical rotary systems available for gutta-percha removal, including rotary file systems such as the ProFile (DENTSPLY Limited) (Fig. 25-32), 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, Savannah, GA), the ProTaper Universal retreatment files (DENTSPLY Limited) (Fig. 25-33), and the Mtwo R (Sweden and Martina, Padova, Italy). These engine-driven instruments mechanically chop up the gutta-percha and sealer 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.[151] In vitro studies have generally shown these systems to be efficient in that they generally need less time to remove the bulk of the gutta-percha filling material

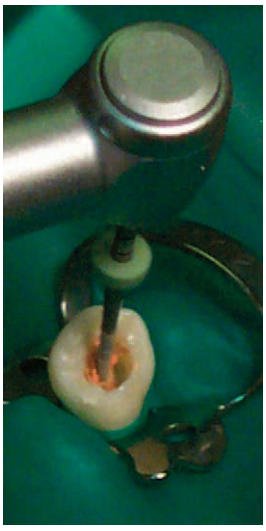


FIG. 25-32 Nickel-titanium rotary Profile thermoplasticizing and removing gutta-percha. Optimum rotary speed is 1500 rpm.

than hand removal,* although in two studies, they were slower to remove the root filling than hand filing.[13,93] Assessments of canal cleanliness and extruded apical debris generally indicated that there were no overall differences between hand and mechanical gutta-percha removal.† In one study using Quantec SC instruments (Tycom, Irvine, CA), however, it was found that hand files with solvent cleaned canals more effectively.[19] This finding has been repeated recently using the ProTaper retreatment files,[77] but in another study in the same journal issue, ProTaper retreatment files were found to leave canals cleaner than hand files.[66] 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, fracture of the mechanical instruments or the tooth root occurred.‡ This result was reported to occur less frequently when the instrument rotary speed was increased from 350 to 1500 rpm.[24] One study showed no separation or other canal defects when using dedicated retreatment files, the ProTaper Universal and Mtwo R instruments.[194] The dedicated retreatment files have end-cutting tips to enhance penetration and removal of the root filling mass, increasing their efficiency[210] (see Fig. 25-33, *B*). This in combination with flute design and techniques advocated may be the reason for the potential reduced risk of separation. While the mechanical gutta-percha removal systems may provide enhanced efficiency, the increased risk of instrument separation, further complicating retreatment, may outweigh this benefit. Dedicated retreatment files may reduce this risk.

Use of the Nd:YAG laser to remove gutta-percha from root-filled teeth has been investigated in vitro.[225] 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, so without further investigation proving safety and efficacy, laser gutta-percha removal cannot be recommended at this time.

Resilon (Resilon Research LLC) (Fig. 25-34) is a thermoplastic polyester polymer that is bonded into the canal space using an unfilled resin bonding system (Epiphany; Pentron). It is also marketed as RealSeal (Sybron Endodontics, Orange, CA). It has been advocated as a root canal obturating material to replace traditional gutta-percha and sealer because of its apparent enhanced sealing ability[185] and potential to strengthen root resistance to fracture as a result of internal bonding.[212] Resin-bonded obturation systems have been advocated in the past,[118] but the inability 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. Recent 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,[38,39,47,175] although this finding has been disputed. Hassanloo et al. 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.[82] This finding has been corroborated[175,211] and indicates that there may be a temporal effect that introduces a methodological bias in these studies. There may also be a problem with removal of the unfilled Epiphany resin sealer, especially since the sealer tags have been shown to penetrate deep into dentinal tubules[185] 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, Kent, UK) (Fig. 25-35) 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), have become very popular since their introduction several years ago. After cleaning and shaping procedures are completed, the clinician 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 into the canal and condenses it while the material is cooling. This system provides a rapid and simple technique for warm gutta-percha endodontic obturation. As with any obturating material, retreatment will be necessary occasionally.

Retreatment of solid core materials is considered to be more complex and difficult than removal of gutta-percha alone, owing 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. There are two types of carriers found in these systems: metal (stainless steel or titanium) and plastic. The plastic carriers are smooth sided,

*References 11, 50, 66, 88, 89, 166, and 213.

†References 13, 52, 88, 89, 94, 145, 166, 193, 213, and 244.

‡References 11, 19, 24, 89, 93, and 213.

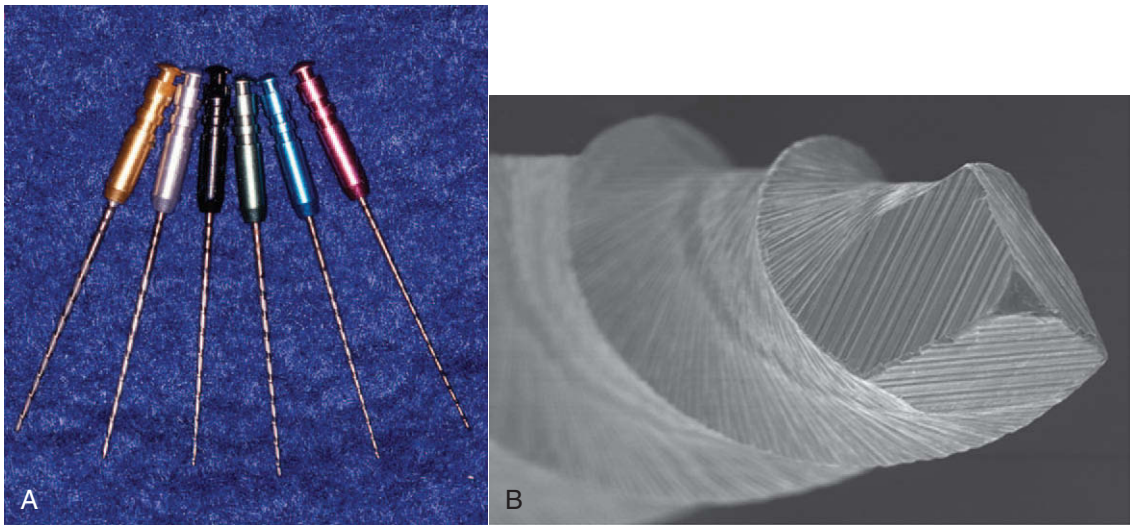


FIG. 25-33 Rotary gutta-percha removal instruments. **A,** Brasseler GPX Instruments. **B,** ProTaper Universal retreatment file has a cutting tip for enhanced penetration of the root filling materials. *(**B** Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*



FIG. 25-34 Epiphany Obturation System (Pentron Clinical Technologies, Wallingford, CT) using Resilon material (Resilon Research LLC, Madison, CT).



FIG. 25-35 Endosolv-E *(left)* and Endosolv-R *(right)* (Septodont, Kent, United Kingdom).

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, since 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,[75] 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. The technique of placing a nick in the midcanal level of the carrier to allow the clinician to rotate the carrier handle and sever the obturator deep in the canal is sometimes used to allow creation of a post space, but 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.[243]

It is advantageous to determine prior to initiating treatment whether there is a solid core obturation in the root-filled tooth. The preoperative radiograph may show this, since stainless steel carriers will exhibit a fluting effect on the radiograph (Fig. 25-36); however, titanium carriers rarely are distinguishable from gutta-percha, and plastic ones never are. In most instances, the clinician finds that they are dealing with a carrier-based obturator after initial access to the pulp chamber. This is why, as was 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

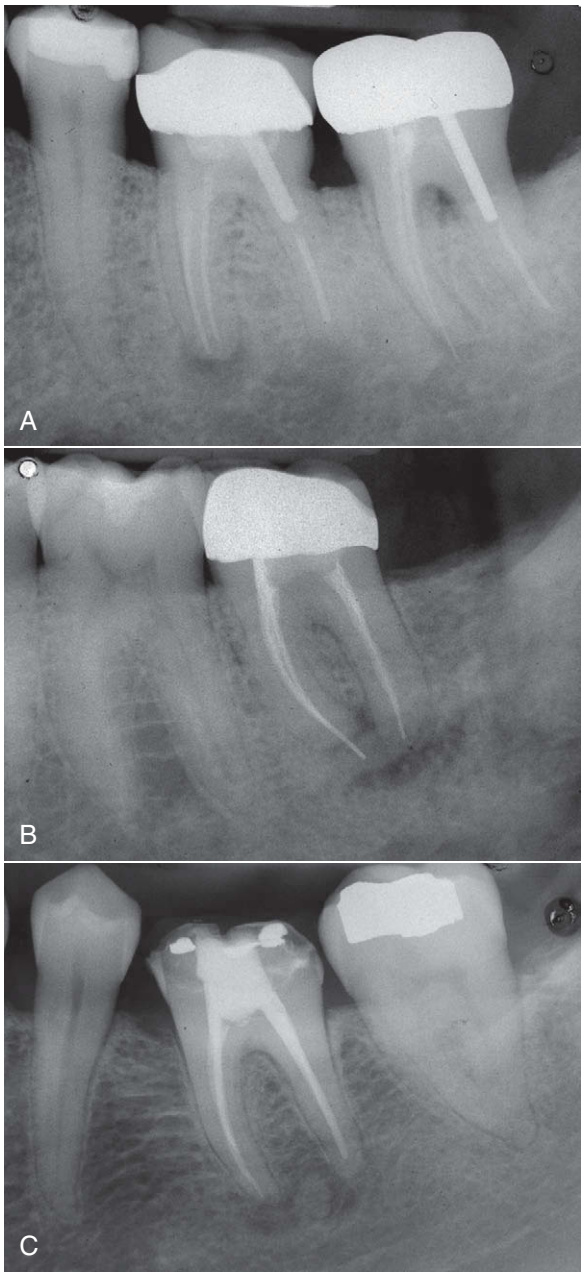


FIG. 25-36 Comparison of radiographic appearances of three different obturating materials. **A,** Gutta-percha. **B,** Stainless steel Thermafil carrier (note subtle fluting effect in the fill). **C,** Plastic Thermafil carrier.

structure embedded in the gutta-percha mass or a black spot, indicating a plastic 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.[75]

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 (Pulpdent Corporation, Watertown, MA)[75,173,232,233] (Fig. 25-37). Often there is not enough of the carrier remaining in the access to grasp with forceps, and 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[75,163] (Fig. 25-38), 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,[53,243] 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 generally, heat should be avoided to minimize the likelihood of damaging the carrier.[75] 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; the larger sizes used polysulfone, which is soluble in chloroform.[152] Solvents, on the other hand, seem not to affect the newer GT plastic carriers, so their use can be recommended[18] (Fig. 25-39). 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 (e.g., #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 separation of the file or the carrier may result.[18] Occasionally, grasping pliers will be needed to remove the carrier if it is accessible[95,163] 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 prone to separation and unable to be retrieved, potentially resulting in the need for apical surgery.[92]

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.[236] The temperature is set at 225° C, and the heat plugger is placed buccal and lingual to the carrier, after which #50 to #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 to using solvent.[236] Concerns regarding heat generation in the periradicular tissues have been raised,[121] 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 turned off. This allows

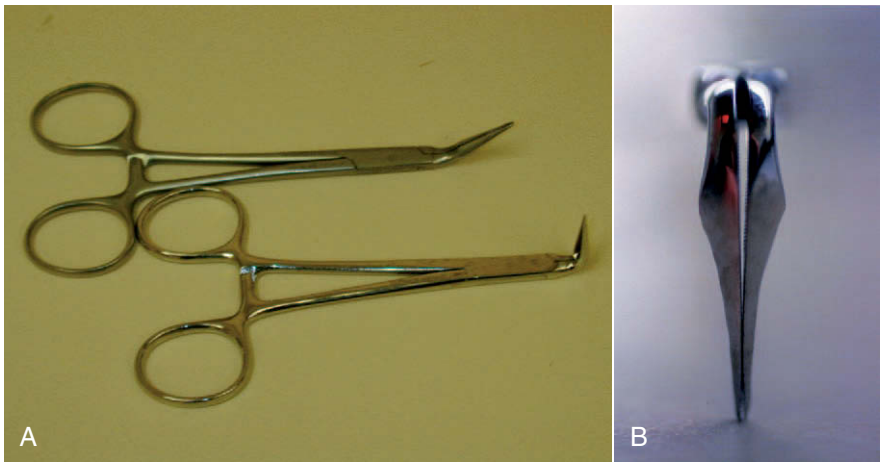


FIG. 25-37 A, Steiglitz forceps (Pulpdent Corporation, Watertown, MA) in 45- and 90-degree head angles. **B,** Tips of the Steiglitz forceps ground to a thinner contour to create the "modified" instrument. This allows deeper penetration into the tooth to enhance removal of obstructions. *(**B** Courtesy Dr. Daniel Erickson.)*

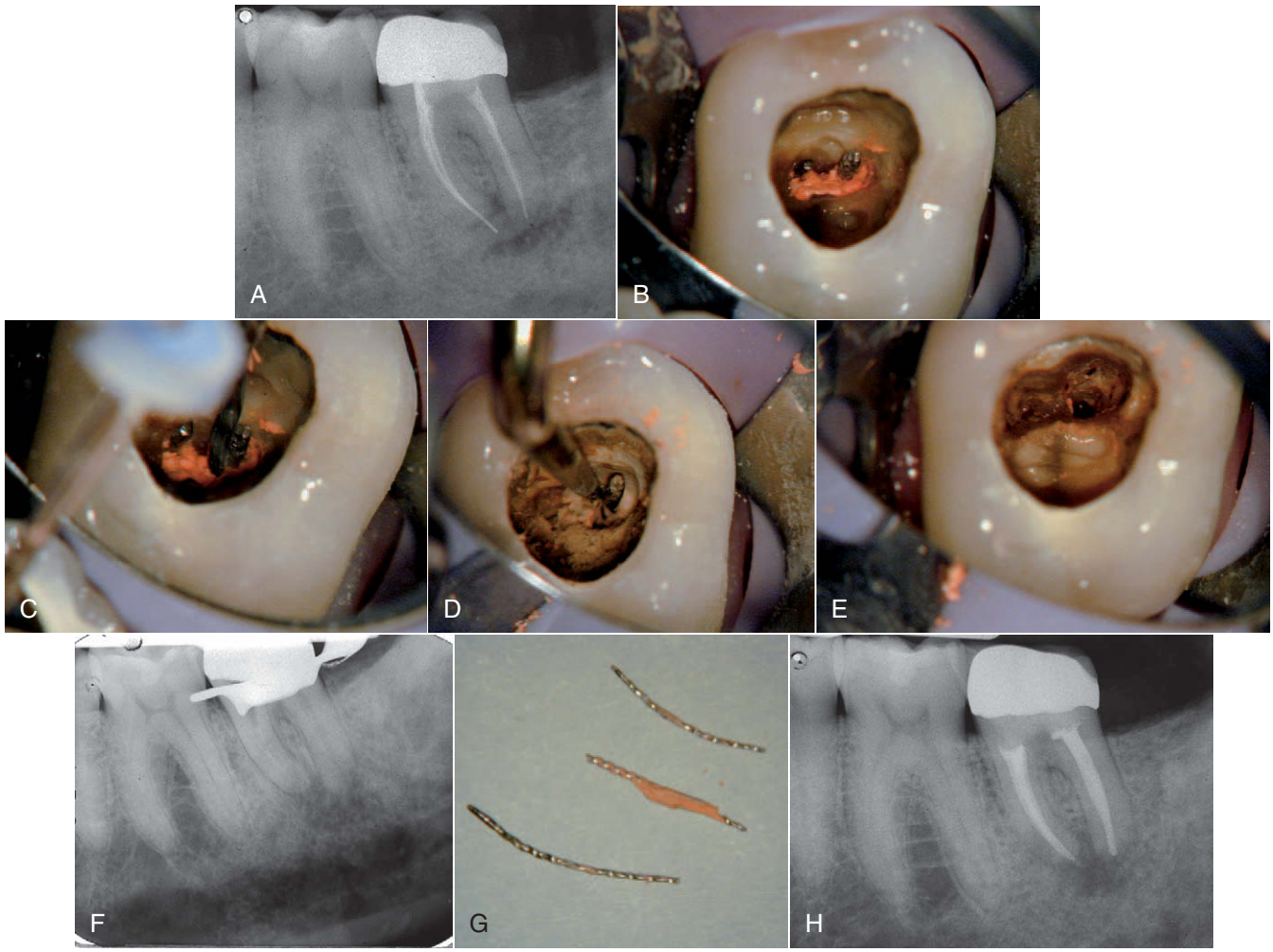


FIG. 25-38 Metal carrier retreatment. **A,** Preoperative radiograph. **B,** Metal carriers exposed by careful excavation of gutta-percha. **C,** Use of the Touch 'n Heat instrument to heat the carriers and soften the gutta-percha. This allowed removal of one of the carriers using modified Steiglitz forceps. The other could not be removed using heat or solvents. **D,** Ultrasonic troughing around the carrier to facilitate grasping it with forceps. **E-F,** Carriers removed and confirmed with a radiograph. **G,** Metal carriers showing gutta-percha still adhering to them. **H,** Final obturation of the tooth.

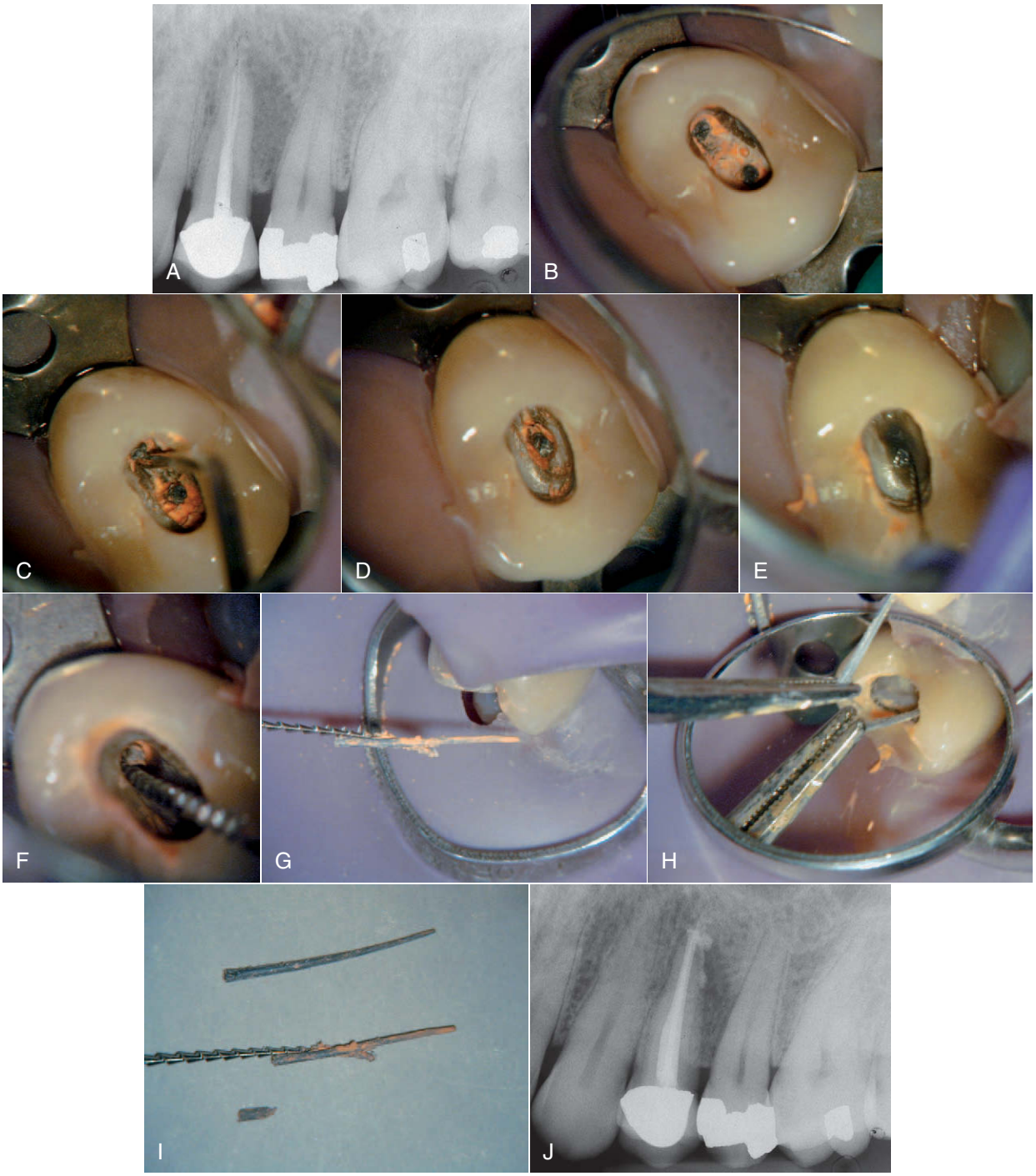


FIG. 25-39 Plastic carrier retreatment. **A,** Preoperative radiograph. At this stage, the nature of the root filling is unknown. **B,** Plastic carriers visible in the access as two black spots in the gutta-percha mass. **C,** Gutta-percha in the chamber is carefully removed from the carriers. **D,** 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-G,** A Hedstrom file is gently screwed into the canal alongside the carrier and withdraws it on removal. **H,** A hemostat removes the other carrier. **I,** Plastic carriers removed. **J,** Final obturation with Resilon and Epiphany sealer.

the plastic carrier to adhere to the tip while cooling and may result in its removal upon withdrawal of the heat tip (Fig. 25-40).

Rotary instruments have been advocated for use in removal of plastic carriers and gutta-percha, and a recent study showed that removal of plastic carriers was successful in all but one of the teeth obturated with them.[11] 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.[162]

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, since the more highly processed gutta-percha used with these carriers may be more difficult to remove than other forms of the material. One study 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.[243] Their findings have not been corroborated, however, and other studies have shown no difference in residual debris remaining after carrier-based removal.[53,95,243] 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.[163]



FIG. 25-40 Plastic Thermafil carrier adhering to a System B heat plugger (SybronEndo, Orange, CA).

Paste Retreatment

Various pastes have been used as root canal obturating materials, especially outside North America. There are such a wide variety of paste compounds used in endodontics that 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, making them toxic and a potential danger to the patient's health, both local and systemic if overextended beyond the confines of the root canal system.[22,150] None has the potential to seal the canal effectively, and many render a tooth impossible to retreat,[176] so their use is strongly discouraged. On radiographic examination, they can usually be discerned by their lack of radiopacity, the presence of voids, and evidence of inadequate canal shaping and poor length control (Fig. 25-41). 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, since 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.

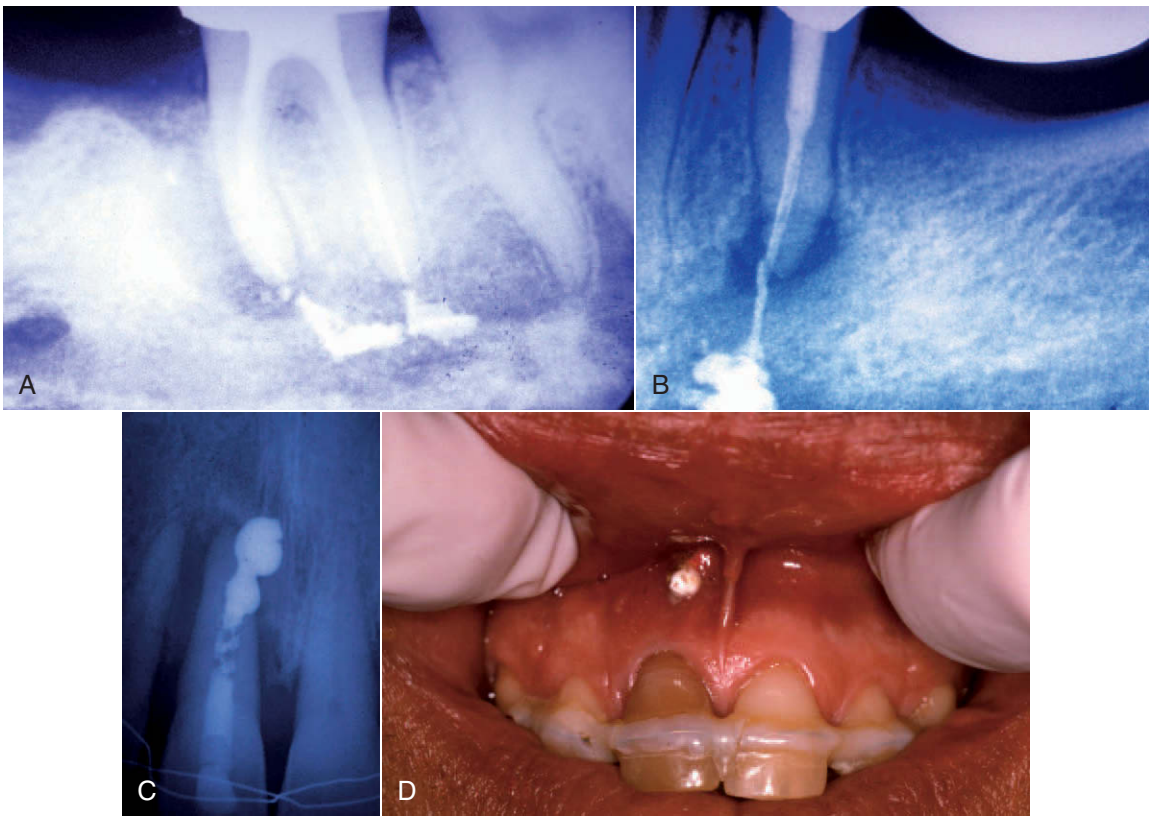


FIG. 25-41 Example of poor length control with paste root fillings. **A,** Overextended paste filling into the inferior alveolar canal. **B,** Overextended paste filling into the mental foramen. **C,** Overextended paste filling extending through a perforation in an upper central incisor. **D,** Clinical appearance of the case in part **C.** Note the material extending out through a sinus tract.

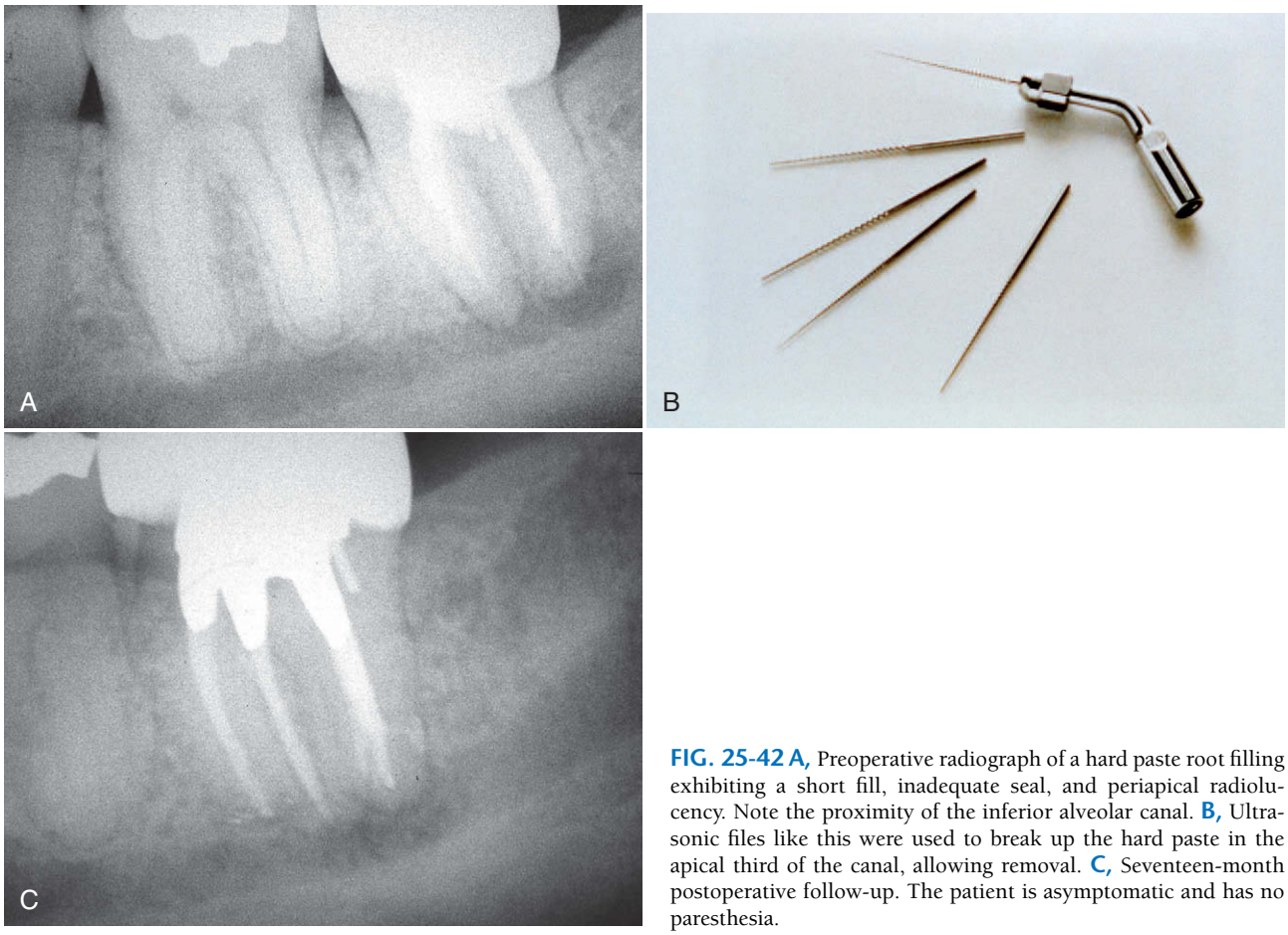


FIG. 25-42 A, Preoperative radiograph of a hard paste root filling exhibiting a short fill, inadequate seal, and periapical radiolucency. Note the proximity of the inferior alveolar canal. **B,** Ultrasonic files like this were used to break up the hard paste in the apical third of the canal, allowing removal. **C,** Seventeen-month postoperative follow-up. The patient is asymptomatic and has no paresthesia.

Great care should be exercised when removing the paste to avoid overextension, potentially severe postoperative pain,[73] and possible paresthesia/dysesthesia from the paste's potential neurotoxicity.[25,183] Soft pastes are generally easy to remove using crown-down instrumentation with copious sodium hypochlorite irrigation to minimize extrusion.[75] Greater difficulty arises when the paste is set hard.[176] Since 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[58] or a straight, tapered ultrasonic tip in the easily accessible straight portions of the canal, using magnification and illumination.[163] 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 penetration to the apex may be possible.[163] 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, Kent, UK) (see Fig. 25-35). 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. 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,[61] and the clinician may elect to leave some solvent in the canals between appointments to soften the paste.[164] Care should be taken in the choice of temporary restoration, since the solvent left in the canal may also soften the temporary, potentially leading to breakdown of the interappointment seal.[135]

Ultrasonically activated files have been advocated for use in penetrating hard-set pastes in the curved apical segments of canals[101,109] (Fig. 25-42). The ultrasonic energy breaks up the paste, and the irrigation floats the fragments in a coronal direction until the apical terminus is reached.[58] This technique is reported to be very 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.[73]

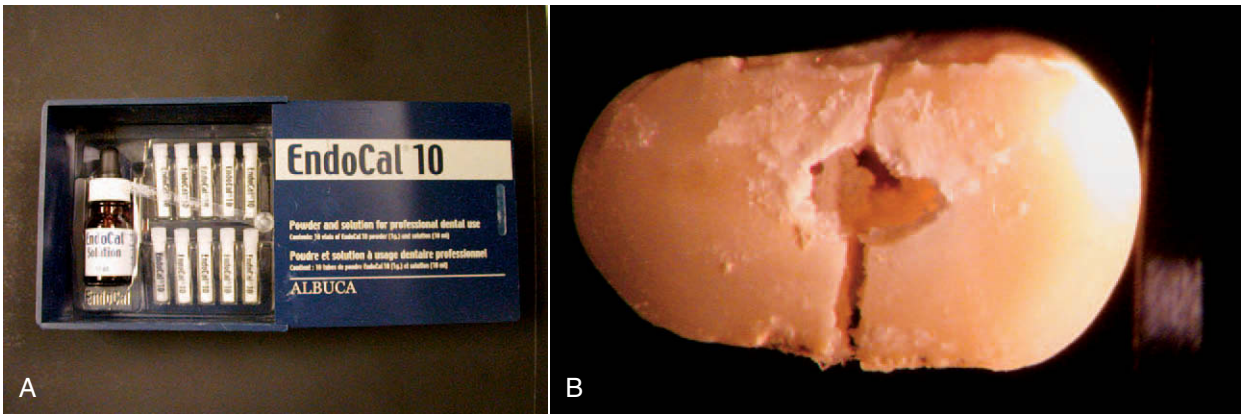


FIG. 25-43 A, Endocal 10 (formerly Biocalyx [Albuca Inc, Montreal, Canada]). **B,** Split root in a case filled with Endocal 10. *(Courtesy Dr. Rob Goldberg.)*

Biocalex 6.9 (currently known as Endocal 10) (Fig. 25-43) is a hard-setting calcium oxide paste that has been popular in Europe for over 30 years but is now being used in North America since its recent FDA approval.[69] 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.[69] Retreatment will be complicated by the hard-setting nature of this material, but since it is a calcium oxide paste, ethylenediamine tetra-acetic acid (EDTA) may soften it, facilitating its removal. Because EDTA also softens dentin, care must be taken not to 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 because silver appears to have some antibacterial activity.[178] However, over the past few decades, the usage of silver points has dramatically diminished. Presently they are considered a deviation from the standard of care. The main reason for this change is because they corrode over time (Fig. 25-44), and the apical seal may be lost.[23] 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[123,174] (Fig. 25-45). The corrosion of silver points occurs when they come into contact with tissue fluids and certain chemicals used in endodontics, including sodium hypochlorite and some sealers.[74] This corrosion produces chemicals such as silver sulfide, silver sulfate, silver carbonate, and silver amine hydrate,[181] which have been shown to be cytotoxic in tissue culture.[178] Corrosion occurs mainly at the apical and coronal portions of the points, indicating that leakage is responsible.[181] Gutta-percha root-filling techniques do not suffer from these disadvantages and have replaced the use of silver points in endodontics. Owing to this decrease in usage over the past quarter century, 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, are smooth-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. 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.[163]

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 to not 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, since fresh solvent enhances the efficiency of cement removal. At this point, the easiest technique, which is also very predictable, is to grasp the exposed end of the silver point with a Stieglitz pliers (Henry Schein, Melville, NY) (see Fig. 25-37) or other appropriate forceps and gently pull it out of the access cavity preparation. If too much extraction force is needed, 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[75] (Fig. 25-46). 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

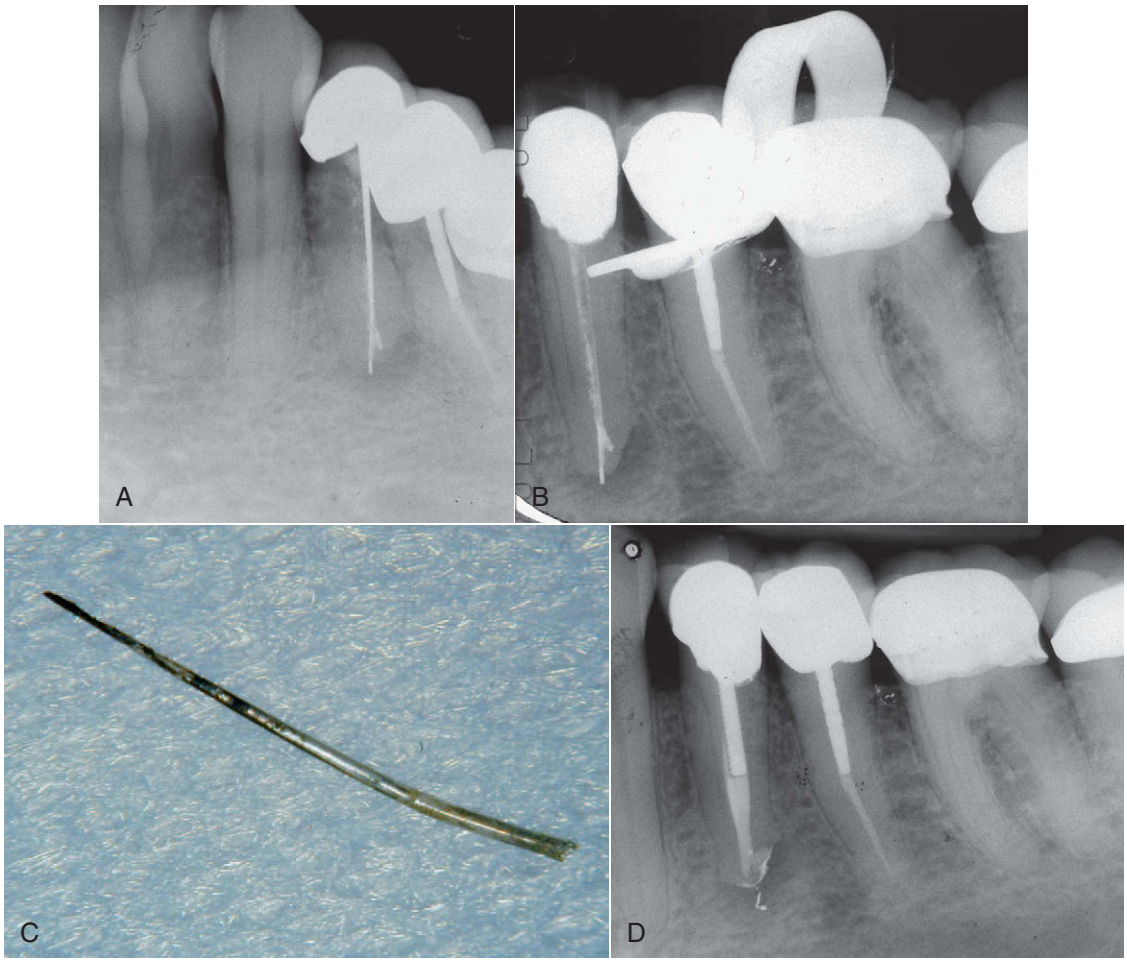


FIG. 25-44 A, Persistent disease in a silver point filled tooth. **B,** Silver point removed. Note the radiopaque material in the apical portion of the canal system. This represents corrosion products remaining in the canal and a possible separated apical segment of the cone. **C,** Removed silver point 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.

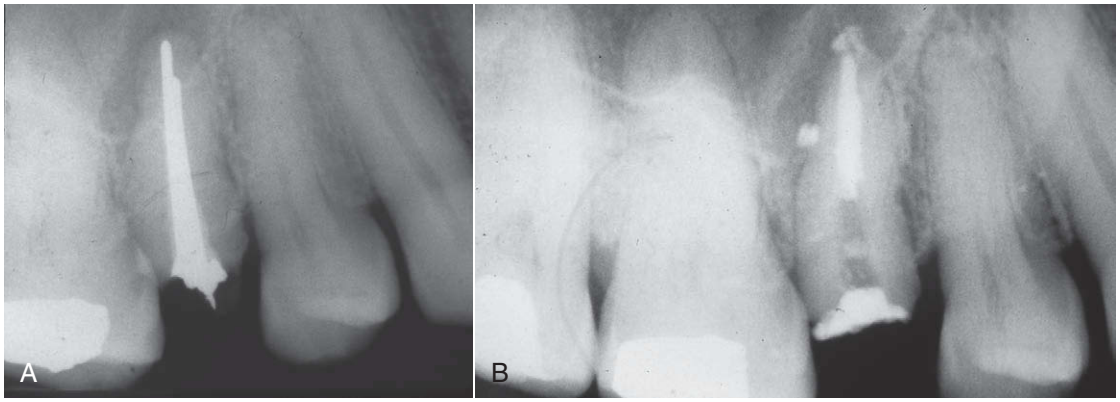


FIG. 25-45 A, Preoperative view of a silver point case with persistent disease. Note that the periapical radiolucency extends coronally on the distal aspect of the root end, indicating the presence of an unfilled lateral canal. **B,** Postobturation radiograph showing the cleaned and filled distal canal branch.



FIG. 25-46 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.

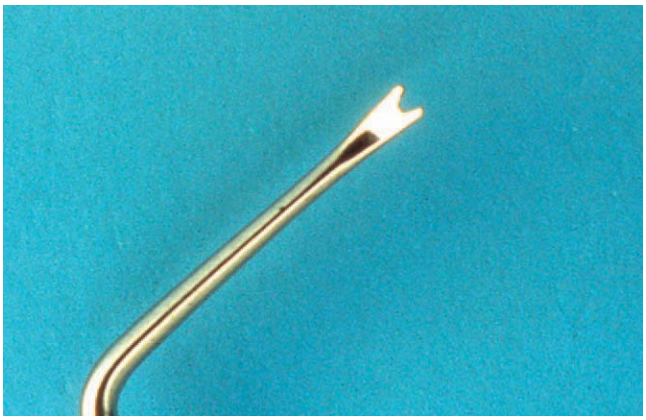


FIG. 25-48 Caufield elevator tip, useful in gripping and elevating silver points that are protruding a small amount into the pulp chamber.

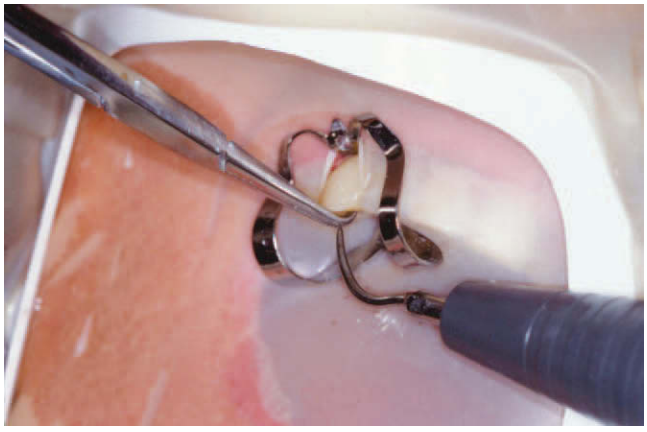


FIG. 25-47 Application of indirect ultrasonic energy to a silver point by placing the ultrasonic tip against forceps that are holding the silver point.

ultrasonic energy is applied to the forceps, not the point (Fig. 25-47). 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 (Miltex Inc. York, PA). This instrument is a spoon with a groove in the tip (Fig. 25-48) 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.[75] The 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.[122] The sealer is dissolved as previously mentioned, then files are negotiated as far apically as possible 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 small Hedstrom files are gently screwed in as far as possible apically. To prevent breakage, they should not be screwed in too tightly. The flute design of the Hedstrom file allows for much better engagement into the silver point, compared to other file designs. The files are then twisted together and pulled out through the access (Fig. 25-49). 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.[58] 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 ultrasonic energy directly to a post or file may prove beneficial in vibrating them loose, silver points are much softer. 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.[163] The ultrasonic instrument is used on tooth structure circumferentially around the silver point. This is a very 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, techniques that involve the use of an end-cutting trephine bur to remove tooth structure around the

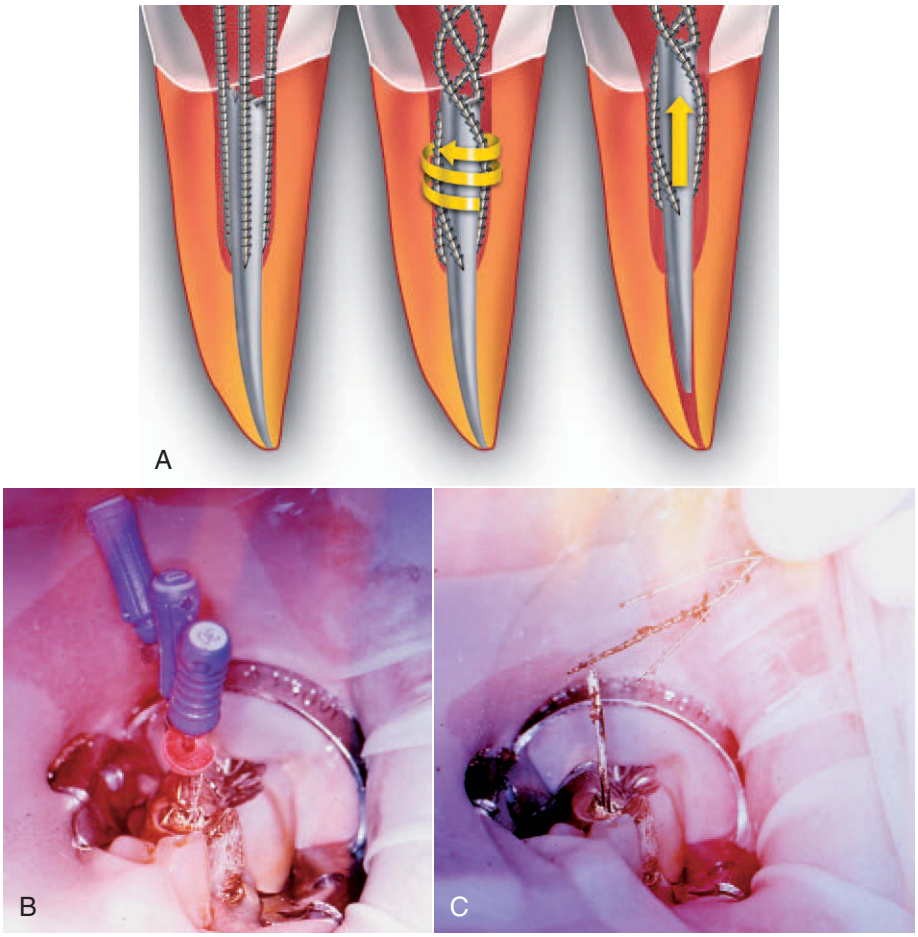


FIG. 25-49 A, Diagram illustrating the braiding of Hedstrom files around a silver point. By twisting the braided files, a gripping force is applied which aids in removal of the obstruction. **B,** Small files being braided around a silver point. **C,** Pulling coronally with the braided files removes the silver point. *(A Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

point and then use of an extraction device to remove it may be employed (Fig. 25-50). There are many kits that use these principals with slight variations from one another, including the Endo Extractor (Brasseler),[198] the Masserann Kit (Medidenta International, Woodside, NY), and the Extractor System (Roydent, Troy, MI) (Fig. 25-51). Additional techniques that 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,[163] and the Instrument Removal System (DENTSPLY Tulsa Dental, Tulsa, OK).

These kits are not only effective in the removal of silver points but also in the removal of separated instruments. Because of this common approach to removing both during endodontic retreatment, these techniques will be discussed in detail in the following section on separated instrument removal.

After the silver point is removed, it is very 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 as a result of 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, the case should be completed and followed carefully (Fig. 25-52). Apical surgery or extraction could be necessary in the future (Fig. 25-53).

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. 25-54). It is useful to expose a check radiograph after removal of the root filling to see if there is any metallic

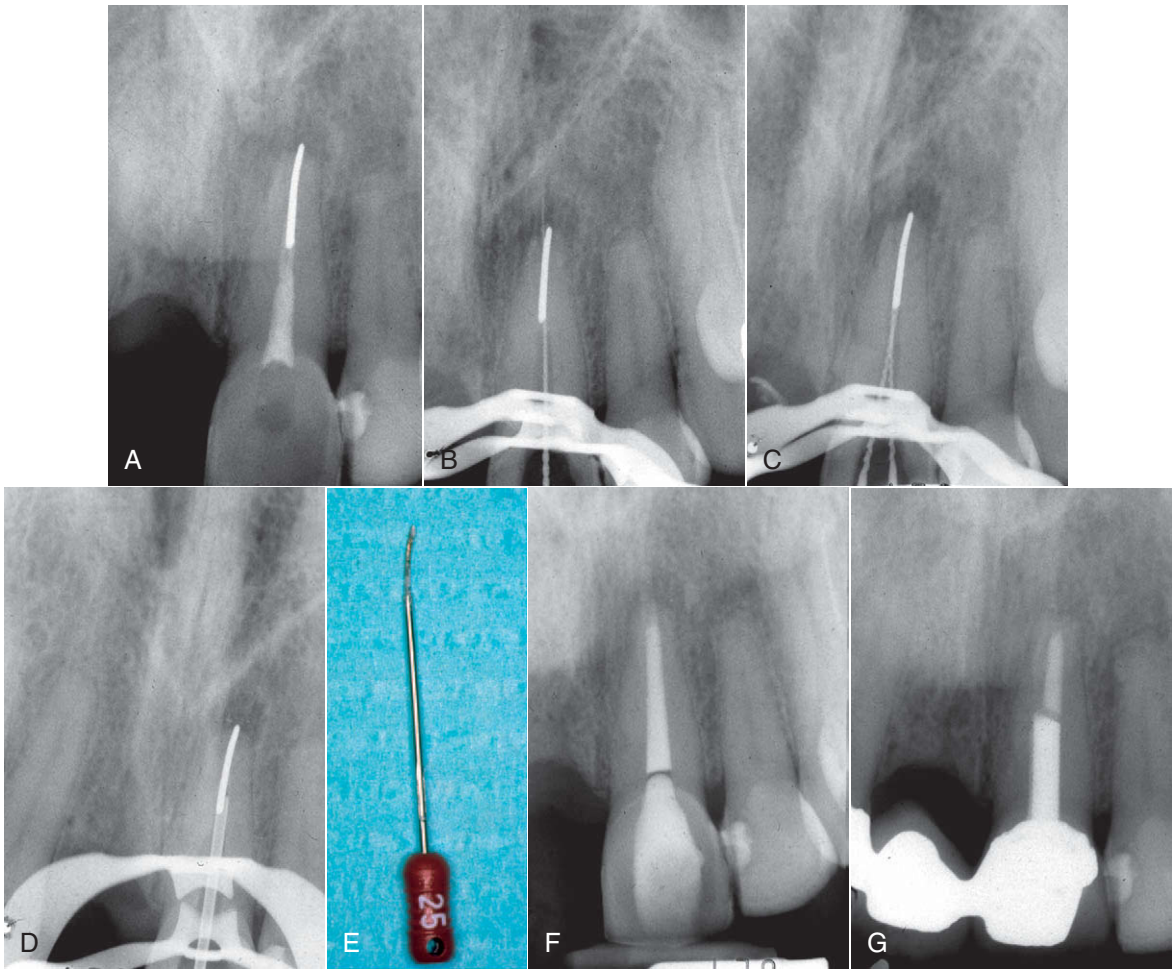


FIG. 25-50 Twist-off silver point case. **A,** Preoperative radiograph showing apical periodontitis and a split silver cone (twist-off) obturation technique. **B,** The cone was initially bypassed but could not be loosened. **C,** The braided Hedstrom file technique was attempted but was unsuccessful. **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,** One-year follow-up showing apical healing. *(From Gutmann JL, Dumsha TC, Lovdahl PE (eds):* Problem solving in endodontics: prevention, identification, and management, *ed 4, St Louis, Mosby, 2006.)*

obstruction in the canal. Regardless of which type of instruments the clinician uses, whether stainless steel or nickel-titanium, and 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%[98]; for rotary instruments, it ranges from 1.68% to 2.4%.[98,237]

The most common causes for file separation are improper use, limitations in physical properties, inadequate access, root canal anatomy, and possibly manufacturing defects.

A very common cause for instrument separation is improper use. Included in this are overuse or 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.[75]

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 usage.
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.[75]

Another type of improper use is to apply too much apical pressure during instrumentation,[218] 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 use them in a dry canal, because attempting to instrument a dry canal will cause excessive frictional stresses on an instrument.[218] Continual lubrication of the canal with either irrigating solutions or lubricants is required.[75] 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.

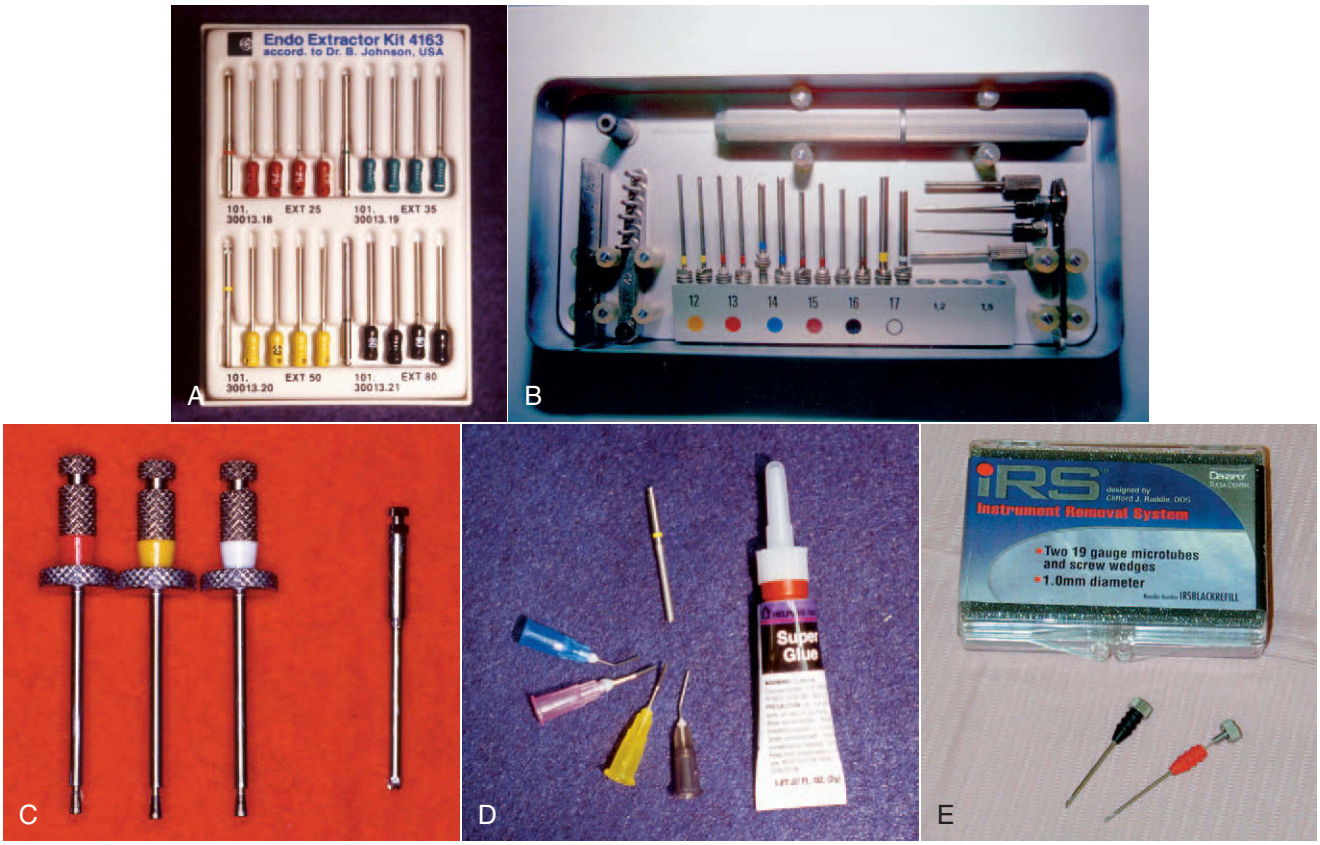


FIG. 25-51 A, Brasseler Endo Extractor kit (Brasseler USA, Savannah, GA). **B,** Masserann kit (MICRO-MEGA Dentalvertrieb GmbH & Co. KG, Germany). **C,** Roydent Extractor System (Roydent Dental Products, Johnson City, TN). **D,** Separated Instrument Retrieval System (SIR) (Vista Dental Products, Racine, WI). **E,** Instrument Removal System (IRS) (DENTSPLY Tulsa Dental, Tulsa, OK). (***B** Courtesy Dr. Daniel Erickson.*)

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 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 the file must negotiate. This under prepared 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, since traversing an S curve greatly stresses the rotating file, leading to separation (Fig. 25-55).

Anatomy, such as abrupt curvatures or anatomic ledges, increases the likelihood of instrument fracture. When the file's progress is hindered, it is quite 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.[218]

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.[218] 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 and its effect on the prognosis.[36] In addition, when a file separates, as with other procedural accidents, detailed documentation is necessary for medicolegal considerations,[218] and the remaining segment of the file should not be discarded but placed in a coin envelope and kept in the patients record.[36]

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.[197] 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

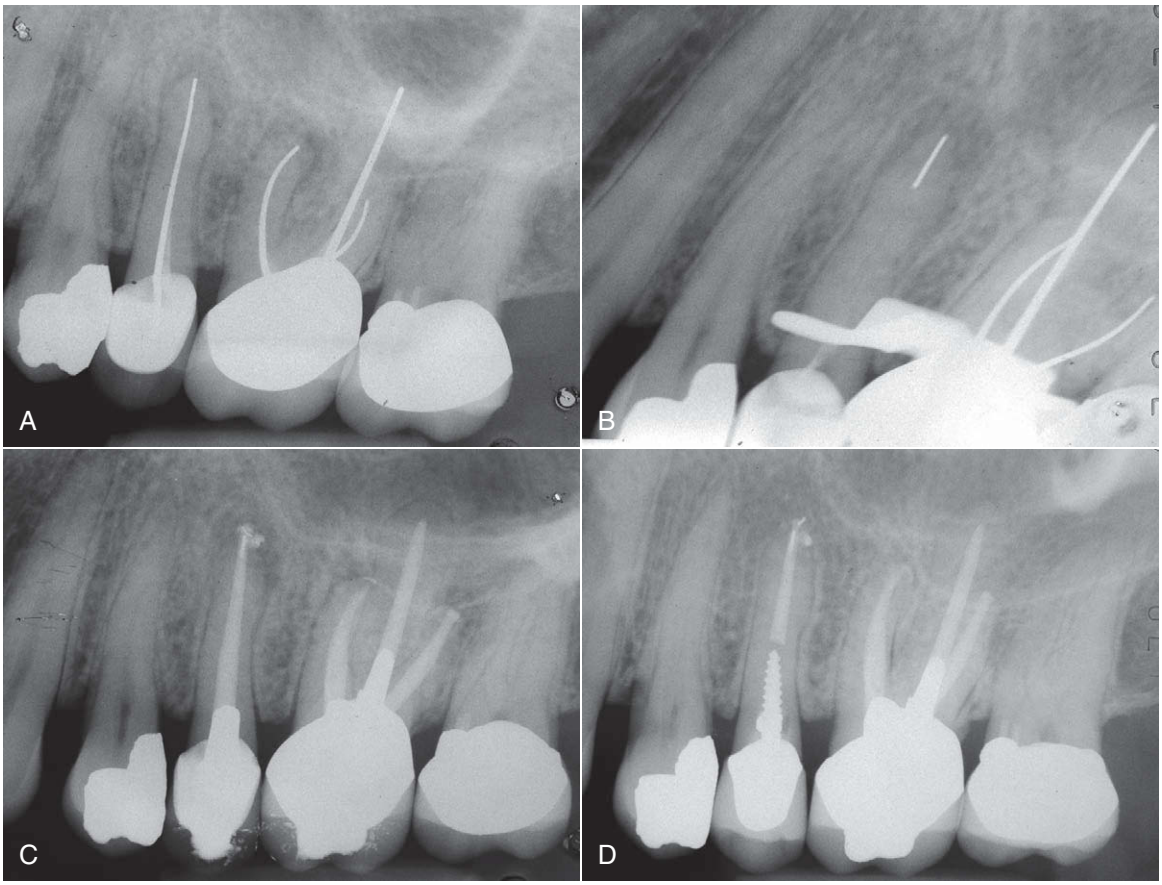


FIG. 25-52 Case illustrating healing despite inability to remove a separated silver point. **A,** Preoperative radiograph showing persistent disease in an upper premolar and molar. **B,** Working film showing the separated cone that could not be retrieved despite extensive clinical efforts. **C,** Final obturation after two-appointment procedure using a calcium hydroxide interappointment medicament. **D,** Four-year follow-up showing apical healing.

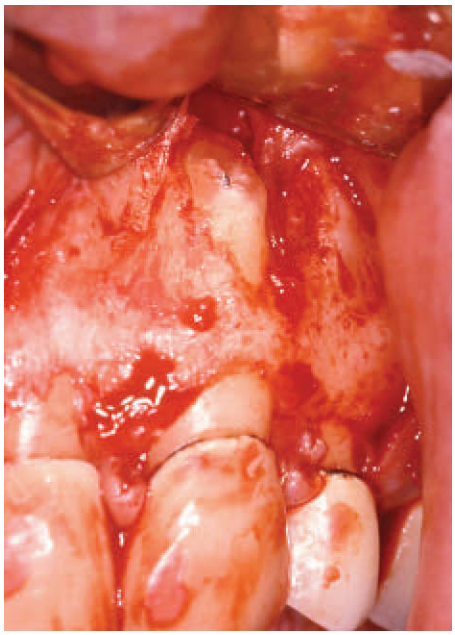


FIG. 25-53 If silver point retreatment is unsuccessful, apical surgery may be needed. Note the silver point visible on the resected root end. If the point cannot be pulled out in a retrograde direction, ultrasonic root-end preparation may be complicated by its presence; root-end preparation using rotary burs may be necessary.

occurs.[218] If the preoperative pulp was vital and uninfected (irreversible pulpitis, for example), and there was no apical periodontitis, the presence of the separated instrument should not affect the prognosis.[37] If the file can be removed without excessive overenlargement 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 affect 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. If symptoms persist, apical surgery or extraction should be considered.[218,240]

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 of critical importance. If the separated instrument extends into the straight coronal portion of the canal, retrieval is likely, but if the instrument has separated deep in the canal and the entire broken segment is apical to the canal curvature, orthograde removal will not be possible, and attempts to do so could lead to a much higher rate of iatrogenic complication.[184,195] 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 cur-

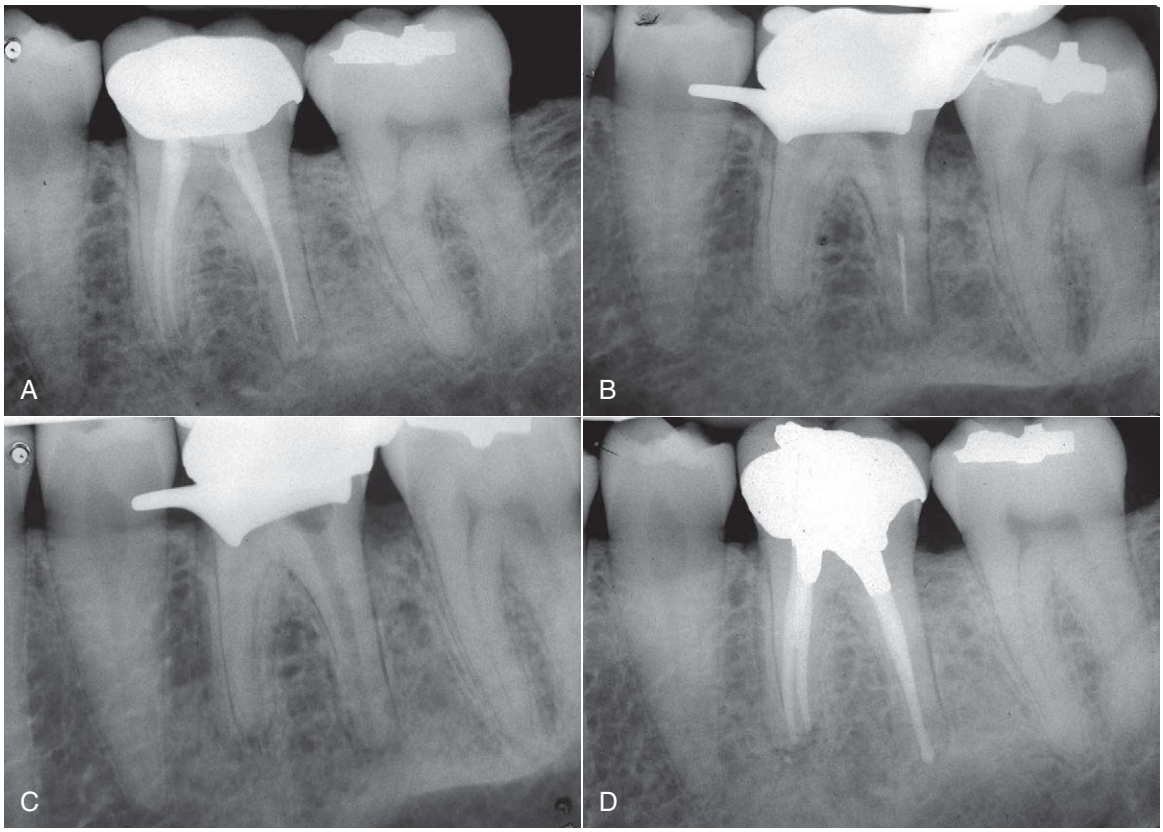


FIG. 25-54 A, Preoperative radiograph of a tooth with symptomatic posttreatment disease. **B,** Although not readily apparent on the preoperative film, there is a separated nickel-titanium instrument in the distal canal. **C,** Check film showing that ultrasonics has removed the separated file. **D,** Thirteen-month recall film. The patient was asymptomatic.

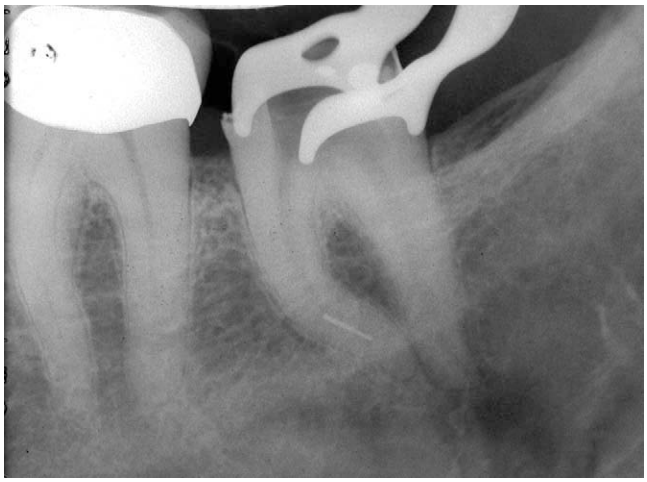


FIG. 25-55 Complicated canal anatomy can increase stress on rotary instruments, leading to separation such as in this S-shaped canal.

vatures, 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 energy is applied to them, hindering removal, whereas stainless steel instruments are more robust and more easily removed with ultrasonics.[163]

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. 25-56). This instrument will not only increase visibility by the use of magnification and light, 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, but the enhanced lighting and magnification the operating microscope offers has caused a quantum leap in visualization.[107] Many of the techniques to be described should not even be attempted without the use of this valuable tool.[205]

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 (Henry Schein, Melville, NY) (see

Fig. 25-37), these should be used to obtain a firm hold of the file and extract it out through the access cavity preparation. Many different 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. Unfortunately, many files separate at a point where these forceps cannot be used.

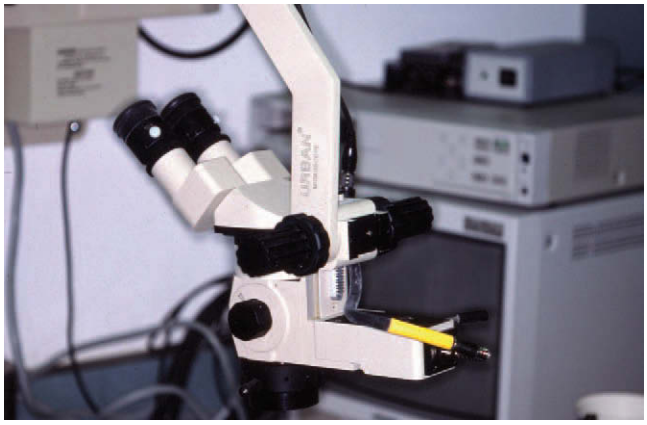


FIG. 25-56 The surgical operating microscope is not only invaluable in helping to remove separated instruments, it is in fact a necessary tool for these procedures.

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. 25-57).[163] A recent study showed that use of a similarly modified Lightspeed nickel-titanium rotary instrument (Lightspeed Technology Inc, San Antonio, TX) created a staging platform that was more centered in curved canals than the Gates-Glidden drills.[99]

Ultrasonic instruments have been shown to be very effective for the removal of canal obstructions.[33,138,163] 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

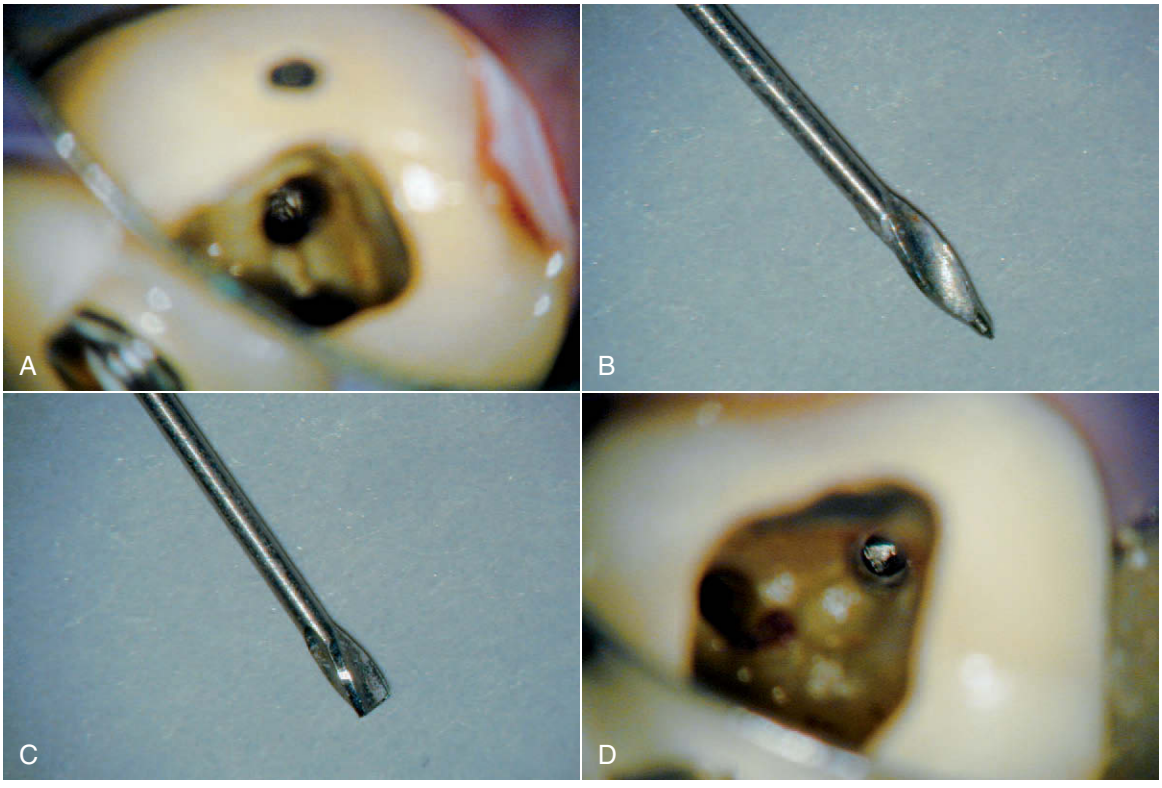


FIG. 25-57 A, Separated instrument in the mesiobuccal canal of a molar. **B,** Unmodified Gates-Glidden drill. **C,** Modified instrument. The tip has been ground off to the maximum diameter of the cutting head. **D,** Staging platform created in the straight coronal section of the canal. Note the enhanced visibility and the triangular cross-section of this rotary instrument.

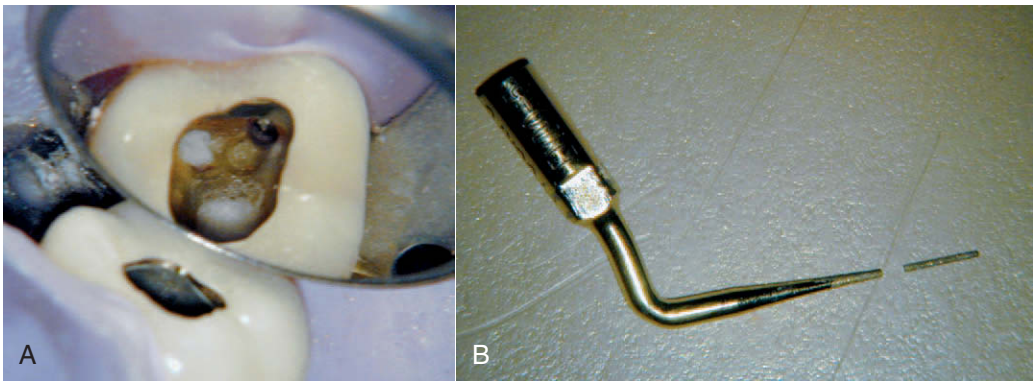


FIG. 25-58 A, Cotton pellets protecting the orifices of the other canals when ultrasonic obstruction removal is needed. **B,** Ultrasonic tip separated during excavation around separated instrument. If the adjacent canals were not protected, further unnecessary complication could result.

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[163] (Fig. 25-58). There are many different sizes and angles of ultrasonic tips 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. 25-59). 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.[87] As mentioned previously, nickel-titanium instruments often break up into fragments when subjected to the energy supplied by an ultrasonic instrument. The clinician may be tempted to use this information to their advantage by applying the tip of the ultrasonic directly onto nickel-titanium files. Although this method may work, 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).[163] 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[204] (Fig. 25-60).

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 both the beveled end and the opposite 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, then the complete assembly is withdrawn from the canal.[161] Occasionally, a larger diameter tube and thinner 0.11-mm ligature wire will facilitate assembly of this extractor (Fig. 25-61).

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 principles with slight variations from one another, including the Endo Extractor (Brasseler USA Inc), the Masserann Kit (Medidenta International Inc), and the Extractor System (Roydent Dental Products) (see Fig. 25-51).

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 mm 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 minutes for a loose fit.[65,198] One disadvantage of this instrument is that the trephine burs are much larger than their ISO equivalents, so the manufacturer has also added a separate smaller trephine bur that is sold separately from the kit. This bur corresponds better with the smaller extractors and removes less dentin, which can decrease the likelihood of weakening the root and the risk of fracture.[52] Another disadvantage is that the burs cut very aggressively when new but dull rather quickly. When new, their aggressive cutting may lead to perforation or even separation of the obstruction, so great care is needed when using this instrument (Fig. 25-62). Once the separated instrument has been removed, the extractors may be reused, either by using the debonding agent included in the kit to remove the embedded instrument from

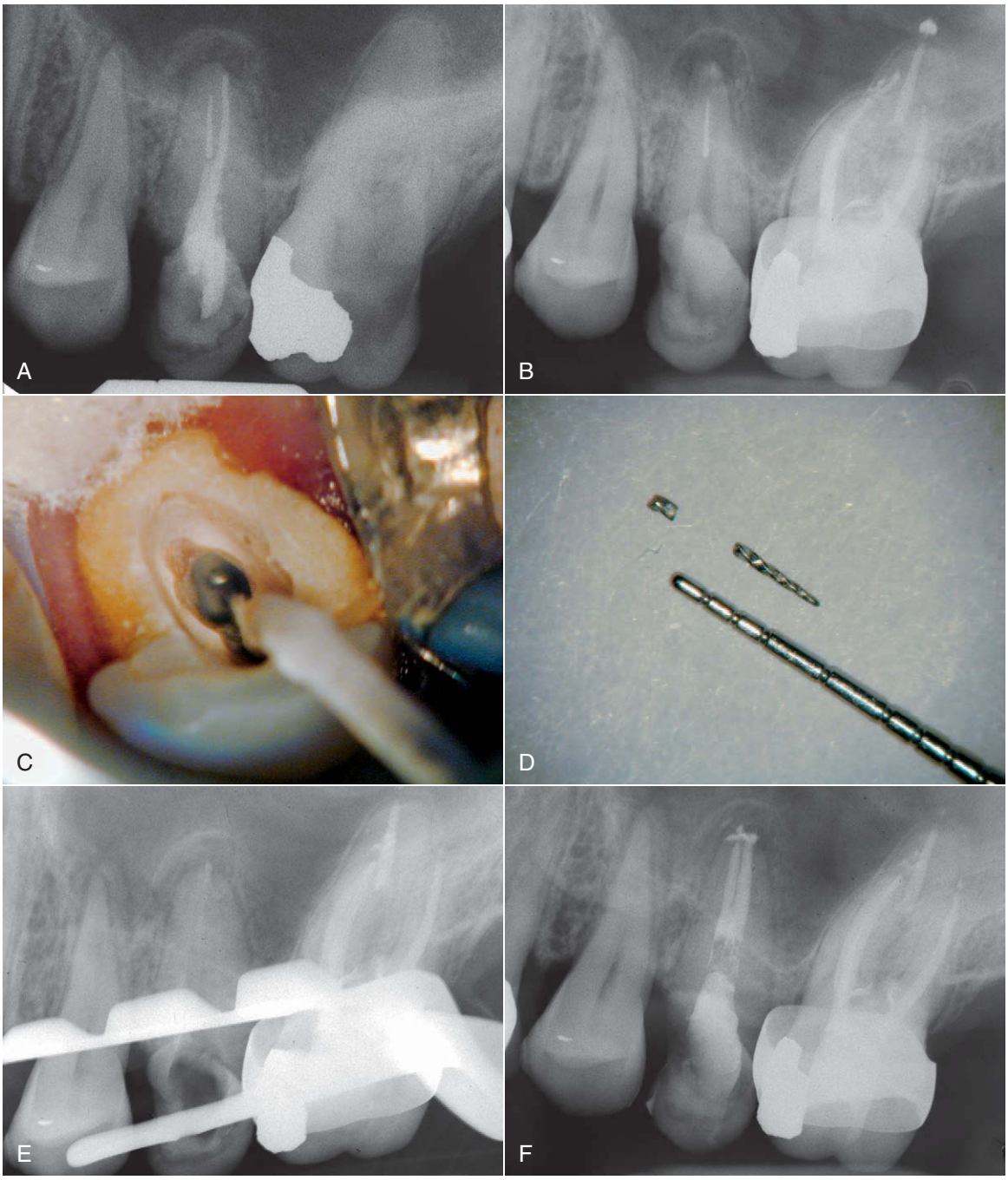


FIG. 25-59 A, Preoperative radiograph showing a separated file in the palatal canal, potential coronal leakage, and apical periodontitis. **B,** Check film showing the separated instrument after gutta-percha removal. **C,** Photograph showing the separated instrument in the palatal canal and a paper 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.

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.[131] This technique is similar to the Endo Extractor in that it uses trephine burs and a specific extraction device. This kit comes with a very convenient gauge that aids in predicting the size of the bur and the extractor to be used, and it contains many different 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,[58] leading to root weakening and

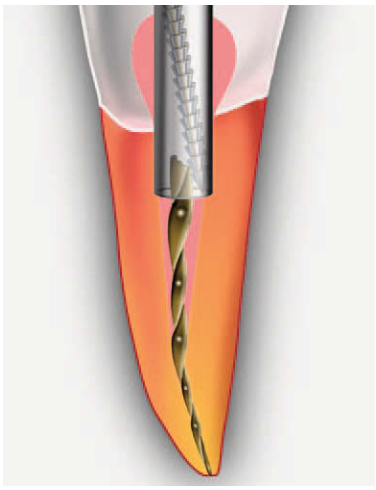


FIG. 25-60 Tube and Hedstrom file removal technique. The tube is slipped over the obstruction, and a Hedstrom file is gently screwed into the space between the tube and the obstruction. Pulling the tube and Hedstrom file together can withdraw the obstruction. *(Diagram courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

the risk of perforation[241]; 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 and removes a minimal amount of tooth structure, enabling access to the obstruction. The extractor tubes are also quite small and 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. 25-63). 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: the Cancellier instrument and the Mounce extractor (SybronEndo) (Fig. 25-64). The Cancellier instrument works in a very similar manner to the Brasseler Endo Extractor

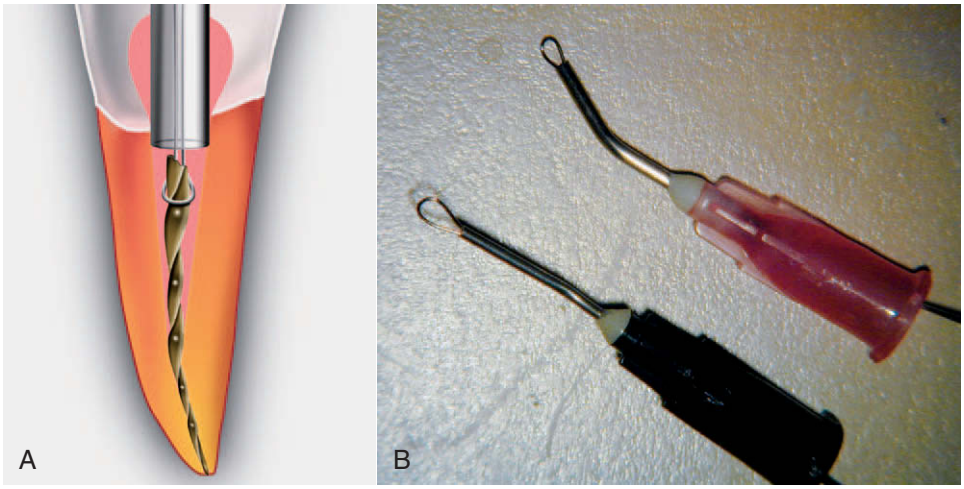


FIG. 25-61 A, Diagram illustrating the wire-loop-and-tube method of obstruction removal. The wire loop is carefully placed around the obstruction, tightened, then removed. **B,** Larger-diameter tubes and smaller-diameter 0.11 mm ligature wire enhances the efficiency of this technique. *(**A** Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

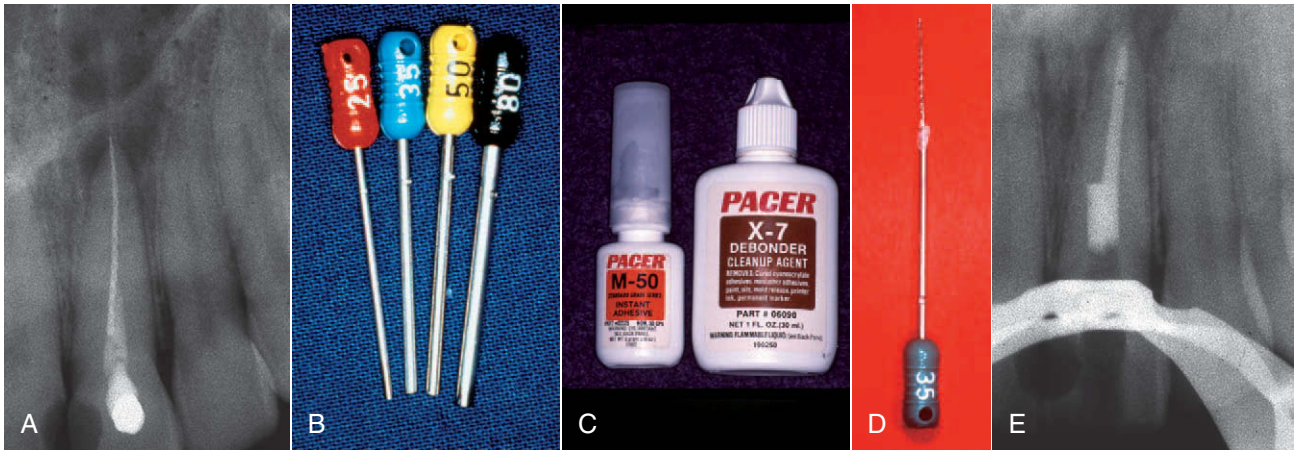


FIG. 25-62 A, Separated file wedged into an upper incisor. **B,** Brasseler Endo Extractor tubes. **C,** Cyanoacrylate cement and debonding agent. **D,** Separated file pulled out by the bonded tube. **E,** Final obturation. Note the excessive amount of tooth structure removal by the trephine bur that was needed to bond the tube.

in that they are 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. There are four sizes of extractor tubes available, each of which corresponds to specific file sizes. The Mounce extractors are also hand instruments that enable usage 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, but the ball tip is relatively large and is only useful in retrieving instruments that are in the most accessible coronal portion of the canal.



FIG. 25-63 Close-up view of the Roydent Extractor tip. The tip is placed over the separated instrument and tightened to grasp the obstruction.

Another device designed specifically for the purpose of separated file removal is the Instrument Removal System (DENTSPLY Tulsa Dental, Tulsa, OK) (see Fig. 25-51). 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 usage 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

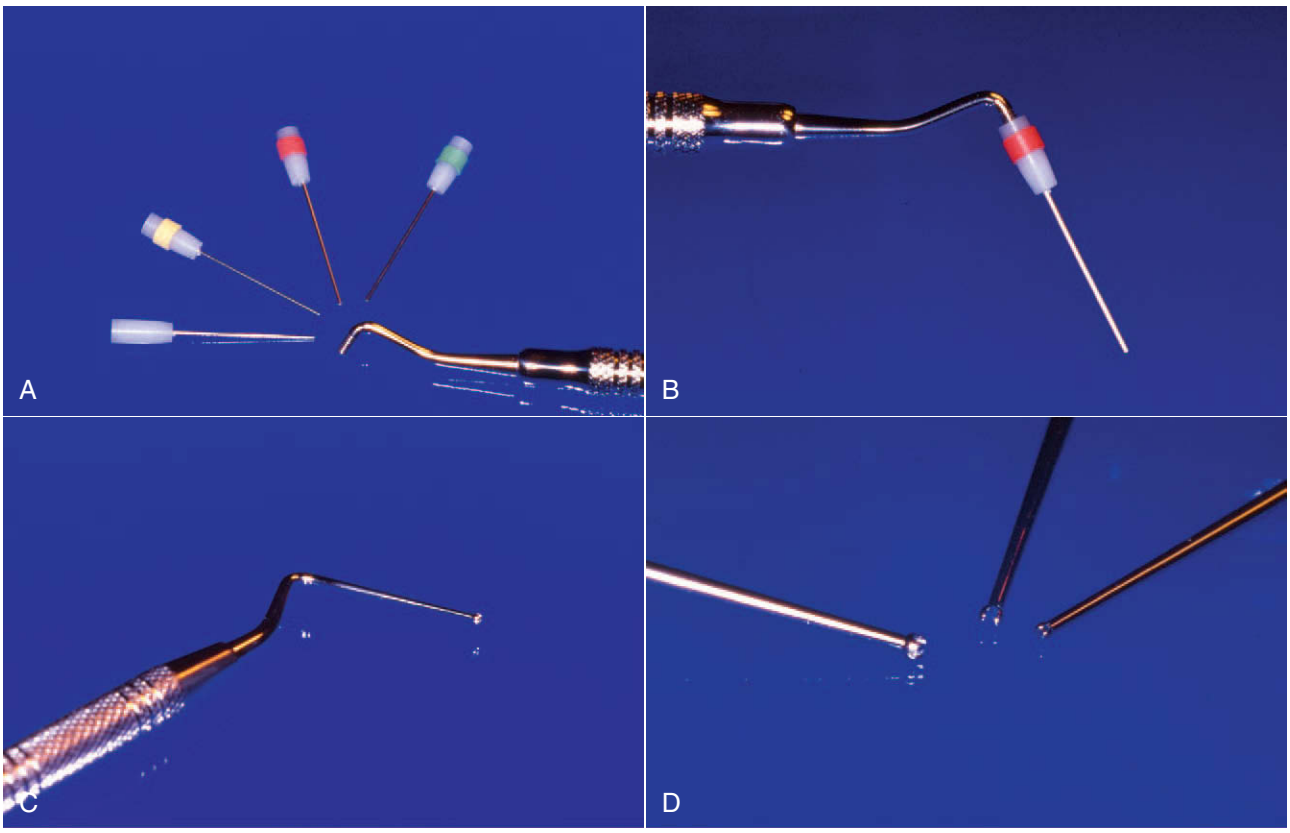


FIG. 25-64 A, The Cancellier Kit (SybronEndo, Orange, CA) with four tube sizes available. **B,** The Cancellier instrument is used with superglue to bond the obstruction, but its design allows for greater visibility during use. **C,** The Mounce instrument. **D,** Varying tip sizes for the Mounce instrument.

the side window. The assembly is then removed.[163] This instrument is very 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. 25-65).

The S.I.R. (Separated Instrument Retrieval) System (Vista Dental Products) (see Fig. 25-51) 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. If the clinician has access to grasp the extractor with their 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[115,121] and use of ultrasonics to dislodge posts[168] and separated instruments[81,126] 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.[9,45,170,171]

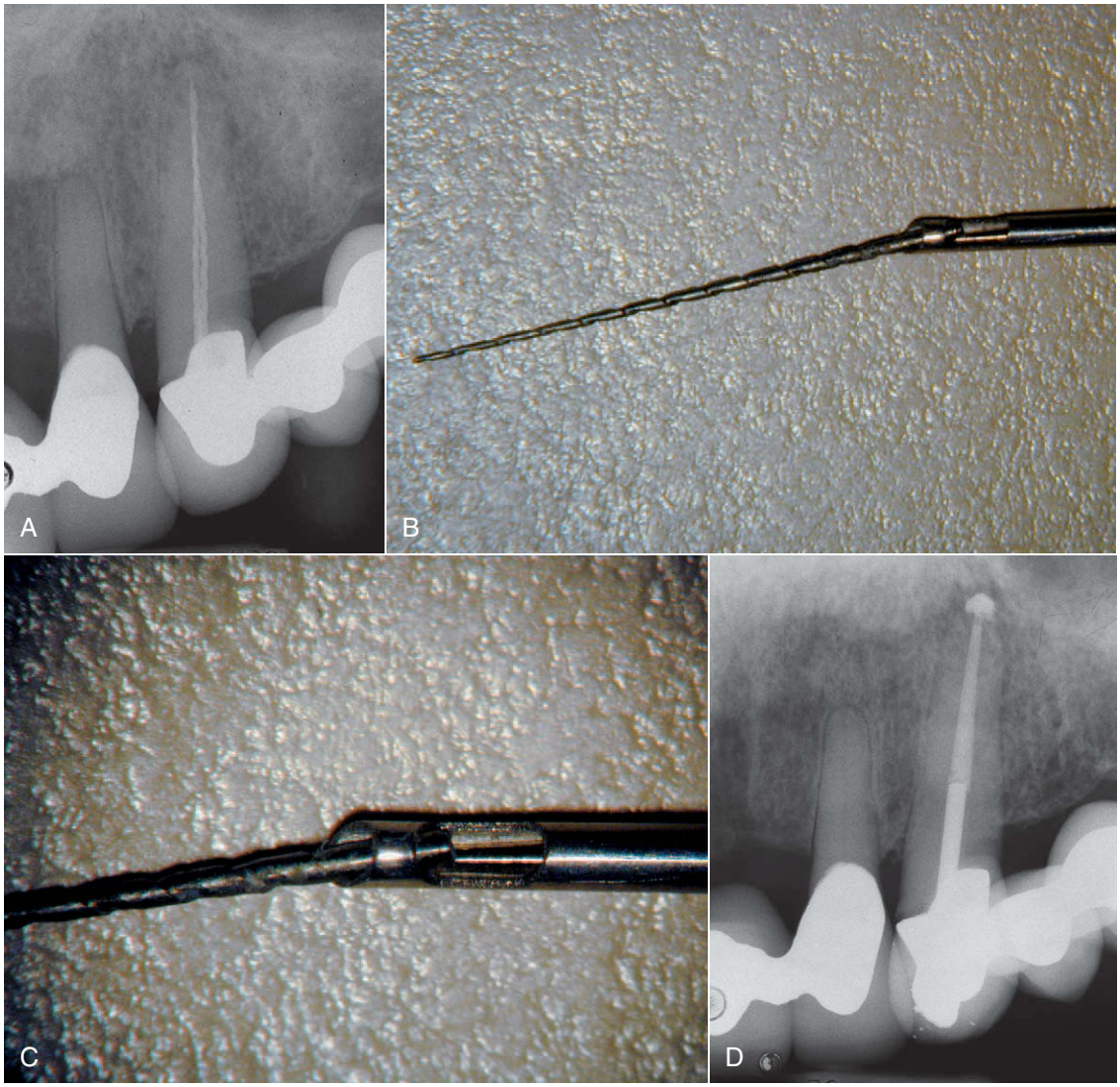


FIG. 25-65 A, Preoperative radiograph showing two separated instruments in one tooth. **B,** IRS instrument with a removed file. **C,** Note that large-size files are difficult to push through the cutout window. **D,** Postoperative radiograph.

The greatest danger of heat-related damage occurs with the use of ultrasonic energy to dislodge foreign objects in the canal space to gain access to the apical portion. As noted earlier, this has recently become an extremely important and useful part of the clinician's armamentarium because of 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. In vitro research and clinical case reports imply that they have the potential to damage the tooth attachment apparatus as a result of the heat generated during usage.[27,67,177] 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.[41] Thermal damage to the periradicular tissues may be so serious as to result in both tooth loss and permanent bone loss (see Fig. 25-26). This is not to say that ultrasound energy should be avoided for the removal of canal obstructions, since 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 to establish heat-reduction protocols based on these variables are needed. Some have proposed that the dentin thickness between the outer surface of the post and the root surface may affect root surface temperature rise,[68,96] and one study showed this.[168] However, a more recent study has shown that the dentin thickness is statistically insignificant as a factor in root surface temperature rise.[86] 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 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 use of ultrasound energy for post removal is time dependent.[27,91] Studies have suggested that cooling the ultrasonic tips can greatly reduce heat buildup,[27,91] but this reduces the efficiency of debonding resin-bonded posts.[41,62] The amount of time the clinician can use these instruments safely is very difficult to determine; specific evidence-based protocols using in vivo research have yet to be established. The authors feel that 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 regards 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.[46]
- Take frequent breaks to let the tooth cool down.
- Avoid using the ultrasound on the high power setting.[46]

Prudent clinicians must take extreme care when applying ultrasound energy to a canal obstruction. It has been shown that even with the use of water coolant, root surfaces can increase in temperature rapidly.[41,168] Until evidence-based heat-reduction protocols are developed, caution when using instruments that can generate heat in the periodontal ligament is required.

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.[163] 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.[75,100] 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 reader is referred to Chapter 9 for strategies to prevent blocks and ledges. 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.[57] This should not deter the clinician from choosing nonsurgical retreatment, however. In a recent 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.[48] 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 Technology), the Profile or GT instruments (DENTSPLY Limited), or the K-3 instrument (SybronEndo) 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 silicone stop should be used so the clinician knows in which direction the tip of the instrument is pointing, which helps in visualizing the three-dimensional layout of the canal

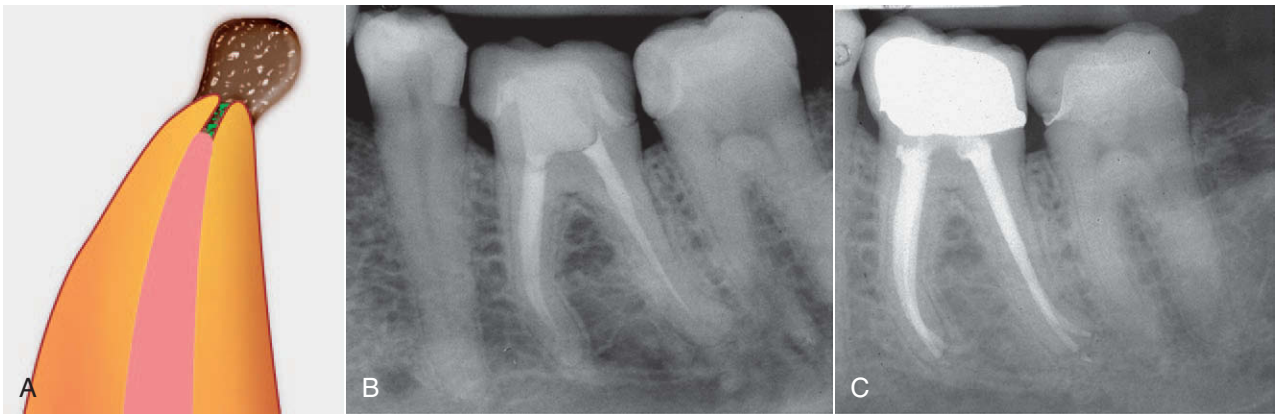


FIG. 25-66 A, Diagrammatic representation of a canal block. Fibrotic and/or calcified pulp and debris that is potentially infected remains in the apical segment of the canal when the canal is instrumented short of the apical constriction. **B,** Preoperative radiograph showing obturation short of ideal length. The patient was symptomatic, and the canals were blocked. **C,** Three months post treatment. Treatment took a total of 3.5 hours over three appointments because of the time-consuming nature of bypassing blocked canals. *(**A** Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

system. Frequently, evacuating the irrigant and using a lubricant such as RC Prep (Premier, Plymouth Meeting, PA) or Pro-Lube (DENTSPLY Tulsa Dental) 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"), the clinician should continue to peck at the sticky spot until further apical advancement is accomplished.[163] 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 (DENTSPLY Maillefer), but there is a risk of deviating from the original canal path, creating a ledge, and ultimately a false canal leading to zip perforation.[75] 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.[163] 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 very gentle reciprocal rotational motion ("twiddling") will aid in 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.[129] 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. 25-66).

If after a reasonable amount of time, no sticky spot can be found, the clinician must consider the possible presence of a

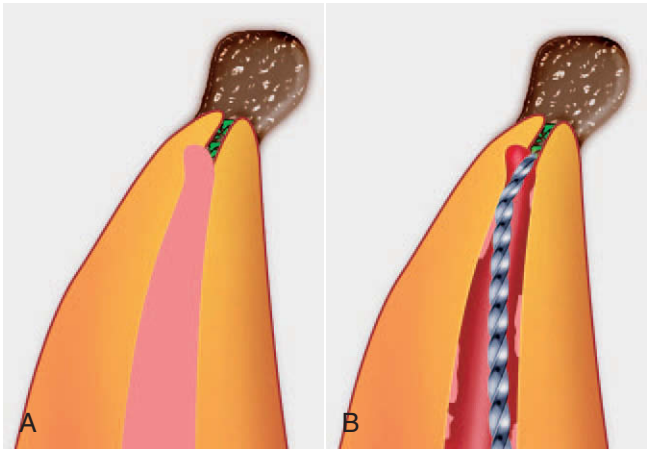


FIG. 25-67 A, Diagrammatic representation of a ledged canal. Potentially infected debris remaining in the apical segment can result in posttreatment disease. **B,** Attempting to bypass the ledge with a small file having a 45-degree bend in the tip. Note that the opening to the apical canal segment is on the inside of the canal curvature and coronal to the level of the ledge. *(**A** Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*

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, whereas finding the original canal is many times impossible. Ledges feel like a hard brick wall, short of length when encountered, and care must be used to prevent worsening them by indiscriminately burrowing into them.[218] 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,[100,218] 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. Since 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. 25-67). The file tip is slowly scraped along the internal wall of the canal curve slightly coronal to

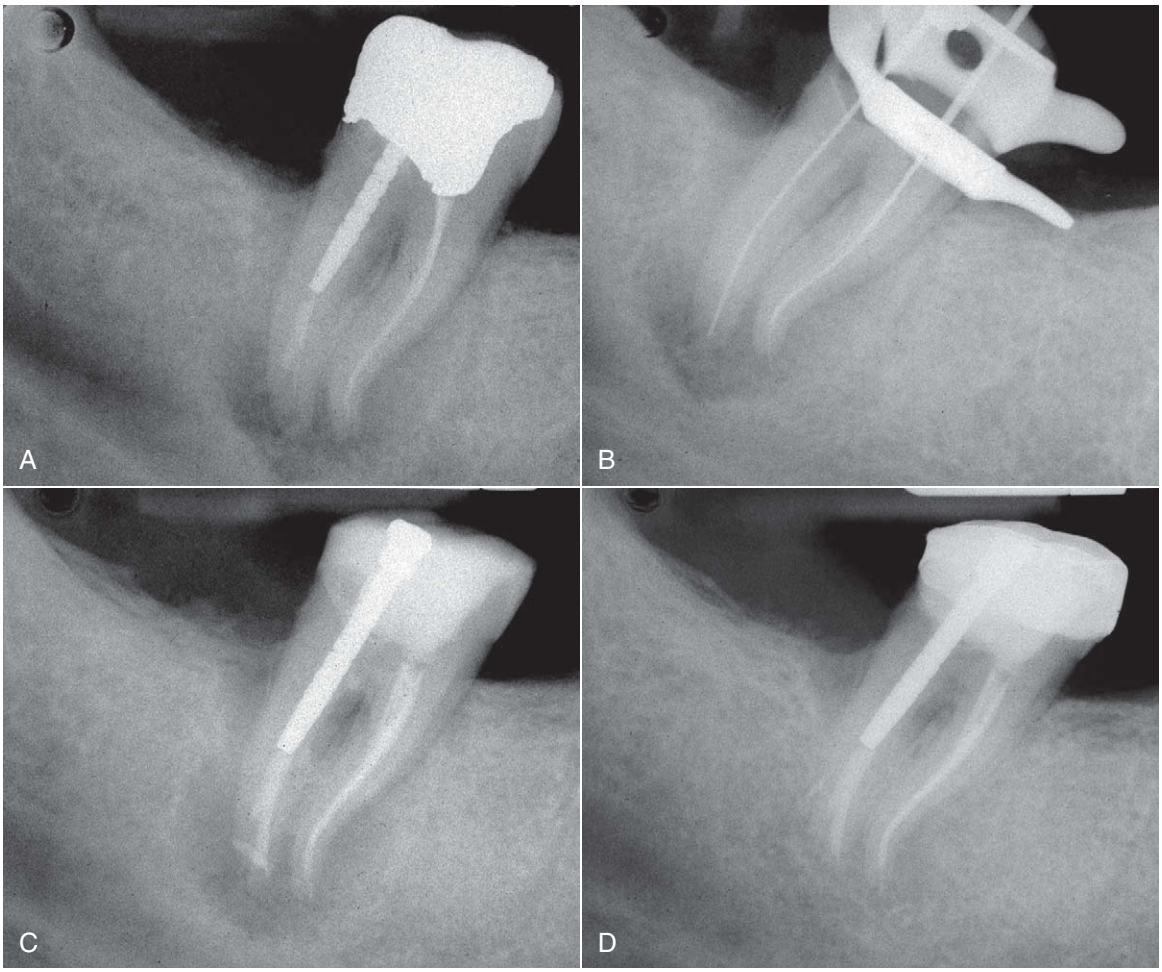


FIG. 25-68 A, Preoperative radiograph showing a distal canal ledge with a small amount of sealer that has entered the apical segment. The ledge prevented proper cleaning and sealing of the canal system, resulting in posttreatment disease. **B,** The ledge has been bypassed. The attempt is made to blend the ledge into the contour of the prepared canal wall. **C,** Final obturation showing the filled ledge and apical segment. **D,** Thirteen-month recall showing healing. The patient was then directed to have a definitive coronal restoration placed.

the level of the ledge,[218] looking for 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 file can be easily negotiated around the ledge, anticurvature filing will enable the clinician to blend the ledge into the canal preparation (Fig. 25-68). Many times this cannot be completely accomplished,[218] but so 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) for the blending of ledges has been advocated.[163] 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 the tip of the GT file can passively follow this glide path beyond the ledge. The GT file must have 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 since 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 instrument's 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. 25-69).

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. 25-70). If symptoms of posttreatment disease arise subsequently, apical surgery or extraction will be needed.[163,218]

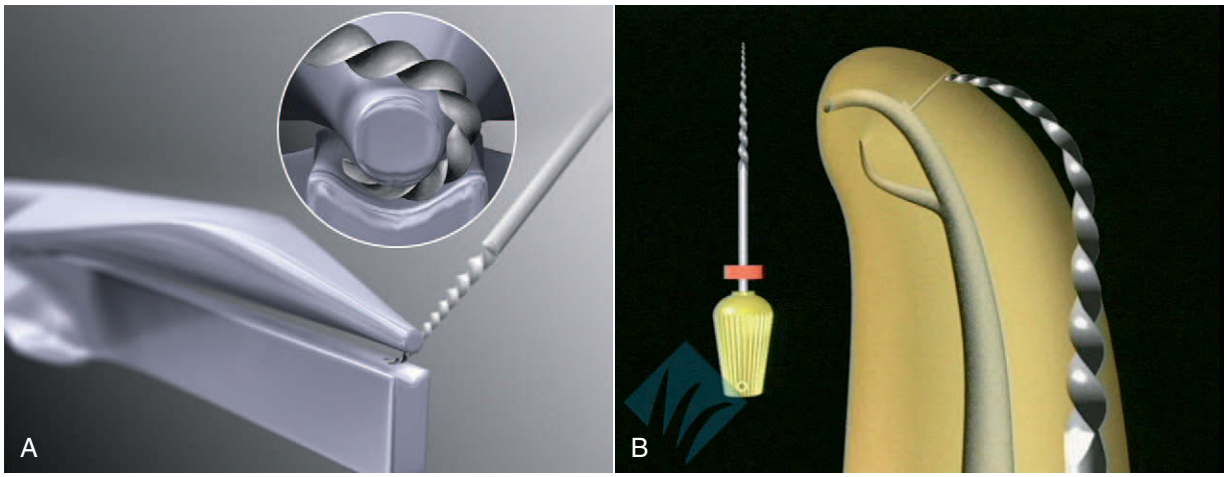


FIG. 25-69 A, Endobender Pliers (SybronEndo) used to over bend a nickel-titanium Hand GT file (DENTSPLY Tulsa Dental). **B,** GT hand file can hold a bend to allow it to bypass ledges. *(Courtesy Dr. Steve Buchanan.)*

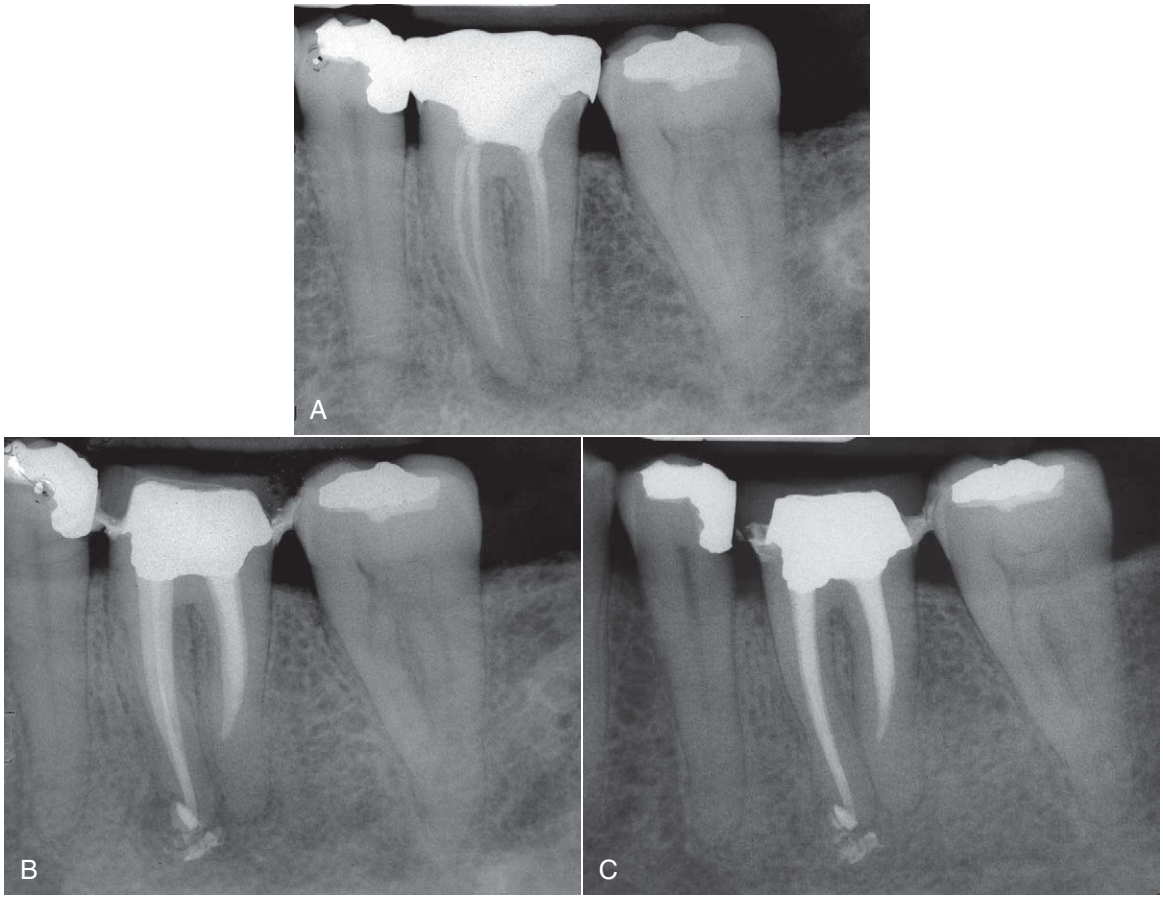


FIG. 25-70 A, Preoperative radiograph showing mesial canal blockages and potential distal root ledging with accompanying posttreatment disease. **B,** Final film showing mesial blockages bypassed but inability to negotiate beyond the distal ledge. The patient elected to pursue no further treatment at this time. **C,** One-year recall. Despite not achieving all 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.

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, microexcavation techniques, and most importantly, the knowledge of canal anatomy that is discussed in another section of this text (Fig. 25-71). 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, keeping the goals of the retreatment procedure in mind. These matters are covered in detail in Chapter 9.

Canal disinfection procedures are, however, paramount after the completion of cleaning and shaping. Since the primary cause of posttreatment disease is usually microbial,[140,218] and these microbes (i.e., *Enterococcus faecalis*) are frequently resistant to traditional canal disinfection regimes,[14] every effort must be made to eliminate these organisms from the canal system. This effort is complicated by the fact that no instrumentation regime can predictably remove the entire previous root filling from the canal space after retreatment.[10] 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 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.[96] Root perforations are created pathologically by resorption and caries and iatrogenically

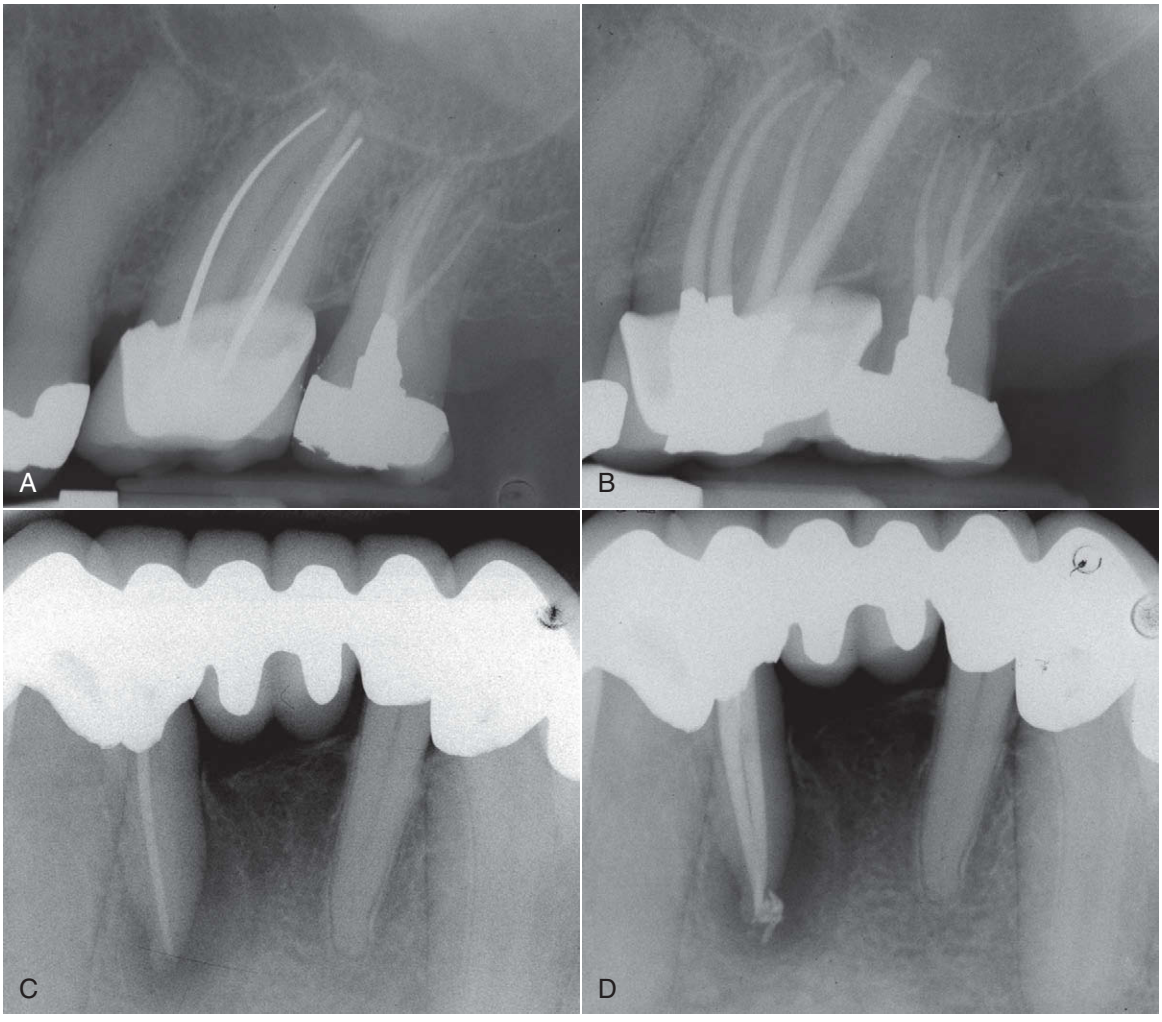


FIG. 25-71 Frequently missed canals that may result in posttreatment disease. **A,** Missed second mesiobuccal canal (MB2) in an upper molar. **B,** Final obturation showing cleaned, shaped, and filled MB2 canal. **C,** Missed lingual canal on a lower incisor results in posttreatment disease. **D,** Immediate postoperative film showing management of the missed canal.

during root canal therapy (zip, strip, and furcation perforations) or its aftermath (e.g., post preparation perforation)[160] (Fig. 25-72). 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 radiographically to have left 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[128,182] (Fig. 25-73). 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, either nonsurgically by approaching the defect internally through the tooth, or surgically by using an external approach

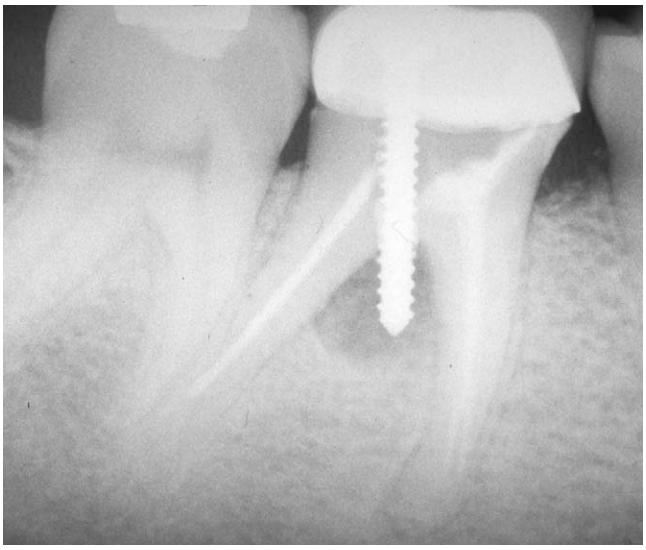


FIG. 25-72 Furcal post perforation resulting in persistent infection and furcal bone loss.

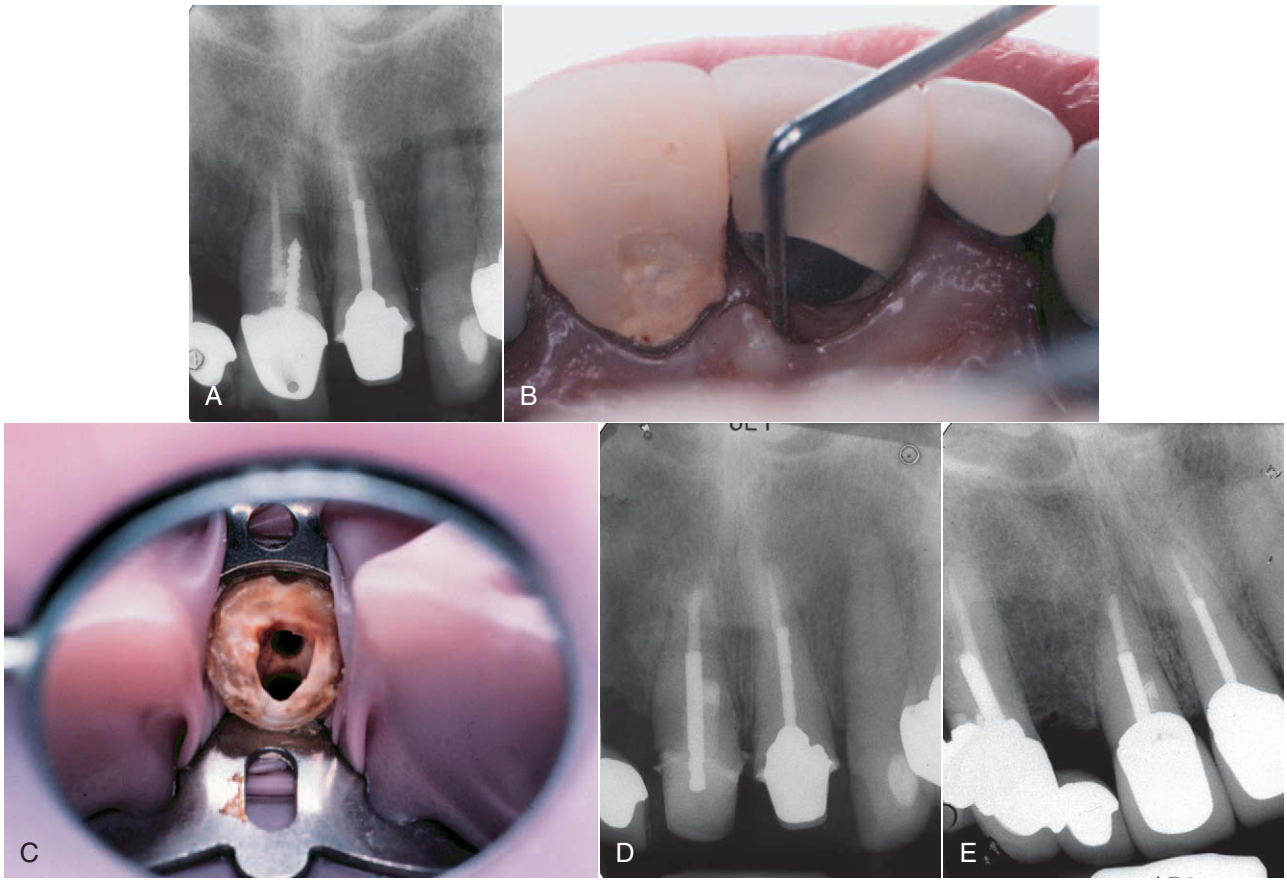


FIG. 25-73 A, Mesially angulated preoperative radiograph showing a palatally oriented post perforation in an upper incisor. **B,** An 8-mm narrow-based probing defect on the mesiopalatal corner of the tooth. **C,** Following coronal disassembly, the true canal can be seen lying in a facial direction 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,** Three-year reevaluation. The tooth is asymptomatic, and mesiopalatal probing depth is 4 mm.

through the periradicular tissues.[160] In general, if all other factors are considered equal, internal nonsurgical perforation repair will be the preferred method, since it is usually less invasive, produces less destruction of periradicular tissues via the surgical access wound needed, and isolation from microbes and disinfection is usually enhanced. 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. 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.[163,229]

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.[112,182,188] Generally, the more apical the perforation site, the more favorable is the prognosis, but 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 third of a straight canal (access perforation), it is considered easily accessible. If it is in the middle third of the canal (strip or post perforations), difficulty increases, and in the apical third of the canal (instrumentation errors), predictable repair is most challenging; frequently apical surgery will be needed.

Immediate repair is better than delayed repair, since delay can cause breakdown of the periodontium, resulting in endoperio lesions that are difficult to manage,[110,186] and elimination of microbial contamination of the defect and sealing it properly are critical to success. Many materials have been advocated for the repair of perforations in the past, but 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 (ProRoot MTA, DENTSPLY Tulsa Dental)[163] (Fig. 25-74).

Since the recent introduction of MTA for perforation repair, the choice of which repair material to use is more clear.[128,160] MTA has many advantages over other restoratives when being used for perforation repair. This material seals well[144,239] even when the cavity preparation is contaminated with blood.[214] It is very biocompatible,[149,155,215,217] 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.[85,155] MTA has also been shown to have a high degree of clinically favorable long-term outcomes when used as a perforation repair material.[128,160] The main disadvantage of MTA is the long time required for setting[216] that makes this material inappropriate for transgingival defects such as those associated with cervical 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, Santa Maria, CA) is recommended for lesions that cross the gingival margin[15,42,173] (Fig. 25-75). MTA is available in the original gray-colored formulation and a newer, more esthetic off-white color for treatments in the esthetic zone of the mouth, although there is little research on the differences between the two formulations. Their sealing ability seems comparable,[51] but questions remain as to whether white MTA exhibits the same biocompatibility[153] and will have the same long-term success as the older variety.



FIG. 25-74 ProRoot MTA is a medical grade of Portland cement that has had the arsenic removed so it can be used in the human body. It is the material of choice for endodontic perforation repair. *(Courtesy DENTSPLY Tulsa Dental, Tulsa, OK.)*



FIG. 25-75 Geristore Kit (Den-Mat, Santa Maria, CA). This and other resin ionomers have been advocated for cervical perforation repair, owing to good biocompatibility and the shorter, more controlled setting times that make them useful for transgingival root fillings.

If the perforation is to be repaired nonsurgically through the tooth, coronal-radicular access to the defect is prepared as stated previously (Fig. 25-76). 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

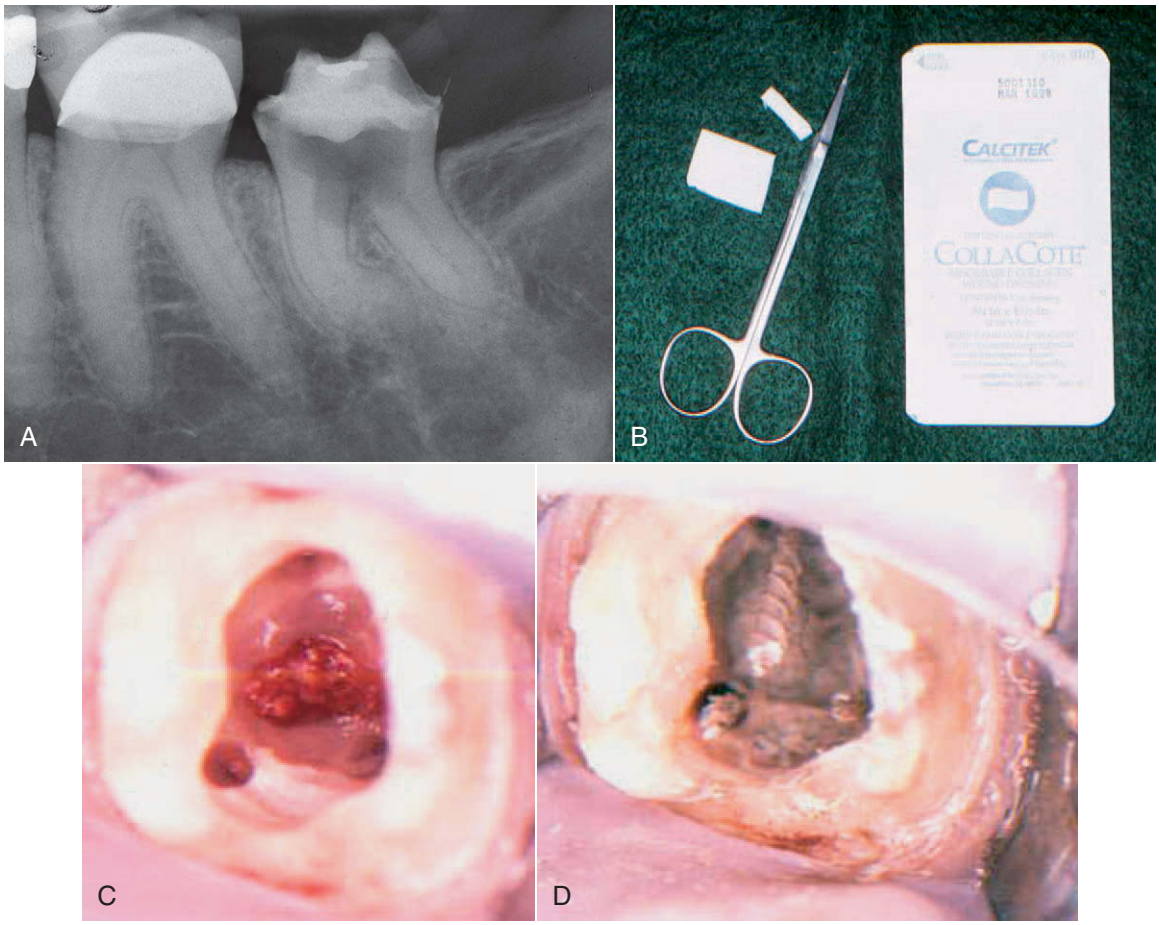


FIG. 25-76 A, Large furcal perforation created during an attempt at endodontic access. **B,** Colla-Cote (Integra Life Sciences, Plainsboro, NJ) to be used as an external matrix material to recreate the external root contour. **C,** Canals have been found, preliminarily instrumented, and the external 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.

the perforation is not so large as to allow the irrigant to significantly damage the periradicular tissues. If the perforation is large, sterile saline should be used as an irrigant, and disinfection of the margins of the defect is performed using mechanical dentin removal. Investigators[8] have advocated the use of copious flushing of the defect with 2.5% hypochlorite, but in light of the potential severe complications of hypochlorite overextension through a perforation,[63] 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) (see Fig. 25-76, *B*), calcium sulfate (Capset, Lifecore Biomedical, Chaska, MN), or calcium hydroxide[163]; however, astringents such as ferric sulfate should be avoided, since the coagulum they leave behind may promote bacterial growth and may compromise the seal of the repair.[117]

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, because removal of the files after placement of the repair material is very difficult, since the material tends to lock into the instrument's flutes (see Fig. 25-76, *F*). After protecting the canals, the perforation site is inspected to determine whether an external matrix is needed to ensure a proper contour of the restoration.[116] If the surrounding bone is closely adapted to the defect margins, minimal to no matrix material will be necessary. 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), hydroxylapatite, gelfoam, or calcium sulfate.[160,163] 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 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 compactor, since they can wick some of the water out of the material, giving it a firmer consistency and

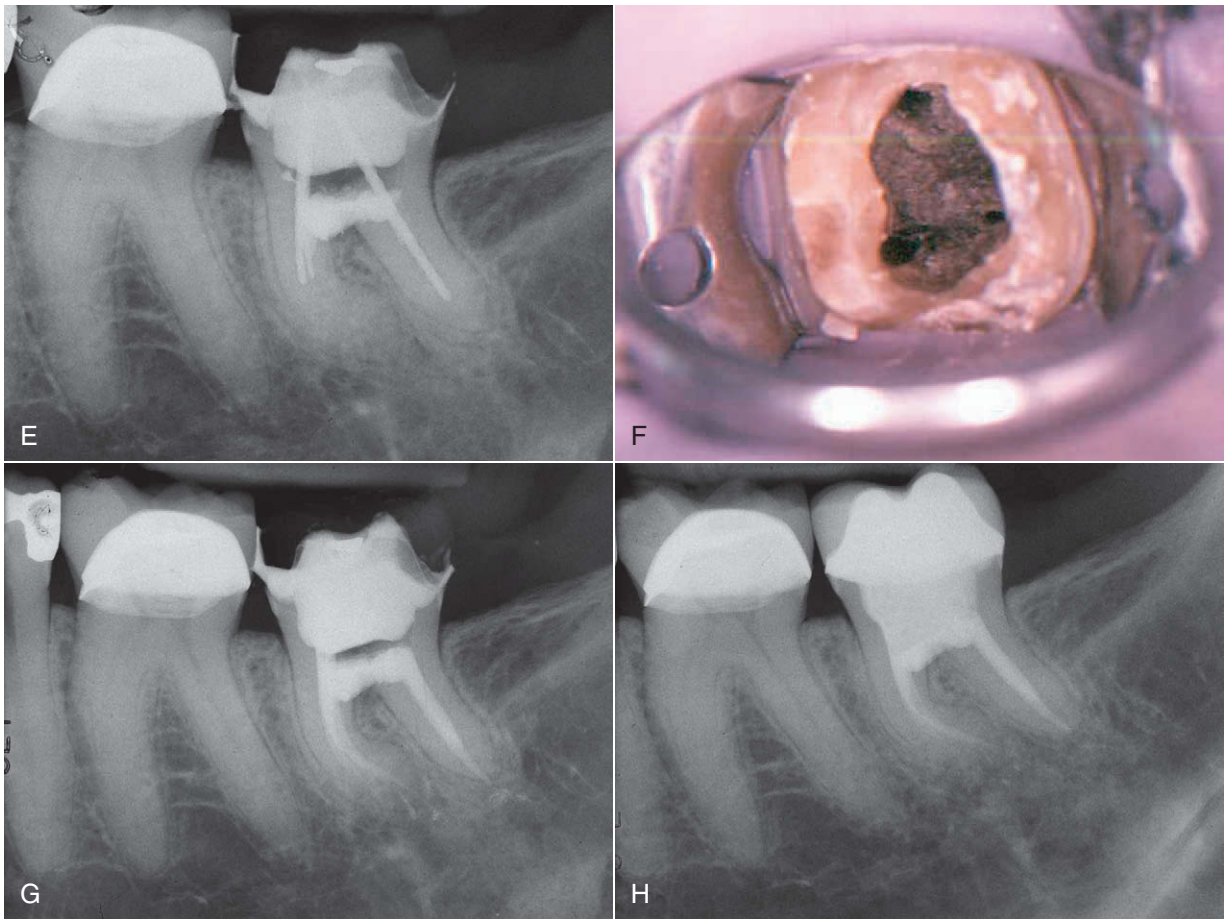


FIG. 25-76, cont'd E, Radiograph showing the initial repair with MTA recreating the vault of the furcation. **F,** On the second appointment, the blocking files are removed with difficulty, since the MTA has flowed into the flutes and set. At this time, endodontic therapy is completed as normal. **G,** Postobturation radiograph. Note the radiolucency in the furcal vault, which represents the Colla-Cote external matrix material. **H,** Nineteen-month reevaluation. The patient is asymptomatic, and there is evidence of healing in the furcal vault area.

aiding compaction. When the MTA has been placed, 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[192] (see Fig. 25-76, *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.[8,160]

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. 25-77). Protecting the canal from blockage is somewhat more difficult, and placement of the repair material requires the enhanced vision provided by the surgical operating microscope. Ideally, the canal should be fully shaped prior to the repair attempt,[163] and a canal patency protector should be placed apical to the defect. In some of these instances, protecting the canal can be accomplished by using a severed file, notwithstanding the previous warning, since not only can it protect the canal from blockage, 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 compaction is impossible. The file is placed into the canal to a level well below the defect, and the MTA is carried to place. Once compaction 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[163] (Fig. 25-78). There is some evidence that ultrasonic placement of MTA may enhance the seal against bacteria in an apexification model,[113] although other researchers have not agreed with this conclusion and found poorer canal wall adaptation when filling the apical extent of canals using ultrasonic compaction compared with hand filling.[6] Further study of ultrasonic MTA placement is warranted, but clinical observation suggests that this method has merit.

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, since 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.

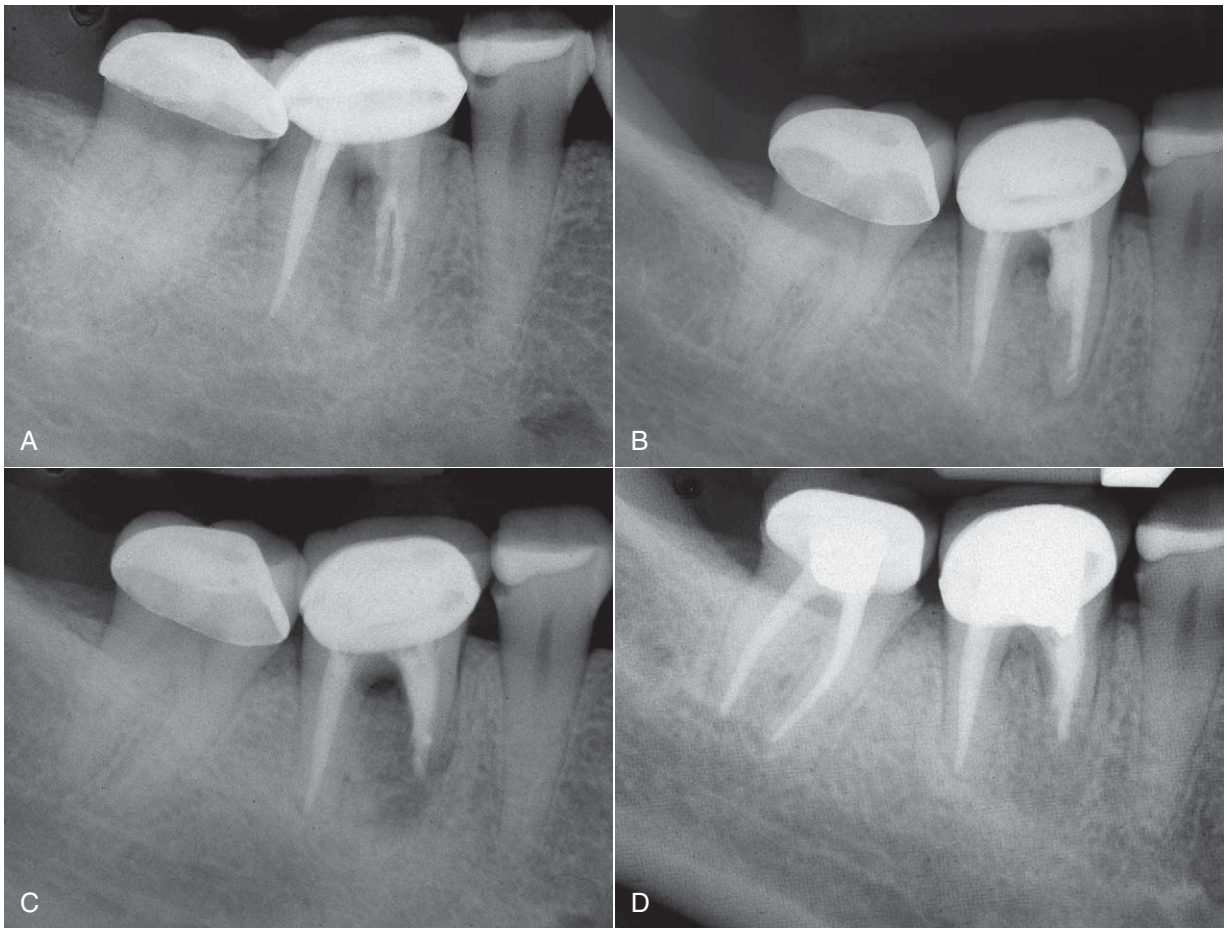


FIG. 25-77 Midroot-level perforation repair. **A,** Preoperative film showing mesial strip perforation with bone loss. **B,** Nonsurgical internal repair with inability to negotiate canals to the apical terminus and MTA overextension into the furca. **C,** Apical and perforation repair surgery performed. **D,** One-year follow-up showing complete healing.

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, 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. Generally, 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.

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. 25-79). The prognosis depends to a large extent on whether apical periodontitis exists prior to retreatment.[146] In a recent systematic review of outcomes studies,[55] investigators report 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 state 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."[55]

Unfortunately, these numbers mean that the desired outcome will not occur in potentially a fourth 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.

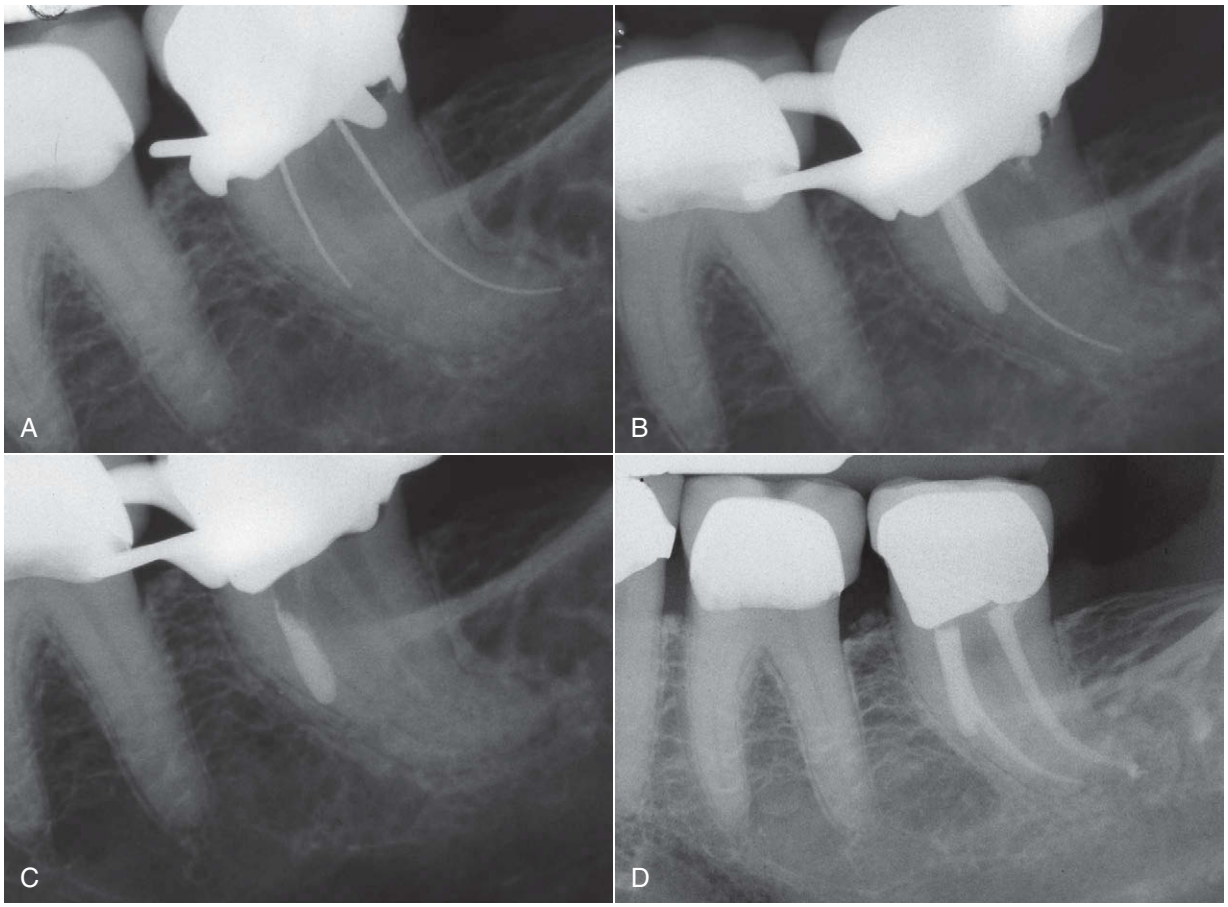


FIG. 25-78 A, This patient was in extreme pain following initial endodontic instrumentation by her dentist. Midroot-level perforation found on access. **B,** The original canal was found and protected with an endodontic file. MTA was vibrated using ultrasonic energy applied to the file, and it flowed into the defect. Then the file is 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 is withdrawn easily, since it was detached from the MTA repair material. Now the endodontic therapy is concluded as normal. The patient has been asymptomatic since the end of the first appointment. **D,** Follow-up at 27 months, showing complete healing.

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*.[55] 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 (e.g., progressive enlargement of a periapical 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.[134]

Endodontic surgery (Fig. 25-80) is a very predictable procedure[55,76] that can be performed on most teeth, but there are some anatomic and medical concerns regarding treatment planning for this procedure which are covered in detail in Chapter 21. Extraction-replantation (Fig. 25-81), also referred to as *intentional replantation*,[147] 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 another chapter. Extraction and replacement should be the treatment of last resort, to be selected only when the tooth has been shown to be irreparable. 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 vast 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, since 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.

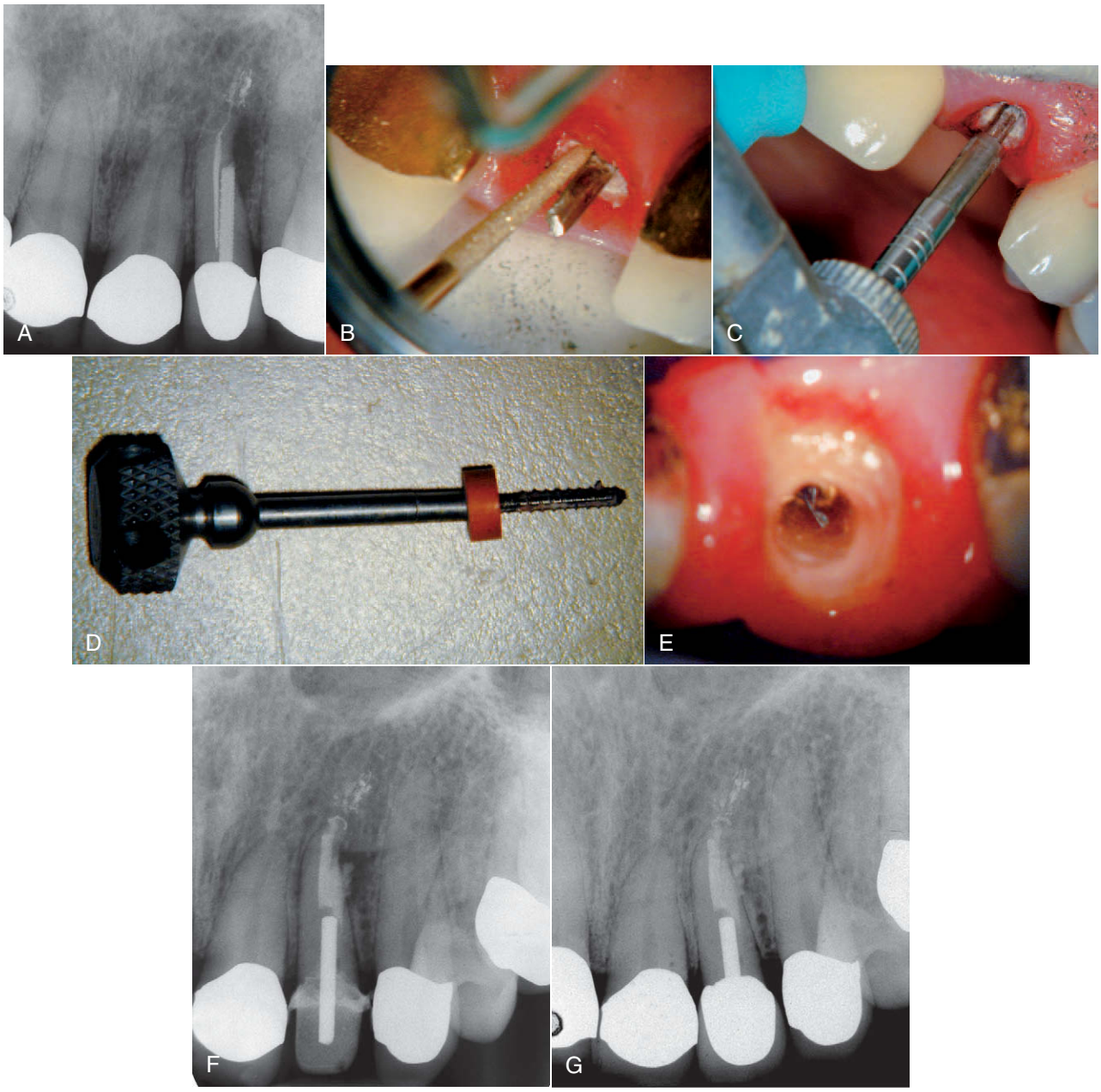


FIG. 25-79 A, Preoperative radiograph showing midroot-level post perforation and associated periradicular periodontitis. **B,** The crown was removed, and ultrasonic energy was applied to the post. **C,** Using the trephine bur from the Ruddle Post Removal kit to mill the endo of the post. **D,** The 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,** Thirteen-month recall showing healing around the perforation repair site.

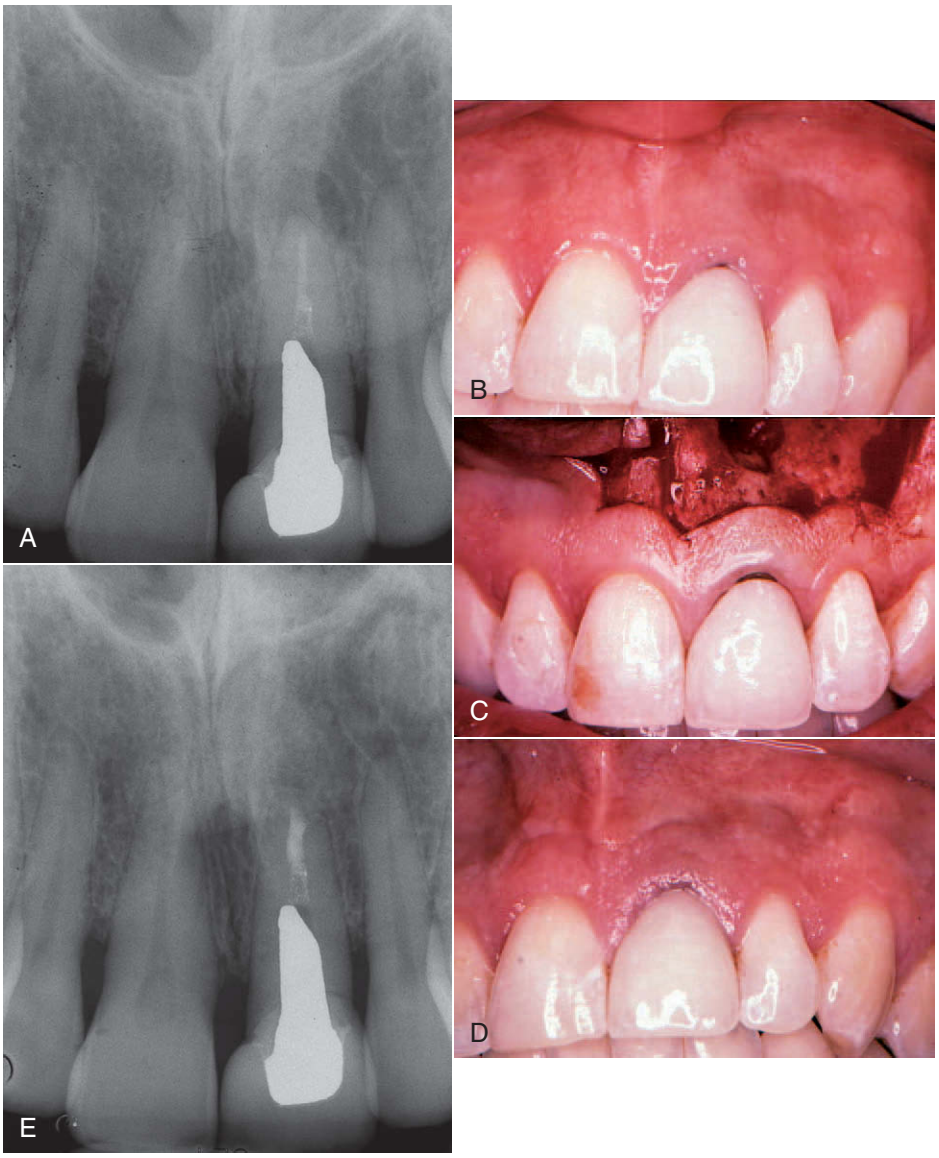


FIG. 25-80 A-B, Preoperative images showing posttreatment disease in the upper left central incisor and a large custom cast post. The patient elected to leave the post and perform surgery rather than risk damage to his new crown. **C,** Submarginal rectangular flap design. **D,** Three-week follow-up showing excellent soft-tissue healing. **E,** Eighteen-month follow-up showing excellent healing of the periradicular tissues.

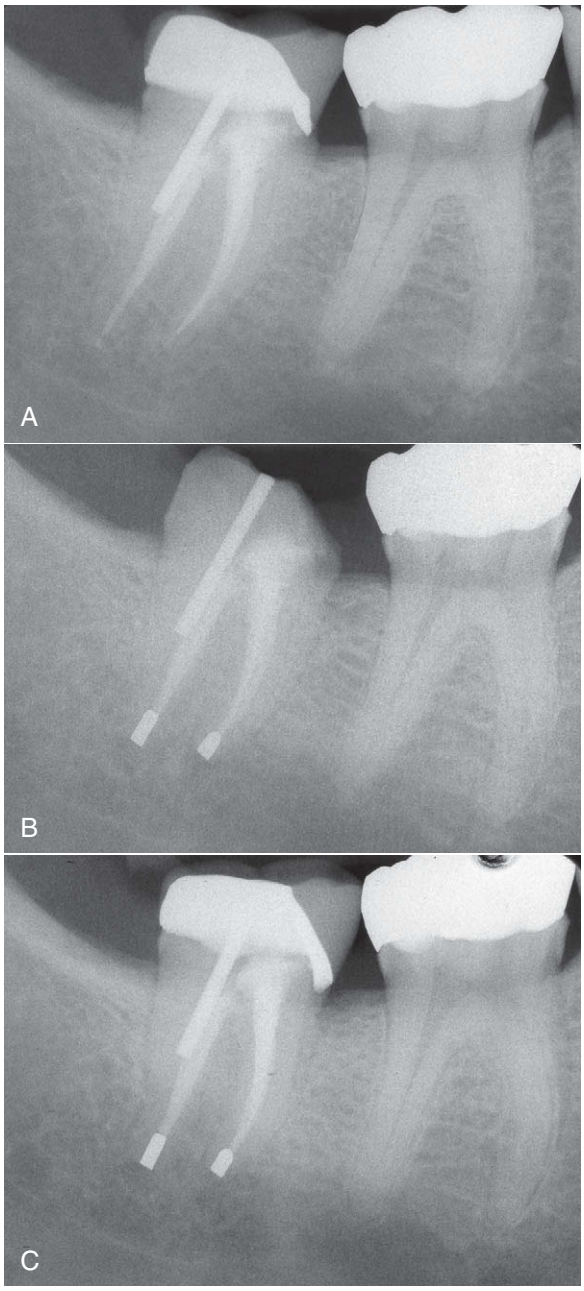


FIG. 25-81 A, This lower second molar remained symptomatic despite nonsurgical retreatment. Surgery was precluded by the poor access and proximity of the inferior alveolar canal. **B,** Intentional replantation was performed using amalgam alloy retrograde fillings. Better materials such as MTA or Super-EBA are available now. **C,** Fifteen-month reevaluation showing apical healing. The patient was asymptomatic.

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CHAPTER 26

Understanding and Managing the Fearful Dental Patient

HENRIETTA L. LOGAN

CHAPTER OUTLINE

ANXIETY AND FEAR
MECHANISMS
INTER- AND INTRAVARIABILITY IN RESPONSES
Variability in Stress Response
Variability in Pain Response
MANAGEMENT OF PAIN AND ANXIETY
Patient Communication
Behavioral Interventions

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 rushed 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."

The scenario is familiar to many people. The mere act of making a dental appointment requires greater courage than is available, and except for the moments when oral pain becomes unbearable, pursuing dental treatment is out of the question.[78]

Many fears are regarded as socially unacceptable. The presence of dental fear, however, is widely accepted and carries little social stigma. Several studies confirm that dental anxiety and fear are common among the general U.S. population, as well as being a worldwide problem.[68,69,74] About 50% of adults in the United States report some dental fear, with 8% to 12% fearful enough that simply making a dental appointment is problematic.[82] Although women account for more than half of fearful patients, clinicians should not assume 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.[34] 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/fear has remained stable during the past 50 years.[81]

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%,[32] whereas in Iceland, Taiwan, and Japan, adult respondents who report at least some dental fear are 21%, 50%, and 82%, respectively.[26,72,75] Studies of children have shown considerable global variation in the prevalence, with some estimates as high as 43%.[31] 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.[5] 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 biologic and psychologic 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. Short-term reaction proportional to the challenge elicits an appropriate response (often escape and avoidance) and is of fundamental importance as a survival strategy. This survival strategy, however, 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[14,15,86] and activation of latent virus[8,59] to impaired wound healing after surgery[64] and increased mortality.[11]

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] The 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.[69,88]

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, with 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 psychologic 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 one's social 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 caregiver 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 with 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.[22] 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, at the mere mention of the word *dental,* provoke an immediate and intense anxiety response.

MECHANISMS

This 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.[27] 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 are located in the brainstem and the hypothalamus and 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[10] for details of the endocrinology of the stress response.)

INTER- AND INTRAVARIABILITY IN RESPONSES

Variability in Stress Response

The stress system receives and integrates a diversity of inputs that 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,[80] the severity of infectious diseases,[15,60] inflammatory response,[61] chronic diseases such as cancer,[83] and pain levels.[21,71,73,90]

For instance, psychologic stress has been shown to impair human wound healing in a number of well-controlled studies. In one study, investigators[47] placed 3.5-mm punch biopsy experimental wounds on the arms of chronically stressed caregivers and well-matched controls of patients with Alzheimer's disease to study dermal wound healing. Alzheimer caregivers took an average of 24% longer to heal standardized wounds, 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-1β than 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 wounds made in the same students during summer vacation, and the differences were quite reliable. No student healed as rapidly during examinations as during vacation.[64] 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.[63] 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

*References 12, 41, 42, 45, 48, and 85.

of health, which is 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.[24] 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 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.[77] 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.[9] It is not surprising that many highly anxious patients show elevated levels of pain reactivity during dental treatment compared with less anxious patients.[50] 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.[62]

A body of literature exists that shows pain is strongly influenced by factors including affective, cognitive, psychosocial history, and social learning (see also Chapter 19). One study[57] 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.[37] 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.[46] In addition, negative mood predicts greater severity of acute pain and is related to greater pain sensitivity.[38,58]

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.[36] These research findings reinforce the significance of efforts to reduce a patient's level of negative emotion prior to dental treatment.

There is increasing evidence that women with high levels of dental fear are more likely to have a history of sexual/physical abuse 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.[30,91] 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.[92] 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 the clinician can be trusted and not feeling in control.[93] It is not hard to understand the parallel between some elements of abuse and receiving dental care, including discomfort and pain, as well as a reluctance for the abused patients to believe that the clinician can be trusted.[84]

From a more general perspective, one study 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.[29] Not surprisingly, health care utilization and psychologic distress, including the tendency to catastrophize about upcoming events, was greater among women with an abuse history compared with other women.[29] The clinical implications of the association between sexual abuse and dental fear is 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.[90] 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. Specifically for highly anxious dental patients, experiencing treatment tends to make their fear of pain even greater.[90] Evidence of the biologic correlates of the individual differences in pain response reported before is beginning to emerge from a variety of sources, including those using functional magnetic resonance imaging (fMRI). One study found through neural imaging that fear of pain predicted activation of the right lateral orbital prefrontal cortex during a heat pain task.[73] This region of the brain has been linked with 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[5,16,55] 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, psychologic, 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 the receptor tyrosine kinase for nerve growth factor, underscores the role of individual genes in the reaction to painful stimuli.

One study[49] 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. They 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[25] found that the *COMT* gene was related to experimental pain sensitivity and risk of developing temporomandibular pain (TMD).

In a fascinating study,[76] 207 cancer patients were genotyped receiving morphine treatment with respect to the Val-158Met polymorphism and 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 ± 160 mg/24 h) when compared with the Val/Met (117 ± 100 mg/24 h; n = 96) and the Met/Met genotype (95 ± 99 mg/24 h; n = 67) groups ($P = 0.025$). This difference was not explained by other factors such as 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.

There is further evidence of a genetic basis for differences in patients' response to pain control agents. One study[71] demonstrated a significant link between melanocortin-1 *MC1R* gene sequence, pain tolerance, and efficacy of morphine-6-glucuronide in both humans and laboratory animals. In addition, the distinctive molecular processes that are associated with different types of pain and changes in gene expression over time support the idea that heterogeneous responses to pain stimulus is 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. With one in seven patients reporting inadequate anesthesia when his or her teeth are stimulated with a drill, even after administration of local anesthetic,[69] it may be time to launch research into the impact of individual differences in genetic composition on drug efficacy.[87]

Brain imaging studies provide additional support for the need to adapt to individual differences in pain mechanism. One study[13] 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.[13] 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, psychologic, 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 will be 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 the doctor-patient communication to identify, acknowledge, and reassure the distressed patient is one of the most effective anxiety-reducing strategies available to the dental team. It is a three-pronged strategy, involving the establishment of rapport with the patient, the use of a standard interview protocol, plus the use of effective nonverbal communication. Each of the prongs of communication requires a dialogue with the patient, rather than one-sided listening to the patient at one extreme or a monologue, with the clinician not providing opportunities for the patient to comfortably speak up, at the other. For most patients, having the clinician listening attentively sets a context on which to build a trusting doctor-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.[35] 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 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*.[4] An important exception to the use of nondental topics to establish rapport involves 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.,* unless the patient has expressed a preference for use of his or her first name, demonstrates respect. 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

Although listening is sometimes viewed as analogous to sending an audio signal to a receiver that faithfully reproduces the signal as it was transmitted, 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 exhibit 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 doctors 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 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 by being 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 is clarified, and the patient's strong emotions have diminished, the usual techniques for effective communication can be employed using open-ended questions with appropriate follow-up questions based on the content and tone of the message. ***Developing a dialogue of trust between the clinician and the patient is necessary and cannot be delegated to a staff member (see also Chapter 1).***

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.[20] 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.[53] 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 has the potential to go more 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 than their own. It is unreasonable to expect that clinicians can identify, understand, or adopt 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.[20] 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 will facilitate trust. Trust cannot be earned if the patient feels there is a lack of concern from the clinician.

Using A Standard Interview Protocol

The use of a standard interview protocol facilitates the dialogue with distressed patients. Not infrequently, the anxious patient will become "sidetracked" with the emotion-driven recollection of past dental experience, and the response to questions in the patient history will 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 has been 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 such as a reference to when I tell my teenager what time to come home can help make the point.) Since 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 asks: "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(s) of his or her anxiety can lead to fruitful revelation of specific averse 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 will 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 dialogue. 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 the 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 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 will maintain 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.[23] 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.[57] 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 "time out." 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.[3,40] (See de Jongh for a review of individualized dental anxiety management strategies.[23,56])

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.[55] 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.[43,54] (See de Jongh for a review of individualized dental anxiety management strategies.[23])

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 doctor have not agreed on.[23] 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.[51] 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 the patient's anxiety level, patients often report that they prefer to have music in the background because music makes them more comfortable.[4] 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.[3] Thus music can be both distracting and, under certain conditions of choice, provide the patient with a sense of control.

Portions of the office décor can also divert a patient's attention. It has often been reported that interesting artwork, décor, or tropical fish tanks are useful in distracting the patient from feared stimuli.[4] Some clinicians have used video games or a television set in the operatory as an effective and comforting distraction for anxious patients.[17,33] Manual stimulating distraction devices have also been used during dental anesthesia injection to reduce dental anxiety.[89] The clinician should be aware that simple distraction techniques may be ineffective if the perceived intensity of dental pain is too high.[6] 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.[18,19] 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 help to 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. By demonstrating for patients that deep breathing is something they can do on their own, a new sense of control is provided 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 will quickly and effectively improve 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 is directed inward. An altered level of consciousness is reached, similar to "zoning out" while daydreaming or reading a book.[44] Guided imagery is a form of mild hypnosis that can be useful with fearful patients.[7] 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.[79] 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 will 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 and further supports the efficacy of guiding patients to a "safe and comfortable place" of their own choosing.[28,54] 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.[7,52]

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.[39] 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.[52] 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.[28] 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.[2,15] *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/guided imagery are among the most useful. The effectiveness of hypnosis in treating dental fear has been demonstrated.[39] Hypnosis, however, requires specialized training and experience, so a brief "how to" belies the complexity of the strategy. For clinicians who are interested in being trained in hypnosis, resources can be accessed through the Internet[1] 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.

Referring to the Mental Health Professional

In extreme cases, refining the dialogue 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 psychologic 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 dialogue with the therapist during the course of dental treatment. Questions the dental provider and the therapist may discuss are:

- 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 HIPAA standards in discussing the patient's care among themselves and in sharing information unless directly relevant to provision of dental care.

SUMMARY

Individual patients differ dramatically from each other in how they respond to dental treatment. These differences arise from multiple biologic, psychologic, and social sources, which are influenced by both genetic factors and lifestyle. How successfully a clinician adapts to these differences by tailoring treatment options will depend 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 dialogue can produce improved clinical outcomes.

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CHAPTER

27

The Law and Dental Technology: Focus on Endodontics

ARTHUR W. CURLEY

CHAPTER OUTLINE

SCOPE OF DENTAL TECHNOLOGY
- Patient Communications and Marketing
- Dental Continuing Education

SCOPE OF THE ISSUES
- Barriers to Adoption of Dental Technology

LEGAL EXPOSURE
- Traditional Avenues of Exposure
- New Avenues of Exposure

LAW OF COMPUTER RECORDS
- Off-Site Backup
- Protection Against Alteration

IMAGING

INSURANCE SUBMISSIONS
- Office Technology Policies
- E-Mail and Internet Access
- Communications
- Patient Education

THE FUTURE

Dentistry has always been at the forefront of technologic advances in health care. Until recently, those advances came at a steady and moderate rate that allowed the clinician to adapt using a traditional understanding of the laws affecting practice. However, the exponential growth of the Internet and the digitalization of health care systems have required clinicians to revisit their understanding of the laws of dental technology and the resulting changes in the standard of care.[17]

This chapter provides an overview of the nature of the technology and trends in dentistry, the issues that consequently arise, and the applicable laws.

SCOPE OF DENTAL TECHNOLOGY

Recent advances in dental technology have brought about rapid changes in every aspect of the delivery of dental care, including marketing, communications, record keeping, imaging, testing, diagnosis, treatment, education, and billing. Likewise, these advances have increased the capabilities of the dental clinician, and with their growing acceptance in the dental profession, the standard of care also has evolved.

Patient Communications and Marketing

Many homes and businesses in America have Internet access. Patients younger than 30 may have grown up using computers and cell phones as their primary sources of information and communication.[10] Once just the name of another Internet web page, *Google* (www.google.com) has become a de facto verb that defines one of the most common methods of exploring an issue in dentistry, researching a proposed treatment, or even finding a dentist. Patients frequently investigate a dentist by *googling;* they find sites that rate dental offices, or they may go to a state's licensing webpage.[20] After selecting a dentist, the new patient is likely to use *MapQuest* (www.mapquest.com) for directions to the office if the dentist's webpage does not already provide a link to a map page.

Dental Continuing Education

Dentists no longer need to wait for the mail carrier to deliver journals or find the time to attend a convention for an update on recent advances in dentistry. Many clinicians have Internet subscriptions to sites such the American Dental Association's (ADA) (www.ada.org/prof/resources/pubs/adanews/index.asp) that regularly announce developments in dentistry by e-mail and provide links for further investigation. Accordingly, the legal *standard of care,* or the practices expected of a reasonable and prudent dentist, has become a standard that is defined on a national level rather than simply by the care provided in the local community wherein the dentist practices.

Conversion to digital dentistry soon will no longer be optional. It will become necessary because of the elevation of the standards of care, patient demands, and the requirements of the marketplace in general. At all major dental conventions, most trade exhibitors now market some form of digital

dentistry. Also, clinicians increasingly are seeking patients by offering "high-tech" dentistry.

SCOPE OF THE ISSUES

In the 21st century, dental technology affects every aspect of the delivery of dental care at an ever-increasing rate. At the same time, learning curves are flattening, and costs are dropping. State licensing boards and traditional dental societies are finding it difficult to keep up with the advances in dental technology. U.S. lawmakers and courts also are finding it difficult to keep pace; therefore they have taken laws designed for other technology industries and adapted them to meet medical and dental care issues.

In addition, the law has recognized the impact on privacy of the digital communications age and the ability to transfer electronic patient information instantly. The Health Insurance Portability and Accountability Act (HIPAA), passed in 1996, sets forth strict guidelines on confidentiality standards for patient data.[2]

Barriers to Adoption of Dental Technology

Traditional barriers to the adoption of technologic advances in dentistry have included investment limitations, slow approval by the U.S. Food and Drug Administration (FDA), office design issues, conversion costs, and resistance from clinicians who were very comfortable and reasonably productive with conventional dental systems. The old ways were simple, they worked, and they were cheap, but those barriers are falling. With greater community acceptance, the production of technologic devices has increased, lowering the cost of digital dentistry. Adaptation of technologies from other health care industries has increased the rate of regulatory approval for digital dentistry. A reasonable understanding of the laws of dental technology can help remove the last barrier: clinician resistance.

LEGAL EXPOSURE

Traditional Avenues of Exposure

Traditional legal exposures for the dental practice included *malpractice* (negligence or substandard care), *general liability* (also known as *premises liability*), employment claims, and licensure issues. Typical claims against dental offices involve bad results or ineffective treatment arising from poor planning and execution. In many cases, they can be directly traced to poor record-keeping or to communication gaps between dentists and patients and/or between general dentists and specialists. These weaknesses often are readily spotted by lawyers. Particularly by those who specialize in dental malpractice cases.[7]

Digital dentistry, when used correctly, can reduce traditional legal exposures by increasing record-keeping capability, providing twice as much data at half the input time and eliminating legibility problems. E-mail and faxes provide instant, traceable forms of communication, record sharing, and data gathering; as a result, the clinician is better informed and better able to provide ideal treatment.

New Avenues of Exposure

Although digital dentistry has the potential to reduce the risks associated with certain traditional areas of exposure, it has created some new avenues of potential exposure. Misuse of technology, inadequate maintenance of equipment, warranties, and confidentiality of records are emerging areas of potential liability exposure.

Misuse of Technology

The nature of health care, and particularly of dentistry, is such that maximization of production often is the primary business focus. Dentistry has always been creative in finding ways to provide treatment in less time and with less discomfort and fewer complications for the patient. However, that inventiveness may prompt clinicians to use technology for unapproved, or *off-label,* purposes and to take short cuts. In such cases, the guidelines issued by the manufacturer of a specific technology can be used as a statement of the standard of care. Examples include the *Physician's Desk Reference* (www.pdr.net) for medications, the instruction booklet for endodontic electronic apex locators, template overlays for sizing dental implants, the user guides for virtual impressions, and the owner's manual for electric high-speed handpieces.

In the event of a complication or bad result, the patient's attorney may offer the manufacturer's guidelines as evidence the jury should consider in deciding whether a problem suffered by a patient was the result of substandard care for which damages can be awarded. A particular manufacturer's guide or standard that was not followed by a defendant dentist, projected in bold print on an 8 × 8-foot screen, tends to have a very powerful impact on a jury that is trying to decide whether a treatment complication was merely a bad result or evidence of substandard care.

In contrast, evidence of strict adherence to a manufacturer's guidelines may be used by the attorney defending a dental malpractice suit. It may serve as powerful evidence that the complication or problem was not the result of substandard care but rather a risk of the procedure.

Inadequate Maintenance of Equipment

Manufacturers of dental technology can become targets of personal injury suits if a treatment complication or bad result can be traced to a failure or breakdown of the technology. For example, defective bearings in a high-speed handpiece may cause overheating, resulting in a burn on the patient's lip, or an endodontic file can break in a canal during treatment. Therefore manufacturers of dental equipment have issued specific guidelines for the maintenance of dental devices. If those guidelines are not strictly followed, liability for an injury shifts from the manufacturer to the health care provider, even if the patient's injury was caused by a manufacturing failure. The prudent clinician strictly follows the guidelines for maintaining equipment and, more important, documents adherence to those instructions. This policy should include regular and scheduled testing if such is required by the manufacturer. With regard to the example of the overheating handpiece, some manufacturers have issued revised guidelines calling for shorter and shorter recall cycles and for more frequent testing by the dentist. Failure to keep up to date on those changes could expose the user to liability for an equipment breakdown.

Warranties

Breach of warranty presents a different type of legal exposure than breach of the standard of care. Failure to meet the stan-

dard of care is a form of dental negligence, which usually is determined by comparing the testimony of the defendant with the testimony of expert witnesses retained by both parties.

A *warranty* is a statement or promise of a specific result or, in dentistry, a promise of a specific outcome. In the event of a bad result or complication, the patient need not prove substandard or negligent care; the person need only show that he or she received something other than the promised result. Such representations must be avoided.

Dental high tech equipment, such as computer projections of potential treatment results, can create a warranty and the attendant exposure for less than the projected result. The prudent clinician therefore makes sure to have patients review and acknowledge disclaimers notifying the patient that such projections are merely estimates, that no specific result or outcome can be guaranteed, and that individual outcomes may vary. These disclaimers must be charted and also must become part of the informed consent process and the case documentation.

Some clinicians may think it acceptable to offer an expressly stated but limited warranty for dental treatment. In such cases, no evidence of substandard care is required; merely a failed or unsuccessful result, and the dentist who issued the warranty is required to pay for retreatment.[15]

Standards of Care

The traditional standards of care have evolved to include reasonable standards of dental technology, which is subject to specific state and federal laws. A dentist may become liable for failure to suggest the option of a new form of dental therapy and for not allowing the patient to make an informed decision on options, regardless of whether insurance coverage is available for the ideal treatment or test.

The law has embraced a doctrine of *informed refusal,* which specifically requires discussion of the risks of refusing a recommended treatment or test. For example, the clinician would be required to discuss advanced endodontic treatment modalities, cone beam volumetric tomography (see also online Chapter 29), or dental implants, regardless of the patient's insurance coverage. The prudent clinician documents such discussions with the patient. Such documentation should include a mention of the options discussed and states that the risks, benefits, and alternatives (RB&As) were discussed and the specific option was selected by the patient. If the patient chooses an inferior option (higher failure rate, more complications) because of financial limitations, the clinician should specifically document the informed refusal.

Many dental risk management and professional liability companies now provide *informed refusal* forms for patient acknowledgment and documentation.[9]

Webpage Issues

Internet webpages have become a major business tool throughout the world and particularly in America (see also online Chapter 28). They are the new marketplace, post office, and meeting center—and a new potential for liability for the uninformed dentist. Therefore when planning a webpage, dentists should consider the legal issues as well as style and content.

Webpages are created by blending text with images and *links* (click-on pointers that can direct the viewer to another webpage), all of which are separate files that can be copied and downloaded to make another webpage. Such techniques are commonly used by webpage builders, but the material, text, and images are subject to copyright laws in the same fashion as text and pictures in a book. As such, the use of images and text taken from other webpages may subject the owner (dentist) of the recipient data to liability for copyright infringement. The prudent clinician makes sure that permission has been obtained and documented before using images taken from the Internet, unless the material is considered *public domain* and is so marked or labeled. This proviso also applies to links that use the logo of another company or person. Dentists cannot assume that buying a product or endorsing a company constitutes permission to put the company's logo or trademark on the dentist's webpage. Again, *documentation by way of a confirming letter or e-mail should be obtained before such proprietary images or links are used on a webpage.*[18]

Interactive webpages, created through the use of special programs and e-mail connectivity, present another potential legal issue: the remote and electronic practice of dentistry. Patients can submit questions, send digital photographs or scanned radiographs, and request an opinion through the website. However, responding to such an inquiry may be considered the practice of dentistry in the state where the patient resides, which may not be the same state where the dentist holds a license. The courts have seen several cases of prosecution of doctors for giving advice over the Internet to patients in states other than the state where the doctor is licensed. Also, charges have been brought against insurance company consultants for evaluating out-of-state claims for benefits.[13]

Although currently the subject of considerable discussion, nationwide licensure by credential would appear to be years away. Therefore the practice of dentistry is limited to the licensing of individual states (and nation) in which a dentist maintains a license. To avoid potential liability, a dentist with interactive webpages should use disclaimers specifically stating that the communication is to be considered only as data for informational purposes, *not advice or diagnosis,* and in all cases it is not a substitute for a consultation with a dentist in the community where the patient lives.

Confidentiality and Security

All dental health care providers have a basic understanding of the need to maintain the confidentiality of patient records and the information included in them. However, with the advent of paperless digital dental records and imaging, the potential for rapid and widespread distribution of a patient's inadvertently disclosed confidential information has become a significant issue. The problem has increased with the growth of the Internet and the use of computer consultants with remote access. These matters have come to the attention of lawmakers and the courts. Laws have been passed to address such concerns.

Even if a dentist reasonably believes that he or she is not subject to HIPAA, almost all states have laws requiring that reasonable efforts be made to secure patient records and maintain their confidentiality. Coincidentally, many of those requirements mirror the regulations set forth by HIPAA.[4] The following checklist for confidentiality compliance is useful for all clinicians, whether HIPAA qualified or not.

- *Recall cards.* An unsealed, postcard-type communication from a dental office to a patient should not disclose any

information about a patient's health history. It may only indicate the patient's name and a general statement of the need for an appointment (without specifics as to why) or may merely confirm the date of a recall appointment. Information about the need for premedication or other special preparations or specific treatments to be performed should not be included on an unsealed postcard.

- *Records.* Dentists are required to make reasonable efforts to maintain the confidentiality of records. Protocols should be established for record organization, placement, and storage to prevent inadvertent disclosure of patient information. The jackets of dental charts should not have any markings, writing, or indications of the patient's illness specific health status. Stickers warning of a patient's particular medical condition should not be used. Reception desk protocols should prevent a patient's chart from being left open and visible when staff members are speaking with another patient. This rule should be followed in all areas of the office. Day sheets should not include any information other than the patient's name and the time allotted for the appointment, or they should be positioned so that they are not visible to patients (e.g., under a cover sheet or an inbox).
- *Communication.* Reasonable communication protocols should be established that (1) prohibit easily over heard conversations in the reception area between staff and patients regarding a patient's medical condition; (2) prohibit loud conversation between staff members when discussing the medical history or specific treatments of patients; (3) offer patients the opportunity to go to the dentist's private office to discuss sensitive aspects of a medical history.
- *Computers.* In offices with computer screens that might be viewed by patients, protocols should be established to minimize the potential for inadvertent viewing of information about another patient. Such protocols might include something as simple as turning the computer screen away from patient paths of travel or using polarizing filters over the screen that narrow the viewing angle to 15 degrees.

LAW OF COMPUTER RECORDS

Although the laws of individual states may vary, certain basic requirements apply to computer records in health care settings.[1]

Off-Site Backup

All computer systems should have an off-site backup data storage system. Daily or weekly backups of the computer files must be removed from the office and stored at another site; this prevents loss of the backup data in the office as a result of an event such as a flood or fire.

Protection Against Alteration

A valid digital record-keeping system must include programming that prevents alteration of the data after input. Any digital record-keeping system that allows postinput changes to the entries without maintaining evidence of the change may be challenged in court. The same rules apply to digital imaging such as radiographs and photographs.

Signature

The digital record must also note the identity of the person inputting the data, either by a login system or by a designation at the end of each recorded entry.[8]

Confidentiality

The law requires offices with Internet access to use reasonable security methods such as computer firewalls and antivirus programs. In addition, computer consultants with access to digital files should provide written acknowledgment of their obligation to maintain confidentiality.

In the transition from paper to digital records, paper charts can be converted to digital records by scanning the documents into an Adobe portable document format (PDF) file,[3] a graphic file that cannot be easily altered. It is important to remember that after a file has been converted into digital form, the paper records, radiographs, and photographs must be destroyed in a manner that maintains confidentiality (e.g., shredding). The law considers trash and paper given up for recycling to be similar to public property and readily accessible to anyone. Simply tossing old records into the trash is a violation of the laws that require maintenance of patient confidentiality.

The statute of limitations for bringing a lawsuit against a dentist for malpractice is 2 years in most states. However, various exceptions to that law exist, such as when the patient is a minor or is found to be legally incompetent. Because of these exceptions, most risk managers recommend that records, whether paper or digital, be maintained for at least 10 years after the patient's last visit. After that, the records can be discarded, but this must be done in a manner that maintains patient confidentiality. **Tip:** As more health care facilities and offices go digital, be on the lookout for states to increase the time dental offices are required to keep records.

IMAGING

The prudent clinician recognizes that as the quality of digital radiographs improves and the cost decreases, digital imaging will become a standard of care. There are several reasons for this. Digital radiographs expose the patient to less radiation, are not subject to the same processing problems as conventional films (underdeveloping, poor fixation), can be downloaded to the chart and instantly sent to other dental offices, and ultimately will be required for online insurance submissions. In the not-too-distant future, clinicians may see the end of the darkroom and the passage of laws that ban or severely restrict the use of the chemicals required to process radiographs, because of toxicity concerns.

These same issues will apply to digital photography versus conventional photography, because of the digital mode's easier storage, transmission, and submission to insurance companies. As digital photography becomes more popular with the public, patients will expect that photographs taken by the dentist will be readily and easily transmitted to other health care providers or the patient.

Perhaps the single most dramatic change in the standard of care has come with the advent of cone-beam three-dimensional (3D) imaging, described in Chapter 29.[12] This imaging system provides the clinician with virtually complete anatomy of a tooth and surrounding structures—information that can be used in planning, evaluating, and performing

endodontics.[12,21] The data obtained can display conditions such as root curvature, sinus anatomy, and instrument location to the point that treatment is more predictable and reliable and complications more avoidable.[16] The prudent clinician must consider giving patients the option of 3D cone-beam imaging if such data would enable better treatment planning, reduce risks and complications, and foster greater success. If a patient declines the use of such imaging, the clinician—after educating the patient about the risks, benefits and alternatives—should obtain and document informed refusal.

INSURANCE SUBMISSIONS

Materials, postage, and labor constitute a significant part of the costs incurred by insurance companies for processing dental insurance claims. In the not-too-distant future, insurance carriers will forbid or add a surcharge for claims submitted as paper documentation, using plastic radiographs, and paper photographs. Other industries already require digital submissions. Airlines now add a surcharge for paper tickets, and banks charge a fee for maintaining and returning canceled paper checks. Some courts, such as the Federal District Court of Northern California, strictly prohibit the submission of paper documents (all filings must be submitted over the Internet as Adobe PDFs). Insurance companies will easily find judicial favor when they start to require digital submission of claims for dental treatment and begin charging a fee to process paper submissions.[5]

Office Technology Policies

As dental practices begin to use online computer services such as e-mail and Internet access, as well as digital record storage, the office should establish and enforce staff management policies regarding the use of those technologic aids.

E-Mail and Internet Access

E-mails and Internet surfing are two common sources of computer viruses, spam, and spyware. Computer viruses can damage or destroy data. They typically and unknowingly are sent by a friend who is sharing a joke or photograph that has been passed around by other computer users. While surfing the Internet, users can unknowingly download computer spyware programs; these programs secretly take up residence on a computer system and then silently begin transmitting information about the user's computer over the Internet to outside individuals and companies. The use of e-mail and the Internet also can result in a person's e-mail address being sent or sold to spam companies and businesses that mass produce millions of unwanted e-mail marketing messages per day.

In most states, the law holds that employee use of e-mail, in the absence of a specific policy to the contrary, is considered private, similar to the use of a telephone, and employers may not access such communications without an employee's permission, just as tapping a phone call is illegal. For these reasons, offices providing employee access to e-mail and the Internet should have written policies that specify the restrictions and violation warning (Box 27-1).

Communications

Some clinicians have considered documenting their patient consultations by recording them with video or audio devices.

BOX 27-1

Sample Written Policy for Employees With E-Mail and/or Internet Access

1. *Use of, and access to, e-mail and the Internet at the offices of _____ is restricted and limited to business purposes only. Personal, nonbusiness use is prohibited.*
2. *Use of e-mail and the Internet is not to be considered private, and _____ reserves the right to, and may at any time, audit, record, trace, and/or monitor employees' use of e-mail or the Internet.*
3. *In order to protect the office computer systems and patient privacy, this office will use various security systems such as antivirus and antispyware programs, as well as spam filters. Employees will be expected to assist, as indicated by their assigned job duties, in the routine application of such security systems.*

Violation of these policies may subject an employee to termination of employment.

Recent developments in digital recording make it possible to record and store huge amounts of video and audio data on relatively compact systems. DVDs can record and store up to 6 hours of video/audio on a single disk, which currently costs less than $2. Devices with hard drives can record and store more than 80 hours of video and audio data.

The laws of most states prohibit the recording of conversations conducted over the phone or in person when anyone in the conversation could have a *reasonable expectation of privacy.* Hidden microphones or cameras are illegal recordings and subject the user to both criminal and civil penalties. However, the law allows recording of a conversation and/or the patient's image if the patient gives expressed consent to the recording.

If the clinician decides to record a consultation (video, audio, or both), the patient's permission should be obtained before recording begins. After this permission has been obtained, the patient's consent to being recorded should be stated, including the time and date, at the beginning of the recording and again at the end of the recording. The fact that the consultation was recorded should be noted in the patient's chart.

Patient Education

Digital systems are available for patient education. The fact that a patient viewed educational material about a proposed treatment option or a diagnosis, whether the material is in the form of a booklet or an audio or video program, should be recorded in the patient's chart. The prudent clinician documents what, where, and when the patient was provided educational material, as well as the fact that the dentist and patient had an opportunity to discuss the material.

Even the education of dental students has gone digital; at some schools, all textbooks are provided to students through laptops rather than on paper.[11]

THE FUTURE

Dental technology is growing at an accelerated rate, and the digitalization of all aspects of dental care is on the near horizon. The process began with electronic records, moved to digital

imaging, and is progressing to digital treatments, such as endodontic electronic apex locators, automatic periodontal probes, virtual impressions, computer-fabricated restorations, and in-office testing ("laboratory-on-a-chip" systems) to evaluate a patient's blood sugar, perform microbial assays, and monitor a patient's stress level. Patients have come to expect complete treatment in one session, such as single-visit root canal therapy (indicated in most cases), preparation and placement of crowns, and same-day surgical placement and restoration of implants.[6] One of the present challenges for clinicians considering endodontics is the growing school of thought that removing the tooth and replacing it with an implant may be the better long-term treatment in cases where saving a tooth with endodontics would also involve a post, build-up, pins, crown lengthening, and a crown. In such cases, the prudent clinician should advise the patient of both options, regardless of financial issues, and allow the patient to decide. If the patient chooses the lesser of two options (say, because of insurance coverage), informed refusal should be obtained and documented.[19]

In the near future, after a patient chooses a dental clinician, the individual will have his or her computer's scheduling program contact the dental office's computer over the Internet to schedule an appointment. At the same time, the patient will authorize a HIPAA-qualified release of digital records and imaging from other health care providers, and the information will be downloaded to the dentist's database patient health history profile, which will place appropriate tags for the staff and dentist to review before the first consultation. The dentist's office will have no darkroom, no record storage area (paper or models or imaging), no laboratory, and in some cases, no waiting room because no paperwork would need to be filled out, and patients who might be at the mall next door could be paged on their cell phones just before an appointment. Sound futuristic? It's already here; some dental service companies are completely digital and provide standard-of-care dentistry in a two- or three-operatory recreational vehicle (RV).[14]

SUMMARY

Dental technology is raising the bar for the standard of care. Very soon, some aspects of conventional methods of record keeping, image gathering, and patient treatment will become substandard. The prudent clinician considers and, when appropriate, adapts to new dental technologies but only with an appreciation of the legal issues involved.

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CHAPTER

28

Digital Technology in Endodontic Practice

MARTIN D. LEVIN

CHAPTER OUTLINE

COMPREHENSIVE TECHNOLOGY PLAN
- Strategic Plan
- Operational Plan
- Measuring Success

TRACKING BITS AND BYTES THROUGH THE "PATIENT LOOP"
- Central Information Store
- Reception Area
- Front Desk
- Operatory
- Sterilization Area
- Administrative Area
- Clinician's Office

COMPUTER INDUSTRY STANDARDS
- Graphics and Imaging
- Open Database Connectivity
- Digital Imaging and Communications in Medicine
- Systematized Nomenclature of Dentistry

INTERNET

IMPORTANT SOFTWARE FEATURES
- Financial Security
- Referral Tracking
- Preregistration

REFERENCE
- Infrastructure and Connectivity
- Power, Cabling, and Wireless
- Central Processing Unit
- Monitors
- Printers
- Document Scanners
- Input Devices
- Consulting and Support
- Pharmacology Reference Software
- Patient Education Software

Endodontic offices rely on words, numbers, and images to communicate with patients, assess patient status, evaluate treatment options, and determine treatment outcomes. Many dental offices already use computer-based programs to manage patient demographic and financial information; the electronic oral health record[23] (EOHR) is the next logical step in the journey to the paperless office.

Although EOHRs are now widely available, integration of this information with an electronic health record (EHR) to create a unified, scalable, searchable, and secure patient database documenting the current health status and lifetime medical history of each patient is still elusive. In a broader sense, the real challenge is to understand dental informatics and the ways the collective efforts of the profession can be applied to improve patients' health care. Two experts in dental informatics observed that[79] "application of computer and information sciences to improve dental practice, research, education, and management" is the holy grail of dental informatics.[34,80]

The goals of this chapter are (1) to improve the dental care delivery organization's understanding of dental informatics and (2) to devise a strategic plan to help design office systems that use the latest computer, digital-radiographic, photographic, charting, and management tools to document vital information. Once this strategic guide has been implemented, continual upgrading based on best-recommended practices is important to ensure continued success.

In the early 1990s, the American Dental Association (ADA) initiated the development of technical reports, guidelines, and standards on electronic technologies used in dental practices. A task group was established to promote the concept of a computerized dental clinical workstation and to allow the integration of different software and hardware components into one system to accommodate all a clinician's information needs. Establishment of interoperability standards for these modules eliminates the need for multiple stand-alone systems in the dental office and provides a seamless information exchange throughout all facets of health care. The American National Standards Institute (ANSI) recognized the ADA as an accredited standards developer in 2000[4]; the ADA is working to ensure that common conventions have worldwide acceptance.[43]

The U.S. government has already begun using the Government Computer-Based Patient Record (GCPR) in federal health care agencies. The advantages of the EOHR were highlighted in a recent study[46] that compared an EHR with manual record

keeping. The study's findings were favorable to the EHR: improved understandability (89.2% versus 69.9%), better legibility (100% versus 64.3%), and the benefits of having at least one diagnosis recorded (48.2% versus 33.2%). Additional findings included improved prescription writing (86.6% versus 66.2%) and improved ability to recall advice given to patients (38.6% versus 26.8%).

COMPREHENSIVE TECHNOLOGY PLAN

Information technology (IT) systems are changing every day and at an ever-increasing rate. Deciding what equipment to purchase for patient use in the reception area, as well as for clinical, front desk, administrative, and clinician needs, can be daunting. Upgrading older office systems, especially while an office is in operation, complicates this process.

Clinicians should begin by establishing clear goals for the systems they want to implement. Generally, such technology planning can be divided into three areas: (1) the strategic plan, (2) the operational plan, and (3) measurement of success.

Strategic Plan

The strategic plan should be based on a list of goals for the seamless integration of all computer functions in the office. Clinicians should focus on what is necessary to provide the best service to patients and establish clear priorities. The more sophisticated the system, the more customization that can be done to provide exactly what patients and clinicians value most.

Dental practice systems are meant to assist the human user. These systems are *work multipliers*—that is, the technology replaces human effort, making tasks the clinician and staff members do easier and more error free and in some cases making possible new tasks that otherwise would be too costly in terms of expense or time. The bottom line for these systems is the value provided in terms of labor savings and quality of services compared with the total cost of acquisition and use. If the value the system produces is greater than the cost of buying and using it, the clinician and the practice benefit.

At its most detailed, or *granular,* level, dental care is delivered differently across the United States. Although many commonalities exist, and best practices generally are agreed upon, the goal of achieving the best outcome for the patient often means tailoring the dental arts and sciences to what works best in the hands of a specific clinician. Clinicians build or modify their practice environments to fit the nature of their practice; the workflow is tailored to allow the clinician to operate at the highest level of excellence in the clinical process.

Clinical and practice management systems are most beneficial when technology helps to make activities in the clinical process more efficient, more economic, and less subject to error. As noted in 1996 in a concept model for the electronic dental record published by the ADA, the fundamental activities in the clinical process are[2]:

- Examination
- Diagnosis
- Treatment planning
- Clinical care delivery

Table 28-1 presents the usual types of computer support available for these activities.

TABLE 28-1

Typical Computer Support for the Clinical Process

Procedure	Task	Typical Automation Support
Examination	Data acquisition	Automated charting, note taking, digital radiography
Diagnosis	Data analysis	Clinical decision support, automated real-time consultation
Treatment	Service planning	Decision support: case planning, automated presentation, documentation of informed selection and consent
Care delivery	Service delivery	Documentation of services provided; digital radiography of completed services

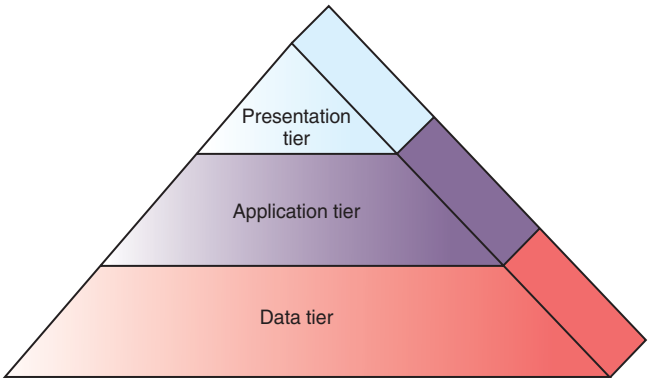


FIG. 28-1 Three basic layers of an integrated office computer system.

The systems that provide this type of support typically are composed of several layers, or *tiers,* that correspond to the basic functions provided by computer technology (Fig. 28-1). Each tier provides the dental practice system with a capability, and for best use, each tier must be configured to support the nature of the practice and its workflow. The tiers usually are categorized as follows:

- *Data tier:* The form and physical storage of data; in clinical practice, this often is the EHR and a variety of clinical and administrative reference data.
- *Application tier:* The internals of the system, which provide logic, calculation, and help with decision-making functions.
- *Presentation tier:* The part of the overall computer system that interacts with people and the outside environment, including other computer systems.

At the data tier, the heart of the clinical system is the EHR. ANSI/ADA Specification No. 1000 provides a data blueprint for digital representation of clinical and support data for an individual human patient.[4] This standard is the only model-based, data-level specification of clinical data for building an

EHR database. In such an EHR data system, not only the individual patient but also family members and significant others are identified. Often this capability is expanded to identify individuals who may provide health care services to the patient. Administrative data such as personal characteristics, employment, and similar information frequently are included in the EHR. The digital structures used to build the dental chart (but not the graphic chart per se) are maintained in the data tier. Also in the data tier, but outside the scope of the EHR, are the reference tables and rules sets for clinical decision support, lists of pharmaceutical items in a formulary database, and code sets for anatomic location, diagnosis, procedures, and so forth. The data tier is the foundation of the practice system.

The application tier is the functional heart of the practice system. All processing of data occurs in this tier, such as clinical decision support whereby the computer acts on rules to alert the clinician to a potential problem with a particular therapeutic agent in the case at hand. The application tier can operate on the data specific to a case, the reference codes, and notations and construct the graphic dental chart for a particular patient. It also operates on any signals from automated diagnostic devices, such as digital periodontal probes, cardiac monitoring devices or root canal depth gauges, converting these into human-understandable measurements that can be displayed to the clinician by the presentation tier.

The presentation tier provides the face of the dental practice system. The application and data tiers usually are neither seen nor heard by the clinician and staff; the presentation tier is by far the most interesting aspect of the computer system because it is the point where the clinician and staff interact with the system. The presentation tier is where the dental chart takes form, where the clinician can watch as its graphic features are recorded, and where the clinician can key in any special notes required. It also is where the clinician receives any patient care advisories, enabling patient safety features such as alerts to prevent adverse events. The presentation tier is where the electronic prescription is sent to a pharmacy. Finally, the presentation tier is the realm of digital radiography, regarded by many as one of the most beneficial aspects of automation in dentistry.[19]

The core of the office IT infrastructure is the central information store (CIS) (Fig. 28-2). The CIS is the "center of the universe" of the IT system, comprising the server and its attendant software and hardware; it also may include data generated in the office or imported from the Internet. The CIS should be located in a secure area and have an always-on connection to the Internet to increase functionality. The establishment of a local area network (LAN) now is considered essential for dealing with the heterogeneous information generated in each physical location and for integrating a wide area network (WAN) to connect several practice locations.

A comprehensive strategic technology plan should address all the information generated and processed in the practice and the ways it will be handled. The clinician must be prepared to manage information from patients and referring clinicians and to document and communicate all pretreatment, treatment, and posttreatment information via LAN, WAN, mail, fax, e-mail, and the web. An ideal system that handles this mix of data and gives all members of the office team what they want integrates the CIS with the reception, clinical, front desk, administrative, and clinician's office components.



FIG. 28-2 Relationship of the central information store (CIS) to functional areas in the office local area network (LAN).

Operational Plan

The newest concept in dental office management is *front-desklessness*. The central tenet of this concept is to minimize office interactions that traditionally take place at the front desk and shift them to the operatories (e.g., procedure code and fee posting, prescription writing, and all associated communications). Some programs now available to endodontists allow pending or posting of all relevant data from any computer in the office, including the generation of postoperative forms, instruction sheets, patient treatment reports, and e-mail correspondence. Advantages of this system include improved workflow and accuracy, because significant office revenues can be lost through posting inaccuracies that are never discovered.[58]

The operational strategy covers the planning and day-to-day practices that ensure a smoothly running IT system. Training and cross-training of staff are essential elements of a complete plan. To begin, at least one staff member must be knowledgeable about basic computer operations, especially word processing. End-user training courses are available online (e.g., globalknowledge.com) and nationwide to teach these basic skills. Books and video self-study materials can augment classroom training. The purchase of most clinical and front desk software systems should include ample opportunity for initial training.

Once initial training has been accomplished, a second training session, preferably on site, is advisable. This second visit, which should be scheduled several months after the initial installation, allows the clinician and the office staff to fine-tune the program by customizing certain preferences and double-checking that all the program's features are used to full advantage. In addition, to get the most out of the software, staff members should periodically review the program's training manual to discover features and shortcuts that may have been overlooked.

All computer workstation environments should be designed with ergonomic considerations in mind, such as work surface, keying, seating (Fig. 28-3), acoustics, lighting, and ventilation (Box 28-1).[42] Special care should be taken to design each area of the office so that employees, patients, and clinicians are comfortable, and all personal health information (PHI) is protected.

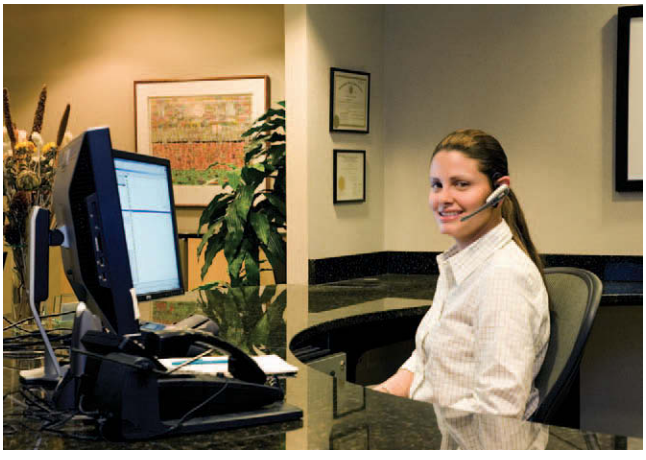


FIG. 28-3 The front desk computer workstation has dual high-resolution 24-inch flat panel monitors, ergonomic seating, and wrist-neutral positioning of the keyboard and pointing devices.

Management of database security is another important aspect of a good operational plan. The security standards mandated by the Health Insurance Portability and Accountability Act (HIPAA) now apply to all covered entities (see also Chapters 11 and 27). Practices are required to (1) perform a risk analysis to safeguard all electronic protected health information (E-PHI); (2) use technical, administrative, and physical safeguards to protect E-PHI; and (3) institute ongoing risk management. Security standards require that each employee have an individual password to access the electronic medical records system. Even in offices that are not covered entities, prudence dictates that clinicians use software that provides multiple levels of protection to reduce the chance of malicious tampering with data. For example, control over deletion of files should be protected to allow only senior staff members and/or the clinician to make changes. Compliance with the security standards requires a framework for ongoing compliance, including the designation of a security official and continual analysis of security needs as office systems change.

The purchase and continuation of hardware and software maintenance contracts are another area where diligence is important. The clinician should create a system for monitoring the status of the maintenance plans for key elements of the IT system, especially digital radiography and front desk software. Establishing insurance coverage for computer hardware, software, and data also requires special attention. General office insurance policies often limit computer coverage; therefore clinicians should discuss policies with a knowledgeable insurance agent. In addition, staff members should keep a file of purchase invoices, including digital photographs of all equipment, to document purchases for insurance purposes.

Marketing the practice is another way in which an IT system can prove invaluable. Referral and educational materials should be upgraded continually. The production of newsletters, e-newsletters, informational mailings, patient education handouts, and management of sponsored study clubs and meetings can be handled with the help of an up-to-date word processing and practice management system (see Fig. 28-1). Marketing is most effective if it is customized, frequent, and consistent.

Measuring Success

Once various technologic systems have been implemented, clinicians should learn how to measure their progress toward achieving strategic goals. For example, if premedication instructions are included in a "welcome kit" sent to new patients or on an Internet "welcome" page, staff members should see a quantifiable improvement in compliance with these instructions. Enhanced communication with patients and referrers should lead to improved satisfaction and increased engagement. Other ways to measure success with regard to operational strategies include surveying patients and referring doctors on the effectiveness of communication materials. A proactive approach to these procedural issues is the hallmark of a well-managed office.

Trend analysis is another key method of evaluating the success of office programs. The ability to better track referral patterns and follow up on collections, treatment efficiency,

BOX 28-1

HEALTH AND SAFETY FACTORS FOR EMPLOYEES

In the past, worker safety primarily involved preventing accidental injury or death from hazards in the factory or other industrial environment. Because the office now is the most common work setting, attention is turning toward subtler but still serious health problems associated with office work, especially cumulative trauma disorders (CTDs) and vision disorders. The incidence of these disorders is growing.

CTDs of the muscles, tendons, or nerves can be caused by repetitive movements of the body. As the frequency and duration of a repetitive task increase, so does the likelihood that a CTD will develop. Although most CTDs can occur in any part of the body, the term usually refers to disorders of the hands, wrists, arms, or shoulders. Carpal tunnel syndrome is one example of a CTD. Typical symptoms of CTDs include pain, swelling, tingling, numbness, or heat around the affected area during both work and rest. These disorders differ from simple fatigue, which disappears after rest. With a CTD, a person may still experience symptoms after days or even weeks of inactivity.

Ergonomists often note that computers themselves are not really to blame; repetitive, unvarying work is the cause of most CTDs. Individual physiology plays a big part in whether a person develops a CTD. However, experts recognize the importance of workstation design and layout in the prevention of CTDs. For example, a work surface's height and forearm support and work process issues (e.g., a company's break policy) affect the incidence of CTDs. For a worker involved in repetitive motion, frequent breaks, even short ones of 30 seconds every 10 minutes, help reduce the probability of a CTD developing. Task variety is also important. Therefore clinicians should design their office duties to allow clerical workers to alternate among different types of work (some of it computer based and some of it not) or to alternate computing tasks that require different motions and muscle groups.

Front Desk, Administrative, and Clinician Computer Workstations: General Specifications

Work Surface

A desktop height of 28.3 inches (71.9 cm), with a keyboard tray below the work surface that places the hands and wrists in a wrist-neutral position, is a good compromise height for front desk and administrative workstations. A bullnose (i.e., curved) edge can be placed on countertops to eliminate sharp corners. CRT monitors should be positioned far enough from the user to reduce extremely low frequency (ELF) radiation exposure.

Keying

Positioning the elbows at 90 degrees and keeping the arms and hands parallel to the floor can help prevent CTDs. With use of Microsoft's "Natural Keyboard" wrist rests,[93] locating mouse and writing platforms in the primary reach zone may also contribute to a more comfortable work environment. Work surface space must allow for efficient organization of documents, papers, and other materials. Peripheral equipment should be easy to reach.

Seating

Seat height should be adjustable to allow for positioning in the range of 16 to 20½ inches (41 to 52 cm) above the floor. The seat should have a "waterfall" or gradually curved contour just behind the user's knee and the underside of the thigh to prevent excessive pressure in this area. A seat back rest and lumbar support should also be provided.

Acoustics and Lighting

Ambient or background noise (i.e., white noise) is desirable at a level that does not interfere with task performance (40 to 55 dB). Experts agree that open-plan acoustics are subject to three main problems: sound level, speech intelligibility, and sound paths.[44]

Lighting sources should be designed and located to minimize glare and provide luminance in the range of 200 to 500 lux in the work area. Task or local lighting may be needed for reading documents. A combination of indirect overhead, natural, and task lighting is preferable. For close-up work, light should be directed sideways onto documents to avoid glare on the monitor; this helps prevent eye fatigue and headaches. Eyestrain is the leading complaint among computer users, according to the American Optometric Association.[7] Computer users should take frequent breaks and have their vision checked, and the work space should be designed so as to reduce visual stress.

Modern LCD monitors are designed to operate at their "native" resolution and a 60 to 70 Hz refresh rate to reduce monitor-induced eye strain. Further improvement can be realized by turning on "ClearType," imbedded in Microsoft Windows XP, Vista, and Windows 7 operating systems. ClearType provides improved font display quality over traditional forms of font smoothing or anti-aliasing. Glare can be reduced by replacing fluorescent lighting diffusers with grids that break up the light pattern. Office color schemes should be neutral and pleasing to the eye.

Ventilation

Proper ventilation requires about two air exchanges per hour. The temperature and humidity level should be kept constant, with temperature range adjustable from 68° F to 75° F (20° C to 24° C).

outcomes, and uncompleted treatments is one of the benefits of a well-designed software program. For example, generating reports that analyze fading referrals can be an important measure of referral satisfaction. Once these data are evaluated, an action plan and global strategy can be devised to address issues that need attention.

TRACKING BITS AND BYTES THROUGH THE "PATIENT LOOP"

The *patient loop* begins when a patient is informed that he or she needs an endodontic procedure and ends with the postoperative check-up visit (Box 28-2). This process begins the

BOX 28-2

THE VIRTUAL OFFICE: READY NOW

The new paradigm for exceptional patient care calls for sophisticated communication and customization that can be accomplished most efficiently in the integrated digital office. The following steps outline the process, or patient loop, from introduction of endodontic treatment to the last follow-up visit.

1. The process begins when patients need an endodontic or implant procedure. The endodontic office should make the following options available:
   - A website for patient education, contact information, referral materials, and patient registration forms
   - A *referral kit* for referring doctors that contains a brochure, a map to the office, customized referral forms, past newsletters, introductory information on the office website, and business reply envelopes
   - Informational meetings with referring clinicians to enhance communication and discuss expectations
2. When patients call for an appointment, the office should be ready to do the following:
   - Preregister patients using the practice management software
   - Set up an appointment
   - Refer patients to the office website, where they can read a welcome letter and complete the patient registration forms using a HIPAA compliant security protocol
   - Mail, fax, or e-mail a *welcome kit* with a welcome letter, brochure, explanation of the procedure, map, biography of the treating endodontist, and statement of the fee policy
3. When patients arrive for their first visit, they can also fill out the personal information sheets on a reception area computer kiosk connected to the network, or they can watch educational material while waiting. Any film-based radiographs can be scanned into the patient database. The patient's status can be indicated in the endodontic time and patient tracking module so that every member of the dental team knows the status of all patients from their presentation visit until they are discharged.
4. When patients are seated in the operatory, an assistant can perform indicated radiographic studies and capture a visible light (VL) image of any teeth or area in question, using an intraoral camera. The assistant then can enter the chief complaint, history of the current illness, list of medications, and medical history into the digital chart and check for drug interactions on the chairside computer workstation. The clinician performs pulp tests and records the endodontic findings in the charting module, followed by an oral and written consent and vital signs check. Treatment then can be started, and each step of the procedure can be recorded by mouse and keyboard input or voice command. If desired, subsequent appointments can be made for patients while they are still in the operatory, treatment reports can be generated, and their insurance information can be sent to the office financial coordinator or directly to the insurance company.
5. Patients are discharged at the front desk departure station, with all pertinent information already entered into the computer. This is an opportunity for patients to receive customized information about their treatment, next appointment, follow-up care, and any medications prescribed or dispensed.
6. Patient treatment reports and other correspondence can be created by the front desk or assisting staff; reviewed by the clinician at a computer terminal; forwarded to the print queue; and sent by mail, fax, or e-mail.
7. When patients return for follow-up visits, digital radiographs can be exposed, and comparisons of immediate postoperative and check-up radiographs can be made side by side on the same screen to assess healing. Reports can then be generated.

patient's journey through different operational steps that result in the successful management of endodontic treatment and completion of the EOHR.

Central Information Store

Creation and management of the EOHR and all its components is the job of the CIS. A CIS requires a single, robust central computer, called a *server,* that links all the computers in the practice. When computer systems are connected in this way, they most often are referred to as a *client/server network*. The server is designed to administer the network efficiently, taking care of management functions such as backup, file sharing, print processing, and web and e-mail tasks.

A server-based system has four primary benefits:

1. It saves time and effort because all information can be stored for instant access by any team member. As files are updated, all the new information is available instantly.
2. Security is better because all data are centrally stored and (normally) automatically backed up.
3. Each user can be better monitored, because each workstation can be uniquely identified at every transaction.
4. Remote access to the entire database is possible, so records can be reviewed from outside the office and more easily maintained.

The Microsoft Windows Server System allows the creation of a single point of contact with the Internet, which improves security as well as security settings that allow for appropriate remote access levels for employees or vendors.

Software

Servers have evolved into microprocessor-based machines dedicated to running certain software applications. Two basic types of software can be purchased to run on a server:

- *Network operating system (NOS)*. Most LANs and WANs in endodontic practices use a Microsoft NOS, which is based on the Microsoft Windows Server System. These sophisticated, stable systems are ubiquitous, and many servers come configured with base installations of the software.
- *Database engine*. One of the most robust and commonly used database management technologies is the structured query language (SQL), an ANSI standard computer language that uses SQL statements for obtaining and manipulating data. SQL is a technology, not a product,

and many vendors offer versions of SQL (e.g., SQL Anywhere, SQL Base, and DB2).

Hardware

Two basic hardware recommendations can be made to ensure a reliable network that can handle both digital imaging and practice management databases:

- *Redundant arrays of inexpensive disks (RAID Level 5)*. To reduce the chance of hardware failure and subsequent data loss, RAID 5 uses an array of multiple hard disk drives that behave like a single storage disk but have increased capacity and improved fault tolerance and performance.
- *Serial computer systems interface (SAS)*. More advanced than the IDE/ATA interface, SAS[82] is really a system-level bus with intelligent controllers that allow hard drives and other peripherals to operate with unmatched performance, expandability, and compatibility. Newer serial ATA (SATA) drives and IDE RAID are not considered as reliable as SAS and should not be used for critical hardware.[49]

Data Security

All data in the office are subject to security threats. Network attacks can interrupt office operations and cause loss of patient data, leading to legal liability. Whether a disgruntled employee decides to delete information, a disk with an infected Word file is brought from home and uploaded on an office computer, or an Internet connection allows unauthorized access, client and server tiers of the network are vulnerable to attack. Scalable security approaches are emerging to address these new challenges in three basic data security categories: integrated security, data backup, and archiving.

INTEGRATED SECURITY

Symantec (Mountain View, CA) and McAfee (Santa Clare, CA) are two leading vendors of enterprise-wide security suites that address malicious code attacks, denial of service attacks, unauthorized network entry, and blended threats.[90] Single-source enterprise protection can automatically update the office server and each client every day via the Internet. This includes:

1. Protection against viruses, worms, and Trojans
2. Software firewall protection to prevent unauthorized access to the network
3. Content filtering to eliminate unwanted materials
4. Connection security for any virtual private networks (VPNs). VPNs are constructed by using public wires (i.e., the Internet) to connect nodes and provide encryption and other security mechanisms.

DATA BACKUP

Data backup is the process of transferring data from the office server to a separate storage medium such as a tape drive or another hard drive. Because data are only as good as the last backup, it is imperative that a consistent backup and recovery protocol be followed. Data loss can be attributed to hard drive failure, data corruption, or physical damage to the server because of fire, flood, or theft. Although many backup strategies exist, even solo clinicians using periapical, panoramic and CBCT digital radiography modalities, and visible light imaging can generate 500 gigabytes of data per year. This means that backing up with CDs and DVDs is not a practical solution. Removable hard drive storage is also not recommended as a primary backup method because it does not allow regression to a previous configuration if an application failure is not evident for several days. Instead, a recommended scheme is a nightly tape backup with a 20-business-day rotation (1 business month) using a different tape for each of 20 days, replacing all tapes at manufacturer's suggested intervals or at least yearly.[12]

All backups should be full backups, rather than incremental or differential backups, to improve efficiency if reinstallation is required. Use of backup-to-disk functions and universal service bus (USB) hard drives can also be useful if multiple point-in-time backups are necessary. A full backup before any software upgrade must always be performed. Backup media should be tested periodically and backup logs examined to make sure the system can be properly restored. Other backup schemes that automatically upload encrypted data to a dedicated Web vendor nightly, ideally SAS 70 Type II certified, and store the data at a minimum of two geo-separate remote sites are available and can also provide a high level of security.

ARCHIVING

An *archive* is a permanent data backup that usually is stored off site. All computer data are subject to loss, and merely making a daily backup of the data does not provide sufficient protection if the data are corrupted because of a previously unknown virus, worm, or Trojan. Removing one tape from the normal backup tape rotation on a monthly basis and placing it in fireproof off-site storage usually ensures that a valid data set is available if a catastrophic event causes data corruption or loss. Another advantage is that this tape can be archived at a local medical records storage facility and logged in and out to ensure that the tape is not altered if required during litigation.

Equipment Protection

The server should be located in a secure area of the office, preferably a computer "closet" with locked access (HIPAA-covered entities are required to provide physical security of E-PHI). Designing lockable front and rear access to the server system or rack, if applicable, enhances access to the CIS when maintenance of upgrades are performed. Although rack-mount systems offer better space efficiency, there is a cost premium for this kind of equipment. All server installations must have adequate cooling, another factor that must be considered when planning the practice's air conditioning and heating systems.

Privacy

Security considerations are recommended for ensuring that data are reliable and safe and for meeting the requirements of Title II of HIPAA. The server and all software, including the wired and wireless LAN and WAN if any, should be protected by a system of individual passwords that allow different levels of security. Server log-in, remote access to the server via the Internet, program security, and especially the practice management system and digital imaging software must be password protected from unauthorized deletions and changes. The server password should be difficult to decipher (e.g., #eio9$kej**) to reduce the chance that the password will be decoded if the server is stolen from the office.



FIG. 28-4 The reception area computer kiosk allows patients to complete registration forms, view educational material, and surf the Internet.

Reception Area

Placement of a computer kiosk in the reception area with Internet-only access can be a convenience for patients who want to complete their registration materials online (Fig. 28-4). A computer installation in the reception area is often overlooked, but this is an ideal place to market the practice, educate patients, and introduce the "high-tech" office. The clinician should plan the location of the kiosk to follow HIPAA guidelines for ensuring patient privacy.

Operationally, this computer can run an Internet kiosk program that prevents users from manipulating the operating system or tampering with the office LAN. The system can serve the following three purposes:

1. The computer can provide new patients with a link to the practice's website, allowing patient registration forms to be completed and sent via the Internet to the office for upload into the office's practice management software, if applicable. This software should use secure file transfer protocol (SFTP) technology to ensure that E-PHI is sent with high-level encryption to maintain privacy.
2. The computer can also be used by patients as a convenience to allow free Internet access if an appointment is delayed.
3. The computer kiosk or a stand-alone computer system can be used to run a PowerPoint or Web-enabled patient education module to help familiarize the patient with services offered by the office and to provide information about the practice and office team members.

Front Desk

In a small office, the computer setup at the front desk is used primarily to process patient accounting and insurance, manage scheduling, and in some cases generate patient treatment reports. Another strategy, especially in multiple clinician practices, is to have separate computer workstations for patient intake and checkout. The addition of Internet functionality is another area where the intake computer can prove valuable.

Production of a customized "welcome letter" sent directly to the patient by mail, e-mail, or fax can help introduce patients to the practice, better inform patients about the office, and set expectations about treatment and billing practices. More offices are now creating websites that offer patient education modules and enhanced functionality; this allows tight integration of the Web materials and office software, as follows:

1. Patients can complete Web forms that transmit demographic data and E-PHI via a HIPAA-compliant protocol directly to the intake computer.
2. TruForm (PBHS, Santa Rosa, CA, and Henry Schein, Inc., Melville, NY) allows a patient to access the office website, complete the registration forms, and transmit them to the office by means of a remote host computer. The data can then populate the preregistration fields in the practice management software automatically.
3. More flexibility can be added with new software program modules that permit greater frontdesklessness. WebConnect, an integrated product by PBHS, allows patients to log on to the practice website, view and change future appointments, review financial information, pay bills online, and communicate automatically by e-mail.

Larger offices, especially in multiple-clinician practices with two or more front desk personnel, may benefit from configuring the front desk with a separate departure area and computer. This computer can be linked to a shared multidrawer laser printer so that both front desk computer stations print to a central printer, with each unit able to print specific documents or envelopes on demand.

Software

Practice management software for an endodontist has some of the same features as software for the generalist. Specialty markets, especially endodontics, have several key requirements. Considerations for hardware and networking, as well as product features (e.g., scheduling, referral tracking, preregistration, advanced reporting, and analysis), have poor analogs in general practice management software and require the selection of a product written specifically for the endodontic specialist.

Another important factor in the selection of software has to do with the recent consolidation trends in the dental software industry.[57] Careful investigation of software companies should include a look at the parent companies in an attempt to determine which ones will survive. When purchasing software, the clinician also should give careful consideration to the basic language and robustness of the program's database engine.

Great software, like a great building, is constructed on a solid foundation. Although there are many techniques for building software, some are superior.

The 2000s saw the delivery of incredible power to the desktop. A client/server model now exists that eclipses all others. Put simply, it features a powerful database engine run on a central computer (i.e., a dedicated server) that controls access to data requested by client workstations. The standard in databases for this model is SQL,[102] which delivers data faster than any other database system and does so with far greater efficiency, safety, and simplicity. Unlike other systems that move data back and forth between the network server and client workstations, client/server systems produce less network

traffic and greater performance. This is because in an SQL system, virtually all data manipulation is done at the server. This also reduces the chance of the data becoming corrupt, because data never leave the server, and the SQL engine actively controls access to the data.

In previous models, each client workstation read data from the server, manipulated it, and then wrote it back. Because no client was aware of the activity of any other client, and the network operated more or less like multiple independent systems, two or more workstations writing data to the server at the same moment was commonplace. In addition, older models looked at data one row at a time, whereas SQL engines grab chunks of data in response to queries issued by the client and send these *result sets* in their entirety to the client. The resulting performance increases in terms of speed are significant, especially with remote offices. Therefore a true 32-bit client/server product with an SQL database is recommended as a minimum requirement.

Hardware

A well-designed front desk workstation includes a computer equipped with a dual monitor card and two large-screen liquid crystal display (LCD) monitors, label printer, and high-speed Internet connection.

Operatory

The core function of an endodontic office is to provide the best clinical care for patients, yet only about 25% of dental offices today use a chairside computer workstation.[81] The chairside clinical workstation (Fig. 28-5) is the natural place to begin the most important gathering of data.

Software

The EOHR begins with thorough documentation of care at an individual site (e.g., a practice or hospital) and eventually becomes part of an individual patient's cradle-to-grave EHR.

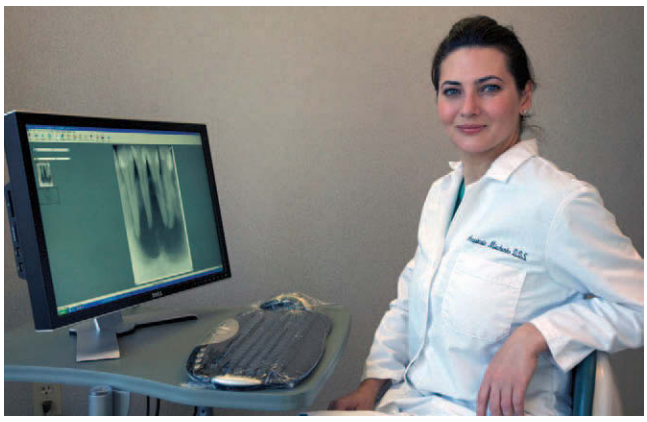


FIG. 28-5 The computer chairside workstation is the information center for today's office. A barrier-protected wireless Bluetooth keyboard and mouse improve asepsis, and the 24-inch flat panel monitor economically supports high-resolution images at 1920 × 1200 dpi with 12-bit internal processing, enabling 1.07 billion colors and improved very low grayscale tones. The remotely positioned operatory central processing unit (CPU) controls three monitors in this configuration.

Electronic charting does everything paper systems do and much more. Numerous systems are available specifically for endodontists.

Electronic charting serves three purposes: (1) accurately and legibly recording diagnostic and treatment data for individual patients,[46] (2) automating many aspects of record taking, and (3) indicating trends by allowing analysis of patient records based on user-defined queries.

An electronic charting system can include the following:

1. Integration with the general practice management software
2. Extensive trend analysis and reporting
3. Voice integration (i.e., the ability of the computer to understand natural speech and record findings)
4. Comprehensive, easy-to-use interface with pick-lists and drop-down boxes that automate entries, many of which are customizable. (Note that any fields that allow customization may not be easily data-mined for groups of users at a later time.)
5. Macroautomation (i.e., the ability of the program to automate common functions by playing back a series of recorded actions)
6. Scanner integration, which allows the user to scan in supporting documents, radiographs, and other paper records

An EOHR for endodontic visits should have the following information, arranged in a logical sequence:

1. General demographic and medical information (integrated with online pharmacologic and medical databases to allow quick lookup of medications and diseases on the Internet in real time)
2. Chief complaint and history of present illness
   a. Chief complaint (which contains a notes field for recording, in the patient's words, what prompted the trip to the office)
   b. History of present illness (notes field for recording the history of the current illness in the patient's words)
3. Clinical examination (describes findings by tooth numbers)
4. Radiographic examination (includes radiographic analysis, which is essential to measurement of treatment outcomes)
5. Etiology (list of causative factors)
6. Diagnosis and treatment plan
7. Treatment notes, which are based on user-defined criteria and include the following information:
   a. Patient's vital signs, (blood pressure, pulse, respiration and temperature)
   b. Tooth number
   c. File lengths
   d. Type of injection, gauge and length of needle, type and amount of anesthetic
   e. Dental dam clamp
   f. Number and names of canals
   g. Trial length, actual length, reference point, measurement methodology, and final instrument size
   h. Instrumentation technique, irrigants
   i. Obturation material and technique, including sealer
   j. Detailed record of restorative treatment
   k. Prescribed medications (may include over-the-counter drugs)
   l. Temporization method, post-and-core procedures and materials
   m. Recommended posttreatment instructions

8. Postoperative report generator
   a. Patient treatment reports can be created automatically and sent to referring clinicians by hard copy or e-mail (using a HIPAA-compliant protocol) (Fig. 28-6)
   b. Documentation of oral and written instructions and a reevaluation plan
   c. Contact log

In keeping with the concept of frontdesklessness introduced earlier, to reduce congestion at the front desk, a customized screen can allow the dental team to post the treatment information in the operatory to generate billing, prescription, customized instruction sheets, and patient treatment reports with a few mouse clicks or the touch of an input pen. The information then can be posted at the front desk or in the operatory, and instructions, prescriptions, and reports can be directed to the appropriate printer for hard copy production or e-mail distribution to any referrers or co-therapists.

Reference software is another area where the Internet-connected concept can prove invaluable. With each chairside computer workstation connected to the Internet, the clinician can subscribe to an online pharmacologic reference that gives specific information about pharmaceuticals as they pertain to dental treatment. One example of such a service is Lexi-Comp Online (Lexi-Comp, Hudson, OH), a site that offers information on medications that are indexed alphabetically and by disease, with specific reference to dental considerations and dental vasoconstrictor precautions.

Hardware

The computer chairside workstation is the "center of the universe" for the endodontic clinician. The central processing unit (CPU) should be placed in a location that allows easy access to connections so that upgrades can be made without major disruption. This computer should be equipped with a dual monitor card to allow two or more monitors to operate simultaneously, as follows: (1) a monitor to display the current schedule, visible only to the clinician and staff; (2) a second output that is then further split by an amplified video splitter to two monitors in "clone" configuration, allowing the clinician and staff to have identical screen views and input tools (Fig. 28-7). This HIPAA-compliant arrangement conceals the schedule and other patients' E-PHI from the view of patients in the dental chair.

In endodontics, clinical input systems represent a very broad category that can be divided into two groups: input devices and charting software. Input devices can be subclassified further into radiographic and photographic or VL imaging equipment.

Imaging

MICROSCOPIC IMAGING

Microscopic imaging has evolved over the past several years to include many choices for high-resolution image capture.

- Medical-grade, single-frame digital cameras are available from several manufacturers that can produce superior images and allow capture directly to the chairside clinical workstation via the USB[95] or the FireWire (IEEE 1394) port or a memory card. Many cameras let the clinician preview an image on the camera's integral display so that images can be quickly retaken if not satisfactory. The cameras attach to the microscope by means of a beam splitter and camera adapter. Professional-grade digital photography has replaced the 35-mm film format for medical and scientific use. It allows an instant picture with image resolution that approximates that of film-based photography.
- Solid-state analog video cameras also can be purchased for microscopic videography (Fig. 28-8). These cameras produce an analog output (either a *composite* signal or an *S-Video* signal). Composite signals with images of 280 lines of resolution usually are less expensive than S-Video models, but the images have inadequate resolution (Box 28-3). Cameras capable of S-Video output can produce images in the range of 480 lines and are preferred for microscopic imaging. They can be connected either to video capture cards for incorporation into imaging databases or to a video splitter to provide several S-Video outputs. One output can be connected to the chairside computer workstation, a second to an LCD flat screen monitor with S-Video inputs, and a third to a videocassette recorder. This arrangement allows legacy systems to operate in a multiscreen mode, but it has been supplanted for the most part by a mostly digital imaging chain. Even more sophisticated cameras with three charged-coupled devices (CCDs) and *component* outputs are available for the highest-level resolution, but they are not necessary for routine clinical documentation.
- A hybrid system that uses the newest consumer-grade video/still cameras with solid state memory allows nonlinear editing, a 16 × 9 recording ratio for widescreen playback, and instant viewing or copying. Many of these cameras can be connected directly to the computer by means of a USB or FireWire and can take credible still images in addition to 30 fps (frames per second) video.

INTRAORAL CAMERAS

Intraoral cameras that can photograph a single tooth, a group of teeth, or a patient's full face are available in digital format, using a USB interface with plug-and-play setup. These cameras are very useful for recording the condition of soft and hard

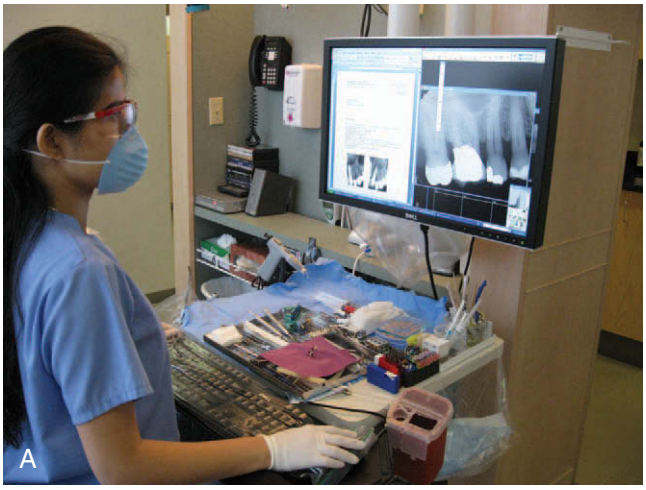


FIG. 28-6 A, Producing patient treatment reports at chairside, shown here, can lead to improved workflow.

Martin D. Levin, DMD
Anastasia Mischenko, DDS, MS
Practice Limited to Endodontics

Martin D. Levin, DMD, LLC
5454 Wisconsin Avenue, Suite 815
Chevy Chase, Maryland 20815-6901
Phone: 301-654-6077
Fax: 301-654-0021
info@endocc.com
www.endocc.com

Philip Jones, MD
1234 Second Street NW, Suite 306
Washington, DC 20036-7689

Dear Dr. Jones:

Thank you for the referral of Ms. Wendy Patient for endodontic evaluation of her maxillary right dentition. If we can be of further assistance, please let us know.
Patient date of birth: 04/11/1948; **Patient gender:** Female
Patient home phone number: 202-333-5555 **Patient office number:** 202-444-6666
Tooth/teeth number/numbers: UR
Date of treatment: 05/14/2009
Diagnosis: necrosis with internal resorption; acute periradicular abscess
Medical history/medications: 05/14/09: NKDA; Synthroid 50 mg, Lipitor 10 mg, clindamycin 300 mg q6h (for current dental infection)
CC/HPI: "Abscess. I have swelling and off-and-on fever. It has been a problem for a few days. I am traveling to Africa tomorrow."
Comments: The patient's oral temperature was 99.4; indurated swelling maxillary right alveolus, extended between teeth #s 2 and 4. Two buccal fistulae were present (a). A periapical radiograph (b) and a focused field CBCT were exposed. See orthogonal CBCT images below: (c), axial; (d), cross sectional; (e), curved planar; and (f), 3D reconstruction. Tooth #2 shows a unilocular radiolucency at the mesio-buccal root that appears to be continuous with the periapical radiolucency at #3 and extends to the periapcial radiolucency at tooth #4 (20.4 mm). An internal resorptive lesion (green arrow) on the palatal root of tooth #3 and two draining sinus tracts to the cortical plate (yellow arrows, one in the axial view of this section) were noted. Teeth #s 2 and 4 were vital.
Plan: Referred to Dr. Oral Surgeon for extraction and biopsy.
Attending endodontist: Martin D. Levin, DMD, Diplomate, American Board of Endodontics
Cc: Dr. Oral Surgeon

05/14/2009

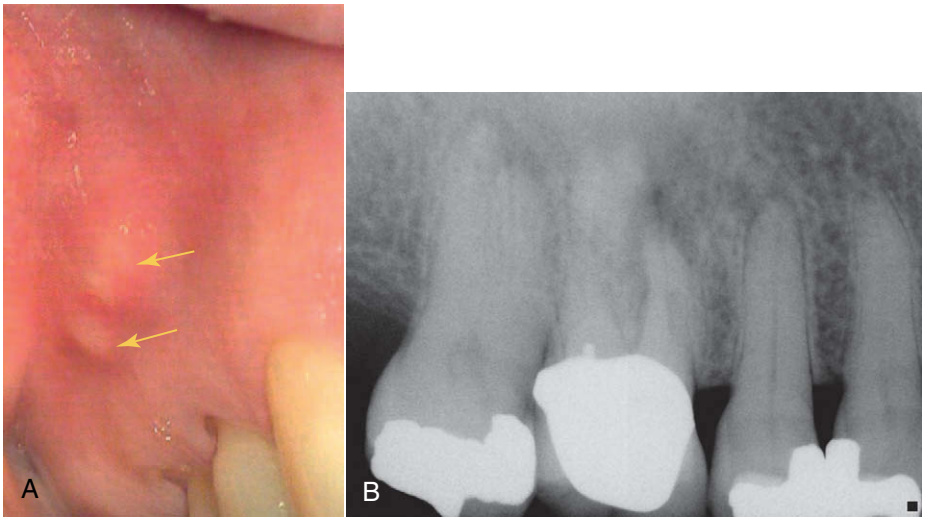


FIG. 28-6, cont'd B, Preformatted patient treatment reports can be automatically created and printed in the office on a variety of printers using a wide array of software. This patient treatment report can be sent by e-mail from a preformatted template using a Health Insurance Portability and Accountability Act (HIPAA) compliant pick-list.[16]

Continued

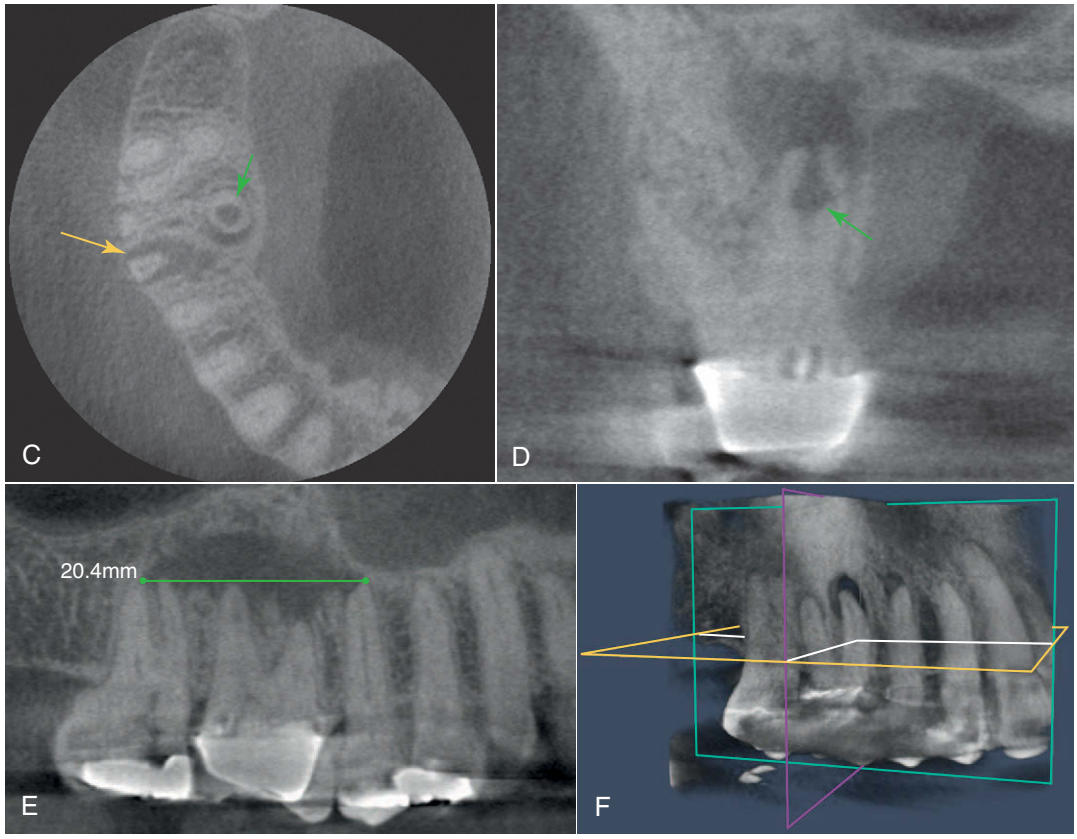


FIG. 28-6, cont'd



FIG. 28-7 This operatory is equipped with four flat panel monitors. This arrangement allows input from the clinician and assistant to any screen. The assistant and schedule screens are located to the rear of the patient in compliance with Health Insurance Portability and Accountability Act (HIPAA) guidelines. The doctor's chairside workstation screen and the ceiling-mounted entertainment/patient education screens are in full view of the patient. The entertainment/patient education screen are able to show music or movie DVDs and VL images from the microscope via a switch.

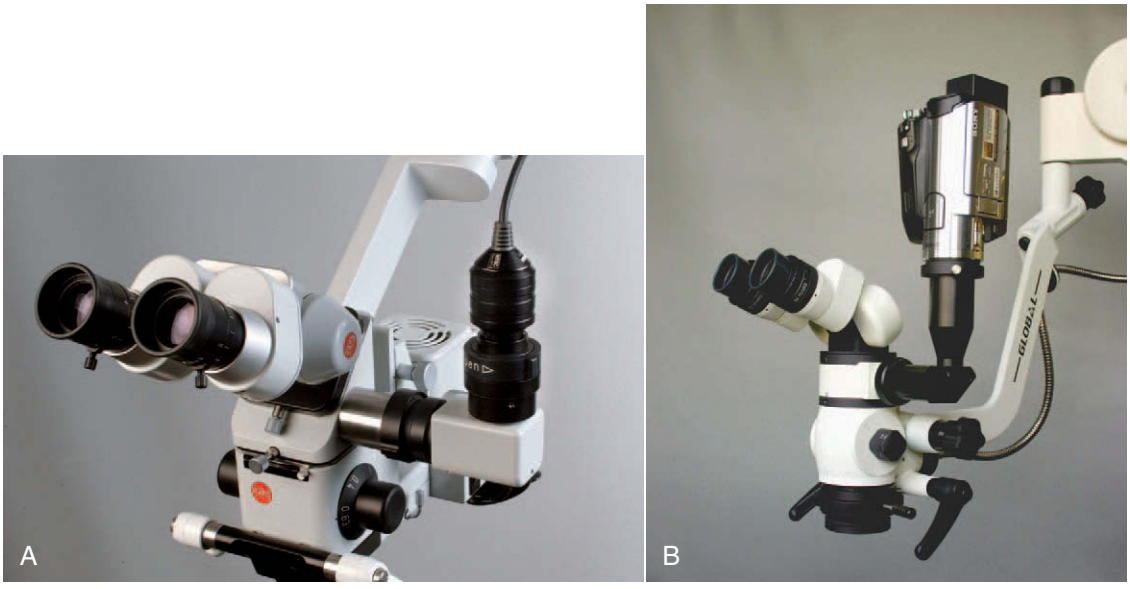


FIG. 28-8 A, This microscope incorporates a miniature analog solid-state camera (Y/C format, 480 lines) into the beam splitter. **B,** A solid-state video camera captures either still images or video at 30 fps (frames per second) in high-definition (HD) format. (***A*** *courtesy JEDMED Instrument Co, St Louis, MO;* ***B*** *courtesy Global Surgical Corp, St Louis, MO.)*

BOX 28-3

Short Primer on Video Signals

Visible light (VL) imaging is one of the most powerful documentation tools available to the clinician for recording the patient's dental condition. Most operating microscopes can accommodate a wide range of analog and digital VL imaging equipment. Although analog cameras are still popular for microscopic documentation, digital technology is increasingly competitive, especially as the consumer video market converts to digital equipment.

Video cameras generate red, green, and blue (RGB) signals to create video images. If the RGB signals were sent as three separate signals, huge storage and bandwidth capacities would be required. Instead, it is possible to take advantage of the human visual system to reduce storage requirements. This is done by transforming the RGB signals into new video signals that can be band limited with minimal loss of perceived picture quality. In Europe, the dominant television standards are PAL and SECAM. The United States uses three common National Television Standards Committee (NTSC) analog signal formats:

- *Composite video* (single 75-ohm cable terminated at each end with RCA connectors). With composite video, the red, blue, and green signals are mixed together. This architecture is present on almost all contemporary home video equipment; when modulated with audio onto a radio frequency (RF) carrier, it is used by over-the-air digital broadcast stations or on coaxial wire by cable TV systems. These systems have the lowest-quality signals.
- *S-Video*, or super-video (looks like a single cable but internally has two 75-ohm coax or twisted pair cables terminating at each end in a four-pin DIN connector). S-Video involves the transmission of video signals over a cable through division of the video information into two separate signals, one for color (i.e., chrominance) and the other for brightness (i.e., luminance). Professionals refer to this as *Y/C video*, rather than S-Video, because the former is more descriptive of the signal format. These systems produce midrange quality signals.
- *Component video* (analog component video uses three 75-ohm coax cables that terminate at each end in RCA connectors, usually color coded and bundled together). Component video reduces artifacts and color errors by minimizing the number of video-signal format conversions between the source and the display device; it transmits its video signal by dividing the Y, R-Y, and B-Y signals separately. These component video signals produce the highest quality signals.

In professional and industrial video equipment, impedance-matched 75-ohm BNC connectors are used for all these signal formats. Also, each of these analog signal formats can be stored, processed, and transported in the digital domain in professional applications. Digital home satellite systems receive MPEG compressed digital component signals and provide component, S-video, HDMI outputs. The digital versatile disk (DVD) format, and the newer "Blu-Ray" format, also based on MPEG technology, is available with analog component video and HDMI interfaces for the highest possible picture quality.

Computer monitors, on the other hand, are designed for RGB signals. Most digital video devices (e.g., digital cameras and game machines) produce video in the RGB format, so the images look best when shown on a computer monitor. When seen on a television, however, these images look better in S-video format than in composite format. To use S-video, the sending and receiving devices and the interconnect cable must be S-video compatible. The newest connections between computers and monitors is the DisplayPort interface standard, which supports both RGB and component encoding formats. DisplayPort connections are interoperable (capable of supporting analog and digital interfaces), can drive display panels directly, eliminating scaling and control circuits that allow cheaper and thinner displays.

tissues for documentation and for patient education and "co-diagnosis" (allowing the patient to visualize problem areas and participate in the evaluation). When VL images from the intraoral cameras are transmitted directly to a clinical chairside workstation, they can be stored along with radiographic images in the patient's imaging database for easy retrieval. This type of camera can be a valuable adjunct to the microscope-mounted camera because of its lower overall cost. Another advantage of intraoral cameras is the ease with which they can be positioned (Fig. 28-9), especially by dental auxiliaries; a microscope, on the other hand, can be somewhat difficult to position and use with mirrors. Intraoral cameras are available with barrier sheaths to prevent cross-contamination.

A new intraoral camera (Fig. 28-10, *A*) is now available for endodontic offices and features a unique liquid lens technology that instantaneously focuses like the human eye. It features a CMOS sensor that captures still images (1024 × 768) and video (640 × 480) with the highest resolution currently available, and supports a focus range from 1 mm to infinity (Fig. 28-10, *B*). The video output includes the following options: USB 2.0, TV-NTSC, TV-PAL, S-Video, and VGA. Using eight light-

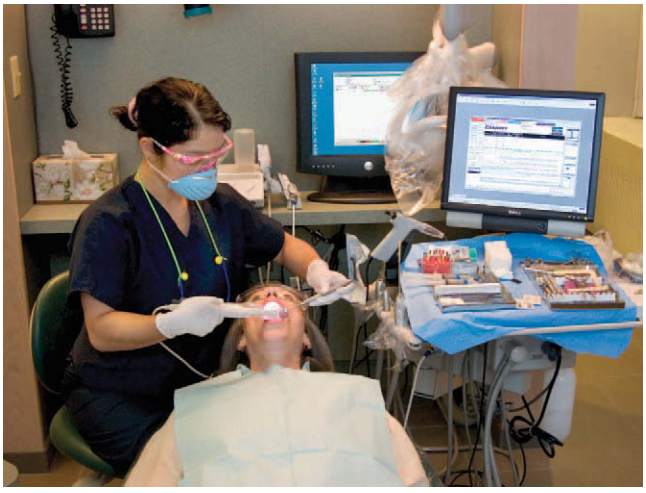


FIG. 28-9 Intraoral cameras can be used efficiently by the staff to deliver images directly to the universal serial bus (USB) port of the chairside computer workstation. *(Courtesy Schick Technologies, Inc., Long Island City, NY.)*



FIG. 28-10 A, The Kodak 1500 wireless intraoral camera captures still images and video using a new liquid lens technology. Coupled with its "WiFi"-enabled docking station, the new camera can transmit high-resolution images anywhere in the office. **B,** An occlusal preoperative image of the mandibular right first molar showing a defective amalgam; buccal, lingual, and distal marginal ridge fractures; and significant occlusal wear. *(Courtesy PracticeWorks, LLC, Atlanta, GA.)*

emitting diodes (LEDs) to provide illumination, the system automatically adjusts to lighting conditions to optimize illumination and contrast levels. The intraoral camera provides the advantages of auto-focus of still images and video, elimination of undesired light reflection by using a unique polarization filter, and automatic adjustment to lighting conditions.

This camera can substitute for microscope-mounted VL imaging hardware. Unlike older wireless technologies, this camera uses "Wi-Fi" connectivity, which enables reliable wireless performance and easy integration with and without a computer. The small docking and charging station allows the camera to be used anywhere in the practice, encouraging camera sharing across multiple operatories.

FIBEROPTIC IMAGING

The introduction of *fiberoptic* and *rod-lens endoscopes* specifically for use in the oral cavity has taken endodontic imaging into the root canal and periapical space.[9,10] Both the fiberoptic and the rod-lens endoscope works in conjunction with a camera, light source, and monitor. The clinician views the treatment field on the monitor, which allows the entire treatment team to see the site. A documentation device can be added to the endoscope's visual system to record the endodontic procedure.

The fiberoptic endoscope is used for intracanal visualization (Fig. 28-11, *A*). The device is composed of 10K flexible fibers that transmit light to a treatment field and images back to the camera. The fiberoptic scope's lens has a diameter of 0.8 mm with 0 degrees of angulation.

Two types of rod-lens endoscopes are used for endodontic procedures (Fig. 28-11, *B*). The model that is 4 cm long and has a lens 4 mm in diameter with 30 degrees of angulation can be used for both conventional and surgical endodontic visualization. Another model, which is 3 cm long and has a lens diameter of 2.7 mm with 70 degrees of angulation, is used primarily for endodontic surgical procedures.

DIGITAL PHOTOGRAPHY

Intraoral and perioral digital imaging provide valuable documentation tools for clinicians. Many clinicians routinely take images of their patients for identification purposes with consumer-grade digital cameras. However, a dedicated model with specific features is recommended for documenting the patient's intraoral and perioral condition for patient education purposes, consultation with colleagues, teaching, and other professional uses.

The following criteria should be considered when purchasing a digital camera:

1. High resolution is important to ensure good image clarity. Although the size of the camera's image sensor and monitor quality are constantly improving, cameras that can produce an image of at least 1600 × 1200 pixels are recommended.
2. The camera should have macro capability (i.e., should be adjustable from about 2 inches [5 cm] to infinity) to ensure that oral and full-face images are distortion free and in focus.

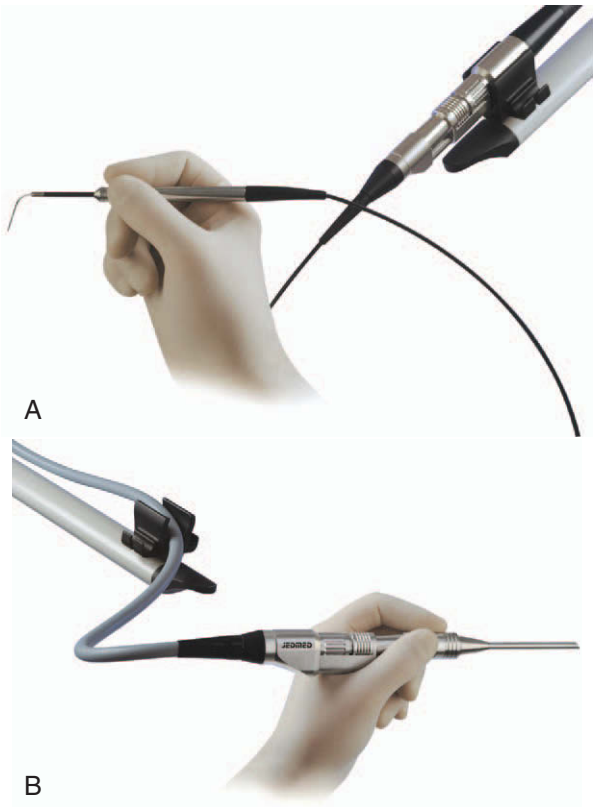


FIG. 28-11 A, Fiberoptic endoscope used for intracanal visualization. *(Courtesy JEDMED Instrument Co, St Louis, MO.)* **B,** Rod-lens endoscope used for conventional and surgical endodontic visualization. *(Courtesy JEDMED Instrument Co, St Louis, MO.)*

3. Lighting is an important consideration in the selection of a digital camera. For this reason, either a flash diffuser or a ring flash is advised to ensure consistent illumination, make sure color values are accurate, and prevent shadows and highlights that are too bright (Fig. 28-12).

The advantages of a digital camera include savings on the cost of film and processing, absolute archiveability, instantaneous evaluation of the image, and the ability to catalog and search the image database. In addition, the clinician can print multiple, identical, inexpensive high-quality copies in just a few minutes. The standard methods of image transfer in the industry are direct wiring via the USB or FireWire port and use of small memory cards. Any TWAIN-compliant[94] (standard software protocol and applications programming interface that regulates communication between software applications and imaging devices) digital radiography or image organizational software allows the clinician to store these images directly in the patient database.

RADIOGRAPHIC IMAGING

The value of digital radiography in dental and medical diagnosis is well known, especially during operative procedures where time to image is important. Key advantages of digital radiography are: reduction in radiation dose to the patient; patient education; image optimization and computer-aided feature extraction;[96a] workflow improvement; the avoidance of shipping, darkroom, or chemical processing errors; environmental



FIG. 28-12 Specially configured digital cameras can record oral and perioral conditions. Cameras can transfer images via USB and memory cards. *(Courtesy PhotoMed International, Van Nuys, CA.)*

waste reduction; improved electronic communications[68]; image archiving; and projecting a technologically advanced practice image.[21,68] Solid-state digital imaging using CCD or complementary metal oxide semiconductor (CMOS) detectors has become an increasingly popular technology for intraoral and extraoral dental radiography. Three methods of producing digital images are available: direct solid-state detectors (e.g., CCD/CMOS detectors[35,77]), indirect photostimulable phosphor plates,[97] and indirect secondary capture of conventional film by scanning.[27] The most useful technology for endodontic practice is the direct model, because digital detectors can produce an image in seconds, often at a lower radiation dose than film.[13]

A CMOS detector is literally a camera on a chip (Fig. 28-13, *A*). Because it has a full digital interface, the analog signals from the scintillator are translated to digital signals by the chip. Simply stated, CCD systems require many supporting components (e.g., timing generators, shuttering and signal-processing chips), whereas CMOS detectors integrate all these functions in a single chip. Any computer with a USB port can accept this detector, and a separate processing board is not needed. Several manufacturers produce CMOS detectors of x-radiation, integrate the radiographic image, and initiate the readout of the pixel data.

Taking this technology a step farther, Schick Technologies (Long Island City, NY) manufactures a wireless sensor, the CDR Wireless (see Fig. 28-13, *B*). This device sends a radio frequency signal to a nearby remote receiver on the 2.4 GHz band that produces an image with the same quality as the wired version. 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.

Storage phosphor plate technology allows indirect production of radiographic images using a semiflexible, phosphor-coated plate. With the DenOptix system (Gendex, Des Plaines, IL), plates are loaded into a sheath, exposed, mounted in a carousel, and then placed in the scanner. As many as eight intraoral images are ready for viewing in about 1 minute if the lowest-resolution scan is used. The plates are available in periapical, panoramic, and cephalometric sizes, and they support a wide range of exposure settings. The advantages of this

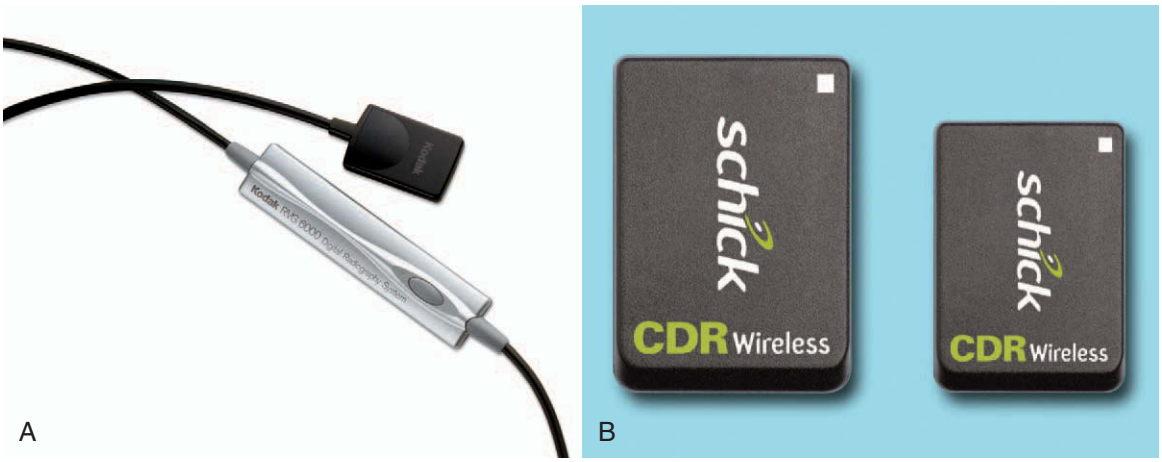


FIG. 28-13 A, Complementary metal oxide semiconductor (CMOS) direct sensor technology is literally a camera on a chip that connects to the chairside workstation via the USB port, thereby eliminating the processing board. **B,** Wireless CMOS sensor transmits images to the chairside workstation by 2.4 GHz radio frequency. *(**A** Courtesy PracticeWorks LLC, Atlanta, GA; **B** courtesy Schick Technologies, Inc., Long Island City, NY.)*

system are that very thin plates are equivalent to film in size, and placement characteristics are very similar to those of film because there is no wire on the plate. The main disadvantages are longer processing times (compared with only seconds for CCD/CMOS sensors), light sensitivity during transfer to the scanner, and the additional handling steps of erasing and sheathing the individual plates between uses. For individual intraoral images, the Air Techniques Scan-X processor (Air Techniques, Hicksville, NY) can process the initial image in less than 20 seconds and subsequent images in about 4 seconds. New intraoral plates have a surface protective layer but can still degrade as a result of scratching during use.

For the truly paperless office, a film scanner (Box 28-4) is necessary because it allows input of historical, film-based radiographic images. Both dedicated film scanners and flatbed scanners are available for digitizing radiographs. The ScanRite dedicated film scanner (ScanRite Systems, Fremont, CA) provides a single image in about 10 seconds (Fig. 28-14). This high-resolution image can be enhanced and imported into any TWAIN-compliant software through the USB port on the computer.

Consumer-level flatbed scanners can be used to scan film-based images, but these require transparency adapters. Flatbed scanners also can be used to scan paper documents and append them to the patient's digital chart. Nevertheless, a dedicated single-purpose scanner with a document feeder is recommended for paper documents.

The value of instant digital imaging in the modern dental office is well known, especially for endodontic and implant procedures. Many researchers have concluded that digital x-ray sensors equal analog film for diagnostic tasks, with the added benefits of immediate image production, image feature enhancement and the increased security of offsite archiving.[24] Conventional film provides a finer grade of detail through continuous shades of grayscale images, rather than the discrete pixels of individual gray levels used by digital systems. Advances in digital imaging continue, such as improved resolution, enhanced digital subtraction radiography,[72] and active surfaces approaching those of film, and in the not-too-distant future, digital image quality may surpass that of film.



FIG. 28-14 The ScanRite (ScanRite Systems, Fremont, CA), a dedicated film scanner, can improve workflow because it is optimized to scan film-based images and automatically correct for brightness, contrast, and color.

To produce the highest quality digital radiographic image, consideration must be given to the entire "imaging chain" (i.e., the x-ray generator, subject, sensor, computer interface, and monitor). *The quality of a digital image depends on each step of the process, and the weakest link degrades the final result.* The following sections discuss basic considerations for a better understanding of digital imaging.

IMAGE QUALITY

One of the most critical and most misunderstood issues concerning digital representation of image data is clinical performance. Computer screen selection, ambient lighting,[52] and image compression all affect the image; however, for the purchasing decision, the most compelling issue should be the image quality of dental structures as perceived by the clinician.

Clinical performance depends on numerous interrelated factors and can be expressed as detective quantum efficiency (DQE), the measure of noise and contrast expressed as a func-

BOX 28-4

Dental Film Scanning

A scanner can be used as a complement or an alternative to a sensor-based or phosphor plate–based digital radiography system. Scanned film images can be saved in digital radiography system software, patient management software, or third-party imaging software (such as that included in the Windows XP and Vista operating software).

Complementary System

To complement a sensor- or phosphor-based digital radiography system, both the scanner and the system software must be TWAIN compliant. This allows images to be scanned directly into the electronic oral health record (EOHR). Some system software even names the file automatically to simplify the process.

Alternative System

Scanners can be used as an alternative to a sensor- or phosphor-based digital radiography system because the investment cost is lower, and the image quality can be excellent. If the office is networked or is wired for monitors in the operatories, the image can be scanned at any location and shown on any computer linked to the network. Two major classes of scanners are of interest to the dental clinician: (1) the relatively inexpensive *flatbed scanner* and (2) the more expensive *dedicated film scanner*.

Flatbed Scanners

Certain models of flatbed scanners offer a transparency adapter to allow scanning of radiographic film. Flatbed scanners typically are best used for documents, photographs, panoramic films, and cephalometric films. Flatbed scanners usually are slow to preview and slow to scan, and their image quality and resolution are inferior to those of dedicated film scanners.

Film Scanners

Dedicated film scanners typically are best used for film-based images. The ScanRite (see Fig. 28-14) is a fast, high-quality film scanner specifically designed to input periapical and bite-wing films, mounted 35-mm color slides, and 35-mm color film. The primary considerations in choosing a dental film scanner are resolution, levels of gray scanned, speed, software, and convenience. Scanner resolution typically is quantified as dots per inch (dpi). Higher resolutions result in better clarity when images are enlarged. *Optical resolution* describes the capability of the hardware. *Interpolated resolution* is the ability of the scanner software to place additional dots among the data scanned. Image quality can be adversely affected by "noise" during image processing of either a scanned or direct digital image.

The levels of gray or number of colors scanned is important because each scanned dot is assigned a "color." The more grays or colors scanned, the better the image quality. With fewer grays or colors, images are "blotchy." This can be demonstrated by adjusting the computer monitor settings to a low color palette or depth while viewing a digital image.

The scanning speed depends on many factors. Better scanners have a built-in buffer memory so that the data do not have to be sent in multiple "bursts" to the computer. Better scanners scan in one pass without stopping. Scanning speed also depends on the resolution selected, the physical size of the original, whether color or grayscale is used, the speed of the microprocessor, and the amount of random access memory (RAM) in the computer.

The better scanners include software with the capability to change the color, select film calibrations, sharpen the image, rotate the image, scale the output, match the output (i.e., gamma level) to other software applications, invert colors or grayscale levels, and save settings for various types of originals. Most scanner software allows an image to be previewed before it is saved to disk; however, this small preview image makes it difficult to make image enhancements accurately. Dedicated film scanners save time by automatically optimizing the brightness, contrast, and color for each scanned image.

tion of object detail. Noise is an inevitable product of the digital imaging chain and is reflected in the signal-to-noise ratio (SNR). Systems with a high SNR produce the best image quality. Compensating for poor SNR is possible—to a point—with increased radiation, but this increasing dose negates one of the key advantages of digital systems. Moreover, excessive dosage can lead to detector saturation and reduced image contrast. *Contrast performance* is the ability of the system to display the actual contrast of an object, and the ability to make window and level adjustments is potentially one of the true benefits of digital radiography over film. Digital detectors generally have a wide dynamic range, with thousands of shades of gray; therefore they might depict areas that might be overexposed or underexposed on film. Window and level adjustments are needed to display all the subtle contrast variations in a digital image, just as a bright light might be used to evaluate an overly dense analog film's radiographic detail.

The ideal is to produce a high DQE by combining low noise and high contrast so that small low-contrast objects can be detected. Another potentially significant advantage of a high DQE is that digital systems can produce better small, low-contrast object detection than film with the same radiation dose. In the future, improving the DQE (high DQE results in low-noise images with high object detectability) coupled with advanced image processing algorithms and other postprocessing improvements will lead to enhanced visualization.[31,40] According to one expert,[28] "DQE is useful when it comes to accurate measurement for endodontic purposes"; however, the DQE is largely theoretical because few dental studies have actually used it.

Simple comparisons of limiting spatial resolution (LSR), the spatial frequency at which the observer can no longer detect a high-contrast test pattern under laboratory conditions, and digital detectors are problematic because there is a point of diminishing returns. The amount of signal captured per pixel is reduced as pixel size becomes smaller, although the amount of noise remains relatively constant for each system. Increasing the number of pixels can result in a lower SNR at each pixel, which limits small object detection. Even though sensors may have LSRs as high as 20 to 22 line pairs per millimeter (lp/mm) when measured using high-contrast lead grids, the smallest object that can be detected is usually larger

in clinical circumstances. In addition, noise and contrast combined with limitations of human vision's poor response to high spatial frequencies limit small object visibility. Nevertheless, the latter problem is somewhat obviated by the ability to zoom-enlarge digital image details. Unfortunately, as low-contrast details such as lateral root canals become even thinner in dimension, there might not be sufficient contrast to detect them, regardless of the system's spatial resolution. There is no free ride. A low DQE, even with a high LSR resulting from high noise and low contrast, may remain a limiting factor.

Furthermore, the quality of many film-based images may be compromised by operational problems such as chemical and film freshness, developing inconsistencies, light leaks, and shipment handling. Gross and moderate caries can be detected with both conventional film and digital imaging systems with a great degree of surety. Studies[50,101] showed relative equality between digital and film-based radiographs for caries detection. Another study[91] compared the accuracy of caries detection in film-based, storage phosphor, and CCD sensors. They found that the diagnostic accuracy of film was comparable to that of digital systems. The study also noted that the dentist's ability to evaluate the radiographs accurately was the most significant variable in radiographic diagnosis. Unfortunately, the presence of caries is always more extensive than depicted by either digital or film systems. Incipient caries and some periapical lesions continue to present a challenge for both film and digital-based systems.

Caries detection on interproximal surfaces has been further refined with the development of smart software that uses a software algorithm approved by the U.S. Food and Drug Administration (FDA), originally developed for the defense industry. This predictive software, the Logicon Caries Detector (PracticeWorks, LLC, Atlanta, GA), produces three types of diagnostic aids to improve caries detection by up to 20%.[38]

In the detection of periapical bone lesions created in cortical and trabecular bone, some have stated that no difference was seen among E-speed film, CCD, and CMOS sensors.[74] Furthermore, the study noted that "cortical bone lesions were detected with significantly higher accuracy once the junction of the cortical plate was involved or perforated."[6] However, caution must be exercised when evaluations are made using artificially created lesions.

Digital detectors produce images with a dynamic range of 8 to 16 bits. A typical 12-bit system records 4096 shades of gray. Because most computer monitors can display only 8 bits, or 256 shades of gray, some of the data are lost in the transformation. If these data are processed linearly instead of by sophisticated algorithms, some useful data are discarded.[26] These types of algorithms are commonly used in most modern digital systems to maintain the clinical usefulness of the images produced.

RADIATION

Silver-halide film records the interaction of x-ray photons with electrons in the film emulsion.[30] The photons are either attenuated (absorbed or scattered) or transmitted (along with some scatter) and reach the film emulsion to create a latent image. This image is then chemically processed to reveal the viewable radiograph. Film images are characterized by continuous shades of gray between the extremes of black and white.[76] However, film requires a relatively high dose of x-radiation to achieve the same result as a direct digital detector. According to van der Stelt,[96] "the primary principles of radiation protection in diagnostic radiology are justification (the patient will experience more benefit than harm from the exposure) and the 'as low as reasonably achievable' (ALARA) principle." Despite the fact that radiation exposure can be reduced by using thyroid shields, the highest-speed films, and rectangular collimation, studies consistently demonstrate that radiation hygiene is not practiced by dentists to its fullest benefit. In 2006, medical x-rays, especially medical CT and nuclear medicine imaging, constituted nearly half of the total radiation exposure of the U.S. population from all sources (Fig. 28-15). New guidelines issued in 2003 by the National Council on Radiation Protection (NCRP) in its Report No. 145 supersedes the previous report published in 1970.[68b]

If the states adopt the newest NCRP recommendations (Box 28-5), as is commonly done, most dental offices would not be in compliance. Two terms used in the report have been specifically defined. The terms *shall* or *shall not* indicate that adherence to the recommendation would be in compliance to the standards of radiation safety. The terms *should* or *should*

BOX 28-5

National Council on Radiation Protection Recommendations

1. Dentists must examine their patients prior to 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.

From the National Council on Radiation Protection and Measurements: Radiation protection in dentistry, Report #145, Bethesda, MD, 2003, NCRP. Available at <ncrponline.org/Publications/145press.html>. Accessed July 17, 2009.

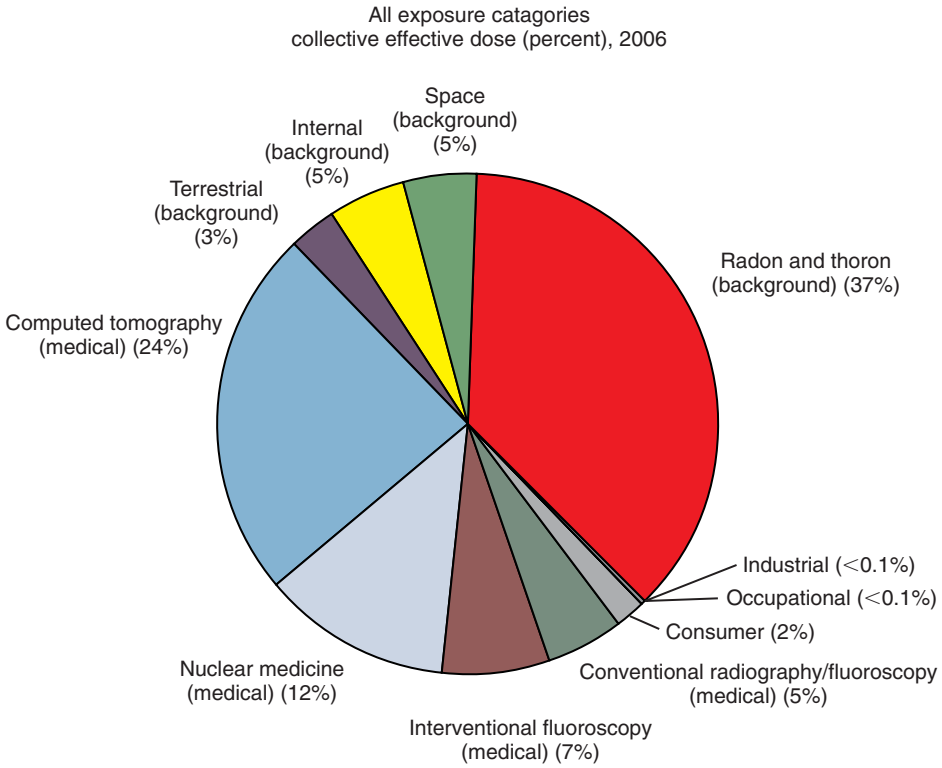


FIG. 28-15 Natural and manmade sources of radiation. *(From National Council on Radiation Protection and Measurements (NCRP) Report No.160, Ionizing Radiation Exposure of the Population of the United States, 2009.)*

not indicate prudent practice and acknowledge that exceptions may be made in certain circumstances. In addition, there are also nine new recommendations for image processing of conventional film in the NCRP report. A strong argument can be made for clinicians to switch to a direct digital radiography system to avoid all the drastic changes necessary to assure compliance with the new recommendations.

Digital intraoral imaging generally requires less radiation than film-based systems, a key public health benefit. According to the ADA Council on Scientific Affairs, digital imaging reduces radiation exposure by 50% to 90%.[5] Film scanning provides no reduction in radiation, because a conventional film image must first be produced.

Although digital radiography promises to reduce radiation for each image exposure, the real radiation a patient receives in relation to the diagnostic information gained remains controversial. In studies by Versteeg et al. and Sommers, technique errors for digital detectors were higher than film-based systems.[87,98] The most common difficulty, according to focus-group studies, was found to be sensor positioning. These findings dramatically point out the need for careful and ongoing training of dental technicians in the art of taking radiographs with digital sensors—a critical step in taking full advantage of the benefits of reduced radiation for each image exposed.

Placement errors aside, most CCD and CMOS-APS detectors have a similar range or exposure latitude to counteract under- or overexposure. Direct digital detectors take about 100 times more exposure to produce a black image than a light gray image, or a 100:1 ratio. In comparison, film has about a 1000:1 ratio, and PSPs have a range of 10,000:1.[99] Although the benefit of PSPs' wider range of exposure latitude may "save" an occasional overexposed image, PSPs can lead to inadvertent excessive radiation exposure, because overexposure will less likely produce poor image quality as a warning.[11] Strict adherence to preset exposure settings will continue to require diligence to reduce radiation exposure. Additional reduction in radiation to special-needs patients (e.g., patients with developmental disabilities or are severely mentally handicapped) may also be realized because of the advantages of digital detectors.[29]

Common dental projections rarely if ever deliver a measurable absorbed dose to an embryo or fetus, but any radiologic procedure on a pregnant patient should be conducted so as to minimize the radiation dose to the pelvic region.[47] This procedure should be conducted only if x-ray exposure is required for adequate diagnosis and treatment. If dental care is to be delayed until after delivery, exposure should also be delayed. Also, usual and customary radiographic selection criteria should be applied for patients who are trying to get pregnant, breastfeeding, or have received radiation therapy to the head and neck for medical purposes (the radiation dose from dental radiography is insignificant when compared to therapeutic dosing).[39,64]

Even though restriction of an intraoral dental x-ray beam is mandated by federal law to be a circle no greater than 7 cm, rectangular collimation has proven to significantly reduce radiation dose to the patient. The ADA Council on Scientific Affairs states that the "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…

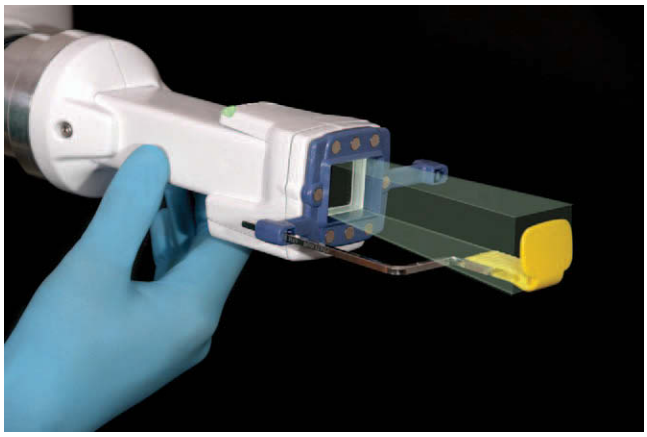


FIG. 28-16 In compliance with the recommendations of the National Council of Radiation Protection and Measurements (NCRP) guidelines, reducing radiation for periapical and bitewing radiographs can be accomplished by using a rectangular collimation device to assure that only productive ionizing radiation reaches the patient. When compared to conventional round collimation, the Tru-Align system restricts the beam size to approximately the size of a #2 periapical film or digital sensor, thereby reducing skin surface–area dose by a factor of 5 and overall radiation to patients by as much as 65% to 70%. *(Courtesy Interactive Diagnostic Imaging Inc, Atlanta, GA.)*

unnecessary radiation exposure to the patient."[5] In a recent study by Seltzer et al.[83] comparing a new rectangular collimation system, Tru-Align (Interactive Diagnostic Imaging Inc, Atlanta, GA), with conventional round collimation, the rectangular collimator (Fig. 28-16) reduced skin surface–area dose by a factor of almost 5. The Tru-Align system, deemed easy to use, generally gave perfect alignment with only occasional cone-cuts.

Personnel monitoring is required in all states to assure compliance with established ionizing radiation limits. According to the Maryland Department of the Environment guidelines,[88] monthly badges are required for the first 6 months of employment to document that radiation will not exceed 10% of the legal radiation limit set by the state. Ionizing radiation dose limits for Maryland of 5000 millirem for workers, 500 millirem in 9 months for pregnant women,[47] and 100 millirem for the general public are mandated by state regulation. All dosimetry badge reports must be maintained at the office for review as proof of minimal radiation exposure. Variations of these requirements are found in each state, so familiarity with the controlling laws is mandatory.

QUALITY ASSURANCE

Clinicians should ensure that their practices take all necessary steps to produce consistently high-quality radiographs with minimal ionizing radiation exposure to patients and staff. Dental office quality assurance involves periodic evaluation of the entire imaging chain, including the x-ray generator, film procurement and storage, developer,[30] darkroom, view boxes, and protective aprons. Administration of quality assurance requires assigning responsibility, training, evaluating results of testing, and taking corrective action.[53] Ohio, for example, requires each registrant to maintain a site-specific written quality assurance program with published requirements for how often x-ray generators need to be tested and calibrated and so on.[73]



FIG. 28-17 Using large interactive displays improves the patient's understanding of their condition, leading to a better treatment experience. Here the clinician is explaining a periradicular lesion shown by focused-field cone-beam computed tomography.

PATIENT EDUCATION

Most people of college age and older are visual learners, and presentations that include both visual and auditory modalities are superior to visual or auditory modalities alone.[32] Consider the educational value for the patient able to view a large, high-resolution radiograph in conjunction with the clinician's diagnostic explanation (Fig. 28-17). Once the patient understands their dental condition (e.g., caries, periradicular pathosis), treatment acceptance becomes more assured, and the patient experience is enhanced.

WORKFLOW IMPROVEMENT: A WORK MULTIPLIER

Digital radiography is a work multiplier; it replaces human effort, making tasks the practitioner and staff members perform easier and more error free. In some cases, it makes new tasks possible.[19] DDR images take less time to expose, duplicate, retake, and transmit, all with less radiation and environmental impact. With solid-state systems, any operative procedure, such as endodontic therapy and implant placement, can be performed in less time. The diagnostic yield of digital imaging systems will improve as enhancements are made to image acquisition, but also to our understanding of human vision and cognition. Image processing can improve diagnostic outcomes by defining diagnostic issues and designing tools to achieve specific goals.[68a] Also, visual information such as digital radiography and visible light images (i.e., intraoral photography, microscopy) is easier to access than film-based images and improves office operations by increasing efficiency. PSP plates take at least 8 seconds to process after the detector is transported to the developing site and unwrapped, whereas CCD/CMOS-APS detectors are virtually instant, with "paint times" usually just a few seconds. Some workflow advantages are shown in the following comparisons.[73a]

OPTIMIZATION

All digital radiographic systems allow for some image optimization (Fig. 28-18, *A*). Postexposure image processing routines differ among manufacturers. Some produce a raw image for which automatic enhancement routines can be turned on and off, whereas others produce images that are filtered automatically. Most software features the ability to change contrast and brightness to view images that are underexposed or overex-

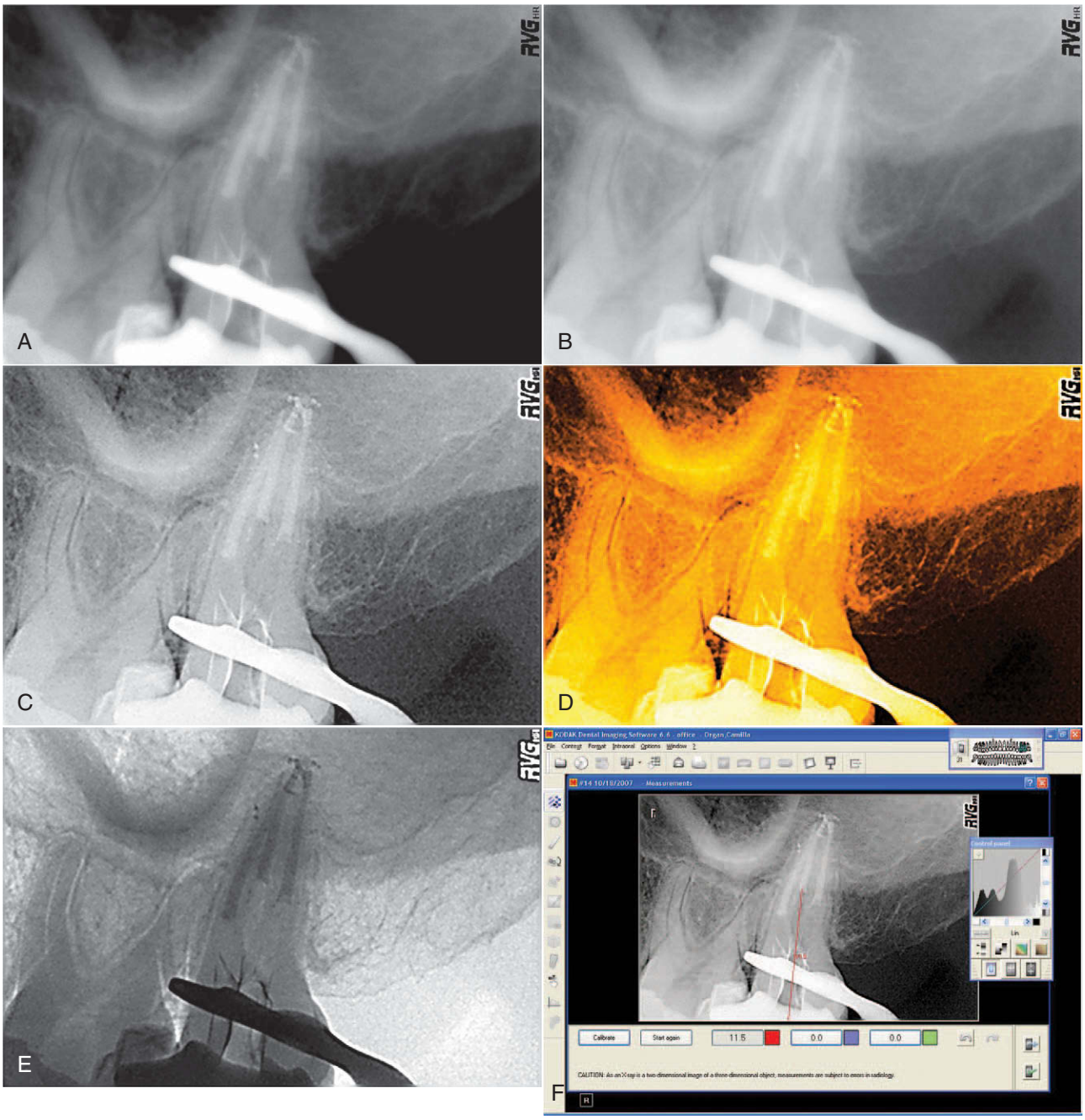


FIG. 28-18 Optimization of digital radiographs. **A,** Raw periapical image. **B,** Window and level adjustments, similar to contrast and brightness, have been used to reduce the effects of overexposure. **C,** A filter has been applied to enhance readability. **D,** The image has been colorized to further differentiate the elements of the radiograph. **E,** Image reversal can sometimes improve readability. **F,** Once calibrated, the measuring tool can be helpful for measuring root lengths and distances to anatomic sites, but accuracy is not assured because of parallax error.

posed, as well as to equalize density, magnify (see Fig. 28-18, B), create an inverse image (see Fig. 28-18, E), and allow for image rotation and mirror imaging. Another enhancement, pseudocoloring, ascribes false colors based on the brightness of pixels and can help with patient visualization of images.[54] A number of systems also have algorithms that can sharpen and enhance caries imaging, and the future promises even more improvements.[70]

Filtering tools are available in all commercially available software, but comparison is difficult because proprietary names do not reveal the underlying algorithms. Investigators[55] studied the functionality of 13 software programs and suggested more standardization of filtering tools to improve exchange, enhancement, processing, and analysis. They further suggested that public-domain image processing and analysis software, similar to software adopted for medicine by the National Institutes

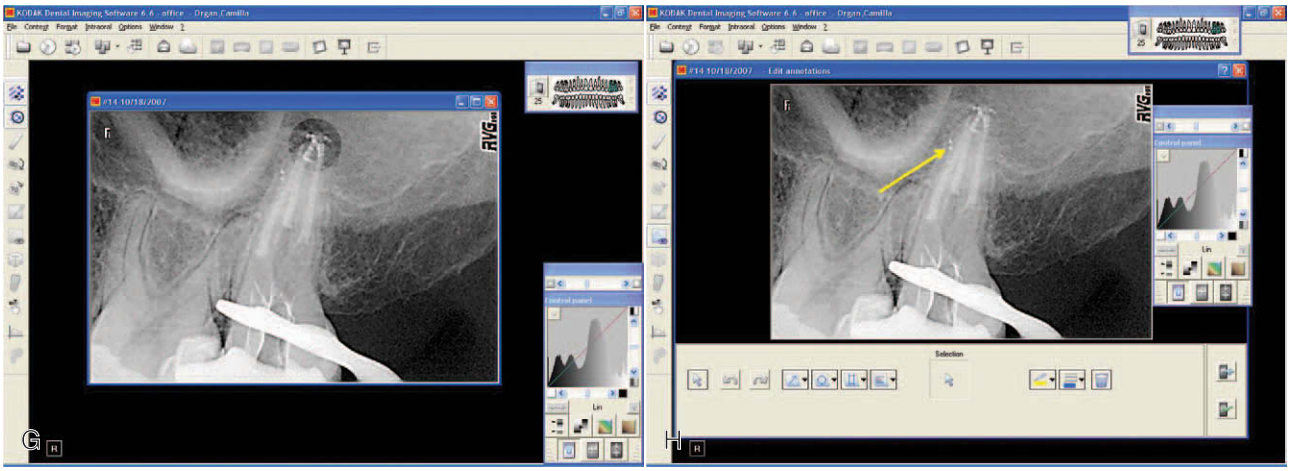


FIG. 28-18, cont'd G, Certain regions of interest (ROI) can be highlighted with a preset contrast tool that can be moved around the image to highlight specific areas. **H,** ROI can be annotated with arrows and drawing tools. *(Courtesy Dr. John D. West, Tacoma, WA, and PracticeWorks, LLC, Atlanta, GA.)*

of Health (NIH), may provide the basis for more standardization.[70]

MEASUREMENT

The three types of measurement generally available with digital imaging software are (1) linear measurement, the distance between two points in millimeters (see Fig. 28-18, *F*); (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 error play a significant role in all two-dimensional radiographic measurement accuracy, both film and digital systems are subject to parallax error. However, a study[22] 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." This was true even though, as the authors pointed out, the measurement differences may not be clinically significant. Sophisticated calibration algorithms are under development, and accurate measurement of parallel images should be more feasible in the future.[13]

SECURITY

Although film-based images can easily be produced in duplicate, each subsequent rendition of the image is reduced in quality. Digital images, on the other hand, can be reproduced in unlimited number because the images can be stored and produced without any loss of detail. Furthermore, digital images can be stored on and off site and on many types of media, which helps prevent permanent loss from theft, fire, or other causes.

Paradoxically, the ease with which digital images can be reproduced and stored allows for alteration of radiographs without a trace.[25,100] Of course, this has always been possible with duplicated analog film images, but the issue has not been commonly addressed. The ease of producing an altered image is controversial, but most digital vendors on the market have safeguards that greatly minimize the possibility of this occurring. In addition, standards such as Digital Imaging and Communications in Medicine (DICOM; discussed later in the chapter) help protect image integrity through the use of digital signatures in the form of tagged attributes attached to each image.[25] Archiving radiographic images in a secure site in DICOM format using removable media such as DVD and tape is one of the current solutions to this problem. These types of magnetic and optical media can be stored off site by a third party archivist for extra protection.

These data are also vulnerable when sent via the Internet to a discussion site. Two common technologies, Secure Socket Layer (SSL)[1] and digital certificates (VeriSign, Mountain View, CA), can provide at least 128-bit encryption and virtually eliminate the chance of alteration or fraud. SSL connections to Internet servers can be recognized by the prefix *https://* in the address line. In any event, such material should be carefully secured to fulfill HIPAA requirements regarding privacy.

Another important security consideration is the database engine used to store the files on the server. Among the most robust systems are SQL-based models, which allow for client-server configuration with industry-standard software protocols. Images that are stored and retrieved cannot be deleted, nor can patient identification be changed, without opening a separate administration utility, which can be password protected on the server.

IMAGE STORAGE AND COMPRESSION

All computer-based images are voracious consumers of hard drive space, and various manufacturers have used a number of techniques to reduce image size. For example, Schick Technologies allows acquisition of each image with the advanced compression technology of the JPEG4 codec. Clinicians can also compress selected groups of images between certain dates with up to four different compression schemes, allowing for *lossless* (no visible change in image clarity with approximately 50% to 75% reduction in file size) to *lossy* (significant reduction in file size with some degradation of image resolution) compression. Lossy compression is not recommended for dental grayscale images.

MARKERS

Occasionally, radiographs and VL images need a tag so the clinician can return to them later to assemble teaching or

patient education materials. Most software today allows markers to be placed so that a computer search can call up the images efficiently. These customized tags can be useful in organizing cases that require special follow-up to evaluate postoperative healing. In addition, readily available software enables the clinician to create notes and diagrammatic annotation of important features of an image (see Fig. 28-18, *D*, *F*, and *G*).

COMPUTER INTERFACE

Many digital sensors connect to the chairside computer workstation via the USB port. As of 2004, only Schick Technologies had produced a wireless sensor that transmits the image data by radio frequency to a nearby receiver and thence to the USB port, eliminating any sensor wire. The direct USB connection eliminates any computer interface board, which simplifies installation and maintenance. Laptop configurations add flexibility if the clinician wants to move the equipment between different operatories and office locations or use it for off-site procedures at hospitals and nursing homes. Laptop configurations also benefit from the elimination of special cards for sensor connection. System backup strategies are particularly important for laptops because of the increased risk of theft or of being dropped.

X-RAY GENERATOR

According to Miles, the image chain is only as good as its weakest link, and digital systems work best with a modern x-ray generator.[66] Images from digital detectors are optimized if taken with an x-ray generator that has (1) an extremely accurate timer, especially for short exposures,[51] (2) low mA and kV settings, and (3) a direct current (DC) circuit. Farman,[28] however, contends that the x-ray generator has not been shown to have a significant effect on digital radiographic quality.

FOCUSED FIELD CONE-BEAM COMPUTERIZED TOMOGRAPHY IN ENDODONTICS

Endodontics is an image-guided specialty, and until recently, endodontic radiographic diagnosis relied on two-dimensional radiographic assessment. The introduction of cone-beam computerized tomography (CBCT), and especially recent refinements such as very small field of view (FOV) or focused-field devices, is revolutionizing the visualization of the dentition and the maxillofacial complex (see online Chapter 29).

The shortcomings of panoramic radiography have been well understood for many years. They are flat, two dimensional, superoinferior or posteroanterior images that suffer from superimposition of all structures imaged and distortion and magnification errors. Therefore, measurements of objects on panoramic images are inaccurate. CBCT images, on the other hand, capture anatomic entities in three dimensions (3D) and can be viewed by digital selection of the region of interest (ROI) with great accuracy. In recent research published by Stratemann et al.,[89] linear measurements of skulls comparing calipers and CBCT imaging revealed only a 1% relative error.

According to one source,[41] "these machines are smaller than medical CTs, less expensive to purchase and operate, produce a fraction of the radiation dose, and possess an open architecture that, generally, eliminates patient complaints of claustrophobia." Unlike periapical radiography, CBCT scans enable the visualization of the "true extent of lesions and their spatial relationship to important anatomic landmarks."[15] Comparing periapical radiographs (PAs) of 156 maxillary posterior roots with small field CBCT images prior to surgery, investigators[61] found that these CBCT images showed 34% more lesions than were visible with PAs alone (Fig. 28-19). Numerous papers have demonstrated that CBCT imaging is superior to PA radiography in detection of periradicular lesions. Researchers[37,67] created artificial osseous defects and conclusively demonstrated the improved detection rate of bony defects with CBCT imaging. Other researchers[59] reported that in 70% of cases that were evaluated with both PA and CBCT, CBCTs provided clinically relevant information not visible on PA radiography. Other researchers have reported that CBCT, which can be used to measure the grayscale values of lesions, may provide a better, more accurate, and faster method to differentially diagnose a solid from a fluid-filled lesion or cavity. This may allow the clinician to decide whether or not periradicular surgery is necessary without waiting for a long recall period to see if healing has occurred.[84]

Newly introduced hybrid units like the Kodak 9000 3D extraoral imaging system (PracticeWorks, LLC, Atlanta, GA) have two digital detectors, one for digital panoramic imaging and one for focused-field digital volumetric tomography (Fig. 28-20). Aside from panoramic radiographs, this unit produces 3D images of an area measuring just 37 mm × 50 mm (approximately a quadrant), with less radiation than a standard CBCT and improved readability because of its new higher-resolution detector. Unique to the Kodak 9000 3D system is the highest-resolution sensor available at this time, with a voxel (*vol*ume pix*el*, representing a quantity of 3D data similar to a pixel representation in 2D data) size of just 0.076 mm. While not necessary for use in every case, when appropriate, this technology will improve visualization and ultimately lead to better care. This is because imaging the maxillary posterior region poses unique challenges, where roots of teeth overlap and anatomic structures such as the zygomatic buttress and maxillary sinus are present.

Medical CTs necessitate a significant radiation dose to patients, with a typical scan of the maxilla and mandible of 2.00 millisieverts (mSv), or as much as 345 panoramic images. On the other hand, large FOV CBCTs will image the same area at 0.068 to 1.073 mSv or 3 to 44 panoramic equivalents.[63] The focused-field FOV CBCT will further reduce radiation exposure to 0.005 to 0.038 mSv[62] because of its limited coverage and low mA technique factors (Table 28-2).

Integration of third-party digital radiography and VL imaging products is very important to the modern endodontic practice, especially if the goal is to create a paperless environment. Several open platform applications specific to dentistry are available that allow the clinician to acquire, store, annotate, manipulate, and output many types of images. Comprehensive packages such as ViperSoft (Integra Medical, American Fork, UT), TigerView (Televere Systems, Los Gatos, CA), and Kodak Dental Imaging Software (PracticeWorks, LLC) work with analog and digital still and video cameras, scanners, and digital radiography programs. Once an image has been acquired, these sophisticated software programs allow the clinician to label and annotate the image, transmit it by e-mail, and even simulate the repair and restoration of teeth (i.e., cosmetic imaging). As operatory-based computers become the norm, dental imaging systems are becoming mainstream, allowing secure

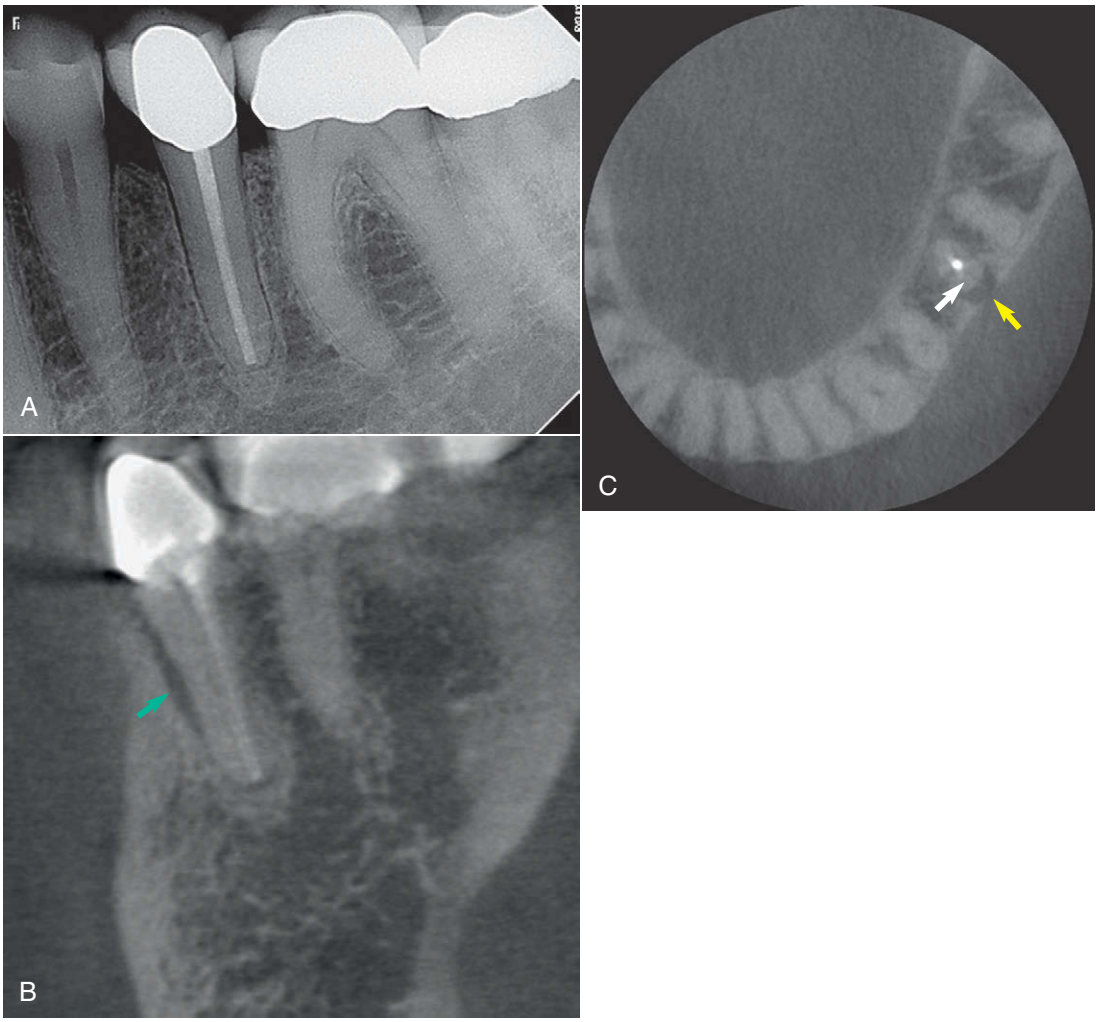


FIG. 28-19 A digital periapical radiograph **(A),** showed previous endodontic treatment, slight widening of the periodontal ligament on the mesial, and a periapical condensing osteitis. Two CBCT images: **B,** sagittal view *(green arrow)* demonstrated significant periradicular widening of the periodontal ligament, which extended to the buccal aspect and appeared to perforate the buccal plate; **C,** axial view *(yellow arrow)*. While the image showed streak artifact on the mesial and distal of tooth #20, a vertical fracture was evident on the direct buccal that extended from the CEJ to the midroot *(white arrow)*. This example demonstrates the improved visualization of periradicular pathosis with 3D technology.

network e-mail transfer of images in several industry standard formats (i.e., DICOM, TIFF, BMP, JPEG, and TGA). Before purchasing any software, including imaging software, the clinician should confirm that it is HIPAA compliant (transaction types, standards, and code sets) and DICOM conformant to the requirements set forth in DICOM Supplement 92 (2004).[18]

Imaging software can be an invaluable aid for cataloging images for easy retrieval, case presentation to other dentists and patients, analysis, and magnification of images for improved diagnosis. Sophisticated practice management software should integrate smoothly with these systems, extending the functionality of the office software and creating a complete EOHR.

Sterilization Area

The computer in the sterilization area can be used to create, print, or transmit patient treatment reports, to order supplies, for general communication duties, and as a backup for the front desk system. One advantage of creating the report in the clinical area is that the staff member who assisted with treatment can individualize the report as necessary. Once a template has been created and the software populates the preset fields, radiographs and VL images can be inserted and then sent to an approval queue to be inspected by the clinician in the private office, or an assigned staff member can print or e-mail it. Some practice management software programs are designed to create these reports at the chairside computer workstation and send them directly to predesignated printers and specific paper trays. Report generation at the chairside promises to further streamline the patient treatment report process.

Administrative Area

The generation of marketing materials; bookkeeping, payroll, and banking functions; bulk scanning; and special projects can be assigned to the administrative computer. Although small offices often use the front desk computer for these tasks, the increased utility of a separate computer for special projects cannot be overemphasized.



FIG. 28-20 The Kodak 9000 3D extraoral imaging system has two digital detectors, one for panoramic imaging and one for focused-field digital volumetric tomography. This DICOM-compliant unit takes 13.5 seconds to capture a CBCT dataset of 461 basis images that are then reconstructed to create a focused-field 3D volume of 37 mm × 50 mm. *(Courtesy PracticeWorks, LLC, Atlanta, GA.)*

Clinician's Office

The clinician's office should be equipped with a workstation that allows entry into all the programs on the network. Ample connections for uploading camera images and digital video sequences, for Internet access, and for a smart phone are also recommended. The smart phone modules available from some vendors allow the clinician to take home a synchronized database of patient and referring clinician information, the appointment schedule, and images (Fig. 28-21). This facilitates after-hours emergency communication and allows notations made to the patient's chart to be synchronized with the office database the next time the smart phone is connected to the LAN.

Real-time video conferencing and collaboration can also be handled from the clinician's office computer. Addition of an Internet camera with audio capability is all that is needed to communicate with a colleague via the Web.

COMPUTER INDUSTRY STANDARDS

Graphics and Imaging

For most clinicians, graphics and imaging is a confusing area. Windows has many graphics standards; it has been difficult to sort out which ones to use, but this is beginning to change. Although some dental imaging software stores graphics data in nonstandard ways, most products support computer industry norms. It is less important that clinicians understand the peculiarities of the different graphics storage categories than which ones to look for in a program. The importance of standards here is simply that if the graphics are stored in a standard manner, clinicians will be able to manipulate an image and drop it directly into the dental software. Some of the common image formats are:

- *Bitmap (BMP):* This is the Microsoft standard for Windows. It is used and understood by almost all imaging software and is the format of most pictures delivered with a Windows computer. Its strengths are universal distribution and very high quality. Its chief weakness is that the files it creates are not compressed and take up more space.

TABLE 28-2

Effective Doses From Various Dental X-Ray Procedures

Radiation Source*	Millisieverts (mSv)	Time Period for Equivalent Effective Dose From Natural Background Radiation (United States is 3 mSv/year)
CBCT (focused FOV)	0.005-0.038	1-5 days
CBCT (large FOV)	0.068-1.073	9-131 days
Panoramic†	0.010-0.026	1.2-3.2 days
Cephalometric	0.026	3.2 days
Full-mouth intraoral (18 F speed films)‡	0.150	18.3 days
Bitewings (4 F speed films)‡	0.038	4.6 days
Intraoral periapical radiograph	0.005	0.61 days
Maxillomandibular CT	2.000	243 days
Maxillary CT	1.400	170 days
Mandibular CT	1.320	160 days
CT abdomen	10.000	3.3 years
Chest (PA film)	0.020	2.4 days
Average galactic radiation in Denver, CO (per year)	0.510	62 days

CBCT, Cone-beam computed tomography; *CT,* computed tomography; *FOV,* field of view; *PA,* posteroanterior.

*Radiation doses vary considerably within and between different brands of equipment.

†With respect to digital panoramic radiographs, there appears to be no dose reduction compared with film-based panoramic images, because there is already a large dose reduction as a result of the use of intensifying screens in the panoramic cassettes.

‡There is approximately a 50% decrease in effective dose when comparing digital imaging and "F" speed film.

Effective doses from various dental x-ray procedures adapted from the Health Physics Society: <http://hps.org/publicinformation/ate/faqs/dentalpatientissuesq&a.html>. Accessed July 17, 2009; American Dental Association: <http://www.ada.org/public/topics/xrays_faq.asp>. Accessed July 17, 2009; and White SC, Pharoah MJ: *Oral radiology: principles and interpretation,* ed 6, St Louis, 2009, Mosby.

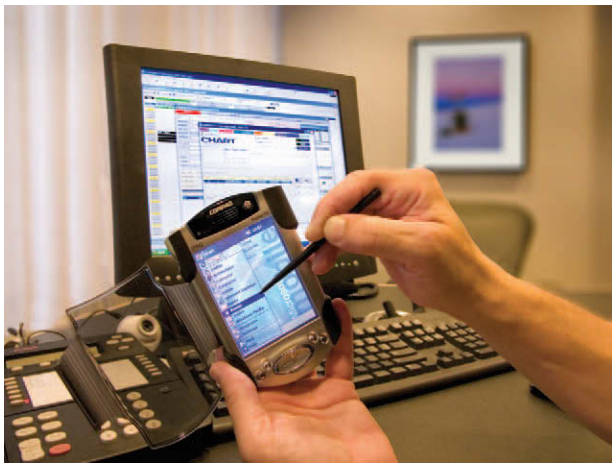


FIG. 28-21 Clinicians can take the patient database out of the office with a smart phone linked to the office database. *(Courtesy Henry Schein, Inc., Melville, NY.)*

- *Joint photographic experts group (JPEG or JPG):* This is probably the most popular standard for PCs and the Internet. It has good quality and supports compressed formats (allowing for smaller files). When JPEGs are resaved, they are recompressed; therefore, care must be taken to limit the number of times the image is saved, or it must be saved in a different format (e.g., TIFF). JPEG 2000, the new image coding system, uses wavelet technology that allows improved compression and smaller-sized files.[92]
- *Tagged image file format (TIFF or TIF):* This is also a very popular standard because the images are highly flexible and can be used with many different image processing applications.[6] The images are usually very high quality and typically support minimal compression.
- *Graphic interchange format (GIF):* This is a very compressed format that is most popular on the Internet and online services, but it is not helpful in storing digital images.

Open Database Connectivity

One of the classic problems in the computer industry is that each vendor stores and manipulates its data differently. As a result, data can be read only if tools created by the same company are used. To correct this problem, Microsoft introduced open database connectivity (ODBC). With ODBC in place, software programs connecting to databases first connect to ODBC (another software product, called a *driver*), and ODBC connects to the database. This makes ODBC (and other derivative standards, such as JDBC and OLE DB[36]) a translator between software programs and databases. The advantage of ODBC is that software developers can plug into different databases without changing their program codes. In addition, ODBC-compliant databases are accessible from many programs.

ODBC is helpful to clinicians because it allows them to read and analyze the database from spreadsheet programs, report writers, graphics programs, and other database programs. In today's open-standards world, *it is unwise to purchase any product that is not ODBC compliant*.

Digital Imaging and Communications in Medicine

DICOM is a set of international standards established in 1985 by the American College of Radiology (ACR) and the National Electrical Manufacturers Association[17,69] (NEMA) to address the issue of vendor-independent data formats and data transfers for digital medical images.[75] 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 or Windows).[19] 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), as well as 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 DICOM standard also supports a form of SSL (see section on security) for the transmission of digital output. 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 continuity. Most software vendors are striving to achieve full DICOM compliance, and some have achieved at least partial compliance.

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.[3] 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.

Systematized Nomenclature of Dentistry

Systematized nomenclature of dentistry (SNODENT) is an ADA-sponsored, comprehensive set of standardized terms for defining dental disease in an electronic environment. SNODENT is part of a larger code set called *systematized nomenclature of medicine* (SNOMED),[86] which was developed by the College of American Pathologists. The codes allow dentists to document a wide range of conditions and risk factors using standard terms to aid communication among dental and medical clinicians. In addition, SNODENT allows dentists and researchers to better assess the long-term benefits of certain treatment protocols by standardizing the measurement of outcomes and the documentation of evidence-based care.[65]

INTERNET

The Internet offers current and prospective patients and referrers an opportunity to learn about the office at their convenience. The Web has consistently proved itself a cost-effective communication and marketing tool in dentistry (Fig. 28-22). The right content mix, one that is unique and well targeted to

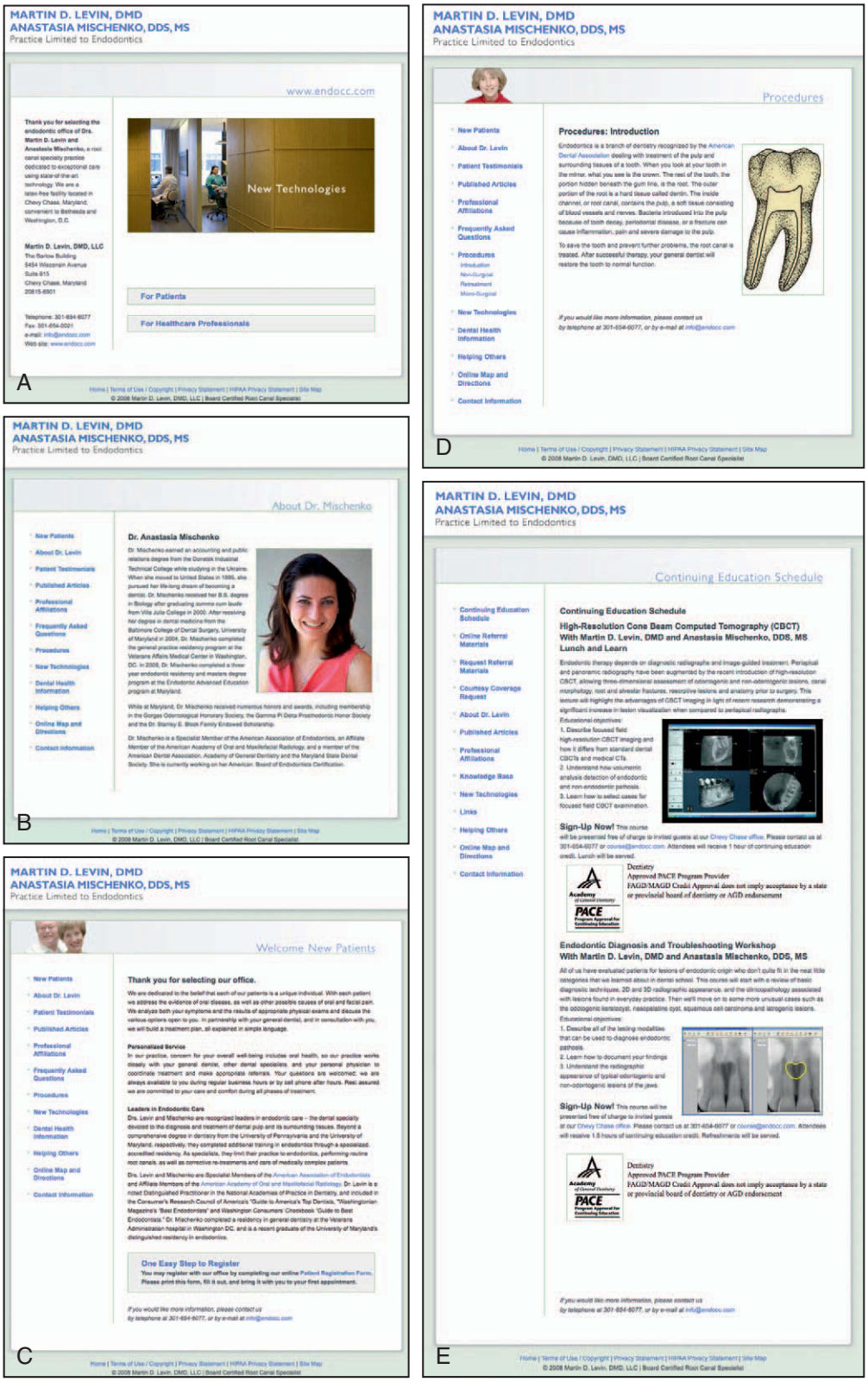


FIG. 28-22 The Internet has revolutionized office communications. **A,** The main index page of www.endocc.com, the author's endodontic office website, shows the esthetic possibilities of a professionally designed site. **B,** This page helps viewers learn about the clinicians, their educational qualifications, and professional accomplishments. **C,** The "Welcome New Patients" page introduces the practice and displays the website navigation bar on the left pane. **D,** Patients can view animated illustrations and video sequences designed to help them understand procedures. Here, the nonsurgical phase of therapy is explained. **E,** "For Healthcare Professionals" module enhances communication with other clinicians by including continuing education offerings, templates for transmission of patient referral materials, professional affiliations, a knowledge base, links to other sites and contact information. *(Courtesy Atlantic Design LLC, Playa del Rey, CA.)*

the needs of prospective and current patients, can improve both the image of the practice and patient satisfaction.

Getting started on a website can be a daunting task, because the possibilities are virtually limitless. The following steps can help the beginner get started:

1. The best way to begin is to visit other medical and dental providers' websites. The clinician should learn how to navigate the sites and should determine the elements that are required and which high-end graphic effects might prove effective. Simple is better, but patients want to see an esthetically pleasing site, and they want to learn something on each visit.
2. The clinician should then decide on a set of goals for the site; for example, should the site generate referrals via Internet search engines or serve as a resource for patients and referrers?
3. A design for the site should then be created. It should project the practice's image or brand and should fulfill patients and referring clinicians' needs. All design elements should be consistent with other marketing materials to further the practice's brand identity.
4. The next step is to outline the proposed site and create a storyboard that includes navigation possibilities for the audience. Each page is designed on a separate piece of paper, and these ideas are tested with friends and selected prospective users.
5. A uniform resource locator (URL) must be chosen so that the site address can be easily remembered and entered in a browser with minimal effort and confusion. Again, the URL should tie in to the practice's desired image.
6. The blanks on the web pages are then filled in with copy, photographs, and an appealing style that projects the practice's vision. Copyright laws apply to most material on the Internet; therefore written permission must be obtained before using protected material.
7. The website must be kept fresh. To draw repeat visitors, it must be updated continually, and all links must be relevant and functional. Some dental Internet vendors allow the clinician to edit the site on an as-needed basis 24 hours a day, 7 days a week, by adding a small applet that allows password-protected entry.

An endodontic office website can contain searchable educational material for study clubs and referrers. Websites can be transformed into a communications center, a scientific reference library, and clinical and business information resource. This can provide fast, convenient access to resources such as MEDLINE, online continuing education courses, product catalogs, practice management information, and pharmacology references.

Many vendors currently provide service for site construction and hosting that include creative patient education modules, maps with customized directions, clinician biographies, and animated procedure descriptions. Some sites also include referral forms that automate attachment of digital radiographs and promote interactive discussions about treatment.

New patients can benefit by submitting forms directly to the practice's management software via an always-on Internet connection. Clinicians can customize referral forms to provide data fields that *require* inclusion of certain information for submission, so that patients can provide more complete registration information.

IMPORTANT SOFTWARE FEATURES

Financial Security

Although fraud in dental offices is not often discussed, even conservative statistics indicate that it is a significant problem. No absolute solution exists; therefore vigilance is the key. However, the clinician should do at least three things[56]:

1. *Become educated.* Clinicians should find out how people embezzle from a dental office. Sometimes this means taking a course or two, hiring a fraud expert to review office systems, or both.
2. *Pay attention to the business.* Too many dental professionals abdicate the function of their front office to trusted "office managers." This dynamic, in which the owner of the business ignores the business, is ill conceived and invites fraud.
3. *Select software with a security log.* The practice should use an office computer system that carefully tracks all deletions, edits, and other potentially suspicious behavior automatically and prevents anyone except the clinician from viewing the security log. In addition, each user is logged on and off the system, so that the software knows when a particular person was on the system; what station the person was using; and which transactions they created, edited, or removed. Most security experts recommend that each financial transaction (i.e., routing slips) be sequentially numbered without the possibility of modification or deletion.

Referral Tracking

Sophisticated referral tracking and trend analysis are crucial to the success of a specialized practice. These capabilities are lacking in software created for the general dentist. Software designed using SQL tracks financial and other statistics associated with referrers in the practice at the moment transactions are posted. Thus the statistics are always current and accurate. Key features of referral tracking include:

1. Real-time statistics. The software should give the clinician up-to-the-second statistics (without the need to run reports) in several key areas:
   - Production
   - Collections
   - Adjustments
   - New patients
   - Patient visits
   - Teeth treated
   - Production/collections ratio (the collections percentage)
2. Real-time ranking of all referrers relative to all others in any of the previously mentioned categories.
3. Sophisticated, flexible reports based on production, collections, procedures, and other factors that can be filtered and sorted.
4. Generation of correspondence, labels, and envelopes to one referrer or a selection of referrers based on queries.
5. Availability of referrer data from the patient screen, the schedule, and other key areas so that the clinician can

make quick decisions and see the juxtaposition of referrer and other system information.
6. Charts and graphs on referrer and patient activity associated with referrers.
7. Tracking of multiple-referrer categories:
   - Referral source (i.e., general dentist, patient, phone book, website, etc.)
   - General dentist (track multiple office locations and group name identity of referring doctor)
   - Dental specialists (i.e., periodontist, oral surgeon, orthodontist, etc.)
8. Collection of the previously mentioned data by multiple facilities of the referrer. In other words, if the referral source has multiple facilities, the clinician should be able to track statistics for each of those facilities and summarize a consolidated set of numbers for all facilities.
9. Collection of the previously mentioned data by practice. If the practice uses several referral sources, this feature allows the clinician to assess statistical data by practice.

Preregistration

Using a specialized system, some software can preregister a patient (i.e., the process of recording information for patients referred but not yet seen by the clinician) at almost any level of detail. The major features associated with preregistration are:

1. The ability to record all pertinent data about a referred patient—including insurance, referral source, and medical conditions—before an appointment is made. Ample notes fields are available for recording comments from both the patient and other clinicians. A direct link to e-mail and home and work phone icons facilitates quick communication. A "welcome letter" and information about the patient's first appointment can be produced automatically and sent out with a brochure.
2. An integrated link to the schedule allows the clinician to easily drag and drop the preregistration record into the appointment book, complete with all the relevant information from preregistration.
3. Automatic correspondence can be sent to the patient after creation of the preregistration record.
4. An integrated link to the registration module means that the system can automatically create both the patient record and the insurance records without reentering the data. Sophisticated algorithms continually update insurance data based on previous cases submitted.

REFERENCE

Infrastructure and Connectivity

Single Locations: Local Area Networking

LANs allow the sharing of data and network-attached devices to improve workflow efficiency. The simplest form of LAN is the peer-to-peer model. Connected by a hub and simple wiring, two or more computers can network to share resources and data. The drawbacks of this type of system are the limited number of workstations that can be adequately supported and the necessity to restart the entire system if a program fault occurs. Peer-to-peer systems are not recommended. A much more robust solution requires a dedicated server connected with cabling to each workstation via a central hub. The utility of a network cannot be underestimated. For example, a central repository of digital radiographs allows clinical chairside workstations in each operatory to call up all patient images so that a patient does not have to be in the same operatory for each subsequent visit. A server-based LAN has several additional advantages. It provides greater reliability and faster overall speed; it allows individual stations to be rebooted without restarting the entire system; it secludes the server in a safe location; and it supports a large number of workstations at one or multiple sites. Addition of enterprise-level virus, Trojan, and worm protection is another benefit of the server. With this feature, the server can go online daily to automatically download the latest virus definitions from the manufacturer and allow client machines to self-update as preset by the clinician.

Multiple Locations: Wide Area Networking

The efficiency of the client/server model also allows practices with multiple facilities to set up high-speed communications between those locations, keeping the database on a single server. Maintaining a single database at one location means that updates to patient and billing data are instantly visible practice-wide, reducing errors and consequent liability. But maintaining a single database also requires high-speed access to that data from all practice and remote (e.g., laptop) locations.

The features, power, and complexity of today's software solutions have forever retired dial-up modem banks as a remote-access option. High-speed access is no longer an option, but a requirement. Interconnected offices have access to various options when it comes to high-speed wide-area connectivity. Typically, there is a trade-off between speed, cost, and reliability. For example, a T1 circuit, which is available in "point-to-point" format or as a conduit to the Ethernet, has moderate speed, is generally highly reliable, and is reasonably expensive. Lower-cost options such as DSL or broadband (cable, FIOS), often targeted more at consumers, offer higher speed but often with less reliability than their more-expensive counterparts. Keep in mind that offices in remote or rural locations will have fewer connectivity options than offices in cosmopolitan, higher-tech areas.

Virtual Private Networks

As part of an office's connectivity package, Internet access is now effectively a requirement. Leveraging the Internet as a communication medium makes data security concerns imperative. This is easily accomplished by implementing firewalls at each internet-connected office location and deploying industry-standard, affordable Virtual Private Networks (VPNs)[48] between these locations (see Fig. 28-23).

VPNs use the Internet as their long-distance backbone, leveraging TCIP/IP 60 internet protocols to effect communication. Essentially, the process looks like this:

1. Each office obtains local internet service, connecting it to an Internet service provider (ISP). A VPN is configured at each office's firewall to encrypt data going to other offices.
2. The ISP passes the signal through the Internet, occasionally through another ISP, to the second office. VPNs are private because the signal that passes through the very

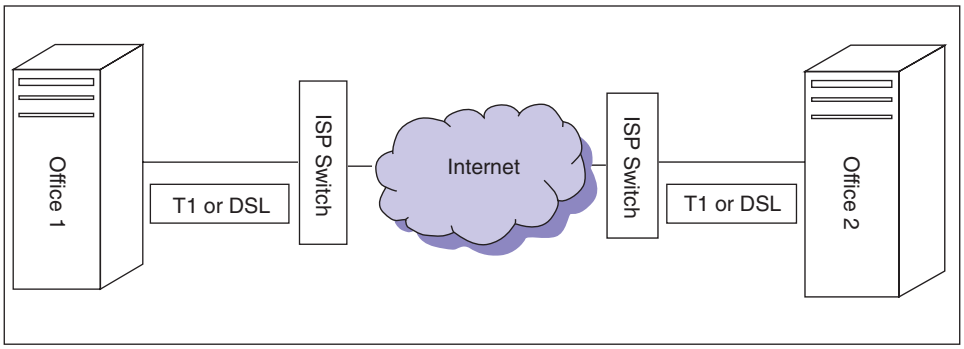


FIG. 28-23 Virtual private networks (VPNs) use wide bandwidth technology to connect two or more locations.

public Internet is encrypted to prevent others from intercepting and reading the traffic. VPN solutions are often packages with Internet routers or firewalls.

Citrix and Windows Terminal Server

Just a few years ago, all networking was wide area, combining mainframe-like servers at one location with remote dumb terminals at other locations using the UNIX platform. The difference between these systems and the current technology is that the old networks are just receiving and transmitting stations and are capable only of displaying text. The advantage of these systems is that they are highly efficient. Because only one central computer is involved, no data moves through the network; only screens and key clicks move between server and terminal. These systems require only low-speed, inexpensive data lines. In addition, UNIX is and always was designed to run high-performance WANs. Even today, almost all Internet servers run on a UNIX platform.

By comparison, Windows-based networks are highly inefficient. Because everything in Windows is an image, all workstations are computers; real data move across the network, and higher-performance communications lines are required to carry the signals. Citrix (Citrix Systems, Ft. Lauderdale, FL), has provided the closest approximation (in the Windows world) of the mainframe model of computing. Citrix software (e.g., Xen Server, Xen App, Xen Desktop) offers leading solutions to maintain all data and programs on a single server. In other words, the client workstations (PCs in most cases) are not running any software; they are simply sending and receiving Windows screens and transmitting keyboard and mouse activity. Because a much smaller amount of data is moving around the network, Citrix systems can perform well with much lower-speed communication lines. Microsoft's Windows server platforms (e.g., Windows 2003 Server, Windows 2008 Server) also offer remote "terminal servers" capability.

No matter which WAN model the clinician chooses, the client/server system is recommended because only a very small amount of real data travels on the network. This is vastly different from other dental software systems, which transmit data to the workstation for change and then transmit the data back for storage. In these non–client/server systems, the server is simply a passive storage system. Client/server systems feature a dynamic, active server that controls access. This can be likened to a busy intersection. A client/server system has "traffic lights" and "police officers" (i.e., the SQL database engine) which control access. No such controls exist in a non–client/server system. Each car (i.e., workstation) entering the intersection makes its own decisions as to when to stop and when to go. As a result, "collisions" and "accidents" (i.e., corrupted data) are more common.

Fat- and Thin-Client Computing and the Internet

Software written for PCs and Windows often is referred to as a *fat-client model*. This unattractive label signifies that software written for the PC must be installed and run separately from each client computer; only the database is centralized. Each fat-client must be a powerful PC with the software individually installed. Because data must travel to and from the client, the bandwidth of the communications channel must be significant. *Bandwidth* is a term used to specify the speed of a communications channel. For example, a high-speed cable connection offers wide bandwidth because it can transmit 5 megabits of data per second; a T1 connection transmits at 1.544 megabits per second. A 56k modem connection is narrow bandwidth because it can only transmit at speeds up to 56 kilobits per second. A cellular broadband connection may offer 768 kilobits per second. Specialists in communications often refer to bandwidth as "the size of the pipe," with fat pipes being higher speed than small pipes.

Another disadvantage of the fat-client model is the cost of maintaining it. Imagine for a moment that you worked in a large law office with 200 desktop computers, all running the same software. If the company making the law office's software released an update, the practice would have to install the update separately on each workstation. In addition, each of those 200 workstations would have to be powerful PCs, which produces its own maintenance headaches.

The thin-client model[85] is much simpler. This is not a new model; UNIX networks have always been thin, and most mainframe networks are thin. In fact, the fat-client model is a product of the PC age. The Internet, the ultimate thin-client network, has become so pervasive that thin-client computing has again become the preferred model. Within the next few years, developers of software for endodontists probably will reengineer their software and hardware to run on the Internet as a thin-client solution, with imaging, charting, and patient demographic data stored and archived remotely, and running on a simple Internet browser (e.g., Internet Explorer or Firefox).

The Internet represents a great deal more than just a different place to run office software. Remarkable innovations available now to clinicians include (to name just a few):

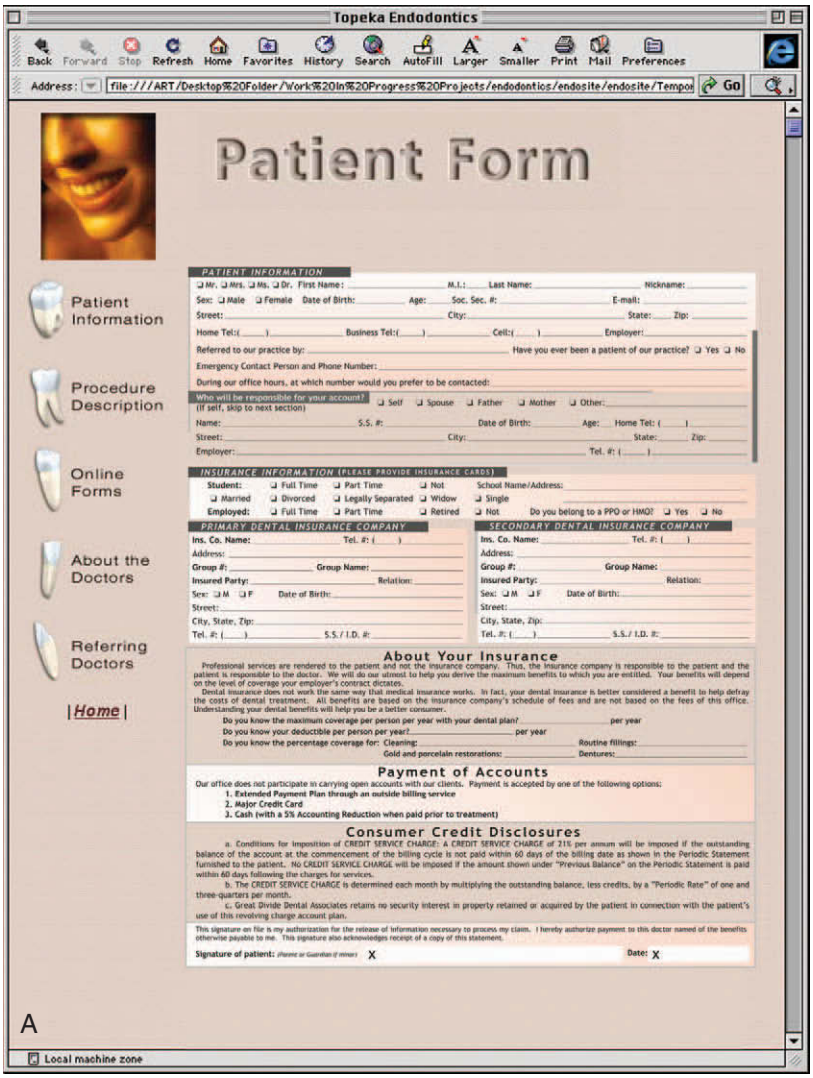


FIG. 28-24 A, Patients can receive pretreatment instructions or a "Welcome Letter" and complete their preregistration forms on the office website in this fictitious example.

- *Automated preregistration over the Internet.* Several vendors provide online patient registration forms (Fig. 28-24, *A*), which can securely transfer information over the Internet directly into an endodontic software database (see Fig. 28-24, *B*).
- *Multiprovider networks.* Multiprovider networks allow clinicians to send patient records and images to colleagues in a secure area on the Internet.
- *Online scheduling.* Patients can have limited use of the schedule online, 24 hours a day, 7 days a week. For example, patients can enter their name and appointment date, and the website will allow cancellation of the appointment and selection of preferences for a new appointment (i.e., specific days of the week and time of day).
- *Online payments.* Patients can pay their bills online.
- *Data backup.* With this feature, which is offered by numerous vendors, data can be automatically transferred at night to geo-separate servers at different locations.
- *Online ordering.* Clinicians can order most supplies over the Internet.
- *Data analysis.* In the future, clinicians may provide anonymous data to data banks that will analyze the information and report statistics regarding their practices.

Early PCs and mainframe computers were isolated and therefore limited islands of information storage. Later, networked systems with some sharing of data allowed exchanges within each business entity only. Today, using software protocols such as HTTP and HTML, the Internet has revolutionized interconnectivity between millions of computer networks, allowing cross-platform connectivity. Software is now the chief driver of distributed computing, characterized by technologic advances such as extensible markup language (XML), a system of small applications that can be connected for specific functions to enhance Web services. With XML, information now can travel to and from different systems regardless of hardware and location. The integration across many platforms provides numerous benefits, including better connections among offices, patients, referrers, and vendors. Finding ways to allow patients, referrers, and vendors to conduct exchanges that remain private and secure, however, has been a continuing challenge. Microsoft.NET is the software tech-

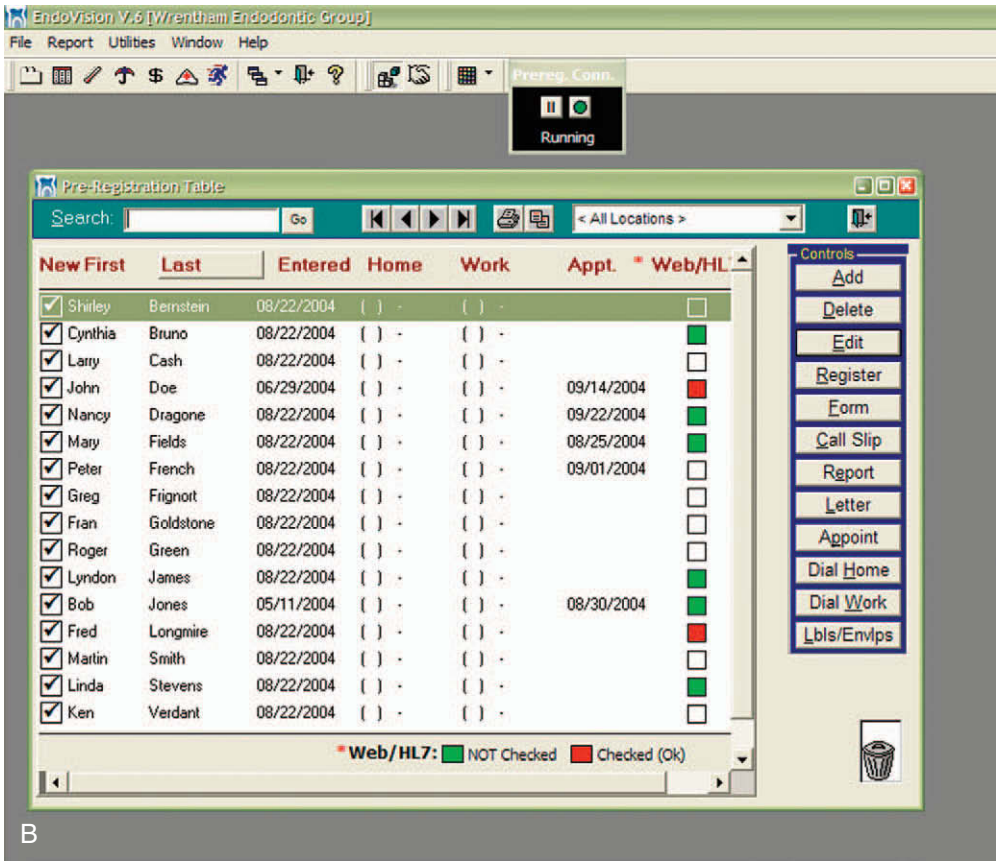


FIG. 28-24, cont'd B, When submitted by the patient, the forms are transmitted to the office computer via a web server and are automatically entered into the practice management software (green square under Web/HL7). (***A** courtesy PBHS, Santa Rosa, CA. **B** courtesy Henry Schein, Inc., Melville, NY.)*

nology for connecting disparate systems and the watchword for the next generation of software platforms. That said, the IT plan for endodontic offices should include provision for each computer workstation to be connected to the Internet to improve functionality.

Power, Cabling, and Wireless

Power

All computers, especially the server, should be protected from voltage sags, interruptions, and surges in power and should allow for an orderly shut-down and saving of all open files if a power aberration occurs. Although many uninterruptible power supply (UPS) topologies are available for small business applications, the *line interactive UPS* is especially well suited for servers. In this scheme, the battery to AC power inverter is always on, and the battery is charged when input AC power is normal, and the inverter is operated in reverse. This design provides power filtering and reduced power transients compared with the *standby UPS* topology recommended for workstations[8] (Table 28-3). Because the primary cause of hard drive failure is power spikes and sags, dedicated and properly specified UPS equipment is highly recommended.

The computer and monitor must be plugged into only the surge/battery backup–protected sockets. Laser printers should not be plugged into the battery positions, just the surge suppression sockets, because they draw too much current. Any system that receives telephone service also should be protected from "back door" lightning strikes that can destroy the office's system and data.

Cabling and Wireless

Wall jacks for power and Ethernet,[71] a LAN technology that transmits information between computers at speeds of 1000 Mbps (million bits per second), should be placed near the computer workstations. A double duplex power outlet should be installed at each location, along with a combination wall plate containing an Ethernet and a telephone jack. Internal wiring for the Ethernet should be run with copper unshielded twisted-pair (UTP) Cat 6 wire terminated in RJ 45 blocks. Cabling used inside walls is different from that used in patch cords. Wall cabling is much more susceptible to breakage and therefore should be terminated in a fixed block mounted on the wall (i.e., a wall jack) and not run out of the wall directly to the CPU. The Cat 6 can operate at a bandwidth of 1000 MHz and carry voice and data at rates of up to 1 Gbps.

Wireless networks are becoming more common, but they can be somewhat problematic because of sometimes inconsistent reception. Some wireless capability is necessary if tablet or slate PCs are part of the practice's computer hardware. Wireless networks must be carefully configured so that security features effectively shield against hacking. Before installation, the clinician should fully understand the advantages and disadvantages of a wireless LAN.

TABLE 28-3

Examples of Uninterruptible Power Supply (UPS) Systems Commonly Used by Small Businesses

UPS Type	APC Product Name	Benefits	Recommendations
Standby	Back-UPS	Low cost, high efficiency, compact	Best value for workstations
Line interactive	Smart-UPS, Back-UPS Pro, and Matrix	High reliability, high efficiency, good voltage conditioning	Most popular type of UPS because of its high reliability; ideal for servers and harsh power environments

Modified from APC Corp: *White Paper #1, The Different Types of UPS Systems*, www.apcmedia.com/salestools/SADE-5TNM3Y_R5_EN.pdf. Accessed Nov 21, 2009.

Central Processing Unit

Two kinds of CPUs are commonly used in dental offices: the standard desktop, or minitower, and the laptop. CPUs designed for businesses, such as Dell's OptiPlex line (Dell, Austin, TX), are network-optimized for improved manageability and reliability. These units also support multiple monitor configurations, which increases available "desktop" space for displaying multiple application windows to improve productivity.

Desktop CPUs can be used in almost any area or for almost any task, and the clinician should choose one of the fastest processors available. It should have enough random access memory (RAM) to allow several programs to be run at once and should include at least two internal expansion slots and one drive bay for additional peripherals. As a general rule, a faster processor adds to a machine's useful life, and upgrading the system's RAM to at least one level more than required to run the computer's operating system (OS) is advisable. Increasing RAM to at least 4 GB (gigabytes) improves speed for today's graphic-intensive software. Savings are often available on RAM, and it is well worth the additional investment.

Software applications, the OS, and the data created by the office are stored on the hard drive. As hard drive capacity increases and the price per megabyte declines, it becomes easier to choose a large hard drive that is adequate for most purposes. Because data are stored on the server in the CIS, the office generally does not need to buy individual workstation CPUs with ultralarge-capacity hard drives.

Monitors

The monitor's size and image quality are critical to image resolution. Clinicians should invest in the highest quality unit they can afford. In all likelihood, the monitor will last well past any single CPU. LCDs are free from geometric image distortions, flicker, and screen brightness variations, which may reduce the incidence of Computer Vision syndrome (eyestrain, headaches, etc.).

LCD screens require little in the way of maintenance. The clinician should make sure that all equipment in clinical areas is protected from cross-contamination with disposable barrier techniques, if applicable, and that manufacturer's recommendations are followed regarding ventilation, electrical and shock hazards (all clinical equipment should be powered through hospital-grade electrical connections), and cleaning. Many vendors make cleaning solutions, wipes, and cloths specifically for LCD computer screens.

LCD Screens

The gold standard for administrative, front desk, and imaging applications in the operatory are LCD[20] screens with high-resolution characteristics. All flat screens are not created equal. The following tips can aid the clinician in selecting a screen:

1. The screen should have the Video Electronics Standards Association's (VESA) mounting hole pattern on the back so that it can be attached to an industry-standard mounting arm. This mounting hole pattern is square and measures 3 × 3 inches (75 × 75 mm) or 4 × 4 inches (100 × 100 mm).
2. LCD monitors are measured by the size of the viewable area. A flat panel screen of 20 to 24 inches is recommended. Most screens list horizontal and vertical viewing angles of at least 140 degrees. The ideal horizontal and vertical viewing angle is 160 degrees or more.
3. LCD is a transmissive technology. A fixed-intensity fluorescent or light-emitting diode (LED) backlight is filtered by the liquid crystals arranged between two finely grooved surfaces. These new LEDs produce greater dynamic contrast, are thinner, consume 40% less power, have a wider color gamut, but cost more than fluorescents at this time. Organic light-emitting diodes (OLED), another new monitor technology, are composed of a film of organic compounds, are the thinnest technology, and do not require a backlight to function, providing deep black levels. The screen purchased should have a 3-year warranty on the backlight. Most LCD fluorescent screens last for about 60,000 hours (6.8 years) of continuous use.
4. An LCD panel can display only one resolution at full screen size, because each pixel is hard wired (e.g., a 1280 × 1024 panel has 1,310,720 hard-wired pixels in its native mode). A minimum dot pitch of 0.26 mm is recommended. If an LCD panel has a native resolution of 1280 × 1024 pixels, it can display at a resolution of 640 × 480 by using less of the screen area. Most LCD screens can rescale a lower-resolution image using rathiomatic expansion, which works adequately for photographs but not very well for text or images with fine detail. Objects become jagged, and artifacts fill the extra pixels. When resampling is done for scaling the image up, antialiasing fills the extra pixels, resulting in better results. For the clinician, the bottom line is to choose a screen with a clear understanding of the native resolution and plan to use it as specified.

5. Only LCD monitors that support digital (binary pulse) signal inputs (i.e., DVI) should be purchased for general office use. Because LCD screens process information digitally, fluttering and ghosting can result if digital information from the computer must be sent to the video card, converted to analog, sent on to the LCD screen, and then converted back into a digital signal. Currently, LCD units that include both analog video and digital inputs such as high definition multimedia interface (HDMI), digital visual interface–digital (DVI-D) with high definition content protection (HDCP), DisplayPort, video graphics array (VGA), component video, S-video, and composite video are recommended for certain applications.
6. The refresh rate (i.e., how quickly the screen paints the image) is an important indicator of how well the screen will depict moving images. A refresh rate of 6 msec is a minimum standard.
7. Special attention should be paid to contrast ratios, to ensure that the screen depicts a large range of light and dark tones. A contrast ratio of at least 80,000:1 should help paint an image with excellent appearance quality.
8. Integral stereo speakers are a nice feature on any monitor. Although most speakers do not take up much room, built-in speakers reduce clutter and make the computer system easier to move.

Printers

Dental offices strive to run compliant, cost-effective operations that minimize cost and maximize productivity. Printers are available in a dizzying array of formats and output types; therefore careful selection for each use is recommended. Although increasingly popular multifunction products (MFP) promise "significant advances in the areas of document distribution and management, security, wired and wireless connectivity, and color,"[45] most dental offices use single-function machines. Because most MFP devices use proprietary software drivers, the prospective buyer must make sure that the machines are compatible with all the software used in the office. Selection of the right printers is crucial to the usefulness of the entire computer system.

Monochrome and Color Laser Printers

The front desk, administrative, and clinician's office computers should be linked to a monochrome or color laser printer for printing of patient instructions, insurance materials, receipts, and general word processing tasks. Clinicians who follow the frontdesklessness concept should consider a multidrawer printer model so that stationery, plain paper, and envelopes are all preloaded. For example, EndoVision software (Henry Schein, Inc., Melville, NY) allows the clinician to use a "rapid post" module to select tasks that can be directed to multiple individual and networked printers and specific drawers.

Label Printers

Sterilization area and front desk computers benefit from dedicated label printers for printing chart labels and mailing labels for general mailing duties. Although the use of industry-standard window envelopes can greatly reduce the need for envelope printing, label printers are often a useful adjunct, especially for larger items.

Inkjet Printers

Production of patient treatment reports and radiographs are the primary functions of the inkjet printer. A high-quality printer with individual archival inks that use a variety of paper formats can produce stunning reports that can improve communication with co-therapists. Epson's (Epson America Inc, Long Beach, CA) new pigment-based inks with resin-coated 0.1-µm particle size and long-lasting permanence ratings are the gold standard for professional image printing.

Document Scanners

Scanning of documents improves dental processes by increasing security and efficiency and reducing storage space. Scanners can transmit documents in a variety of formats, so careful attention should be given to compatibility with dental management software. Documents should be scanned into a patient's individual file so that all the documents for that particular patient are easily accessible. Accounting and other miscellaneous office documents can be scanned into a file on the server or client workstation for easy retrieval. Recommended scanners include native support for Adobe portable document format (PDF). The PDF file format has become the document exchange standard because of its wide industry acceptance. PDF files can be read independently from other application software and can be viewed as thumbnails in Microsoft Office application software; each document can be password protected.

Portable Document Format

Unlike TIFF and JPEG image formats, PDF encapsulates other compression formats so that documents become application independent. These files have embedded metadata, or data within the image, that can authenticate the document. As an open specification, PDF also maintains documents in a consistent format, which makes it the best choice for long-term archiving and for preserving the original appearance.

Security

PDF files improve security through digital signatures and document encryption features that improve HIPAA compliance and address general business security demands. All PDF files can be password protected to limit access to only those authorized to view the contents.

Versatility

PDF files can be viewed on every major computing platform, including Microsoft Windows, Mac OS, and UNIX.

Searchable Images

PDF creates image files that can be searched and indexed by optical character recognition (OCR). Processing a document with OCR produces a searchable bitmap that is hidden behind the image while preserving the look of the original image.

Input Devices

With pointing devices and keyboards, location and ease of use should be considered along with infection control. In the operatory, clinicians should choose wireless keyboards and optical mice that can be protected by barriers to eliminate cross-contamination. Logitech manufactures a wide range of wireless

input devices, including Bluetooth models with wireless hub/battery charger, keyboard, and rechargeable mouse that allows a range of up to 30 feet (9 m) using the 2.4 GHz ISM band. Care must be taken with the addition of any radio frequency (RF) wireless devices to guard against interference with other wireless equipment, such as the wireless radiographic sensors, tablet and slate computers, and wireless telephones.

Graphic tablets are also useful adjuncts that allow annotation of drawings in the patient's chart. Graphic tablets can be purchased in several sizes and usually require a USB interface. The pen, or stylus, can be wired or wireless, although batteries add to the weight of the pen. Another solution to graphic input, albeit a more expensive one, is a wireless tablet PC, which has all of the functionality of a PC with wireless RF capability and true tablet functionality. These devices can operate as a laptop and, with "digital ink" technology, can input hand-written notes and graphics into any enabled application.

Consulting and Support

IT systems require thorough planning and careful implementation to ensure trouble-free installation. Clinicians should begin planning with local vendors in close cooperation with the practice management vendor. Because technology changes constantly, clinicians need someone who can update the system's hardware and software, upload "patches" and "firmware updates," and generally be available for help with equipment purchase decisions. These vendors usually work on an hourly basis and may offer blocks of time per month or per quarter at discounted rates and guaranteed response times.

Pharmacology Reference Software

Many programs are available that can serve as clinical pharmacology references. As mentioned previously, Lexi-Comp Online is a Web-enabled or installable product that features extensive indexing, cross-referencing, and linking of drugs for optimum quick search functions. For example, the clinician simply types in the name of the drug, and the program calls up every reference to it in the compendium. The search can be done by alphabetic listings, a main index of drug classifications, product identification (by description of the drug), adverse reactions, disease description, drug interactions, and other parameters. This product contains clinically oriented monographs on all drugs used in the United States and allows for quick, accurate identification of drug interactions and contraindications. The program also promotes a quick search of dental and dental vasoconstrictor implications for each drug listed.

Patient Education Software

Patient education is an important part of modern dental practice. One study[14] found a positive statistical correlation between patient education and their endodontic treatment experience. To this end, several vendors have created informational software programs for the Web and local computer use. The patient can access these programs on the Internet, at a computer kiosk in the reception area, or at the clinical chairside workstation.

Health Insurance Portability and Accountability Act

Confusion about transmission of protected health information by electronic means has created the myth that clinicians cannot communicate by e-mail or the Internet. The U.S. Department of Health and Human Services has stated[16]:

> *The Privacy Rule allows covered health care providers to share protected health information for treatment purposes without patient authorization, as long as they use reasonable safeguards when doing so. These treatment communications may occur orally or in writing, by phone, fax, e-mail, or otherwise.*

SUMMARY

Creation of the EOHR is the next logical step in the journey to the paperless office. Integration of this information with an EHR to create a unified patient database that documents the current health status and lifetime medical history for each patient is the pot of gold at the end of the rainbow. As the functionality of digital systems improves, every dental care delivery organization should consider incorporating at least some of these advances. However, technology is not a substitute for treatment excellence, and clinicians must continue to strive to provide quality treatment for patients regardless of the equipment they use.

ACKNOWLEDGMENTS

Mr. Brendan Sheehan, Mr. Guy Wassertzug, Mr. Joshua R. Walty, Dr. Robert Lapp, Dr. Allan Farman, Dr. Bruce Lieberthal, Mr. Jay Levine, Dr. David Gain, Mr. David Taylor, Dr. John Goyette, Dr. Mark Diehl, and Mr. Paul Bralower for their invaluable help with IT systems, digital imaging, and dental informatics. Also, Mr. Bruce Dale and Mrs. Katherine Levin Sheehan for their exceptional photographic skills and Mrs. Deborah Lebbin and Mrs. Susan Levin for their editorial help.

DISCLAIMER

Dr. Levin receives honoraria or equipment or has a financial relationship with the following vendors mentioned in the text: Atlantic Design, LLC; PracticeWorks, LLC; and Henry Schein, Inc.

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CHAPTER 29

Cone Beam Imaging for Endodontic Procedures

DALE A. MILES and THOMAS V. McCLAMMY

CHAPTER OUTLINE

PRINCIPLES OF CONE BEAM VOLUMETRIC TOMOGRAPHY
- Voxels and Voxel Sizes
- Field of View

IMAGING TASKS IMPROVED OR SIMPLIFIED BY CONE BEAM VOLUMETRIC TOMOGRAPHY
- Apical Morphology and Suspected Lesions of Endodontic Origin
- Root Canal System Morphology
- Presurgical Visualization
- Suspected Root Fractures and Trauma
- Internal and External Root Resorption

Although at first glance, it may seem that cone beam volumetric tomography (CBVT), also known as *cone beam computed tomography* (CBCT), may be "overkill" for diagnostic evaluation of endodontically compromised teeth, the interest and adoption of this advanced imaging modality is now becoming very popular with clinicians performing endodontic procedures—and for good reason. Two-dimensional (2D) grayscale images, whether they are conventional film or digital images, cannot accurately depict the full three-dimensional (3D) representation of the teeth and supporting structures. In fact, traditional images are very 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. 29-1, *A*).

On the other hand, CBVT 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 pathology and root morphology previously impossible (see Fig. 29-1, *B*).

Several tools available in CBVT, such as changing the vertical or horizontal angulation of the image "on the fly" or in real time, as well as thin-slice, grayscale data of 0.1 mm thickness, will never be available for conventional or even digital radiographic assessment.[1] In addition, the ability to use CBVT data to view the region of interest in three anatomic planes of section at very low x-ray doses was never as easy or accessible as it is today. Most clinicians probably never even anticipated that such a precise imaging solution would be introduced to the dental profession during their careers.

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

PRINCIPLES OF CONE BEAM VOLUMETRIC TOMOGRAPHY

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

1. Voxel size
2. Field of view (FOV)
3. Slice thickness/measurement accuracy

Voxels and Voxel Sizes

Unlike medical computed tomography (CT) scanning, cone-beam scanning machines acquire their x-ray information using low kV and low mA exposure parameters in a single pass from 180 to 360 degrees of rotation. Medical scanners use kilovoltages of 120 kV or more and milliamperages of about 400 mA. The highest exposure factors for any dental cone-beam scanner is the Imaging Sciences i-CAT (Imaging Sciences International, Hatfield, PA) (Fig. 29-2), which uses 120 kV and 24 mA. In contrast, dental scanners like the Planmeca Promax® (Planmeca Oy, Helsinki, Finland) (Fig. 29-3, *A*) or Veraviewepocs 3D (J. Morita Corporation, Kyoto, Japan) (Fig. 29-3, *B*) use typical dental exposure factors of about 5 to 7 mA and 70 to 80 kV. The x-ray dose for all cone-beam machines is significantly lower than the dose received from a medical computed axial tomography (CAT) scan.

Besides lower exposure factors, the image data in cone-beam imaging is reconstructed from isometric voxels; that is, the images are constructed from pixels that are cubic and have



FIG. 29-1 A, A well-angulated periapical radiograph of tooth #3 taken during diagnostic appointment for endodontic evaluation in maxillary right quadrant. At first glance, there is little radiographic evidence of significant periapical and/or periradicular change. **B,** Cone beam volumetric tomography of same tooth at same appointment gives an entirely different perspective; periapical changes are visible on all three roots in all three anatomic planes of section. (***B,*** *Image taken with J. Morita Veraviewepocs 3D [J. Morita, Osaka, Japan].*)



FIG. 29-2 The Imaging Sciences i-CAT. (*Courtesy Imaging Sciences International, Hatfield, PA.*)

the same dimensions for length, width, and depth. These voxel sizes are as small as 0.1 to 0.6 mm.[1] By comparison, medical CAT scan slice data is 1 mm to 1 cm. Fig. 29-4 illustrates the difference between a pixel and a voxel, the difference between an ***anisometric pixel*** of medical CT and an ***isometric pixel (voxel)*** of CBVT, and how the pixel data is acquired from both modalities.

A patient in a medical CT scanner is moved in the scanner as little as 1 mm at a time as the image data is acquired. But for each slice acquired, the patient is exposed to ionizing radiation. If it takes 20 or 30 slices to acquire the data for the head region, the patient is exposed during the gathering of each slice. This results in significantly higher absorbed x-ray doses for the patient. A typical CBVT examination would expose the patient to only about 20 to 500 μSv in one rotation, whereas a typical medical examination of the head would approach 2100 μSv[2] because the image data is gathered one section or layer at a time. Consequently, CBVT data has much higher resolution than medical CT data and is acquired at a fraction of the radiation dose. Because of the smaller voxel size, there is also greater image resolution.

Field of View

The FOV ranges from as small as a portion of a dental arch to an area as large as the entire head. Currently, the smallest FOV available is 37 × 50 mm (Fig. 29-5), which may be appropriate for a single arch. The largest, 220 × 220 mm (Fig. 29-6), is probably far larger than an endodontist would require, but many machines with larger FOVs can be collimated down to as little as 40 × 40 mm, making them very suitable for endodontic procedures. The selection of the FOV depends on several factors. Among the most important are:

1. Diagnostic task
2. Type of patient
3. Spatial resolution requirements
4. Clinician's confidence interpreting the acquired data volume

Diagnostic Task

Since patients may have more than one tooth requiring an endodontic procedure, it may be desirable to have a machine capable of acquiring a CBVT volume greater than 40 × 40 mm.

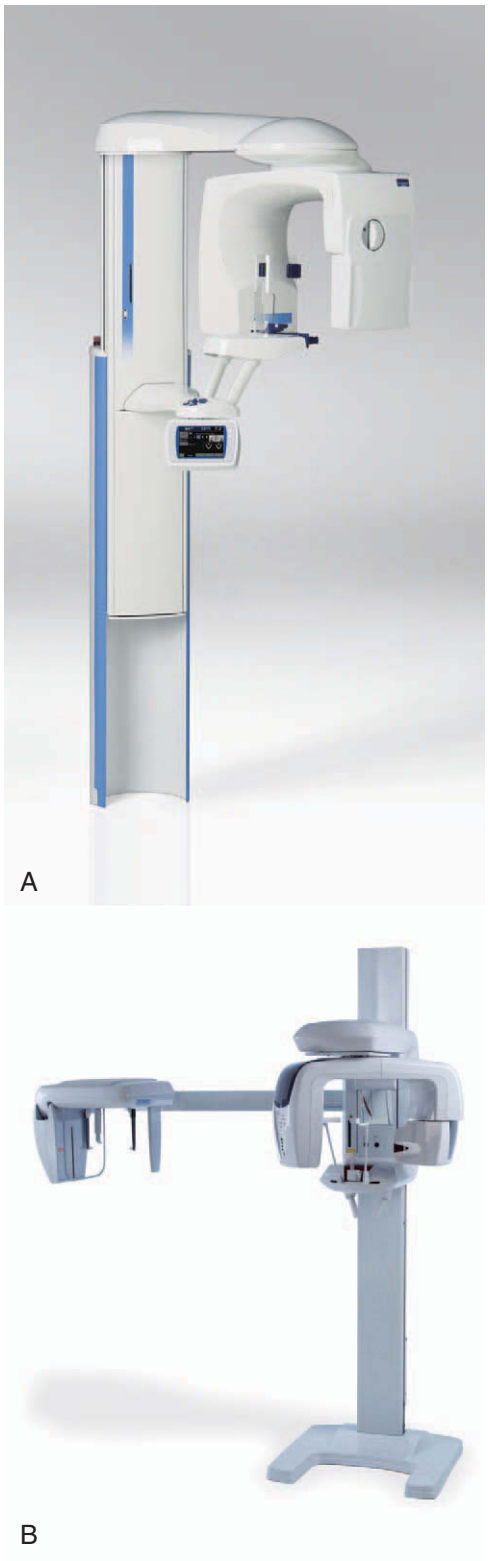


FIG. 29-3 A, The Planmeca ProMax 3D. **B,** The J. Morita Veraview-epocs 3D. (***A,*** *Courtesy Planmeca Oy, Helsinki, Finland.* ***B,*** *Courtesy J. Morita Corp, Osaka, Japan.)*

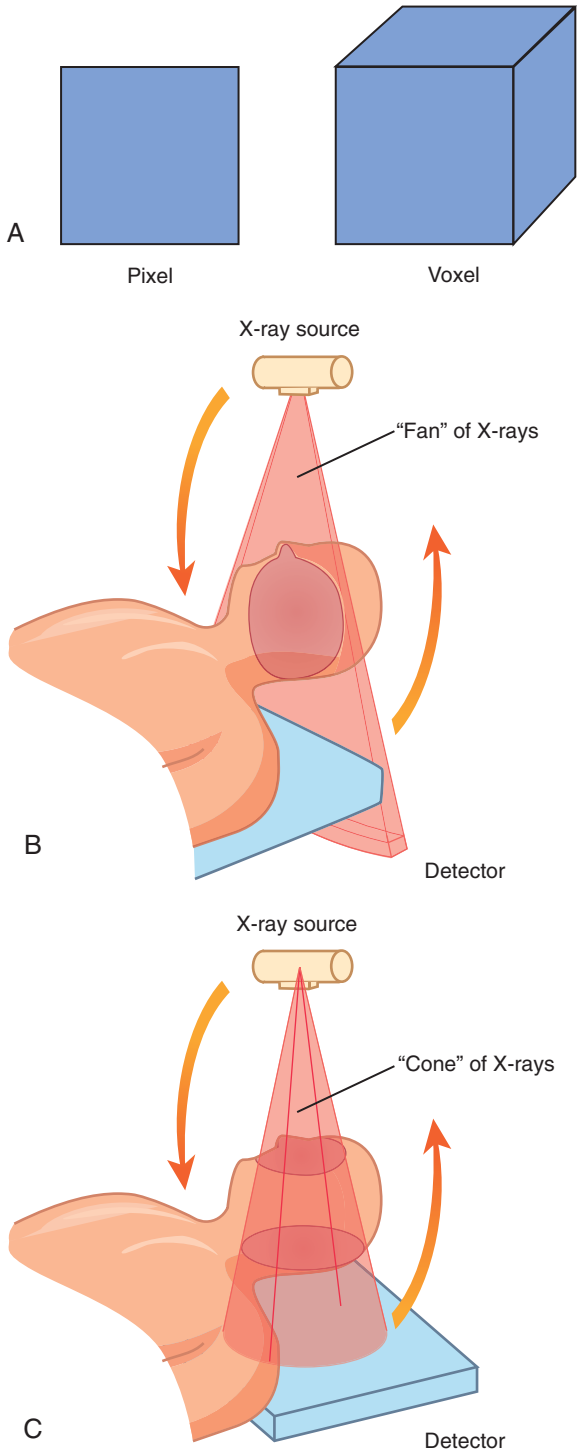


FIG. 29-4 A, Drawing on the left represents a *pixel* (picture element), the image capture and display element of any traditional digital image displayed on the computer. Shades of gray or color will be displayed in these pixels to represent a 2D image. Drawing on the right represents a *voxel* (volume element). Voxels are isometric pixels having the same dimension or length on all sides. They are very small (from 0.10 to 0.60 microns) and are the capture element for cone beam imaging devices. They too can have gray shades or colors in them (displayed on the computer in pixels, but with image-reconstruction software and postprocessing, can be made to look 3D in grayscale or color images. Principles of conventional fan beam **(B)** and cone beam **(C)** tomography. (***B-C*** *from Babbush et al:* Dental implants: the art and science, *ed 2, St Louis, Elsevier/Saunders, 2011.)*

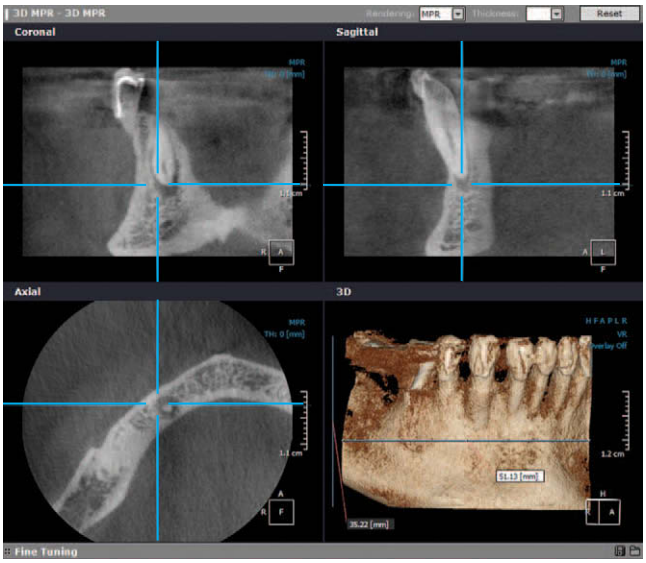


FIG. 29-5 Multiplanar and 3D color reconstructed views of right mandible taken on a cone beam volumetric tomography machine with a volume size of 37 × 50 mm. *(Image taken with Carestream Health Kodak 9000 3D cone-beam machine [Rochester, NY].)*

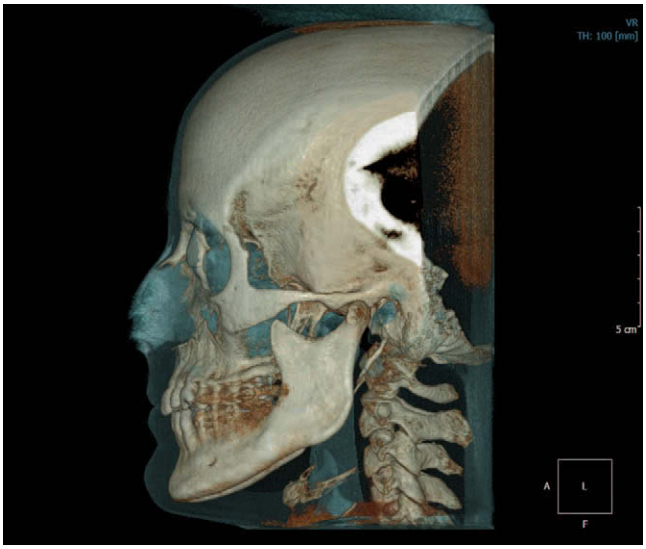


FIG. 29-6 Image of entire head (17 × 23 cm) from a large-volume machine. *(Image taken with Imaging Sciences i-CAT [Hatfield, NY]).*

TABLE 29-1

Details of Machines Currently on the Market (October 2009)

Machine	Type of Image			Smallest Cone Beam FOV (cm)	Largest Cone Beam FOV (cm)	Native Voxel Size (microns)
	"True" Panoramic	Bitewing	Periapical			
Planmeca: ProMax 3D	Yes	Yes	Yes*	4 × 4	13 × 17	0.16
Soredex: Scanora 3D	Yes	No	No	6 × 6	7.5 × 14.5	0.15
Carestream: Kodak 9000	Yes	No	No	3.7 × 5	3.7 × 5	0.076
J. Morita: Veraviewepocs 3D	Yes	No	No	4 × 4	8 × 8	0.12
PreXion: PreXion 3D	?	No	No	7.62 × 8.12800 (cm) (3 × 3.2 inches)	7.62 × 8.12800 (3 × 3.2 inches)	0.2
Gendex: GXCB-500	No; reconstructed	No	No	4 × 4	13 × 13	0.125

3D, Three-dimensional; *FOV,* field of view.

*Bitewing mode also depicts periapical regions of teeth in all quadrants from canine to third molar.

In addition, many clinicians may prefer more than one imaging modality to more fully evaluate their patients' needs. There are several multifunctional cone beam machines available that will allow the clinician to acquire several image types with only one machine. Table 29-1 details the machines currently available that allow several types of images to be reconstructed from the data set. Fig. 29-7 illustrates the advantages of using multiple image types for an endodontic case.

Type of Patient

Just because you own a cone beam machine does NOT mean that every patient will require a cone-beam scan.[2] More importantly, some patients requiring an endodontic procedure may be children or adolescents in whom the need to minimize the absorbed x-ray dose is more critical. 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)[3] exhibiting apical change are all important tasks requiring minute detail. If CBVT is employed, 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,

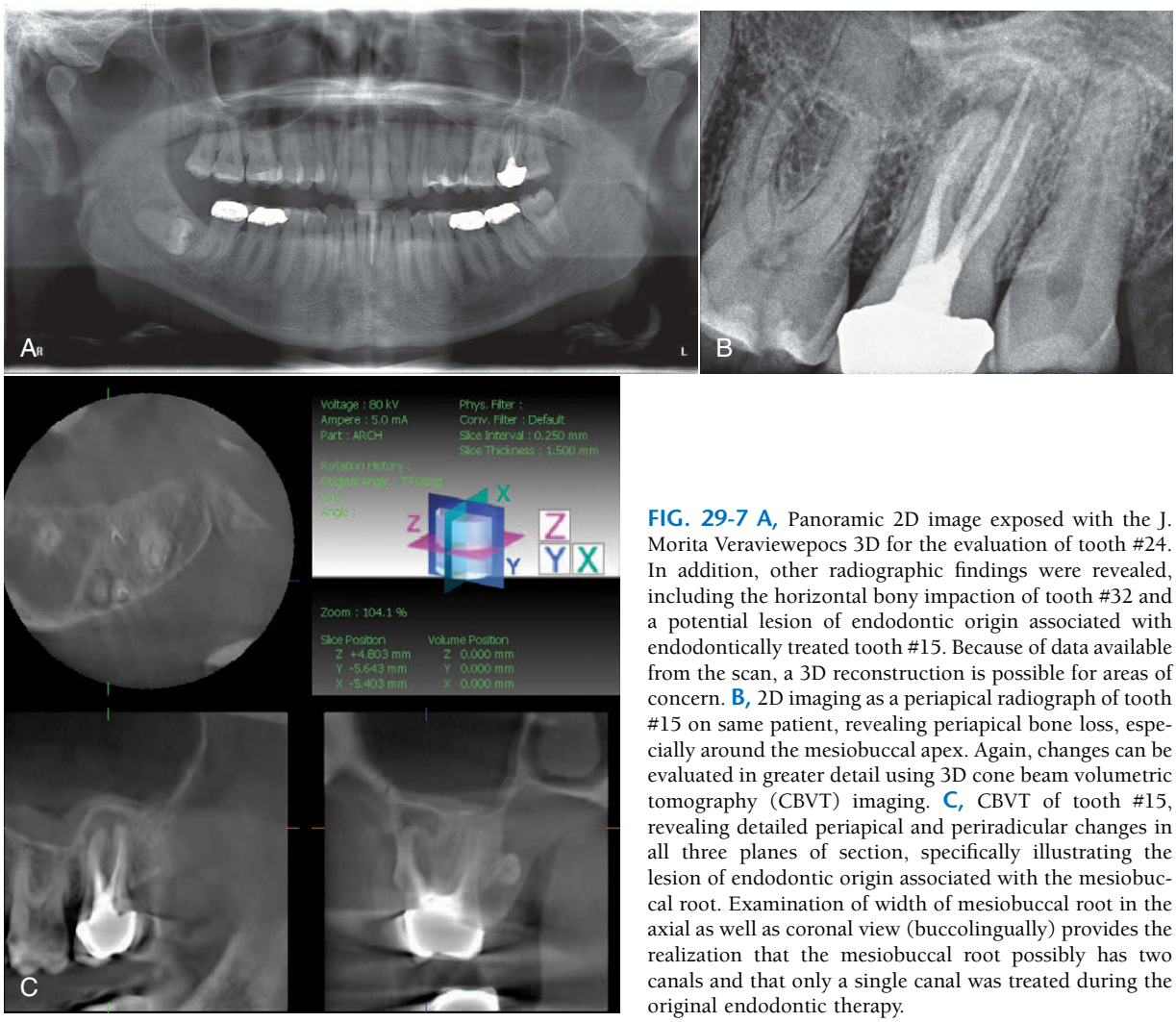


FIG. 29-7 A, Panoramic 2D image exposed with the J. Morita Veraviewepocs 3D for the evaluation of tooth #24. In addition, other radiographic findings were revealed, including the horizontal bony impaction of tooth #32 and a potential lesion of endodontic origin associated with endodontically treated tooth #15. Because of data available from the scan, a 3D reconstruction is possible for areas of concern. **B,** 2D imaging as a periapical radiograph of tooth #15 on same patient, revealing periapical bone loss, especially around the mesiobuccal apex. Again, changes can be evaluated in greater detail using 3D cone beam volumetric tomography (CBVT) imaging. **C,** CBVT of tooth #15, revealing detailed periapical and periradicular changes in all three planes of section, specifically illustrating the lesion of endodontic origin associated with the mesiobuccal root. Examination of width of mesiobuccal root in the axial as well as coronal view (buccolingually) provides the realization that the mesiobuccal root possibly has two canals and that only a single canal was treated during the original endodontic therapy.

such as the Imaging Sciences i-CAT, set their voxel size defaults to 0.4 mm. This voxel size is inadequate for high spatial detail. However, these same machines often have a voxel size selection program that allows smaller voxel size during the image acquisition. The absolute minimum voxel size for endodontic imaging should be 0.2 mm. Machines such as those cited in Table 29-1 typically use voxel sizes of 0.076 to 0.16 mm for their native image capture. *Fig. 29-8* shows two images taken at the axial slice level of the mental foramen. Fig. 29-8, *A* shows a slice using a voxel size during capture of 0.4 mm. Fig. 29-8, *B* shows an axial slice at the same level using a voxel size of 0.16 mm.

Clinician's Confidence Interpreting the Acquired Data Volume

As previously stated, endodontic imaging procedures can be appropriately performed with small volume data sets. Unless there are multiple teeth in different quadrants requiring endodontic diagnostics or procedures, a small FOV is probably sufficient. This also reduces the diagnostic responsibility for occult pathology outside the region of interest (ROI). Large-FOV machines will capture structures like the paranasal sinuses, airway, and vertebral column, so the clinician has an ethical and legal responsibility to examine these structures for potential pathoses.[4,5] To ignore these non-odontogenic regions would be considered negligence. Clinicians have the option to perform the radiographic evaluation themselves or refer the data volume to a qualified specialist such as an oral and maxillofacial radiologist or medical radiologist.[5,6]

For endodontists, and indeed most general clinicians, a CBVT scanner with a smaller FOV may be more appropriate.

IMAGING TASKS IMPROVED OR SIMPLIFIED BY CONE BEAM VOLUMETRIC TOMOGRAPHY

For endodontic treatment and assessments, there are at least five primary imaging tasks whereby CBVT scans have a distinct advantage over traditional 2D radiographs. These include evaluation of the following:

1. Apical morphology and suspected lesions of endodontic origin



FIG. 29-8 A, Axial slice from data gathered with a 0.4-mm voxel size. Compare the trabecular pattern and outline of the mental foramina to the same location using 0.16-mm voxel size in Part B. **B,** Axial slice from data gathered with a 0.16-mm voxel size.

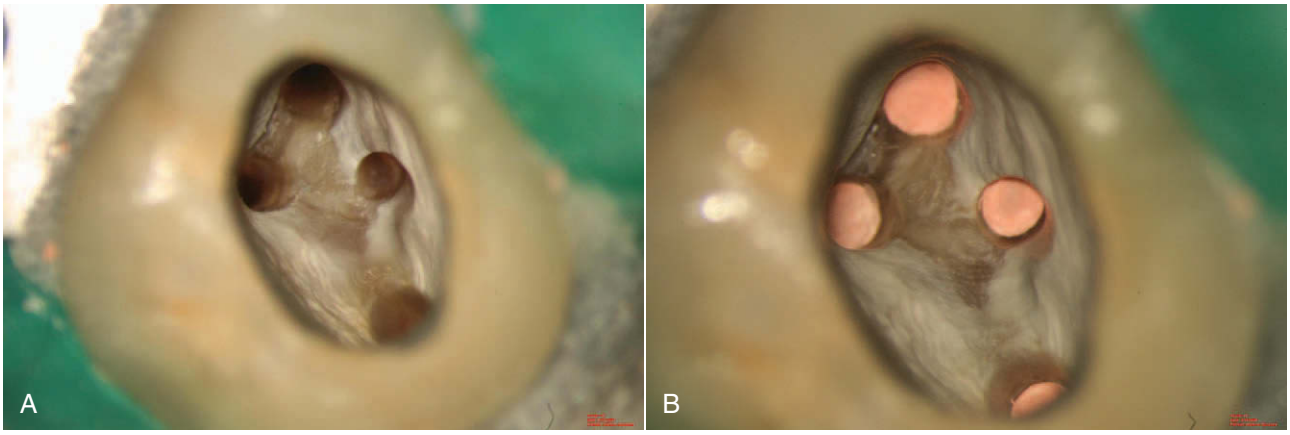


FIG. 29-9 Tooth #15 was endodontically retreated nonsurgically. As suspected, the original treatment did not access and treat the entire root canal system. Seen here are images taken of the access cavity just prior to **(A)** and after **(B)** final obturation of the entire system, including the missed mesial buccal canal.

2. Root canal system morphology
3. Presurgical visualization
4. Suspected root fractures and trauma
5. Internal and external root resorption

Apical Morphology and Suspected Lesions of Endodontic Origin

The precise location and visualization of root morphology is vastly improved with cone-beam data. Root curvature, root path, 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 0.1 mm (Fig. 29-9). Visual obstruction from anatomic features such as buccal bone and the malar process over the apices of maxillary roots simply "disappear" when you can scroll through the slices of the bone from facial to palatal in 0.1-mm sections while also changing axial orientations.

Additional benefits of some CBVT imaging software include allowing the clinician to create a pseudopanoramic image—that is, a thin-slice grayscale image of the mandible, maxilla, or both. Conventional panoramic machines, though not commonly employed by endodontists, use the mandible to select the focal trough, or zone of sharpness. This means that the palatal roots of the maxillary posterior teeth may not be centered in the focal trough. With CBVT imaging software, a central plane through the maxilla or mandible can be drawn so that the structures of interest can always be centered. This facilitates apical lesion assessment, even with a panoramic image (Fig. 29-10).

Radiographically, it is fundamental to understand that lesions of endodontic origin arise secondary to pulpal breakdown products and form adjacent to canal portals of exit.[7,8] These lesions can and do form three-dimensionally anywhere along root surface anatomy.[9] Prior to CBVT, clinicians were unable to visualize the specific location and extensiveness of periapical bone loss.

Fig. 29-11, *A* is a 2D periapical radiograph of the previous endodontic treatment of tooth #30, revealing no objective radiographic pathosis. The patient reported that tooth # 30 had

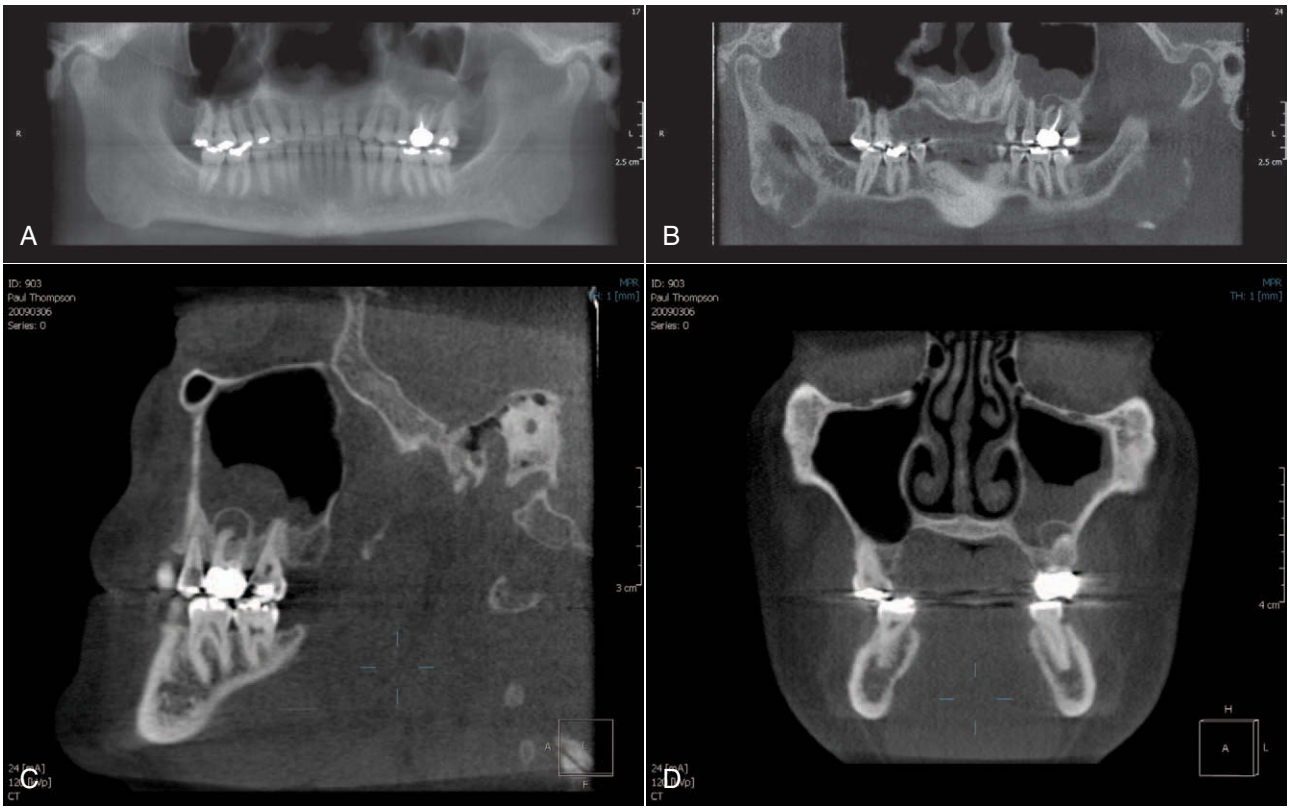


FIG. 29-10 A, Even though this is a reconstructed panoramic from the CBVT data, it approximates the view one would see on a conventional panoramic radiograph. It is somewhat difficult to see the lesion on tooth #14. **B,** This image is a thin slice pseudopanoramic image which shows the lesion more precisely because it is a very thin slice (0.10 mm). **C-D,** These slices of tooth #14 show the lesion in sagittal **(C)** and coronal **(D)** views, confirming the pseudopanoramic view.

been treated by an endodontist 5 years ago. The patient had returned to the treating clinician numerous times over the last 5 years complaining of tooth pain in this area. The patient was last advised that the area "had not enough time to heal." However, an additional radiograph exposed with a different angulation revealed that only one of the two mesial canals were cleaned and obturated (Fig. 29-11, *B*). Using available CBVT technology, additional radiographs would not be necessary to elucidate this information (Fig. 29-11, *C-D*).

Fig. 29-11, *E* shows multiplanar views of tooth #30 taken with a contemporary CBVT machine. This image set reveals:

1. A very high probability of a missed ML canal
2. A lesion of endodontic origin developing on the mid-furcation side of the mesial root (large white arrows)
3. Early periapical and periradicular changes of tooth # 31 (small white arrow)

Tooth #30 was sensitive to percussion and buccal alveolar bone palpation. While it can be concluded from the information available for the CBVT image that tooth #30 is the source of the patients' pain, the clinician can also inform the patient that tooth #31 may also need endodontic therapy and that a further evaluation of this tooth is necessary.

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 when evaluating root canal system morphology. With ever-present unusual and atypical root shapes and numbers, there is a need to look further than what a clinician can see or imagine with 2D radiography (Fig. 29-12).

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 periapical radiographs have been the only way to assess the apical region. Unfortunately, the information available from these images may not adequately prepare the clinician to surgically resolve the pathosis. For example, the clinician may be unable to observe whether the lesion has perforated the buccal and/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 to visualize the entire defect before the incision is created. This is an immense improvement over conventional imaging (Fig. 29-13).

Suspected Root Fractures and Trauma

Fig. 29-14 shows 2D and 3D images of the endodontic and restorative treatment of tooth #30. The 2D periapical

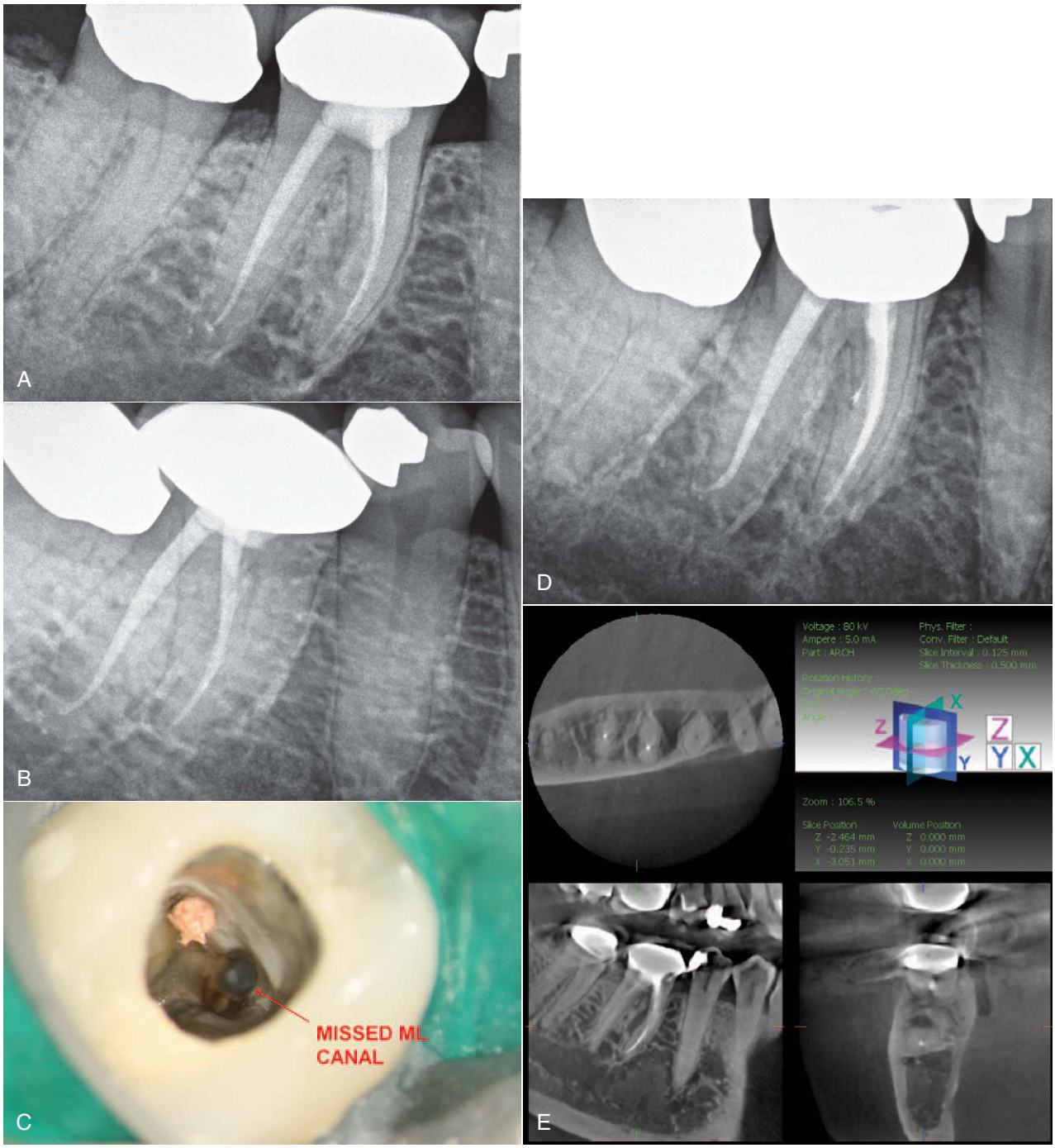


FIG. 29-11 A, Tooth #30. **B,** Exposure of tooth #30 with a different angulation, confirming that original root canal therapy for tooth #30 only addressed a single mesial canal. **C-D,** Initial access for nonsurgical retreatment provided visual confirmation that original endodontic therapy did not address the mesial lingual canal **(C).** Part **D** illustrates a posttreatment radiograph that addresses cleaning and obturation of entire root canal system. **E,** Multiplanar views of tooth #30.

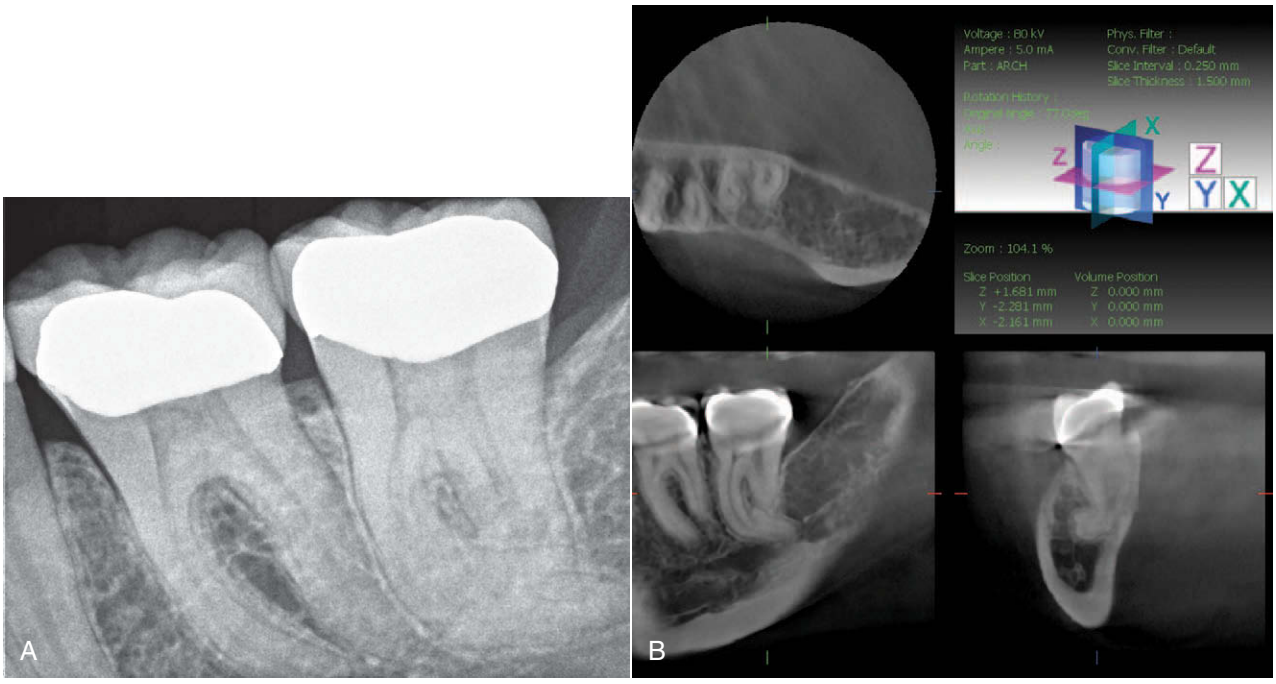


FIG. 29-12 A, Tooth #18, referred for endodontic evaluation and potential treatment. This 2D radiograph reveals significant pulp stones and canal calcification developing not only in coronal aspect of root canal system but extending down the visible distal canal. Apical third of the canal system appears unusual and dilacerated. Cone beam volumetric tomography (CBVT) imaging would be beneficial in visualizing root canal anatomy prior to creating ideal endodontic access. **B,** Single slice of the CBVT for tooth #18. Information regarding direction of root canal anatomy is provided in all three planes of section, axial, coronal, and sagittal. It is interesting to note that axial slice shows that the mesial lingual root actually traverses buccally as it approaches its terminal extent. This is valuable information for the clinician, available prior to cleaning and shaping of entire root canal system, permitting a higher degree of treatment predictability.



FIG. 29-13 Axial slice **(A)** and sagittal slice **(B)** reveal that there may not be any cortical bone perforation. This is because these slices are very thin (0.10 mm), and it depends which level or section of the slice is being viewed; lesion is observed to have spread palatally.

Continued

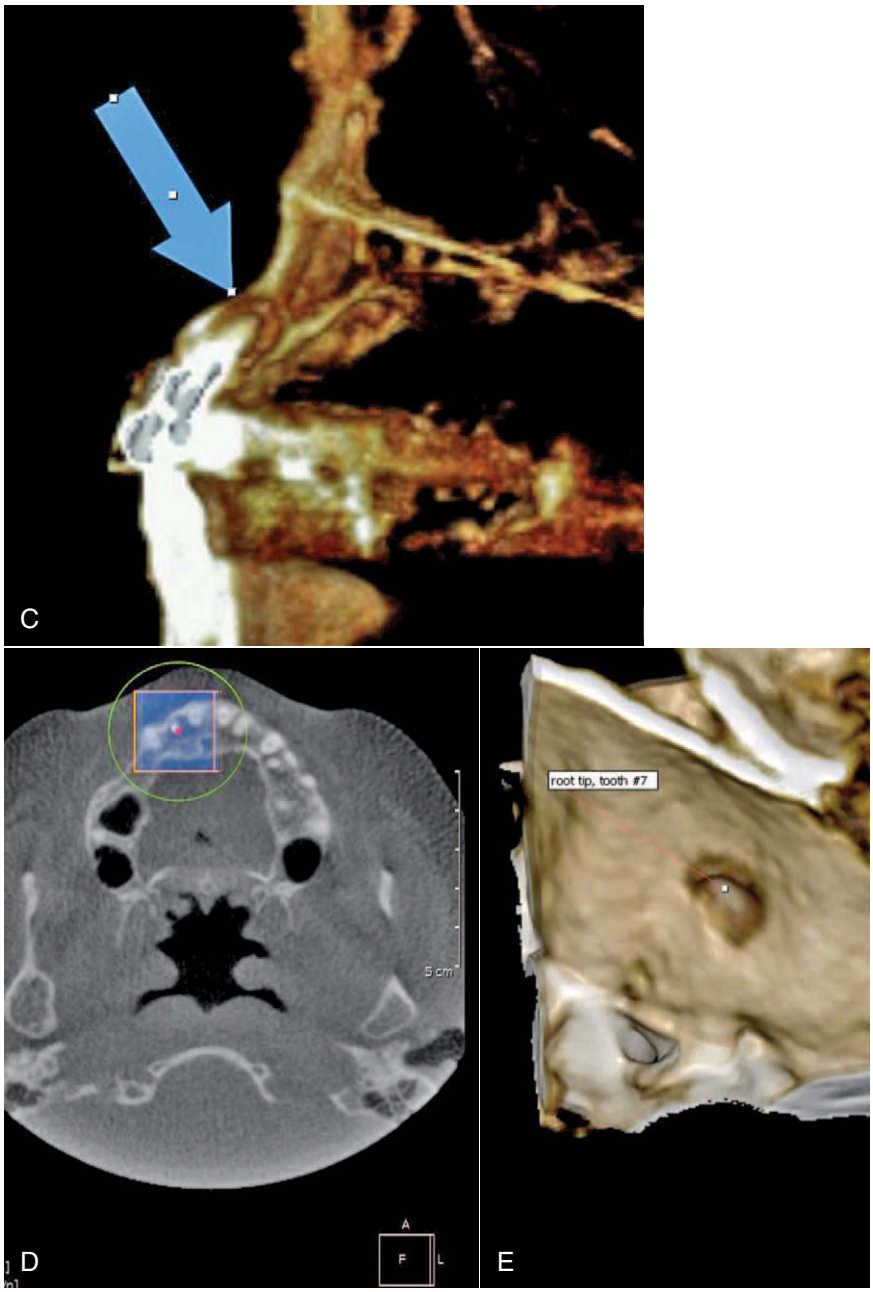


FIG. 29-13, cont'd C, When an oblique-slice tool (found in OnDemand 3D, CyberMed International, Seoul, Korea) is used and the slice thickness thickened, a 3D color image reveals a buccal cortical bone perforation (*arrow*). **D-E,** Using another tool called a *cube tool* and rotating the image to look from the top down on the lesion, the clinician can actually visualize the root apex of tooth #7 through the lesion perforation.

radiograph illustrates incomplete distal canal obturation, with associated periapical bone loss in the area of the apical third of the distal root. The CBVT image reveals a horizontal root fracture in the distal root at the apical extent of the post. Without this valuable information, the clinician may have attempted nonsurgical retreatment or an apicoectomy. Instead the patient can be informed of a hopeless prognosis, and valuable treatment time, expenses, and patient and clinician frustration can be avoided because of the enhanced information provided from the CBVT imaging.

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, with early diagnosis and treatment typically providing an improved prognosis. CBVT imaging of these resorption lesions provides the clinician with enhanced information so that more appropriate treatment-planning options are available (Fig. 29-15).

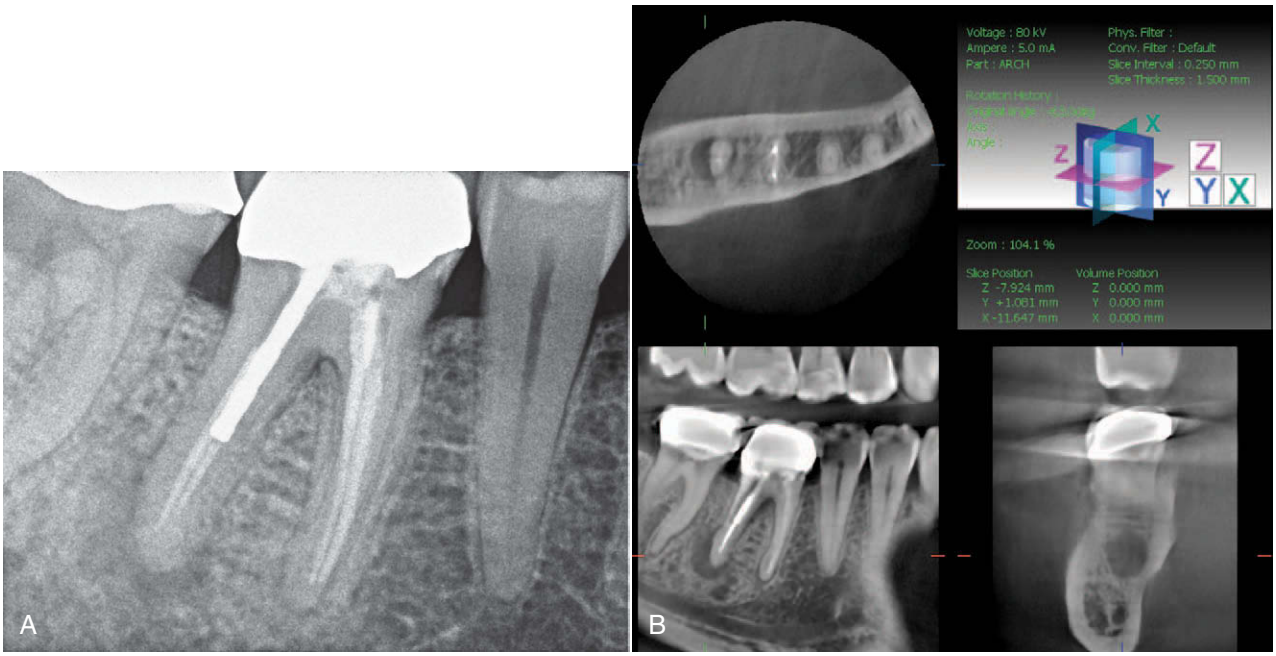


FIG. 29-14 A, Incomplete obturation of distal root canal complex. **B,** CBVT slice of tooth #30 shows the lesion of endodontic origin (LEO) on the distal root more dramatically than the PA. While axial and coronal views do not provide significant information, sagittal view reveals a horizontal root fracture at the apical extent of the post.

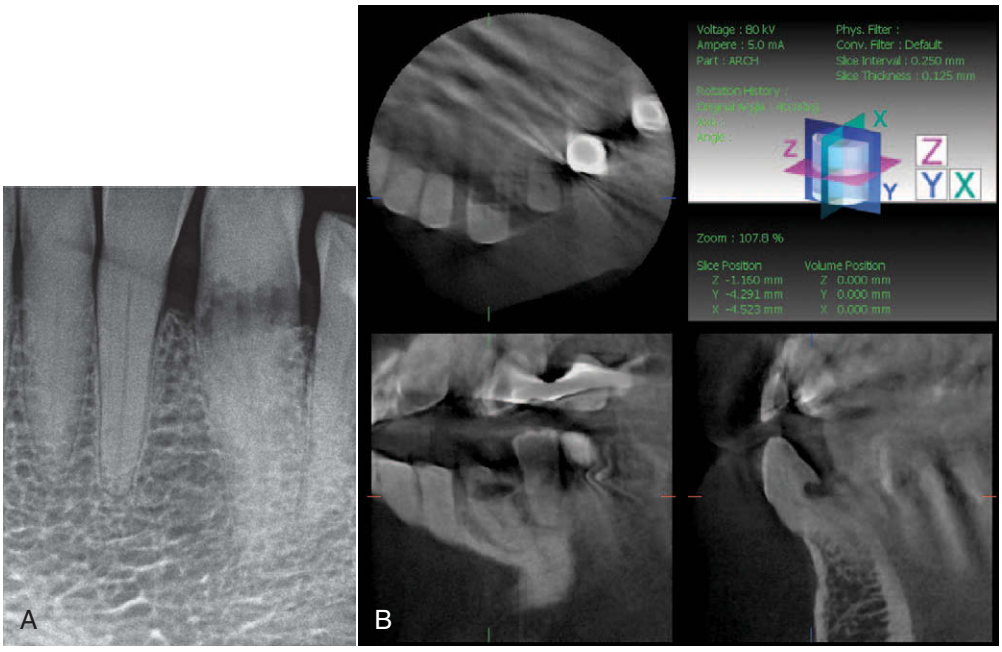


FIG. 29-15 A, Patient referred for evaluation and potential treatment of tooth #22. The periapical (PA) image reveals some significant cervical resorption. Tooth is asymptomatic, and patient was unaware of the problem until clinician discovered the defect upon routine clinical examination. Clinically, the resorptive defect appears primarily on lingual aspect of tooth but extends both to the mesial and distal aspects of tooth and slightly subgingival towards the level of crestal bone. **B,** CBVT slice confirms that resorption defect is significant and congruent with findings of the pretreatment PA images. The defect does communicate with the pulp of the tooth. Endodontic therapy may be indicated, but CBVT imaging suggests a questionable endodontic and restorative prognosis.

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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
- Key Performance Indicators
- Staff Training and Development
- Scheduling
- Customer Service

MARKETING
- Endodontic Referral Marketing

LEADERSHIP
- The Endodontic Leader

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 in recent years.

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 clinicians 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 clinicians believe that dental implants are a better treatment choice 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 the necessary 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 operate successfully as both a provider of patient care and a business enterprise. Documented systems reduce unnecessary stress and allow endodontists to remain chairside 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 a practice begins to grow, the office has a solid management foundation in place to allow for the achievement of more challenging goals. If the practice waits until it gets busier, there is less time to design effective systems, and the endodontist often becomes less motivated to do so owing to a higher level of "busyness" and other responsibilities in life.

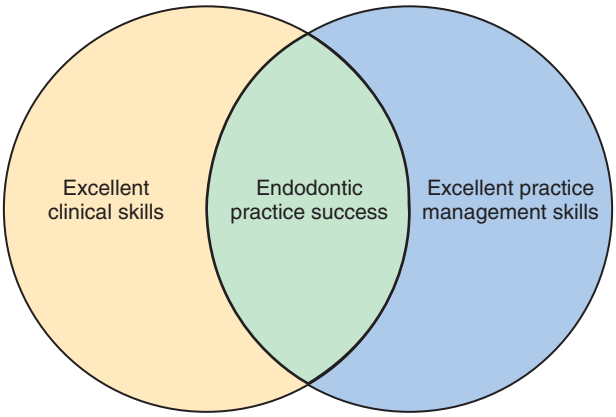


FIG. 30-1 The keys to achieving practice management success.

Systems Design

New practice owners have an excellent opportunity to design systems when capacity is available. 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- to 7-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.

Systems should be designed based on the specific characteristics of each practice. Areas to be considered include:

- Days of operation
- Hours of operation
- Number of staff
- Number of patients
- Number of emergencies
- Average daily patient flow
- Types of procedures offered
- Number of chairs

For example, a practice that sees a higher volume of patients will probably need more staff than a practice that sees a lower number of patients. The same would be true for a practice that participates heavily with insurance companies versus a fee-for-service practice.

Effective management systems can make the difference between a successful endodontic 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 a similar situation.

Assuming a sufficient patient flow, the answer always comes down to management systems. It is not about working harder or faster, but 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:

1. Vision
2. Mission
3. Goal setting
4. Key performance indicators
5. Staff training and development
6. Scheduling
7. 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 to 5 years. Whereas some vision statements may reflect a longer period, this shorter timeframe works well for most endodontists because it is difficult to predict changes in dentistry, the economy, and other events more than 5 years into the future. In addition, after 3 to 5 years, it is time for the clinician 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 to 5 years?
- Does the doctor want to be in solo practice or group practice?
- What 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 a sampling 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 staff to fully grasp such a lengthy and complex vision.

The vision guides the long-term development and growth of the endodontic practice. Once the vision statement has been created, it should be shared with the endodontic 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 5 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 20% 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 5 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 or late 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 5, we will engage an associate doctor.

Mission

Once the vision statement has been created, the next step is the development of a mission statement.

While 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 top of 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 bonus of displaying the mission statement is the opportunity to let patients know of their importance to the practice.

The endodontist should reference 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 is 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 a reality. Goal setting is a step-by-step methodology that leads to the achievement of the overall vision. To be effective as a goal, it 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 hectic pace of today's endodontic practice obliges many doctors to focus on day-to-day operations and fail to step back and see the big picture until many years have gone by. Goal setting and regular goal review 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 achieve their goals and ultimately make their practice vision and mission a reality.

Key Performance Indicators

Without data, an endodontic practice cannot be well managed. To obtain a clear picture of current and potential practice performance, practice goals must be monitored and measured. Key performance indicators (KPIs) provide an accurate assessment of the practice's most critical functions, including:

- Production
- Collections
- Profit
- Overhead
- New patients
- Account receivables
- Case acceptance
- Average production per patient
- Number of re-treats versus initial root canal treatment
- 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 frequency of analysis 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 the team understands 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 needed to perform each staff position. Bear in mind that the majority of endodontic 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 and responsibilities as independently and effectively as possible. During training, it is critical that practices document training policies and procedures. When policies and protocols are documented, they can be clearly communicated to new staff and regularly reinforced by current team members.

One frequent problem is that many team members fail to fully understand their job responsibilities and are not held accountable for their performance, which means the endodon-

tist 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 endodontic practice, endodontists who become effective leaders learn how to delegate appropriate tasks to the endodontic team. The goal for every endodontist 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 will perform and all responsibilities that will be assigned to the endodontic staff. This exercise will allow the endodontist to clearly identify which activities should be handled by the doctor only and which can and 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 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 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 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 the practice 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 goals.

Schedules are created to achieve specific practice production in a low-stress environment. This means that an endodontic practice must establish a target for its annual production and design the schedule accordingly. Each year, endodontic practices should complete an exercise of determining the desired level of growth and create a schedule that allows for that growth. The greatest challenge to increasing production is scheduling limitations that make it all but impossible for the practice to grow.

The following principles are essential when designing the ideal schedule for any individual endodontic office.

Schedule for Production

Design the schedule to achieve a specific production goal. Endodontic practices are typically capable of growing between 5% and 15% annually. Less than 4% growth represents moving into a plateau. There are endodontic practices that can grow by as much as 25% to 30% 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: *How many of each service 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 Goal

Setting the daily goal is a reflection of the annual production goal. To determine the daily goal, simply take the annual production goal and divide it by the number of days the practice will be operating 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 practices are unrealistic about how much time they actually need for procedures. One common example is for root canals to simply be scheduled at one per hour, even though the practice is becoming more stressed and rarely runs on time. Performing "procedural time studies" to determine the exact amount of time needed for each procedure is recommended.

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, either by 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 cases in the morning whenever possible. This can be extremely challenging owing to the number of emergencies seen by endodontists during a normal day. By scheduling larger procedures in the morning, practices can avoid the fatigue factor that occurs later in the day for both the endodontist and the team. 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:

- Handle minor to moderate types of cases in the early afternoon.
- Perform consults and diagnoses in the midafternoon.

- Schedule all minor procedures for the end of the day. These include post-ops, quick checks, and the like.

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 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 a focus on emergency management. 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, enjoyable environment for any endodontic practice.

The first step in managing emergencies is to develop a schedule that allows some flexibility throughout the day. It does not benefit the practice to create a schedule that is 100% maximized without accounting for emergencies. Instead, analyze how many emergencies occur on an average day, and then leave time for that number of emergencies in the schedule. Many high-level endodontic practices will often 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 AM. The practice does not have an opening until 12 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 manages the emergency patient during an emergency time slot, the opportunity to create an excellent flow for the day and a positive customer service experience for every patient seen is maximized. The main point is to provide optimal care and exceptional customer service without disrupting the daily schedule.

The keys to effective emergency management are:

- 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 proper 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 practices tell every emergency patient to come in immediately. The problem arises in 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 a high level of 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 while lowering customer service. This same type of stress is indicative of inefficient practice systems, which means that even though the endodontist is actually working harder, profitability will be lower. An efficient schedule enables the endodontist to provide a high level of care and customer service to all patients.

Customer Service

Customer service is 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 vast majority of endodontic patients are referred to the practice by general dentists. The growth of the endodontic practice 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 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 and/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 in a timely fashion, is running late when the patient arrives, or does not

seem prepared for the appointment, patient confidence and trust disappears, as does their perception of quality care.

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:

- 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 the Yellow Pages, Internet, or recommendation from a friend, but the majority of endodontic patients will be referred by other clinicians. For endodontic practices to reach their true production and profitability potential, a referral marketing program should be in place. The major challenges with implementing a referral marketing program are:

Expertise

Referral marketing is a specialized form of marketing. Endodontists are trained clinicians 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

Endodontists are extremely busy. They are not only the main producer in the practice but often the only producer. It is not unusual for an endodontist to work 8 hours a day highly focused on providing patient care and managing the practice and have little time to think about referral marketing. This is problematic because effective referral marketing is not a few isolated activities but a complete program that must be carried out in a systemized manner to achieve desired results.

Consistency

Perhaps the chief reason referral marketing programs fail is inconsistency. 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 type of marketing leaves practices more susceptible to the effects of 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 in that 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 practices. 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 going to 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 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 extremely appropriate, given that doctors have an obligation to provide

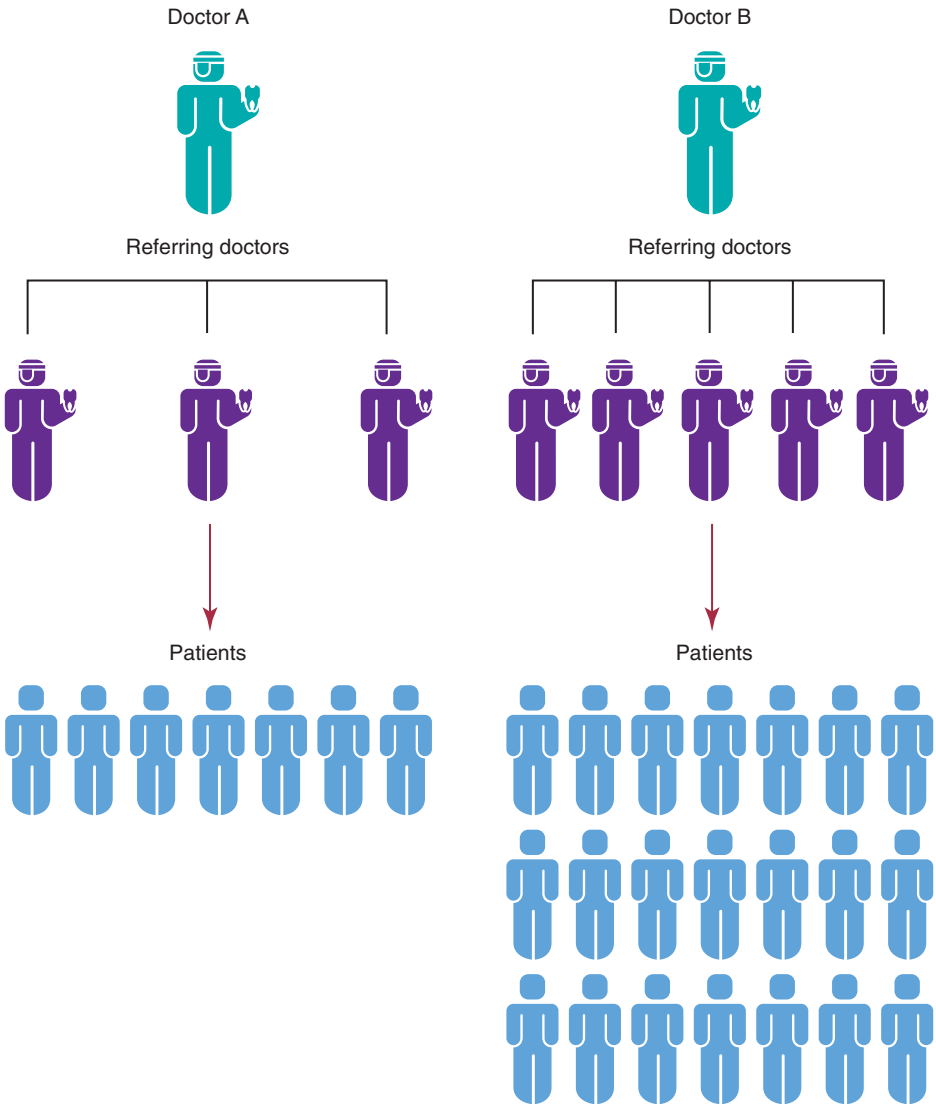


FIG. 30-2 The impact of referral marketing. *Doctor A* does *not* have a formal referral marketing program in place. *Doctor B* does have a formal referral marketing program in place. The results are dramatic. *Doctor B* has many more referring doctors, who refer more patients for treatment.

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 an endodontist can master, the better leader he or she will 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. Unfortunately, being tough or inflexible is not always the best strategy, especially in dentistry. Few endodontists can be tough leaders and still expect to have happy team members and a successful practice. The best practice leaders can motivate their staffs, accomplish goals, and are well respected by their teams. 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 CEO of the practice, endodontists must make business decisions. There are many people who do not progress in business simply because they teeter between two ideas 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 base. They

are confident and have the ability to make decisions that are in their best interest and that of their teams.

Motivation

Motivation is one of the most important skills for a leader. The endodontic leader is able to bring together a team of 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. It is easier to motivate others and work as a team if the leader is trusted and respected.

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 learn sporadically but tend to seek out opportunities to learn new ideas and strategies on an ongoing basis.

Teamwork

Endodontic leaders never work on their own. They 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 leadership 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.

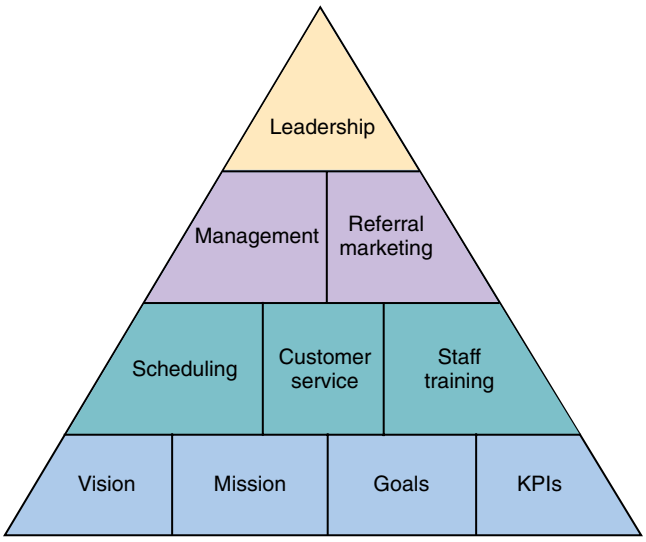


FIG. 30-3 Key principles of endodontic practice management.

CONCLUSION

Effective practice management involves management systems and referral marketing that allows an endodontic practice to provide outstanding patient care while operating as a successful business enterprise (Fig. 30-3). The #1 source of practice stress is the lack of efficient systems. When an endodontic 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.

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