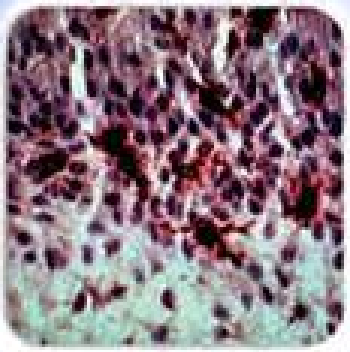

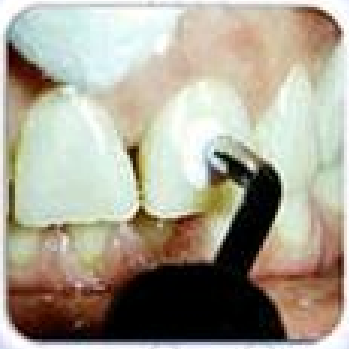

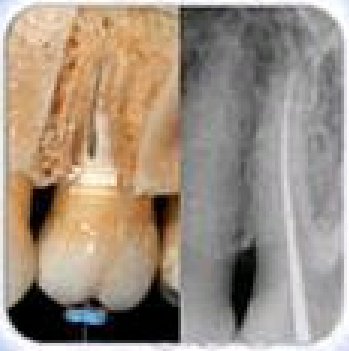

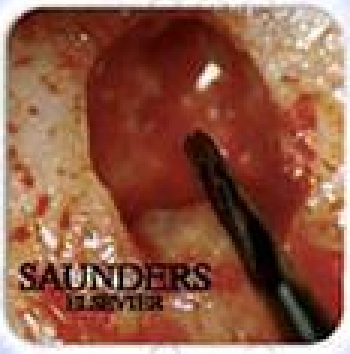

DVD
VIDEOS AND CHAPTER REVIEW QUESTIONS

4th Edition

ENDODONTICS

PRINCIPLES AND PRACTICE

Mahmoud Torabinejad
Richard E. Walton

Endodontics

Principles and Practice

Fourth Edition

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PREFACE

The primary objective of dentists has always been to prevent tooth loss. Despite this effort, many teeth develop caries, suffer traumatic injury, or are impacted by other diseases and disorders, often requiring endodontic care. Endodontics is a discipline of dentistry that deals with the morphology, physiology, and pathology of the human dental pulp and periapical tissues, as well as the prevention and treatment of diseases and injuries related to these tissues. Its scope is wide and includes diagnosis and treatment of pain of pulpal and/or periapical origin, vital pulp therapy, nonsurgical root canal treatment, retreatment of unsuccessful treatment, internal bleaching and endodontic surgery. Ultimately, the primary goal in endodontics is to preserve the natural dentition. Root canal treatment is a well-tested procedure that has provided pain relief and has restored function and esthetics to patients. Millions of patients expect preservation of their natural dentition; if root canal treatment is necessary, they should be aware that the procedure is safe and has a high success rate if properly performed.

As with other dental specialties, the practice of endodontics requires two inseparable components: art and science. The *art* consists of executing technical procedures during root canal treatment. The *science* includes the basic and clinical sciences related to biological and pathological conditions that guide the art of endodontics through the principles and methods of evidence-based treatment. Evidence-based treatment integrates the best clinical evidence with the practitioner's clinical expertise and the patient's treatment needs and preferences. A principal objective of our textbook is to incorporate evidence-based information when available and when appropriate.

Because there are not enough endodontists to manage the endodontic needs of the public, general dentists must assist endodontists to preserve natural dentition. Their responsibility is to diagnose pulpal and periapical diseases and to perform noncomplicated root canal treatments. Our textbook, written specifically for dental students and general dentists, contains the information necessary for those who would like to incorporate endodontics in their practice. This includes diagnosis and treatment planning as well as management of pulpal and periapical diseases. In addition, the general dentist must be able to determine the case complexity and whether she or he can perform the necessary treatment or if referral is the better option.

Although many advances have been made in endodontics in the past decade, the main objectives of root canal therapy continue to be the removal of diseased tissue, the elimination of microorganisms, and the prevention of recontamination after treatment. This new edition of *Endodontics: Principles and Practice* has been systematically organized to simulate the order of procedures performed in a clinical setting. It contains information regarding normal structures, etiology of disease, diagnosis and treatment planning, local anesthesia, emergency treatment, root canal instruments, access preparations, cleaning and shaping, obturation, and temporization. In addition, it covers etiology, prevention, and treatment of accidental procedural errors, as well as treatment of inadequate root canal–treated teeth using nonsurgical and surgical approaches. Furthermore, it provides guidelines regarding the assessment of outcomes of these procedures. Finally, we have added an appendix containing self-assessment questions. Except for one chapter, these questions were developed by two endodontists not involved in the actual writing of the chapters, our belief being that having questions asked by people independent of the text itself adds additional value to the questions: This process (1) ensures that the reader understands the purpose of the writer and (2) assesses the knowledge of the reader.

The other distinctive features of the new edition are (1) presentation of color figures, (2) new trim size of the book, (3) updated relevant and recent references, (4) information regarding new scientific and technological advances in the field of endodontics, and (5) a revised table of contents. Appendix A provides colorized illustrations that depict the size, shape, and location of the pulp space within each tooth. There is also a DVD with video clips for selected procedures and an interactive version of the self-assessment questions that appear in Appendix B, along with rationales for each question, to test subject comprehension. These features provide the reader with a textbook that is concise, current, and easy to follow in an interactive manner.

The new format gives the reader an opportunity to learn the scope of the contemporary principles and practice of endodontics. This textbook is not intended to include all background information on the art and science of endodontics. At the same time, it is not designed to be a "cookbook" or a preclinical laboratory technique manual. We have tried to provide the reader with the basic information to perform root canal treatment and to give the reader background knowledge in related areas. This textbook should be used as a building block for understanding the etiology and treatment of teeth with pulpal and periapical diseases; then the reader can expand her or his endodontic experiences with more challenging cases. Providing the best quality of care is the guiding light for treatment planning and performing appropriate treatment.

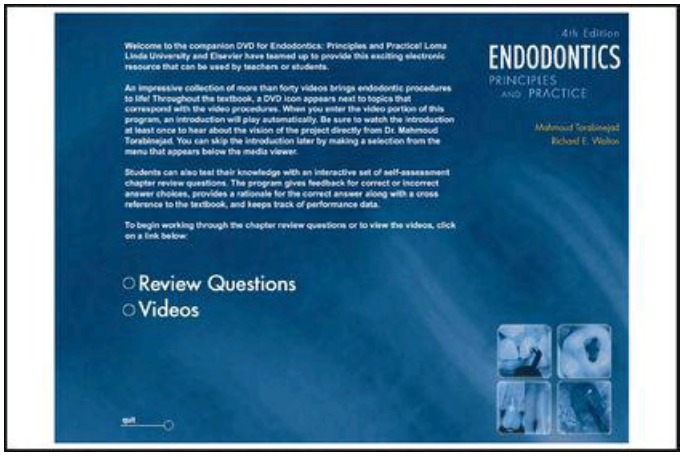
We thank the contributing authors for sharing their materials and experiences with our readers and with us. Their contributions improve the quality of life for millions of patients. We also express our appreciation to the editorial staff of Elsevier, whose collaboration and dedication made this project possible. In addition, we acknowledge our colleagues and students who provided cases and gave us constructive suggestions to improve the quality of our textbook. Because much of their material is incorporated into the new edition, we also would like to acknowledge the contributors to the third edition: Frances M. Andreasen, Jens O. Andreasen, J. Craig Baumgartner, Stephen Cohen, Shimon Friedman, Kenneth M. Hargreaves, Gerald W. Harrington, Jeffrey W. Hutter, Thomas R. Pitt Ford, Gerald L. Scott, Denis E. Simon III, David R. Steiner, Calvin D. Torneck, James A. Wallace, and Peter R. Wilson. We also would like to thank Laura Walton, Harriet M Bogdanowicz, and Mohammad Torabinejad for editing and proofreading of the manuscripts.

Mahmoud Torabinejad, Richard E. Walton

HOW TO USE THE COMPANION DVD

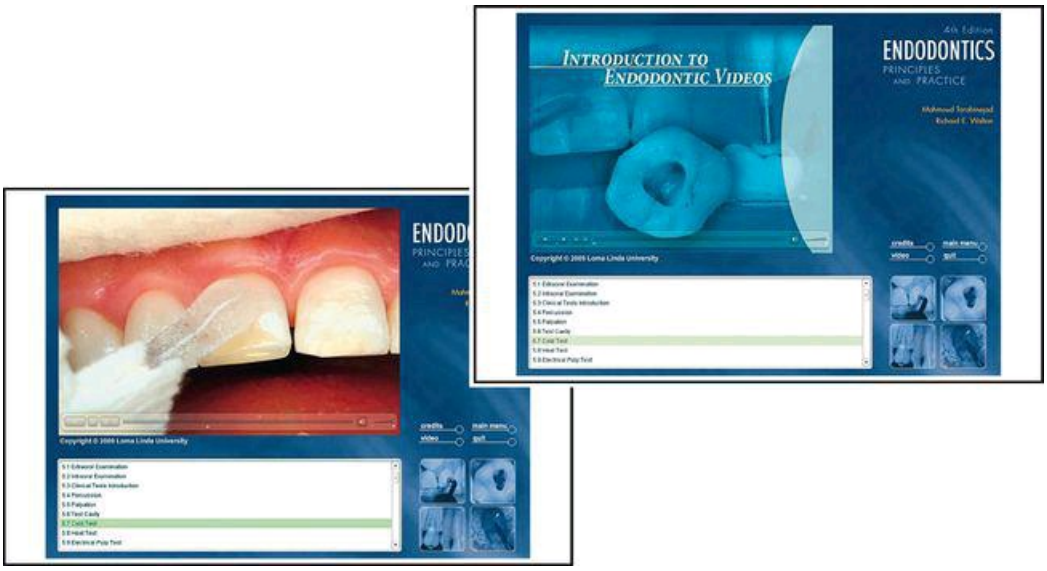
Elsevier and Loma Linda University are pleased to provide this exciting electronic resource that can be used as a teaching tool for classroom or individual student use. For system requirements, see the card that is packaged with the disk. This DVD includes a video collection of endodontic procedures that was produced at Loma Linda University and interactive review questions for each chapter.

On the main menu, use the cursor to click on the section you wish to view:



VIDEOS

When you enter the video portion of this program, an introduction will play automatically. Be sure to watch the introduction at least once to hear about the vision of the project directly from Dr. Mahmoud Torabinejad.



As you visit the video collection subsequent times and want to skip the introduction, simply make a selection from the menu that appears below the media viewer to move to the topic of your choice:

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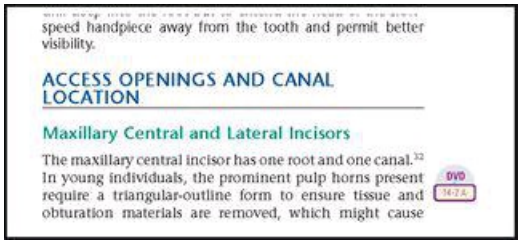

speed handpiece away from the tooth and permit better visibility.

ACCESS OPENINGS AND CANAL LOCATION

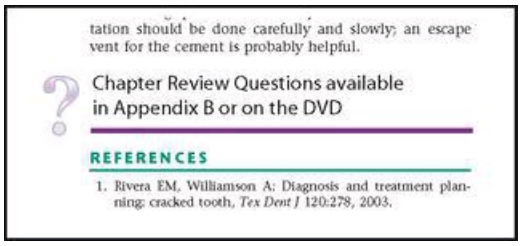
Maxillary Central and Lateral Incisors

The maxillary central incisor has one root and one canal.[32] In young individuals, the prominent pulp horns present require a triangular-outline form to ensure tissue and obturation materials are removed, which might cause

DVD

CHAPTER REVIEW QUESTIONS

When you complete a chapter in the textbook, you will find a reminder to work through the chapter review questions. The questions can be found in the back of the textbook or in an interactive format on the DVD:

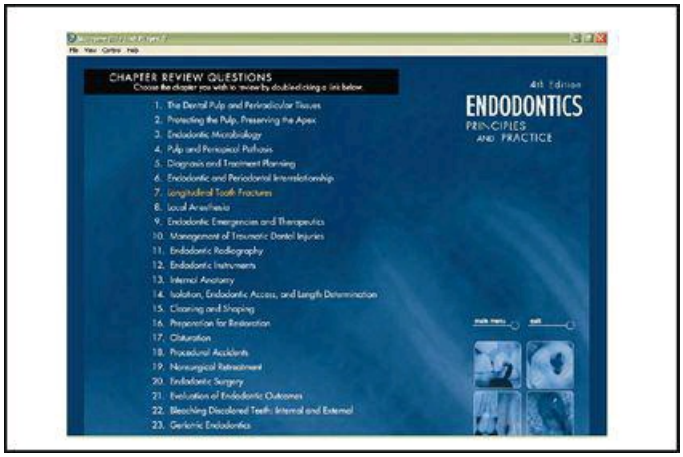

tation should be done carefully and slowly; an escape vent for the cement is probably helpful.

Chapter Review Questions available in Appendix B or on the DVD

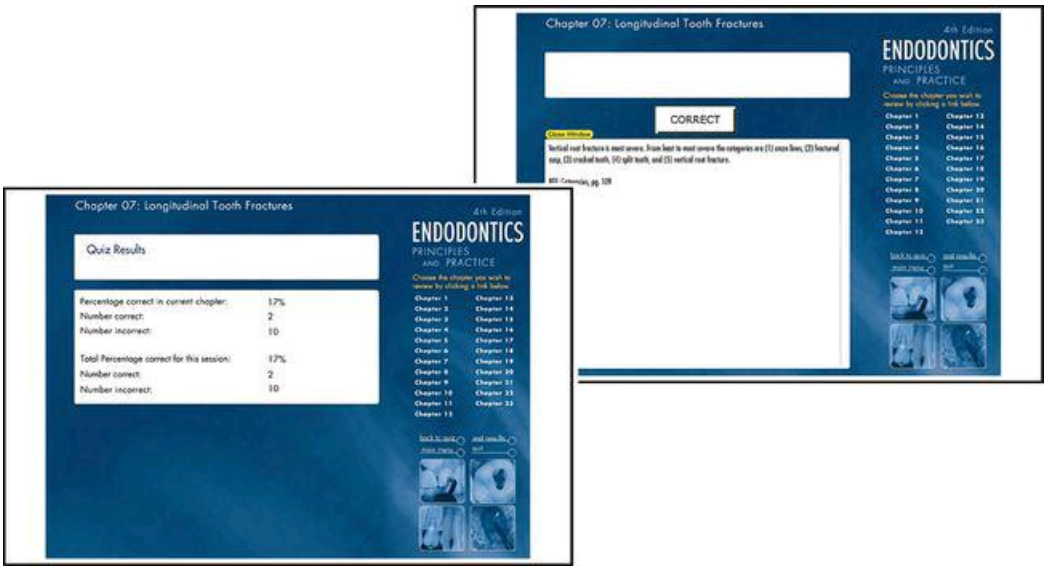
REFERENCES

1. Rivera EM, Williamson A: Diagnosis and treatment planning: cracked tooth, *Tex Dent J* 120:278, 2003.

To access the review questions for a specific chapter, click on the title from the list of chapters:



As you work through the questions for each chapter, the program will provide a rationale for correct answer selections and a cross reference to the textbook.



The program also keeps track of performance data for each chapter:

Table of Contents

The Dental Pulp and Periradicular Tissues

Graham Rex Holland, Mahmoud Torabinejad

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe the development of pulp.

2. Describe the process of root development.

3. Recognize the anatomic regions of pulp.

4. List all cell types in the pulp and describe their function.

5. Describe both fibrous and nonfibrous components of the extracellular matrix of pulp.

6. Describe the blood vessels and lymphatics of pulp.

7. List the neural components of pulp and describe their distribution and function.

8. Discuss theories of dentin sensitivity.

9. Describe the pathway of efferent nerves from pulp to the central nervous system.

10. Describe the changes in pulp morphology that occur with age.

11. Describe the structure and function of the periradicular tissues.

Dental pulp is the soft tissue located in the center of the tooth. It forms, supports, and is an integral part of the dentin that surrounds it. The *primary function* of the pulp is formative; it gives rise to odontoblasts that not only form dentin but also interact with dental epithelium early in tooth development to initiate the formation of enamel. Subsequent to tooth formation, pulp provides several *secondary functions* related to tooth sensitivity, hydration, and defense. Injury to pulp may cause discomfort and disease. Consequently, the health of the pulp is important to the successful completion of restorative and prosthetic dental procedures. In restorative dentistry, for example, the size and shape of the pulp must be considered to determine cavity depth. The size and shape of the pulp depend on the tooth type (e.g., incisor, molar), the degree of tooth development related to the age of the patient, and any restorative procedures the tooth may have had. The stage of development influences the type of pulp treatment rendered when pulp injury occurs. Procedures routinely undertaken on a fully developed tooth are not always practical for a tooth that is only partially developed. In these cases, other special procedures rarely used on mature teeth are applied.

Because endodontics is the diagnosis and treatment of diseases of the pulp and their sequelae, a knowledge of the biology of the pulp is essential for the development of a rational treatment plan. Lesions that do not arise from the pulp may be mistaken for those that do. For example, the appearance of periodontal lesions of endodontic origin can be similar to that of lesions induced by primary disease of the periodontium, or by injury or disease that does not arise from the tooth. An inability to differentiate apparently similar lesions may lead to misdiagnosis and incorrect treatment.

Comprehensive descriptions of pulp embryology, histology, and physiology are available in several dental texts. This chapter presents an overview of the biology of the pulp and the periodontium, including development, anatomy, and function, which affect pulp disease, as well as periradicular disease and its related symptoms.

EMBRYOLOGY OF THE DENTAL PULP

Early Development of Pulp

The tooth originates as a band of epithelial cells, the dental lamina (Figure 1-1, *A*), in the embryonic jaws. Downgrowths from this band will ultimately form the teeth. The stages of tooth formation are described by the shapes of these downgrowths, the tooth germs. Initially, they look like the bud of a forming flower (Figure 1-1, *B*) and become invaginated at what is at first called the cap stage (Figure 1-1, *C*) but then, as the tooth germ grows in size and the invagination deepens, becomes the bell stage (Figure 1-1, *D*). The tissue within the invagination will ultimately become the dental pulp, which is known as the *dental papilla,* during the early stages of development. The papilla and thus the pulp are derived from cells that have migrated from the neural crest (ectomesenchymal cells) and mingled with cells of local mesenchymal origin. During the bell stage, the inner layer of cells of the enamel organ will differentiate into ameloblasts (Figure 1-2, *A*). This is followed by the outer layer of cells of the dental papilla, which will differentiate into odontoblasts (Figure 1-2, *B*) and begin to lay down dentin (Figure 1-2, *C*). From this point on, the tissue is known as the dental pulp.

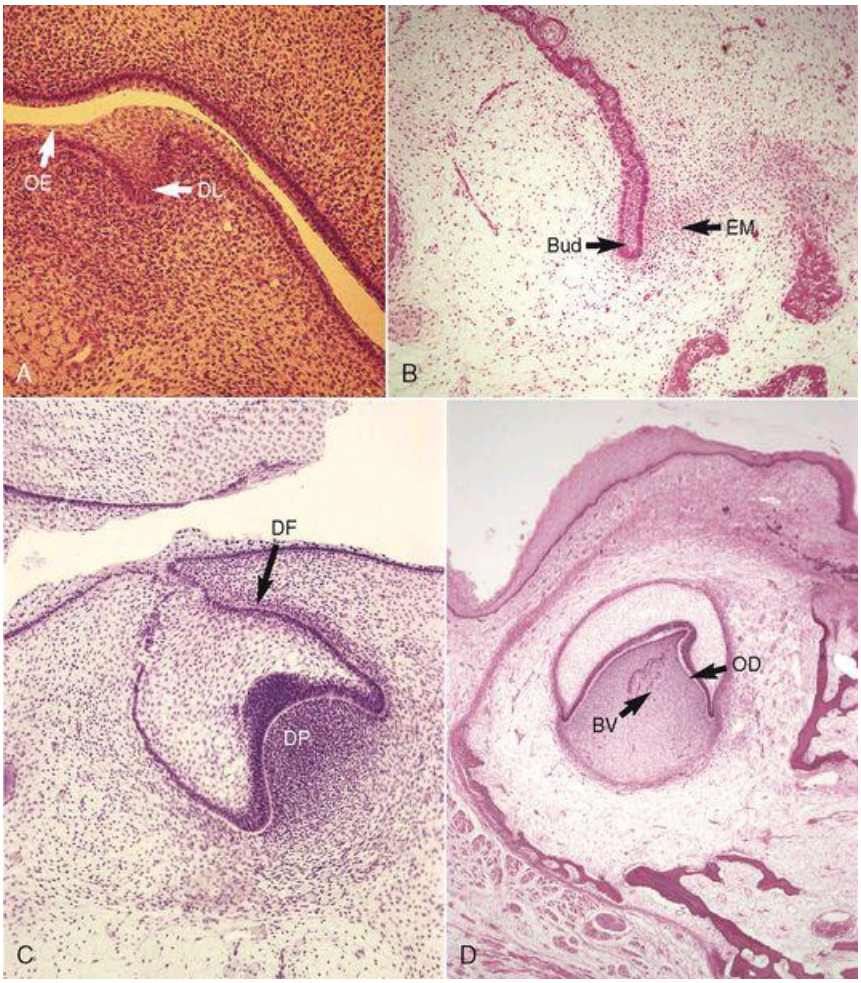


Figure 1-1 A, Earliest stage of tooth development. The dental lamina *(DL)* invaginates from the oral epithelium *(OE).* **B,** Bud stage of tooth development. Ectomesenchyme *(EM)* is beginning to condense around the tooth germ. **C,** The cap stage of tooth development. The condensed ectomesenchyme within the invagination is the dental papilla *(DP).* The dental follicle *(DF)* is beginning to develop around the tooth germ. **D,** Early bell stage. The odontoblast layer *(OD)* and blood vessels *(BV)* are visible in the dental pulp.

(Courtesy Dr. H. Trowbridge.)

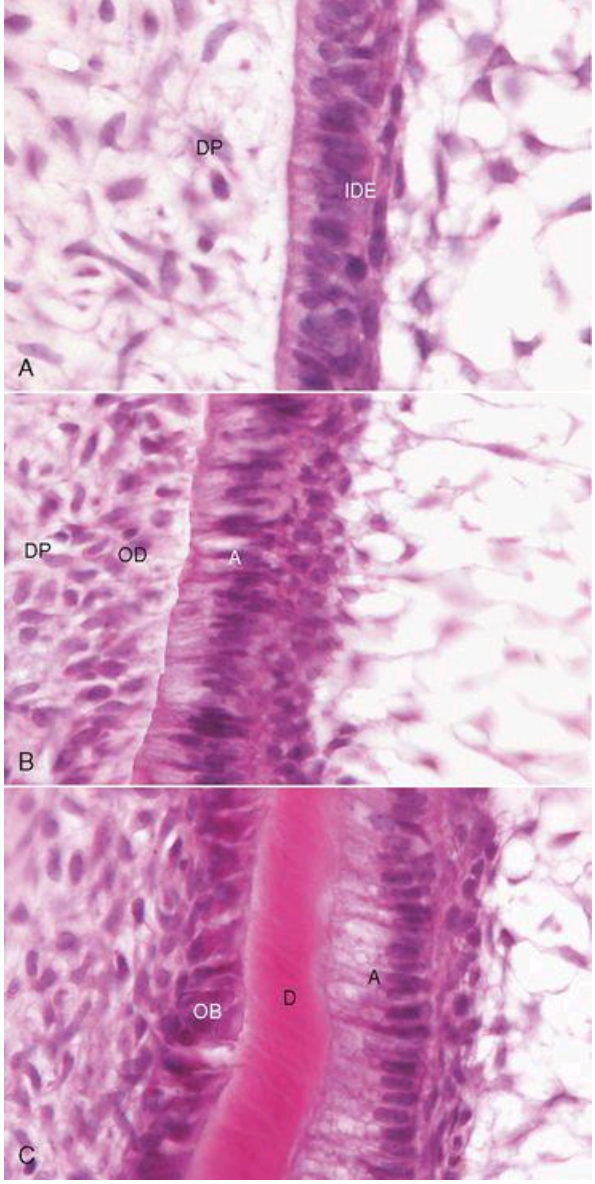


Figure 1-2 A, At the late cap stage the internal dental epithelium *(IDE)* has differentiated into a layer of ameloblasts but not laid down enamel. The outer layer of the dental papilla *(DP)* has not yet differentiated into odontoblasts. **B,** Slightly later than Figure 1-2, *A*, the outer cells of the dental papilla are beginning to become odontoblasts *(OD)* at the periphery of what now is the dental pulp *(DP).* The ameloblasts *(A)* are fully differentiated, but no enamel has yet formed. **C,** In the bell stage the odontoblasts *(OB)* are laying down dentin *(D),* but the ameloblasts *(A)* have laid down little, if any, enamel.

(Courtesy Dr. H. Trowbridge.)

The differentiation of odontoblasts from undifferentiated ectomesenchymal cells is initiated and controlled by the ectodermal cells of the inner dental epithelium. These produce growth factors and signaling molecules that pass into the basal lamina of the epithelium and from there to the cells that are becoming odontoblasts. The cells beneath the forming odontoblasts remain as undifferentiated stem cells and retain the potential throughout life to differentiate into odontoblasts.

Once the odontoblast layer has differentiated, the basal lamina of the inner dental epithelium that contained the signaling molecules disappears, and the odontoblasts, now linked to each other by tight junctions, desmosomal junctions, and gap junctions, begin to lay down dentin (see Figure 1-2, *C*).[1] Once dentin formation has begun, the cells of the inner dental epithelium begin to deposit enamel. The back and forth signaling controlling differentiation and the initiation of hard tissue formation is an example of epithelial-mesenchymal interaction, a key developmental process that has been heavily studied in the tooth germ model. The deposition of unmineralized dentin matrix begins at the cusp tip. Deposition progresses in a cervical (apical) direction in a regular rhythm at an average of 4.5 µm/day.[2] Crown shape is genetically predetermined by the proliferative pattern of the cells of the inner dental epithelium. The first thin layer of dentin formed is called *mantle dentin.* The direction and size of the collagen fibers in mantle dentin differ from those in the subsequently formed circumpulpal dentin. The pattern of matrix formation followed by its mineralization continues throughout dentin deposition. Between 10 and 50 µm of the dentin matrix immediately adjacent to the odontoblast layer remains unmineralized at all times and is known as *predentin.*

As crown formation occurs, vascular and sensory neural elements begin migrating into the pulp from the future root apex in a coronal direction. Both elements undergo branching and narrowing toward the odontoblast layer, and each will at a late stage form plexuses beneath the layer.

Root Formation

The cells of the inner and outer dental epithelia meet at a point known as the *cervical loop.* This delineates the end of the anatomical crown and the site where root formation begins. Root formation is initiated by the apical proliferation of the two fused epithelia, now known as *Hertwig's epithelial root sheath.*[3] The function of the sheath is similar to that of the inner enamel epithelium during crown formation. It provides signals for the differentiation of odontoblasts and thus acts as a template for the root (Figure 1-3, *A*). Cell proliferation in the root sheath is genetically determined; its pattern regulates whether the root will be wide or narrow, straight or curved, long or short, or single or multiple. Multiple roots result when opposing parts of the root sheath proliferate horizontally as well as vertically. As horizontal segments of Hertwig's epithelial root sheath join as the "epithelial diaphragm," the pattern for multiple root formation is laid down. This pattern is readily discernible when the developing root end is viewed microscopically (Figure 1-3, *B*).

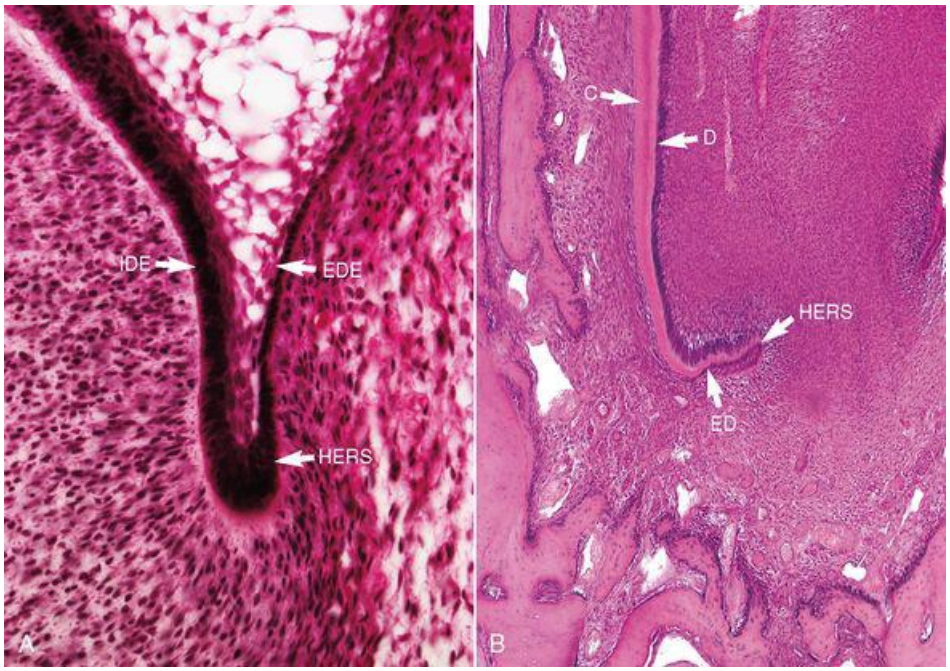


Figure 1-3 A, The formation of Hertwig's epithelial root sheath *(HERS)* from the internal *(IDE)* and external *(EDE)* epithelia. **B,** Hertwig's epithelial root sheath *(HERS)* has extended. Both dentin *(D)* and cementum *(C)* have been deposited. HERS has changed direction to form the epithelial diaphragm *(ED).*

After the first dentin in the root has formed, the basement membrane beneath Hertwig's sheath breaks up and the innermost root sheath cells secrete a hyaline material over the newly formed dentin. After mineralization has occurred, this becomes the *hyaline layer of Hopewell-Smith,* which helps bind the soon-to-be-formed cementum to dentin. Fragmentation of Hertwig's epithelial root sheath occurs shortly afterward. This fragmentation allows cells of the surrounding dental follicle (the future periodontium) to migrate and contact the newly formed dentin surface, where they differentiate into cementoblasts and initiate acellular cementum formation (Figure 1-4).[4] This cementum ultimately serves as an anchor for the developing principal fibers of the periodontal ligament (PDL). In many teeth, cell remnants of the root sheath persist in the periodontium in close proximity to the root after root development has been completed. These are the *epithelial cell rests of Malassez.*[5] Normally functionless, in the presence of inflammation, they can proliferate and may under certain conditions give rise to a radicular cyst.[6]

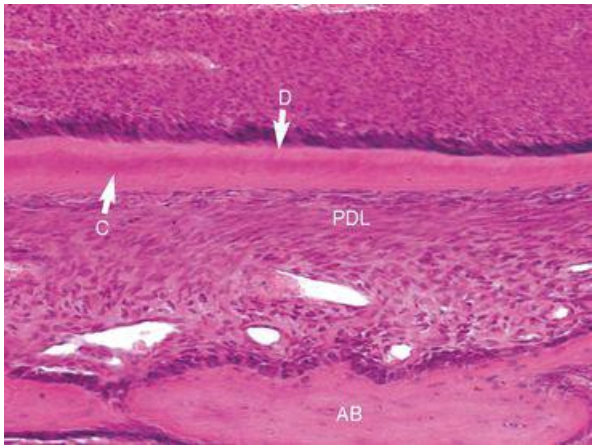


Figure 1-4 Developing dentin *(D),* cementum *(C),* periodontal ligament *(PDL),* and alveolar bone *(AB)*.

Formation of Lateral Canals and Apical Foramen

Lateral Canals

Lateral canals (or, synonymously, accessory canals) are channels of communication between pulp and PDL (Figure 1-5). They form when a localized area of root sheath is fragmented before dentin formation. The result is direct communication between pulp and the PDL via a channel through the dentin and cementum that carries small blood vessels and perhaps nerves. Lateral canals may be single or multiple or large or small. They may occur anywhere along the root but are most common in the apical third. In molars, they may join the pulp chamber PDL in the root furcation. *Lateral canals are clinically significant; like the apical foramen, they represent pathways along which disease in the pulp may extend to periradicular tissues and occasionally allow disease in the periodontium to extend to the pulp.*

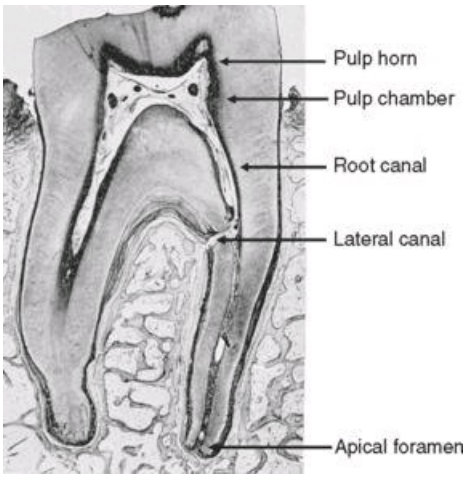


Figure 1-5 Anatomic regions of the root canal system highlighting the pulp horn(s), pulp chamber, root canal, lateral canal, and apical foramen. The pulp, which is present in the root canal system, communicates with the periodontal ligament primarily through the apical foramen and the lateral canal(s).

(Courtesy Orban Collection.)

Apical Foramen

The epithelial root sheath continues to extend until the full, predetermined length of the root is reached. As the epithelial root sheath extends, it encloses more dental papilla until only an apical foramen remains through which pulpal vessels and nerves pass. During root formation, the apical foramen is usually located at the end of the anatomic root. When tooth development has been completed, the apical foramen is smaller and found a short distance coronal to the anatomic end of the root.[7] This distance increases as later apical cementum is formed.

There may be one foramen or multiple foramina at the apex. Multiple foramina occur more often in multirooted teeth. When more than one foramen is present, the largest one is referred to as the apical foramen and the smaller ones as accessory canals (in combination they constitute the *apical delta*). The diameter of the apical foramen in a mature tooth usually ranges between 0.3 and 0.6 mm. The largest diameters are found on the distal canal of mandibular molars and the palatal root of maxillary molars. Foramen size is unpredictable, however, and cannot be accurately determined clinically.

Formation of the Periodontium

Tissues of the periodontium develop from ectomesenchyme-derived fibrocellular tissue that surrounds the developing tooth *(dental follicle).* After the mantle dentin has formed, enamel-like proteins are secreted into the space between the basement membrane and the newly formed collagen by the root sheath cells. This area is not mineralized with the mantle dentin but does mineralize later and to a greater degree to form the hyaline layer of Hopewell-Smith. After mineralization has occurred, the root sheath breaks down. This fragmentation allows cells from the follicle to proliferate and differentiate into cementoblasts, which lay down cementum over the hyaline layer. Bundles of collagen, produced by fibroblasts in the central region of the follicle *(Sharpey's fibers),* are embedded in the forming cementum and will become the principal fibers of the periodontal ligament. At the same time, cells in the outermost area of the follicle differentiate into osteoblasts to form the bundle bone that also will anchor the periodontal fibers. Later, periodontal fibroblasts produce more collagen that binds the anchored fragments together to form the principal periodontal fibers that suspend the tooth in its socket. Loose, fibrous connective tissue carrying nerves and blood vessels remains between the principal fibers. Undifferentiated mesenchymal cells (tissue-specific stem cells) are plentiful in the periodontium and possess the ability to form new cementoblasts, osteoblasts, or fibroblasts in response to specific stimuli. Cementum formed after the formation of the principal periodontal fibers is cellular and plays a lesser role in tooth support.

The blood supply to the periodontium is derived from the surrounding bone, gingiva, and branches of the pulpal vessels.[8] It is extensive and supports the high level of cellular activity in the area. The pattern of innervation is similar to that of the vasculature. The neural supply consists of small, unmyelinated sensory and autonomic nerves and larger myelinated sensory nerves. Some of the latter terminate as unmyelinated neural structures thought to be nociceptors and mechanoreceptors.

ANATOMIC REGIONS AND THEIR CLINICAL IMPORTANCE

The tooth has two principal anatomic divisions, root and crown, that join at the cervix *(cervical region).* The pulp space is similarly divided into coronal and radicular regions. In general, the shape and the size of the tooth surface determine the shape and the size of the pulp space. The coronal pulp is subdivided into pulp horn(s) and pulp chamber (see Figure 1-5). Pulp horns extend from the chamber into the cuspal region. In young teeth, they are extensive and may be inadvertently exposed during routine cavity preparation.

The pulp space becomes asymmetrically smaller after root lengthening is complete because of the continued, albeit slower, production of dentin. There is a pronounced decrease in the height of the pulp horn and a reduction in the overall size of the pulp chamber. In molars, the apical-occlusal dimension is reduced more than the mesial-distal dimension. Excessive reduction of the size of the pulp space is clinically significant and can lead to difficulties in locating, cleaning, and shaping the root canal system (Figure 1-6).

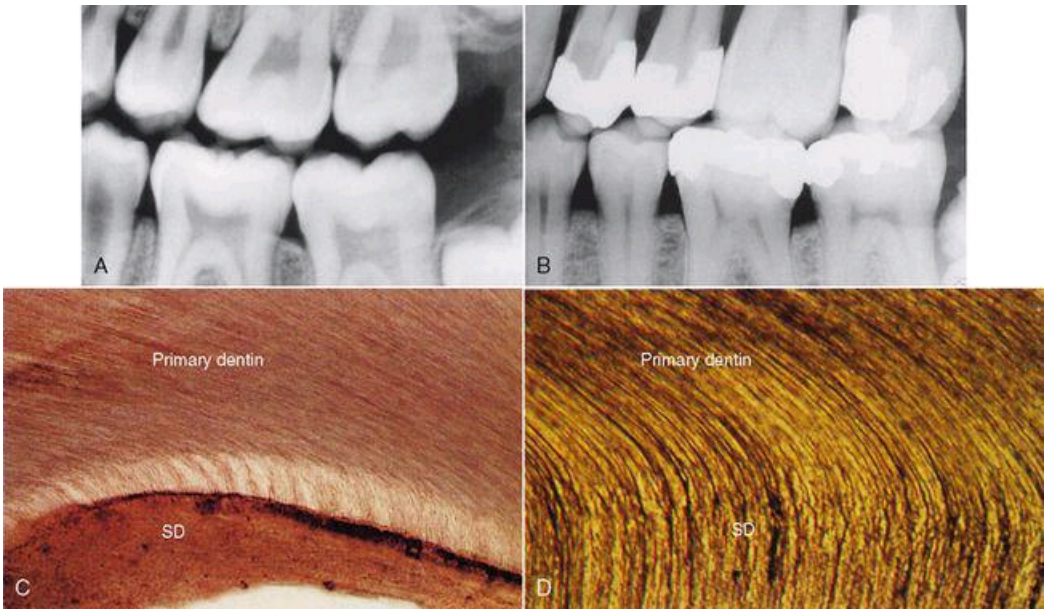


Figure 1-6 A and **B,** Radiographic changes noted in the shape of the pulp chamber over time. The posterior bitewing radiographs were taken 15 years apart. The shapes of the root canal systems have been altered as a result of secondary dentinogenesis and by the deposition of tertiary dentin in instances where deep restorations are present. **C,** Secondary dentin *(SD).* Ground section at low power. **D,** Secondary dentin *(SD)* at high power.

The anatomy of the root canal varies not only between tooth types but also within tooth types. Although at least one canal must be present in each root, some roots have multiple canals of varying sizes. *Understanding and appreciating all aspects of root canal anatomy are essential prerequisites to root canal treatment.*

Variation in the size and location of the apical foramen influences the degree to which blood flow to the pulp may be compromised after a traumatic event. *Young, partially developed teeth have a better prognosis for pulp survival than teeth with mature roots* (Figure 1-7).

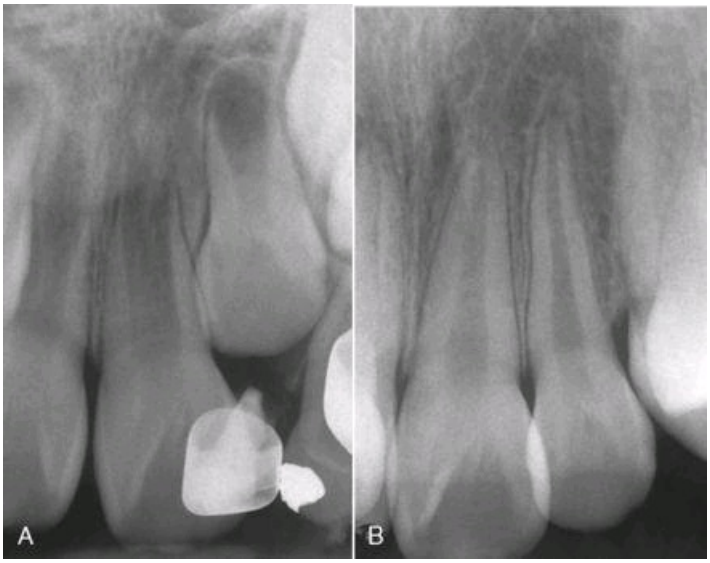


Figure 1-7 Changes in the anatomy of the tooth root and pulp space. **A,** A small crown-root ratio, thin dentin walls, and divergent shape in the apical third of the canal are seen. **B,** Four years later, a longer root, greater crown-root ratio, smaller pulp space, and thicker dentin walls with a convergent shape are seen.

Posteruptive deposition of cementum in the region of the apical foramen creates a disparity between the radiographic apex and the apical foramen. It also creates a funnel-shaped opening to the foramen that is often larger in diameter than the intraradicular portion of the foramen. The narrowest portion of the canal is referred to as the *apical constriction.* However, a constriction is not clinically evident in all teeth. Cementum contacts dentin inside the canal coronal to the cementum surface. This is the *cementodentinal junction (CDJ).* The CDJ level varies not only from tooth to tooth but also within a single root canal. One study estimated the junction to be located 0.5 to 0.75 mm coronal to the apical opening.[7] Theoretically, that is the point where the pulp terminates and the PDL begins. However, histologically and clinically, it is not always possible to locate that point. Cleaning, shaping, and obturation of the root canal should terminate short of the apical foramen and remain confined to the canal to avoid unnecessary injury to the periapical tissues. *The determination of root length and the establishment of a working length are essential steps in root canal preparation. The radiograph and electronic apex locators are helpful in establishing the root length.*

PULP FUNCTION

The pulp performs five functions, some formative and others supportive.

Induction

Pulp participates in the initiation and development of dentin.[9] When dentin is formed, it leads to the formation of enamel. These events are interdependent, in that enamel epithelium induces the differentiation of odontoblasts, and odontoblasts and dentin induce the formation of enamel. Such epithelial-mesenchymal interactions are the core processes of tooth formation.

Formation

Odontoblasts form dentin.[10] These highly specialized cells participate in dentin formation in three ways: (1) by synthesizing and secreting inorganic matrix, (2) by initially transporting inorganic components to newly formed matrix, and (3) by creating an environment that permits mineralization of matrix. During early tooth development, primary dentinogenesis is generally a rapid process. After tooth maturation, dentin formation continues at a much slower rate and in a less symmetric pattern *(secondary dentinogenesis).* Odontoblasts can also form dentin in response to injury, which may occur in association with caries, trauma, or restorative procedures. Generally, this dentin is less organized than primary and secondary dentin and mostly localized to the site of injury. This dentin is referred to as *tertiary dentin*. Tertiary dentin has two forms. Reactionary tertiary dentin is tubular, with the tubules continuous with those of the original dentin. It is formed by the original odontoblasts. Reparative dentin is formed by new odontoblasts differentiated from stem cells after the original odontoblasts have been killed. It is largely atubular (Figure 1-8).

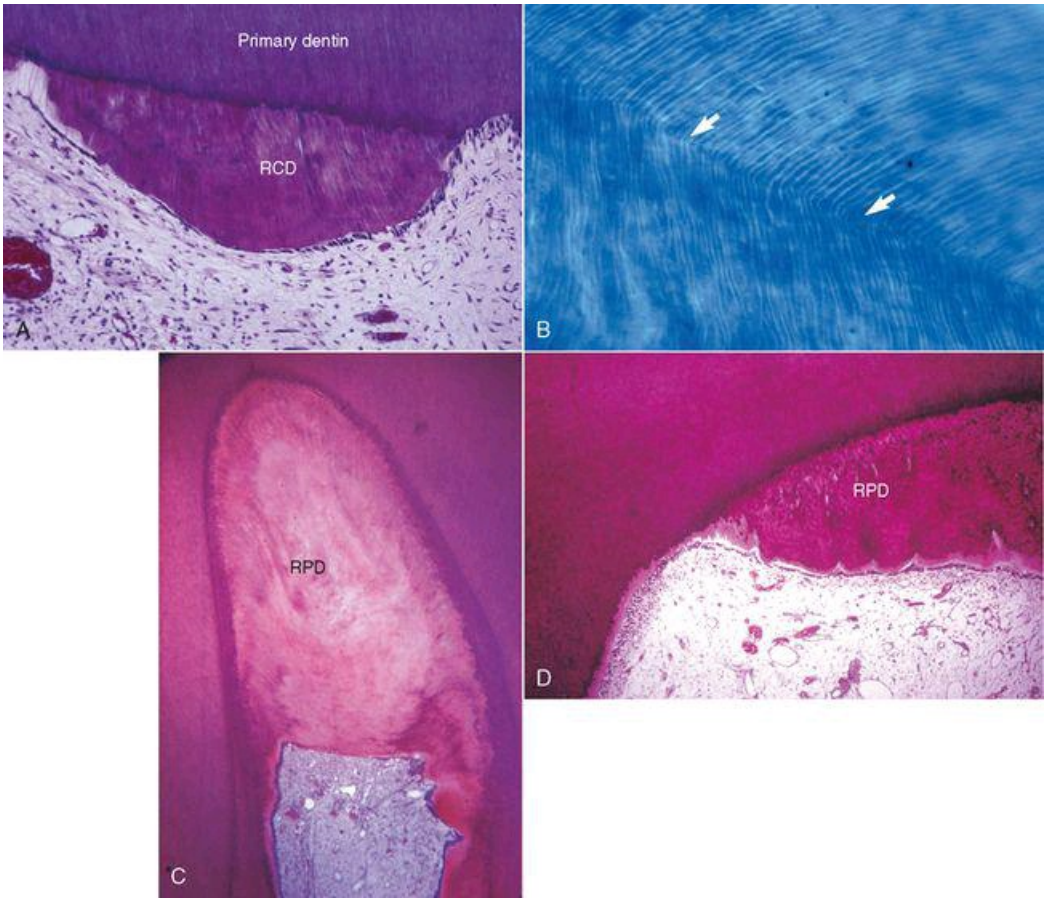


Figure 1-8 A, Reactionary dentin *(RCD)* at low power. **B,** RCD at high power showing change in direction of tubules *(arrows).* **C,** Reparative dentin *(RPD)* at low power. **D,** RPD at high power.

(Courtesy Dr. H. Trowbridge.)

Nutrition

The pulp supplies nutrients that are essential for dentin formation and for maintaining the integrity of the pulp itself.

Defense

In the mature tooth, the odontoblasts form dentin in response to injury, particularly when the original dentin thickness has been reduced by caries, attrition, trauma, or restorative procedures. Dentin can also be formed at sites where its continuity has been lost, such as at a site of pulp exposure. Dentin formation occurs in this situation through the induction, differentiation, and migration of new odontoblasts to the exposure site (Figure 1-9).

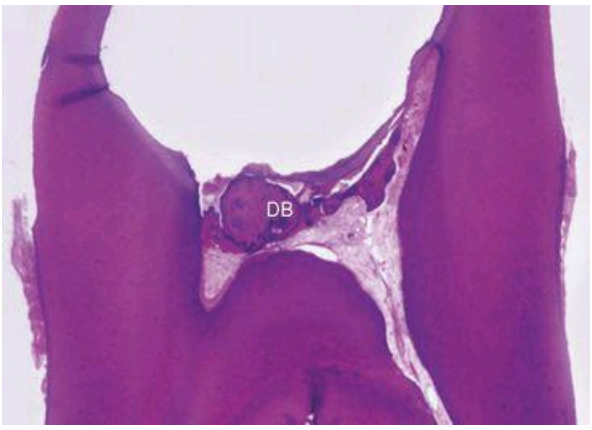


Figure 1-9 Reparative dentin bridge *(DB)* formed over a cariously exposed pulp.

(Courtesy Dr. H. Trowbridge.)

Pulp also has the ability to process and identify foreign substances, such as the toxins produced by bacteria of dental caries, and to elicit an immune response to their presence.

Sensation

Nerves in the pulp can respond to stimuli applied to the tissue itself directly, or reaching it through enamel and dentin. Physiological stimuli can only result in the sensation of pain. The stimulation of myelinated sensory nerves in the pulp results in fast, sharp pain. Activation of the non-myelinated pain fibers results in a duller, slower pain. Pulp sensation through dentin and enamel is usually fast and sharp and is transmitted by Aδ fibers *(myelinated fibers).*

MORPHOLOGY

Dentin and pulp are really a single-tissue complex whose histologic appearance varies with age and exposure to external stimuli.

Under light microscopy, a young, fully developed permanent tooth shows certain recognizable aspects of pulpal architecture. In its outer (peripheral) regions subjacent to predentin there is the odontoblast layer. Internal to this layer is a relatively cell-free area *(the zone of Weil).* Internal to the cell-free zone is a higher concentration of cells (cell-rich zone). In the center is an area containing mostly fibroblasts and major branches of nerves and blood vessels referred to as the *pulp core* (Figure 1-10).

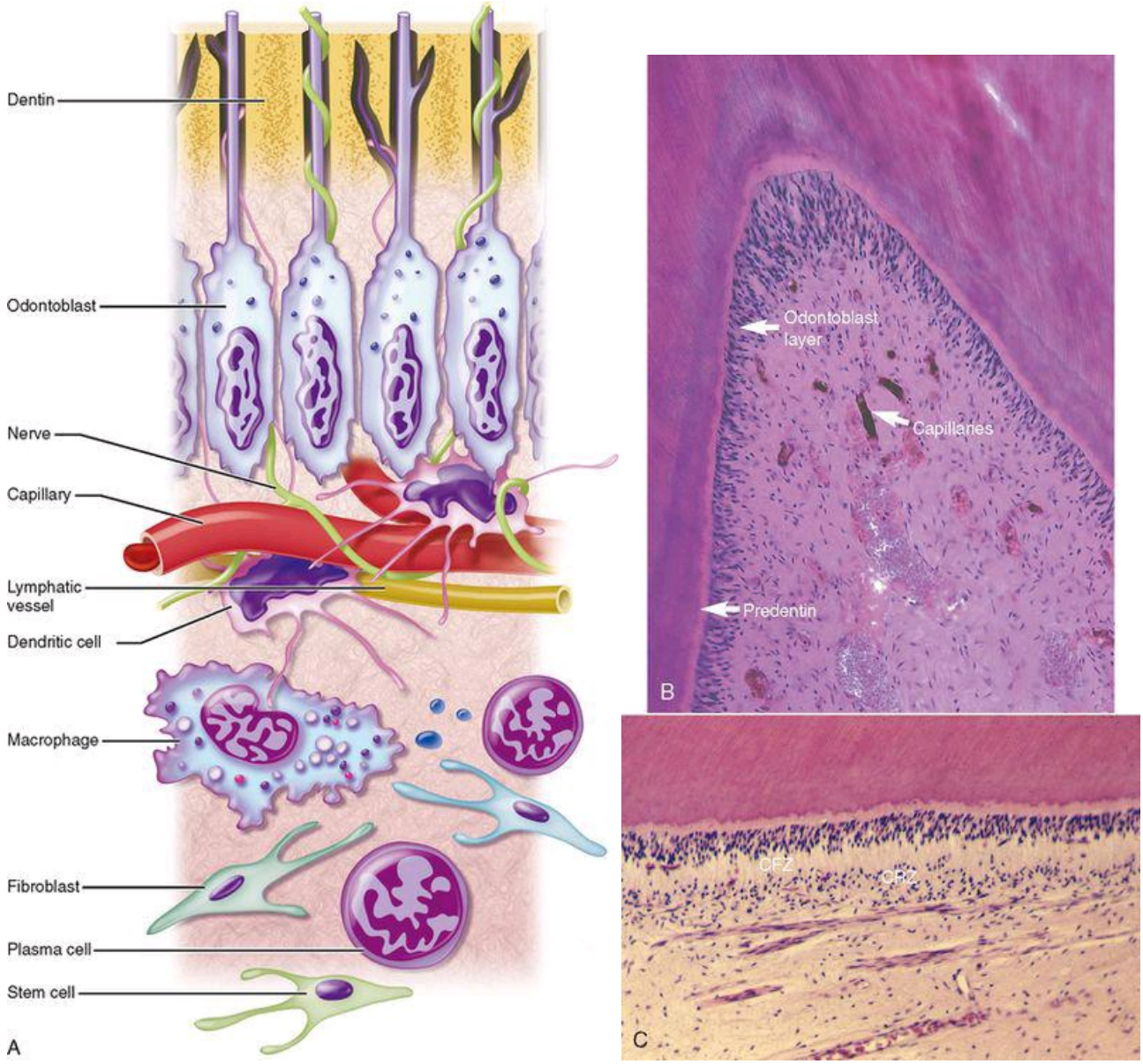


Figure 1-10 A, Diagram of the organization of the peripheral pulp. **B,** Peripheral pulp at low power. **C,** Peripheral pulp showing cell-free zone *(CFZ)* and cell-rich zone *(CRC).*

CELLS OF THE DENTAL PULP

Odontoblasts

Odontoblasts are the characteristic cells of pulp. They form a single layer at its periphery, synthesize the matrix, and control the mineralization of dentin.[11] In the coronal part of the pulp space the odontoblasts are numerous, relatively large, and columnar in shape. They number between 45,000 and 65,000/mm^2 in that area. In the cervical portion and midportion of the root, their numbers are fewer and they appear more flattened. The morphology of the cell reflects its level of activity; larger cells have a well-developed synthetic apparatus and the capacity to synthesize more matrix. Odontoblasts are end cells and as such do not undergo further cell division. During their life cycle, they go through functional, transitional, and resting phases, all marked by differences in cell size and organelle expression.[12] Odontoblasts can continue at varying levels of activity for a lifetime. Some do die by planned cell death (apoptosis)[13] as the volume of the pulp decreases. Disease processes, principally dental caries, can kill odontoblasts, but if conditions are favorable, these cells can be replaced by new odontoblasts that have differentiated from stem cells.

The odontoblast consists of two major components, the cell body and the cell process. The *cell body* lies subjacent to the unmineralized dentin matrix (predentin). The *cell process* extends outward for a variable distance through a tubule in the predentin and dentin. The cell body is the synthesizing portion of the cell and contains a basally located nucleus and an organelle structure in the cytoplasm that is typical of a secreting cell. During active dentinogenesis, the endoplasmic reticulum and the Golgi apparatus are prominent and there are numerous mitochondria and vesicles (Figure 1-11). Cell bodies are joined by a variety of membrane junctions, including gap junctions, tight junctions, and desmosomes. Each junction type has specific functions. Desmosomal junctions mechanically link the cells into a coherent layer. Gap junctions allow communication between cells in the layer. Tight junctions control the permeability of the layer. The secretory products of the odontoblasts are released through the cell membrane at the peripheral end of the cell body and through the cell process.

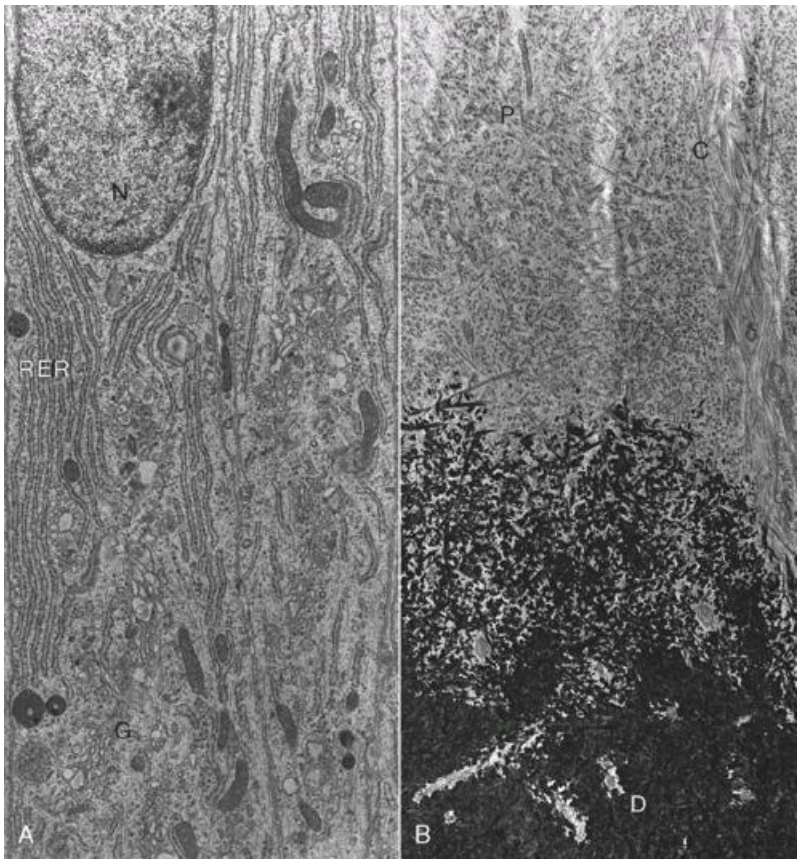


Figure 1-11 A, Odontoblast cell body. The nucleus *(N)* is proximal, and the numerous organelles, such as rough endoplasmic reticulum *(RER)* and Golgi apparatus *(G),* which are responsible for synthesis of matrix components, occupy the central-distal regions. **B,** Predentin *(P)* shows the orientation of collagen *(C)* to the odontoblastic process, which is the secretory organ that extends through the predentin into the dentin *(D).*

(Courtesy Dr. P. Glick and Dr. D. Rowe.)

Stem Cells (Preodontoblasts)

Newly differentiated odontoblasts develop after an injury that results in the death of existing odontoblasts. They develop from stem cells (also known as undifferentiated mesenchymal cells), which are present throughout the pulp, although they are densest in its core.[14] Under the influence of signaling molecules released in response to injury and cell death, these precursor cells migrate to the site of injury and differentiate into odontoblasts.[15] The key signaling molecules in this process are members of the bone morphogenetic protein (BMP) family and transforming growth factor β.

Embryonic stem cells can, with the appropriate signals, differentiate into any cell type. Stem cells in the adult are more restricted and are usually described as tissue-specific, meaning that they can only differentiate into the cell types found in the tissue from which they originate. This is the case with the pulpal stem cells.

Fibroblasts

Fibroblasts are the most common cell type in the pulp and are seen in greatest numbers in the coronal pulp. They produce and maintain the collagen and ground substance of the pulp and alter the structure of the pulp in disease. As with odontoblasts, the prominence of their cytoplasmic organelles changes according to their activity. The more active the cell, the more prominent the organelles and other components necessary for synthesis and secretion. Like odontoblasts, these cells undergo apoptotic cell death and are replaced when necessary by the maturation of less differentiated cells. Routine histologic preparations detect only prominent morphologic differences between cell types. Many of the cells usually recognized as “fibroblasts” may, in fact, be stem cells.

Cells of the Immune System

The most prominent immune cell in the dental pulp is the dendritic cell.[16] These are antigen-presenting cells present most densely in the odontoblast layer and around blood vessels. They recognize a wide range of foreign antigens and initiate the immune response. Many other cells (macrophages, neutrophils) have antigen-presenting properties, but dendritic cells in the pulp are, in terms of numbers (estimated at 8% of the pulp) and position, the most prominent in the pulp. Special stains are needed to recognize them histologically.

Macrophages in a resting form (histiocytes) and some T lymphocytes are also found in the normal pulp.[17]

EXTRACELLULAR COMPONENTS18

Fibers

The predominant collagen in dentin is type I, whereas both type I and type III collagens are found within pulp in a ratio of approximately 55: 45. Odontoblasts produce only type I collagen for incorporation into the dentin matrix, whereas fibroblasts produce both types I and III. Pulpal collagen is present as 50-nm-wide fibrils several microns long. They form bundles that are irregularly arranged, except in the periphery where they lie approximately parallel to the predentin surface. The only noncollagenous fibers present in the pulp are tiny, 10- to 15-nm–wide beaded fibrils of fibrillin, a large glycoprotein. Elastic fibers are absent from the pulp.

The proportion of collagen types is constant in the pulp, but with age there is an increase in the overall collagen content and an increase in the organization of collagen fibers into collagen bundles. Normally, the apical portion of pulp contains more collagen than the coronal pulp, facilitating pulpectomy with a barbed broach or endodontic file during root canal treatment.

Noncollagenous Matrix[19]

The collagenous fibers of the pulp matrix are embedded in a histologically clear gel made up of glycosaminoglycans and other adhesion molecules. The glycosaminoglycans link to protein and other saccharides to form proteoglycans, a very diverse group of molecules. They are bulky hydrophilic molecules that with water make up the gel. At least six types of adhesion molecules have been detected in the pulp matrix. One of these, fibronectin, is responsible for cell adhesion to the matrix.

Calcifications

Pulp stones or denticles (Figure 1-12) were once classified as true or false depending on the presence or absence of a tubular structure. However, this classification has been challenged, and a new nomenclature based on the genesis of the calcification has been suggested. Pulp stones have also been classified according to location. Three types of pulp stones have been described: *free stones,* which are surrounded by pulp tissue; *attached stones,* which are continuous with the dentin; and *embedded stones,* which are surrounded entirely by dentin, mostly of the tertiary type.

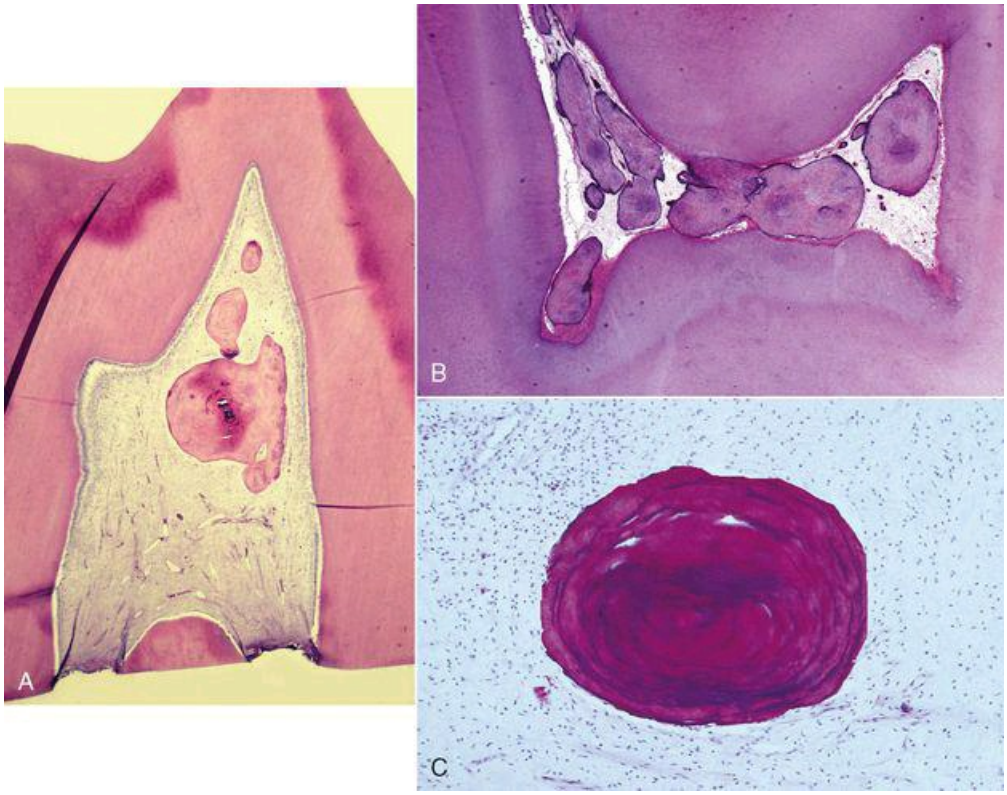


Figure 1-12 A, Multiple stones in coronal pulp. **B,** Stones occluding a pulp chamber. **C,** Lamellated pulp stone.

(Courtesy Dr. H. Trowbridge.)

Pulp stones occur in both young and old patients and may occur in one or several teeth. A recent radiographic (bitewing) survey of undergraduate dental students found that 46% of them had one or more pulp stones; 10% of all the teeth contained a pulp stone. They occur in normal pulps, as well as in chronically inflamed pulps. They are not responsible for painful symptoms, regardless of size.

Calcifications may also occur in the form of diffuse or linear deposits associated with neurovascular bundles in the pulp core. This type of calcification is seen most often in the aged, chronically inflamed, or traumatized pulp. Depending on shape, size, and location, pulp calcifications may or may not be detected on a dental radiograph (Figure 1-13). *Large pulp stones are clinically significant in that they may block access to canals or the root apex during root canal treatment.*

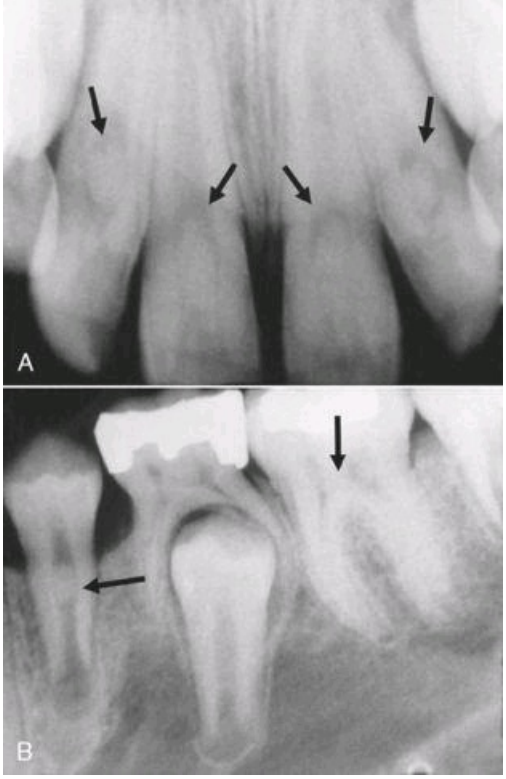


Figure 1-13 Multiple pulp stones *(arrows)* in the pulp chamber and root canals of the anterior **(A)** and posterior **(B)** teeth of a young patient.

BLOOD VESSELS

Mature pulp has an extensive[20] and specialized vascular pattern that reflects its unique environment. The vessel network has been examined using a variety of techniques, including India ink perfusion, transmission electron microscopy, scanning electron microscopy, and microradiography.

Afferent Blood Vessels (Arterioles)

The largest vessels to enter the apical foramen are arterioles that are branches of the inferior alveolar artery, the superior posterior alveolar artery, or the infraorbital artery.

Once inside the canal the arterioles travel toward the crown. They narrow, then branch extensively and lose their muscle sheath before forming a capillary bed (Figure 1-14). The muscle fibers before the capillary bed form the "precapillary sphincters," which control blood flow and pressure. The most extensive capillary branching occurs in the subodontoblastic layer[21] of the coronal pulp, where the vessels form a dense plexus (Figure 1-15). The loops of some of these capillaries extend between odontoblasts.[22] The exchange of nutrients and waste products takes place in the capillaries (Figure 1-16).[23] There is an extensive shunting system composed of arteriovenous and venovenous anastomoses; these shunts become active after pulp injury and during repair.

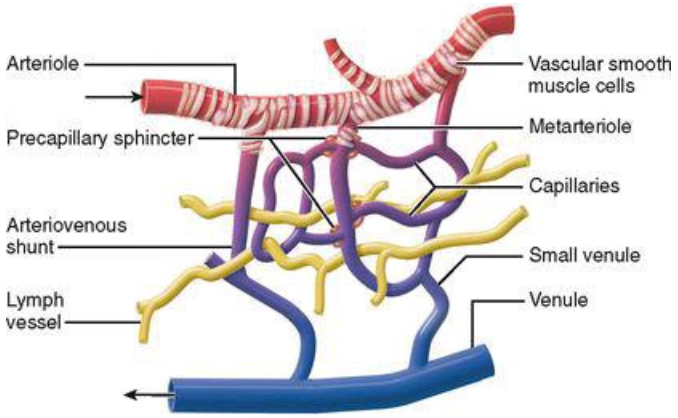


Figure 1-14 Schematic of the pulpal vasculature. Smooth muscle cells that surround vessels and precapillary sphincters selectively control blood flow. Arteriovenous shunts bypass capillary beds.

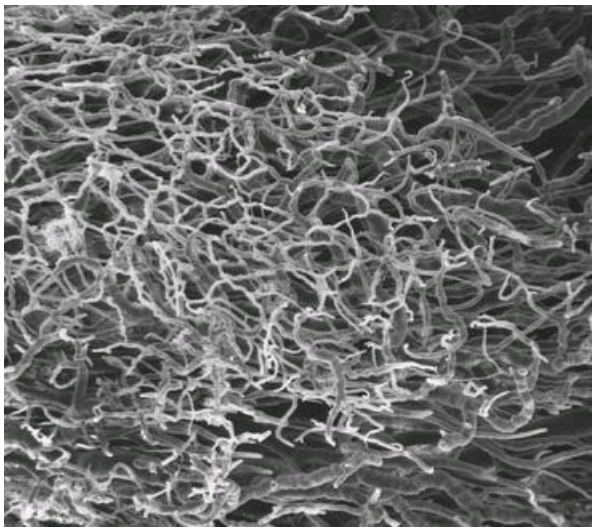


Figure 1-15 The dense capillary bed in the subodontoblastic region is shown by resin cast preparation and scanning electron microscopy.

(Courtesy Dr. C. Kockapan.)

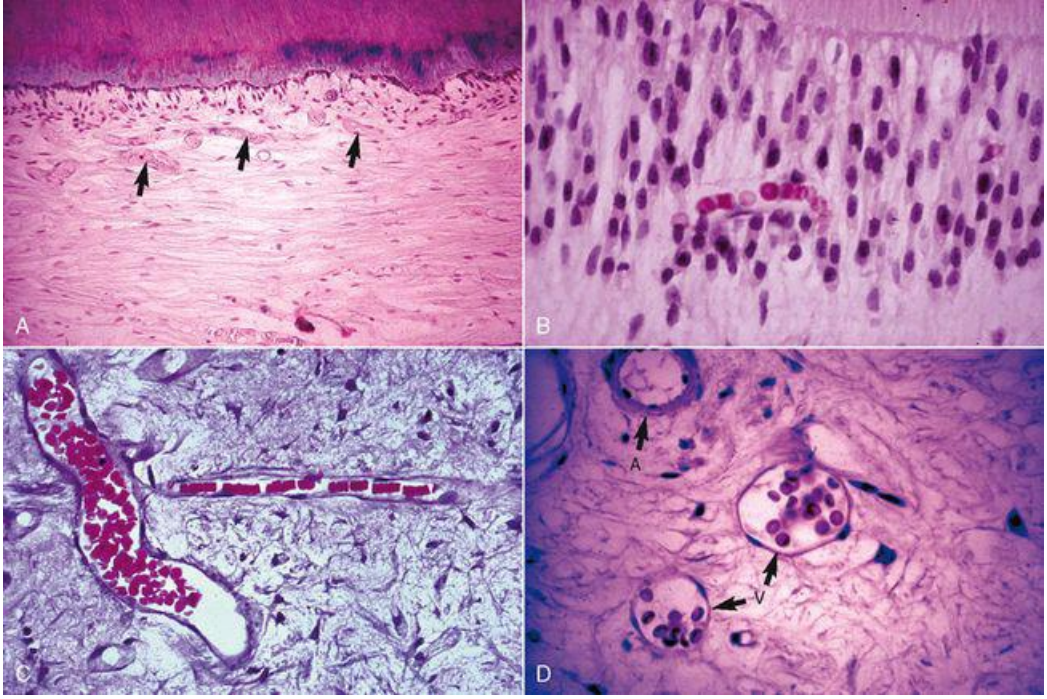


Figure 1-16 A, Subodontoblastic capillary plexus. **B,** Capillary within the odontoblast layer. **C,** Branching capillaries in subodontoblastic plexus. **D,** Arteriole *(A)* and venules *(V)* in the peripheral pulp.

(Courtesy Dr. H. Trowbridge.)

Efferent Blood Vessels

Venules constitute the efferent (exit) side of the pulpal circulation and are slightly larger than the corresponding arterioles. Venules are formed from the junction of venous capillaries and enlarge as more capillary branches unite with them.[24] They run with the arterioles and exit at the apical foramen to drain posteriorly into the maxillary vein through the pterygoid plexus or anteriorly into the facial vein.

Lymphatics

Lymphatic vessels arise as small, blind, thin-walled vessels in the periphery of the pulp.[28] They pass through the pulp to exit as one or two larger vessels through the apical foramen (Figures 1-17 and 1-18). The lymphatic vessel walls are composed of an endothelium rich in organelles and granules. There are discontinuities in the walls of these vessels and also discontinuities in their basement membranes. This porosity permits the passage of interstitial tissue fluid and, when necessary, lymphocytes into the negative-pressure lymph vessel. The lymphatics assist in the removal of inflammatory exudates and transudates, as well as cellular debris. After exiting from the pulp, some vessels join similar vessels from the PDL[29] and drain into regional lymph glands (submental, submandibular, or cervical) before emptying into the subclavian and internal jugular veins. *An understanding of lymphatic drainage assists in the diagnosis of infection of endodontic origin.*

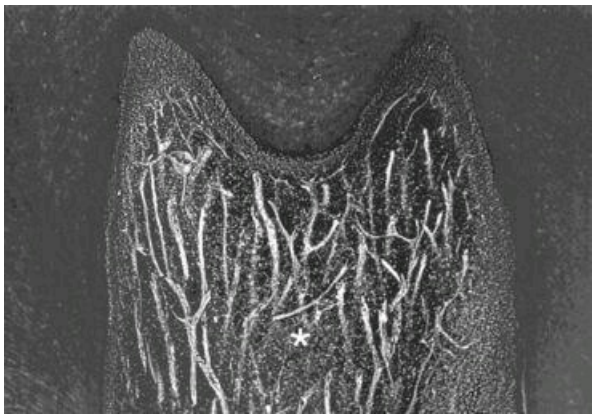


Figure 1-17 Distribution of lymphatics. Scanning electron micrograph of secondary and back-scattered electrons after specific immune staining.

(From Matsumoto Y, Zhang B, Kato S: Microsc Res Tech 56:50, 2002.)

Figure 1-18 Transmission electron micrograph of a lymphatic vessel *(L)* in the peripheral pulp.

(From Matsumoto Y, Zhang B, Kato S: Microsc Res Tech 56:50, 2002.)

Vascular Physiology

The dental pulp, at least when young, is a highly vascular tissue. Capillary blood flow in the coronal region is almost twice that of the radicular region. Blood supply is regulated largely by the precapillary sphincters and their sympathetic innervation.[25] Other local factors and peptides released from sensory nerves also affect the vessels but most prominently during inflammation.[26]

As in other tissues, the volume of the vascular bed is much greater than the volume of blood that is normally passing through it. Only part of the vascular bed is perfused at any one time. This capacity allows for sizeable local increases in blood flow in response to injury.

The factors that determine what passes in and out between the blood and the tissue include concentration gradients, osmosis, and hydraulic pressure. Concentration gradients vary along the capillary bed as oxygen, for example, diffuses out into the depleted tissue and CO_2 enters from high to low concentration. The hydraulic pressure in the pulpal capillaries falls from 35 mm Hg at the arteriolar end to 19 mm Hg at the venular end. Outside the vessel, the interstitial fluid pressure varies, but a normal figure would be 6 mm Hg.[27]

Vascular Changes During Inflammation

When the dental pulp is injured, it responds in the same way as other connective tissues with a two-phase immune response. The initial immune response is nonspecific but rapid, occurring in minutes or hours. The second response is specific and includes the production of specific antibodies. Before the detailed nature of the immune response was known, the phenomena associated with the response to tissue injury, including redness, pain, heat, and swelling, was known as *inflammation.* Although much more is now known about the response to injury at the cellular level, these "cardinal signs" remain important. Except for pain, they are all vascular in origin. Heat and redness are results of increased blood flow, and swelling results from increased formation of interstitial tissue fluid because of increased permeability of the capillaries. In other tissues, such as skin (in which inflammation was first described), the increased production of tissue fluid results in swelling. Because the dental pulp is within a rigid, noncompliant chamber, it cannot swell, and the increased interstitial fluid formation results in an increase in tissue fluid pressure. At one time, it was thought that this rise in interstitial fluid pressure would spread rapidly and strangle vessels entering the root canal at the apical foramen. Closer study has revealed that this is incorrect. Elevations in tissue fluid pressure remain localized to the injured area. A short distance from the injury, tissue fluid pressure is maintained within normal limits. As interstitial fluid pressure rises, the intraluminal (inside) pressure of the local capillaries increases to balance this, so that the vessels remain patent. During the response to injury, the gradients by which nutrients and wastes leave and enter the capillaries change to allow greater exchange. At the same time these changes occur in the capillaries, lymphatic vessels become more heavily employed, removing excess tissue fluid and debris. In addition, anastomoses in the microvascular bed allow blood to be shunted around an area of injury, so that the oxygenation and nutrition of nearby uninjured tissue are not compromised. If the cause of the injury is removed, these processes will gradually return the vasculature to normal, and repair or regeneration can take place. If the injury persists and increases in size, this tissue will necrose. This necrosis can remain localized as a pulpal abscess, although it more often spreads throughout the pulp.

The vascular changes seen in inflammation are largely mediated by local nerves. The sympathetic fibers through the precapillary sphincters can alter the pressure, flow, and distribution of blood. Sensory nerve fibers release a number of neuropeptides but most prominently calcitonin gene–related peptide (CGRP) and substance P. (These names are of historic origin and unrelated to the function of these molecules in this setting.) The release of these neuropeptides comes about through axon reflexes, whereby one branch of a sensory nerve stimulated by the injury causes the release of the peptides by another branch. This mechanism, in which excitation of sensory elements results in increased blood flow and increased capillary permeability, is known as *neurogenic inflammation.*

INNERVATION

The second and third divisions of the trigeminal nerve (V^2 and V^3) provide the principal sensory innervation to the pulp of maxillary and mandibular teeth, respectively. Mandibular premolars also can receive sensory branches from the mylohyoid nerve of V^3, which is principally a motor nerve. Branches from this nerve reach the teeth via small foramina on the lingual aspect of the mandible. Mandibular molars occasionally receive sensory innervation from the second and third cervical spinal nerves (C2 and C3). This can create difficulties in anesthetizing these teeth with an inferior dental block injection only.

Cell bodies of trigeminal nerves are located in the trigeminal ganglion. Dendrites from these nerves synapse with neurons in the trigeminal sensory nucleus in the brainstem. Second-order neurons here travel to specific nuclei in the thalamus. Third-order neurons and their branches reach the sensory cortex, as well as a number of other higher centers.

Pulp also receives sympathetic (motor) innervation from T1 and to some extent C8 and T2 via the superior cervical ganglion. These nerves enter the pulp space alongside the main pulp blood vessels and are distributed with them. They maintain the vasomotor tone in the precapillary sphincters, which control the pressure and distribution of blood. The presence of parasympathetic nerve fibers in the pulp has been controversial. The current consensus is that there is no parasympathetic innervation of the pulp. This is not unusual. All tissues have an autonomic innervation but not always from both divisions.

Pulpal and Dentinal Nerves

Sensory nerves supplying the dental pulp contain both myelinated and unmyelinated axons (Figure 1-19). The myelinated axons are almost all narrow, slow-conducting Aδ axons (diameter 1 to 6 μm) associated with nociception. A small percentage of the myelinated axons (1% to 5%) are faster-conducting Aβ axons (diameter 6 to 12 μm). In other tissues, these larger fibers can be proprioceptive or mechanoreceptive. Their role in the pulp is uncertain, but it is now known from other tissues that in inflammation, these Aβ can be recruited to the pain system. Before they terminate, all the myelinated axons lose their myelin sheath and terminate as small, unmyelinated branches either below the odontoblasts, around the odontoblasts, or alongside the odontoblast process in the dentinal tubule (Figure 1-20).[30] Beneath the odontoblast layer, these terminating fibers form the subodontoblastic plexus of Raschkow (Figure 1-21).

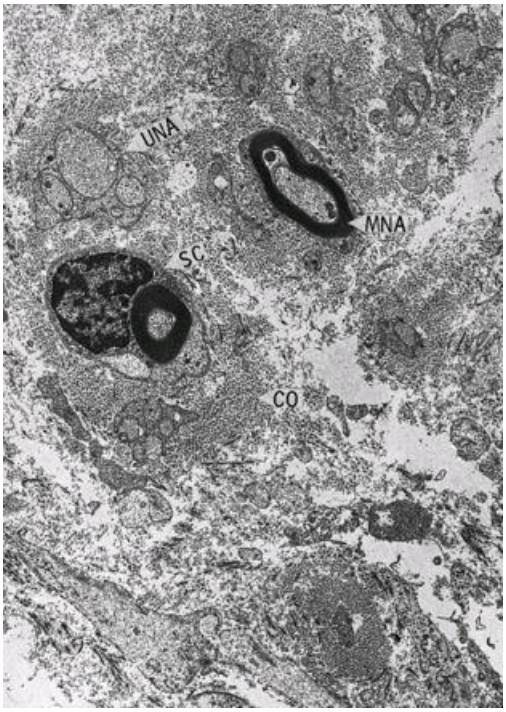


Figure 1-19 Pulp nerves in region of the pulp core. A group of unmyelinated *(UNA)* and myelinated *(MNA)* nerve axons are shown in cross-section. A Schwann cell *(SC)* associated with one of the myelinated axons is evident. Nerves are surrounded by collagen fibers *(CO).*

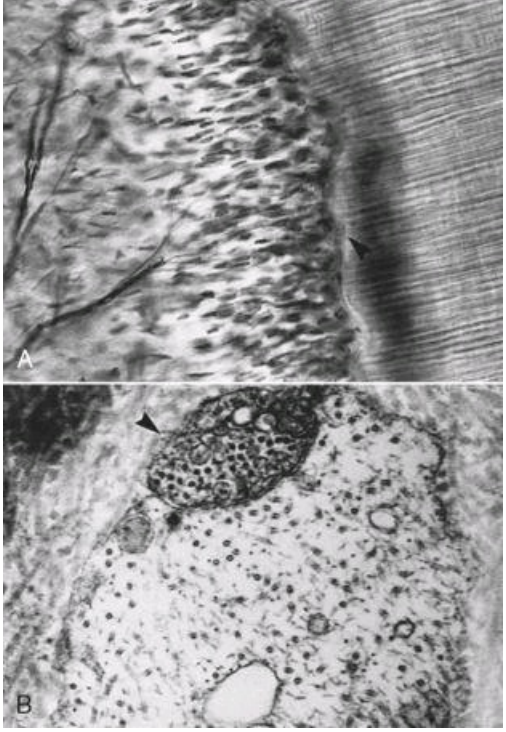


Figure 1-20 A, Silver-stained section of pulp in a young human molar demonstrates arborization of nerves in the subodontoblastic region and a nerve *(arrow)* passing between odontoblasts into the predentin area. **B,** Transmission electron micrograph demonstrates an unmyelinated nerve axon *(arrow)* alongside the odontoblast process in the dentin tubule at the level of the predentin.

(Courtesy Dr. S. Bemick.)

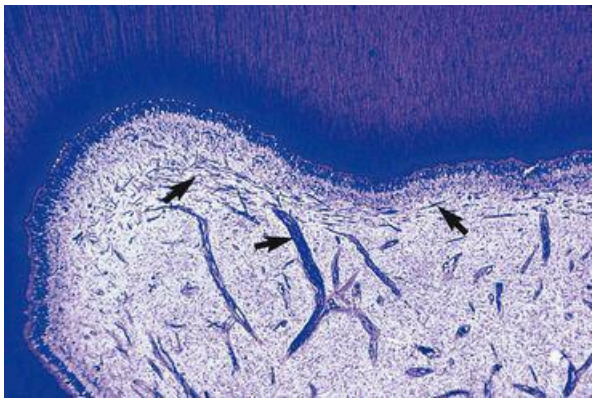


Figure 1-21 Rashkow's subodontoblastic neural *(arrows)* stained with silver.

The nerves that enter the dentinal tubules do not synapse with the process but remain in close proximity with it for part of its length. Approximately 27% of the tubules in the area of the pulp horn of a young, mature tooth contain an intratubular nerve. These nerves occur less often in the middle (11%) and cervical portions (8%) of the crown and not at all in the root.[31] Their incidence is higher in predentin than in mineralized dentin.

Developmental Aspects of Pulp Innervation

The types and relative number of nerves depend on the state of tooth maturity. Myelinated nerves enter the pulp at about the same time as unmyelinated nerves, but in most instances do not form the subodontoblastic plexus of Raschkow until some time after tooth eruption. As a result, there are significant variations in the responses of partially developed teeth to pulp vitality tests. *This undermines the value of stimulatory tests for determining pulp status in young patients, particularly after trauma.*

The number of pulpal nerves diminishes with age. *The significance of this reduction in terms of responses to vitality testing is undetermined.*

Pathways of Transmission from Pulp to Central Nervous System

Mechanical, thermal, and chemical stimuli initiate an impulse that travels along the pulpal axons in the maxillary (V^2) or mandibular (V^3) branches of the trigeminal nerve to the trigeminal (Gasserian) ganglion, which contains the cell body of the neuron. Dendrites from the ganglion then pass centrally and synapse with second-order neurons in the trigeminal nuclear complex located at the base of the medulla and the upper end of the spinal cord. Most of the activity that originates in the dental pulp is conducted along axons that synapse with neurons in the spinal portion of the complex, most notably the subnucleus caudalis.

Many peripheral axons from different sites synapse on a single secondary neuron, a phenomenon known as *convergence.* Activity in a single synapse does not result in excitation of the second-order neuron. Activity in many synapses must summate to reach the threshold of the second-order neuron. The activation of the second-order neuron is also affected by fibers from the midbrain that belong to the endogenous opioid system. These, when active, reduce the activity of the second-order neurons. Thus noxious input is modulated, explaining why the pain experience is not always closely related to the degree of peripheral noxious stimulation. Axons from the second-order neurons cross the midline and synapse in thalamic nuclei. From here, third-order neurons pass information to a variety of higher centers, the sensory cortex being only one of them. The broad distribution of noxious input centrally and the presence of a pain-modulating system descending from higher centers provide the broad framework for understanding and controlling pain. As a result of persistent noxious input, the properties of second-order neurons can change. These changes can be used to explain some of the complexities of diagnosing and treating pain as described in other sections of this text.

Theories of Dentin Hypersensitivity

Pain elicited by scraping or cutting of dentin or by the application of cold or hypertonic solutions to exposed dentin gives the impression that there may be a nerve pathway from the central nervous system to the dentinoenamel junction (DEJ). However, no direct pathway is present. The application of pain-producing substances, such as histamine, acetylcholine, or potassium chloride, to exposed dentin surface fails to produce pain. Eliciting pain from exposed dentin by heat or cold is not blocked by local anesthetics.

At one time it was thought that dentin sensitivity was due to sensory nerves within the dentinal tubules. Currently two explanations for peripheral dentin sensitivity have broad acceptance (Figure 1-22). One is that stimuli that are effective in eliciting pain from dentin cause fluid flow through the dentinal tubules; this disturbance results in the activation of nociceptors in the inner dentin and peripheral pulp.[32] Several observations support this "hydrodynamic hypothesis." In experiments on extracted teeth, it has been shown that hot, cold, and osmotic stimuli do cause fluid flow through dentin. In human subjects, the success of solutions in inducing pain is related to the osmotic pressure of the solution. Exposed dentin that is sensitive in patients has patent dentinal tubules.[33] In exposed dentin that is not sensitive, the dentinal tubules are occluded. Substances and techniques that occlude dentinal tubules in sensitive dentin eliminate or reduce the sensitivity. A second explanation is that some substances can diffuse through the dentin and act directly on pulpal nerves. Evidence for this largely comes from animal experiments, which show that the activation of pulpal nerves is sometimes related to the chemical composition of a stimulating solution rather than its osmotic pressure. These are not mutually exclusive hypotheses. Both may occur and both should be addressed in treating sensitive dentin.

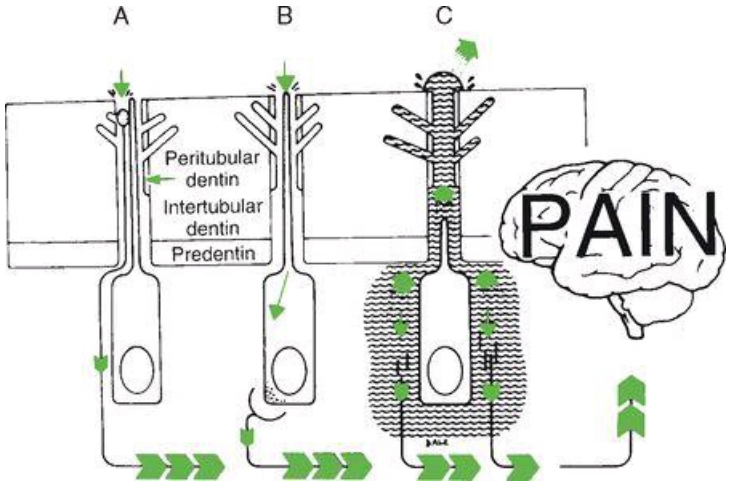


Figure 1-22 Schematic drawing of theoretic mechanisms of dentin sensitivity. **A,** Classic theory (direct stimulation of nerve fibers in the dentin). **B,** Odontoblasts as a mediator between the stimuli and the nerve fibers. **C,** Fluid movement as proposed in hydrodynamic theory.

(Modified from Torneck CD: Dentin-pulp complex. In Ten Cate AR, editor, Oral histology, ed 4, St. Louis, 1994, Mosby.)

AGE CHANGES IN THE DENTAL PULP AND DENTIN

Secondary dentin is laid down throughout life; as a result, both the pulp chamber and root canals become smaller, sometimes to the point where they are no longer visible on radiographs. More peritubular dentin is laid down, often completely occluding the dentinal tubules in the periphery (sclerotic dentin). As a result of these processes the permeability of the dentin is reduced. The pulp tissue itself becomes less cellular and less vascular and contains fewer nerve fibers. Between the ages of 20 and 70, cell density decreases by approximately 50%. This reduction affects all cells, from the highly differentiated odontoblast to the undifferentiated stem cell.

PERIRADICULAR TISSUES

The periodontium, the tissue surrounding and investing the root of the tooth, consists of the cementum, PDL, and alveolar bone (Figure 1-23). These tissues originate from the dental follicle that surrounds the enamel organ; their formation is initiated when root development begins. After the tooth has erupted, the cervical portion of the tooth is in contact with the epithelium of the gingiva, which in combination with reduced dental epithelium on the enamel forms the *dentogingival junction.* When intact, this junction protects the underlying periodontium from potential irritants in the oral cavity.

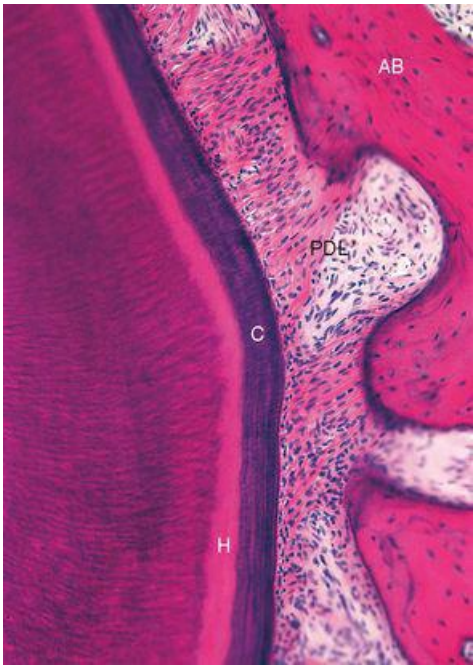


Figure 1-23 Peripheral radicular dentin (*H,* hyaline layer), cementum *(C),* periodontal ligament *(PDL),* and alveolar bone *(AB).*

The pulp and the periodontium form a continuum at sites along the root where blood vessels enter and exit the pulp at the apical foramen and lateral and accessory canals (Figure 1-24).

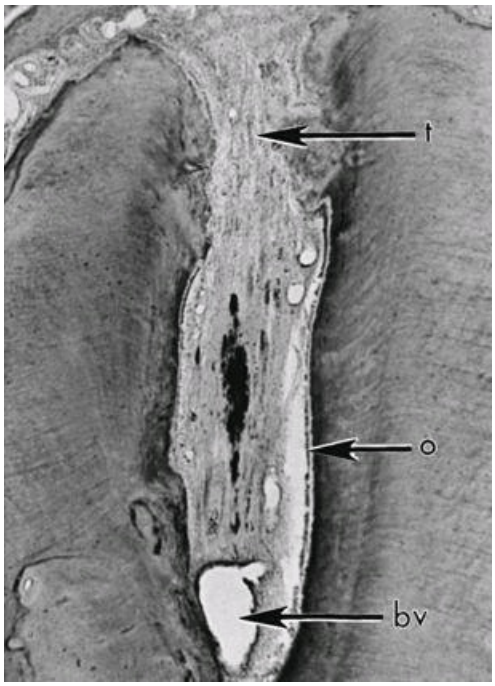


Figure 1-24 Apical region of maxillary incisor showing apical foramen. *t,* Transitional tissue between periodontal ligament and pulp; *o,* odontoblasts; *bv,* blood vessel.

Cementum

Cementum is a bonelike tissue that covers the root and provides attachment for the principal periodontal fibers. The several types of cementum that have been identified are as follows:

1. *Primary acellular intrinsic fiber cementum*. This is the first cementum formed, and it is present before principal periodontal fibers are fully formed. It extends from the cervical margin to the cervical third of the tooth in some teeth and around the entire root in others (incisors and cuspids). It is more mineralized on the surface than near the dentin and contains collagen produced initially by cementoblasts and later by the fibroblasts.

2. *Primary acellular extrinsic fiber cementum*. This is cementum that continues to be formed about the primary periodontal fibers after they have been incorporated into primary acellular intrinsic fiber cementum.

3. *Secondary cellular intrinsic fiber cementum.* This cementum is bonelike in appearance and only plays a minor role in fiber attachment. It occurs most often in the apical part of the root of premolars and molars.

4. *Secondary cellular mixed fiber cementum.* This is an adaptive type of cellular cementum that incorporates periodontal fibers as they continue to develop. It is variable in its distribution and extent and can be recognized by the inclusion of cementocytes, its laminated appearance, and the presence of cementoid on its surface.

5. *Acellular afibrillar cementum.* This is the cementum sometimes seen overlapping enamel, which plays no role in fiber attachment.

Cementum is similar to bone but harder and thus resists resorption during tooth movement. The junction between the cementum and the dentin (CDJ) that forms the apical constriction is ill defined and not uniform throughout its circumference. Biologic principles suggest that the most appropriate point to end a root canal preparation is at the junction of the pulp and periodontium, which occurs at the apical constriction. *Although many practitioners debate the probabilities and practicalities of achieving this goal, most agree that it is essential to measure canal length accurately and to restrict all procedures to a canal length that estimates this point as closely as possible.*

Although dentin is harder than bone and resorbs more slowly, it does resorb in periapical inflammatory lesions, often resulting in loss of the apical constriction. Occasionally, more rapid resorption of unknown cause is seen (idiopathic resorption) but is often self limiting.

Cementoenamel Junction

The junction of cementum and enamel at the cervix of the tooth varies in its arrangement even around a single tooth. Sometimes cementum overlies enamel and vice versa. When there is a gap between the cementum and the enamel, the exposed dentin may be sensitive.

Periodontal Ligament

PDL, like dental pulp, is a specialized connective tissue.[34] Its function relates in part to the presence of specially arranged bundles of collagen fibers that support the tooth in the socket and absorb the forces of occlusion from being transmitted to the surrounding bone. The PDL space is small, varying from an average of 0.21 mm in young teeth to 0.15 mm in older teeth. The uniformity of its width (as seen in a radiograph) is one of the criteria used to determine its health.

Lining the periodontal space are cementoblasts and osteoblasts. Interwoven between the principal periodontal fibers is a loose connective tissue that contains fibroblasts, stem cells, macrophages, osteoclasts, blood vessels, nerves, and lymphatics. Epithelial cell rests of Malassez are also present (Figure 1-25). As already noted, these cells are of no known significance in the healthy periodontium but can, during inflammatory states, proliferate and give rise to cyst formation.

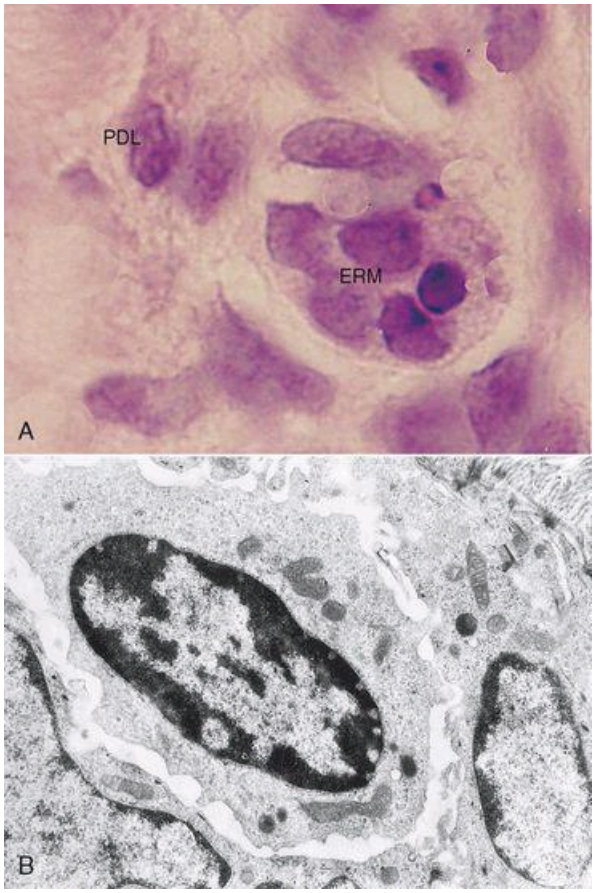


Figure 1-25 A, Epithelial rest of Malassez *(ERM)* in periodontal ligament *(PDL).* **B,** Transmission electron micrograph of epithelial rests.

(From Cerri PS, Katchburian E: J Perio Res 40:365, 2005.)

The vasculature of the periodontium is extensive and complex. Arterioles that supply the PDL arise from the superior and inferior alveolar branches of the maxillary artery in the cancellous bone. These arterioles pass through small openings in the alveolar bone of the socket, at times accompanied by nerves, and extend upward and downward throughout the periodontal space. They are more prevalent in posterior than anterior teeth. Other vessels arise from the gingiva or from dental vessels that supply the pulp; these latter vessels branch and extend upward into the periodontal space before the pulpal vessels pass through the apical foramen. The degree of collateral blood supply to the PDL and the depth of its cell resources impart an excellent potential for its repair subsequent to injury, a potential that is retained for life in the absence of systemic or prolonged local disease.

The periodontium receives both an autonomic and a sensory innervation. Autonomic nerves are sympathetics arising from the superior cervical ganglion and terminating in the smooth muscle of the periodontal arterioles. Activation of the sympathetic fibers induces constriction of the vessels. As in the pulp, there is no convincing evidence that a parasympathetic nerve supply exists.

Sensory nerves that supply the periodontium arise from the second and third divisions of the trigeminal nerve (V^2 and V^3). They are mixed nerves of large and small diameter. Unmyelinated sensory fibers terminate as nociceptive free endings. Large fibers are mechanoreceptors and terminate in special endings throughout the ligament, but are in greatest concentration in the apical third of the periodontal space. These are highly sensitive and record pressures in the ligament associated with tooth movement. They allow patients to identify teeth with acute periodontitis with some precision.

Alveolar Bone

The bone of the jaws that supports the teeth is referred to as the *alveolar process.* Bone that lines the socket and into which the principal periodontal fibers are anchored is referred to as *alveolar bone proper* (bundle bone, cribriform plate). Alveolar bone is perforated to accommodate vessels, nerves, and investing connective tissues that pass from the cancellous portion of the alveolar process to the periodontal space. Despite these perforations, alveolar bone proper is denser than the surrounding cancellous bone and has a distinct opaque appearance when seen in periapical radiographs. On the radiograph, alveolar bone proper is referred to as lamina dura (Figure 1-26). Its continuity is equated with periodontal health and its perforation with disease. Radiographic changes associated with periradicular inflammatory disease usually follow rather than accompany the disease. Significant bone loss is necessary before a radiographic image is seen.

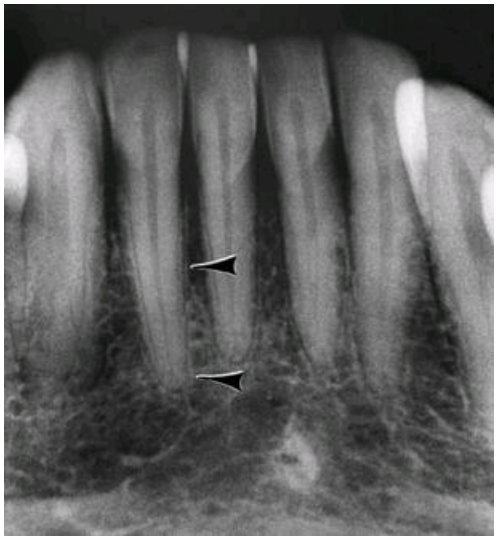


Figure 1-26 Mandibular anterior teeth with normal, uniform periodontal ligament space and identifiable lamina dura *(arrows).* This usually but not always indicates the absence of periradicular inflammation.

Alveolar bone proper is principally lamellar and continually adapts to the stress of tooth movements. Because pressures are not constant, bone is constantly remodeling (by resorption and apposition).

Chapter Review Questions available in Appendix B or on the DVD

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Protecting the Pulp, Preserving the Apex

Graham Rex Holland, Henry O. Trowbridge, Mary Rafter

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe pulp protection and pulp therapy.
2. Understand the special physiologic and structural characteristics of the pulp-dentin complex and how they affect the pulpal response to injury.
3. Describe the reparative mechanisms of the pulp, including immune responses and tertiary dentin formation.
4. Describe the effect of dental procedures and materials on the pulp.
5. Appreciate the significance of microleakage and smear layer on pulp response.
6. Describe the indications for and procedures for vital pulp therapy.
7. Discuss the effects of pulpal injury in teeth with developing roots.
8. Describe diagnosis and case assessment of immature teeth with pulp injury.
9. Describe the techniques for vital pulp therapy (apexogenesis) and root-end closure (apexification).
10. Describe the prognosis for vital pulp therapy and root-end closure.
11. Consider restoration of the treated immature tooth.
12. Recognize the potential of tissue engineering techniques in regenerating pulpal tissue.

DEFINITIONS

Pulp Protection

The principal threat to the health of the dental pulp is dental caries. The second most significant threat is the treatment of dental caries. Heat generation and desiccation during cavity preparation, the toxicity of restorative materials, and, most significantly, the leakage of bacteria and their products at the margins of restorations can cause damage that is added to that caused by the original caries. This damage can convert a reversible pulpitis into an irreversible pulpitis. In consideration of this, operative dentistry can be considered “preventive” or “interceptive” endodontics. Restorative procedures should be designed not only to restore the mechanical integrity and appearance of the tooth but also to avoid further harm, allow a compromised pulp to recover, and protect the pulp from further damage.

A key element in pulp protection is the recognition that the pulp is always inflamed when dental caries is present. Even in teeth in which there are white spot lesions and where restorative procedures are not indicated, pulpal inflammation is frequently present (Figure 2-1).[1] In designing treatment plans where several teeth have carious lesions and especially when lesions are extensive, a “triage” approach is preferred in which active caries is removed and good temporary restorations are placed at an early stage, allowing the pulp the maximum opportunity for recovery.

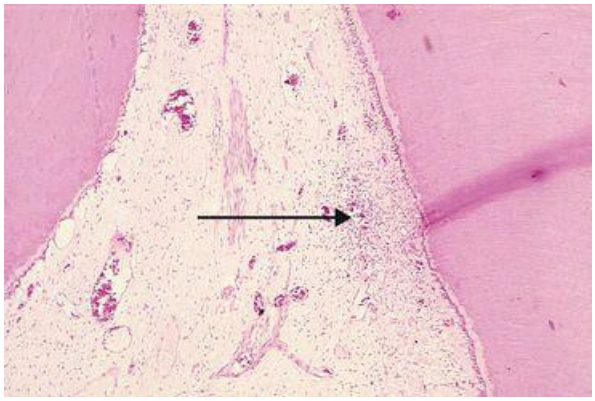

Figure 2-1 Pulpal inflammation *(arrow)* at the base of the tubules beneath a white spot lesion in enamel.

Pulp Therapy

When the dental pulp is mechanically exposed by trauma or during cavity preparation, it may by appropriate treatment be possible to maintain pulp vitality and avoid root canal treatment. The exposed pulp may be protected immediately by covering it (pulp capping) and placing a restoration. If the exposure is large or seriously contaminated, it may be possible to remove the diseased part of the pulp (pulpotomy), cap the remaining pulp, and place a restoration. This procedure is of greatest value when the root has not reached its full length (apexogenesis). In teeth with necrotic pulps and incomplete root formation, apical closure (but not root elongation) can be obtained by apexification. Future tissue engineering techniques may allow replacement of part or all of the pulp with new tissue. Table 2-1 defines the principal terms used in pulpal protection and vital pulp therapy.[2]

Table 2-1 Definitions of the Principal Terms Used in Pulpal Protection and Vital Pulp Therapy

Term	Definition
Pulp cap	Treatment of an exposed vital pulp in which the pulpal wound is sealed with a dental material, such as calcium hydroxide or MTA, to facilitate the formation of reparative dentin and maintenance of a vital pulp.
Direct pulp cap	A dental material placed directly on a mechanical or traumatic vital pulp exposure.
Step-wise caries excavation	A material is placed on a thin partition of remaining carious dentin that if removed might accidentally expose the pulp (for immature permanent teeth).
Pulpectomy (pulp extirpation)	The complete surgical removal of the vital pulp.
Pulpotomy (pulp amputation)	The surgical removal of the coronal portion of a vital pulp as a means of preserving vitality of the remaining radicular portion is usually is performed as an emergency procedure for temporary relief of symptoms or therapeutic measure.
Partial pulpotomy (shallow pulpotomy; Cvek pulpotomy)	The surgical removal of a small diseased portion of vital pulp as a means of preserving the remaining coronal and radicular pulp tissues.
Apexification	Inducing a calcified or artificial barrier in a root with an open apex or the continued apical development of an incompletely formed root in teeth with a necrotic pulp.
Apexogenesis	A vital pulp therapy procedure performed to enable continued physiologic development and formation of the root end; term frequently used to describe vital pulp therapy that encourages the continuation of this process.

From American Association of Endodontists: Glossary of endodontic terms, Chicago, 2003, The Association. MTA, Mineral trioxide aggregate.

IATROGENIC EFFECTS ON THE DENTAL PULP

When most local anesthetics containing vasoconstrictors are used in restorative dentistry, the blood flow to the pulp is reduced to less than half of its normal rate (Figure 2-2).[3] In the case of lidocaine with epinephrine, this effect is entirely a result of the vasoconstrictor.[4] The local anesthetic without vasoconstrictor may in fact induce a small increase in blood flow and will be much less effective as an anesthetic. The epinephrine-induced reduction in blood flow will persist for some time after the anesthetic is injected. In procedures on teeth whose pulps are already compromised, this may be an additional stressor. Fortunately, the rate of oxygen consumption in the healthy pulp is relatively low, and if necessary, pulp cells can produce energy anaerobically through the pentose phosphate pathway of carbohydrate metabolism.[5] A healthy pulp may survive episodes of ischemia lasting for 1 hour or more. An already ischemic pulp subjected to severe injury may hemorrhage (blush) when subjected to trauma such as that associated with full crown preparation without the use of an adequate coolant.

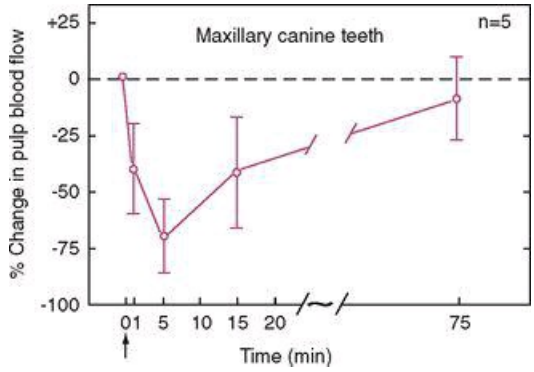

Figure 2-2 Effects of infiltration anesthesia (2% lidocaine with 1 : 100,000 epinephrine) on pulpal blood flow in the maxillary canine teeth of dogs. The *arrow* indicates the time of injection. The bar depicts SD.

(From Kim S, Edwall L, Trowbridge H, et al: J Dent Res 63(5):650, 1984.)

Heat

Frictional heat is produced whenever a revolving bur or stone contacts tooth structure. Until the advent of the high-speed handpiece, enamel and dentin preparation involved heavy torque, low rotational speeds, and steel burs that were not cooled with water. Consequently, vital dentin was often scorched, and pulps were injured. as a result of extreme heat (Figure 2-3).[6]

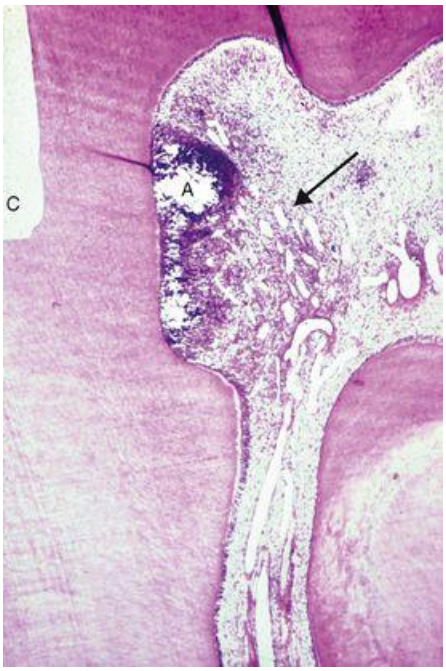

Figure 2-3 Localized inflammation *(arrow)* and abscess *(A)* formation beneath a deep cavity preparation *(C)* without adequate coolant.

(Courtesy Dr. H. Trowbridge).

Dentin is an effective insulator; for this reason, judicious cutting is not likely to damage the pulp unless the thickness of dentin between preparation and pulp is less than 1.0 mm.[7] Even then, the response should be mild (Figure 2-4). The greatest amount of frictional heat is generated with a large diamond stone when teeth are prepared for a full crown. The heat generated may also have a desiccating effect by "boiling" away dentinal tubule fluid at the dentin surface.

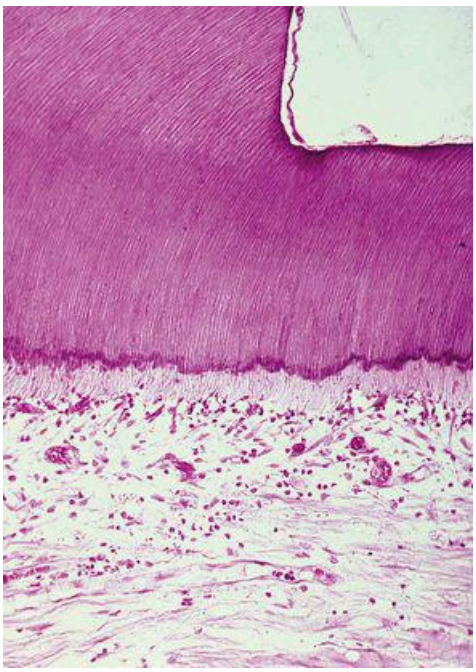

Figure 2-4 Mild inflammation beneath a deep cavity preparation with adequate coolant.

(Courtesy Dr. H. Trowbridge).

The “blushing” of dentin during cavity or crown preparation is thought to be due to frictional heat resulting in vascular injury (hemorrhage) in the pulp.[8] The dentin takes on an underlying pinkish hue soon after the operative procedure. Crown preparation performed without the use of a coolant leads to a marked reduction in pulpal blood flow, presumably because of vascular stasis and thrombosis. The amount of heat produced during cutting is determined by sharpness of the bur, the amount of pressure exerted on the bur or stone, and the length of time the cutting instrument contacts tooth structure. The safest way to prepare tooth structure is to use ultra-high speeds of rotation (100,000 to 250,000 rpm), with an efficient water cooling system, light pressure, and intermittent cutting. During cutting at high speeds, the revolving bur creates an area of turbulence that tends to deflect a stream of water. Therefore an air-water spray with sufficient volume and pressure must be used if the coolant is to overcome the rotary turbulence. *The bur-dentin interface should be constantly wet.*

Cavity preparation with a low-speed handpiece, sharp bur, and light, intermittent pressure is only slightly more injurious than cutting at high speeds. *Hand instruments and low-speed cutting are relatively safe ways to finish a cavity preparation,* rather than using a high-speed handpiece with the water coolant shut off.

The use of laser beams to fuse enamel and reduce the likelihood of carious invasion has been suggested.[9] Different lasers with different energy levels may also be used to remove caries. Lasers generate heat and increase intrapulpal temperature. The heat generated varies with a number of parameters but can be reduced by water cooling to a level similar to that caused by a water-cooled high-speed drill.[10]

Cavity Depth/Remaining Dentin Thickness

Dentin permeability increases exponentially with increasing cavity depth, as both diameter and density of dentinal tubules increase with cavity depth (Figure 2-5).[11] Thus the deeper the cavity the greater the tubular surface area into which potentially toxic substances can penetrate and diffuse to the pulp. The length of the dentinal tubules beneath the cavity is also important. The farther substances have to diffuse, the more they will be diluted and buffered by the dentinal fluid. A remaining dentin thickness of 1 mm is usually sufficient to shield the pulp from most forms of irritation. In noncarious teeth, tertiary reactive dentin is formed most rapidly when the remaining dentin thickness is between 0.5 and 0.25 mm.[12] With a narrower remaining dentin thickness, odontoblasts die and reparative dentin would be formed by newly differentiated cells.[13]

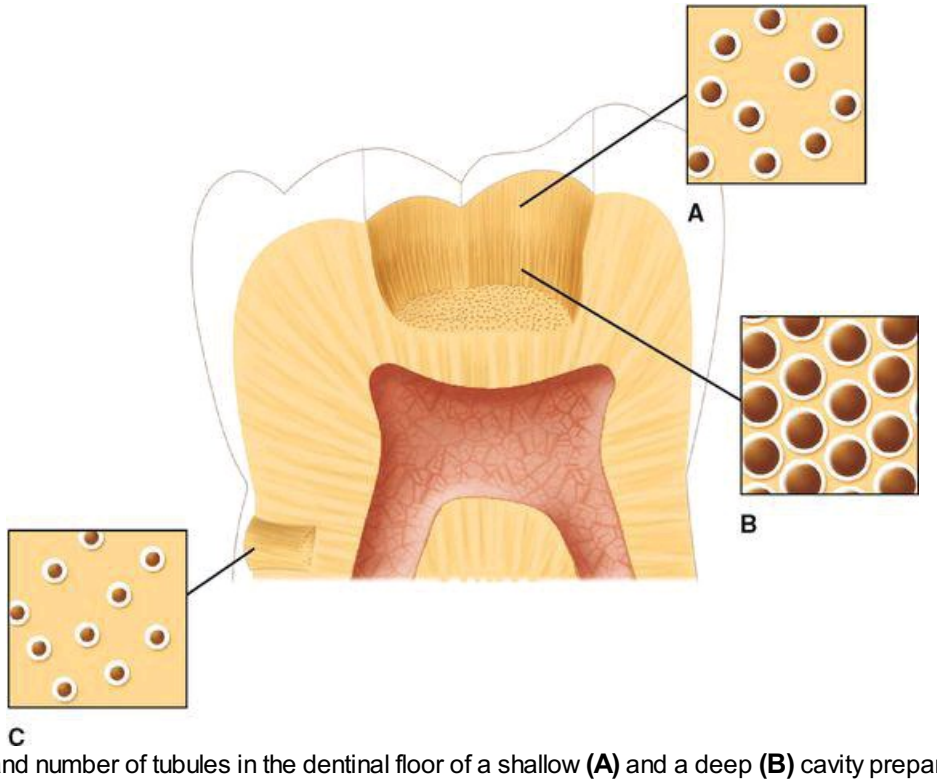

Figure 2-5 Difference in size and number of tubules in the dentinal floor of a shallow **(A)** and a deep **(B)** cavity preparation.

(From Trowbridge HO: Dentistry 82:22, 1982.)

Cavity Drying and Cleansing[12]

A prolonged blast of compressed air aimed onto freshly exposed vital dentin will cause a rapid outward movement of fluid in patent dentinal tubules. Tubule diameter midway between the pulp and the dentinoenamel junction (DEJ) is only 1.5 µm.

Therefore the removal of fluid from the tubules by a blast of air activates strong capillary forces. These in turn lead to a rapid outward flow of dentinal fluid. Fluid removed from the tubules at the dentin surface is replaced by fluid from the pulp.

As discussed previously, rapid outward flow of fluid in the dentinal tubules stimulates nociceptors in the dentin pulp, thus producing pain. Rapid outward fluid movement may also result in *odontoblast displacement* (Figure 2-6).[14] Odontoblasts are dislodged from the odontoblast layer and drawn outward into the tubules. Within a short time the displaced cells undergo autolysis and disappear. Providing the pulp has not been severely injured by either caries or other factors, displaced odontoblasts are replaced by new cells that are derived from stem cells deeper in the pulp. In this way the odontoblast layer is reconstituted by "replacement" odontoblasts capable of producing tertiary dentin. Even vigorous drying of dentin alone does not result in severe injury to the underlying pulp. Drying agents containing lipid solvents, such as acetone and ether, have been used to clean cavity floors. Because of their rapid rate of evaporation, application of these to exposed dentin produces strong hydrodynamic forces in the tubules, causing odontoblast displacement. *Cavities should be dried with cotton pellets and short blasts of air rather than harsh chemicals.* Disinfectants have in the past been used in cavity cleansing. There seems to be no particular benefit to this, and they are now rarely used as they are potentially toxic to the pulp.

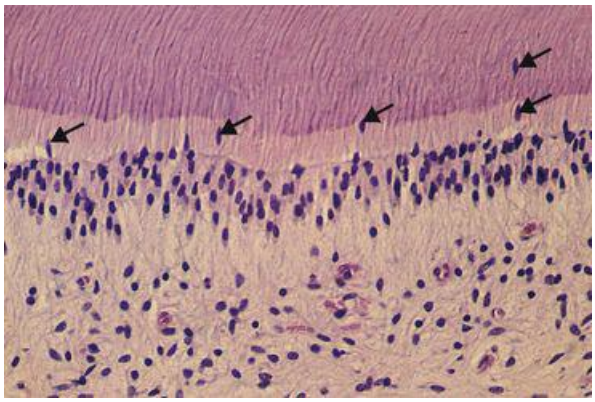

Figure 2-6 Aspiration of odontoblasts *(arrows)* into dentinal tubules following desiccation of a cavity.

(Courtesy Dr. H. Trowbridge).

Etching Dentin/Smear Layer Removal[15-17]

Cutting dentin results in a smear layer on the cut surface, consisting of fragments of microscopic mineral crystals and organic matrix produced. This layer may interfere with the adherence of adhesive restorative materials, although some newer bonding agents reportedly bond well to the smear layer. Acidic cavity-cleansing products and chelating agents have been used to remove the smear layer, but the use of these depends on the nature of the restorative material. The smear layer does have one desirable property. By blocking the orifices of dentinal tubules, the smear plugs greatly decrease the permeability of dentin. While the smear layer is largely impervious to bacteria, it is not a barrier to bacterial products.

Complete dissolution of the smear layer opens the dentinal tubules, significantly increasing the permeability of dentin. If the dentin is left unsealed, the diffusion of irritants to the pulp may intensify and prolong the severity of pulpal reactions. Available experimental evidence is contradictory. Some evidence indicates that etching as a step in restoration may reduce microleakage.[17] Other work suggests that etching when there is less than .3 mm of remaining dentin thickness causes pulpal damage.[15]

In the absence of microleakage, acid etching of dentin does not appear to produce injury to the pulp because calcium and phosphate ions are released, producing a buffering action. Even when placed in deep cavities, acid etchants produce only a small increase in hydrogen ion concentration in the pulp.[18]

Pins[19,20]

Pins used to retain amalgam require caution; it is preferable to use other, safer means of retention. Pulp damage may result from pinhole preparation or pin placement. Coolants do not reach the depth of the pin preparation. During pinhole preparation, there is always the risk of pulp exposure. Furthermore, friction-locked pins often produce microfractures that may extend to the pulp, subjecting the pulp to irritation and the effects of microleakage.

Impressions, Temporary Crowns, and Cementation[21-25]

Rubber base and hydrocolloid materials do not injure the pulp. Modeling compound may be damaging as a result of the combination of heat and pressure. Temperatures of up to 52° C have been recorded in the pulp during impression taking with

modeling compound.

The heat generated during the exothermic polymerization of autopolymerizing resins may injure the pulp. Cooling is strongly recommended when provisional crowns are fabricated directly. Before cementing provisional crowns, the crown preparation should be carefully lined with temporary cement to minimize microleakage. *The temporary crown/cement should be in place for a short period of time;* temporary cements are not stable and will eventually wash out. Microleakage around temporary crowns is a common cause of postoperative sensitivity.

During the cementation of crowns, inlays, and bridges, strong hydraulic forces may be exerted on the pulp as cement compresses the fluid in the dentinal tubules. In deep preparations, this can result in a separation of the odontoblast layer from the predentin. Vents in the casting will allow cement to escape and facilitate seating.

Polishing Restorations[26]

Enough frictional heat may be generated during the polishing of a restoration to seriously injure the pulp. Continuous polishing of amalgam or other metallic restorations with rubber cups at high speeds causes a damaging temperature increase of up to 20° C. *Polishing with rubber wheels, points, or cups should be at low speeds using intermittent pressure and a coolant.*

Removing Old Metallic Restorations

The use of burs to remove metallic restorations can produce very high levels of frictional heat. A coolant, such as water spray or a combination of water and air, avoids a burn lesion in the pulp.

Postrestorative Hypersensitivity[27,28]

Many patients complain of hypersensitivity after a restorative procedure. This may be due to any of the factors previously listed. Discomfort is usually of short duration. If pain is prolonged, a preexisting pulpitis may have been exacerbated. If delayed in onset by days, the cause may be microleakage of bacterial toxins under a poorly sealed temporary restoration. The absence of postoperative sensitivity after restoration with modern composites of both class I and II preparations has been demonstrated in clinical studies, suggesting that variations in technique may be responsible for the anecdotal reports.[29,30] The use of hydroxymethacrylate/glutaraldehyde “desensitizer” does not reduce the incidence of sensitivity.[31] Self-etching, self-priming dentin bonding systems reduce the incidence of sensitivity after the restoration of deep carious cavities.[32]

If pain is evoked by biting on a recently restored tooth, an intracoronal restoration may be exerting a strong shearing force on the dentin walls of the preparation. It is more likely to be caused by an injury to the periodontal ligament resulting from hyperocclusion. Hyperocclusion from an extracoronal restoration does not injure the pulp but may cause a transient hypersensitivity.

Microleakage[33-35]

The most important characteristic of any restorative material in determining its effect on the pulp is its ability to form a seal that prevents the leakage of bacteria and their products onto dentin and then into the pulp.

Cytotoxicity

Certain restorative materials are composed of chemicals that have the potential to irritate the pulp. However, when placed in a cavity, the intervening dentin usually neutralizes or prevents leachable ingredients from reaching the pulp in a high enough concentration to cause injury. For example, eugenol in zinc oxide–eugenol (ZnOE) is potentially irritating but very little can reach the pulp. Phosphoric acid is a component of silicate and zinc phosphate cements and was thought to be highly injurious to the pulp. However, the buffering capacity of dentin greatly limits the ability of hydrogen ions to reach the pulp. It is now clear that the problems following use of these materials were a result of their high degree of shrinkage and subsequent microleakage.[36]

Clearly, the thickness and permeability of dentin between a material and the pulp affect the response to the material. In addition, the penetration of some materials through dentin may be limited by the outward flow of fluid through the tubules, which will be increased if the pulp is inflamed.[37] This factor has been overlooked in many in vitro studies that investigate the passage of materials through dentin.

Many cytotoxicity studies examine isolated cell types in culture and do not take into account the immunocompetent cells present in the intact pulp. Materials may have a differential effect on these cells by either stimulating or inhibiting their activity.[38]

Materials are more toxic when they are placed directly on an exposed pulp. Cytotoxicity tests carried out on materials in vitro or in soft tissues may not predict the effect of these materials on the dental pulp. The toxicity of the individual components of a material may vary.[39,40] A set material may differ in toxicity from an unset material. The immediate pulpal response to a material is much less significant than the long-term response. A few days after placement the pulp may show a strong inflammatory response. A few months later, the inflammatory response may have done its job and repair has taken over. The best measure of long-term response is the thickness of tertiary dentin laid down by the affected pulp (Figure 2-7).

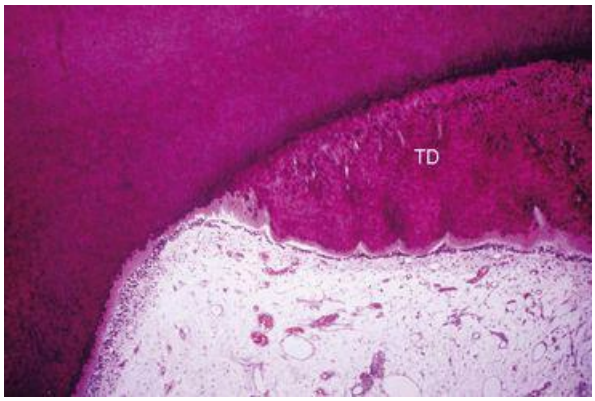

Figure 2-7 Tertiary dentin *(TD)* formed under a deep preparation and irritating material.

(Courtesy Dr. H. Trowbridge.)

Depth of Preparation

Deep cavity preparations are likely to destroy odontoblasts. These will be replaced by new odontoblasts that often form an irregular, reparative dentin that has few tubules (see Figure 1-8, *A*).

Heat on Setting[41]

Some luting cements generate heat during setting; it has been suggested that this might cause pulpal injury. The most exothermic luting material is zinc phosphate (ZnOP) cement. However, during setting an intrapulpal temperature increase of only 2° C was recorded. Heat of this magnitude is not sufficient to injure the pulp.

Desiccation by Hygroscopy[42]

Some hygroscopic materials may potentially cause injury by withdrawing fluid from dentin. However, little relationship exists between the hydrophilic properties of materials and their effect on the pulp. Moisture absorbed by materials is probably much less than that removed from dentin during cavity drying, which is a procedure that produces a small amount of pulpal inflammation.

Specific Materials

Zinc Oxide–Eugenol[12,43-45]

ZnOE has many uses in dentistry, having had a long history as a temporary filling material, cavity liner, cement base, and luting agent for provisional cementation of castings. Before the introduction of calcium hydroxide, ZnOE was the material of choice for direct pulp capping.

Eugenol, biologically the most active ingredient in ZnOE, is a phenol derivative and is toxic when placed in direct contact with tissue.[46] It also possesses antibacterial properties.[47] Eugenol's usefulness in pain control is attributed to its ability to block the transmission of nerve impulses.[48] Researchers have found that a thin mix of ZnOE significantly reduces intradental nerve activity when placed in a deep cavity preparation in cats' teeth; however, a dry mix of ZnOE has no effect.[49]

When included in cements to temporize crown preparation, some eugenol does reach the pulp, but the amounts are small and unrelated to remaining dentin thickness. "Desensitizing" agents do not seem to reduce the penetration.[33] The release of eugenol is by a hydrolytic mechanism, which depends on the presence of water. With little water available, release is low.[44,45]

The most important property of ZnOE is that it provides a tight marginal seal and thus prevents microleakage, thereby reducing hypersensitivity. Its superiority as a temporary restorative material is enhanced by its antimicrobial properties.

Zinc Phosphate Cement[27,50-52]

ZnOP is a popular luting and basing agent. It has a high modulus of elasticity and therefore is commonly used as a base beneath amalgam restorations. The phosphoric acid liquid phase was formerly thought to injure the pulp. However, recent studies have shown that this is not the case. Cementation of castings with ZnOP is well tolerated by the pulp. Researchers reported that ZnOP is more likely to produce pulpal sensitivity at the time of cementation and 2 weeks after cementation as compared with glass ionomer. However, 3 months after cementation there is no difference in sensitivity between the two luting agents.

Polycarboxylate Cement[13,52,53]

When placed in cavities or used as a luting cement, zinc polycarboxylate does not irritate the pulp. In cementing well-fitting crowns and inlays, neither polycarboxylate nor ZnOP cements contract enough to permit the ingress of bacteria. Consequently, it is unnecessary to apply a varnish or liner to cavity walls; doing so only reduces cement adhesion.

Restorative Resins[54-56]

Early adhesive bonding and resin composite systems contract during polymerization, resulting in gross microleakage and bacterial contamination of the cavity. Bacteria on cavity walls and within axial dentin are associated with moderate pulpal inflammation. Over a period of time, some composites absorb water and expand; this tends to compensate for initial contraction. To limit microleakage and improve retention, enamel margins are beveled and acid etched to facilitate mechanical bonding. When compared with unfilled resins, the newer resin composites present a coefficient of thermal expansion similar to that of tooth structure. With recently developed hydrophilic adhesive bonding composite systems, the problem of marginal leakage appears to have been diminished.

Glass Ionomer Cements

Glass ionomer cements were originally used as esthetic restorative materials, but these cements are now being used as liners, luting agents, and pulp capping agents (sometimes in conjunction with calcium hydroxide). When placed on exposed pulps in noncarious teeth, glass ionomer cement shows a degree of bacterial microleakage similar to composite resins but less than half that of calcium hydroxide cement.[57,58] The incidence of severe pulpal inflammation or necrosis caused by glass ionomer cement on exposed healthy pulps is similar to that for calcium hydroxide but greater than that for composite resins.[58] When placed in cavities in which the pulp is not exposed and there is a narrow remaining dentin thickness (0.5 to 0.25 mm) both calcium hydroxide and composite resin show faster deposition of tertiary dentin than glass ionomer cements.[12]

When used as a luting agent, the pulpal response to glass ionomer cements is similar to that of polycarboxylate and ZnOP cements.[51] For some time after their introduction as luting agents, glass ionomer cements were associated with postcementation sensitivity. A more recent clinical trial has shown that the incidence of sensitivity using these agents with appropriate technique is no greater than other commonly used luting agents.[27]

Amalgam

Amalgam alloy is still a widely used material for restoring posterior teeth. Shrinkage during setting results in microleakage.[59] This decreases as corrosion products accumulate between restoration and cavity walls and can be reduced by the use of liners.[60] Amalgam is the only restorative material in which the marginal seal improves with time. Esthetics and public concern with the mercury content of amalgams have led to an accelerated use of composite resins as posterior restorative materials. Their use is more technique sensitive than amalgams. In deep cavities in posterior teeth, composites are associated with more pulpal injury than amalgams because of microleakage.[54]

Orthodontic Tooth Movement

Orthodontic tooth movement of a routine nature does not cause clinically significant changes in the dental pulp. Responses to pulp testing, especially electrical testing, may be unreliable.[61] The heavy forces used to reposition impacted canines frequently lead to pulp necrosis or calcific metamorphosis.[62] Intrusion but not extrusion reduces pulpal blood flow for a few minutes as the pressure is applied.[63] Capillaries proliferate in the pulps of moving teeth.[64] A variety of growth factors are produced, including vascular endothelial growth factor, which may explain this increase in vessels.[65]

Vital Tooth Bleaching

Overnight external bleaching of anterior teeth with 10% carbamide peroxide causes mild pulpitis, which is reversed within 2 weeks.[66] In vitro studies show that the principal bleaching agent, hydrogen peroxide, can reach the pulp[67] after application to the enamel. Whether this occurs in vivo is unknown. Outward fluid flow in dentinal tubules and other factors would reduce the effect. Both short-term[68] and long-term (9 to 12 years) clinical observation on bleached teeth report no significant pulpal changes.[69] Heat-activated bleaching agents can cause intrapulpal temperatures to rise by 5° to 8° C when measured in vitro.[70]

PROTECTING THE PULP FROM THE EFFECT OF MATERIALS

Cavity Varnishes, Liners, and Bases

A liner is routinely placed between restorative materials and the dentin to improve the overall performance of a restoration. The main concern is to reduce or eliminate microleakage. In vitro studies suggest that most liners show some degree of leakage,[60] but it is unknown what level of dye leakage would relate to clinical problems. One 3-year clinical study[71] compared three common dentin treatments but found no recurrent caries around any of the restorations, including those where no liner was used. All liners and bases reduce dentin permeability but to different extents. Bases provide the largest reduction, varnishes the least.[72]

"Insulating" Effect of Bases

A common misconception is the necessity of placing an insulator beneath metallic restorations to protect the pulp from thermal shock (hypersensitivity). Dentin is an excellent insulator; additional thermal insulation is rarely if ever needed. In fact, thick cement bases are no more effective than just a thin layer of varnish in preventing thermal sensitivity, indicating that postrestorative sensitivity is at least partly a result of microleakage.[73]

VITAL PULP THERAPIES

Maintaining an intact healthy pulp is preferable to root canal treatment or other endodontic procedures that are complex, expensive, and time consuming. When dealing with a deep carious lesion, indirect pulp capping, a procedure that avoids accidental pulp exposure during the removal of carious dentin, may be attempted. Another approach is to remove all carious dentin. If there is a carious exposure, the exposed pulp tissue is covered with a biocompatible liner (direct pulp capping). Others advocate a procedure involving surgical removal of inflamed pulp tissue (pulpotomy or pulpectomy); the remaining tissue is then covered with dressing that hopefully allows healing. The success rate of these procedures is variable and depends on proper diagnosis and clinical judgment but primarily on the status of the pulp before the procedure.

Removal of Dental Caries

Caries is a localized, progressive destruction of tooth structure and the most common cause of pulp disease. It is now generally accepted that for caries to develop, specific bacteria must become established on the tooth surface. Products of bacterial metabolism, notably organic acids and proteolytic enzymes, cause the destruction of enamel and dentin. Bacterial metabolites diffusing from the lesion to the pulp are capable of eliciting an immune response and inflammatory reaction. Eventually, extensive dentin involvement results in bacterial infection of the pulp, particularly after carious exposure.

Removal of gross caries and preparation for restoration are generally accomplished with rotary instruments. Hand instruments are avoided near the pulpal wall to prevent accidental mechanical pulp exposure. Final removal of carious dentin is best accomplished with a large, sharp round bur at slow speed to remove the last layer of softened dentin.

Capping the Vital Pulp

Step-Wise Excavation of Caries

Step-wise excavation of caries is a technique in which *caries is removed in increments in two or three appointments over a few months to a year* rather than removing the caries in a single sitting, which might result in an accidental exposure and contamination of the pulp. The deeper affected but noninfected dentin may remineralize,[74] and tertiary dentin may form. *Each time caries is removed, glass ionomer base is placed, which may contribute to mineralization,[75] followed by a well-sealing temporary restoration.*

For this technique to be successful, careful case selection is necessary. There must be no signs or symptoms of irreversible pulpitis, recognizing that irreversible pulpitis is frequently asymptomatic. If the caries has already penetrated to the pulp, this will be unsuccessful; the pulp is already irreversibly damaged. *Therefore it is critical that follow-up evaluation include pulp testing and radiographs, as pulp necrosis may occur even years later.*

There are important considerations. One is that the term *indirect pulp cap* is a misnomer. A material, such as calcium hydroxide, placed on caries will not have a beneficial effect on the pulp. Another is that, if the caries has already penetrated to the pulp, the pulp is infected and will not survive (irreversible pulpitis).

The controversy occurs between those who believe all caries should be removed and those who believe that the pulp is capable of recovery from lesser insults. Each case must be assessed on its own merits and within an overall treatment plan.

Direct Pulp Cap

There are two considerations for direct pulp capping: accidental mechanical pulp exposure during cavity preparation and exposure caused by caries. In either case the pulpal health should be normal or if signs of reversible pulpitis are present. These two types of exposure differ in that the condition of the pulp is likely to be reversible pulpitis in the case of an accidental mechanical exposure, whereas it is likely to be severely inflamed beneath a deep carious lesion. When a small mechanical exposure of the pulp is encountered during cavity preparation, after hemorrhage is controlled, capping the exposed pulp with hard-set calcium hydroxide or preferably mineral trioxide aggregate (MTA) covered by glass ionomer cement should be followed by a permanent restoration with a good marginal seal. The long-term success rate for direct pulp capping of small, clean mechanical exposures is high (80%) but very low for carious exposures[76] and considered unacceptable by most practitioners. Teeth in which the pulp is cariously exposed should be treated by root canal therapy.

Pulpotomy[77,78]

Pulpotomy is an alternative to direct pulp capping or root canal treatment when carious pulp exposures occur in young permanent teeth and the inflammation may be restricted to the crown. The pulp must be vital, with a diagnosis of normal pulp or reversible pulpitis.[79,80] All the carious dentin and the pulp to the level of the radicular pulp are removed. Bleeding from the vital pulp stump is controlled, the surface is rinsed with sodium hypochlorite, and then it is capped with calcium hydroxide or preferably mineral trioxide aggregate (MTA). This is covered by glass ionomer cement, and the tooth is restored with a restoration that seals completely and forever. The remaining pulp should continue to be vital after partial pulpotomy. Follow-up examination should show no adverse clinical signs or symptoms such as severe pain or swelling. There should be no radiographic sign of internal or external resorption, abnormal canal calcification, or periapical radiolucency postoperatively. Teeth with immature roots should continue normal root development and apex formation and closure (Figure 2-8).[81-85]

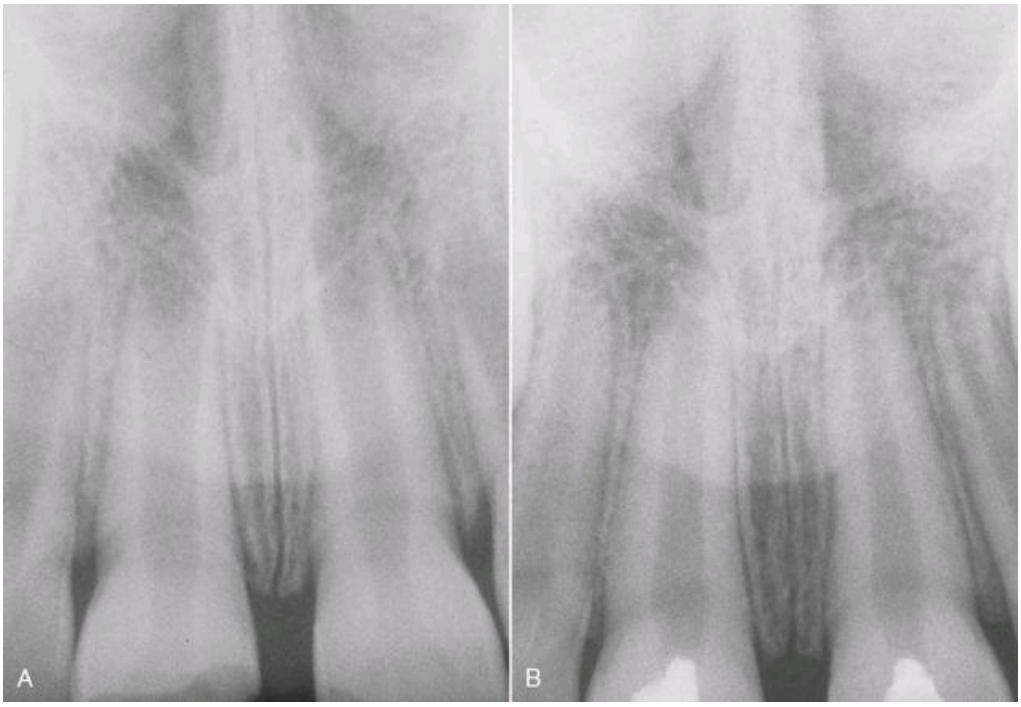

Figure 2-8 Preoperative **(A)** and postoperative **(B)** radiographs demonstrate continued root development after pulpotomy procedures on both incisors. If these have good sealing restorations, the pulps should remain healthy; root canal treatment would be unnecessary.

THE OPEN APEX[86,87]

An open apex is in the developing root of immature teeth until apical closure occurs, which is approximately 3 years after eruption. In the absence of pulpal or periapical disease, such an open apex is normal. However, if the pulp becomes necrotic before root growth is complete, dentin formation ceases and root development is arrested. The resultant root is short with thin and consequently weakened dentin walls. The walls may diverge (Figure 2-9), be parallel, or converge slightly, depending on the stage of root development. The apex is comparatively large and lacks constriction (Figure 2-10). An open apex may also develop as a result of extensive resorption of a previously mature apex after orthodontic treatment or severe periapical inflammation (Figure 2-11). The presence of an open apex provides significant challenges in the treatment of pulpal injury. When the apex is not closed, routine root canal procedures cannot be performed; the results of treatment are unpredictable. Depending on the vitality of the affected pulp, two approaches are possible—apexogenesis (vital pulp therapy) or apexification (root-end closure).

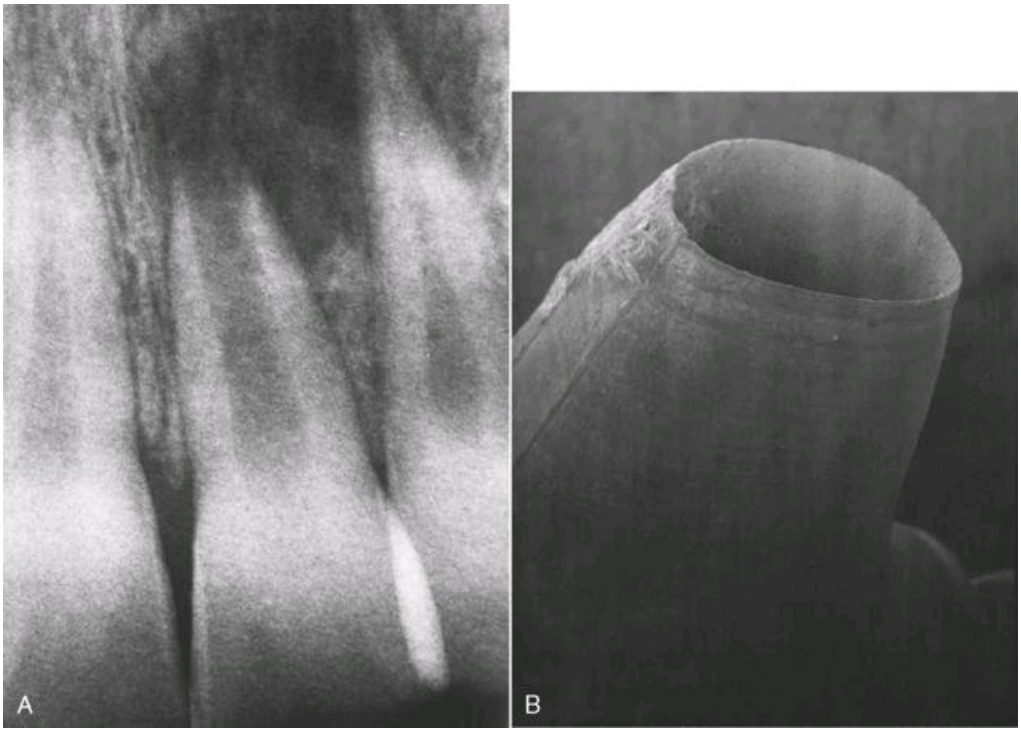

Figure 2-9 A, Incisor (history of luxation injury) with an open apex (divergent walls), necrotic pulp, and apical pathosis. Root-end closure is indicated. **B,** Apical region of an immature central incisor extracted from a 7-year-old. Besides being open, the apical dentin walls are egg-shell thin. These teeth are difficult to treat; long-term prognosis is questionable.

(Courtesy Dr. L. Baldassari-Cruz.)

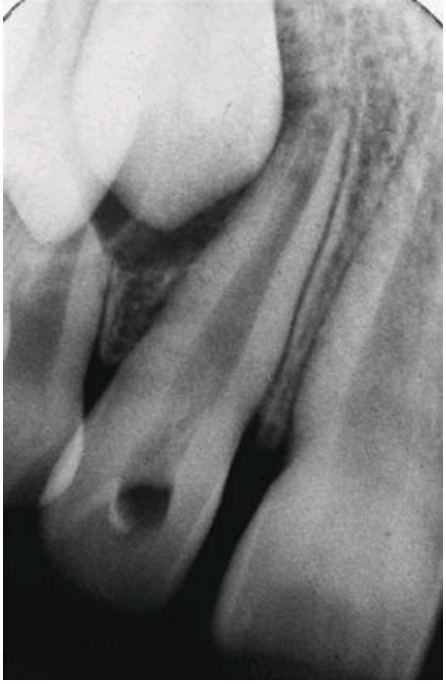

Figure 2-10 Incisor with a necrotic pulp, but with substantial dentin formation and an open apex (parallel walls). An access opening has been made into the pulp chamber. Root-end closure with a barrier is indicated. Long-term prognosis is good.

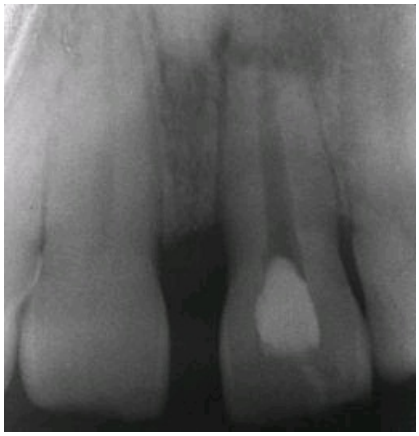

Figure 2-11 Resorbed apex (now open) caused by periapical inflammation resulting from pulpal necrosis.

The importance of careful case assessment and accurate pulpal diagnosis in the treatment of immature teeth with pulpal injury cannot be overemphasized. Clinical assessment of pulpal status requires a thorough history of subjective symptoms, careful clinical and radiographic examination, and performance of diagnostic tests as described in Chapter 5.

Radiographic interpretation may be difficult in the case of the developing, immature root. A radiolucent area usually surrounds the apex of an immature root with a healthy pulp. It may be difficult to differentiate between this finding and a pathologic radiolucency resulting from a necrotic pulp. A radiolucent lesion tends to have a noncorticated, diffuse border. Also, comparison with the periapex of the contralateral tooth may be helpful. A radiograph represents a 2-dimensional picture of a 3-dimensional situation. Therefore only the mesiodistal aspect is seen in a routine radiograph. Frequently, the apex appears almost completely closed in this view, but in actuality is wide open when observed from the proximal aspect (Figure 2-12).

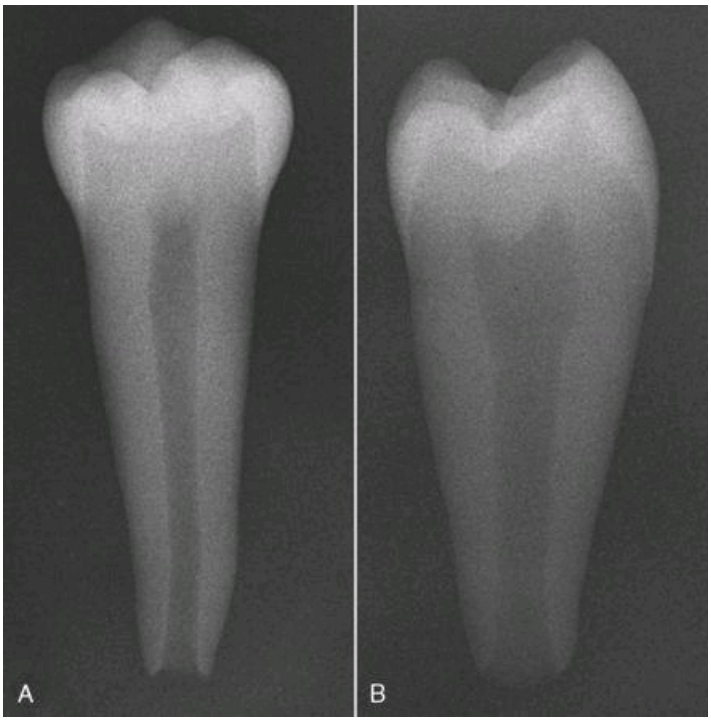

Figure 2-12 A, The apex appears to be near parallel as seen from the facial aspect.

B, *From the proximal, the apical walls diverge.*

Unfortunately, a close correlation between the results of these individual tests and the actual histologic diagnosis of pulpal status does not exist. However, by combining the results of the history, clinical examination, and diagnostic tests, an accurate diagnosis of pulpal and periapical condition can usually be established.

Treatment Planning

The major considerations in treatment planning are pulpal status and degree of root development (Figure 2-13). If the pulpal diagnosis is reversible pulpitis, the appropriate treatment is vital pulp therapy, or apexogenesis, regardless of the degree of root development. Depending on the extent of pulp damage, pulp capping or shallow or conventional pulpotomy may be indicated.

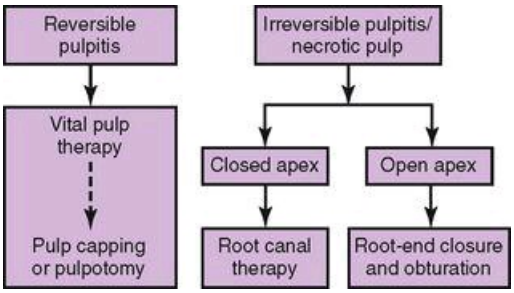

Figure 2-13 Case selection decision tree.

If the diagnosis is irreversible pulpitis or pulpal necrosis, the appropriate treatment is determined by the degree of root development. If root development is complete and the apex is closed, conventional root canal therapy can be performed. However, when root development is incomplete, root-end closure must be induced before obturation.

In devising a treatment plan, the considerations are whether the tooth can be restored and the potential for root fracture because of the thin-walled roots. Patient compliance is important, as treatment of the immature root by induced closure (usually not the preferred approach) may require a number of visits over a prolonged period of time. Alternatives to induction of root-end closure include apical barrier techniques (single-visit apexification), endodontic surgery, or extraction.

Apexogenesis[88-93]

Apexogenesis is defined as a vital pulp therapy procedure performed to encourage continued physiologic development and formation of the root end. The objective is to maintain the vitality of the radicular pulp. Therefore the pulp must be vital and capable of repair, which is often the case when an immature tooth sustains a small coronal exposure after trauma. A small exposure can be treated by pulp capping. The steps involved in pulp capping and apexogenesis with MTA can be viewed.

With more extensive pulpal exposures an attempt is made to remove the inflamed tissue, leaving the rest of the pulp intact. It has been demonstrated that, for up to 168 hours after the traumatic incident, inflammation is limited to the most superficial 2 mm of the pulp. Treatment in these cases is a shallow pulpotomy (Cvek pulpotomy) in which only the superficial 2 to 4 mm of pulp is removed.

When there is a larger exposure, the pulp must be amputated at the level of the cervical constriction (conventional pulpotomy). With both pulpotomy techniques the remaining pulp can be then be capped with a hard-set calcium hydroxide or preferably MTA.

Technique

1. Anesthesia is obtained, and the rubber dam placed.

2. The inflamed pulp tissue is removed. This may involve removal of the most superficial 2 to 4 mm of pulp (Cvek pulpotomy),[81,94-96] using a sharp round bur in a high-speed handpiece with water cooling, or removal of the entire coronal pulp to expose the radicular pulp (conventional pulpotomy) using a sharp spoon excavator.

3. Hemorrhage is controlled by pressure on a cotton pellet moistened with saline. Failure to achieve hemostasis may indicate that inflamed tissue remains and that more pulp tissue must be removed.

4. The exposed pulp is rinsed with 2.5% sodium hypochlorite.

5. A material is placed over the amputated pulp. MTA is the preferred material, although historically hard-set calcium hydroxide has been widely used.[97-100] The tissue response to MTA is excellent, whereas there is always a zone of necrosis beneath calcium hydroxide.[79,88,101]

6. The MTA is prepared immediately before use by mixing the powder with sterile water or saline at a ratio of 3 : 1 on a glass or paper slab. The mixture is placed on the exposed pulp and patted in place with a moist cotton pellet. Because MTA sets in the presence of moisture over a 3-hour period, a wet cotton pellet is placed over the material and the rest of the cavity is filled with a temporary filling material. Alternatively, the entire cavity can be filled with MTA and protected with a wet piece of gauze for 3 to 4 hours. The coronal 3 to 4 mm of MTA is removed, and a final restoration placed immediately.

The primary goal of apexogenesis is to maintain pulp vitality, thus allowing dentin formation and root-end closure. Then the remaining odontoblasts can form dentin, producing a thicker root that is less prone to fracture. The time required to produce a thicker root varies between 1 and 2 years, depending on the degree of root development at the time of the procedure. The patient should be recalled at 6-month intervals to determine the vitality of the pulp and the extent of apical maturation. Absence of symptoms does not indicate absence of disease. At each recall appointment, signs and symptoms are monitored, pulp vitality tested, and radiographs obtained to determine periapical status. One advantage of pulp capping and shallow pulpotomy is the ability to test pulp vitality.

The ideal outcome of apexogenesis therapy is continued apical growth of the root with a normal apex (Figure 2-14). Vital tissue may be maintained for long periods of time, often indefinitely. After pulp capping and shallow pulpotomy, histologic examination of the pulp usually shows normal tissue. Therefore histologic evidence does not support routine pulp extirpation and root filling after apical closure. The success rate is lower after conventional pulpotomy; calcific metamorphosis is a common occurrence. When there is evidence of such calcification, it has been suggested that root canal therapy should be initiated. This is probably unwarranted, as calcific metamorphosis is not in itself pathologic. However, should the pulp become necrotic at some future date, the canals may not be negotiable; surgery would be necessary.

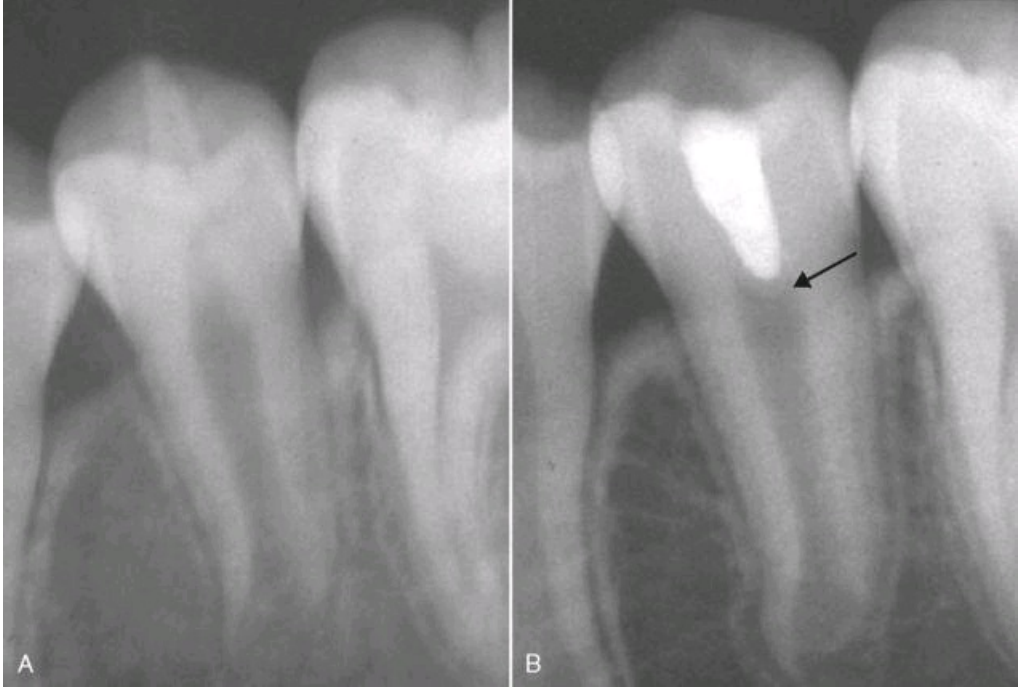

Figure 2-14 A, Premolar with dens evaginatis resulting in pulp exposure. **B,** One-year postoperative after pulpotomy with MTA demonstrating evidence of a dentin barrier *(arrow)* and continued root development.

If it is determined that the pulp has become irreversibly inflamed or necrotic before root development is complete, or if internal resorption is evident, the pulp is removed and apexification therapy initiated.

Apexification[92,102]

Apexification is the induction of a calcific barrier (or the creation of an artificial barrier) across an open apex. Apexification involves removal of the necrotic pulp followed by debridement of the canal and placement of an antimicrobial medicament (Figure 2-15). In the past, much emphasis has been placed on the type and properties of the medicament and many materials have been proposed for the induction of an apical barrier. However, it has been demonstrated that the critical factors in apical barrier formation are thorough debridement of the root canal system and establishment of a complete coronal seal. The nature of the medicament is less important.

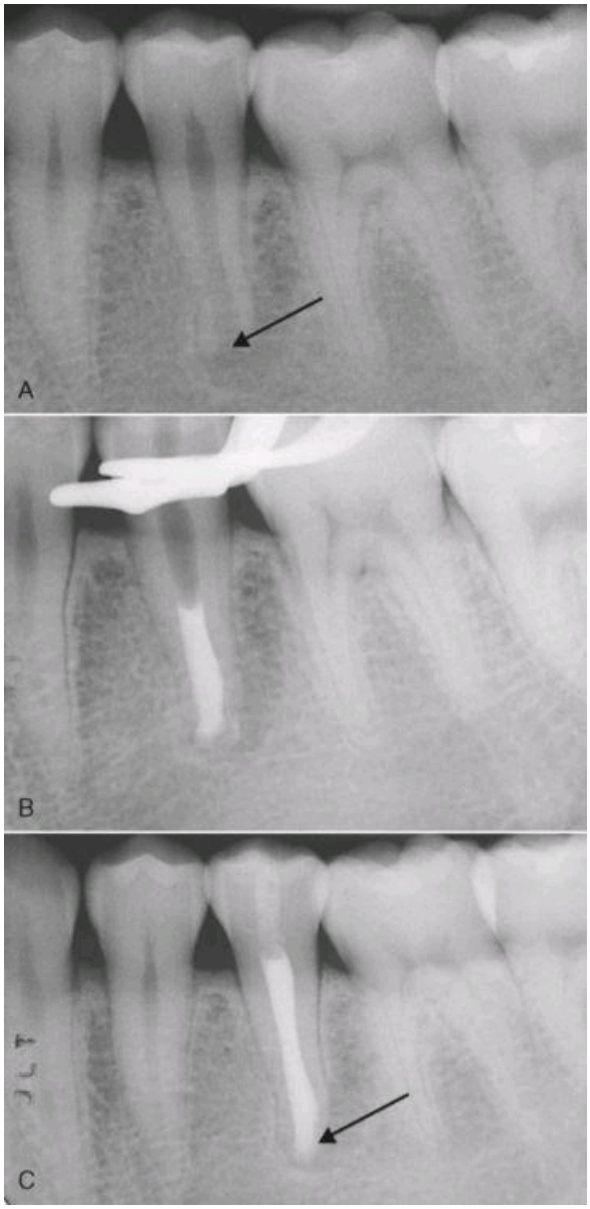

Figure 2-15 Apical closure with an artificial barrier. **A,** Pulp necrosis with arrested root development and an open apex *(arrow)*. **B,** After cleaning and shaping, MTA powder is compacted into the apical half. **C,** The coronal half is back-filled with gutta percha, and the access is restored with composite resin. MTA has formed an apical barrier *(arrow)*.

(Courtesy Dr. A. Williamson.)

Calcium hydroxide has been the most widely accepted material for induction of an apical barrier. The mechanism of action of calcium hydroxide remains controversial in spite of much research on its effect on pulp tissue. It has, however, been demonstrated that the reaction of the periapical tissues to calcium hydroxide is similar to that of pulp tissue. Calcium hydroxide produces a multilayered, sterile necrosis permitting subjacent mineralization.[103]

Recently, interest has centered on the use of MTA for apexification.[99,104] This material has demonstrated good biocompatibility and ability to seal, and its high pH may impart some antimicrobial properties.

Technique

1. After isolation, a large access cavity is prepared to allow removal of all necrotic tissue.

2. It may be possible at this point to remove some of the necrotic pulp tissue by inserting, rotating, and withdrawing a barbed broach or Hedstrom file.

3. Working length is established, slightly short of the radiographic apex. Instrumentation beyond the apex is not advocated as it may damage the tissue that will ultimately form the barrier.

4. Instrumentation is performed with gentle circumferential filing, starting with a relatively large file and progressing through the sizes. The objective is to maximize cleaning by copious irrigation with sodium hypochlorite and minimal dentin removal.

5. Large paper points are used to dry the canal.

6. MTA is introduced into the canal as either a powder or paste (by mixing with sterile saline) and packed with endodontic pluggers.

MTA produces an artificial barrier against which an obturating material may be condensed. If calcium hydroxide were placed, it would allow the tissue to form a biologic barrier. While calcium hydroxide has been widely used, it has serious disadvantages. It has been shown to weaken the dentin and must be replaced at monthly intervals and removed some months after placement before final obturation. MTA is clearly the material of choice.

After placement of the MTA barrier, a radiograph is made to confirm that the canal space close to the apex is adequately filled (see Figure 2-15). A wet cotton pellet is placed above the MTA to ensure setting, and a well-sealing temporary restoration is placed. The patient is recalled when the MTA has set (at least 24 hours) for obturation and placement of the permanent restoration.

Restoration After Apexification[103,105]

Because of the thin dentinal walls, there is a high incidence of root fracture in teeth after apexification. Restorative efforts should be directed toward strengthening the immature root. The use of newer dentin bonding techniques can significantly increase the resistance to fracture of these teeth to levels close to those of intact teeth. The reinforcing effect of resin glass ionomer in immature teeth has recently been demonstrated.[105]

Success or Failure of Apexification

Failure can occur either during or after treatment. The most common cause of failure is bacterial contamination, usually caused by loss of the coronal restoration or inadequate debridement of the canal. After apparently successful treatment, all patients should be recalled at 12-month intervals for 4 years. At the recall appointment the tooth should be carefully examined clinically and radiographically. Some apexification cases that were initially successful may subsequently fail, even though a barrier across the apex was present and an appropriate root canal filling was placed. Infected necrotic material trapped in the barrier may contribute to these failures, particularly if treatment was not performed under strict aseptic conditions. A further cause of failure is an undetected root fracture.

Treatment of the immature tooth with pulpal injury provides significant challenges. It is possible that advances in tissue engineering techniques may permit regeneration of the pulp and dentin without removing the entire pulp. Tissue engineering is the science of design and manufacture of new tissues to replace tissues lost to disease or trauma. It involves the following three key elements:

- □ Stem/progenitor cells
- □ Signals or morphogens that induce morphogenesis
- □ A scaffold that provides a 3-dimensional microenvironment for cell growth and differentiation

The pulp contains adult *stem/progenitor cells* that can potentially differentiate into odontoblasts. Such differentiation requires a morphogen and a scaffold. A morphogen is an inductive signal that functions as a growth/differentiation factor in odontoblast differentiation. Recombinant human bone morphogenetic protein (BMP)[110,111] will cause adult pulp cells to differentiate into odontoblasts. A *scaffold* is a biocompatible, 3-dimensional structure for cell adhesion and migration.[112] Possible scaffolds include natural polymers, such as collagen and glycosaminoglycans, which are found in the pulp and offer good biocompatibility and bioactivity, while synthetic polymers, such as polylactic acid (PLA) and polyglycolic acid (PGA), provide superior mechanical strength. Scaffolds containing inorganic compounds, such as hydroxyapatite and calcium phosphate, enhance bone conductivity.

There are two possible approaches to regeneration of pulp and dentin. First, there is in vivo therapy, in which BMP or BMP genes are applied directly to the exposed, amputated pulp. The in vivo approach has, thus far, demonstrated no success in the presence of inflammation. In the ex vivo approach, the stem/progenitor cells are isolated from the pulp and are differentiated into odontoblasts with recombinant BMP and BMP genes. The BMP transfected cells are then autogenously transplanted into the exposed pulp. The techniques for regeneration of pulp and dentin have progressed rapidly in recent years, but a number of challenges remain. Although promising, these techniques must be proved clinically and must demonstrate that they provide a reliable and costeffective alternative to currently available methods for treatment of pulp.

Chapter Review Questions available in Appendix B or on the DVD

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Endodontic Microbiology

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Understand the microbial etiology of apical periodontitis.
2. Describe the routes of entry of microorganisms to the pulp and periradicular tissues.
3. Recognize the different types of endodontic infections and the main microbial species involved in each one.
4. Understand the bacterial diversity within infected root canals.
5. Describe the factors involved with symptomatic endodontic infections.
6. Understand the ecology of the endodontic microbiota and the features of the endodontic ecosystem.
7. Discuss the role of microorganisms in the outcome of endodontic treatment.
8. Understand the development and implications of extraradicular infections.

MICROBIAL CAUSATION OF APICAL PERIODONTITIS

Apical periodontitis is an inflammatory disease of microbial etiology primarily caused by infection of the root canal system (Figure 3-1).[1-3] The unequivocal role of microorganisms in the causation of apical periodontitis was established nearly 40 years ago; a huge amount of new information about the microbiology of endodontic infections has been learned in the last decade. Endodontic infections usually develop after pulpal necrosis or in cases in which the pulp was removed for treatment. Although fungi and most recently archaea and viruses have been found in endodontic infections, bacteria are the major microorganisms implicated in the etiology of apical periodontitis. Bacteria colonizing the root canal system contact the periradicular tissues via apical and lateral foramina. 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.

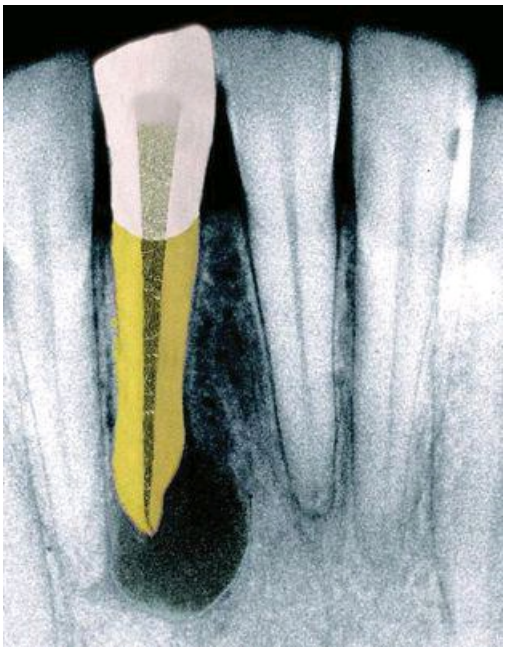

Figure 3-1 Microorganisms infecting the root canal system are the major causative agents of the different forms of apical periodontitis.

The ultimate goal of the endodontic treatment is either to prevent the development of apical periodontitis or to create adequate conditions for periradicular tissue healing. Taking into account the microbial etiology of apical periodontitis, the rationale for endodontic treatment is unarguably to eradicate the occurring infection or to prevent microorganisms from infecting or reinfecting the root canal or the periradicular tissues. The purpose of this chapter is to describe the microbiological aspects of endodontic infections.

ROUTES OF ROOT CANAL INFECTION

Under normal conditions, the dental pulp and dentin are sterile and isolated from oral microorganisms by overlying enamel and cementum. There are situations in which the integrity of these protective layers is breached (e.g., as a result of caries, trauma-induced fractures and cracks, restorative procedures, scaling and root planing, attrition, or abrasion) or naturally absent (e.g., because of gaps in the cementoenamel junction at the cervical root surface). As a consequence, the dentin-pulp complex is exposed to the oral environment and put at risk of infection by oral microorganisms. Microorganisms from subgingival biofilms associated with periodontal disease or present in the circulation during bacteremia may also reach the pulp via lateral and apical foramina. The main portals of pulp infection are dentinal tubules, direct pulp exposure, periodontal disease, and anachoresis.

Dentinal Tubules

Whenever dentin is exposed, the pulp is put at risk for infection as a consequence of the permeability of normal dentin dictated by its tubular structure. Dentin permeability is increased near the pulp because of the larger diameter and higher density of tubules.[4] Exposed dentin can be challenged by microorganisms present in carious lesions, in saliva bathing the exposed area, or in dental plaque formed onto the exposed area.

Dentinal tubules traverse the entire width of the dentin and have a conformation of inverted cones, with the smallest diameter in the periphery, near to enamel or cementum (mean of 0.9 µm).[5] 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. Thus one might well assume that once exposed, dentin offers an unimpeded access pathway for bacteria to reach the pulp via tubules. However, this is not the case. Bacterial invasion of dentinal tubules occurs more rapidly in nonvital teeth than in vital ones.[6] In vital teeth, outward movement of dentinal fluid and the tubular contents influence dentinal permeability and can conceivably delay intratubular invasion by bacteria. Other factors, such as dentinal sclerosis beneath a carious lesion, reparative dentin, smear layer, and intratubular accumulation of host defense molecules, also limit or even impede bacterial progression to the pulp via dentinal tubules.[4] Thus, as long as the pulp is vital, dentinal exposure does not represent a significant route of pulpal infection, except when dentin thickness is considerably reduced and then dentin permeability is significantly increased. On the other hand, if the pulp is necrotic, exposed dentinal tubules can become true avenues for bacteria to reach and colonize the pulp.

Direct Pulp Exposure

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 pulpal exposure, but microorganisms may also reach the pulp via direct pulpal exposure as a result of iatrogenic restorative procedures or trauma. The exposed pulp tissue develops direct contact with oral microorganisms from carious lesions, saliva, or plaque accumulated onto the exposed surface (Figure 3-2). 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.[7]

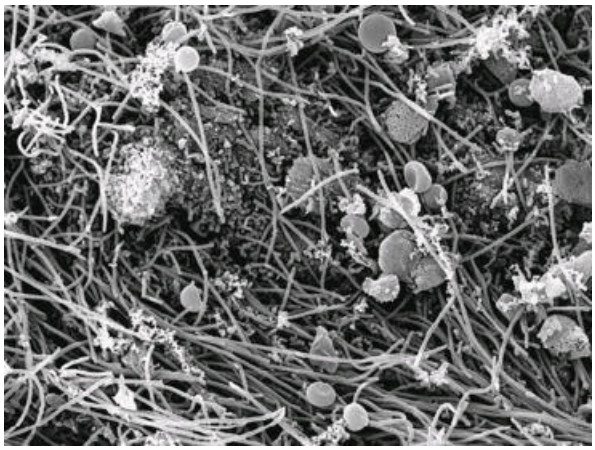

Figure 3-2 Scanning electron micrograph showing bacteria in a carious lesion. Note the presence of different bacterial morphotypes.

Periodontal Disease

Microorganisms in subgingival biofilms associated with periodontal disease could reach the pulp by the same pathways that intracanal microorganisms reach the periodontium and thereby could exert harmful effects on the pulp. Nevertheless, it has been demonstrated that although degenerative and inflammatory changes of different degrees may occur in the pulp of teeth with associated periodontal disease, pulpal necrosis as a consequence of periodontal disease only develops if the periodontal pocket reaches the apical foramen, leading to irreversible damage to the main blood vessels that penetrate through this foramen.[8] Once the pulp becomes necrotic, periodontal microorganisms can reach the root canal system via ramifications, exposed dentinal tubules, and apical foramina and establish an infectious process (see Chapter 7).

Anachoresis

Anachoresis is a process by which 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. There is no clear evidence showing that this process can represent a route for root canal infection. It has been shown 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.[9] Although anachoresis has been suggested to be the mechanism through which traumatized teeth with seemingly intact crowns become infected,[10] current evidence indicates that the main pathway of pulpal infection in these cases is dentinal exposure as a result of enamel cracks.[11]

TYPES OF ENDODONTIC INFECTIONS

Endodontic infections can be classified according to the anatomic location (intraradicular or extraradicular). Intraradicular infections can in turn be subdivided into three categories: primary, secondary, or persistent infection, depending on the time participating microorganisms established themselves within the root canal. The composition of the microbiota may vary depending on the different types of infection and different forms of apical periodontitis.

Intraradicular Infection

Microorganisms colonizing the root canal system cause intraradicular infection, which can be classified as primary, secondary, or persistent.

Primary Intraradicular Infection

Microorganisms that initially invade and colonize the necrotic pulp tissue cause primary intraradicular infection. It has also been referred to as initial infection or "virgin" infection. Participating microorganisms can have been involved in the earlier stages of pulpal invasion, which culminated in inflammation and further necrosis, or they can be latecomers that took advantage of the environmental conditions in the canal after pulp necrosis. Primary infections are characterized by a mixed consortium composed of 10 to 30 bacterial species and 10^3 to 10^8 bacterial cells per canal.[2,12,13] The involved microbiota is conspicuously dominated by anaerobic bacteria, but some facultative or microaerophilic species can also be commonly found in primary intraradicular infections.

Secondary Intraradicular Infection

Microorganisms that were not present in the primary infection but that were introduced into the root canal system at some time after professional intervention cause secondary intraradicular infections. The entry can be during treatment, between appointments, or even after root canal filling. Species involved can be oral or nonoral microorganisms, depending on the cause of infection.

The main causes of microbial introduction in the canal *during treatment* include remnants of dental plaque, calculus, or caries on the tooth crown; leaking rubber dam; or contamination of endodontic instruments, irrigating solutions, or other intracanal medications. Microorganisms can enter the root canal system *between appointments* by loss or leakage of temporary restorative materials, by fracture of the tooth structure, and in teeth left open for drainage. Microorganisms can also penetrate the root canal system *after root canal filling* by loss or leakage of temporary or permanent restorative materials, fracture of the tooth structure, recurrent decay exposing the root canal filling material, or delay in placement of permanent restorations.

Persistent Intraradicular Infection

Microorganisms that can resist intracanal antimicrobial procedures and endure periods of nutrient deprivation in a prepared canal cause persistent intraradicular infections. This is also termed *recurrent infection.* Involved microorganisms are remnants of a primary or secondary infection. The microbiota associated with persistent infections is usually composed of fewer species than primary infections, and gram-positive facultative or anaerobic bacteria are predominant.[14-16] Fungi can also be found in frequencies significantly higher when compared with primary infections.[17]

Persistent and secondary infections are for the most part clinically indistinguishable and 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.

Extraradicular Infection

Extraradicular infection is characterized by microbial invasion of and proliferation in the inflamed periradicular tissues and is almost invariably a sequel to intraradicular infection. Extraradicular infection can be dependent on or independent of the intraradicular infection. The most common form of extraradicular infection dependent on the intraradicular infection is the acute apical abscess (Figure 3-3). The most common form of extraradicular infection that can be independent of the intraradicular infection is the apical actinomycosis (Figure 3-4, *A* and *B*).[18] The question as to whether the extraradicular infection is dependent on or independent of the intraradicular infection assumes special relevance from a therapeutic standpoint because the former can be successfully managed by root canal therapy, whereas the latter can only be treated by endodontic surgery.

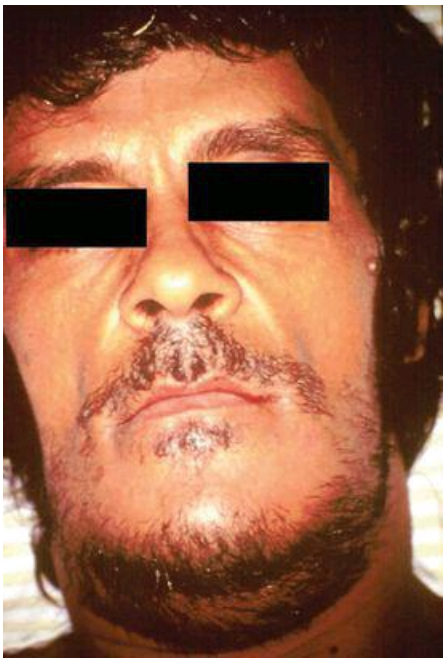

Figure 3-3 Acute apical abscess with severe swelling. Cases like this represent the most common form of extraradicular infection dependent on the intraradicular infection.

(Courtesy Dr. Henrique Martins.)

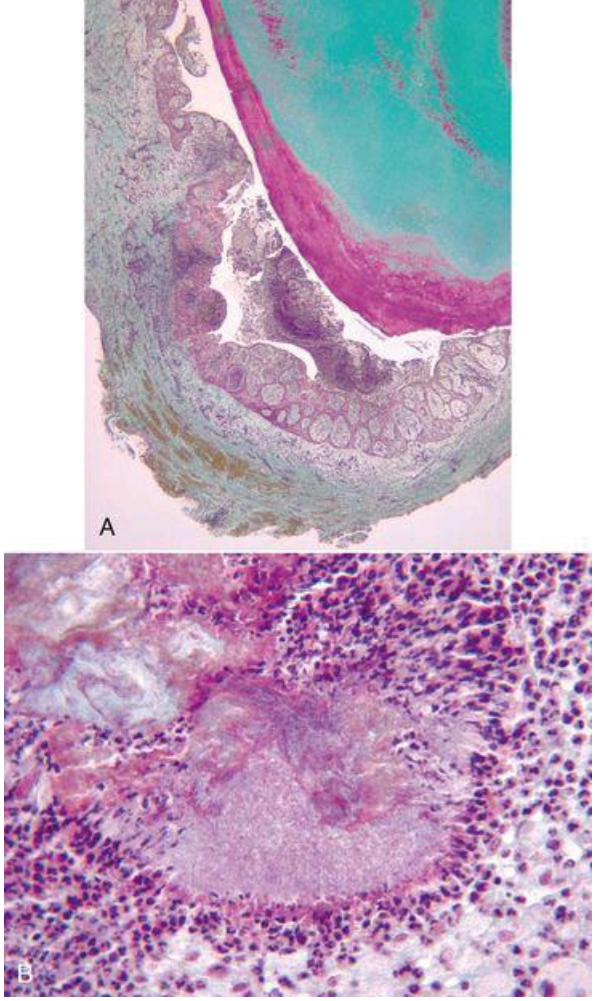

Figure 3-4 Apical actinomycosis. **A,** Bacterial aggregate in an epithelialized apical periodontitis lesion, suggestive of actinomycosis. **B,** Higher magnification of the actinomycotic aggregate, which is surrounded by inflammatory cells.

(Courtesy Dr. Domenico Ricucci.)

THE ENDODONTIC MICROBIOTA

Studies using culture-dependent approaches have allowed recognition of several candidate endodontic pathogens. More recently, with the advent of culture-independent molecular biology techniques, not only have the findings from culture studies been confirmed, but a great deal of new information has also been added to the knowledge of the microbiota associated with different types of endodontic infections. Molecular technology has enabled the recognition of new putative pathogens that had never been previously found in endodontic infections.[12] Moreover, many species that had already been considered as putative pathogens because of their frequencies as reported by culture-dependent methods have been found in similar or even in higher prevalence values by molecular approaches, strengthening their association with causation of apical periodontitis. As a consequence, the endodontic microbiota has been clearly redefined by molecular biology methods.

Primary Intraradicular Infections

Sophisticated culture and molecular biology techniques have revealed the polymicrobial nature of endodontic infections, with a conspicuous dominance of obligate anaerobic bacteria in primary infections. Current evidence reveals that endodontic bacteria fall into 8 of the 13 phyla that have oral representatives, namely *Firmicutes, Bacteroidetes, Spirochaetes, Fusobacteria, Actinobacteria, Proteobacteria, Synergistes,* and TM7.[19-21] Noteworthy is the high prevalence of as-yet-uncultivated species—about 40% to 55% of the endodontic microbiota are composed of bacteria that have yet to be cultivated and fully characterized.[19,20] In addition, bacterial profiles of the endodontic microbiota also vary from individual to individual,[19,22] suggesting that apical periodontitis has a heterogeneous etiology in which multiple bacterial combinations can play a role in disease causation. Table 3-1 shows the bacterial genera with common representatives in endodontic infections, and Figure 3-5 displays the most prevalent bacterial taxa found in primary intraradicular infections associated with different forms of apical periodontitis.

Table 3-1 Bacterial Genera Represented in Endodontic Infections

GRAM-NEGATIVE BACTERIA		GRAM-POSITIVE BACTERIA	
Anaerobes	**Facultatives**	**Anaerobes**	**Facultatives**
Rods		**Rods**	
Dialister	*Capnocytophaga*	*Actinomyces*	*Actinomyces*
Porphyromonas	*Eikenella*	*Pseudoramibacter*	*Corynebacterium*
Tannerella	*Haemophilus*	*Filifactor*	*Lactobacillus*
Prevotella		*Eubacterium*	
Fusobacterium		*Mogibacterium*	
Campylobacter		*Propionibacterium*	
Synergistes		*Eggerthella*	
Catonella		*Olsenella*	
Selenomonas		*Bifidobacterium*	
Centipeda		*Slackia*	
		Atopobium	
		Solobacterium	
		Lactobacillus	
Cocci		**Cocci**	
Veillonella	*Neisseria*	*Micromonas*	*Streptococcus*
Megasphaera		*Peptostreptococcus*	*Enterococcus*
		Finegoldia	*Granulicatella*
		Peptoniphilus	
		Anaerococcus	
		Streptococcus	
		Gemella	
Spirilla			
Treponema			

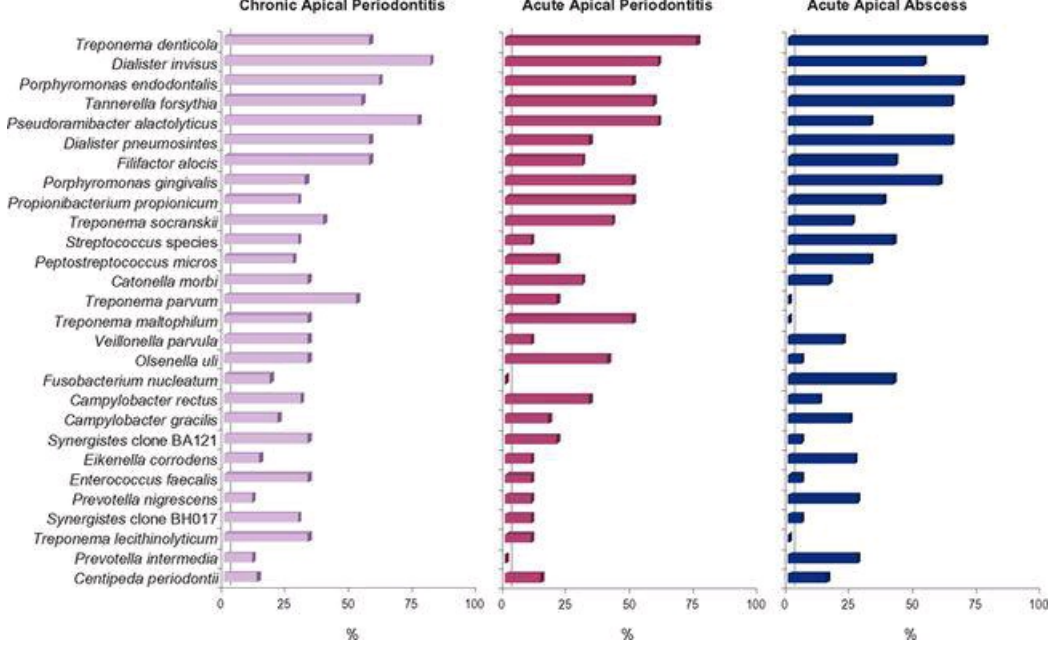

Figure 3-5 Prevalence of bacteria detected in primary endodontic infections of teeth with different forms of apical periodontitis. Compilation of data from authors' studies using a molecular biology technique.

Gram-Negative Bacteria

Gram-negative bacteria appear to be the most common microorganisms in primary endodontic infections. Species belonging to several genera of gram-negative bacteria have been consistently found in primary infections associated with different forms of apical periodontitis, including abscesses. These genera include *Dialister* (e.g., *D. invisus* and *D. pneumosintes*), *Treponema* (e.g., *T. denticola* and *T. socranskii*), *Fusobacterium* (e.g., *F. nucleatum*), *Porphyromonas* (e.g., *P. endodontalis* and *P. gingivalis*), *Prevotella* (e.g., *P. intermedia*, *P. nigrescens,* and *P. tannerae*), and *Tannerella* (e.g., *T. forsythia*).[19-21,23-41] Other gram-negative bacteria detected more sporadically in primary infections are shown in Table 3-1.

Gram-Positive Bacteria

Even though anaerobic gram-negative bacteria are reported to be the most common microorganisms in primary infections, several gram-positive bacteria have also been frequently detected in the endodontic mixed consortium, some of them in prevalence values as high as the most commonly found gram-negative species. The genera of gram-positive bacteria often found in primary infections include *Pseudoramibacter* (e.g., *P. alactolyticus*), *Filifactor* (e.g., *F. alocis*), *Micromonas* (e.g., *M. micros*), *Peptostreptococcus* (e.g., *P. anaerobius*), *Streptococcus* (e.g., *S. anginosus* group), *Actinomyces* (e.g., *A. israelii*), *Olsenella* (e.g., *O. uli*), and *Propionibacterium* (e.g., *P. propionicum and P. acnes*).[20,34,42-48] Other gram-positive bacteria found more sporadically in primary intraradicular infections are shown in Table 3-1.

As-Yet-Uncultivated Bacterial Phylotypes

Data from culture-independent molecular biology studies have indicated that several bacterial phylotypes can participate in endodontic infections. Phylotypes can be regarded as species that have not yet been cultivated and validly named and are known only by a 16S rRNA gene sequence. Uncultivated phylotypes belonging to the genera *Synergistes*, *Dialister, Prevotella*, *Solobacterium, Olsenella, Eubacterium*, and *Megasphaera,* as well as phylotypes related to the family *Lachnospiraceae* have been frequently detected in samples from primary endodontic infections.[19-21,32,49,50] These phylotypes are previously unrecognized bacteria that may play a role in the pathogenesis of apical periodontitis. The fact that they have yet to be cultivated and phenotypically characterized does not mean that they are not important.

Other Microorganisms in Endodontic Infections

Microorganisms other than bacteria have been occasionally found in endodontic infections. Fungi are eukaryotic microorganisms that have been only sporadically found in primary infections.[35,51] Archaea comprise a highly diverse group of prokaryotes, distinct from bacteria, with no known human pathogen. One study found methanogenic archaea in the canals of teeth with chronic apical periodontitis.[52] Viruses are not cells but inanimate particles that have no metabolism on their own. Because viruses require viable host cells to infect and replicate themselves, they cannot survive 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.[53] On the other hand, human cytomegalovirus and Epstein-Barr virus have been detected in apical periodontitis lesions,[54] where living host cells are abundant.

Symptomatic Infections

It has been suggested that the probability of symptoms is increased when certain bacterial species are part of the infective endodontic microbiota.[2,19,26,47] Nevertheless, the same species can be evenly distributed between symptomatic and asymptomatic cases.[23,24,27,29,55] This raises the suspicion that factors other than the mere presence of a given putative pathogenic species can influence the development of symptoms. These factors include differences in virulence ability among strains of the same species, number of occurring species and interactions among them resulting in additive or synergistic pathogenic effects, number of bacterial cells (load), environmental cues regulating expression of virulence factors, host resistance, and concomitant herpesvirus infection.[56] Association of some or all of these factors (instead of an isolated event) is likely to dictate the occurrence and intensity of symptoms.

Ecology of the Endodontic Microbiota

A root canal with necrotic pulp provides a space for bacterial colonization and provides bacteria a moist, warm, nutritious, and anaerobic environment, which is by and large protected from the host defenses because of lack of active microcirculation in the necrotic tissue. Intuitively, the necrotic root canal might be considered a rather fertile environment for bacterial growth and it might be realized that colonization should not be a difficult task for virtually every oral bacterial species. Although more than 700 different bacterial taxa have been reported to occur in the oral cavity and each individual's mouth can harbor about 100 to 200 taxa,[57] only a restricted assortment of these bacteria is found in an infected canal. This indicates that selective pressures must occur in the root canal system that favor the establishment of some species and inhibit others.[58] The key ecologic factors that influence the composition of the microbiota in the necrotic root canal include oxygen tension and redox potential, type and amount of available nutrients, and bacterial interactions.

Oxygen Tension and Redox Potential

The root canal infection is a dynamic process, and different bacterial species apparently dominate at different stages of the infectious process. In the very initial phases of the pulpal infectious process, facultative bacteria predominate.[59] After a few days or weeks, oxygen is depleted within the root canal as a result of pulp necrosis and consumption by facultative bacteria. An anaerobic milieu with consequent low redox potential develops, which is highly conducive to the survival and growth of obligate anaerobic bacteria. With the passage of time, anaerobic conditions become even more pronounced, particularly in the apical third of the root canal, and as a consequence, anaerobes will dominate the microbiota and outnumber facultative bacteria.

Available Nutrients

In the root canal system, bacteria can utilize the following as sources of nutrients: (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 in the root canal, and (4) products of the metabolism of other bacteria. Because the largest amount of nutrients is available in the main canal, which is the most voluminous part of the root canal system, most of the infecting microbiota, particularly fastidious anaerobic species, are expected to be located in this region. At later stages of the infection process, nutritional conditions favor the establishment of bacteria that metabolize peptides and amino acids.

Bacterial Interactions

The establishment of certain species in the root canal is also influenced by interactions with other species. Positive interactions (mutualism and commensalism) enhance the survival capacity of the interacting bacteria and increase the probability of certain species to coexist in the habitat. Negative interactions (competition and antagonism) limit population densities.

Patterns of Microbial Colonization

A better understanding of the disease process and the development of effective antimicrobial therapeutic strategies depend on the knowledge of the anatomy of infection (i.e., the way microbial cells are distributed throughout the infected tissue). Bacteria in the root canal system can exist as planktonic (unattached) cells suspended in the fluid phase of the main root canal and as

aggregates or coaggregates adhered to the root canal walls, sometimes forming multilayered biofilms.[60,61]

In long-standing root canal infections, microorganisms propagate in the entire root canal system. Lateral canals and isthmuses connecting main canals can be clogged with bacterial cells, primarily organized in biofilm structures.[62] Bacteria forming dense accumulations on the root canal walls are often seen penetrating the dentinal tubules (Figure 3-6). The diameter of dentinal tubules is large enough to permit penetration of most oral bacteria, and tubular infection is observed in most teeth, evincing apical periodontitis lesions. Although a shallow intratubular penetration is more common, bacterial cells can be observed reaching approximately 300 µm in some teeth (Figure 3-7).[61]

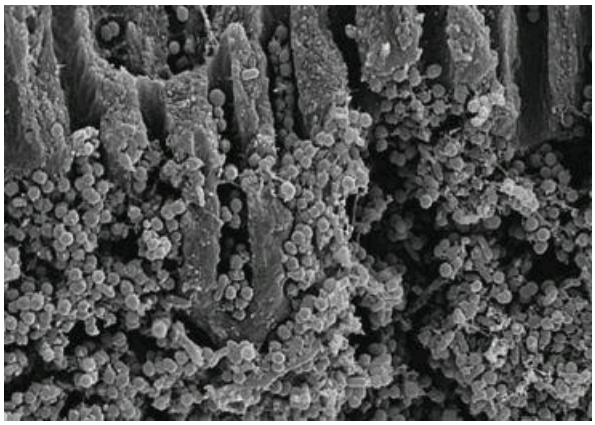

Figure 3-6 Heavy infection of the root canal walls mainly by cocci, but some small rods are also seen. Cocci are penetrating into dentinal tubules.

(From Siqueira JF, Jr., Rôças IN, Lopes HP: Oral Surg Oral Med Oral Pathol Oral Radiol Endod 93:174, 2002.)

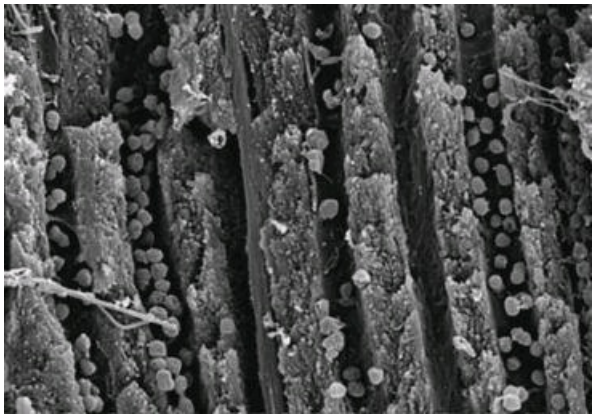

Figure 3-7 Cocci in dentinal tubules approximately 300 µm from the main root canal. Dividing cells are seen within tubules.

(From Siqueira JF, Jr., Rôças IN, Lopes HP: Oral Surg Oral Med Oral Pathol Oral Radiol Endod 93:174, 2002.)

Since the ideal outcome of the endodontic treatment relies on elimination of the cause of apical periodontitis, effective antimicrobial strategies to eradicate endodontic infections should take into account the patterns of microbial colonization. Microorganisms present as planktonic cells in the main root canal can be easily accessed and eliminated by instruments and substances used during treatment. On the other hand, microorganisms present in biofilms adhered to the canal walls or located into isthmuses, lateral canals, and dentinal tubules are indisputably more difficult to be eliminated and may require special therapeutic strategies.

Persistent/Secondary Endodontic Infections

Most root canal–treated teeth with persistent apical periodontitis lesions have been demonstrated to harbor an intraradicular infection.[15,16,63,64] Microorganisms present in root canal–treated teeth can be "persisters" that survived the effects of intracanal disinfection procedures and were present in the canal at the root canal–filling stage (persistent intraradicular infection) or they can have infected the canal after filling as a result of coronal leakage (secondary intraradicular infection). In fact, there is an increased risk of adverse treatment outcome when microorganisms are present in the canal at the time of filling.[65,66] For residual microorganisms to be the cause of persistent apical periodontitis lesions they have to adapt to the modified environment induced by treatment, acquire nutrients, survive the antimicrobial effects of filling materials, reach critical numbers and exhibit virulence attributes sufficient to sustain periradicular inflammation, and have unrestrained access to the periradicular tissues to exert their pathogenicity.

Bacteria at the Root Canal–Filling Stage

Diligent antimicrobial treatment can occasionally fail to totally eradicate bacteria from root canals, with consequent selection of the most resistant segment of the microbiota. Gram-negative bacteria, which are common members of primary intraradicular infections, are usually eliminated after endodontic treatment. Most studies on this subject have clearly revealed a higher occurrence of gram-positive bacteria (e.g., streptococci, lactobacilli, *Enterococcus faecalis*, *O. uli*, *M. micros*, *P. alactolyticus,* and *Propionibacterium* species) in both post-instrumentation and post-medication samples.[66-70] 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 canals.

Microbiota in Root Canal–Treated Teeth

The microbiota in root canal–treated teeth with persistent apical periodontitis lesions is composed of a more restricted group of microbial species in comparison to primary infections, with a mean number of one to three species per canal.[15,16] *E. faecalis* is a facultative anaerobic gram-positive coccus that has been frequently found in root canal–treated teeth in prevalence values ranging from 30% to 90% of the cases.[14-16,64,71,72] Root canal–treated teeth are about 9 times more likely to harbor *E. faecalis* than cases of primary infections.[71] *Candida* species are fungi only sporadically found in primary infections, but detection frequencies in persistent/secondary infections range from 3% to 18% of the cases.[14-16,51,73,74] Both *E. faecalis* and *C. albicans* possess a series of attributes that may allow them to survive in treated canals, including resistance to intracanal medications and ability to form biofilms, invade dentinal tubules, and endure long periods of nutrient deprivation.[75-79]

Streptococcus species and some anaerobic species commonly found in primary infections, such as *P. alactolyticus, P. propionicum*, *F. alocis*, *T. forsythia, D. pneumosintes,* and *D. invisus*, can also be involved in persistent/secondary intraradicular infections (Table 3-2).[15,16,21,64]

Table 3-2 Microorganisms Detected in Root Canal–Treated Teeth Associated with Persistent Apical Periodontitis

Taxa	Frequency (%)*
Enterococcus faecalis	77
Pseudoramibacter alactolyticus	55
Propionibacterium propionicum	50
Filifactor alocis	48
Dialister pneumosintes	46
Streptococcus spp.	23
Tannerella forsythia	23
Dialister invisus	14
Campylobacter rectus	14
Porphyromonas gingivalis	14
Treponema denticola	14
Fusobacterium nucleatum	10
Prevotella intermedia	10
Candida albicans	9
Campylobacter gracilis	5
Actinomyces radicidentis	5
Porphyromonas endodontalis	5
Micromonas micros	5
Synergistes oral clone BA121	5
Olsenella uli	5

* Percent of cases harboring each taxon.

Data from Siqueira JF, Jr., Rôças IN: Oral Surg Oral Med Oral Pathol Oral Radiol Endod 97:85, 2004; Siqueira JF, Jr., Rôças IN: J Clin Microbiol 43:3314, 2005.

Extraradicular Infections

Apical periodontitis is formed in response to intraradicular infection, and in most situations, it succeeds in preventing microorganisms from gaining access to 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. There is, however, another form of extraradicular infection that, unlike the acute abscess, is usually characterized by absence of overt symptoms. This condition encompasses the establishment of microorganisms in the periradicular tissues, either by adherence to the apical external root surface in the form of biofilm structures[80] or by formation of cohesive actinomycotic colonies within the body of the inflammatory lesion.[81] Extraradicular microorganisms have been discussed as one of the causes of persistence of apical periodontitis lesions in spite of diligent root canal treatment.[82]

Conceivably, the extraradicular infection can depend on or be independent of the intraradicular infection.[18] For example, the presence of a sinus tract (fistula) usually indicates the extraradicular occurrence of bacteria. The fact that most sinus tracts close after proper root canal treatment suggests an extraradicular infection fostered by and dependent on the intraradicular infection. Also, 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. Apical actinomycosis is a pathologic entity caused by some *Actinomyces* species and *P. propionicum* and is the main example of an extraradicular infection independent of the intraradicular infection. Because of this independence, apical actinomycosis is successfully treated only by periradicular surgery.[18,83]

Except for apical actinomycosis and cases evincing a sinus tract, it is still controversial whether asymptomatic apical periodontitis lesions can harbor bacteria for very long beyond initial tissue invasion. The incidence of extraradicular infections in untreated teeth is reportedly low,[60,84] which is congruent with the high success rate of nonsurgical root canal treatment.[85] Even in root canal–treated teeth with recalcitrant lesions, in which a higher incidence of extraradicular bacteria has been reported, a high rate of healing after retreatment[85] indicates that the major cause of posttreatment disease is located within the root canal system, characterizing a persistent or secondary intraradicular infection.[14-16,64] Based on this, one can assume that most of the extraradicular infections observed in root canal–treated teeth are indeed fostered by an intraradicular infection.

Chapter Review Questions available in Appendix B or on the DVD

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Pulp and Periapical Pathosis

Mahmoud Torabinejad, Shahrokh Shabahang

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Identify etiologic factors causing pulp inflammation.
2. Explain the mechanism of spread of inflammation in the pulp.
3. Explain why it is difficult for the pulp to recover from severe injury.
4. List specific and nonspecific mediators of pulpal inflammation.
5. Classify pulpal diseases and their clinical and histologic features.
6. Describe the mechanisms and explain the consequences of the spread of pulpal inflammation into periradicular tissues and the subsequent inflammatory and immunologic responses.

7. Classify periradicular lesions of pulpal origin.

8. Identify and distinguish between histologic features and clinical signs and symptoms of acute apical periodontitis, chronic apical periodontitis, acute and chronic apical abscesses (suppurative apical periodontitis), and condensing osteitis.

9. Describe the steps involved in repair of periradicular pathosis after successful root canal treatment.

10. Identify and describe, in general, nonendodontic pathologic lesions that may mimic endodontic periradicular pathosis.

IRRITANTS

Irritation of pulpal or periradicular tissues can result in inflammation. The major irritants of these tissues can be divided into those that are living and nonliving. The living irritants are various microorganisms and viruses. The nonliving irritants include mechanical, thermal, and chemical irritants.

Microbial Irritants

Microorganisms present in dental caries are the main sources of irritation of the dental pulp and periradicular tissues. Carious dentin and enamel contain numerous species of bacteria such as *Streptococcus mutans,* lactobacilli, and *Actinomyces.*[1] The population of microorganisms decreases to few or none in the deepest layers of carious dentin.[2] However, direct pulp exposure to microorganisms is not a prerequisite for pulpal response and inflammation. Microorganisms in caries produce toxins that penetrate into the pulp through tubules. Studies have shown that even small lesions in enamel are capable of attracting inflammatory cells in the pulp.[3,4] In response to the presence of microorganisms and their byproducts in dentin, the pulp is infiltrated locally (at the base of tubules involved in caries), primarily by chronic inflammatory cells such as macrophages, lymphocytes, and plasma cells.[5] As the decay progresses toward the pulp, the intensity and character of the infiltrate change.

When actual exposure occurs, the pulp tissue is infiltrated locally by polymorphonuclear (PMN) leukocytes to form an area of liquefaction necrosis at the site of exposure (Figure 4-1).[6] After pulp exposure, bacteria colonize and persist at the site of necrosis. Pulpal tissue may remain inflamed for long periods of time and may undergo eventual or rapid necrosis. This depends on several factors: (1) the virulence of bacteria, (2) the ability to release inflammatory fluids to avoid a marked increase in intrapulpal pressure, (3) the host resistance, (4) the amount of circulation, and most importantly, (5) the lymph drainage. Yamasaki and associates created pulp exposures in rats and showed that necrosis extended gradually from the upper portion of the pulp to the apical portions. A periapical lesion ensued after pulpal inflammation and necrosis. The lesions extended first horizontally and then vertically before their expansion ceased.[7]

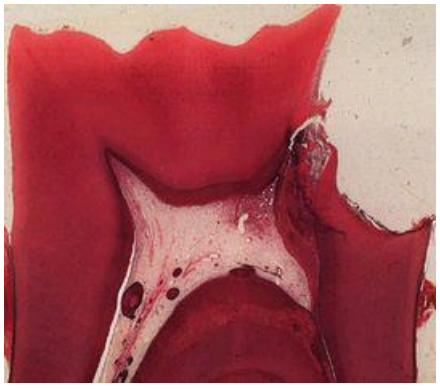

Figure 4-1 A localized inflammatory reaction containing mainly polymorphonuclear leukocytes at the site of a carious pulpal exposure. The remainder of the coronal pulp is almost free of inflammatory cells.

(Courtesy Dr. J.H. Simon.)

As a consequence of exposure to the oral cavity and to caries, pulp harbors bacteria and their byproducts. The dental pulp usually cannot eliminate these damaging irritants. At best, defenses temporarily impede the spread of infection and tissue destruction. If the irritants persist, the ensuing damage will become extensive and will spread throughout the pulp. Subsequently, bacteria, or their byproducts, and other irritants from the necrotic pulp will diffuse from the canal periapically, resulting in the development of inflammatory lesions (Figure 4-2).

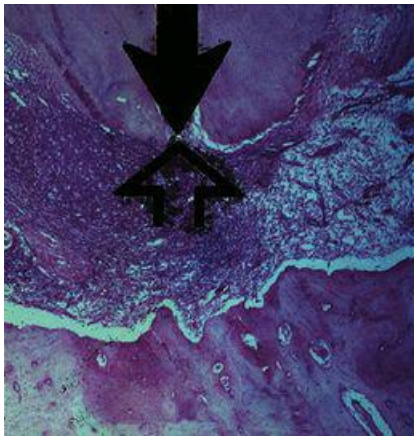

Figure 4-2 Egress of irritants *(closed arrow)* from the root canal into the periapical tissue causes inflammation *(open arrow)* and replacement of normal periapical structures with a granulomatous tissue.

Bacteria play an important role in the pathogenesis of pulpal and periradicular pathoses. A number of investigations have established that pulpal or periradicular pathosis does not develop without the presence of bacterial contamination.[8-10]

Kakehashi and associates[8] created pulp exposures in conventional and germ-free rats. This procedure in the germ-free rats caused only minimal inflammation throughout the 72-day investigation. Pulpal tissue in these animals was not completely devitalized but rather showed calcific bridge formation by day 14, with normal tissue apical to the dentin bridge (Figure 4-3, *A*). In contrast, autoinfection, pulpal necrosis, and abscess formation occurred by the eighth day in conventional rats (Figure 4-3, *B*). Other investigators have examined the importance of bacteria in the development of periradicular lesions by sealing noninfected and infected pulps in the root canals of monkeys.[9] After 6 to 7 months, clinical, radiographic, and histologic examinations of teeth sealed with noninfected pulps showed an absence of pathosis in apical tissues, whereas teeth sealed with necrotic pulps containing certain bacteria showed periapical inflammation. The bacteriologic investigation by Sundqvist[10] examining the flora of human necrotic pulps supports the findings of Kakehashi and associates,[8] as well as Moller and coworkers.[9] These studies examined previously traumatized teeth with necrotic pulps, with and without apical pathosis. Teeth without apical lesions were aseptic, whereas those with periapical pathosis had positive bacterial cultures.

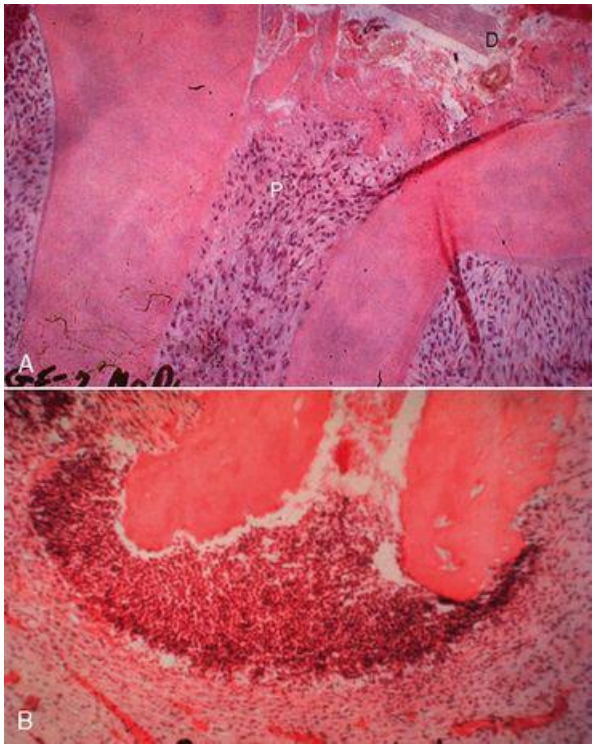

Figure 4-3 A, No inflammation is seen in an exposed pulp *(P)* of a germ-free rat. Food particles and other debris *(D)* are packed into the chamber. **B,** Periapical lesion is apparent in a conventional rat after pulp exposure.

(Courtesy Dr. H. Stanley.)

Recent studies have shown a positive correlation between the presence of certain viruses and symptomatic apical pathoses.[11] In fact, periapical lesions that contain *Cytomegalovirus* and Epstein-Barr virus are more likely to be symptomatic than lesions that do not yield these viruses. While a direct etiologic role has been suggested by some investigators,[12] this cause and effect relationship has yet to be demonstrated in experimental models.

Mechanical Irritants

In addition to bacterial irritation, pulp or periradicular tissues can also be irritated mechanically. Deep cavity preparations, removal of tooth structure without proper cooling, impact trauma, occlusal trauma, deep periodontal curettage, and orthodontic movement of teeth are the main thermal and physical irritants of the pulp tissue. As changes to the underlying pulp (such as aspiration of odontoblasts into the dentinal tubules) are generally transient in nature, a completely healthy pulp can potentially recover from this injury. Unfortunately, pulps associated with teeth that have undergone extensive restorative procedures are generally inflamed. If proper precautions are not taken, cavity or crown preparations may damage subjacent odontoblasts (Figure 4-4). The number of tubules per unit of surface area and their diameter increase closer to the pulp. As a result, dentinal permeability is greater closer to the pulp than near the dentinoenamel junction (DEJ) or cementodentinal junction (CDJ).[13] Therefore the potential for pulp irritation increases as more dentin is removed (i.e., as cavity preparation deepens). Pulp damage is roughly proportional to the amount of tooth structure removed, as well as to the depth of removal.[14] Also, operative procedures without water coolant cause more irritation than those performed under water spray.[15] A study of the reactions and vascular changes occurring in experimentally induced acute and chronic pulpitis demonstrated increased permeability and dilation of blood vessels in the early stages of pulpitis.[16]

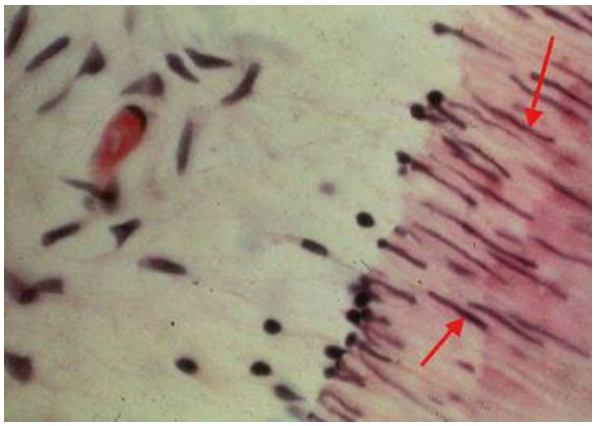

Figure 4-4 Crown preparation through enamel and into 1 mm of dentin resulted in aspiration of odontoblasts *(arrows)* into the tubules and infiltration of the pulp by PMN leukocytes and lymphocytes. The specimen was taken 48 hours after crown preparation.

Impact injuries with or without crown or root fractures may cause pulpal damage (see Chapter 11). The severity of trauma and degree of apical closure of the root are important factors in the pulp's ability to recover. Teeth undergoing mild to moderate trauma and those with immature apices have a better chance of pulpal survival compared with those suffering severe injury or those with closed apices. Application of forces beyond the physiologic tolerance of the periodontal ligament (PDL) during orthodontic tooth movement results in disturbance of the blood and nerve supply of the pulp tissue.[17,18] The resulting changes include atrophy of cells and alteration of nerve axons. In addition, orthodontic movement may initiate resorption of the apex, usually without a change in vitality. Deep scaling and curettage may injure apical vessels and nerves, resulting in pulpal damage (see Chapter 6).

Periradicular tissues can be mechanically irritated and inflamed by impact trauma, hyperocclusion, endodontic procedures and accidents, pulp extirpation, overinstrumentation of root canals, perforation of the root, and overextension of the root canal filling materials. Mechanical irritation by instruments may occur during canal preparation. Inaccurate determination of canal length is usually the cause of overinstrumentation and the subsequent inflammation. In addition, lack of an adequate apical resistance form created during cleaning and shaping can cause overextension of filling materials into the periapical tissues, causing physical and chemical damage (Figure 4-5).

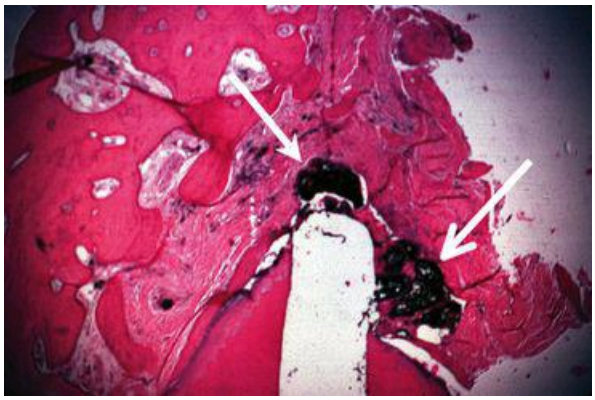

Figure 4-5 Improper instrumentation and extrusion of filling materials into the periapical tissues causing periradicular inflammation *(arrows)*.

Chemical Irritants

Chemical irritants of the pulp include various dentin cleansing, sterilizing, and desensitizing substances, as well as some of the substances present in temporary and permanent restorative materials and cavity liners. Antibacterial agents, such as silver nitrate, phenol with and without camphor, and eugenol, have been used in an attempt to “sterilize” dentin after cavity preparations. However, their effectiveness as dentin sterilizers is questionable,[19] and their cytotoxicity can cause inflammatory changes in the underlying dental pulp.[20] Other irritating agents include cavity cleansers, such as alcohol, chloroform, hydrogen peroxide, and various acids; chemicals present in desensitizers; cavity liners and bases; and temporary and permanent restorative materials.

Antibacterial irrigants used during cleaning and shaping of root canals, intracanal medications, and some compounds present in obturating materials are examples of potential chemical irritants of periradicular tissues. Most irrigants and medicaments are toxic and are not biocompatible.[21,22]

PULPAL PATHOSIS

Apart from anatomic configuration and diversity of inflicted irritants, pulp reacts to these irritants as do other connective tissues. Pulpal injury results in cell death and inflammation. The degree of inflammation is proportional to the intensity and severity of tissue damage. Slight injuries, such as incipient caries or shallow cavity preparations, cause little or no inflammation in the pulp. In contrast, deep caries, extensive operative procedures, or persistent irritants usually produce more severe inflammatory changes. Depending on the severity and duration of the insult and the host capacity to respond, the pulpal response ranges from transient inflammation (reversible pulpitis) to irreversible pulpitis and then to total necrosis. These changes often occur without pain and without the knowledge of the patient or dentist.

Inflammatory Process

Irritation of the dental pulp results in the activation of a variety of biologic systems such as nonspecific inflammatory reactions mediated by histamine, bradykinin, and arachidonic acid metabolites.[23] Also released are PMN lysosomal granule products (elastase, cathepsin G, and lactoferrin),[24] protease inhibitors such as antitrypsin,[25] and neuropeptides such as calcitonin gene–related peptide (CGRP) and substance P (SP).[26]

Unlike the connective tissues in other parts of the body, normal and healthy dental pulps lack mast cells. However, these cells are found in inflamed pulps (Figure 4-6).[27] Mast cells contain histamine, leukotrienes, and platelet-activating factors. Physical injury to mast cells, or the bridging of two immunoglobulin E (IgE) molecules by an antigen on their cell surfaces, results in the release of histamine and/or other bioactive substances present in mast cell granules. The presence of histamine in the blood vessel walls and a marked increase in histamine levels indicate the importance of histamine in pulpal inflammation.[28]

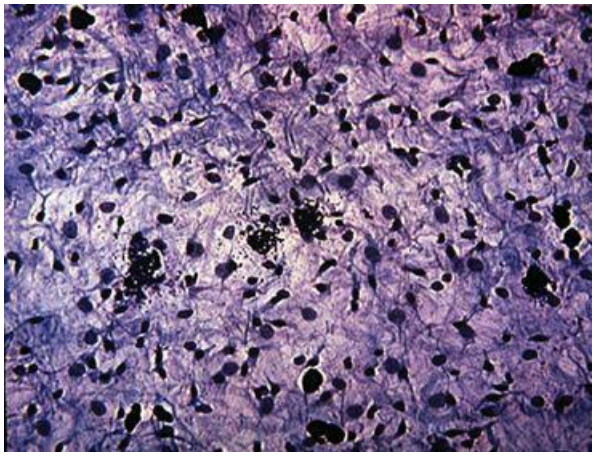

Figure 4-6 Mast cells are readily visible as dark-stained cells in this inflamed human dental pulp.

Kinins, which cause many signs and symptoms of acute inflammation, are produced when plasma or tissue kallikreins contact kininogens. Bradykinin, SP, and neurokinin A have been identified in dental pulp tissue using high-performance liquid chromatography.[29] In an in vitro study, bradykinin evoked immunoreactive CGRP (iCGRP) release from bovine dental pulps[35]; this activity is enhanced by prostaglandin E_2 (PGE_2).[30] As a result of cellular damage, phospholipase A_2 causes release of arachidonic acid from cell membranes. Metabolism of arachidonic acid results in the formation of prostaglandins, thromboxanes, and leukotrienes. Various arachidonic acid metabolites have been found in experimentally induced pulpitis.[31] The presence of these metabolites in inflamed pulps[32] indicates that arachidonic acid metabolites participate in inflammatory reactions of the dental pulp.

The dental pulp is densely innervated with sensory fibers containing immunomodulatory neuropeptides such as SP and CGRP. Studies have shown that denervation of the rat molar pulp, caused by axotomy of the inferior alveolar nerve, results in increased pulp tissue damage and a diminished infiltration of immunocompetent cells.[5] These findings indicate that pulpal nerves are protective in nature and that they may be involved in the recruitment of inflammatory and immunocompetent cells to the injured pulp.[5]

Mild to moderate pulpal injuries result in the sprouting of sensory nerves with an increase in iCGRP.[26,33] However, severe injuries have the opposite effect, resulting in either reduction or elimination of iCGRP and SP.[33] These experiments indicate that pulpal neuropeptides undergo dynamic changes after injury. In addition, recent studies have shown that stimulation of the dental pulp by caries results in the formation of various interleukins and recruitment of inflammatory cells to the site of injury.[34-36]

Immunologic Responses

In addition to nonspecific inflammatory reactions, immune responses also may initiate and perpetuate deleterious pulpal changes.[23] Potential antigens include bacteria and their byproducts within dental caries, which directly (or via the dentinal tubules) can initiate various types of reactions. Normal and uninflamed dental pulps contain immunocompetent cells such as T and B (fewer) lymphocytes, macrophages, and a substantial number of class II molecule-expressing dendritic cells, which are morphologically similar to macrophages.[5] Elevated levels of immunoglobulins in inflamed pulps (Figure 4-7) show that these factors participate in the defense mechanisms involved in protection of this tissue.[37] Arthus-type reactions do occur in the dental pulp.[38] In addition, the presence of immunocompetent cells such as T lymphocytes, macrophages, and class II molecule-expressing cells appearing as dendritic cells (Figure 4-8) in inflamed pulps indicates that delayed hypersensitivity reactions can also occur in this tissue.[5] Despite their protective mechanisms, immune reactions in the pulp can result in the formation of small necrotic foci and eventual total pulpal necrosis.

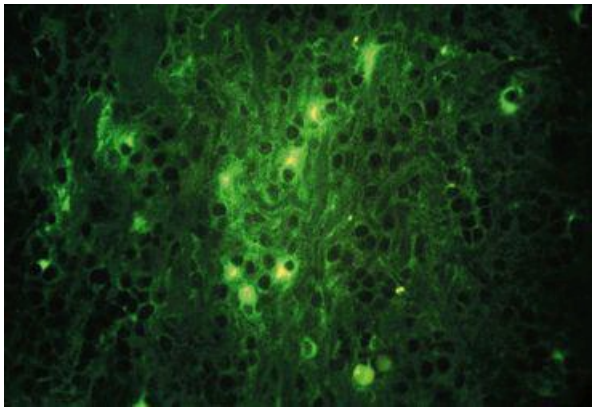

Figure 4-7 Some plasma cells stain positively for IgM in inflamed human dental pulp, indicating immunologic activity.

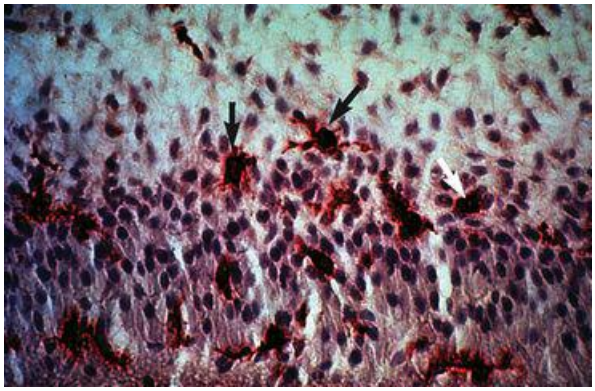

Figure 4-8 Many dendritic cells *(arrows)* are present in an inflamed dental pulp.

(Courtesy Dr. M. Jontell.)

Lesion Progression

Mild injuries may not result in significant pulpal changes. However, moderate to severe injuries to the pulp result in localized inflammation[39] and the release of a high concentration of inflammatory mediators. An increase in protease inhibitors in moderately to severely inflamed pulps indicates the presence of natural modifiers.[25] As a consequence of the release of a large quantity of inflammatory mediators, increased vascular permeability, vascular stasis, and migration of leukocytes to the site of injury occur. Current research data show that the sensory neuropeptide, CGRP, is responsible for the increase in blood flow during pulpal inflammation.[40]

Elevated capillary pressure and increased capillary permeability move fluids from blood vessels into the surrounding tissues. If removal of fluids by venules and lymphatics does not coincide with the filtration of capillaries, an exudate forms. Pulp is encased in rigid surrounding tissues, forming a low-compliance system; therefore a small increase in tissue pressure causes passive compression and even complete collapse of the venules at the site of pulpal injury.[41] Pressure increases occur in small "compartmentalized" regions and progress slowly (see Figure 4-1). Therefore the dental pulp does not degenerate by extensive increases in pressure with subsequent strangulation.[41,42]

Pain is often caused by several factors. The release of mediators of inflammation causes pain *directly* by lowering the sensory nerve threshold. These substances also cause pain *indirectly* by increasing both vasodilation in arterioles and vascular permeability in venules, resulting in edema and elevation of tissue pressure. This pressure acts directly on sensory nerve receptors.

Increased tissue pressure, the inability of the pulp to expand, and the lack of collateral circulation may result in pulpal necrosis and the development of subsequent periradicular pathosis.

CLASSIFICATION OF PULPAL DISEASES

Because there is little or no correlation between the histologic findings of pulpal pathosis and clinical symptoms,[43] the diagnosis and classification of pulpal diseases are based on clinical signs and symptoms rather than histopathologic findings. Pulpal conditions can be classified as normal pulp, reversible and irreversible pulpitis, hyperplastic pulpitis, necrosis, and previously treated pulp. Hard tissue responses include calcification and resorption.

Normal Pulp

A tooth with a normal pulp is clinically symptom free and responds normally to vitality tests. Such a tooth does not reveal any radiographic signs of pathosis.

Reversible Pulpitis

By definition, reversible pulpitis is a clinical condition associated with subjective and objective findings indicating presence of mild inflammation in the pulp tissue. If the cause is eliminated, inflammation will reverse and the pulp will return to its normal state.

Mild or short-acting stimuli such as incipient caries, cervical erosion, or occlusal attrition; most operative procedures; deep periodontal curettage; and enamel fractures resulting in exposure of dentinal tubules can cause reversible pulpitis.

Symptoms

Reversible pulpitis is usually asymptomatic. However, when present, symptoms usually follow a particular pattern. Application of stimuli, such as cold or hot liquids, as well as air, may produce sharp, transient pain. Removal of these stimuli, which do not normally produce pain or discomfort, results in immediate relief. Cold and hot stimuli produce different pain responses in normal pulp.[44] When heat is applied to teeth with uninflamed pulp, the initial response is delayed; the intensity of pain increases as the temperature rises. In contrast, pain in response to cold in normal pulp is immediate; the intensity tends to decrease if the cold stimulus is maintained. Based on these observations, pulpal responses in both health and disease apparently result largely from changes in in-trapulpal pressures.

Treatment

The removal of irritants and sealing and insulating the exposed dentin or vital pulp usually results in diminished symptoms and reversal of the inflammatory process in the pulp tissue (Figure 4-9). However, if irritation of the pulp continues or increases in intensity for reasons stated earlier, moderate to severe inflammation develops with resultant irreversible pulpitis and eventually pulpal necrosis.

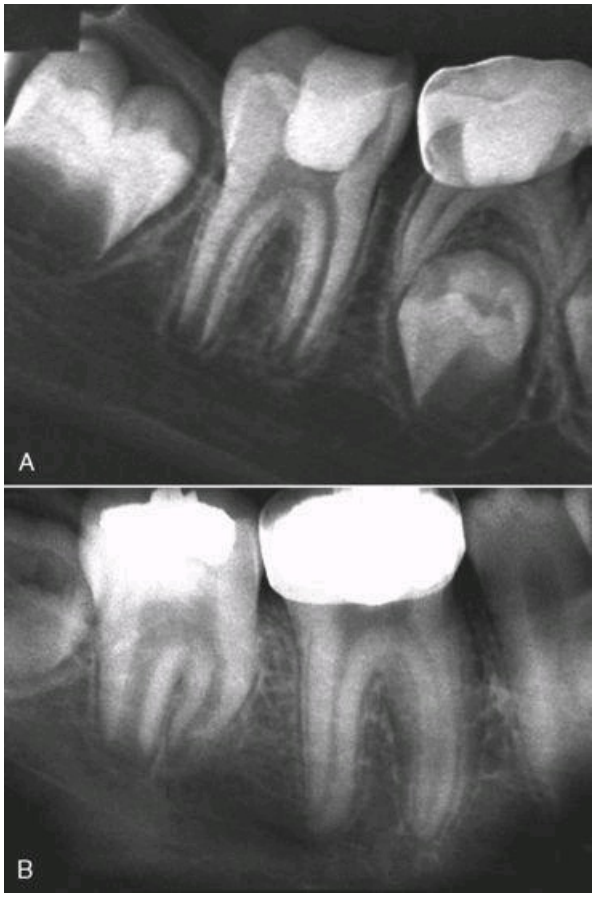

Figure 4-9 A, Mechanically exposed pulp horns of a mandibular molar with signs of reversible pulpitis were capped with mineral trioxide aggregate. **B,** On recall, a follow-up radiograph 5 years later shows no calcific metamorphosis in the pulp chamber, closure of apexes, and the presence of normal responses during clinical examination.

Irreversible Pulpitis

Irreversible pulpitis may be classified as symptomatic or asymptomatic. It is a clinical condition associated with subjective and objective findings indicating presence of severe inflammation in the pulp tissue. Irreversible pulpitis is often a sequel to and a progression from reversible pulpitis. Severe pulpal damage from extensive dentin removal during operative procedures or impairment of pulpal blood flow as a result of trauma or orthodontic movement of teeth may also cause irreversible pulpitis. Irreversible pulpitis is a severe inflammatory process that will not resolve even if the cause is removed. The pulp is incapable of healing and slowly or rapidly becomes necrotic. Irreversible pulpitis can be symptomatic with spontaneous and lingering pain. It can also be asymptomatic with no clinical signs and symptoms.

Symptoms

Irreversible pulpitis is usually asymptomatic. Patients may, however, report mild symptoms. Irreversible pulpitis may also be associated with intermittent or continuous episodes of spontaneous pain (with no external stimuli). Pain resulting from an irreversibly inflamed pulp may be sharp, dull, localized, or diffuse and can last anywhere from a few minutes up to a few hours. Localization of pulpal pain is more difficult than localization of periradicular pain and becomes more difficult as the pain intensifies. Application of external stimuli, such as cold or heat, may result in prolonged pain.

Accordingly, in the presence of severe pain, pulpal responses differ from those of uninflamed teeth or teeth with reversible pulpitis. For example, application of heat to teeth with irreversible pulpitis may produce an immediate response; also, occasionally with the application of cold, the response does not disappear and is prolonged. Application of cold in patients with painful irreversible pulpitis may cause vasoconstriction, a drop in pulpal pressure, and subsequent pain relief. Although it has been claimed that teeth with irreversible pulpitis have lower thresholds to electrical stimulation, Mumford found similar pain perception thresholds in inflamed and uninflamed pulps.[45]

Tests and Treatment

If inflammation is confined to the pulp and has not extended periapically, teeth respond within normal limits to palpation and percussion. The extension of inflammation to the PDL causes percussion sensitivity and allows better localization of pain. Root canal treatment or extraction is indicated for teeth with signs and symptoms of irreversible pulpitis.

Hyperplastic Pulpitis

Hyperplastic pulpitis (pulp polyp) is a form of irreversible pulpitis that originates from overgrowth of a chronically inflamed young pulp onto the occlusal surface. It is usually found in carious crowns of young patients (Figure 4-10, *A*). Ample vascularity of the young pulp, adequate exposure for drainage, and tissue proliferation are associated with the formation of hyperplastic pulpitis. Histologic examination of hyperplastic pulps shows surface epithelium overlying the inflamed connective tissue (Figure 4-10, *B*). Cells of the oral epithelium are implanted and grow over the exposed surface to form an epithelial covering.

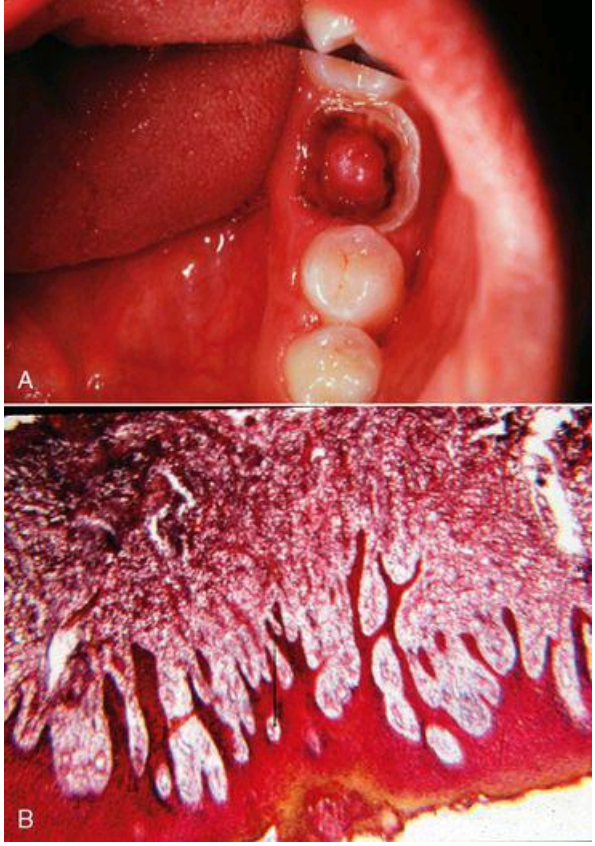

Figure 4-10 A, Pulp polyp is also known as hyperplastic pulpitis. The involved tooth is usually carious with extensive loss of tooth structure and the pulp remains vital and proliferates from the exposure site. **B,** Histologic examination of hyperplastic pulpitis shows surface epithelium and underlying inflamed connective tissue.

Hyperplastic pulpitis is usually asymptomatic. It appears as a reddish cauliflower-like outgrowth of connective tissue into caries that has resulted in a large occlusal exposure. It is occasionally associated with clinical signs of irreversible pulpitis, such as spontaneous pain, as well as lingering pain to cold and heat stimuli. The threshold to electrical stimulation is similar to that found in normal pulps. These teeth respond within normal limits when palpated or percussed. Hyperplastic pulpitis can be treated by pulpotomy, root canal treatment, or extraction.

Hard Tissue Changes Caused by Pulpal Inflammation

As a result of irritation, two distinct hard tissue changes may occur: calcification or resorption.

Pulp Calcification

Extensive calcification (usually in the form of pulp stones or diffuse calcification) occurs as a response to trauma, caries, periodontal disease, or other irritants. Thrombi in blood vessels and collagen sheaths around vessel walls are possible sources for these calcifications.

Another type of calcification is the extensive formation of hard tissue on dentin walls, often in response to irritation or death and replacement of odontoblasts. This process is called *calcific metamorphosis* (Figure 4-11). As irritation increases, the amount of calcification may also increase, leading to partial or complete radiographic (but not histologic) obliteration of the pulp chamber and root canal.[46] A yellowish discoloration of the crown is often a manifestation of calcific metamorphosis. The pain threshold to thermal and electrical stimuli usually increases; often the teeth are unresponsive.

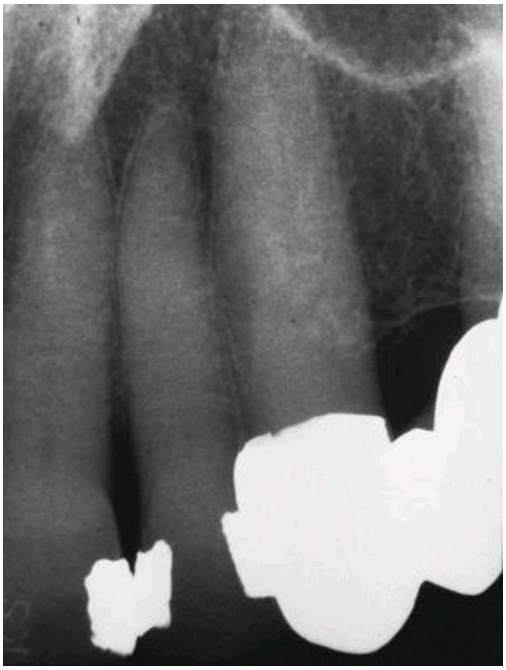

Figure 4-11 Calcific metamorphosis does not represent pathosis per se and may occur with aging or low-grade irritation.

Palpation and percussion are usually within normal limits. In contrast to soft tissue diseases of the pulp, which have no radiographic signs and symptoms, calcification of pulp tissue is associated with various degrees of pulp space obliteration. A reduction in coronal pulp space followed by a gradual narrowing of the root canal is the first sign of calcific metamorphosis. This condition is not pathologic in nature and does not require treatment.

Internal (Intracanal) Resorption

Inflammation in the pulp may initiate resorption of adjacent hard tissues. The pulp is transformed into a vascularized inflammatory tissue with dentinoclastic activity; this condition leads to the resorption of the dentinal walls, advancing from its center to the periphery.[47] Most cases of intracanal resorption are asymptomatic. Advanced internal resorption involving the pulp chamber is often associated with pink spots in the crown.

Teeth with intracanal resorptive lesions usually respond within normal limits to pulpal and periapical tests. Radiographs reveal presence of radiolucency with irregular enlargement of the root canal compartment (Figure 4-12). Immediate removal of the inflamed tissue and completion of root canal treatment are recommended; these lesions tend to be progressive and eventually perforate to the lateral periodontium. When this occurs, pulp necrosis ensues and treatment of the tooth becomes more difficult.

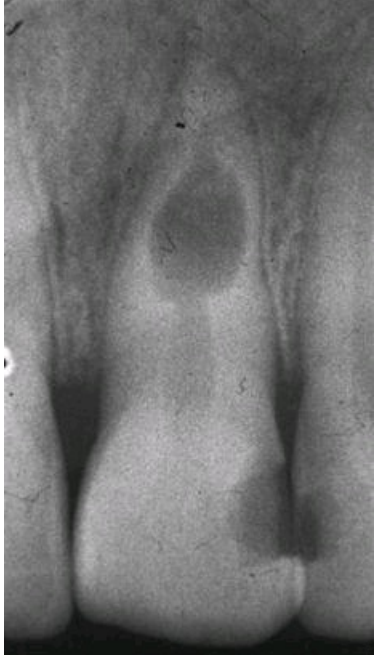

Figure 4-12 Hard tissue resorption that causes disappearance of normal radiographic evidence of the root canal *usually* indicates an internal resorption defect.

Pulpal Necrosis

As stated before, pulp is encased in rigid walls, it has no collateral blood circulation, and its venules and lymphatics collapse under increased tissue pressure. Therefore irreversible pulpitis leads to liquefaction necrosis. If exudate produced during irreversible pulpitis is absorbed or drains through caries or through a pulp exposure into the oral cavity, necrosis is delayed; the radicular pulp may remain vital for long periods of time. In contrast, closure or sealing of an inflamed pulp induces rapid and total pulpal necrosis and periradicular pathosis.[48] In addition to liquefaction necrosis, ischemic necrosis of the pulp occurs as a result of traumatic injury from disruption of the blood supply. Necrotic pulp is a clinical condition associated with subjective and objective findings indicating death of the dental pulp.

Symptoms

Pulpal necrosis is usually asymptomatic but may be associated with episodes of spontaneous pain and discomfort or pain (from the periradicular tissues) on pressure. In teeth with necrotic pulps, pain provoked with application of heat is not due to an increase in intrapulpal pressure as is the case in teeth with vital pulps. This pressure registers zero after heat application to teeth with necrotic pulps. It is commonly believed (but is unlikely) that applying heat to teeth with liquefaction necrosis causes thermal expansion of gases present in the root canal space, which provokes pain.[49] In fact, cold, heat, or electrical stimuli applied to teeth with necrotic pulps usually produce no response.

Tests and Treatment

By definition, the pulp of a tooth with necrotic pulp should be nonresponsive to vitality testing.

However, the presence of various degrees of inflammatory response ranging from reversible pulpitis to necrosis in teeth with multiple canals is possible and may occasionally cause confusion during testing for responsiveness. Furthermore, effects of necrosis are seldom confined within canals. Because of the spread of inflammatory reactions to periradicular tissues, teeth with necrotic pulps are often sensitive to percussion. Sensitivity to palpation is an additional indication of periradicular involvement. Root canal treatment or extraction is indicated for these teeth.

Previously Treated Pulp

This condition represents a clinical diagnostic category in which the tooth has had either partial or complete endodontic therapy. The teeth in this category can be symptomatic or asymptomatic, depending on pulpal and periapical conditions. Completion of partial root canal therapy or retreatment of failed root canal treatment, endodontic surgery, or extraction is indicated for these teeth.

PERIAPICAL PATHOSIS

As a consequence of pulpal necrosis, pathologic changes can occur in the periradicular tissues. In contrast to pulp, periradicular tissues have an almost unlimited source of undifferentiated cells that participate in inflammation and repair. In addition, these tissues have a rich collateral blood supply and lymph drainage system. The interaction between the irritants emanating from the canal space and the host defense results in the activation of an extensive array of reactions to protect the host. Despite its benefits, some of these reactions are associated with destructive consequences such as periradicular bone resorption. Resorption of the bone provides a separation between the irritants and the bone, thereby preventing osteomyelitis. Depending on the severity of irritation, duration, and host response, periradicular pathoses may range from slight inflammation to extensive tissue destruction. The reactions involved are highly complex and are usually mediated by nonspecific mediators of inflammation, as well as specific immune reactions (Figure 4-13).[28]

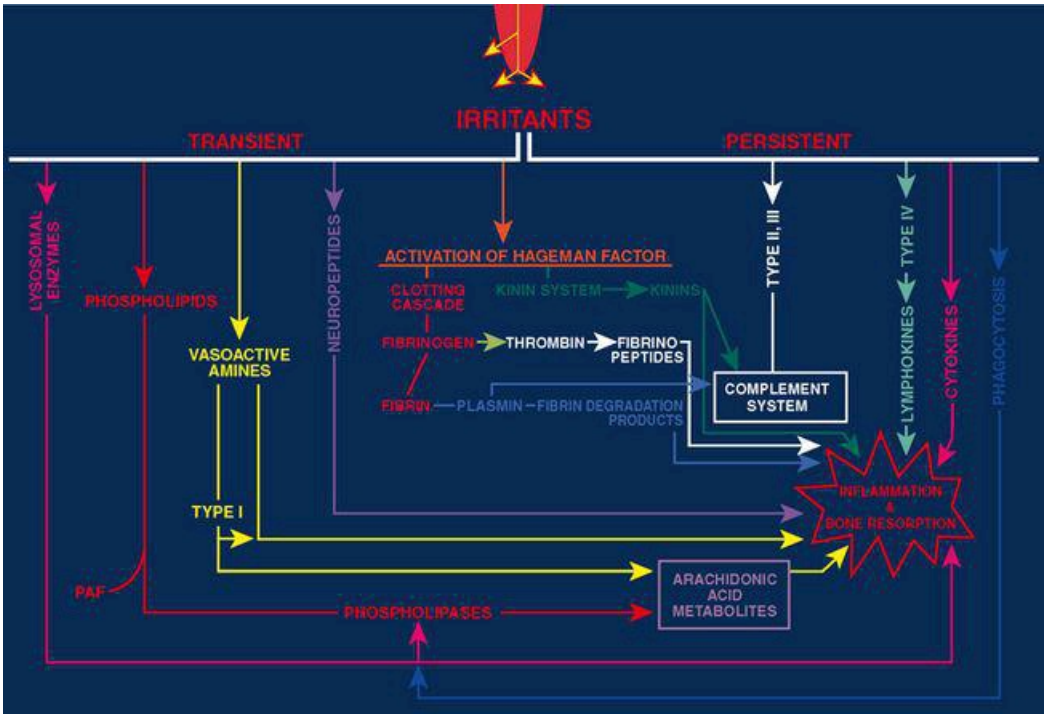

Figure 4-13 Pathways of inflammation and bone resorption by nonspecific inflammatory mediators and specific immune reactions.

Nonspecific Mediators of Periapical Lesions

Nonspecific mediators of inflammatory reactions include neuropeptides, fibrinolytic peptides, kinins, complement fragments, vasoactive amines, lysosomal enzymes, arachidonic acid metabolites, and various cytokines.[23] Neuropeptides have been demonstrated in inflamed periapical tissues of experimental animals, and it appears that these substances play a role in the pathogenesis of periradicular pathosis.[26]

Severance of blood vessels in the PDL or bone during canal instrumentation can activate intrinsic and extrinsic coagulation pathways. Contact between the Hageman factor and the collagen of basement membranes, enzymes such as kallikrein or plasmin, or endotoxins from inflamed root canals activates the clotting cascade and the fibrinolytic system. Fibrinopeptides released from fibrinogen molecules and fibrin degradation products released during the proteolysis of fibrin by plasmin contribute to inflammation. Trauma to the periapical tissues during root canal treatment can also activate the kinin system and consequently the complement system. C3 complement fragments have been found in periradicular lesions.[28] Products released from the activated systems contribute to the inflammatory process and cause swelling, pain, and tissue destruction.

Mast cells are normal components of connective tissues and are present in a normal PDL. They are also found within periradicular lesions.[50] Physical or chemical injury causes the release of vasoactive amines such as histamine, which are chemotactic for leukocytes and macrophages. In addition, lysosomal enzymes cause cleavage of C5 and generation of C5a, a potent chemotactic component, and liberation of active bradykinin from plasma kininogen.[23] Periradicular lesions show increased levels of lysosomal hydrolytic arylsulfatase A and B compared to normal tissues.[51] Significant levels of PGE_2 and leukotriene B_4 are also present in these lesions.[23] Other studies have confirmed these findings, demonstrating cessation of symptoms subsequent to emergency cleaning and shaping.[52] When utilizing immunohistochemical staining, PGE_2, prostaglandin F_{2a} (PGF_{2a}), and 6-keto-PGF_{1a} (a stable metabolite of prostaglandin I_2 [PGI_2]) have been observed in inflamed pulp tissue and periradicular lesions.[53] Regions staining positively for prostaglandins gradually extend apically into areas of the pulp tissue that are not yet inflamed. Use of indomethacin, a prostaglandin inhibitor, experimentally reduces bone resorption, indicating that prostaglandins are also involved in the pathogenesis of periradicular lesions.[23,54]

Various cytokines such as interleukins, tumor necrosis factors, and growth factors are involved in the development and perpetuation of periradicular lesions.[34,55-58] Kawashima and Stashenko[57] examined the kinetics of expression of 10 cytokines in experimentally induced murine periapical lesions. Their results showed that a cytokine network is activated in the periapical tissues in response to root canal infections and that T helper 1–modulated proinflammatory pathways predominate during periapical bone resorption.

Specific Mediators of Periapical Lesions

In addition to the nonspecific mediators of inflammatory reactions, immunologic reactions also participate in the formation and perpetuation of periradicular pathosis (see Figure 4-13). Numerous potential antigens may accumulate in the necrotic pulp, including several species of microorganisms, their toxins, and altered pulp tissue. Root canals are a pathway for sensitization.[23] The presence of potential antigens in root canals, immunoglobulin E (IgE), and mast cells in pathologically involved pulp and periradicular lesions indicate that a type I immunologic reaction may occur.

Various classes of immunoglobulins have been found in inflamed lesions.[23,59] These include specific antibodies against a number of bacterial species in infected root canals.[60,61] In addition, numerous types of immunocompetent cells, such as antigen-presenting cells (Ia antigen-expressing nonlymphoid cells), macrophages,[62] PMN leukocytes, and B and T cells, have been found in human periradicular lesions.[63] The presence of immune complexes (Figure 4-14) and immunocompetent cells such as T cells (Figure 4-15) indicates that various types of immunologic reactions (types II to IV) can initiate, amplify, or perpetuate these inflammatory lesions.[23]

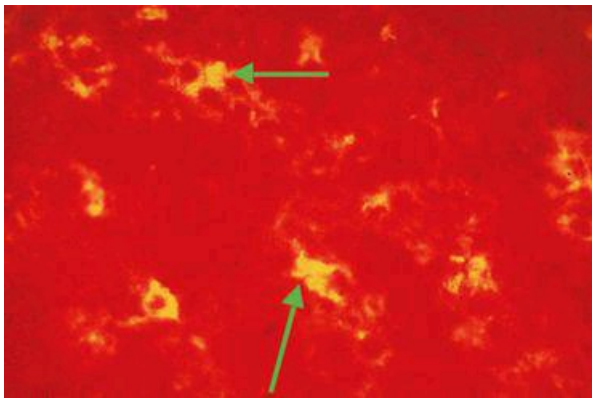

Figure 4-14 Using the anticomplement immunofluorescence technique, immune complexes are identified *(arrows)* in human periapical lesions.

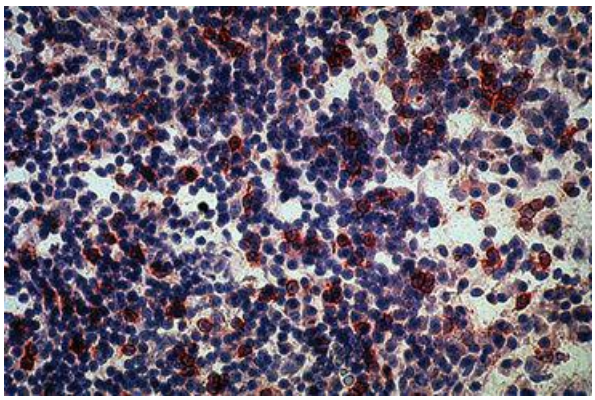

Figure 4-15 T lymphocytes (red membrane cells) are identified in a human periapical lesion by an immunohistochemical technique.

CLASSIFICATION OF PERIAPICAL LESIONS

Periapical lesions have been classified on the basis of their clinical and histologic findings. As with pulpal disease, little correlation exists between the clinical signs and symptoms and duration of lesions compared with the histopathologic findings.[64] Because of these discrepancies and for convenience, these lesions are classified into the following six main groups: normal periapical tissues, symptomatic (acute) apical periodontitis, asymptomatic (chronic) apical periodontitis, condensing osteitis, acute apical abscess, and chronic apical abscess. Lesions associated with significant symptoms, such as pain or swelling, are referred to as acute (symptomatic), whereas those with mild or no symptoms are identified as chronic (asymptomatic).

Normal Periapical Tissues

This condition represents a clinical and radiographic diagnostic category in which the tooth has normal periapical tissues and will not be abnormally sensitive to percussion or palpation testing. The teeth in this category have normal lamina dura and periodontal ligament structures.

Symptomatic Apical Periodontitis

Etiology

The first extension of pulpal inflammation into the periradicular tissues is called symptomatic apical periodontitis (SAP). Eliciting irritants include inflammatory mediators from an irreversibly inflamed pulp or egress of bacterial toxins from necrotic pulps, chemicals (such as irrigants or disinfecting agents), restorations in hyperocclusion, overinstrumentation of the root canal, and extrusion of obturating materials. The pulp may be reversibly inflamed, irreversibly inflamed or necrotic.

Signs and Symptoms

Clinical features of SAP are moderate to severe spontaneous discomfort as well as pain on biting or percussion. If SAP is an extension of pulpitis, its signs and symptoms will include responsiveness to cold, heat, and electricity. Cases of SAP caused by a necrotic pulp do not respond to vitality tests. Application of pressure by the fingertip or tapping with the butt end of a mirror handle (percussion) can cause marked to excruciating pain. SAP may or may not be associated with an apical radiolucent area. “Thickening” of the PDL space may be a radiographic feature of SAP (Figure 4-16). However, usually there is a normal PDL space and an intact lamina dura.

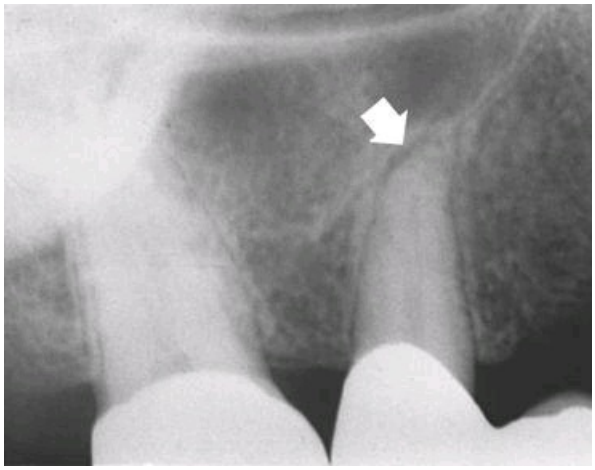

Figure 4-16 After cementing a three-unit bridge, the premolar developed clinical signs and symptoms of acute apical periodontitis, and the radiograph shows a widened periodontal ligament space *(arrow)*.

Histologic Features

With SAP, PMN leukocytes and macrophages are visible within a localized area at the apical region of the pulp. At times, there may be a small area of liquefaction necrosis (abscess). Bone and root resorption may be present histologically; however, resorption is usually not visible radiographically.

Treatment

Adjustment of occlusion (when there is evidence of hyperocclusion), removal of irritants or a pathologic pulp, or removal of periapical exudate usually results in relief.

Asymptomatic Apical Periodontitis

Etiology

Asymptomatic apical periodontitis (AAP) results from pulp necrosis and usually is a sequel to SAP.

Signs and Symptoms

By definition, AAP is a clinical asymptomatic condition of pulpal origin associated with inflammation and destruction of periapical tissues. Because the pulp is necrotic, teeth with AAP do not respond to electrical or thermal stimuli. Percussion produces little or no pain. There may be slight sensitivity to palpation, indicating an alteration of the cortical plate of bone and extension of AAP into the soft tissues. Radiographic features range from interruption of the lamina dura to extensive destruction of periapical and interradicular tissues (Figure 4-17).

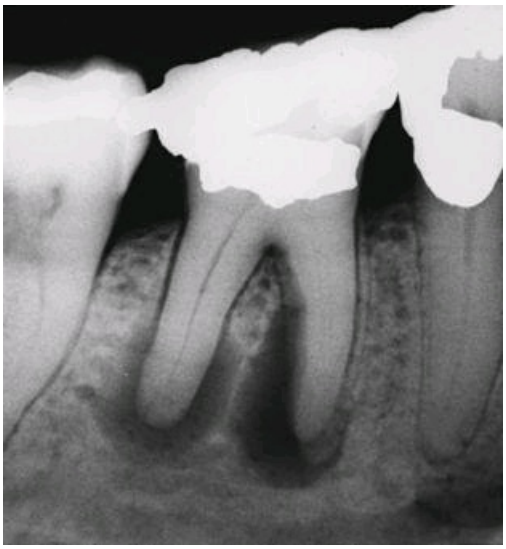

Figure 4-17 Chronic apical periodontitis. Extensive tissue destruction in the periapical regions of a mandibular first molar as a result of pulpal necrosis. A lack of symptoms and radiographic lesions is diagnostic.

Histologic Features

Histologically, AAP lesions are classified as either granulomas or cysts. A periapical *granuloma* consists of granulomatous tissue infiltrated by mast cells, macrophages, lymphocytes, plasma cells, and occasionally, PMN leukocytes (Figure 4-18). Multinucleated giant cells, foam cells, cholesterol clefts, and epithelium are often found.

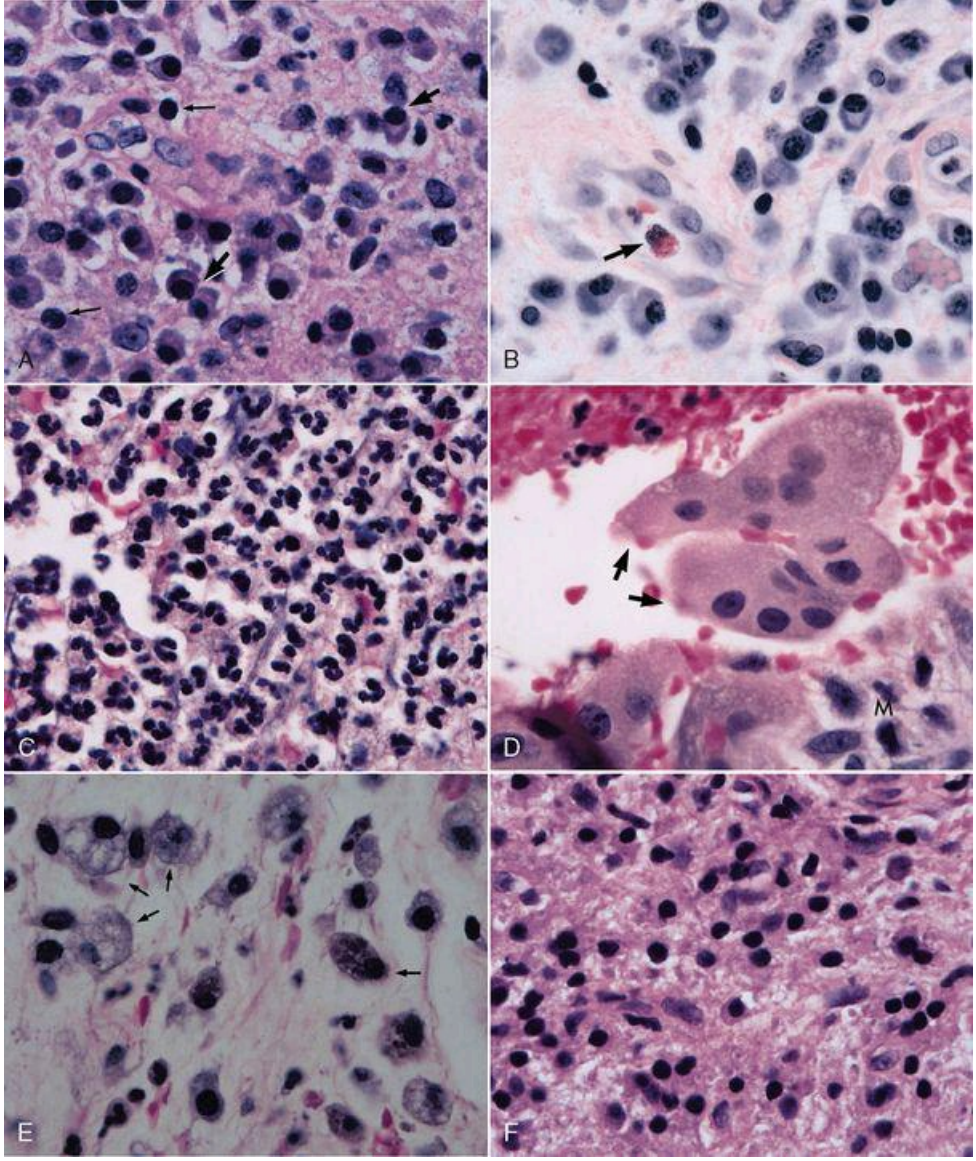

Figure 4-18 A, Lymphocytes *(small arrows).* Plasma cells *(large arrows)* have an eccentric nucleus with adjacent "clear zone" and a basophilic outer rim of cytoplasm. **B,** Eosinophil *(arrow)* with distinct eosinophilic granules and bilobed nucleus. Plasma cells and lymphocytes are also visible. **C,** PMN leukocytes are concentrated in this field. These have multilobed nuclei; many of these are degenerating and have disrupted cell walls. **D,** Giant cells *(arrows)* with multiple nuclei. Macrophages *(M)* with lighter-stained nuclei and diffuse cytoplasm. **E,** Macrophages *(arrows)* are larger and often have ingested material, as indicated by a "foamy" cytoplasm in these cells. **F,** Lymphocytes, with their densely basophilic nuclei, dominate this field.

(Courtesy Dr. C. Kleinegger.)

The apical (radicular) *cyst* has a central cavity filled with an eosinophilic fluid or semisolid material and is lined by stratified squamous epithelium (Figure 4-19). The epithelium is surrounded by connective tissue containing all cellular elements found in the periapical granuloma. Therefore an apical cyst is a granuloma that contains a cavity or cavities lined with epithelium. The origin of the epithelium is the remnants of Hertwig's epithelial sheath, the cell rests of Malassez. These cell rests proliferate in response to inflammatory stimuli. The actual genesis of the cyst is unclear.

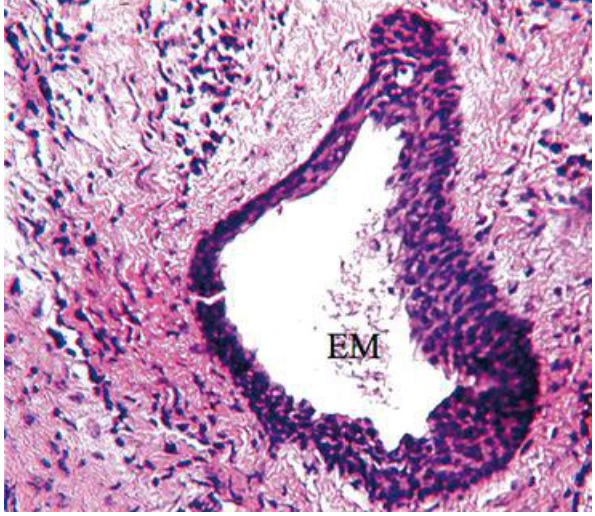

Figure 4-19 A region of human apical cyst consists of a central cavity filled with eosinophilic material *(EM)* and a wall lined with epithelium.

(Courtesy Dr. N. Perrini.)

The reported incidence of various classes of endodontic lesions is inconsistent. Variations may be due to sampling methods and the histologic criteria used for diagnosis. Nobuhara and Del Rio examined periapical biopsies that were refractory to root canal treatment and showed most (59%) to be granulomas, with some (22%) cysts, a few (12%) scars, and a scattering (7%) of other types of lesions.[65] Percentages such as these are misleading. Many lesions share combined features of granulomatous inflammatory lesions, cysts, and areas of scarring. The samples usually do not include abscesses, because their intact recovery is difficult during surgery. In fact, in the majority of cases, the entire lesion is typically not recovered for biopsy and only fragments are obtained during curettage.

Treatment

Removal of inciting irritants (necrotic pulp) and complete obturation of the root canal system usually result in resolution of AAP (Figure 4-20). There is no evidence that apical cysts resist resolution after adequate root canal treatment or extraction.

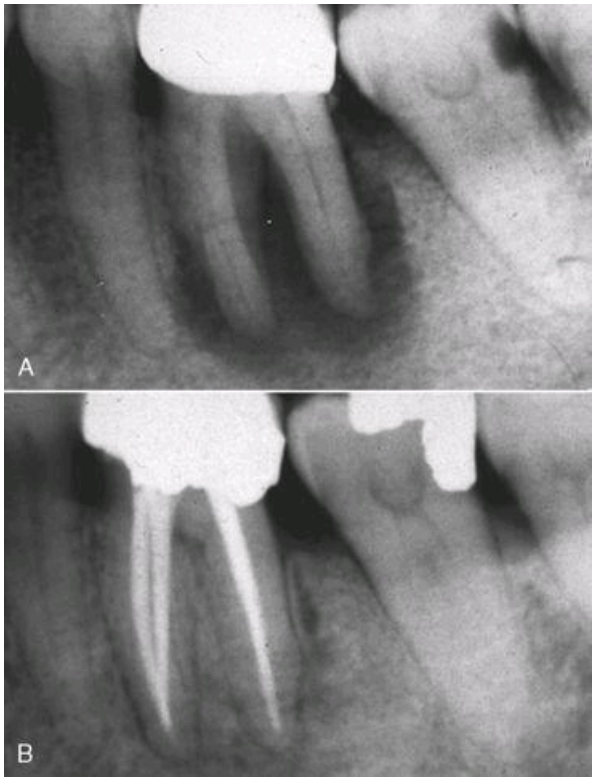

Figure 4-20 A, Preoperative radiograph of a first molar shows pulpal necrosis and chronic apical periodontitis. **B,** Postoperative radiograph 2 years after root canal therapy shows complete resolution of the periradicular pathosis.

Condensing Osteitis

Etiology

Condensing osteitis, a variant of asymptomatic apical periodontitis, represents an increase in trabecular bone in response to persistent irritation. The irritant diffusing from the root canal into periradicular tissues is the main cause of condensing osteitis. This lesion is usually found around the apices of mandibular posterior teeth, which show a probable cause of pulp inflammation or necrosis. However, condensing osteitis can occur in association with the apex of any tooth.

Signs and Symptoms

Depending on the cause (pulpitis or pulpal necrosis), condensing osteitis may be either asymptomatic or associated with pain. Pulp tissue of teeth with condensing osteitis may or may not respond to electrical or thermal stimuli. Furthermore, these teeth may or may not be sensitive to palpation or percussion. Radiographically, the presence of a diffuse concentric arrangement of radiopacity around the root of a tooth is pathognomonic (Figure 4-21). Histologically, there is an increase in irregularly arranged trabecular bone and inflammation.[66]

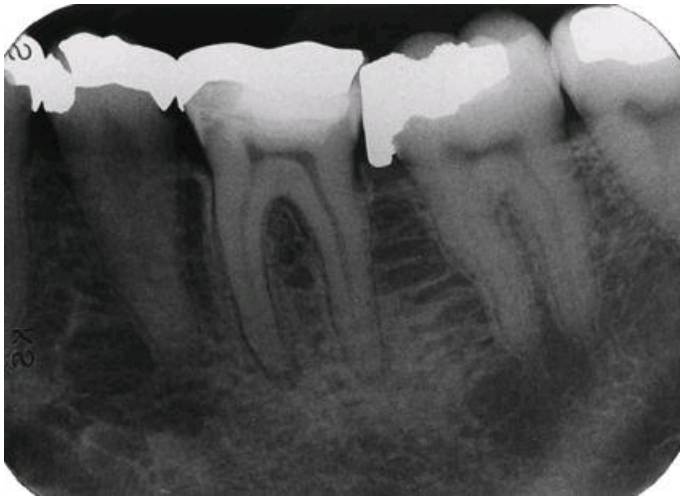

Figure 4-21 Condensing osteitis. Inflammation followed by necrosis in the pulp of the first molar has resulted in the diffuse radiopacity of periapical tissue.

Root canal treatment, when indicated, may result in the complete resolution of condensing osteitis.[67] Condensing osteitis is often confused with enostosis (sclerotic bone), a nonpathologic entity.

Acute Apical Abscess

Etiology

Acute apical abscess (AAA) is a localized (Figure 4-22, *A*) or diffuse (Figure 4-22, *B*) liquefaction lesion of pulpal origin that destroys periradicular tissues and a severe inflammatory response to microbial and nonbacterial irritants from a necrotic pulp.

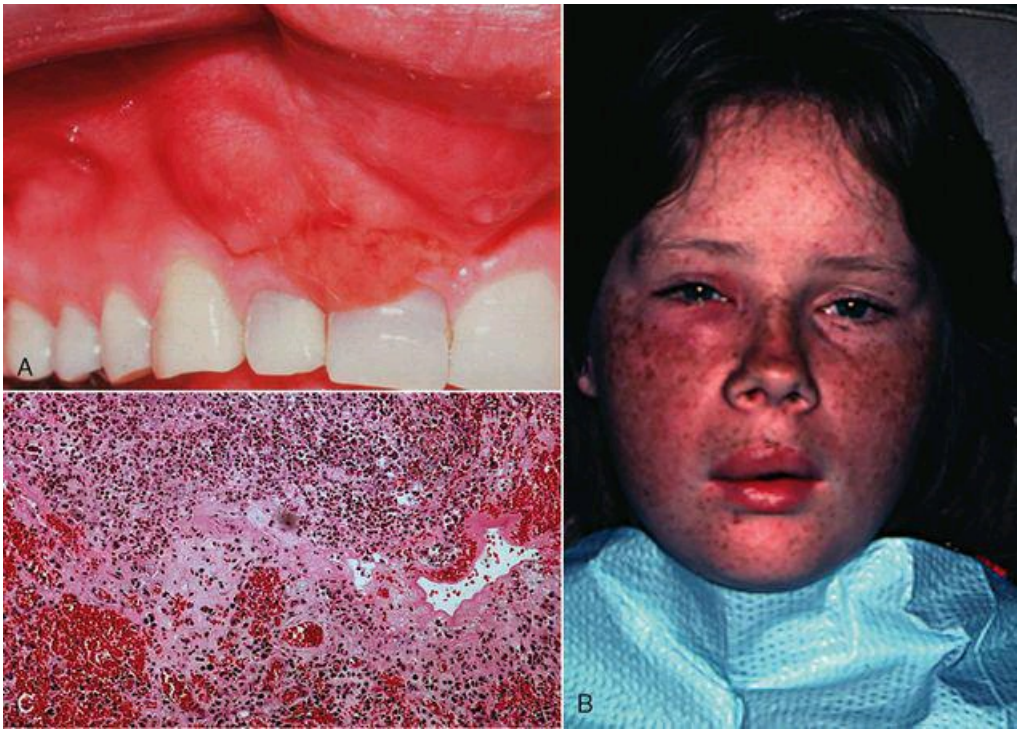

Figure 4-22 A, The localized vestibular swelling is a result of the necrotic pulp in the right lateral incisor. **B,** An acute apical abscess has created a diffuse facial swelling. **C,** Histologic examination of AAA shows edematous tissue heavily infiltrated by degenerating PMN leukocytes.

Signs and Symptoms

AAA is characterized by rapid onset and spontaneous pain. Depending on the severity of the reaction, patients with AAA usually have moderate to severe discomfort and/or swelling. There often is no swelling if the abscess is confined to bone. In addition, they occasionally have systemic manifestations of an infective process such as elevated temperature, malaise, and leukocytosis. Because these findings are only observed in association with a necrotic pulp, electrical or thermal stimulation produces no response. However, these teeth are usually painful to percussion and palpation. Depending on the degree of hard tissue destruction inflicted by irritants, radiographic features of AAA range from no changes to widening of the PDL space to an obvious radiolucent lesion.

Histologic Features

Histologic examination of AAA usually shows a localized destructive lesion of liquefaction necrosis containing numerous disintegrating PMN leukocytes, debris and cell remnants, and an accumulation of purulent exudate (Figure 4-22, *C*). Surrounding the abscess is granulomatous tissue; therefore the lesion is best categorized as an abscess within a granuloma. Notably, the abscess often does not communicate directly with the apical foramen; frequently an abscess will not drain through an accessed tooth.

Removal of the underlying cause, release of pressure (drainage where possible), and routine root canal treatment will lead to resolution of most cases of AAA.

Chronic Apical Abscess

The chronic apical abscess (CAA) is an inflammatory lesion of pulpal origin that is characterized by the presence of a long-standing lesion that has resulted in an abscess that is draining to a mucosal (sinus tract) or skin surface.

Etiology

CAA has a pathogenesis similar to that of AAA. It also results from pulpal necrosis and is usually associated with chronic apical periodontitis that has formed an abscess. The abscess has "burrowed" through bone and soft tissue to form a sinus tract stoma on the oral mucosa (Figure 4-23, *A*) or sometimes onto the facial dermis. The histologic findings in these lesions are similar to those found in SAP (Figure 4-23, *B*). CAA may also drain through the periodontium into the sulcus and may mimic a periodontal abscess or pocket (see Chapter 7).

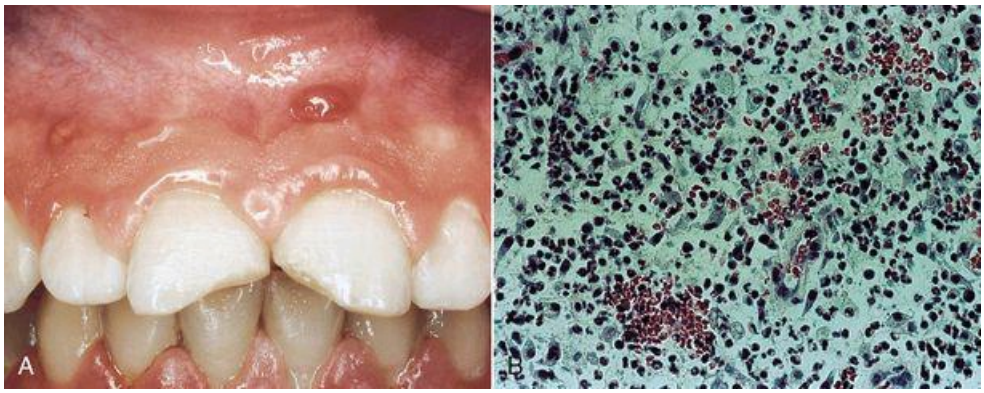

Figure 4-23 A, A sinus tract stoma associated with a necrotic pulp in the left central incisor. **B,** Histologic examination of the periapical tissue shows numerous lymphocytes, plasma cells, and macrophages (foam cells)

Signs and Symptoms

Because drainage exists, CAA is usually asymptomatic, except when there is occasional closure of the sinus pathway, which can cause pain. Clinical, radiographic, and histopathologic features of CAA are similar to those described for AAP. An additional feature is the sinus tract, which may be lined partially or completely by epithelium surrounded by inflamed connective tissue.[68]

HEALING OF PERIAPICAL LESIONS AFTER ROOT CANAL TREATMENT

Regeneration is a process by which altered periapical tissues are completely replaced by tissues native to their original architecture and function. Repair is a process by which altered tissues are not completely restored to their original structures. Histologic examination of most tissue sections in experimental animals and humans shows that healing of periradicular lesions after root canal therapy is by repair rather than regeneration of the periradicular tissues. Inflammation and healing are not two separate entities; in fact, they constitute part of one process in response to tissue injury. On the molecular and cellular levels, it is impossible to separate the two phenomena. Inflammation dominates the early events after tissue injury, shifting toward healing after the early responses have subsided. However, for convenience and to simplify the complex inflammatory-resorptive process, they are studied as two separate entities.

Extent of Healing

The level of healing is proportional to the degree and extent of tissue injury and the nature of tissue destruction. When injury to periradicular tissues is slight, little repair or regeneration is required. However, extensive damage requires substantial healing (see Figure 4-20). In other words, periradicular repair ranges from a relatively simple resolution of an inflammatory infiltrate in the PDL to considerable reorganization and repair of a variety of tissues.

Process of Healing

The sequence of events leading to resolution of periapical lesions has not been studied extensively. Based on the processes involved in the repair of extraction sites,[69] after removal of irritants, inflammatory responses decrease and tissue-forming cells (fibroblasts and endothelial cells) increase; and finally, tissue organization and maturation ensue. Bone that has resorbed is replaced by new bone; resorbed cementum and dentin are repaired by cellular cementum. The PDL, which is the first tissue affected, is the last to be restored to normal architecture. Histologic examination of healing of periapical lesions shows evidence of healing in the form of cementum deposition, increased vascularity, and increased fibroblastic and osteoblastic activities.[70] Studies have shown that some cytokines play an important role during healing of periapical lesions.[71,72]

Some lesions do not completely regain all of the original structures. Variations are seen in different fiber or bone patterns. These may be obvious radiographically with a widened lamina dura or altered bony configuration. Certain factors, such as the size of the defect or the extent of injury to the underlying stroma, may impact complete regeneration of the original tissue architecture. Boyne has shown that these critical-sized defects do not heal unless stimulated by inductive factors such as bone morphogenetic proteins.[73]

Factors Influencing Healing

Other factors that may impact healing of periapical lesions include inherent host factors such as leukopenia, impairment of blood supply, inadequate nutrition, corticosteroids, and other systemic diseases. For instance, patients with insulin-dependent diabetes mellitus have a significantly lower healing rate after root canal therapy of teeth with apical lesions than nondiabetic patients.[74]

NONENDODONTIC PERIRADICULAR PATHOSIS

Differential Diagnosis

A number of radiolucent and radiopaque lesions of nonendodontic origin simulate the radiographic appearance of endodontic lesions. Because of their similarities, dentists must use their knowledge and perform clinical tests in a systematic manner to arrive at an accurate diagnosis and avoid critical mistakes. Pulp vitality tests are the most important aids in differentiating between endodontic and nonendodontic lesions. Teeth associated with radiolucent periradicular lesions have necrotic pulps and therefore generally do not respond to vitality tests. In contrast, lesions of nonpulpal origin usually do not affect the blood or nerve supply to adjacent tooth pulp; therefore the vitality (responsiveness) of these teeth remains unaffected.

Unfortunately, many clinicians rely solely on radiographs for diagnosis and treatment, without obtaining a complete history of the signs and symptoms and performing clinical tests. Many nonendodontic radiolucencies (including those resulting from pathoses and those with normal morphology) mimic endodontic pathoses and vice versa. To avoid grievous mistakes, all relevant vitality tests, radiographic examinations, clinical signs and symptoms, and details of the patient history should be utilized.

Normal and Pathologic Entities

Most radiographic changes arise from pathologic changes in the pulp. However, other radiographic variations, such as anatomic variations, as well as benign and malignant lesions, may simulate the appearance of periradicular lesions.[75]

Normal Structures

Anatomic variations include large marrow spaces adjacent to the apices of teeth, submandibular fossae, maxillary sinus, apical dental papillae of developing teeth, nasopalatine foramen, mental foramen (Figure 4-24), and lingual depressions in the mandible. Associated teeth respond to vitality tests and show no clinical signs and symptoms of any disease process. In addition, by changing the cone angulation, the location of these radiolucent lesions can be altered relative to their original positions and to the root apices.

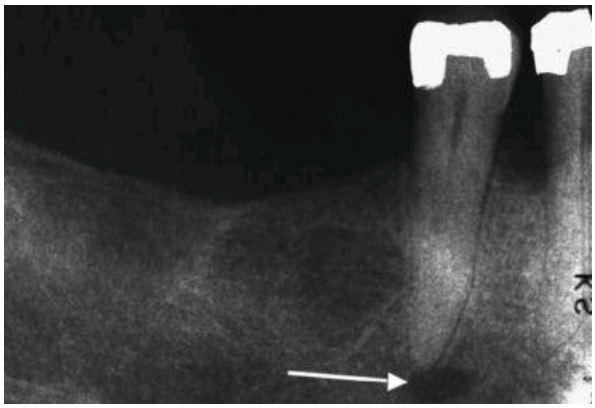

Figure 4-24 A mental foramen *(arrow)* simulating a periapical lesion of pulpal origin. Pulp tests are within normal limits, indicating that this radiolucency could not be endodontic pathosis.

Nonendodontic Pathoses

Benign lesions with radiographic appearances similar to periradicular lesions include (but are not limited to) the initial stages of periapical cemental dysplasia (Figure 4-25), early stages of monostotic fibrous dysplasia, ossifying fibroma, primordial cyst, lateral periodontal cyst, dentigerous cyst, median maxillary or mandibular cyst, bone cyst, central giant cell granuloma, central hemangioma, hyperparathyroidism, myxoma, and ameloblastoma. Usually (but not always), radiographically, the lamina dura around the root apices is intact and responses to pulp tests are normal. The final diagnosis of these lesions is often based on surgical biopsy and histopathologic examination.

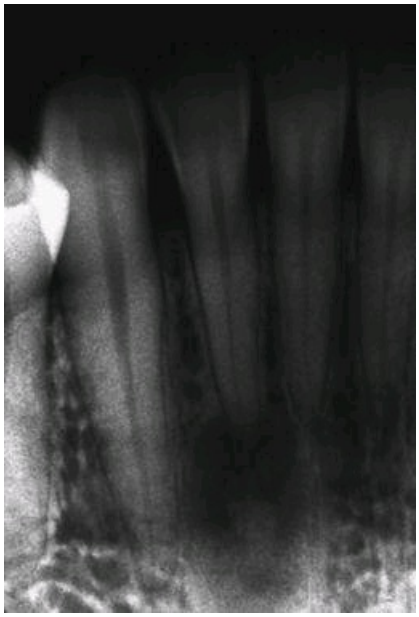

Figure 4-25 A periapical radiolucency in the early stages of cementoma can simulate a periapical lesion of pulpal origin. However, the pulps are responding within normal limits.

Malignant lesions that may simulate periapical lesions of pulpal origin and are often metastatic include lymphoma (Figure 4-26), squamous cell carcinoma, osteogenic sarcoma, chondrosarcoma, and multiple myeloma. Unlike endodontic lesions, these lesions are usually associated with rapid and extensive hard tissue (bone and tooth) destruction. Ordinarily, the teeth in the affected region remain responsive to vitality tests, although occasionally the pulps or sensory nerves are disrupted and nonresponsive. For a more complete list and description of lesions that may mimic the radiographic appearance of endodontic lesions of pulpal origin, an oral pathology text should be consulted.[75]

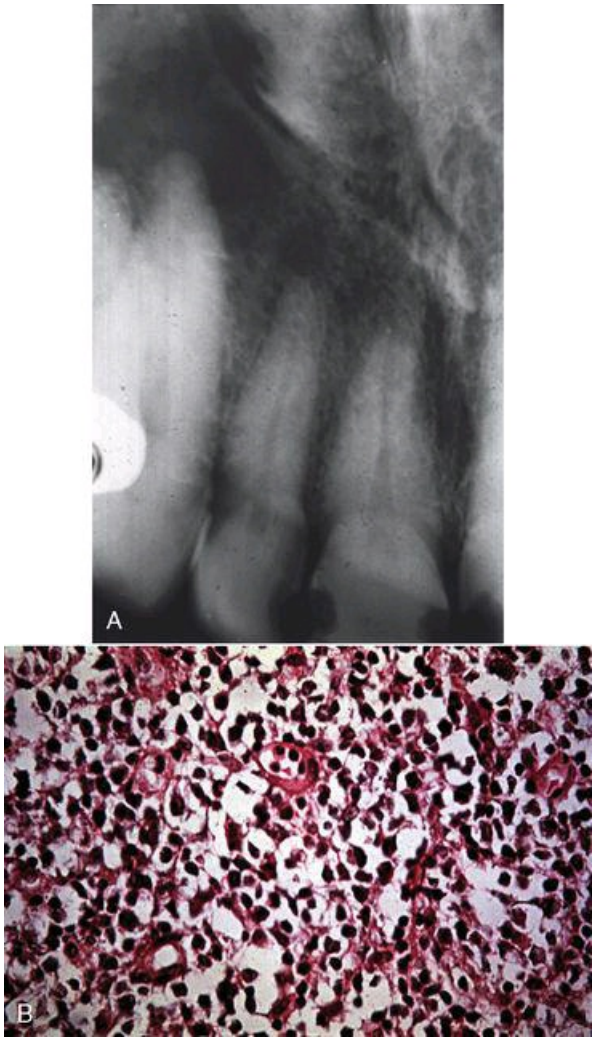

Figure 4-26 A, A periapical radiolucent lesion of nonpulpal origin. **B,** Positive results of vitality tests and histologic examination of the tissue confirmed a diagnosis of lymphoma.

(Courtesy Dr. J Simon.)

Chapter Review Questions available in Appendix B or on the DVD

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Diagnosis and Treatment Planning

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Recognize that diagnosis and treatment planning for pulpal and periapical conditions should be part of a broader examination and treatment plan.

2. Identify emergency conditions that require immediate treatment and integrate their treatment into an overall treatment plan.

3. Understand the importance of the medical and dental history to endodontic diagnosis.

4. Address the correct questions regarding the history and symptoms of the present complaint.

5. Describe clearly to the patient the diagnostic procedure to be followed.

6. Conduct an intraoral examination of both soft and hard tissues focused on determining the pulpal and periapical health.

7. Apply, interpret, and understand the limitations of vitality tests.

8. Know when and how to utilize special approaches such as a test cavity and selective anesthesia.

9. Interpret appropriate radiographs.

10. Understand the mechanisms of pain and how variable the pain experience can be.

11. Understand and detect when pain is referred and when hyperalgesia and allodynia are present.

12. Consolidate all data from the history, symptoms, examination, and tests to form a diagnosis of pulpal and periapical conditions using appropriate terminology.

13. Identify conditions for which root canal treatment is indicated and contraindicated.

14. Recognize the indications for adjunctive treatments such as vital pulp therapy, bleaching, root amputation, endodontic surgery, intentional replantation, autotransplantation, hemisection, apexification, orthodontic extrusion, and retreatment.

15. Identify problems (operative complications, cracked tooth, periodontal problems, isolation difficulties, restorability, strategic value, patient management, medical complications, abnormal root or pulp anatomy, impact trauma, and restricted opening) that require treatment modifications.

16. Design an endodontic treatment plan that is integrated into the overall treatment plan.

17. Present the patient with the preferred treatment plan and any alternatives and explain its development from the diagnostic data.

18. Discuss the prognosis of any suggested treatment.

19. Classify complications of endodontic procedures.

20. Identify which procedures are ordinarily not within the graduating dentist's realm of training or experience and which patients should be considered for referral.

Endodontic diagnosis and treatment occur in two scenarios. The first occurs when the emergency patient presents with pain and possibly swelling or with displaced or avulsed teeth. The second scenario is part of the restorative treatment plan when pulpal or periapical disease is detected but without symptoms. In the first case, urgent treatment is needed to relieve pain, prevent the spread of infection, and immobilize avulsed or loosened teeth. In the second scenario, definitive endodontic treatment will be carried out as part of a comprehensive treatment plan. Once an emergency has been relieved, scenario one will become scenario two.

In both scenarios a thorough history and examination leading to a diagnosis are essential to provide appropriate and effective care (Figure 5-1). *The key to effective treatment is accurate diagnosis.* The key to accurate diagnosis is an understanding of the pathologic processes occurring in the affected tissue (see Chapters 1 and 4).

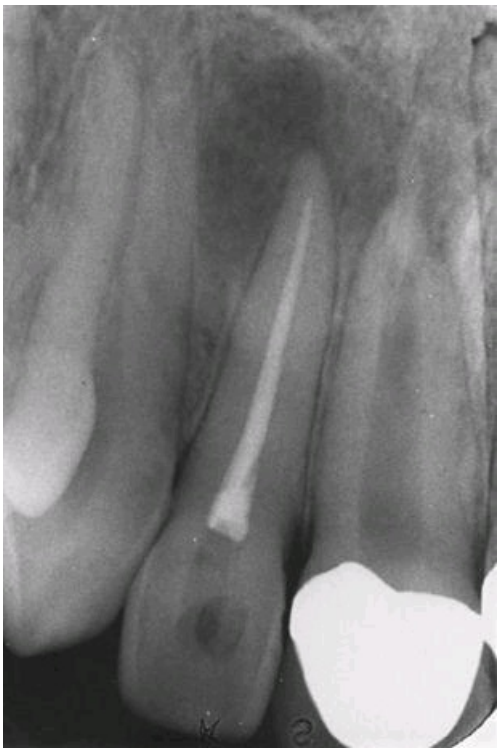


Figure 5-1 A reliance on "clinical experience" rather than on adequate tests resulted in the wrong treatment. The dentist relied on a radiograph only (no tests) and concluded that the lateral incisor was the painful problem tooth. After treatment, with no change in the level of pain, the patient was referred for root-end surgery. Examination of preoperative and postoperative radiographs, as well as clinical tests, showed that treatment had been performed on a tooth with normal pulp. The central incisor was found to have pulp necrosis and an acute apical abscess. Immediate pain relief followed root canal treatment on the correct tooth.

Diagnosis is the science of recognizing disease by means of signs, symptoms, and tests. Often, diagnosis is straightforward; sometimes it is not. The basic steps in the diagnostic process are as follows:

1. Chief complaint

2. History: medical and dental

3. Oral examination

4. Data analysis → differential diagnosis

5. Treatment plan

A limited number of possible diagnoses exist for conditions of the pulp and periapical conditions (Table 5-1). These diagnoses are:

- □ Pulpal diagnoses
  - □ Normal
  - □ Reversible pulpitis
  - □ Irreversible pulpitis
  - □ Necrosis

- □ Periapical diagnoses
  - □ Normal
  - □ Symptomatic apical periodontitis
  - □ Asymptomatic apical periodontitis
  - □ Acute apical abscess
  - □ Chronic apical abscess
  - □ Condensing osteitis

The pathosis of these conditions is described in Chapter 4.

Table 5-1 Diagnostic Terminology

	Symptoms	Radiographic	Pulp Tests	Periapical Tests
Pulpal				
Normal	None of any significance	No periapical changes	Responds	Not sensitive
Reversible	May or may not have slight symptoms to thermal stimulus	No periapical changes	Responds	Not sensitive
Irreversible	Similar to reversible; also may have spontaneous or severe pain to thermal stimuli	No periapical changes; one exception: occasional condensing osteitis	Responds (possibly with extreme pain on thermal stimulus)	May or may not have pain on percussion or palpation
Necrotic	None to thermal stimulus Other symptoms: see under *Periapical*	See under *Periapical*	No response	Depends on periapical status
Periapical				
Normal	None of significance	No significant changes	Response or no response (depends on pulp status)	Not sensitive
Symptomatic (acute) apical periodontitis	Significant pain on mastication or pressure	No significant changes	Response or no response (depends on pulp status)	Pain on percussion or palpation
Asymptomatic (chronic) apical periodontitis and apical cyst	None to mild	Apical radiolucency	No response	None to mild on percussion or palpation
Acute apical abscess	Swelling or significant pain	Usually a radiolucent lesion	No response	Pain on percussion or palpation
Chronic apical abscess (suppurative apical periodontitis)	Draining sinus or parulis	Usually a radiolucent lesion	No response	Not sensitive
Condensing osteitis	Varies (depends on pulp or periapical status)	Increased trabecular bone density	Response or no response (depends on pulp status)	May or may not have pain on percussion or palpation

Although diagnosis is a science, it is an imperfect science and sometimes a detailed, definitive diagnosis is difficult or impossible. Importantly, *significant pulpal or periapical pathosis is frequently without marked symptoms at present or in the past.*[1] The clinician must be alert for other indicators of pathosis that will require careful examination.

Pain as a presenting symptom is obviously of key concern to the patient who is having the experience as well as to the dentist who is eager to deal with the pain. Unfortunately, this symptom often leads to a misdiagnosis because pain is often referred from its true origin to another site. A well-known example is appendicitis; the pain seems to originate from the region of the navel, which is a long way from the diseased organ. A similar phenomenon occurs in the mouth; pain may be referred from one tooth to another. In addition, pain that arises from the temporomandibular joint and associated musculature, from sinus infections, and even from cardiac problems[2] may refer to the teeth. The mechanism for this is discussed later, but diagnosing a

painful condition in the orofacial region must begin with these complicating factors in mind.

Other pathologic conditions, such as neuralgia, multiple sclerosis, myocardial ischemia, or psychiatric disorders, may produce the same symptoms. *A thorough understanding of the complicated, multifactorial, and versatile nature of pain is essential to an accurate diagnosis and successful treatment.*

DIAGNOSIS

Chief Complaint

The chief complaint is the first information obtained and is usually volunteered by the patient. They will express their complaint in their own words, which should be recorded in the chart as such. This, after all, is why the patient seeks attention, and the patient will judge the outcome of treatment by whether the problem as they see it is managed. To capture the patient's confidence, attention must be paid to their chief complaint. Having achieved this, the patient can then understand that diagnosis requires a thorough and methodical approach and follow this approach.

Health and Medical History

Whether the patient is returning to the practice or has filled out a new health questionnaire, the medical history is reviewed with the patient directly; this is recorded in the clinical record. The population seeking and requiring endodontic treatment is older on average than the general population and shows a higher and more complex incidence of systemic medical problems.[3] Some issues are of concern in diagnosing endodontic problems. Acute respiratory infections, particularly of the maxillary sinus, often produce toothache-like symptoms. Stress commonly leads to neuromuscular pain in the masticatory apparatus whose presentation includes tooth pain.

Although no medical conditions contraindicate endodontic treatment, there are some conditions that can reduce the patient's ability to respond to treatment.[4] Acquired immunodeficiency syndrome (AIDS) clearly compromises the immune system, as may hepatitis.[5,6] Drugs used to prevent the rejection of transplants and grafts, as well as those used combat glucocorticosteroid deficiency and serious allergies, may have a similar effect. The incidence of type II diabetes is increasing rapidly in the general population. It is related to a greater incidence of endodontic pathosis[7]; there is some evidence that healing is less predictable.[8,9] Patients with active ischemic heart disease may need special consideration, which should be based on a consultation with their cardiologist.[10,11]

There is considerable interest in the relationship between oral disease (periodontal disease in particular) and cardiovascular disease. There does not seem to be a significant relationship between the endodontic pathosis and cardiovascular disease.[12,13]

Other factors related to therapeutics may complicate diagnosis. Bisphosphonates are a factor to consider in diagnosis and treatment planning. These are commonly prescribed drugs, particularly in postmenopausal females (but occasionally in males) with osteoporosis or osteopenia. Administration is generally oral. Cancer that involves bone resorption is also frequently treated by bisphosphonates, usually intravenously in much higher dosages than when used orally. An uncommon but serious side effect of this drug is osteonecrosis of the jaws (ONJ), which may occur with either intravenous (uncommon) or oral (rare) administration. Although the occurrence rate is very low, it can be destructive and difficult to treat.[14] Patients on this therapy should always be informed of the risks. In addition, surgical procedures, including extractions, should be avoided whenever possible. Although there have been very rare (not well-documented) reports of ONJ after root canal treatment, this approach is preferable to extraction, even when a tooth is severely compromised.[15] The root canal treatment must be performed carefully, confining instruments, irrigants, and obturating materials to the canal space. Rubber dam clamps should be avoided if there are other means of retaining the dam. When their use is necessary, the clamps should be carefully placed to avoid injury to the soft tissues and alveolar bone.

A more frequent concern is that patients who have experienced pain and/or swelling may already be taking antibiotics and analgesics that can mask symptoms.

Antibiotic Prophylaxis

A very recent update on the guidelines for antibiotic prophylaxis has been released by a task force of the American Heart Association with input from the American Dental Association.[16] These recommendations greatly reduce the indications for coverage to a limited number of cardiac conditions that include (1) artificial heart valve; (2) previous history of infective endocarditis; (3) incomplete or repaired congenital heart tissue repair; and (4) some heart transplants. In these patients, the regimen is 2 grams of amoxicillin 30 to 60 minutes preoperatively for adults. Children's dosage is 50 mgm/kg. For allergic patients, a good choice is clindamycin 600 mgm 30 to 60 minutes before the procedure.

The American Society of Orthopaedic Surgeons guidelines for prophylaxis in joint replacement patients remain unchanged from 1997. Patients considered to be at risk include those who have had joint replacements within the past 2 years, immunocompromised/immunosuppressed or hemophiliac patients, insulin-dependent diabetics, and those with previous joint prosthesis infections. The same regimen is recommended as with cardiac patients.

In these cardiac or joint prosthesis patients, endodontic procedures considered to be a risk include instrumentation beyond the apex, periapical surgery, or other procedures that may produce bleeding, such as aggressive rubber dam placement or incision for drainage.

Dental History

Endodontic problems usually have a history (Figure 5-2). Recent trauma is obviously relevant as are recent restorations and previous treatment for temporomandibular dysfunction. A longer overview may indicate the type of treatment that would be most appropriate. A patient with a poorly maintained dentition and several missing teeth may not be an ideal candidate for endodontic treatment and subsequent restorative procedures.

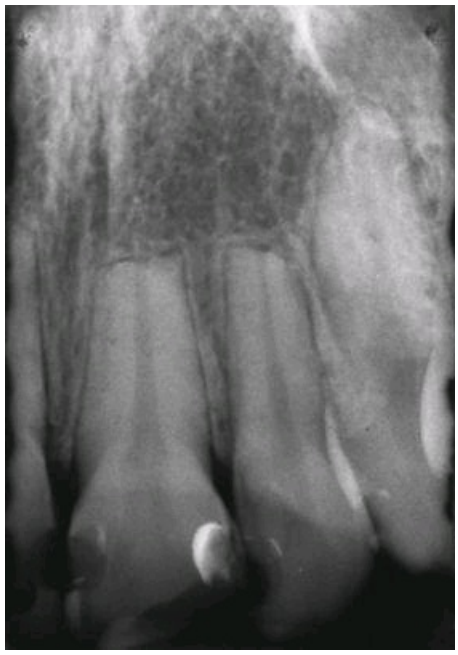


Figure 5-2 Short roots, an absence of pathosis in the periapical tissues, and a history of orthodontics indicate that resorption is due to tooth movement in the past.

History of the Present Complaint

If the patient has already described why they are seeking care in their own words, details must now be established by methodical questioning. There are a limited number of complaints of endodontic consequence. The patient may have more than one complaint. Pain and swelling, for example, often occur together. The most common complaints are as follows:

□ Pain

□ Swelling

□ Broken tooth

□ Loose tooth

□ Tooth discoloration

□ Bad taste

If there are two complaints presented at the same time, such as pain and swelling, then the history of each complaint must be obtained.

Pain is the most obvious and most important of these complaints. A good understanding of the physiology of pain and the anatomy of nociceptive pathways is essential to the diagnosis and treatment of painful conditions.[17] The following paragraphs contain a brief synopsis of pain mechanisms. Boxes 5-1 and 5-2 present the key elements in list form.

Box 5-1 What Is Pain?

□ "An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."

□ A dedicated "pain system" includes nociceptors (receptors preferentially sensitive to a noxious stimulus), small diameter fibers, tracts, and central processing areas.

□ Noxious stimuli activate nociceptors, but this activation does not inevitably result in pain.

□ Activity in pain pathways can be modulated upward or downward, both peripherally and centrally. In particular, descending opioid influences can facilitate or impede the transmission of activity from first-order neurons to higher centers, thus reducing the pain experience.

□ Affective, motivational, and cultural factors contribute substantially to the experience of pain.

□ Hyperalgesia (an increased response to a stimulus that is normally painful), allodynia (pain from a stimulus that does not normally provoke pain), and spontaneous pain (pain experienced without a stimulus) result from both peripheral and central changes after inflammation or injury. Central changes may persist after peripheral injury has resolved.

□ Pain may be acute or chronic. *Acute pain* arises from inflammation or injury to the pulp and periapex. Trigeminal neuralgia and other long-term pain of unknown origin are chronic. Acute pain is protective. It leads to avoidance and escape to prevent or minimize tissue damage. When it continues, it forces an injured area to be rested. *Chronic pain* is nonprotective. It may continue long after an injury has healed or may not be associated with an injury.

Box 5-2 What Is Central Sensitization?

□ Prolonged nociceptive input leads to functional alterations in subnucleus caudalis, the spinal dorsal horn, and probably the thalamus

□ Lower thresholds (hyperalgesia)

□ Wider receptive fields

□ Spontaneous activity

□ Recruitment of nonpain fibers (allodynia)

□ One of the major changes is the upregulation of NMDA receptors on the second-order neuron

NMDA, N-Methyl d-aspartate.

Pain is a multifactorial experience subject to modulation. The basic mechanism of nociception is well established. The pulp is innervated largely by nociceptive fibers, either Aδ (fast-conducting, sharp pain) or C fibers (slow-conducting, dull throbbing pain). During inflammation, the C fibers dominate and pulpal pain is characteristically dull, throbbing, and poorly localized. There are also some Aβ fibers in the pulp that are not normally nociceptive but may be recruited to the pain system following tissue injury as a component of central sensitization.

The periodontal ligament (PDL) has a much greater large fiber innervation than the pulp and many of these fibers are mechanosensitive, which explains why pain from the tooth is more easily localized when inflammation has spread into the supporting tissues. First-order nociceptive fibers connect with second-order neurons in the dorsal horn of grey matter in the spinal cord or into its equivalent in the brainstem, the subnucleus caudalis of the trigeminal system. This is a key relay; it is here that much of the modulation of pain will take place. Descending tracts from midbrain areas (locus coeruleus, periaqueductal grey, and raphe magnus) can by endogenous opioid mechanisms reduce or eliminate the activity from traveling further centrally. The degree to which this mechanism is involved is variable but much of the affective/motivational component of the pain experience is explained by descending modulation. This explains why similar levels of tissue damage may be related to very different levels of pain.

Sustained and continuous noxious input may cause changes in the second order neurons, reducing their threshold and increasing their receptive fields.[18,19] These are elements of central sensitization, a group of changes that contribute to the presentation of long-term and chronic pain (see Box 5-2). Central sensitization explains the nature of hyperalgesia, allodynia, and spontaneous pain. Once sensitized, these second-order neurons may be activated by inputs from multiple areas converging on them, allowing for the phenomenon of referred pain (Box 5-3).

Box 5-3 What Are Key Features of Referred Pain?

□ Never crosses the midline.

□ Can be referred from other teeth or extraoral structures.

□ Anesthetizing the true origin reduces or eliminates the pain.

□ Common, not a rare occurrence.

With referred pain, the pain originating in a tooth can seem to come from another tooth or another area, even outside the mouth.[2,20-24] It also allows teeth to appear painful when the true origin of the pain is in another tooth, in the neuromuscular system (Box 5-4), in the upper respiratory tract, or even in cardiac muscle.

Box 5-4 Case: Pain Referred to Muscles of Mastication

COMPLAINT

Moderately severe but continuous dull ache in left lower jaw.

HISTORY

The pain has lasted several weeks and is most severe in the morning. It is not intensified by hot and cold stimuli and is relieved temporarily by mild analgesics. A three-unit mandibular-posterior bridge had been placed a few months earlier. There was neither recent acute infection nor trauma. The referring dentist had completed root canal treatment on the premolar abutment with no change in symptoms.

EXAMINATION

No visible or palpable intraoral soft tissue abnormalities are present. All teeth on the left side respond within normal limits to vitality tests and percussion. Radiographs show no hard tissue lesions. Discomfort is not relieved by an inferior alveolar nerve block. Palpation of the left masseter muscle shows it to be acutely tender, particularly on the anterior border. Occlusal examination shows imbalances and premature contacts on the left side. Injecting a local anesthetic into the tender region of the muscle relieves the pain.

DIAGNOSIS

Acute myofascial pain in the left masseter muscle after dental treatment.

ETIOLOGY

Afferents from the muscle (probably in tendons or fascia) converge on the same second-order neuron in the brainstem trigeminal nucleus as periodontal afferents from the mandibular abutment teeth. The higher centers to which the second-order neuron projects are unable to differentiate between the two inputs. The higher nerve centers “assume” that the new input (from the muscle) originates from the same site as the original input (the teeth).

TREATMENT

A night guard was worn for 1 week, followed by occlusal adjustment. Short-term splint therapy was necessary to ensure that occlusal changes did not occur while the muscle was in spasm.

When neurons from several teeth or other structures converge on a second-order neuron that is sensitized, non-nociceptive levels of activity from these structures may induce firing and activity in the higher levels of the pain system. The higher centers may then identify this as painful activity in these areas. The reverse is also true. Painful input from, for example, the maxillary sinus may seem to originate from a tooth (Box 5-5).

Box 5-5 Sites from Which Pain Is Commonly Referred to Teeth

- □ Other teeth[23]
- □ Muscles of mastication[22]
- □ Sinuses/respiratory system[24]
- □ Cardiac muscle[1]

The Pain Referral Phenomenon

The pain referral phenomenon must be taken into account when making a diagnosis. The identification and location of an injured tooth should be straightforward in the early stages of the injury when activity in nociceptors predominates. Identification of the source will be more complex in the longer term when modulation can modify the presentation by the referral of pain (Box 5-6).

Box 5-6 Pain Issues in Endodontics

- □ Pain may be referred from another site.
- □ The true origin of the pain is often silent.
- □ Tooth pain is not clearly related to the condition of the pulp.
- □ Pain may be referred to the site of recent dental treatment.
- □ Pulp pain is often poorly localized.
- □ Periodontal pain is more easily localized than pain from the pulp.
- □ Recent upper respiratory tract infections may refer sinus pain to the pulp.
- □ Stress and insomnia are often related to bruxism and temporomandibular disorder pain, which can be referred to the pulp.
- □ Painful pulps are difficult to anesthetize.
- □ Patients in pain often have used analgesics.
- □ There may be more than one source of pain.
- □ Different treatment strategies may be needed to control different pain presentations.

When pain is one of the complaints, the following questions should be asked:

1. When did the pain begin?
2. Where is the pain located?
3. Is the pain always in the same place?
4. What is the character of the pain (short, sharp, long lasting, dull, throbbing, continuous, occasional)?
5. Does the pain prevent sleeping or working?
6. Is the pain worse in the morning?
7. Is the pain worse when you lie down?
8. Did or does anything initiate the pain (trauma, biting)?
9. Once initiated, how long does the pain last?
10. Is the pain continuous, spontaneous, or intermittent?
11. Does anything make the pain worse (hot, cold, biting)? Does anything make the pain better (cold, analgesics)?

If the complaint is swelling or includes swelling, a similar list of questions such as the following should be asked:

1. When did the swelling begin?
2. How quickly has the swelling increased in size?
3. Where is the swelling located?
4. What is the nature of the swelling (soft, hard, tender)?
5. Is there drainage from the swelling?

6. Is the swelling associated with a loose or tender tooth?

If a fractured tooth is part of the complaint, the time and nature of the trauma should be determined, particularly whether other teeth were involved in the trauma even though they were not visibly damaged. Was there any injury to lips or gingiva? Similarly, with a loose or discolored tooth, the time when it was first noticed should be recorded along with any history of any trauma that might be involved and whether it was recently restored. A bad taste can result from a number of causes, but the most significant from an endodontic viewpoint arises from purulence draining through a sinus tract from a chronic periapical or periodontal abscess. The patient should be questioned as to when this drainage was first noticed, whether it was preceded by pain or swelling, and whether there is a loose or tender tooth.

At this point, it may be possible to generate a tentative diagnosis from the history. This is really a hypothesis that the objective component of the examination will test. The clinician's mind must remain open to other possibilities.

Objective Examination

During this stage, extraoral and intraoral tissues are examined, tested, and compared bilaterally for the presence or absence of pathosis.

Extraoral Examination

General appearance, skin tone, facial asymmetry, swelling, discoloration, redness, extraoral scars or sinus tracts, and lymphadenopathy are indicators of physical status. A careful extraoral examination helps to identify the cause of the patient's complaint, as well as the presence and extent of an inflammatory reaction in the oral cavity or even extraoral (Figure 5-3).

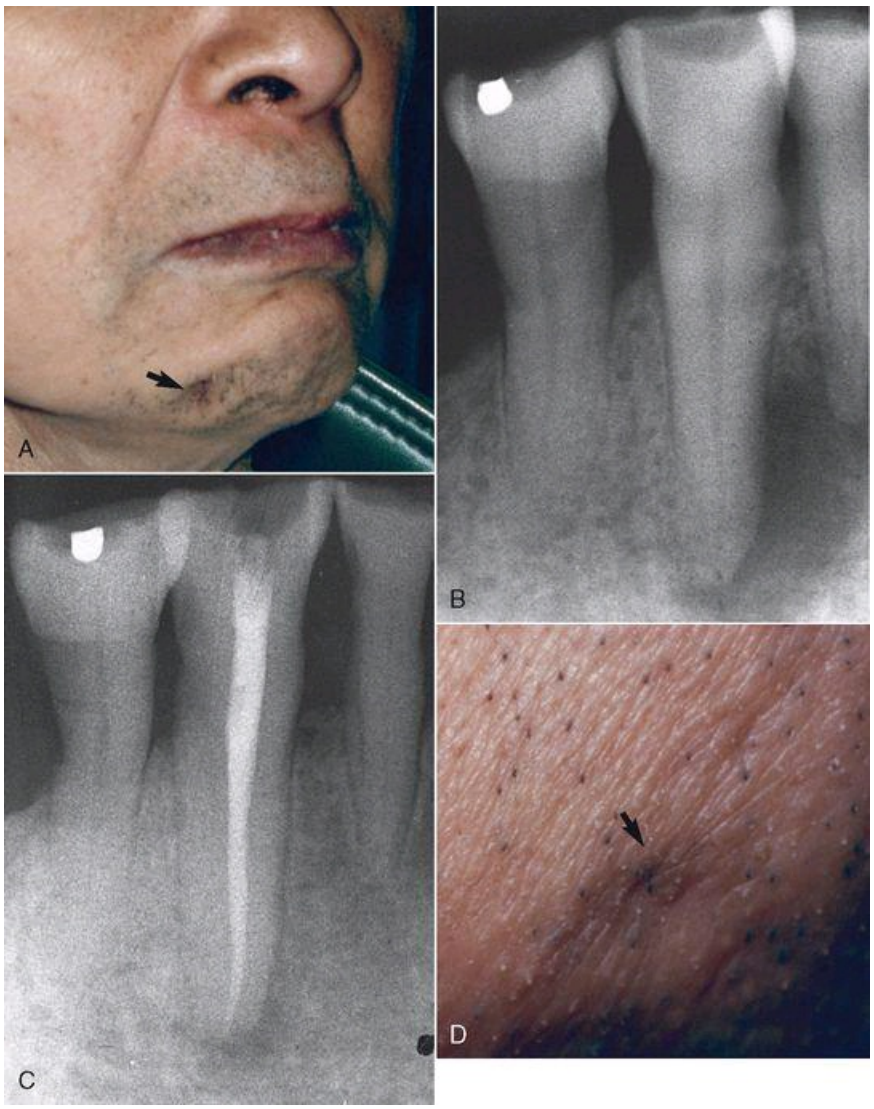


Figure 5-3 Extraoral sinus tract. **A,** This surface lesion *(arrow)* was misdiagnosed and treated unsuccessfully by a dermatologist for several months. Fortunately, the patient's dentist then recognized it to be a draining sinus tract and its source was a mandibular anterior tooth. **B,** The pulp was necrotic because of severe attrition with pulp exposure. **C**, After proper root canal treatment only, **D**, the sinus tract and surface lesion resolved completely (*arrow*).

Intraoral Examination

Soft Tissue

Soft tissue examination includes a thorough visual, digital, and probing examination of the lips, oral mucosa, cheeks, tongue, periodontium, palate, and muscles. These tissues are evaluated, and abnormalities are noted. Alveolar mucosa and attached gingiva are examined for the presence of discoloration, inflammation, ulceration, and sinus tract formation. Sinus tracts are common. A stoma (parulis) usually indicates presence of a necrotic pulp or chronic apical abscess (Figure 5-4) and sometimes

a periodontal abscess. Gutta-percha placed in the sinus tract occasionally assists in tactile and radiographic localization of the source of these lesions. Probing determines the presence of deep periodontal defects (Figure 5-5).

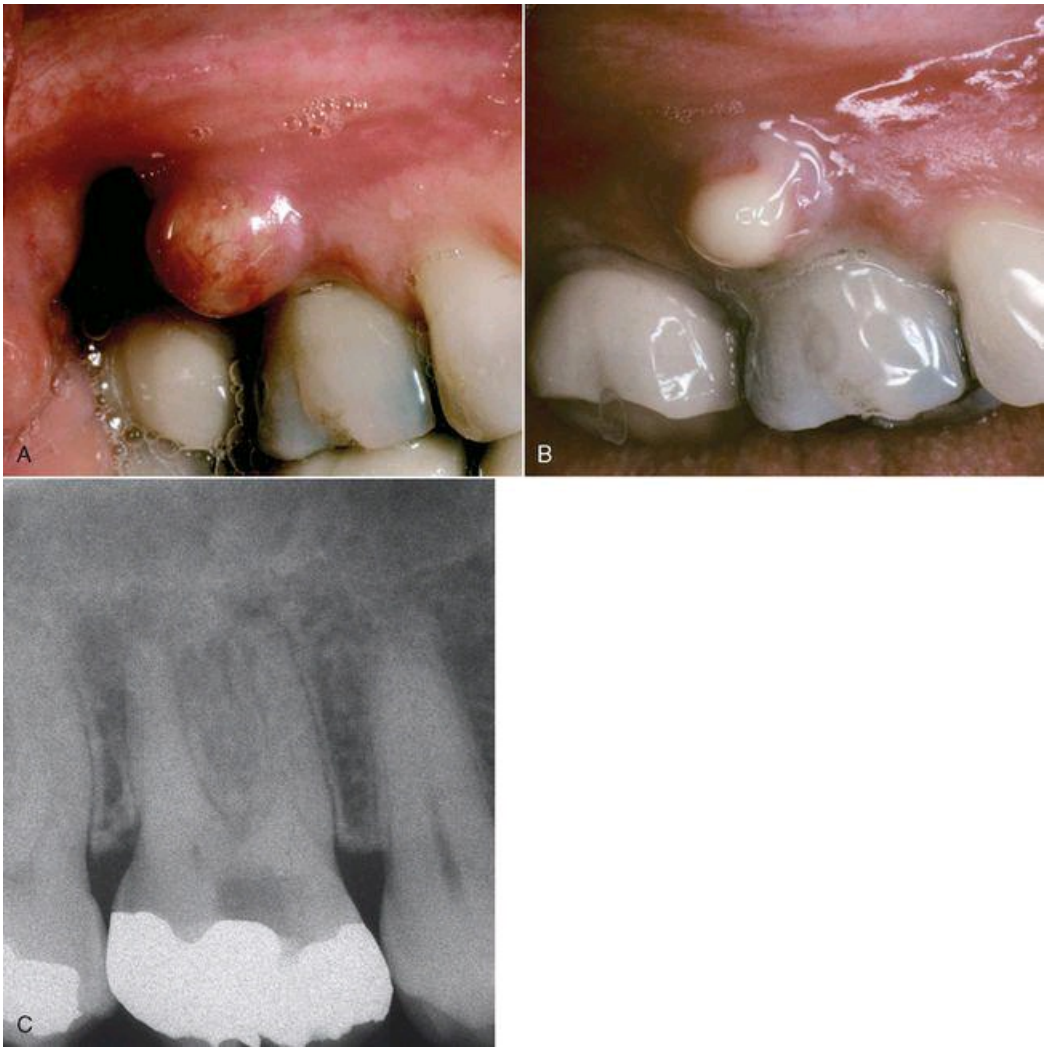


Figure 5-4 Sinus tract and parulis. **A,** Asymptomatic, intraoral swelling on mucosa near first molar. **B,** Purulence can be expressed. **C,** The first molar is nonresponsive and there is a radiolucency apical to the mesiobuccal root.

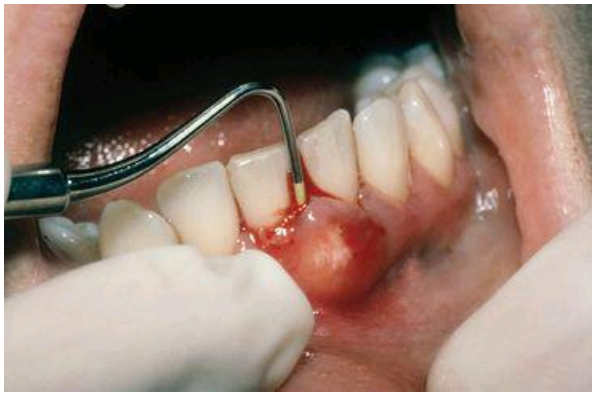


Figure 5-5 Periodontal probing reveals a deep defect. Pulp necrosis suggests that this lesion is endodontic and not periodontic.

Dentition

Teeth are examined with a mirror and an explorer for discolorations, fractures, abrasions, erosions, caries, failing restorations, or other abnormalities. A discolored crown is often pathognomonic of pulpal pathosis or is the sequela of earlier root canal treatment. Although in some cases the diagnosis is very likely known at this stage of examination, a prudent diagnostician should never proceed with treatment before performing appropriate confirmatory clinical and radiographic examinations.

Clinical Tests

There are a number of special tests that can be applied to individual teeth suspected of pathologic change. These tests have inherent limitations; some cannot be used on each tooth and the test results themselves may be inconclusive. The data they provide must be interpreted carefully and in conjunction with all the other information available. Importantly, these are *not* tests of *teeth;* they are tests of a patient's *response* to a variety of applied stimuli, which can be highly variable.

Control Teeth

In using any test, it is important to include control teeth of similar type (e.g., upper molar, lower incisor) to the suspect tooth or teeth. The result of tests on these teeth "calibrates" and provides a baseline for the patient's responses to tests on suspected teeth. The patient should not be told whether the tooth being tested is a control or suspect tooth. The clinician should be aware that a patient may not respond in the same way or to the same extent when tests are repeated. The first application of the test is the most significant.

The tests used fall into two groups, percussion and palpation, which look at the condition of the supporting tissues. Vitality tests provide information on the condition of the pulp.

Percussion and Palpation

Percussion is performed by tapping on the incisal or occlusal surface of the tooth with the end of a mirror handle held either parallel or perpendicular to the crown. This should be preceded by gentle digital pressure to detect teeth that are very tender and should *not* be tapped with the mirror handle. If a painful response is obtained by pressing or by tapping on the crown, this indicates the presence of periapical inflammation. If a painful response is obtained by tapping on the facial surface but not the occlusal/incisal surface, periodontal inflammation is suspected. The pain related to *periodontal inflammation* is likely to be in the mild-to-moderate range. *Periapical inflammation* is more often a sharp pain. An additional approach, useful if the patient complains of pain on chewing, is the biting test in which the patient bites down on a cotton swab between each tooth in turn (Figure 5-6).

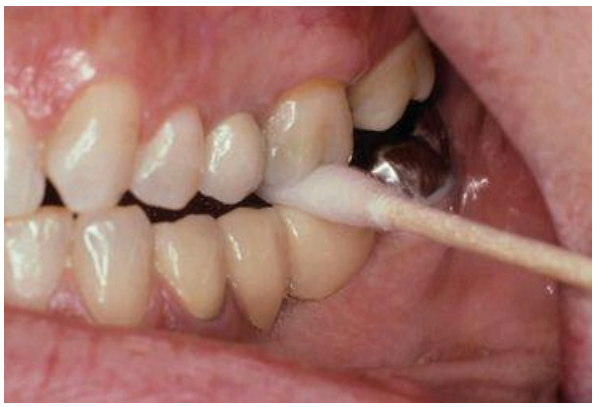


Figure 5-6 Biting test. Firm pressure on a cotton swab that produces definite pain is a good indicator of apical periodontitis.

Neighboring teeth, as well as contralateral control teeth, should also be percussed. Teeth adjacent to the diseased tooth also often show some tenderness because of the local spread of algesic cytokines.

At the same time that the tooth is being percussed, its mobility should be estimated by placing a finger lightly on the lingual surface of the tooth and pushing on the facial surface with the end of the mirror handle. The degree of movement can be visualized and felt tactilely. A healthy periodontium allows movement of only a fraction of a millimeter. Increased mobility is usually the result of periodontal disease, but in teeth with periapical inflammation, the tooth may be elevated in the socket, allowing greater movement. This movement will reduce after the periapical problem resolves. Teeth with advanced periodontal disease are poor candidates for endodontic treatment.

Palpation is firm pressure on the mucosa overlying the apex. Like percussion, palpation determines how far the inflammatory process has extended periapically. A painful response to palpation indicates periapical inflammation (see Figure 5-6).

Pulp Vitality Tests

Vitality tests are an important, often critical, component of the examination. Studies comparing the histologic condition of the pulp to the results of vitality tests have shown that there is only a limited correlation between the two. As a consequence, the results of these tests require very careful interpretation. There are several types of vitality tests, and each can be applied by a

variety of techniques. Not all tests are appropriate for every case, and not every type of test has the same level of reliability. There are five basic types of vitality tests. Four apply either a cold, hot, electrical, or dentin stimulus to the tooth; the patient's verbal response is recorded. The fifth attempts to measure pulpal blood flow on the principle that blood flow increases in inflamed tissue. Selecting which tests to use is based on the patient's complaint and reliability and should be the one that reproduces that stimulus.

An electric pulp test, conducted correctly, can usually determine whether there is vital tissue within the tooth. It cannot determine whether that tissue is inflamed nor can it indicate whether there is partial necrosis. The cold test can also detect vital tissue and does provide an indication of inflammation, although not a very accurate one. The heat test is the least reliable. Measuring blood flow is difficult under routine conditions, but as the technology improves may become less difficult.

Selecting the Appropriate Pulp Test

The selection depends on the situation. Additional meaningful information is collected when stimuli similar to those that the patient reports will provoke pain are used during clinical tests. When cold (or hot) food or drink initiates a painful response, a cold (or hot) test is conducted in place of other vitality tests. Replication of the *same* symptoms in a tooth often indicates the offender. Overall, electrical stimulation is similar to cold (refrigerant) in identifying pulp necrosis; heat is the least reliable stimulus.

When other tests are inconclusive or cannot be used and a necrotic pulp is suspected, dentin stimulation with a *test cavity* is helpful. For example, a tooth with a porcelain-fused-to-metal crown often cannot be tested accurately by standard thermal or electrical tests. After careful subjective examination and an explanation of the nature of the test to the patient, preparation without anesthesia is done with a small, sharp bur. With a vital pulp, the surface of the restoration or the enamel can be penetrated without too much discomfort. If the pulp is vital, there will be a sudden sensation of pain when dentin is reached. In contrast, if discomfort or pain is absent, the pulp is probably necrotic; an access preparation has already been started and the procedure may be continued.

Cold Tests[25-29]

Three methods are generally used for cold testing: regular, carbon dioxide (CO_2) ice (dry ice), and refrigerant. CO_2 ice requires special equipment (Figure 5-7), whereas refrigerant in a spray can is more convenient (Figure 5-8). Regular ice delivers less cold and is not as effective as refrigerant or CO_2 ice. One study found that refrigerant sprayed on a large cotton pellet was the most effective in reducing temperature within the chamber under full-coverage restorations.[25] Overall, refrigerant spray and CO_2 ice are equivalent for pulp testing.

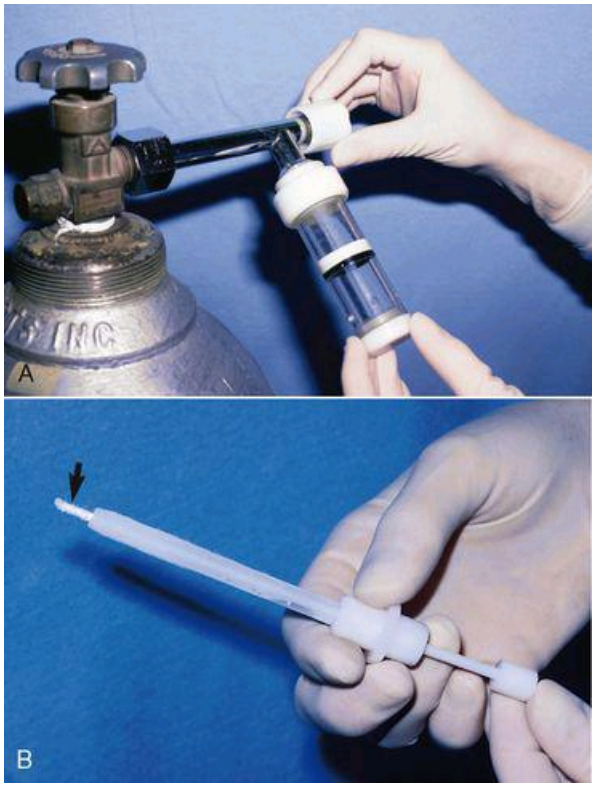


Figure 5-7 Carbon dioxide ice testing. **A,** A carbon dioxide tank and special "ice maker" are required. **B,** A carbon dioxide ice stick is formed *(arrow)*. The stick is held in gauze and touched to the facial surfaces of suspect and control teeth. This technique can be used in teeth with various types of restorations.

(Courtesy Dr. W. Johnson.)

Figure 5-8 A, Refrigerant is available in a pressured can. **B,** Refrigerant sprayed on a large cotton pellet is convenient and effective for determining pulp responsiveness.

After the tooth is isolated with cotton rolls and dried, an ice stick or large cotton pellet saturated with refrigerant is applied. This stimulus applied over a vital pulp usually results in sharp, brief pain. This short response may occur regardless of pulp status (normal or reversible or irreversible pulpitis). However, an intense and prolonged response is usually taken to indicate irreversible pulpitis. In contrast, necrotic pulps do not respond. A *false-negative* response is often obtained when cold is applied to teeth with calcified canals, whereas a *falsepositive* response may result if cold contacts gingiva or is transferred to adjacent teeth with vital pulps.

Cold is more effective on anterior than posterior teeth. Lack of a response to cold on a posterior tooth indicates another type of vitality test (electrical) should be included.

Heat Tests[30-32]

Teeth are best isolated by a rubber dam to prevent false-positive responses. Various techniques and materials are used. The best, safest, and easiest technique is to rotate a dry rubber prophy cup to create frictional heat (Figure 5-9) or to apply hot water. Gutta-percha heated in a flame can be applied to the facial surface of the tooth after first coating the surface of the crown with petroleum jelly. A flame-heated instrument is difficult to control, and its use is best avoided. Battery-powered devices are better controlled and will deliver heat safely and effectively.

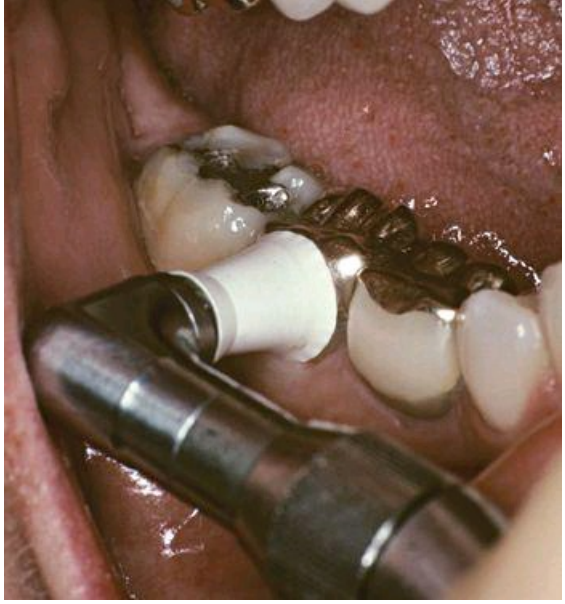


Figure 5-9 A prophy cup run at high speed without lubricant generates controlled heat for pulp testing.

Heat is not used routinely but is helpful when the major symptom is heat sensitivity, and the patient cannot identify the offending tooth. After applying heat, the temperature is gradually increased until pain is elicited. As with cold, a sharp and nonlingering pain response indicates a vital (not necessarily normal) pulp.

Electrical Pulp Testing[31,33]

All the electrical pulp testers currently available produce a high-frequency electrical current whose amperage can be changed. These testers are also *monopolar,* which means that the current flows from the probe through the tooth and then through the patient back to the testing unit. Thus nerves in any part of the pulp will be stimulated. In the past, *bipolar* units with probes that could be placed on either side of the tooth crown were available. This arrangement ensured that the current passed through the crown of the tooth and only stimulated vital pulp tissue there. Although this is useful information and possibly more useful than the results from a monopolar tester, their use has never been widespread because of the difficulty of use; none are available commercially.

Electrical pulp testers with digital readouts are popular (Figure 5-10). These testers are not inherently superior to other electrical testers but are more user-friendly. High readings indicate necrosis. Low readings indicate vitality. Testing normal control teeth establishes the approximate boundary between the two conditions. The exact number of the reading is of no significance and does not detect subtle degrees of vitality, nor can any electrical pulp tester indicate inflammation.

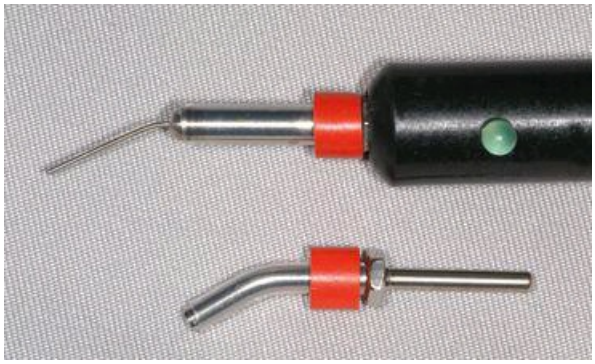


Figure 5-10 Two types of electrodes are available. The lower is a conventional tip. The upper is a “minitip,” which is useful when small areas of tooth structure are available for electrode contact.

All electrical pulp testers are used in a similar manner. It is important to clean, dry, and isolate the teeth. The surface is scrubbed with a cotton roll, isolated with the same roll, and dried thoroughly with the air syringe. A small amount of toothpaste is placed on the electrode. The electrical circuit is completed by using a lip clip or having the patient touch the metal handle. The electrode is placed on the facial or lingual surface (Figure 5-11), and the level of current gradually increased until the patient detects it. The sensation may be tingling, stinging, “full,” or hot. The presence of a response usually indicates vital tissue, whereas the absence of such a response usually indicates pulpal necrosis. The electrical pulp tester is not infallible and may produce false-positive or false-negative responses 10% to 20% of the time.[31] Calcified canals in particular may lead to a false-negative response.

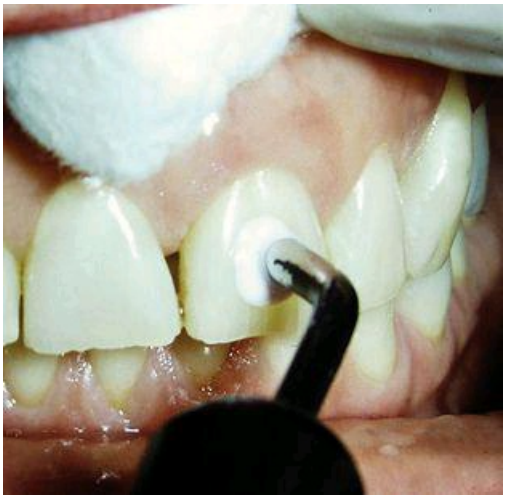


Figure 5-11 The tooth surface is carefully scrubbed, dried, and isolated. A small spot of conductive medium is placed on the electrode, which is applied to tooth structure.

Blood Flow Determination[34-39]

Instruments that detect pulp circulation are part of a developing technology that is likely to produce new approaches for determining the presence of vital pulp tissue and possibly the extent of any inflammation present. Sensors are applied to the enamel surface, usually on both the facial and lingual nerves. Blood flow is shown by beams of light (dual wavelength spectrophotometry),[37] pulse oximetry,[38,39] or laser Doppler flowmetry.[35-37] Blood components are demonstrated by detecting oxyhemoglobin levels in blood or pulsations in the pulp. These approaches are still more experimental than clinically practical, and the devices are expensive. As the technology improves and becomes less costly and more experience is gained, their use as sensitive pulp testers in the future is likely.

Significance of Thermal Tests

An exaggerated and lingering response is accepted as an indication of irreversible pulpitis. Absence of response in conjunction with other tests compared with results on control teeth usually indicates pulpal necrosis.

One test alone is seldom conclusive for the presence or absence of pulpal or periapical disease. The data obtained from vitality testing must be processed in conjunction with the history of the complaint, the intraoral examination, and radiographs.

Periodontal Examination

Periapical and periodontal lesions may mimic each other and therefore require differentiation (see Chapter 6). It is also important to establish the periodontal health of the tooth or teeth as part of overall treatment planning.

Probing

Probing is an important clinical test that is often overlooked and underused for diagnosing periapical lesions. Bone and periodontal soft tissue destruction are induced by both periodontal disease and periradicular lesions and may not be easily detected or differentiated radiographically.

A periodontal probe determines the level of connective tissue attachment. Also, the probe penetrates into an inflammatory periapical lesion that extends cervically. Probing is a diagnostic aid that has prognostic value. The prognosis for a tooth with a necrotic pulp that induces cervically extending periapical inflammation is good after adequate root canal treatment. However, the outcome of root canal treatment on a tooth with severe periodontal disease usually depends on the success of periodontal treatment. Teeth with marked periodontal disease involvement are poor candidates for endodontic procedures. Probing depths should be recorded for future comparison.

Mobility

The mobility test partially determines the status of both the PDL and the prognosis. Teeth with extreme mobility usually have little periodontal support. Occasionally an extensive periapical lesion may alter the periodontal support markedly; mobility usually decreases dramatically after successful root canal treatment.

Mobility is determined by placing the index finger on the lingual aspect and applying pressure with the mirror handle on the opposite facial surface. Movement of more than 2 to 3 mm or depression indicates that the tooth is a poor candidate for root canal treatment if the mobility is due primarily to periodontal disease and not to apical pathosis.

Radiographic Examination[40-44]

Radiographs are essential for the examination of mineralized tissue. Their value, however, is often overestimated. During an examination, clinicians will often study a radiograph first and do little in the way of a visual and digital examination. This is a mistake. The logical place for radiographic observation is after the full history and direct examination. Then the data derived are interpreted as part of the overall examination rather than as a replacement. Radiographs are compressed 2-dimensional images in which much detail may be hidden by superimposition. This limitation can be reduced by making more than one radiograph at different angles. Recent advances in technology allow 3-dimensional (3D) techniques, such as 3D computed tomography (CT), to be explored experimentally and should soon become available for clinical practice.

Radiographs allow the evaluation of carious lesions, defective restorations, root canal treatments, abnormal pulpal and periapical appearances, malpositioned teeth, the relationship of the neurovascular bundle and maxillary sinuses to the apexes, and any bone loss from periodontal disease. They may also reveal structural changes and bony disease unrelated to the teeth (Figure 5-12).

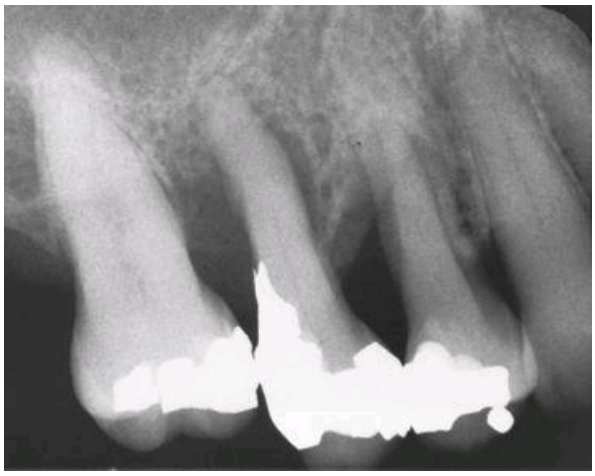


Figure 5-12 Horizontal, as well as vertical, bone loss is evident in this quadrant. All teeth are responsive to vitality tests; therefore the resorptive defects represent a severe periodontal condition and not pulp or apical pathosis. Root canal treatment is not indicated.

Periapical Lesions[45-53]

The primary value of radiographs in endodontic diagnosis is to determine the health of the periapical tissues. Inflammation results in bone resorption and creation of a radiolucent area (a “lesion”) around the apex. There are, however, other radiolucencies that are either normal or pathoses of nonendodontic origin. Periapical lesions of endodontic origin usually have four characteristics: (1) the lamina dura of the tooth socket is lost apically, (2) the lucency remains at the apex in radiographs made at different cone angles, (3) the lucency tends to resemble a hanging drop, and (4) the tooth usually has a necrotic pulp (Figure 5-13).

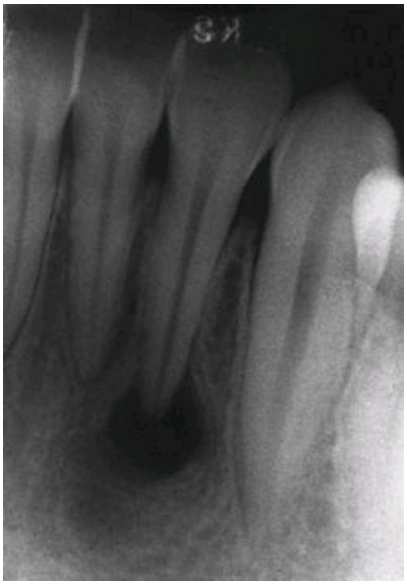


Figure 5-13 Characteristic appearance of an endodontic lesion. This radiograph shows loss of lamina dura and a hanging-drop appearance. There was a history of trauma in this patient. A film made at a different angle would show the lesion remaining at the apex.

(Courtesy Dr. L. Wilcox.)

A sizable radiolucency in the periapical region of a tooth with a vital pulp is *not* of endodontic origin and is either a normal structure or nonendodontic pathosis.

Radiopaque changes also occur. Condensing osteitis is a reaction to pulp or periapical inflammation and results in an increase in the density of bone. It has a diffuse circumferential medullary pattern with indistinct borders (Figure 5-14). The well-circumscribed, more homogeneous normal structure that occurs commonly in the mandibular posterior region is enostosis (or sclerotic bone); this is a nonpathologic condition. Similarly, tori may sometimes be seen over the roots of maxillary molars. Contrary to popular belief, an opaque (corticated) margin to a lesion does not necessarily indicate a cyst.[54]

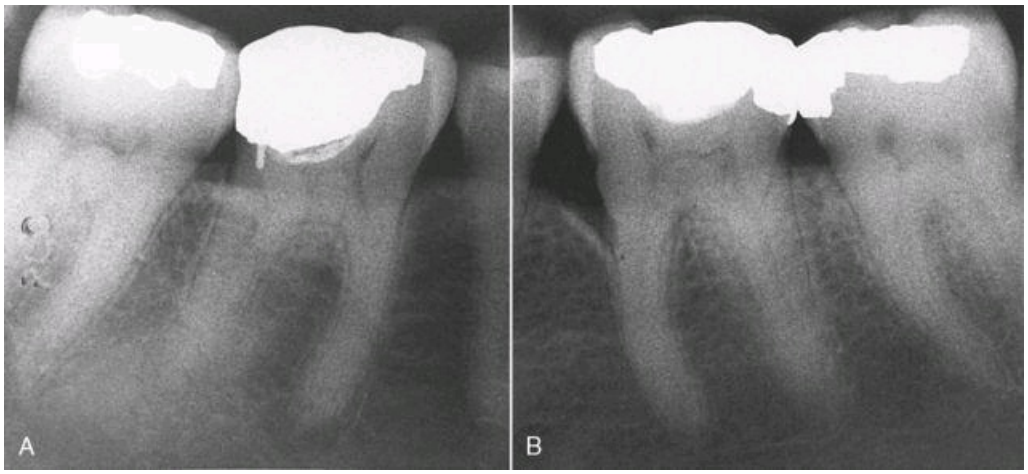


Figure 5-14 Condensing osteitis. **A,** Surrounding the distal root apex is diffuse trabeculation. **B,** This contrasts with the contralateral molar, which demonstrates a normal, sparse trabecular pattern.

New radiographic technology permits early detection of bony changes, allowing new approaches to differential diagnosis.[55,56] Digital subtraction radiography "reads" subtle early changes with periapical bone resorption. Digital radiometric analysis and CT scans may differentiate bony pathoses such as granulomas and cysts. RadioVisioGraphy and magnetic resonance imaging demonstrate changes of resorptive lesions over time. However, these technologies are neither perfected nor cost effective. Plus, at the moment, they do not offer significant diagnostic benefits for routine clinical application. Undoubtedly, these or similar devices will prove useful in the future.

Pulpal Lesions

An inflamed pulp with dentinoclastic activity may show abnormally altered pulp space enlargement and is pathognomonic of internal resorption (Figure 5-15). Extensive diffuse calcification in the chamber may indicate long-term, low-grade irritation (not necessarily of irreversible pulpitis). Dentin formation that radiographically "obliterates" the canals (usually in patients with a history of trauma) does not indicate pathosis (Figure 5-16). These teeth ordinarily require no treatment, but when treatment is necessary, they can be managed with reasonable success. Pulp stones are discrete calcified bodies found occasionally in pulp chambers and are sometimes visible on radiographs. They are of no pathological significance, although they may block access to the root canals in teeth that require endodontic treatment.

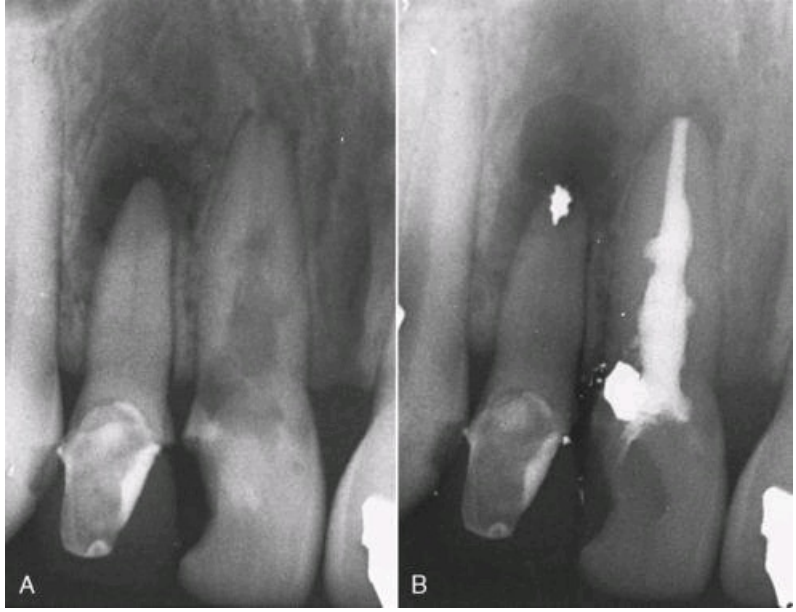


Figure 5-15 Differing pulp responses to injury. **A,** Central incisor shows extensive, perforating internal resorption; the lateral incisor has calcific metamorphosis. **B,** Special techniques manage these problems with both surgical and nonsurgical treatment.

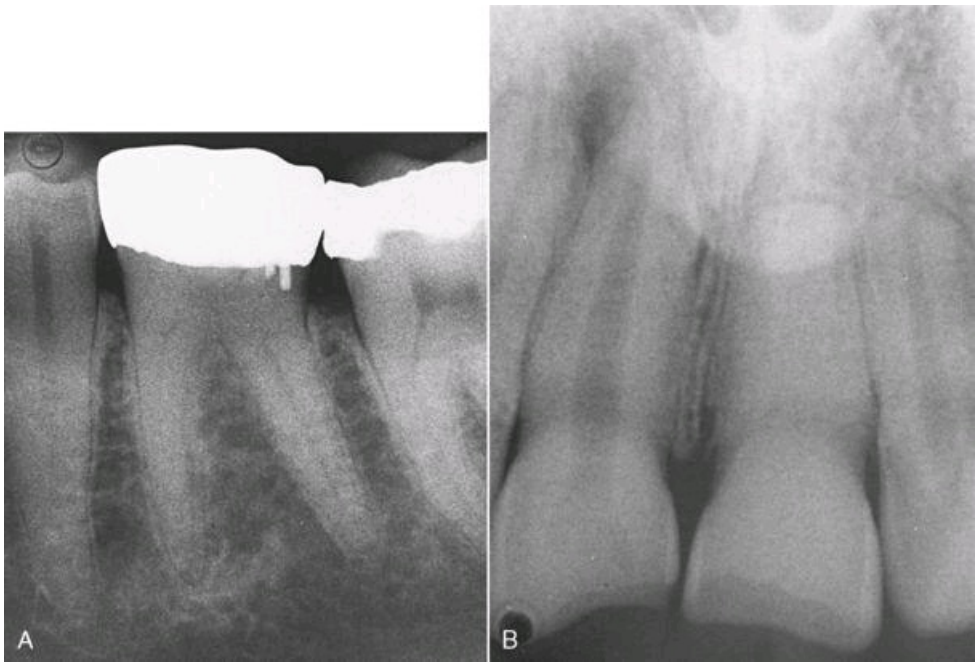


Figure 5-16 Calcific metamorphosis. **A,** This lesion has resulted from repeated insults from caries and restorations. **B,** This dentin formation ("obliteration") resulted from trauma and irritation to the pulp. Neither **A** nor **B** is pathosis. Occasionally, apical pathosis does develop; this is a treatment challenge.

*(**B** courtesy Dr. L. Wilcox.)*

Additional Diagnostic Procedures

After subjective and objective examinations and clinical tests have been completed, it is usually possible to make an accurate diagnosis and create a reasonable treatment plan. However, if special circumstances prevent making a definitive diagnosis, additional tests such as caries removal, selective anesthesia, and transillumination are options.

Caries Removal

Determining the depth of caries penetration is necessary in some situations for definitive pulp diagnosis. A common clinical situation is the presence of deep caries on radiographs with no significant history or presenting symptoms and a pulp that responds to clinical tests. All other findings are normal. The final definitive test is complete caries removal to establish pulp status. Exposure by soft caries is irreversible pulpitis; nonexposure is reversible pulpitis.

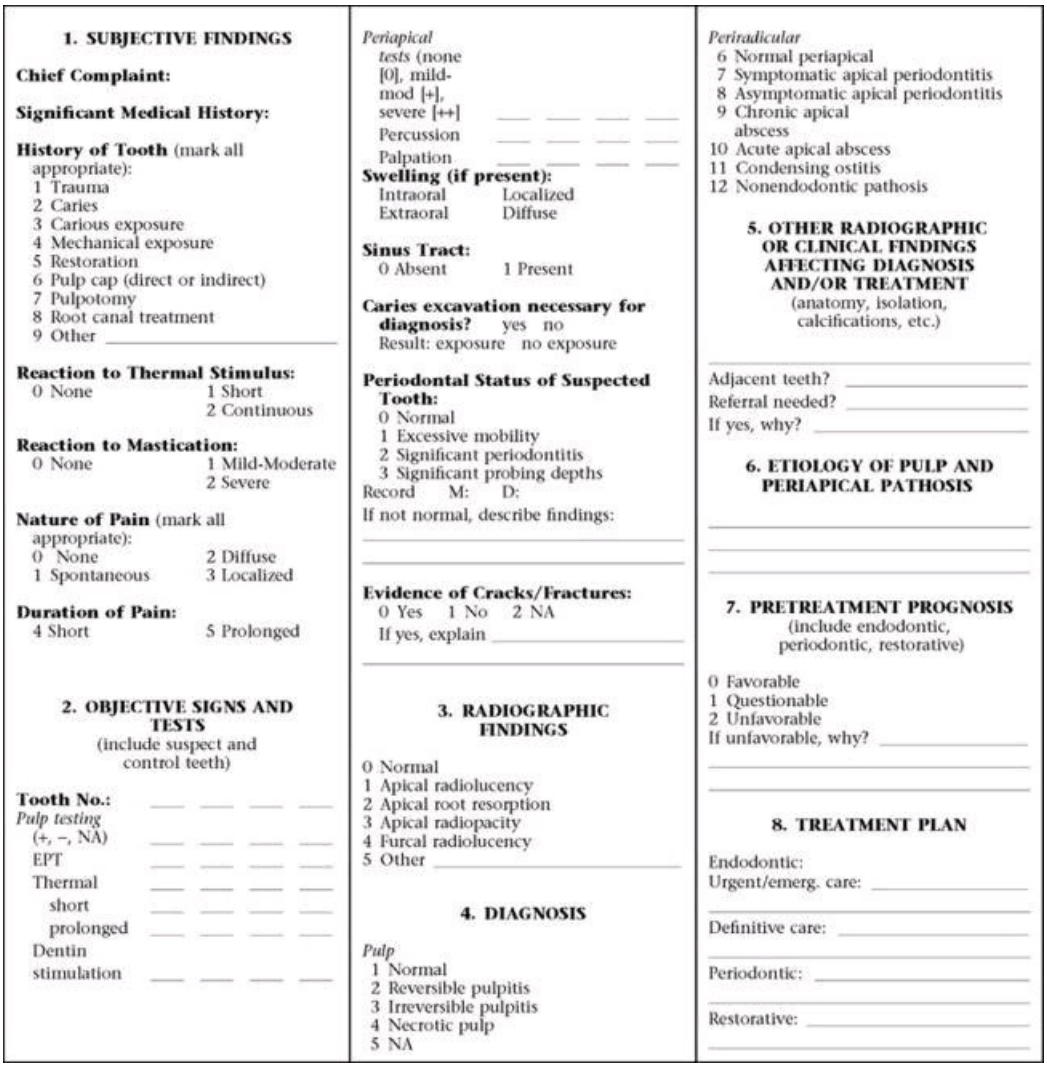
Selective Anesthesia

Selective anesthesia can be useful in localizing a painful tooth when the patient cannot identify the offender. If a mandibular tooth is suspected, a mandibular block will confirm at least the region if the pain disappears after the injection. Selective anesthesia of individual teeth is not useful in the mandible. The PDL injection will often anesthetize several teeth. It is, however, marginally more effective in the maxilla. Anesthetic should be administered to individual teeth in an anterior to posterior sequence because of the pattern of distribution of the sensory nerves.

Transillumination

Transillumination helps identify longitudinal crown fractures because a fracture will not transmit the light. This test produces contrasting vertical dark and light segments of the tooth at the fracture site. Teeth with longitudinal coronal fractures are often tender to biting, particularly if an object is placed between the cusps.

The process of conducting the history and examination takes much longer to describe than to conduct, particularly if an organized format is taken to recording it. Using a form such as in Figure 5-17 ensures that nothing is omitted, and that the various phases are carried out in a logical sequence to provide an accessible record of conditions at the first appointment. Once all the data are obtained and assembled, they must be processed.



1. SUBJECTIVE FINDINGS

Chief Complaint:

Significant Medical History:

History of Tooth (mark all appropriate):
1 Trauma
2 Caries
3 Carious exposure
4 Mechanical exposure
5 Restoration
6 Pulp cap (direct or indirect)
7 Pulpotomy
8 Root canal treatment
9 Other _____

Reaction to Thermal Stimulus:
0 None 1 Short 2 Continuous

Reaction to Mastication:
0 None 1 Mild-Moderate 2 Severe

Nature of Pain (mark all appropriate):
0 None 2 Diffuse
1 Spontaneous 3 Localized

Duration of Pain:
4 Short 5 Prolonged

2. OBJECTIVE SIGNS AND TESTS
(include suspect and control teeth)

Tooth No.: ___ ___ ___ ___
Pulp testing (+, –, NA) ___ ___ ___ ___
EPT ___ ___ ___ ___
Thermal ___ ___ ___ ___
short ___ ___ ___ ___
prolonged ___ ___ ___ ___
Dentin stimulation ___ ___ ___ ___
Periapical tests (none [0], mild-mod [+], severe [++] ___ ___ ___ ___
Percussion ___ ___ ___ ___
Palpation ___ ___ ___ ___

Swelling (if present):
Intraoral Localized
Extraoral Diffuse

Sinus Tract:
0 Absent 1 Present

Caries excavation necessary for diagnosis? yes no
Result: exposure no exposure

Periodontal Status of Suspected Tooth:
0 Normal
1 Excessive mobility
2 Significant periodontitis
3 Significant probing depths
Record M: D:
If not normal, describe findings:

Evidence of Cracks/Fractures:
0 Yes 1 No 2 NA
If yes, explain _____

3. RADIOGRAPHIC FINDINGS

0 Normal
1 Apical radiolucency
2 Apical root resorption
3 Apical radiopacity
4 Furcal radiolucency
5 Other _____

4. DIAGNOSIS

Pulp
1 Normal
2 Reversible pulpitis
3 Irreversible pulpitis
4 Necrotic pulp
5 NA

Periradicular
6 Normal periapical
7 Symptomatic apical periodontitis
8 Asymptomatic apical periodontitis
9 Chronic apical abscess
10 Acute apical abscess
11 Condensing ostitis
12 Nonendodontic pathosis

5. OTHER RADIOGRAPHIC OR CLINICAL FINDINGS AFFECTING DIAGNOSIS AND/OR TREATMENT
(anatomy, isolation, calcifications, etc.)

Adjacent teeth? _____
Referral needed? _____
If yes, why? _____

6. ETIOLOGY OF PULP AND PERIAPICAL PATHOSIS

7. PRETREATMENT PROGNOSIS
(include endodontic, periodontic, restorative)

0 Favorable
1 Questionable
2 Unfavorable
If unfavorable, why? _____

8. TREATMENT PLAN

Endodontic:
Urgent/emerg. care: _____
Definitive care: _____
Periodontic: _____
Restorative: _____

Figure 5-17 Sample form used for diagnosis and treatment planning.

The findings are arranged in a rational order to arrive at both a pulpal and a periapical diagnosis (see Table 5-1). This process is summarized in Figure 5-17. Consistency in all findings does not always occur because of the limitations of the diagnostic techniques available and because of variations in patient responses.

The Difficult Diagnosis

Fortunately, on most occasions, if there is a thorough and methodical approach to the history and examination, the diagnosis of endodontic conditions is fairly straightforward. Some cases are more challenging and may require referral to a specialist.[57] Most often, it is the source of the patient's pain that is difficult to localize. This occurs most often with inflamed pulp tissue. Once the inflammation has reached the periodontium with its heavy supply of mechanoreceptors, localization becomes obvious. Thus, in some cases, it may be necessary to treat the patient symptomatically and wait until the source of pain becomes clear. Some findings are particularly suggestive of a difficult diagnosis. Box 5-7 lists factors that require approaching a case with more caution.

Box 5-7 What Are Indicators of a Difficult Diagnosis?

- □ Patient cannot localize the pain or the site seems to vary.
- □ No local dental cause for the pain can be identified.
- □ Pain is spontaneous or intermittent and not necessarily related to an initiating stimulus.
- □ Stimulation of a suspected tooth does not reproduce the symptoms.
- □ A suspected tooth shows no clear etiology (caries, fracture).
- □ More than one tooth seems to be involved.
- □ Symptoms are bilateral.
- □ Selective anesthesia fails to localize the source of pain.

In addition to diagnosing pathoses and determining what treatment is indicated, the approach used must fit into the patient's overall needs and treatment plan. To accomplish this, the practitioner must know the indications and contraindications for root canal therapy and recognize conditions that make treatment difficult. This knowledge, combined with the diagnosis, determines the treatment plan.

TREATMENT PLANNING

The decision as to whether a tooth requires endodontic therapy follows directly from the diagnosis (see Table 5-1). Once the need for endodontic treatment has been established, in the majority of cases, the choice of procedures is straightforward and should be intracoronal (sometimes described as "conventional" or "nonsurgical" root canal treatment). There are indications for surgical treatment when coronal access to the canal system in the apical root is impossible. Surgical endodontics and its indications are discussed in Chapter 20. The difficulty of intracoronal procedures varies considerably but should be assessed before the general practitioner considers undertaking the treatment. Once the decision has been made that treatment is needed and that the practitioner will perform it, the patient's overall treatment plan should be considered and the positioning of endodontic treatment determined. If a patient is in pain or is swollen, immediate resolution of these symptoms is imperative even if the ultimate disposition of the case is referral to a specialist. Endodontic emergencies are considered in Chapter 9.

Patients are interested in the number of appointments it will take to complete the procedure. The available evidence suggests that single and multiple appointment root canal therapy have the same success rate and the same level of posttreatment complications.[58-66] Most patients prefer single appointment treatment; this is a common practice. There are, however, situations that require more than a single appointment. Some conditions are complex or time consuming. Related to this and most important is patient management and the tolerance level of patient and operator. If fatigue or frustration on the part of either occurs, the appointment is terminated, a temporary filling placed, and another appointment scheduled.

Another situation is the patient with severe periapical symptoms or persistent canal exudation. These are often emergencies, and the tolerance level of the patient may be low. Also, flare-ups between appointments occur more often in these situations and are considerably more difficult to manage if the canals have been obturated.

Specific Treatments

Pulpitis

Normal or Reversible Pulpitis

Root canal treatment is not indicated (unless elective). In patients with reversible pulpitis, the cause is usually removed and restoration follows.

Irreversible Pulpitis

Root canal treatment with total pulp removal (pulpectomy) is the treatment of choice with irreversible pulpitis. In emergency situations and in cases where apexification is being attempted, partial pulp removal (pulpotomy) may be indicated. Intracanal medications are not beneficial and should not be used.

Necrosis

Root canal treatment is indicated when necrosis is present.

Periapical Diagnosis

Normal

No special treatment approach is required.

Symptomatic Apical Periodontitis

It is critical to remove the inflamed pulp or necrotic tissue with a diagnosis of symptomatic apical periodontitis. With the cause of the disease process removed, it will resolve.

Asymptomatic Apical Periodontitis

Treatment for asymptomatic apical periodontitis is the same as that for acute apical periodontitis. The size of the lesion seen on radiograph is of little concern. Lesions of different sizes will heal after appropriate treatment (Figure 5-18).

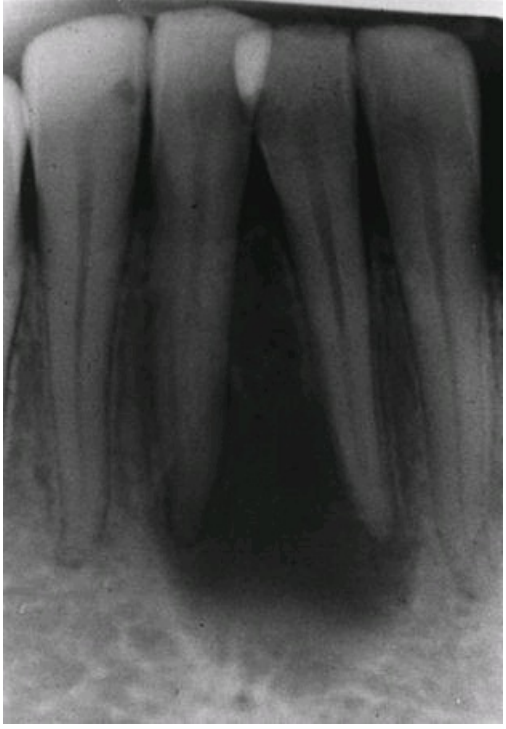


Figure 5-18 Because of its size, this lesion is likely to be an apical radicular cyst. The lesion is related to pulp necrosis in the left central incisor. Although superimposed over the apex of the adjacent incisor, the pulp is not affected and therefore does not require treatment. Proper root canal treatment of the left incisor would lead to resolution without surgery.

Acute Apical Abscess

The basic treatment for acute apical abscess is the same as for the preceding conditions with the addition that drainage of the abscess is attempted either through the tooth or by incision of the soft tissue. Occasionally, such drainage cannot be achieved, so resolution of symptoms is slow.

Chronic Apical Abscess

The basic treatment for chronic apical abscess is the same as for the previous diagnoses except that drainage has already been established naturally. The sinus tract usually resolves a few days to a month after débridement and obturation. If the sinus tract persists, there may have been a misdiagnosis and the lesion is a periodontal abscess. Calcium hydroxide should be placed in the canal(s) and in the access cavity, which is always closed between appointments.

Condensing Osteitis

This entity requires no special treatment. Because it occurs with different pulp conditions, treatment will vary. Condensing osteitis resolves in approximately 50% of teeth after *successful* root canal treatment. There is no apparent problem if the condensing osteitis does *not* resolve; no further treatment is required unless there are other findings that indicate failure.

Extraction as an Alternative to Endodontic Therapy

The only definitive alternative to root canal therapy is extraction, which may be indicated by coexisting severe periodontal disease, by cost factors, and perhaps for strategic reasons in an overall treatment plan. If pain has been present, there will be no difficulty in convincing the patient that some treatment is needed. A patient may opt for extraction but should not be allowed to do this until the clinician has described the negative sequelae of this self-destructive decision.

Patients who are asymptomatic may not be as easily guided toward endodontic therapy. Again, the best approach is to advise the patient that disease is present and likely to progress to a point where symptoms will occur and treatment may be more difficult.

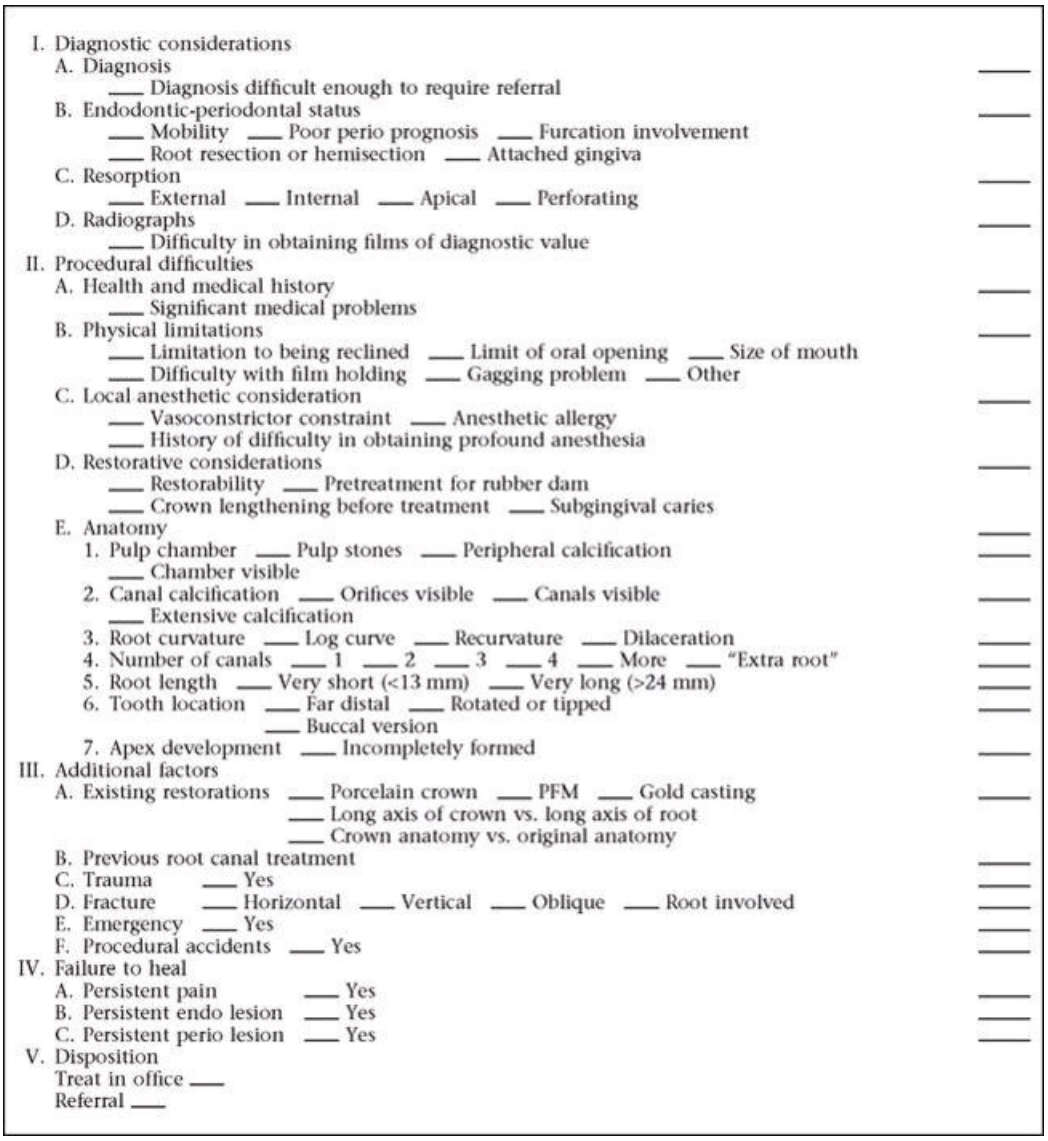
RECOGNIZING WHEN CASES SHOULD BE REFERRED

A patient deserves the best quality of care possible. Referrals are part of a comprehensive health care management and are appropriate when the patient's needs are beyond the generalist's capabilities.[67,68] By definition, *standard of care* means that the *quality of endodontic care provided to patients by the general dentist should be similar to that provided by an endodontist.* Outcome surveys show that success rates for root canal treatment are lower for generalists than for specialists.[69] One possible factor contributing to this is that some general dentists may attempt treatment that is beyond their abilities. Clearly, objective criteria for referral are needed.

Determining the Difficulty of a Case

An objective system has been developed by the American Association of Endodontists for determining the degree of difficulty of any endodontic case. It consists of a checklist of significant factors in a logical sequence. Each factor is scored on a scale of 1 to 3: 1 = uncomplicated, 2 = moderately complicated, and 3 = complicated.

Cases in which all factors score 1 can be treated by a general dentist. Cases with one or more scores of 2 may be completed by an experienced generalist or referred. Cases in which any factors score 3 should be referred to a specialist. Initially, this process may seem cumbersome, but with use it takes only a few minutes and assures a methodological approach (Figure 5-19).



I. Diagnostic considerations
A. Diagnosis ____
___ Diagnosis difficult enough to require referral
B. Endodontic-periodontal status ____
___ Mobility ___ Poor perio prognosis ___ Furcation involvement
___ Root resection or hemisection ___ Attached gingiva
C. Resorption ____
___ External ___ Internal ___ Apical ___ Perforating
D. Radiographs ____
___ Difficulty in obtaining films of diagnostic value
II. Procedural difficulties
A. Health and medical history ____
___ Significant medical problems
B. Physical limitations ____
___ Limitation to being reclined ___ Limit of oral opening ___ Size of mouth
___ Difficulty with film holding ___ Gagging problem ___ Other
C. Local anesthetic consideration ____
___ Vasoconstrictor constraint ___ Anesthetic allergy
___ History of difficulty in obtaining profound anesthesia
D. Restorative considerations ____
___ Restorability ___ Pretreatment for rubber dam
___ Crown lengthening before treatment ___ Subgingival caries
E. Anatomy ____
1. Pulp chamber ___ Pulp stones ___ Peripheral calcification ____
___ Chamber visible
2. Canal calcification ___ Orifices visible ___ Canals visible ____
___ Extensive calcification
3. Root curvature ___ Log curve ___ Recurvature ___ Dilaceration ____
4. Number of canals ___ 1 ___ 2 ___ 3 ___ 4 ___ More ___ "Extra root" ____
5. Root length ___ Very short (<13 mm) ___ Very long (>24 mm) ____
6. Tooth location ___ Far distal ___ Rotated or tipped ____
___ Buccal version
7. Apex development ___ Incompletely formed ____
III. Additional factors
A. Existing restorations ___ Porcelain crown ___ PFM ___ Gold casting ____
___ Long axis of crown vs. long axis of root
___ Crown anatomy vs. original anatomy
B. Previous root canal treatment ____
C. Trauma ___ Yes ____
D. Fracture ___ Horizontal ___ Vertical ___ Oblique ___ Root involved ____
E. Emergency ___ Yes ____
F. Procedural accidents ___ Yes ____
IV. Failure to heal
A. Persistent pain ___ Yes ____
B. Persistent endo lesion ___ Yes ____
C. Persistent perio lesion ___ Yes ____
V. Disposition
Treat in office ___
Referral ___

Figure 5-19 Case difficulty/case selection form.

Treatment Planning Considerations

Diagnosis

Appropriate treatment follows accurate diagnosis. Many procedures are done inappropriately (or not done) because of diagnostic errors. The endodontist is experienced, and the generalist may be unfamiliar with that particular problem. *Referred pain is a good example of a condition that often presents the practitioner with a significant diagnostic challenge.* Unless a definitive diagnosis is obtained, no treatment should be rendered and the patient should be referred (Box 5-8).

Box 5-8 Case: Psychogenic Pain

COMPLAINT

A 45-year-old woman was seen with a continuous, moderate, dull, and occasionally severe ache from bilateral temporomandibular joints and molars. Discomfort began with the onset of marital problems and financial hardship. Root canal treatment of tooth No. 3 provided temporary relief, as did occlusal splint therapy and pharmacologic treatment for depression. However, the discomfort returned.

EXAMINATION

Clinical and radiographic examinations of teeth show no abnormalities. Treatment of tooth No. 3 appears successful. Palpation of the temporomandibular joint reveals no abnormality. Further questioning reveals extended emotional stress after marital break-up.

DIAGNOSIS

Orofacial pain of psychogenic origin (tentative).

ETIOLOGY

Pain originates from higher nerve centers and is probably entirely affective. Various forms of treatment are only transiently effective because they affect higher central nervous system centers.

TREATMENT

Long-term relief depends on the emotional problems sustaining the central nervous system changes being removed or on the patient adopting suppressive strategies. A careful nonjudgmental explanation of psychogenic pain was given to the patient. In particular, the contribution of both organic and psychologic components to the pain experience was described. Clearly, the patient was experiencing pain, but because no organic cause was evident, interventional dental treatment would not bring long-term relief. Addressing the emotional component of the pain with the help of a physician, family, and other support services may eliminate it. The prescription of antidepressants by the physician is virtually always a component of treatment.

Endodontic-Periodontal Status

If there is doubt about whether the problem is endodontic or periodontal (or has another cause), the patient should be referred. Differential diagnosis of periodontal and endodontic lesions is discussed in Chapter 6.

Resorption

Resorption may be either internal or external. All instances of perforating (pulp-periodontal communication) resorption are complex. External resorption may take one of the following forms:

1. *Inflammatory* resorption is related to pulp necrosis. A periradicular lesion may cause apical resorption. Lateral resorption usually results from impact trauma.

2. *Replacement* resorption occurs after impact injuries (luxation or avulsion). This resorption is associated with fusion, then replacement, of tooth structure with bone.

3. *Surface* resorption is limited and not detectable clinically.

Patients with tooth resorption, whether internal or external, are high risk and should be referred for evaluation and treatment (Figure 5-20).

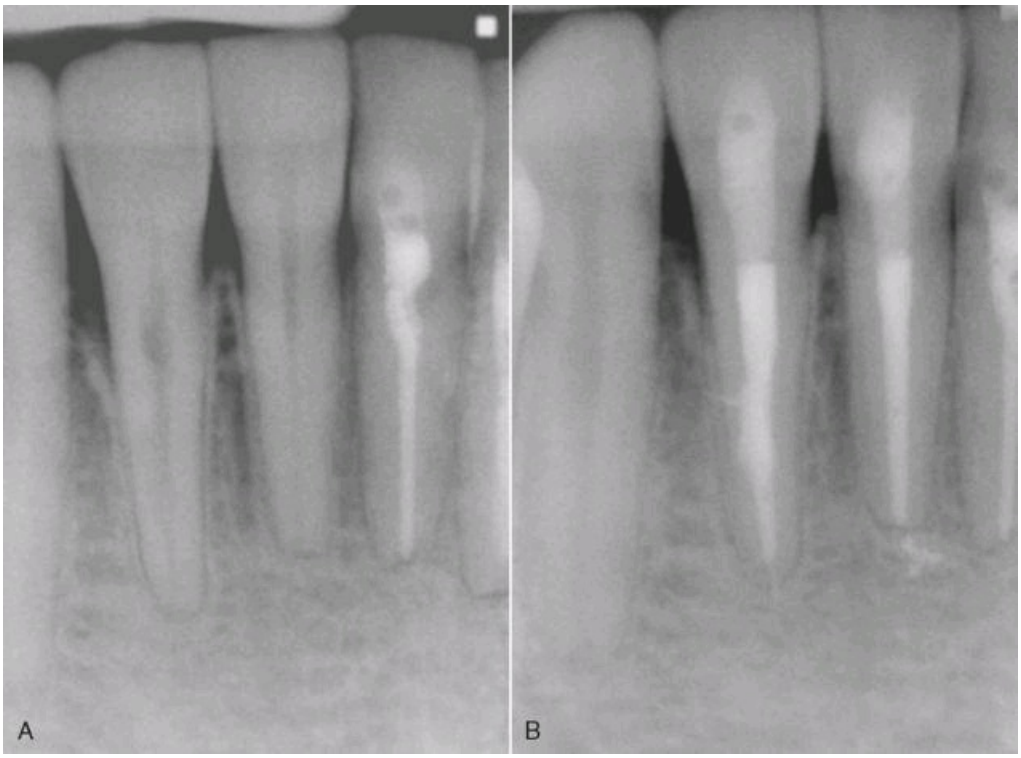


Figure 5-20 A, Complexity caused by trauma. Previous treatment of the lateral and central incisor. The other central incisor is symptomatic, with percussion sensitivity and nonresponsiveness to pulp testing. The adjacent lateral shows internal resorption *(arrow)* that is asymptomatic. Care must be taken to completely clean and shape the irregular canal system, as is demonstrated in the postoperative image. **B,** This situation exhibits a high degree of difficulty.

Limited internal resorption may not present treatment complications, but external apical resorption may drastically alter the geometry of the root canal. A structure that was once round may now be irregular and difficult to clean and seal. Extensive

apical resorption is best referred because the apical anatomy is disturbed.

Radiographs

Diagnostic and treatment films of good quality are critical. Patient characteristics, such as gagging, and oral anatomy (e.g., shallow palatal vault, large and narrow dental arches) may hinder this task.

Procedural Difficulties

Health and Medical History

General practitioners may treat patients who require premedication and patients with medical complications that affect diagnosis and treatment. However, severity must be evaluated. In patients with more serious disorders, an endodontist may best provide treatment. Specialist care is generally more expedient and offers better prevention and management of complications during treatment.

Physical Limitations

If the patient cannot be suitably reclined or if the mouth opening is such that access is compromised, referral should be considered.

Local Anesthetic Consideration

Some patients report an "allergy" to local anesthetics. A true allergic reaction to commonly used local anesthetics, particularly the amides, is almost nonexistent.[7] Tachycardia is often confused with an allergy. A patient may report adverse reactions to an anesthetic with epinephrine. This is probably apprehension and not truly an "allergy."

Difficulty with anesthesia may transform a cooperative patient into a budding dental phobic. When difficulty is encountered in obtaining profound anesthesia, referral should be considered.

Restorative Considerations

Severe caries or fractures from trauma may render the tooth difficult to isolate or restore.

Anatomy

Pulp Chamber

As the tooth ages, pulp chamber space decreases. Chamber size and pulp stones, as well as the extent of calcifications in the canal system, must be considered.

Canal Calcification

Secondary and perhaps tertiary dentin formation leads to narrowing of the canals, sometimes to an extent that they are not visible radiographically (Figures 5-21 and 5-22).

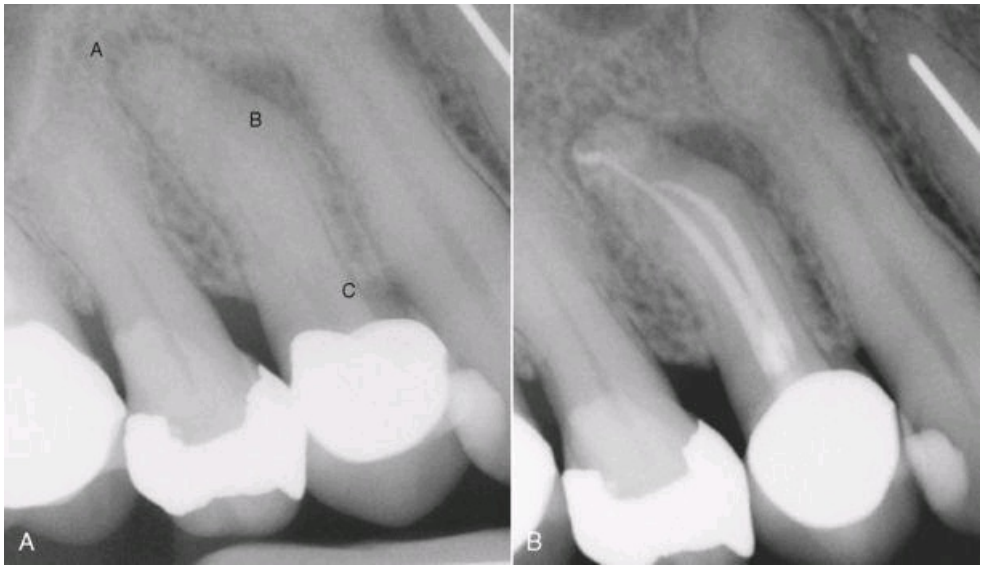


Figure 5-21 A, Periapical radiolucency (*A*) and mesial radiolucency in the apical third (*B*). The canals are calcified, the root is narrow, and there is a hint of a significant mesial concavity in the coronal third (*C*). The tooth is also crowned, thereby increasing access complexity. This case is considered as high risk. **B,** Postoperatively, the mesial radiolucency resulted from the buccal root exiting several millimeters shorter than the palatal root with a significant distal curvature. The practitioner must manage the unexpected, should problems arise during treatment.

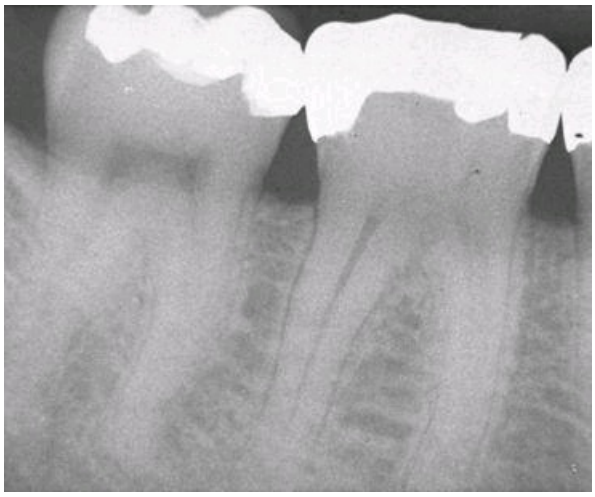


Figure 5-22 Pulp chamber and root canals show calcific metamorphosis; this situation is rated as extreme risk.

Root Curvature

Canals are rarely straight, although they may appear straight on a facial radiograph. Curvature factors include the direction, severity, and number of curves. “Recurvatures” of the mesial roots of mandibular molars in which the apices bend toward each other are especially common. Bayonet curves are difficult to negotiate and prepare (Figures 5-23 and 5-24).

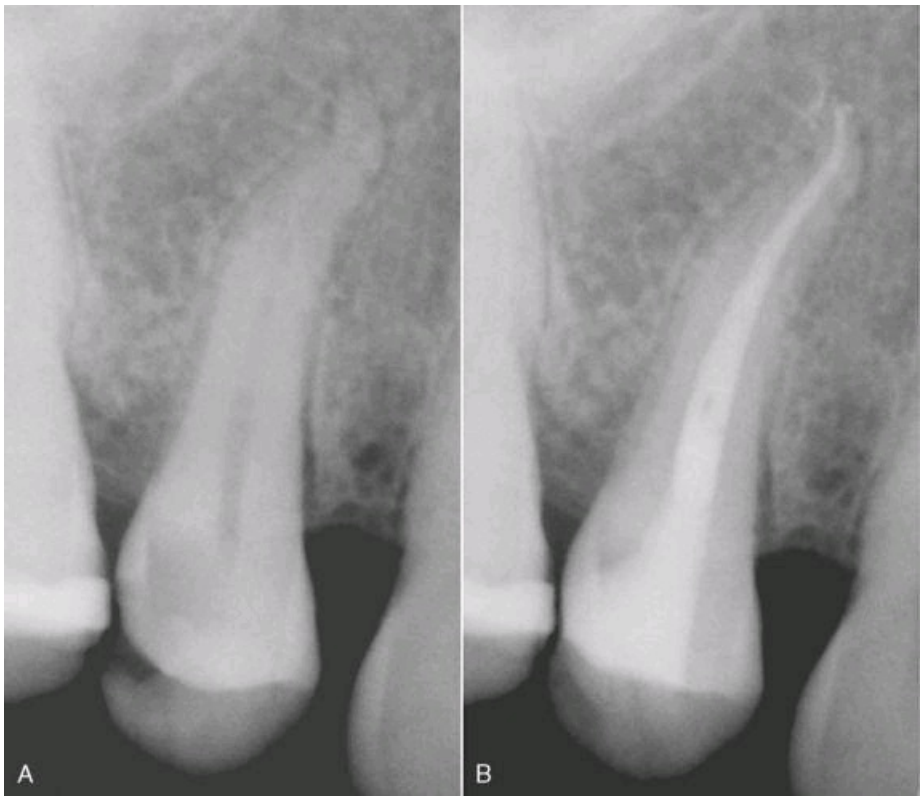


Figure 5-23 A, Dilaceration in the apical third. The degree and location of curvature strongly suggest caution and a consideration for referral. **B,** Note in the postoperative radiograph that the apical third curvature has been maintained.

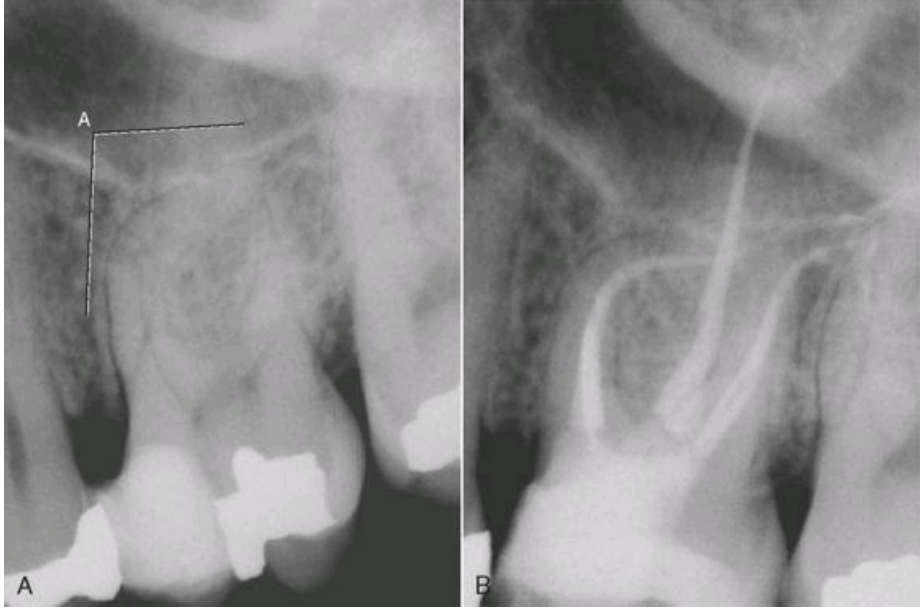


Figure 5-24 A, Extreme curvature of the mesiobuccal root. This nearly 90-degree curve (*A*) would place this situation in the extreme risk category and the patient should be considered for referral. **B,** Note the curvature of the distobuccal root in the postoperative radiograph.

Number of Canals

A good rule is to always expect "extra" canals or roots unless the preoperative film clearly shows a distinct number of canals; the reality is that more may exist (Figure 5-25). There are difficulties with multiple canals. For example, many mandibular incisors have two root canals, and mandibular premolars may have multiple canals. A significant percentage of mandibular first molars have two canals in the distal root, and mesiobuccal roots of maxillary molars often have two canal systems.

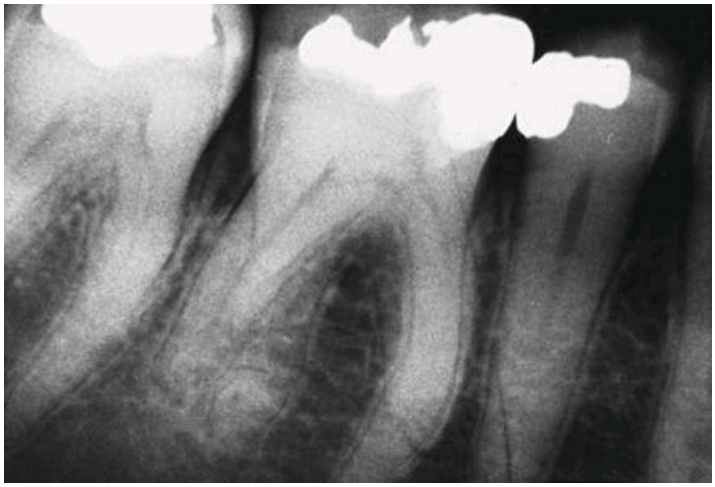


Figure 5-25 Mandibular molar with an "extra" distolingual root; this situation is rated as high risk.

Root Length

Very short or very long roots may be a challenge to treat.

Tooth Location

Generally, second and third molars, especially maxillary molars, are difficult to reach, particularly in a patient with a limited opening. Buccally positioned maxillary second molars are associated with a variety of treatment problems. Rotated, tipped, or crowded teeth may also complicate isolation and access, as well as inhibit adequate cleaning and shaping or obturation.

Degree of Apical Closure

The size of the apical opening correlates with the difficulty of the situation. Recently erupted teeth with immature apices are complicated and often require special procedures. Outcome and duration of treatment are unpredictable; these teeth are difficult to manage (see Chapter 2). Problems with teeth in which the initial apical foramen is between file sizes 50 and 70 are categorized as high risk.

Unusual Anatomy

Different tooth forms often present treatment difficulties (Figure 5-26).

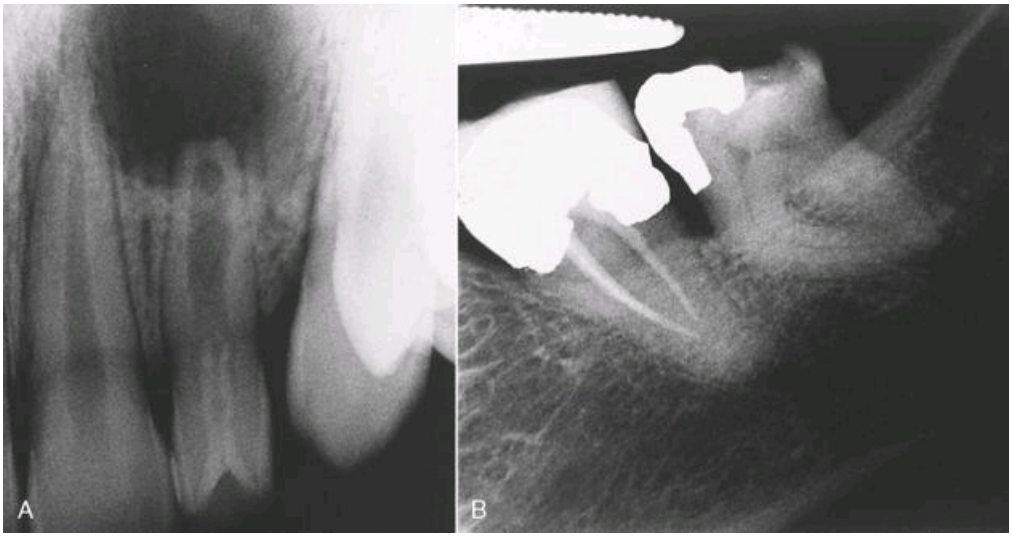


Figure 5-26 Anatomic difficulties. **A,** Dens invaginatus has resulted in communication and resultant necrosis the pulp. Incomplete dentin formation and an irregular internal form make these teeth anatomically difficult to clean, shape, and obturate. The size of the lesion does not contraindicate nonsurgical treatment. **B,** A severely curved root and the posterior position make treatment for this third molar difficult.

Additional Factors

Existing Restorations

Many teeth requiring root canal treatment have castings. Access through gold is easier than that through nonprecious metals. Porcelain is somewhat fragile and can crack or fracture. If the tooth is a bridge abutment, access may weaken the whole unit. Often restoration anatomy does not duplicate the original crown anatomy, and the pulp chamber is difficult to locate. When the chamber and orifices are not visible in the preoperative film or the anatomy of the underlying tooth is questionable, the case should be referred.

Previous Root Canal Treatment

Retreating an endontically treated tooth is often difficult and should be referred. A generalist may perform the retreatment if there is a single canal and the obturating material is gutta-percha. Other conditions are difficult; endodontists have experience with these cases (Figure 5-27).

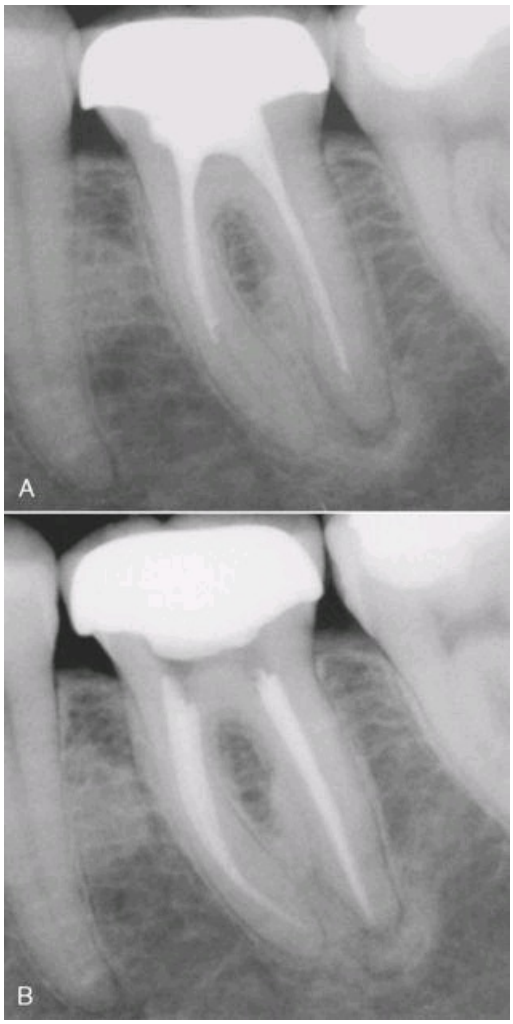


Figure 5-27 A, Previously attempted root canal treatment with very short obturation. The mesial root appears ledged. The tooth is percussion sensitive; also there is apical radiolucency of the distal root. Such retreatment cases should be considered as high or extreme risk. **B,** An experienced practitioner managed this patient; referral was prudent.

Trauma

Impact trauma requires primary (emergency) and secondary (definitive) management. Primary care, usually provided by the general practitioner, may include repositioning, replantation, splinting, dealing with exposed pulps, and suturing of soft tissue lacerations.

For secondary care, the patient should be referred to an endodontist who can determine an appropriate schedule for long-term management, recall, or treatment as necessary. Some changes may have a delayed onset. The possible complications

include crown discoloration, pulpitis, root resorption, pulp chamber and root canal calcification, and root fractures. Some of these changes occur slowly, and these cases need long-term supervision.

Fracture

Cracked or fractured teeth are often difficult to diagnose and treat. Symptoms can vary from vague pain while chewing to extreme pulpal pain or periapical symptoms. Definitive diagnosis must be achieved before treatment; then the prognosis must be determined based on the degree of fracture (see Chapter 7).

Emergency

General practitioners should be able to diagnose and treat most uncomplicated preoperative, interappointment, and postobturation emergencies. However, more difficult problems do occur. For example, if the generalist cannot control the patient psychologically or is unable to obtain profound anesthesia, the patient should be referred. If pain or swelling continues after emergency treatment and the general practitioner cannot assist the patient, an endodontist should be consulted or the patient referred.

Procedural Accidents

Separated Instrument or Canal Blockages

A separated instrument or other blockage has a significant negative effect on prognosis. Different approaches are possible and each requires an endodontist's expertise.

Ledging

If a canal is ledged far from the working length, prognosis is compromised (see Figure 5-27). The generalist should refer the patient to an endodontist. Canals that have not been ledged too far from the working length can be obturated, and the patient can be followed by the general practitioner. If retreatment or surgery is subsequently needed, the patient should be referred.

Perforations

Cases involving root or crown perforations are high risk. Patients with these conditions should be referred. Depending on the size, location, and strategic importance of the tooth, several options are available. These treatment approaches, as well as long-term assessment, are generally beyond the expertise of a general practitioner.

Failure to Heal

Persistent Pain

If pain or swelling persists or develops after treatment, the patient should be referred or an endodontist consulted. The symptoms may have several causes: lack of débridement, coronal leakage, inadequate obturation, missed canals, root fractures, and so on. Retreatment, surgical intervention, or even extraction may be required.

Persistent Lesion

If periapical lesions persist or develop after root canal treatment, consultation may be indicated. The endodontist may retreat, perform surgery, or advise the generalist on appropriate measures to resolve the problem.

Persistent Periodontal Lesion

If a probing defect of endodontic origin or a sinus tract does not resolve after treatment, the patient should be referred. In addition, a new defect or sinus tract indicates treatment failure; referral should be considered.

Referral During Treatment

Timing of referral is important. A poor practice is to initiate treatment with the idea that if problems arise, a referral can be made at that time. However, during treatment, unanticipated problems may arise that require referral; a call to the endodontist and an explanation to the patient are in order. Reasons for possible referral in the middle of treatment include flare-ups (pain or swelling), procedural accidents, inability to achieve adequate anesthesia, and other factors that hinder completion of root canal therapy.

Referral to a Specialist

Most patients understand and accept referral. After briefly explaining the treatment needed and the options, a simple explanation is in order: “Mrs. Jones, because of the type of treatment, you need to obtain the best possible results. I would like you to see Dr. Smith. Dr. Smith is an expert in this field, and it would be to your advantage to go to a specialist. When Dr. Smith has completed the root canal treatment, you will return to me so that I can restore your tooth.”

What Is Expected of a General Practitioner

Explicit written instructions and appropriate radiographs (original or duplicate) should be mailed or e-mailed to the endodontist. These instructions should include how the tooth fits into the overall treatment plan.

What Is Expected of an Endodontist

Specialists serve both the patient and referring dentist. Their responsibilities are to both; they should deliver appropriate treatment and communicate with the practitioner, as well as the patient. When treatment is complete, the referring dentist should receive written confirmation from the endodontist that includes a radiograph of the obturation. A note is included about how the tooth was treated, anticipated recalls, prognosis (both short-term and long-term), and unusual findings or circumstances.

Before and during treatment, the endodontist explains all of the important aspects of the procedure and the anticipated outcome to the patient. After completion of treatment, the patient is informed of prognosis, appropriate follow-up care, and the need to return to the referring dentist for continued care and any possible additional future procedures.

Chapter Review Questions available in Appendix B or on the DVD

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Endodontic and Periodontal Interrelationship

Ilan Rotstein, James H.S. Simon

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Recognize the anatomic pathways of communication between the dental pulp and the periodontal ligament or gingival sulcus.
2. Describe the effects of pulpal disease and endodontic procedures on the periodontium.
3. Describe the effects of periodontal disease and procedures on the pulp.
4. Identify the etiologic factors associated with endodontic-periodontal diseases.
5. State the contributing factors and their role in endodontic-periodontal diseases.
6. Identify the clinical classification of endodontic-periodontal diseases and determine the differential diagnosis.
7. Establish treatment requirements and sequencing according to diagnostic findings.
8. Identify prognosis and assess which cases should be considered for referral.

Treatment and prognosis of endodontic-periodontal diseases depend on the etiology and correct diagnosis of the specific condition. Often, the diagnosis of these conditions presents a challenge to the clinician. One of the more difficult dilemmas is interpretation of the origin of a defect as reflected in the integrity of the periodontal ligament (PDL). Etiologic factors, such as microorganisms, and other contributing factors, such as trauma, root resorptions, perforations, and dental malformations, play an important role in the development and progression of endodontic-periodontal lesions.

This chapter discusses the interrelationship between endodontic and periodontal diseases and provides biological and clinical evidence for diagnosis, prognosis, and decision making in the treatment of these conditions.

PATHWAYS OF COMMUNICATION BETWEEN THE DENTAL PULP AND THE PERIODONTIUM

The dental pulp and periodontal tissues are intimately related. The pulp originates from the dental papilla and the PDL from the dental follicle. They are separated by Hertwig's epithelial root sheath (see Chapter 2). As the tooth matures and the root is formed, three main avenues for communication between the dental pulp and the periodontium are formed: (1) dentinal tubules, (2) lateral and accessory canals, and (3) the apical foramen.

Dentinal Tubules

Cementum acts as a protective barrier, but direct communication may be established between the pulp and the periodontium via patent dentinal tubules if the cementum is missing. The cementum may be missing as a result of developmental defects, disease processes, or periodontal or surgical procedures. Exposed dentinal tubules in areas devoid of cementum may serve as communication pathways between the pulp and the PDL. Recognizing the anatomy of patent dentinal tubules and the changes caused by age or periodontal treatment is essential to the understanding of the permeability of root dentin, as well as clinical conditions such as dentin hypersensitivity.

Radicular dentin tubules extend from the pulp to the cementodentinal junction (CDJ).[1] They run a relatively straight course. The diameter ranges from 1 µm in the periphery to 3 µm near the pulp.[2] The tubular lumen decreases with age or as a response to a chronic, low-grade stimulus. Irritation of dentin can cause apposition of highly mineralized peritubular dentin. The density of dentin tubules varies from approximately 15,000 tubules per square millimeter at the CDJ in the cervical portion of the root, to 8000 near the apex. The number increases to 57,000 per square millimeter at the pulpal ends.[2] When the cementum and enamel do not meet at the cementoenamel junction (CEJ), these tubules remain exposed, thus creating pathways of communication between the pulp and the PDL. Cervical dentin hypersensitivity may be a result of such tubular exposure.

Scanning electron microscopic studies have demonstrated that dentin exposure at the CEJ occurred in about 18% of teeth in general and in 25% of anterior teeth in particular.[3] In addition, the same tooth may have different CEJ characteristics, presenting dentin exposure on one side while the other sides are covered with cementum.[4] Dentin exposure plays an important role when assessing the progression of endodontic pathogens, as well as the effect of scaling and root planing on cementum integrity, trauma, and chemically induced pathosis.[5-7] Other possible dentinal communications may be through developmental grooves, both palatogingival and apical.[8]

Lateral and Accessory Canals

Lateral and accessory canals can be present anywhere along the root, and their incidence and location have been well documented[9-15] (Figure 6-1). It is estimated that 30% to 40% of all teeth have lateral or accessory canals, mostly found in the apical third of the root.[1] De Deus[12] reported that 17% of the teeth examined presented lateral canals in the apical third of the root, about 9% in the middle third, and less than 2% in the coronal third. However, it appears that the incidence of periodontal disease associated with lateral canals caused by irritants in the dental pulp is relatively low. Kirkham[13] examined 1000 human teeth with extensive periodontal disease and found only 2% of lateral canals associated with the corresponding periodontal pocket.

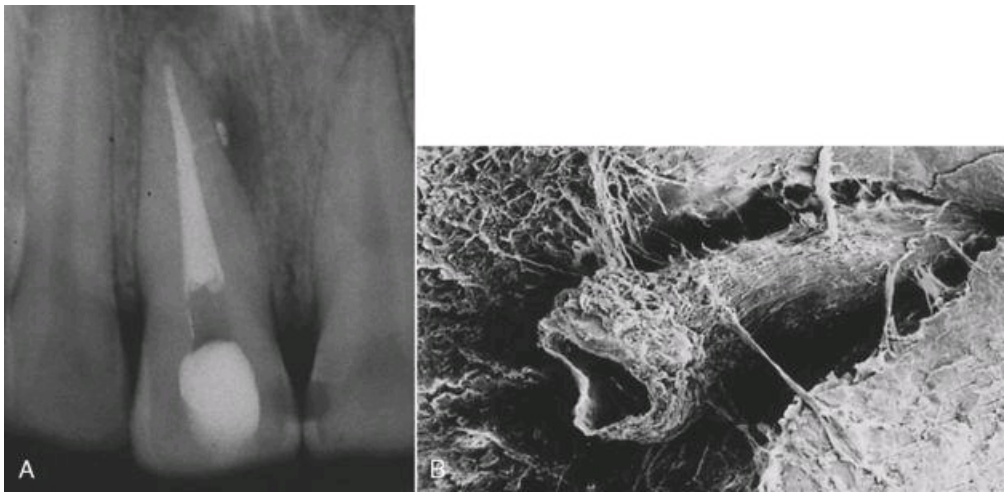

Figure 6-1 A, Postoperative radiograph of endodontic treatment in a maxillary central incisor showing radiopaque material extruding through a lateral canal. **B,** Scanning electron micrograph showing vascular content in lateral canal.

Accessory canals in the furcation of molars may also be a direct pathway of communication between the pulp and the periodontium.[10,14] The reported incidence of accessory canals varies from 23% to 76%.[11,12,16] These accessory canals contain connective tissue and blood vessels that connect the circulatory system of the pulp with that of the periodontium. However, not all of these canals extend the full length from the pulp chamber to the floor of the furcation.[16] Seltzer et al[17] reported that pulpal inflammation may cause inflammatory reaction in the interradicular periodontal tissues. Patent accessory canals are a potential pathway for the spread of microorganisms and their toxic byproducts, as well as other irritants, from the pulp to the PDL and vice versa, resulting in an inflammatory process in the involved tissues (see Figure 6-1).

The presence of a lateral canal is very difficult to detect clinically. Pineda and Kutler,[18] analyzing a large pool of extracted teeth, reported that about 30% of lateral canals could be identified through use of 2-view radiographs, whereas standard radiographs identified only about 8%. These investigators, however, were unable to identify any accessory canals in the furcation area of molar teeth. It therefore appears that under usual clinical conditions, predictable identification of lateral and accessory canals on the basis of radiographic interpretation may be accomplished only in a very small number of cases. However, several clinical aids may be helpful for their identification: (1) a radiographic image of a discrete lateral lesion associated with a necrotic pulp, (2) radiographic identification of a notch on the lateral root surface, suggesting the presence of an orifice; and (3) demonstration of root canal filling material, or sealer, extruding through the patent orifices.

Apical Foramen

The apical foramen is the principal route of communication between the pulp and periodontium. Infectious and inflammatory byproducts of a diseased pulp may permeate readily through the apical foramen, resulting in periapical pathosis. The apical foramen may also be a portal of entry of inflammatory elements from deep periodontal pockets to the pulp. Pulp inflammation or pulp necrosis extends into the periapical tissues, causing a local inflammatory response often associated with bone and root resorption. Elimination of the etiologic factors from the root canal is therefore essential to promote healing.

EFFECT OF ENDODONTIC DISEASE ON THE PERIODONTIUM

When the pulp becomes infected, it elicits an inflammatory response of the PDL at the apical foramen or adjacent to openings of lateral and accessory canals.[19] Inflammatory byproducts of pulpal origin may permeate through the apex, lateral or accessory canals, and dentinal tubules to trigger an inflammatory vascular response in the periodontium. Among those products are living pathogens, such as certain bacteria strains, fungi, and viruses,[20-28] as well as several nonliving pathogens.[29-32] In certain cases, pulpal disease will stimulate epithelial growth that will affect the integrity of the periradicular tissues.[33,34]

The results of pulp inflammation can range in extent from a minimal inflammatory process confined to the PDL to extensive destruction of the PDL, tooth socket, and surrounding bone. Such a lesion may result in a localized or diffuse swelling that occasionally may involve the gingival attachment. A lesion related to pulpal necrosis may also result in a sinus tract that drains through the alveolar mucosa or attached gingiva. It may occasionally also drain through the gingival sulcus of the involved tooth or through the gingival sulcus of an adjacent tooth (discussed later in this chapter). After appropriate root canal treatment, lesions resulting from pulpal necrosis resolve uneventfully in most instances.[35] Subsequently, the integrity of the periodontal tissues will be reestablished.

Certain procedures involved in root canal treatment, as well as irrigants, intracanal medicaments, sealers, and filling materials have the potential to cause an inflammatory response in the periodontium. The inflammatory response resulting from commonly used root canal treatment methods and materials, however, is usually transient in nature and resolves quickly if the materials are confined within the canal space.

Procedural errors during root canal treatment can also cause inflammatory response in the periodontium. Periodontal defects resulting from attachment breakdown may occur after procedural mishaps, such as perforations of the floor of a pulp chamber or the root surface apical to the gingival attachment, strip perforations or root perforations related to cleaning and shaping procedures, and vertical root fractures associated with uncontrolled forces used for canal obturation (Figure 6-2).

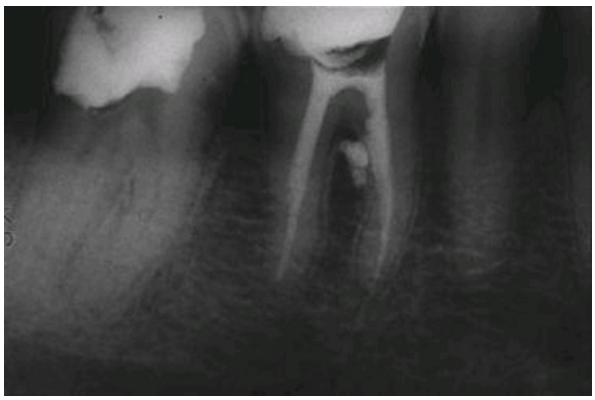

Figure 6-2 Inadequate canal access and instrumentation in a mandibular first molar. As a result, strip perforation and gross extrusion of sealer has occurred.

EFFECT OF PERIODONTAL DISEASE ON THE PULP

The effect of periodontal inflammation on the pulp is controversial.[17,36-42] It has been suggested that periodontal disease has no major effect on the pulp, at least until it involves the apex.[37] In contrast, several studies suggest that the effect of periodontal disease on the pulp is degenerative in nature, causing an increase in calcifications, fibrosis, collagen resorption, and direct inflammatory sequelae.[43,44] It appears that the pulp is usually not severely affected by periodontal disease until the defect has exposed an accessory canal to the oral environment.[9] At this stage, pathogens leaking from the oral cavity through the accessory canal into the pulp may cause an inflammatory reaction followed by pulp necrosis. However, if the microvasculature of the apical foramen remains intact, the pulp may test positive to pulp vitality tests. The effect of periodontal treatment on the pulp is similar during scaling, curettage, or periodontal surgery if accessory canals are severed and/or opened to the oral environment. In such cases, pathogenic invasion and secondary inflammation and necrosis of the pulp can occur.[45]

Blomlöf et al[46] created defects on root surfaces of extracted monkey teeth with either open or mature apices. The canals were either infected or filled with calcium hydroxide and replanted back in their sockets. After 20 weeks, marginal epithelial downgrowth was found on the denuded dentin surface of the infected teeth. Jansson et al[47] assessed the effect of endodontic pathogens on marginal periodontal wound healing of denuded dentinal surfaces surrounded by healthy PDL. Their results suggested that in infected teeth, the defects were covered by 20% more epithelium, whereas the noninfected teeth showed only 10% more connective tissue coverage. The same investigators,[48] in a 3-year, retrospective radiographic study, evaluated 175 endodontically treated single-rooted teeth of 133 patients. Patients who were more prone to periodontitis and exhibited evidence of endodontic treatment failures showed an approximately threefold increase in marginal bone loss as compared to patients without endodontic infection. In addition, the effect of endodontic infection on periodontal probing depth and the presence of furcation involvement in mandibular molars were also investigated.[49] It was found that endodontic infection in mandibular molars was associated with more attachment loss in the furca. These authors suggested that endodontic infection in molars associated with periodontal disease might enhance periodontitis progression by spreading pathogens through accessory canals and dentinal tubules. However, other investigators[50] failed to observe a correlation between a reduced marginal bone support and endodontic status. This issue still merits further investigation.

ETIOLOGICAL FACTORS

Live Pathogens

Among the live pathogens encountered in a diseased pulp that can cause lesions in the periodontium are bacteria,[26,51-60] spirochetes,[61-65] fungi,[27,66-76] and viruses.[77-79] These pathogens and their byproducts, which may affect the integrity of the periodontium, need to be eliminated during endodontic treatment.

Nonliving Pathogens

Nonliving pathogens can be either extrinsic (such as foreign bodies) or intrinsic, including a variety of tissue components. These include foreign bodies such as dentin and cementum chips, root canal filling materials, food debris, calculus-like deposits, inflamed epithelium,[33,34,80] cholesterol crystals,[30,81] Russell bodies,[31] Rushton hyaline bodies,[29,82] and Charcot-Leyden crystals[32] (Figures 6-3 and 6-4).

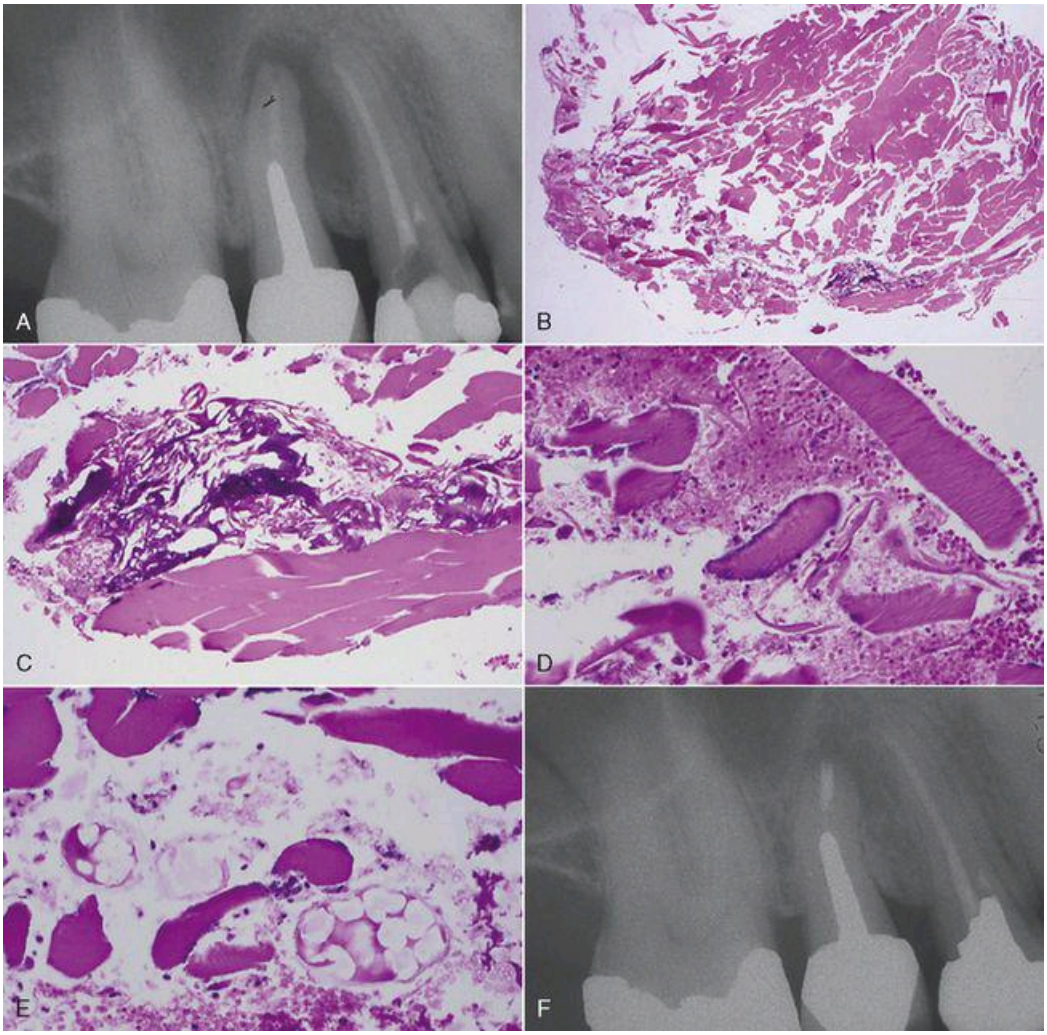

Figure 6-3 Multietiologic pathogenesis of the periapical tissues. **A,** Radiograph showing nonhealing of a maxillary second premolar. The tooth was treated by intentional replantation during which the apical lesion was removed. **B,** Photomicrograph of the apical lesion showing presence of foreign material. **C,** Higher magnification shows purple unidentified foreign material and necrotic muscle tissue. **D,** A different area of the lesion showing necrotic muscle with viable bacterial colonies. **E,** Presence of lentil beans in necrotic muscle tissue infected by bacteria (pulse granuloma). **F,** One-year follow-up radiograph showing evidence of bony healing. The tooth is asymptomatic and firm.

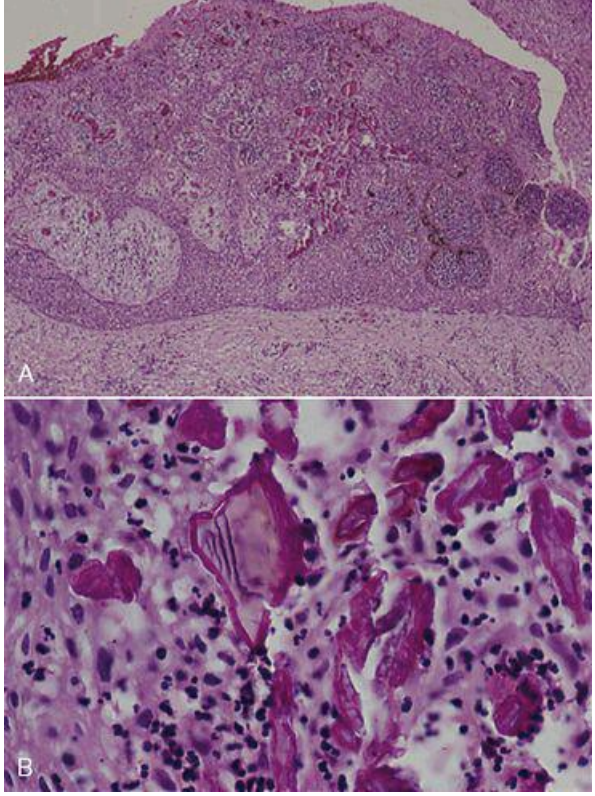

Figure 6-4 A, Photomicrograph showing Rushton bodies in the epithelial lining of a periapical cyst (hematoxylin-eosin stain). **B,** Higher magnifications demonstrating pleomorphism of these bodies.

CONTRIBUTING FACTORS

An important contributing factor is inadequate endodontic treatment. Proper endodontic procedures and techniques are key factors for treatment success. When assessing the retention rate of endodontically treated teeth, nonsurgical endodontic treatment is a predictable procedure with excellent long-term prognosis.[83-85] It is essential to clean, shape, and obturate the canal system well to enhance successful outcomes. Poor endodontic treatment allows canal reinfection, which may often lead to treatment failure and inflammatory reaction in the periodontal tissues.

Endodontic failures can be treated either by orthograde or retrograde retreatment with good success rates. In recent years, retreatment techniques have improved dramatically because of the use of the operating microscope and the development of new armamentarium.

In addition, other factors, such as coronal leakage,[86-88] traumatic injuries,[89] root resorptions,[90-94] root perforations,[95] and developmental malformations,[96] may play a role in both endodontic and periodontal inflammatory processes. An excellent and conservative treatment modality for perforations, root resorptions, and certain root fractures in the cervical third region is controlled root extrusion[97-99] (Figure 6-5). The procedure has excellent prognosis and a low risk of relapse. It can be performed either immediately or over a few weeks, depending on each individual case. The goal of controlled root extrusion is to modify the soft tissues and bone; it is therefore used to correct gingival discrepancies and osseous defects of periodontally involved teeth.[98] It is also used in the management of nonrestorable teeth and as a nonsurgical alternative to crown lengthening.[99]

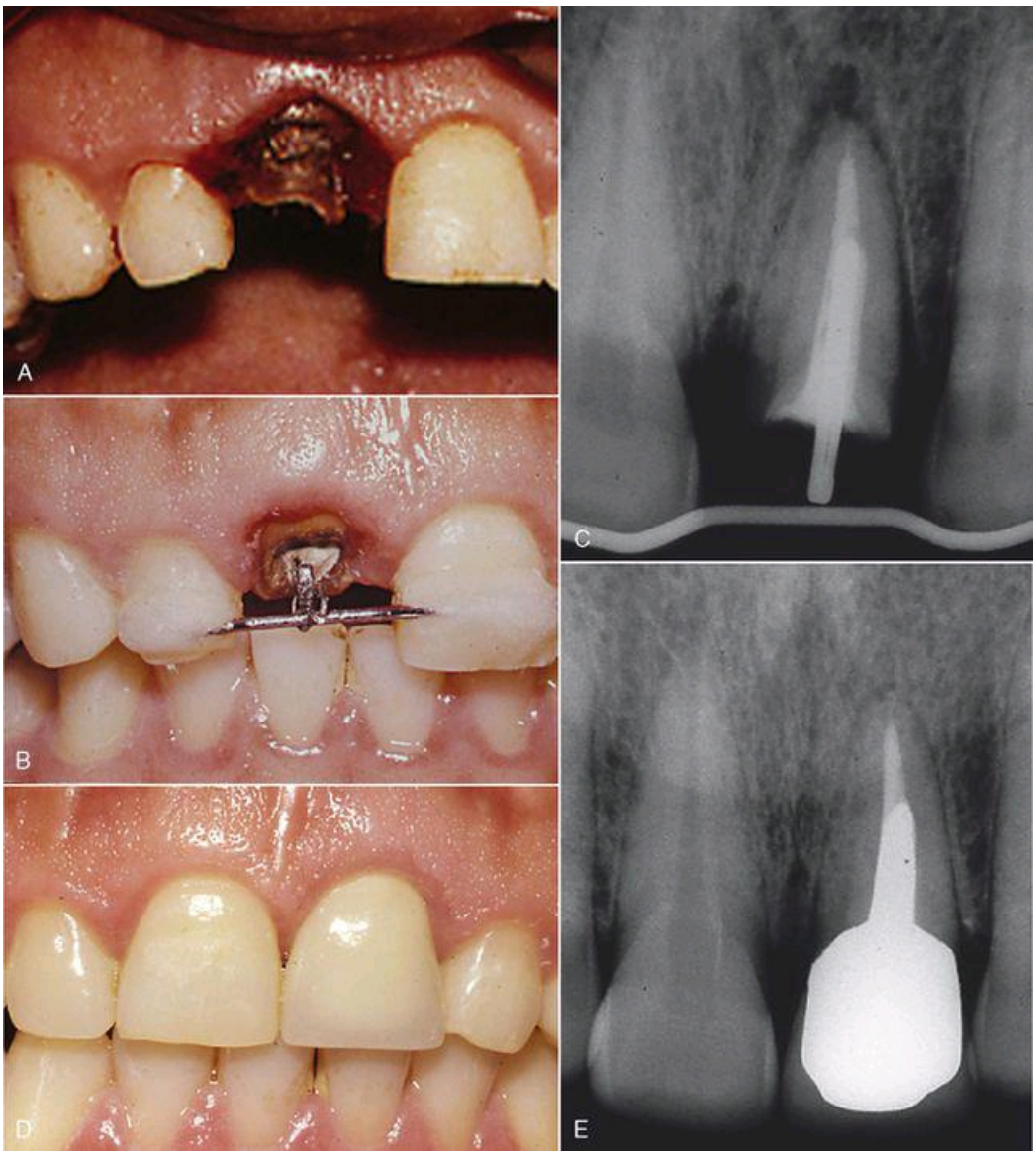

Figure 6-5 Coronal fracture in a maxillary central incisor treated with controlled root extrusion. Preoperative clinical view **(A)** reveals deep horizontal fracture well below the gingival line. Endodontic treatment is performed, and a temporary post is inserted in the canal. The post was then hooked with rubber bands to a bar placed between the adjacent teeth. The bands were replaced periodically, thereby maintaining a tight pressure as the root extrudes. Clinical **(B)** and radiographic **(C)** views show root extrusion in progress. After 2 weeks of root extrusion and 6 weeks of stabilization, the root was extruded to a point that enabled the placement of a prosthetic restoration. Clinical **(D)** and radiographic **(E)** views show the affected tooth restored to esthetics and function with a post and crown. Controlled extrusion is a predictable noninvasive technique and usually does not require surgical intervention.

CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS

For differential diagnosis and treatment purposes, the so-called endodontic-periodontal (endo-perio) lesions can be classified as endodontic, periodontal, or combined diseases.[100] These include: (1) primary endodontic diseases, (2) primary periodontal diseases, and (3) combined diseases. The combined diseases include: (1) primary endodontic disease with secondary periodontal involvement, (2) primary periodontal disease with secondary endodontic involvement, and (3) true combined diseases. This classification is based on how these lesions are formed. By understanding the pathogenesis, the clinician can offer an appropriate course of treatment and assess the prognosis. Fully developed endo-perio lesions may present a similar radiographic picture, making differential diagnosis challenging.

Clinical Findings

An acute exacerbation of a chronic apical lesion in a tooth with a necrotic pulp may drain coronally through the PDL into the gingival sulcus. Clinically, this condition may mimic the presence of a periodontal abscess with the presence of a pseudopocket. In reality, it is a sinus tract of pulpal origin that opens through the PDL area. A deep, solitary pocket in the absence of periodontal disease may indicate the presence of a lesion of endodontic origin or a vertical root fracture. For diagnosis purposes, a gutta-percha cone or other tracking instrument should be inserted into the sinus tract, and radiographs should be taken. This will determine the origin of the lesion (Figures 6-6 and 6-7).

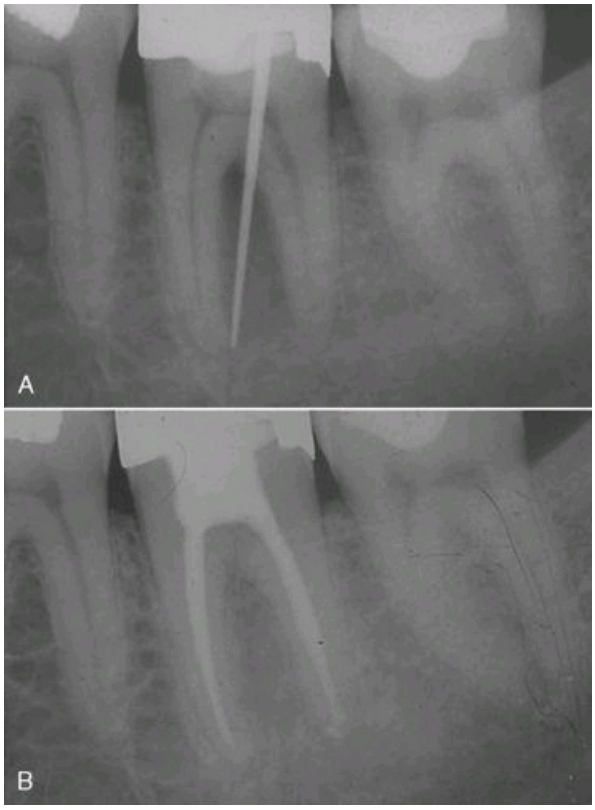

Figure 6-6 Primary endodontic disease in a mandibular second molar. **A,** Preoperative radiograph showing large furcal radiolucency. A deep, narrow periodontal pocket can be probed all the way to the apex of the mesial root. Negative responses to pulp vitality tests confirmed pulp necrosis, and endodontic treatment was carried out. **B,** One year after root canal treatment, resolution of the furcal radiolucency is evident.

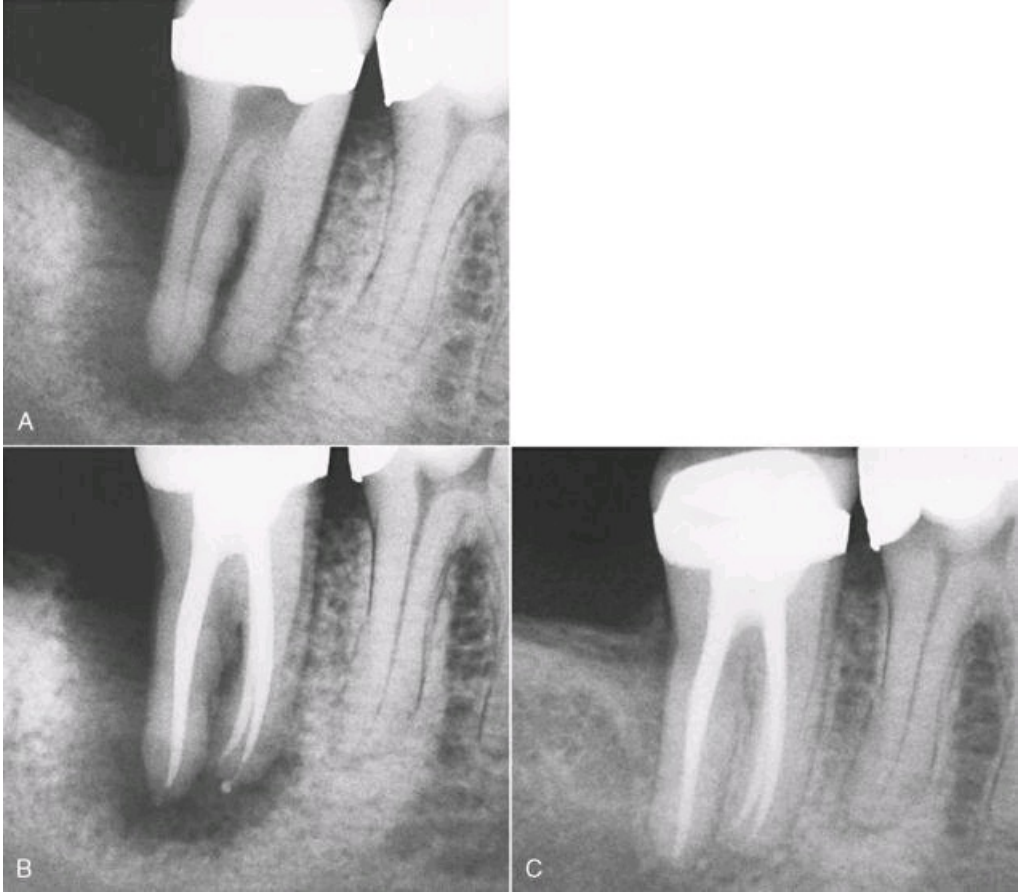

Figure 6-7 Primary endodontic disease with radiographic appearance of a periodontal lesion. **A,** Preoperative radiograph indicates bone loss from the crest of the ridge around the apices of the tooth. Periodontal probing demonstrates that the gingival sulcus is intact. There is no response to pulp tests. **B,** Root canal treatment completed. **C,** Four-year recall shows resolution of the radiolucency.

Radiographic Findings

In the early stages of the disease, radiographic changes may not be noticeable. As the disease progresses, a widening of the PDL space of the affected tooth, a periapical radiolucency, or a bony lesion extending from apical to cervical can be observed.

Pulp Tests

Pulp tests will reveal an abnormal or completely absent response.

Probing

When the pocket is probed, it is narrow and lacks width. A similar condition occurs where drainage from the apex of a molar tooth extends coronally into the furcation area. This may also occur in the presence of lateral canals extending from a necrotic pulp into the furcation area.

Prognosis

Primary endodontic diseases usually heal after root canal treatment. The sinus tract extending into the gingival sulcus or furcation area disappears at an early stage once the affected pulp has been removed and the root canals have been well cleaned, shaped, and obturated.

Primary Periodontal Disease

Clinical Findings

Periodontal lesions are caused primarily by periodontal pathogens. In this process, chronic marginal periodontitis progresses apically along the root surface. There is frequently an accumulation of plaque and calculus, and the pockets are wider.

Radiographic Findings

Bony lesions associated with angular bone loss are usually found and extend from the cervical region toward the apex (Figure 6-8). The lesions may not be limited to just one tooth and often involve adjacent teeth as well. In contrast to primary endodontic lesions, the absence of buccal, lingual, or both plates may clearly show root and canal to the level of the bone loss.[1] The clinician must also be aware of the radiographic appearance of periodontal disease associated with developmental radicular anomalies.

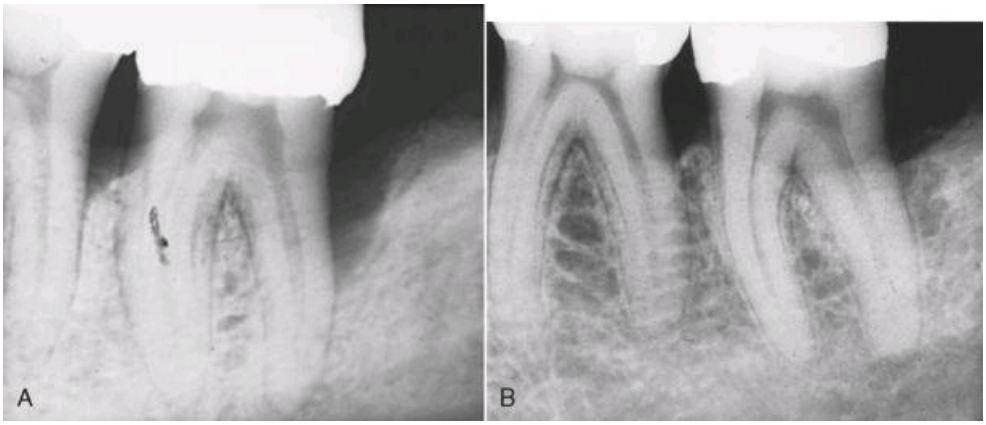

Figure 6-8 Primary periodontal disease in a mandibular second molar. **A,** Preoperative radiograph of a periodontal lesion. All teeth responded within normal limits to pulp tests. **B,** Two-year recall radiograph of successful periodontal treatment.

Pulp Tests

In most cases, pulp tests indicate a clinically normal pulpal response. This is an important test to distinguish between primary endodontic disease and primary periodontal disease conditions.

Probing

Probing will reveal wide pockets that do not necessarily extend toward the apex.

Prognosis

The prognosis depends on the stage of periodontal disease and the efficacy of periodontal treatment.

Combined Diseases

Primary Endodontic Disease with Secondary Periodontal Involvement

If a suppurating primary endodontic disease remains untreated after a period of time, it may become secondarily involved with marginal periodontal breakdown.

Clinical Findings

Plaque is found at the gingival margin of the solitary sinus tract, which leads to marginal periodontitis. When plaque or calculus is present, the treatment and prognosis of the tooth are different than for teeth involved with only primary endodontic disease. Adjacent teeth are not necessarily involved.

Radiographic Findings

A clear widening of the PDL space of the affected tooth extending from apical to cervical can be observed.

Pulp Tests

Pulp tests will usually reveal absence of response.

Probing

A solitary but wider pocket extending toward the apex is usually found.

Prognosis

The affected tooth requires both endodontic and periodontal treatments. If the endodontic treatment is adequate, the prognosis depends on the severity of the marginal periodontal damage and the efficacy of periodontal treatment. With endodontic treatment alone, only that part of the lesion that is of endodontic etiology will heal to the level of the secondary periodontal lesion (Figure 6-9).

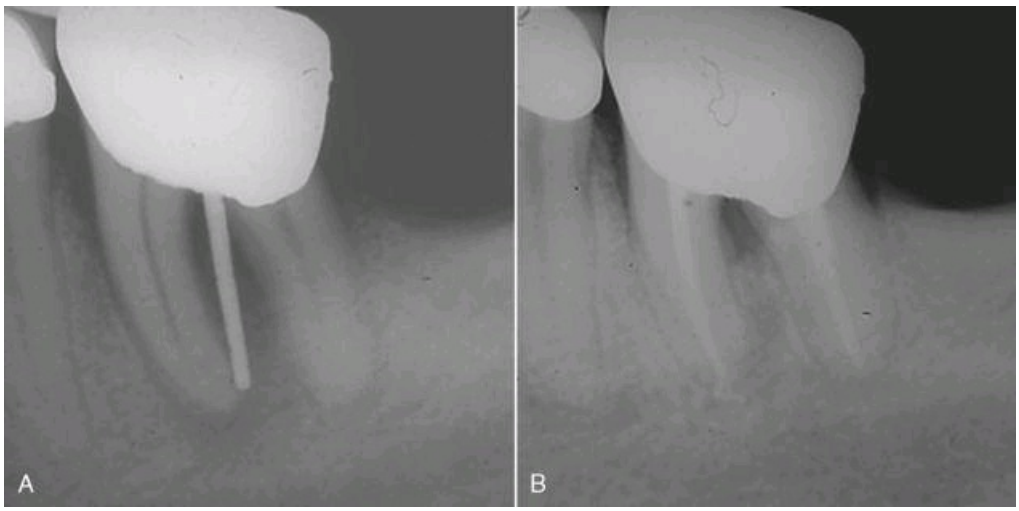

Figure 6-9 Primary endodontic disease with secondary periodontal involvement in a mandibular first molar. **A,** Preoperative radiograph showing deep furcal defect extending to the apex of the mesial root. Negative responses to pulp vitality tests confirmed pulp necrosis; endodontic treatment was carried out. **B,** One-year follow-up radiograph showing resolution of most of the periradicular lesion; however, a bony defect at the furcal area still remained. Note that endodontic treatment alone did not yield complete healing of the defect. Periodontal treatment was necessary for further healing of the furcal area and inflamed gingival tissues.

A similar clinical picture may also occur as a result of root perforation during root canal treatment or misplaced pins or posts during coronal restoration. Sometimes symptoms may be acute, including periodontal abscess formation associated with

pain, swelling, pus exudate, pocket formation, and tooth mobility. A more chronic response may also occur without pain and involves the sudden appearance of a pocket with bleeding on probing or exudation of pus.

Root fractures may also mimic the appearance of primary endodontic lesions with secondary periodontal involvement. These typically occur on endodontically treated teeth with posts and crowns. The signs may range from a local deepening of a periodontal pocket to more acute periodontal abscess formation.

Primary Periodontal Disease with Secondary Endodontic Involvement

The progression of a periodontal pocket may continue until the apical tissues are involved. In this case, the pulp may become necrotic as a result of infection entering via lateral canals or the apical foramen. Bacteria originating from the periodontal pocket can be a source of root canal infection. A strong correlation between the presence of microorganisms in root canals and their presence in periodontal pockets of advanced periodontitis has been demonstrated, indicating that similar pathogens may be involved in both diseases.[101,102] As long as the neurovascular supply of the pulp remains intact, prospects for survival are good. If lost to periodontal disease, pulpal necrosis will likely occur.[43]

Clinical Findings

Bony lesions associated with angular bone loss are usually found and extend from the cervical region toward the apex. Generalized periodontitis or periodontal involvement of adjacent teeth is common. Symptoms associated with pain originating from an inflamed pulp are common at the early stages of the disease. As the disease progresses, the pulp is expected to lose its vitality.

Treatment complications of periodontal disease can also lead to secondary endodontic involvement. Lateral canals and dentinal tubules may be opened to the oral environment by curettage, scaling, or surgical flap procedures. It is possible for a blood vessel within a lateral canal to be severed by a curette and for microorganisms to be introduced into the area during treatment, thereby resulting in pulp inflammation and necrosis (Figure 6-10).

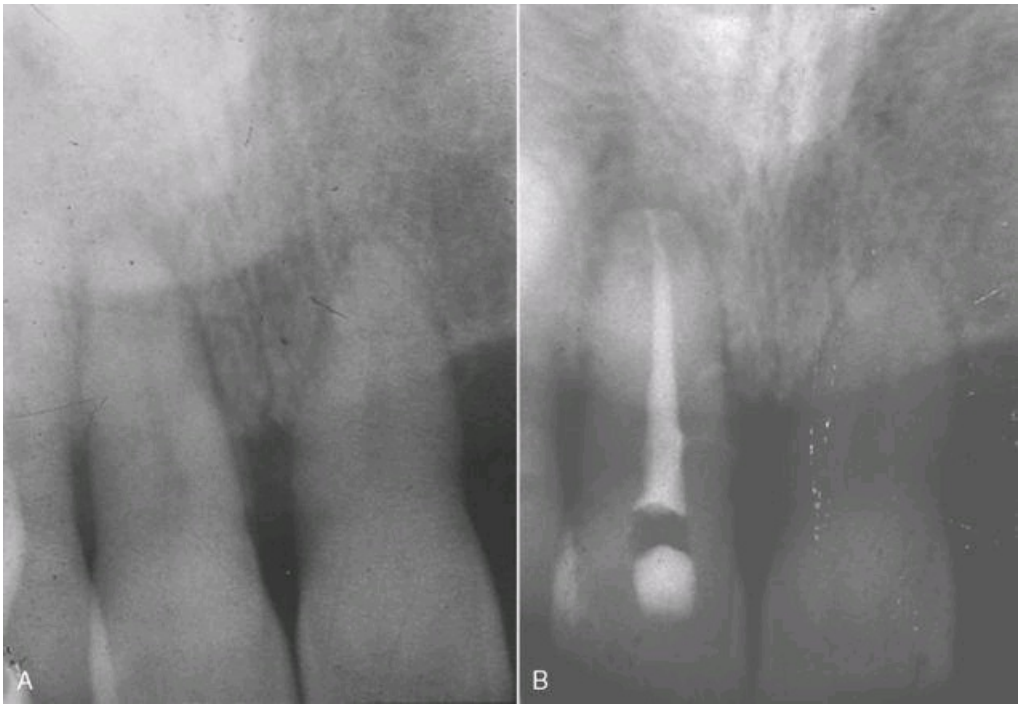

Figure 6-10 Primary periodontal disease with secondary endodontic involvement in a maxillary central incisor. **A,** Radiograph showing bone loss of one-half of the root length and a separate periapical lesion. The crown was intact; however, pulp sensitivity test results were negative. **B,** Radiograph taken immediately after root canal treatment reveals lateral canals exposed to the oral environment because of the bone loss. In this case, the crown of the tooth was intact and the periodontal disease progressed apically, exposing lateral canals to the oral environment. This resulted in infection of the root canal and subsequent pulp necrosis.

Radiographic Findings

Bony lesions associated with angular bone loss are usually found and extend from the cervical region toward the apex. Additional periapical radiolucency can also be detected.

Pulp Tests

Pulp tests will reveal an abnormal or complete absence of response.

Probing

Probing will reveal a wide pocket extending apically. However, probing does not necessarily extend all the way to the apex.

Prognosis

In single-rooted teeth, the prognosis is usually poor. In molar teeth, the prognosis may be better since all the roots may not suffer the same loss of supporting tissues. In such cases, root resection can be considered as a treatment alternative.

True Combined Diseases

True combined endo-perio diseases occur less often.

Clinical Findings

True combined endo-perio diseases occur when an endodontic disease progressing coronally joins with an infected periodontal pocket progressing apically.[17,103] The degree of attachment loss in this type of lesion is great (Figure 6-11).

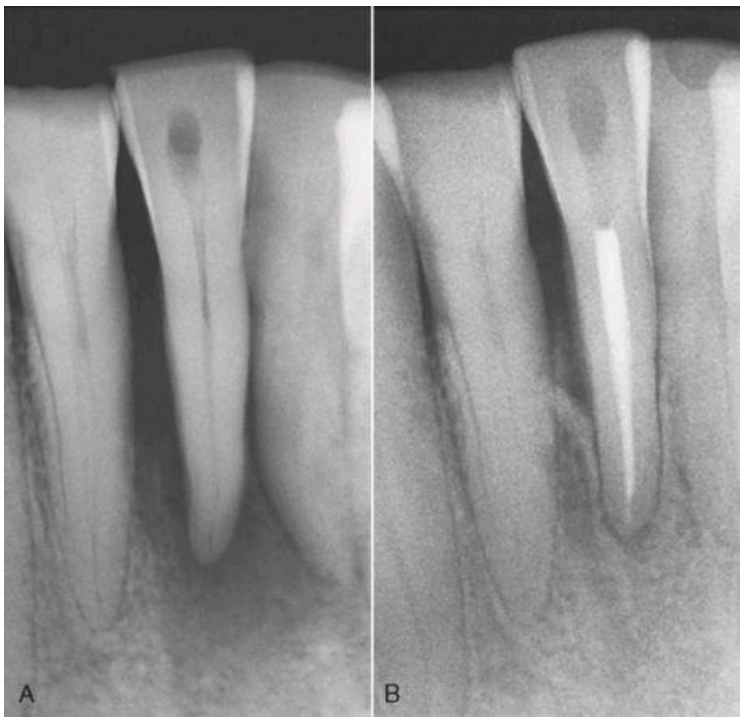

Figure 6-11 True combined perio-endo lesion. **A,** Mandibular incisor with a large lesion at the distal. The lesion probed with a conical contour. Within the conical lesion there was a defect that probed to the apex, demonstrating communication. **B,** An 11-year recall demonstrates resolution of the lesion around the apex. An angular defect remains at the distal.

Radiographic Findings

Extensive bony radiolucencies from endodontic and periodontal origins are found associated with the affected tooth. This is due to the long-standing nature of this condition. Depending on the stage of the disease, the lesions may or may not communicate. The radiographic appearance of combined endo-perio disease may be similar to that of a vertically fractured tooth; correct differential diagnosis is essential.

Pulp Tests

Pulp tests will reveal complete absence of response.

Probing

Probing will show a wide and conical periodontal pocket, characteristic of periodontal disease.

Prognosis

The prognosis is usually guarded. This is particularly true in single-rooted teeth. In molar teeth, root resection can be considered as a treatment alternative if only some of the roots are severely involved. In most cases, periapical healing may be anticipated after successful endodontic treatment. The periodontal tissues, however, may not respond well to treatment, and the outcomes depend on the severity of the condition.

A fracture that has invaded the pulp space with resultant necrosis may also be labeled a true combined lesion, but it may not be amenable to successful treatment. If a sinus tract is present, it may be necessary to raise a flap to establish the diagnosis.

WHEN TO REFER

Correct identification of the etiology of the disease, whether endodontic, periodontal, or combined, will determine the course of treatment and long-term prognosis. Prognosis depends primarily on the diagnosis of the specific endodontic or periodontal disease. The main factors to consider are pulp vitality and extent of the periodontal condition. For example, the prognosis for a tooth with a necrotic pulp, with or without a sinus tract, is excellent after appropriate root canal therapy. However, the prognosis of root canal treatment in a tooth with severe periodontal disease depends on the success of the periodontal therapy.

Primary endodontic disease should only be treated by endodontic therapy. Excellent prognosis is to be expected if appropriate endodontic treatment is carried out. Primary periodontal disease should only be treated by periodontal therapy. In this case, the prognosis depends on the severity of the periodontal disease and the patient's tissue response.

Primary endodontic disease with secondary periodontal involvement should first be treated with endodontic therapy. Treatment results should be evaluated in 2 to 3 months and only then should periodontal treatment be considered. This sequence of treatment allows sufficient time for initial tissue healing and better assessment of the periodontal condition.[15] It also reduces the potential risk of introducing bacteria and their byproducts during the initial phase of healing. Aggressive removal of the PDL and underlying cementum during interim endodontic therapy might adversely affect periodontal healing.[104] Adequate endodontic therapy will cause healing of lesions of pulpal origin.

Primary periodontal disease with secondary endodontic involvement and true combined endo-perio diseases require both endodontic and periodontal considerations. The prognosis of primary periodontal disease with secondary endodontic involvement and true combined diseases depends primarily on the severity of the periodontal disease and the periodontal tissues' response to treatment. True combined diseases usually have a more guarded prognosis. In general, adequate endodontic treatment will result in healing of lesions of pulpal origin. The prognosis of combined diseases depends mainly on the success of periodontal therapy. If the etiology of the condition is unclear, a consultation with a specialist and a referral for appropriate diagnosis and treatment are indicated.

Chapter Review Questions available in Appendix B or on the DVD

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Longitudinal Tooth Fractures

Eric M. Rivera, Richard E. Walton

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Define and differentiate craze line, cusp fracture, cracked tooth, split tooth, and vertical root fracture.
2. Describe the causes of these fractures of tooth structure.

3. List and describe the five considerations (characteristics) of fractures in dentin.

4. Describe in general each of the five categories of fracture regarding incidence, pathogenesis, clinical features, etiologies, diagnosis, treatment, prognosis, and prevention.

5. Identify patients with difficult situations who should be considered for referral.

Cracked teeth and their related entities, as well as vertical root fractures, are longitudinal fractures of the crown and/or root. These contrast with horizontal fractures, which predominate in anterior teeth and result from impact trauma. Longitudinal (vertical) fractures occur in all tooth groups and are caused by occlusal forces and dental procedures. Longitudinal implies a vertical direction and a time component.[1]

There is relatively little research on longitudinal tooth fractures, particularly on clinical outcomes related to diagnosis and treatment. Most treatment modalities are based on opinion and anecdotal information.[2] Therefore many recommendations have not been substantiated in controlled clinical trials but are based on experience. This chapter deals with longitudinal fractures in the vertical plane, or long axis of the crown or root, that propagate over time.[1]

Treating longitudinal fractures is usually challenging. Sometimes these fractures are not difficult to diagnose or manage, whereas at other times they are so devastating that the involved tooth must be extracted. Notwithstanding, many situations present with significant problems in both diagnosis and treatment; these patients should be considered for referral.

INCIDENCE

The incidence of longitudinal fractures is apparently increasing. There are several reasons for this unfortunate occurrence. One is the increasing age of patients with decreased numbers of tooth extractions. Therefore more teeth undergo complex procedures and are present for longer periods of time. These procedures include restorative and endodontic treatments that remove dentin, thereby compromising internal strength. Also, the teeth absorb external forces, usually occlusal, that exceed the strength of dentin and gradually alter tooth structure. When the destructive force is beyond the elastic limit of dentin or enamel, a fracture occurs.[3] Therefore the longer a tooth is present and the more forces it undergoes, the greater the chances of an eventual fracture. Another reason for the increased incidence is more awareness and better diagnosis and identification of the problem. Such fractures are not confined to elderly patients and do not occur only in restored teeth.[4-6]

CATEGORIES

There are five categories of longitudinal fractures. From least to most severe they are (1) craze lines, (2) fractured cusp, (3) cracked tooth, (4) split tooth, and (5) vertical root fracture. Although these fractures differ, they are often confused or combined in clinical articles.[7-11] This leads to misunderstanding, with incorrect diagnosis and inappropriate treatment. Table 7-1 identifies the five entities by findings, diagnostic methods, and treatment. The reader is referred to this table throughout the chapter.

Table 7-1 Categories of Longitudinal Tooth Fractures

	Craze Line	Fractured Cusp	Cracked Tooth	Split Tooth	Vertical Root Fracture
Location	Enamel only, common on marginal ridges	Crown and cervical margin of root	Crown only or crown to root extension (depth varies)	Crown and root; extension to proximal surfaces	Root only
Direction	Occlusogingival	Mesiodistal and faciolingual	Mesiodistal	Mesiodistal	Faciolingual
Origination	Occlusal surface	Occlusal surface	Occlusal surface	Occlusal surface	Root (any level)
Etiologies	Occlusal forces, thermocycling	Undermined cusp, damaging habits	Damaging habits, weakened tooth structure	Damaging habits, weakened tooth structure	Wedging posts, obturation forces, excessive root-dentin removal
Symptoms	Asymptomatic	Sharp pain with mastication and with cold	Highly variable	Pain with mastication	None to slight
Signs	None	None of significance	Variable	Separable segments, periodontal abscess	Variable
Identification	Direct visualization, transillumination	Visualize, remove restoration	Biting, remove restoration	Remove restoration	Reflect flap and transilluminate
Diagnostic tests	None	Visible fractures of cusps, biting test, transillumination	Transillumination, staining, wedge segments (inseparable), isolated/narrow periodontal probing, biting test, magnification	Wedge segments (separable)	Reflect flap and transilluminate
Treatment	No treatment needed, esthetic	Remove cusp and/or restore	Root canal treatment depends on pulpal and periradicular diagnosis, restore with full cuspal coverage	Variable, must remove one segment, restore, or extract	Remove tooth or fractured root, consider fixed and/or removable bridge, or implant
Prognosis	Very good	Very good	Always questionable to poor	Maintain intact (hopeless), remove segment (variable)	Hopeless for fractured root
Prevention	None needed	Place conservative class II restorations, coronal protection (onlay undermined cusps)	Eliminate damaging habits (ice chewing, etc.), coronal protection (onlay undermined cusps)	Eliminate damaging habits, coronal protection (onlay cusps)	Minimize root-dentin removal, avoid wedging posts, reduce condensation forces

Fractures occur primarily in two areas, crown and root. Either area may be the site of initiation, as well as the region of principal damage. In the crown (usually extending to the root), these lesions take the form of a craze line, fractured cusp, cracked tooth, or split tooth (Figure 7-1); the latter three usually extend to the root. Roots show vertical root fracture (Figure 7-2).

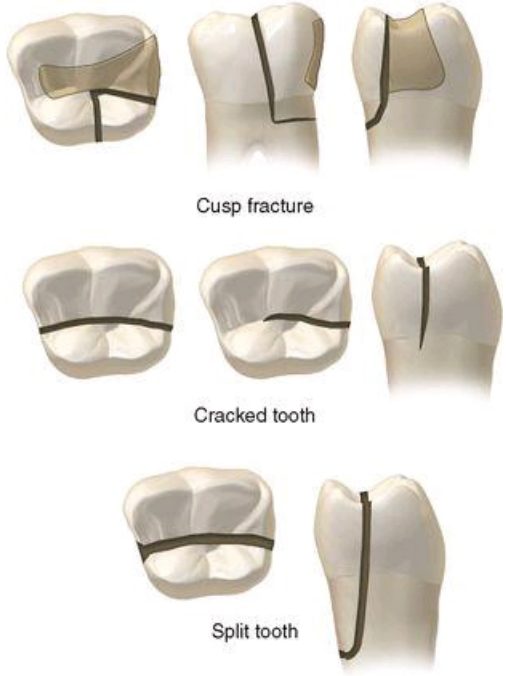

Figure 7-1 The three fracture types that originate occlusally and then extend toward the root.

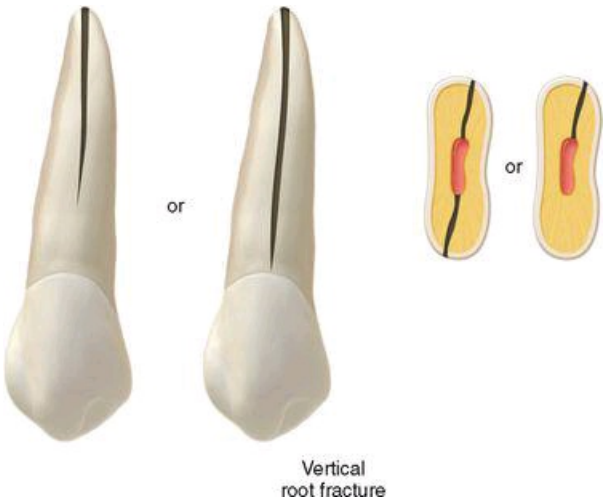

Figure 7-2 This fracture begins and ends on the root. Usually the fracture extends facially and lingually but may not extend to both surfaces or from apical to cervical.

CRAZE LINES

Craze lines are common, particularly in permanent teeth in adults (Box 7-1). They usually extend over marginal ridges and along buccal and lingual surfaces in posterior teeth but also appear as long vertical defects from incisal to cervical aspect on anterior teeth (Figure 7-3). Craze lines are confined to enamel.[2,7]

Box 7-1 Considerations for Longitudinal Fractures

Patients must be informed of the following:

1. Longitudinal fractures result from excessive forces that are usually (but not always) long-term forces.

2. Visualization of the presence or extent of the fracture may be difficult to identify clinically because they are often tiny and are not demonstrable until growth or expansion occurs. Also, they may be hidden under restorations or bone and gingiva and thus not be visible even after flap reflection.

3. With time, fracture spaces tend to acquire stains and become more visible.

4. Fractures have a tendency to grow, although initially they are very small. This propagation may be very slow. An analogy is a small crack in a windshield that may lengthen over months or years.

5. Signs and symptoms often are not present early but become manifest months, years, or decades after fracture initiation.

6. Longitudinal fractures are not diagnoses, they are findings. Pulpal and periradicular diagnoses are usually not affected by longitudinal fractures that do not communicate with the pulp. Longitudinal fractures with pulpal communication allow bacterial contamination, which impacts pulpal and periapical diagnoses.

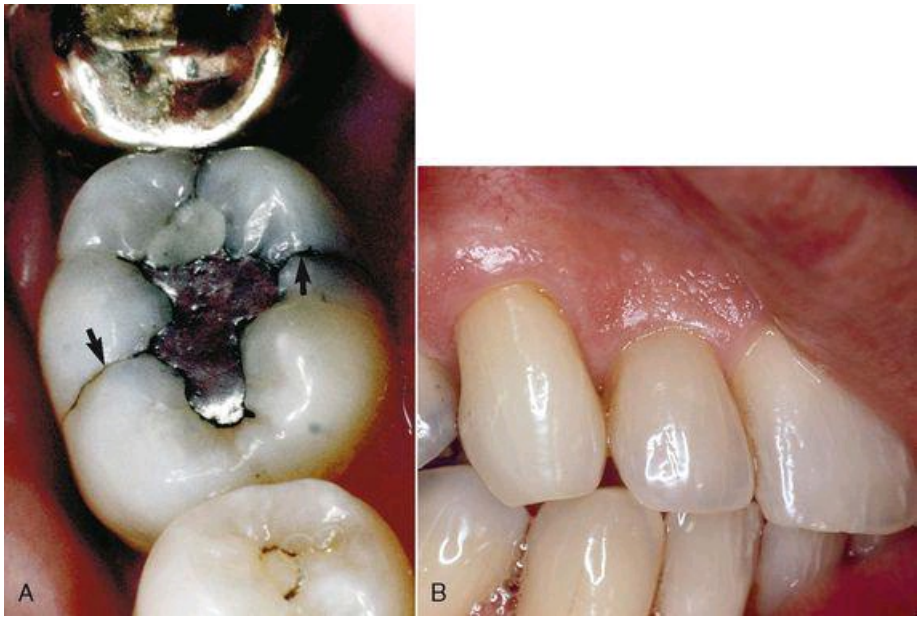

Figure 7-3 Craze lines are common. These are fractures that are limited to the enamel and do not extend to dentin. **A,** This molar shows craze lines over marginal ridges and through buccal and lingual grooves *(arrows)*. **B,** Vertical craze lines are common in anterior teeth, particularly in older patients.

Craze lines occur naturally, but their incidence in-creases in patients who have had restorations or impact injuries. It is unknown (but is unlikely) whether they are precursors to dentin fractures. Craze lines are unimportant other than as a common source of misidentification and confusion with cracked teeth.

FRACTURED CUSP

Fractured cusps are usually relatively easy to diagnose and treat and generally have a good prognosis (see Table 7-1).

Incidence

Fractured cusps are more common than the other major entities discussed in this chapter, which is fortunate because these are the least devastating and the most manageable.[2] This fracture occurs often in teeth with extensive caries or large restorations that do not protect undermined cusps.[12]

Pathogenesis

These fractures are related to lack of cusp support, especially when restorations are placed after loss of tooth structure because of extensive caries. A confusing entity is the type of cusp fracture that occurs as the result of a traumatic injury, which usually is a traumatic upward blow to the mandible resulting in a sharp impact between the maxillary and mandibular teeth. These fractures occur immediately (i.e., not over time), so they are technically not classified as a longitudinal fracture even though the end result is the loss of one or more cusps. A reminder is that the term *longitudinal* implies both a vertical direction and a time component. Fortunately, this mishap does not happen often. A single injury may cause fracture or loss of several cusps, particularly of the maxillary premolars.

Clinical Features

Cusp fractures are usually associated with a weakened marginal ridge in conjunction with an undermined cusp. These compromise dentin support for the cusp, which is supplied primarily by the marginal ridge.[13] Either a single cusp or two cusps (in molars) are involved. The single cusp fracture includes a mesiodistal and a faciolingual component (Figure 7-4). Therefore the crack lines (see Figure 7-1) cross the marginal ridge and then extend down a facial or lingual groove and often into the cervical region parallel to the gingival margin (or somewhat subgingivally, which is more common). If two cusps are involved, the fracture lines will be mesial and distal, without a facial or lingual component. Two mesial or two distal cusps are seldom fractured together.

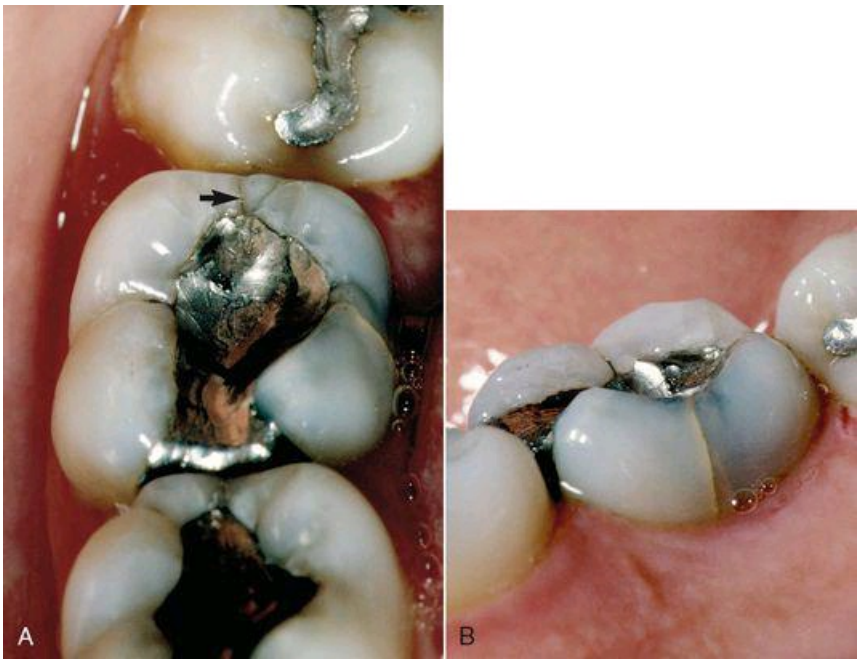

Figure 7-4 Cusp fracture. **A,** This fracture is usually associated with a class II restoration, extending across at least one marginal ridge *(arrow)* and often **(B)** down a lingual or buccal surface. These fractures tend to acquire stain with time, are usually symptomatic, and will stop light with transillumination.

Cusp fractures are oblique shearing fractures extending from the occlusal, often from a line angle at the base of a cavity (Figure 7-5). The defect often includes the region of epithelial attachment and usually does not extend beyond the cervical third of the root.[14] There usually is no pulp exposure, particularly in older teeth with smaller pulp chambers.

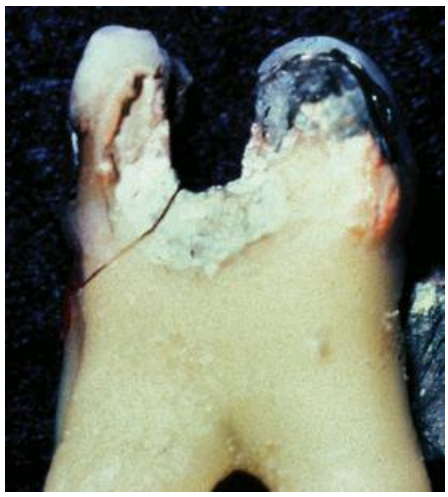

Figure 7-5 Cusp fracture. Typically, the separation occurs from the line angle of the cavity to the cervical surface.

Etiologies

Typically, there is a history of extensive deep interproximal caries or a subsequent class II restoration. Occasionally, these cusp fractures occur in nonrestored teeth with extensive undermining caries.

Diagnosis

Subjective Findings

Frequently, the patient reports brief, sharp pain on mastication. There may be sensitivity to temperature changes, particularly cold. Often the pain is more distinct with masticatory release (not with closure but with separation of teeth after biting). Pain is neither severe nor spontaneous and occurs only on stimulus. Interestingly, the symptoms are often relieved when the cusp finally breaks off.

Objective Tests

The most indicative test is biting. The patient may close onto a cotton swab applicator, a rubber polishing (Burlew) wheel, or a specially designed bite-testing instrument (Tooth Slooth or Frac Finder) (Figure 7-6). An occlusal, gnashing force on the involved cusp elicits pain. Patients ordinarily respond to pulp testing, unless the pulp has been exposed to bacteria for prolonged periods, which may result in pulp necrosis.

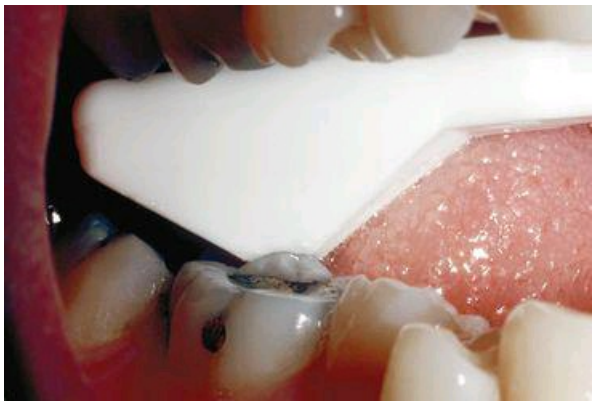

Figure 7-6 Special diagnostic “biting” instruments, such as a Tooth Slooth, are placed on one cusp at a time while the patient grinds with opposing teeth. Sharp pain on pressure or release may indicate a cusp fracture or cracked tooth.

Radiographic Findings

Radiographs are not useful because cusp fractures are usually not visible radiographically.

Other Findings

The restoration often has to be removed to observe the underlying dentin. The fracture may then be readily visible, or it may be disclosed by either staining or transillumination. Older fractures may have already acquired stain (see Figure 7-4). The cusp fracture line usually originates at the cavity floor at a line angle. A surgical microscope is very useful for identification.

Treatment

Retaining the fractured cusp is often not indicated. The cusp is removed, and the tooth is restored as appropriate. The restoration will probably be a three-fourths or full crown extending apical or to the fracture margin. Root canal treatment is often not required because the pulp is usually not exposed. Occasionally, restoration is unnecessary, and the tooth functions minus a cusp.

If the cusp is not mobile, the fracture line probably does not extend to a root surface subgingivally. In these cases, the cusp need not be removed, and a crown should be placed to hold the segments.

Prognosis

Long-term success is good because fractures tend to be shallow. Cusp fractures occasionally extend deeper, below the gingival attachment; treatment of these is more challenging. An approach to restoring deep-extending cusp fractures is described later in this chapter.

Prevention

Extensive removal of dentin support should be avoided. The width and particularly the depth of restorations should be minimized.[15] Those restorations that wedge, such as inlays, require adequate dentin support. Cusps should be reduced and onlayed if undermined; both amalgam and gold onlays provide fracture resistance.[16] Composite resins that are bonded to enamel or dentin and improperly placed may shrink excessively on polymerization. This contraction may displace and weaken cusps, rendering them susceptible to occlusal forces and fracture.

In terms of bonding, adhesive resins, if placed with special techniques, may reinforce weakened cusps.[17-19] However, resin-based composites are equivalent to amalgams as to cusp fracture occurrence in patients.[20-23] Therefore bonded restorations may provide only temporary reinforcement.[24]

CRACKED TOOTH

Cracked teeth are defined as an incomplete fracture initiated from the crown and extending subgingivally, usually directed mesiodistally.[6] The fracture may extend through either or both of the marginal ridges and through the proximal surfaces. The fracture is located in the crown portion of the tooth only or may extend from the crown to the proximal root (see Table 7-1). Cracked teeth are also described as incomplete (greenstick) fractures, which describes their form.[7,25] Cracked tooth is a variation of the cusp fracture, but the associated fracture is centered more occlusally (see Figure 7-1). The effects of cracked teeth tend to be more devastating because their extent and direction are more centered and more apical (see Table 7-1).

Incidence

The occurrence of cracked tooth is unknown but is apparently increasing.[5,7,26] Cracked teeth are predominantly seen in older patients, although they may occur at any age in adults.[4,6] The longevity and complexity of restorations are related factors, although cracked teeth often are minimally restored or not restored at all.[6,7] Mastication for many years, particularly of hard objects, is also a factor. Continued and repeated forces finally cause fatigue of tooth structure, resulting in a small fracture followed by continued growth of that fracture.

The teeth usually involved are mandibular second molars (both restored and nonrestored), followed closely by mandibular first molars, and then by either maxillary second molars or maxillary premolars, depending on the study.[6,26] Anterior teeth occasionally develop true cracks, usually as a result of weakened tooth structure from a traumatic impact or from restorations. Cracks rarely occur on mandibular premolars. Furthermore, class I restored teeth fracture as often as do class II restored teeth, particularly molars. Therefore the phenomenon is not always dependent on violation of tooth structure by access preparations, caries, or restorations. There has been speculation that teeth treated by root canals are more brittle and weakened and therefore are more susceptible to fracture. Evidence does not support this assumption.[13,27-30]

Pathogenesis

As stated previously, cracks in teeth tend to depend on time and patient habits. Obviously, forces in excess of dentin strength are responsible. These forces are greater in the posterior region (i.e., close to the fulcrum of the mandible), invoking the "nutcracker" effect.[6,31]

Although occlusal anatomy (deep fissures or prominent or functional cusps) and occlusal dysfunction might render a tooth more susceptible to cracking, these factors are only speculative because no relationship to cracked teeth has been demonstrated.

Clinical Features

Cracks in teeth are almost invariably mesiodistal fractures[6] (Figure 7-7), although mandibular molars occasionally (rarely) fracture toward the faciolingual surface. The diagnosis of a faciolingual cracked molar is a common misinterpretation because of visualization of facial and lingual fractures (see Figure 7-3, *A*). These are actually craze lines, which follow the buccal and lingual grooves.

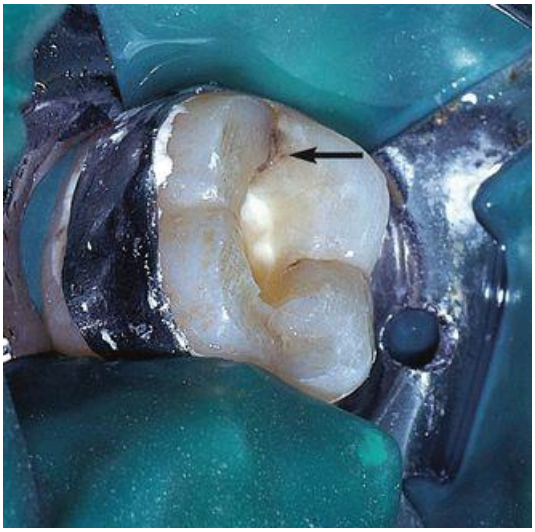

Figure 7-7 Cracked tooth. The fracture extends across both marginal ridges but is most evident on the distal *(arrow)*. Fractures in the mesiodistal direction are by far the most common.

Cracks cross one or both marginal ridges. They generally shear toward the facial or lingual side toward a root surface, usually lingual. Because the fracture begins on the occlusal surface, it grows from this surface toward the cervical surface and down the root. The more centered the fracture (initiated on the midocclusal surface), the more it has a tendency to extend deeper before it shears toward the root surface. The fracture is considered “greenstick” because it is incomplete (either to the mesial or distal surface) or does not extend to the facial or lingual root surface.[7] Wedging forces produce no separable segments that would indicate a complete fracture, as with split teeth (see section on Split Tooth). The direct midocclusal fracture may be very deep. On maxillary molars, it may extend toward the furcation (Figure 7-8) or occasionally toward the apex on mandibular molars (Figure 7-9).

Figure 7-8 Cracked tooth. The fracture extends across the marginal ridge and down the proximal close to the furcation (greenstick fracture).

Figure 7-9 Centered fracture on this lightly restored second molar extends toward the apex. Treatment of these fractures is hopeless.

The fracture may or may not include the pulp. The more centered the fracture, the greater the chance of pulp exposure now or later. Occasionally, fractures oriented toward the faciolingual surface shear away from the pulp, although this is not likely and difficult to determine clinically. Therefore many cracked teeth require root canal treatment, preferably before restoration for coronal protection. Wedging forces must be minimized during both root canal treatment and restoration to avoid aggravating the fracture.

Etiologies

Cracked teeth are often found in patients who chew hard, brittle substances (ice, unpopped popcorn kernels, hard candy, and so on). These patients may have prominent masticatory muscles and show excessive occlusal wear as a result of heavy occlusal forces.[31]

However, cracked teeth may occur in patients without these damaging parafunctional habits and who do not display heavy musculature. If these teeth are restored, the restorations may be class I or a deep class II. Interestingly, cracks associated with wide class II restorations are more likely to be cusp fractures and their effects are not as devastating.[32]

Thermal stresses are also thought to be a cause of fractures, although the evidence of this is inconclusive. Supposedly, differences in expansion and contraction of restorations versus tooth structure may weaken and crack dentin.[33]

Diagnosis

Cracked teeth show a variety of test results, radiographic findings, and signs and symptoms, depending on many factors. This variety and unpredictability often make the cracked tooth a perplexing diagnostic and treatment entity.

Subjective Findings

Often cracked teeth manifest as the so-called cracked tooth syndrome.[31] This syndrome is characterized by acute pain on mastication (pressure or release) of grainy, tough foods and sharp, brief pain with cold.[32] These findings are also related to cusp fracture. However, cracked teeth may present with a variety of symptoms ranging from slight to very severe spontaneous pain consistent with irreversible pulpitis, pulp necrosis, or apical periodontitis.[34] Even an acute apical abscess, with or without swelling or a draining sinus tract, may be present if the pulp has undergone necrosis. In other words, once the fracture has extended to and exposed the pulp, severe pulp and/or periapical pathosis will be present. This explains the variation in signs and symptoms and therefore should not be termed a syndrome.

Objective Tests

Pulp and periapical tests also have variable results. The pulp is usually responsive (vital)[31] but may be nonresponsive (necrosis). Periapical tests also vary, but usually pain is not elicited with percussion or palpation if the pulp is vital. Directional percussion is also advocated. Percussion that separates the crack may cause pain. Opposite-direction percussion usually is asymptomatic. This pain is probably related to stimulation of the periodontal ligament proprioceptors.

Radiographic Findings

Because of the mesiodistal direction of the fracture, it is not visible radiographically. Radiographs are made to help determine the pulp/periapical status. Usually there are no significant findings, although occasionally different entities occur. At times, loss of proximal (horizontal, vertical, or furcal) bone is related to the fracture; bone loss increases as the severity of the crack increases.

Newer methods of analysis are currently being studied, such as cone-beam computed tomography (CT), to help identify longitudinal fractures in a nondestructive fashion.[35-38]

Other Findings

Craze lines in posterior teeth that cross marginal ridges or buccal and lingual surfaces must be differentiated with transillumination. With craze lines, transilluminated light from the facial or lingual surface is not blocked or reflected and the entire tooth in a faciolingual orientation is illuminated.

When a crack is suspected, it is important to try to visualize the length and location of the fracture. Direct inspection (again, a microscope is helpful), staining, and transillumination are usually effective.[34] Occlusal and proximal restorations are first removed.[39] Then transillumination (Figure 7-10), which often shows a characteristic abrupt blockage of transmitted light, is performed. With transillumination the portion of the tooth where the light originates illuminates to the fracture. A fracture contains a thin air space, which does not readily transmit light. Therefore the crack (or fracture) blocks or reflects the light, causing the other portion to appear dark.

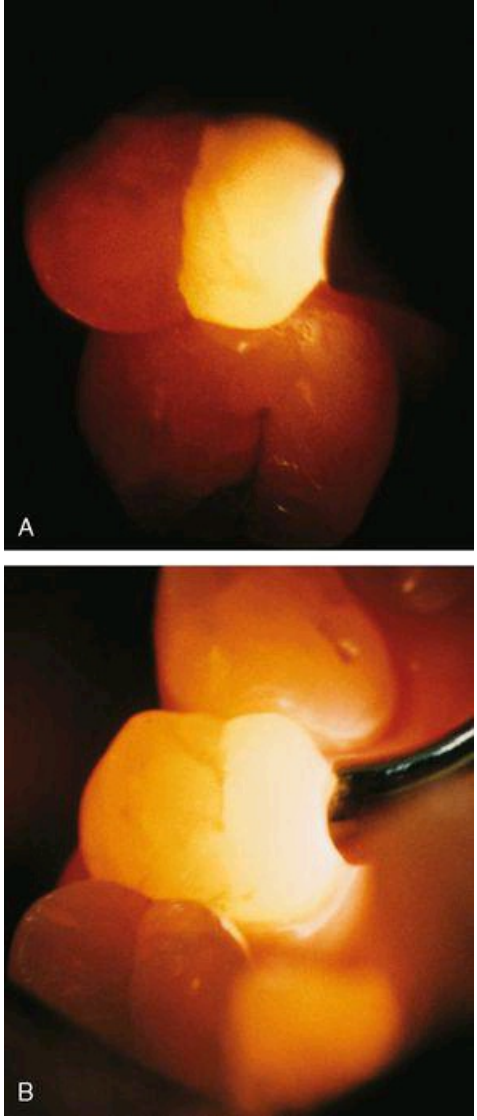

Figure 7-10 Cracked tooth. **A,** Fracture through dentin reflects transilluminated light showing abrupt change in brightness. **B,** For comparison, an adjacent noncracked premolar transmits light readily.

Staining with methylene blue or iodine may also disclose the fracture, although not predictably. A cotton pledget soaked with methylene blue or other dye is placed against the cavity floor. The dye may be washed away immediately to reveal the crack or is held in by a sealing temporary such as intermediate restorative material (IRM). The temporary restoration and pledget are removed after a few days. The dye may have contacted the crack long enough to disclose it clearly (Figure 7-11). Patients should be advised that the tooth may temporarily turn blue (see Figure 7-11); they may wish to forego this test.

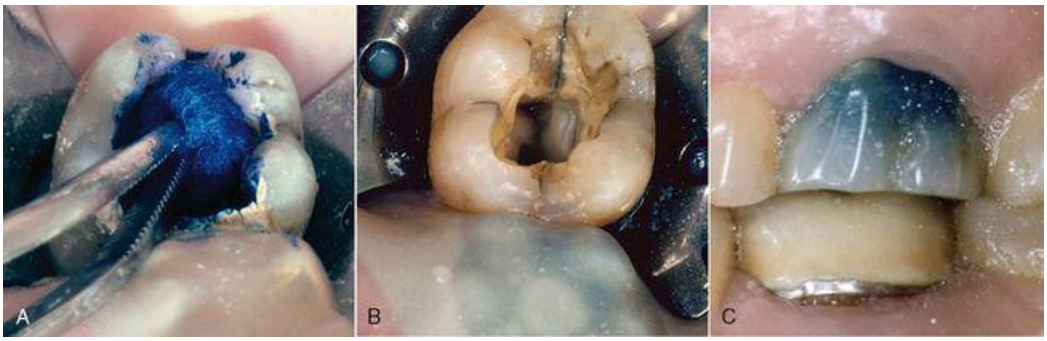

Figure 7-11 A, Disclosing solution on a cotton pellet (in this case, methylene blue) is placed in the cavity for a few minutes or sealed in for a week. **B,** This technique may clearly disclose the fracture and its extent. **C**, Staining solutions may discolor the tooth.

Viewing with a surgical microscope is particularly useful to both identify the presence and extent of the fracture (Figure 7-12).

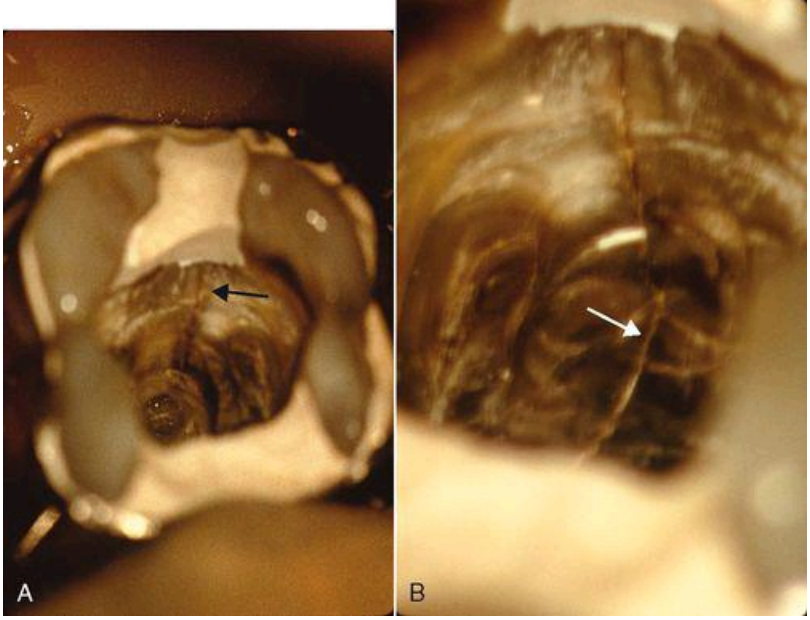

Figure 7-12 Evaluation with magnification and illumination. **A,** To minimize movement of segments, an orthodontic band has been placed. The access is now completed to determine the depth of the fracture *(arrow)*. **B**, The fracture *(arrow)* extends across the floor. The prognosis is poor.

Occasionally (particularly if the crack is centered), an access preparation is necessary to disclose the extent (see Figure 7-12) of the crack. After the chamber roof and coronal pulp have been removed, the floor is transilluminated as for a fracture (not to be confused with anatomic grooves). Sealing in a disclosing dye for a few days may be helpful. Again, visualization with the microscope allows for more definitive identification.

Removal of the fracture line in the area of the cavity floor that would include an ideal endodontic access opening is helpful in removing and/or determining the apical extent of the crack and whether the pulp is involved (Figure 7-13). However, the fracture is small and invisible at the furthest extent (even after staining). Therefore the crack probably continues deeper into dentin than can be visualized. Removal of the fracture line in the proximal portion of the tooth may provide information on the extent but also may cause the tooth to become nonrestorable. Both of these procedures, particularly removal of proximal marginal ridge and tooth structure, remove sound tooth structure, thereby decreasing tooth strength and resistance to fracture.[40,41]

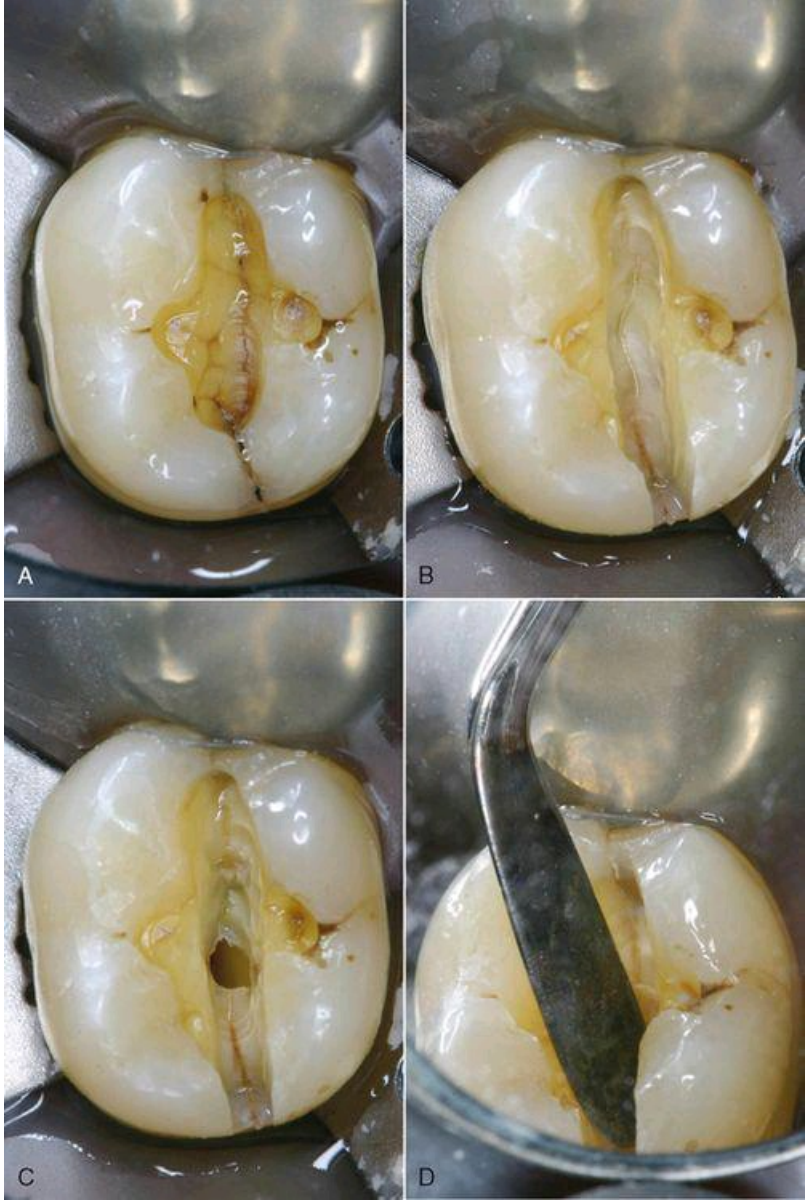

Figure 7-13 A, Removal of amalgam restoration reveals cracked tooth in mesiodistal direction. **B**, Removal of fracture line in the area of the cavity floor that would include an ideal endodontic access opening. **C**, Further removal determines fracture extending into pulp chamber. **D**, Wedging forces used to determine mobility of segments.

Wedging forces are used to determine if the tooth segments are separable (see Figure 7-13). If a fracture is detected, any restorations are removed and an instrument is placed in the cavity with moderate pressure exerted on opposing walls to try to separate the segments. If no movement is detected, the classification is a *cracked tooth.* When the segments separate, it is a *split tooth* (discussed later). The patient must be informed of possible sequelae before performing this test. Clinicians and patients may be hesitant to perform wedging of the segments for fear of splitting the tooth iatrogenically or causing pain. However, if controlled force exacerbates the crack, the tooth is predisposed to a later split anyway and the patient is best served to know this expeditiously.

Periodontal probing is important and may disclose the approximate depth and severity of the fracture. Removal of interproximal restorations is helpful because it allows improved access for placement of the periodontal probe. However, subgingival fractures often do not create a probing defect. Therefore the absence of deep probing does not preclude a cracked tooth. The presence of deep probing is serious and indicates a more adverse prognosis.[42]

Selective biting on objects (see section on Fractured Cusp) is helpful (see Figure 7-6), particularly when pain is reported on mastication.

Treatment

Six important considerations for longitudinal fractures are listed in Box 7-1. When both clinician and patient are aware of the complications and questionable outcomes, a treatment plan is formulated. Extraction is a reasonable solution in many situations. Much depends on the nature (depth and location) of the fracture (Box 7-2). Again, the segments must not separate on wedging. If they do not separate, there are many treatment alternatives to retain the tooth intact. If the occlusal-proximal fracture is centered in the faciolingual aspect and involves the floor of the cavity preparation, there are treatment options. If there are no symptoms of irreversible pulpitis, a crown may be placed, although some of these teeth will eventually manifest irreversible pulpitis or pulp necrosis.[26] They will then require treatment through the crown.

Box 7-2 Prognosis and Treatment Suggestions for Cracked Teeth

1. Pulpal and periapical diagnoses are established. The diagnosis determines the treatment. Included in this assessment is a determination of how the finding of a cracked tooth relates to the diagnosis, prognosis, and treatment (see Box 7-1, number 6).

2. Treatment alternatives are provided to the patient.

3. The patient's questions are answered, and the patient elects which treatment will be performed.

4. Prognosis is more variable with cracks than with other longitudinal fractures. Determining the position and extent may be helpful in determining prognosis and when to recommend extraction. Techniques that provide more information related to the extent of cracks internally and on the proximal surfaces below the cementoenamel junction are needed. It is hypothesized that the prognosis decreases from questionable to poor when cracks involve the following (in order):

- □ One marginal ridge limited to crown
- □ Two marginal ridges limited to crown
- □ Marginal ridge(s) and internal proximal cavity wall only
- □ Marginal ridge(s) and floor of cavity preparation (may involve restoration removal)
- □ One marginal ridge extending from crown to root surface (difficult to visualize)
- □ Two marginal ridges extending from crown to root surface (difficult to visualize)
- □ Marginal ridge(s) and into canal orifice(s)
- □ Marginal ridge(s) and pulpal floor
- □ Furcation involvement (only confirmed after exploratory surgery or extraction)

Further Examination

After endodontic access, the pulp chamber floor is examined. If the fracture extends through the chamber floor, generally further treatment is hopeless and extraction is preferred (see Figure 7-12).[43] An exception is the maxillary molar, which may be hemisected along the fracture, saving half (or both halves) of the crown and supporting roots. Many of these treatments are complex, and the patient should be considered for referral to an endodontist. If a partial fracture of the chamber floor is detected, the crown may be bound with a stainless steel or an orthodontic band (see Figure 7-12) or temporary crown to protect the cusps until final restoration is performed.[25,44] This also helps to determine whether symptoms decrease during root canal treatment. The rationale (unsupported) is that if pain symptoms are not relieved, the prognosis is significantly poorer and extraction may be necessary.

Restoration

If the fracture appears to be incomplete (not terminating on a root surface), the tooth is restored to bind the fractured segments (barrel-stave effect) and also to protect the cusps. For a permanent restoration, a full crown is preferred, although an onlay with bevels may suffice. Posts and internally wedging foundations are to be avoided. Acid-etch dentin bonding resins may help to provide a foundation for the crown to prevent crack propagation, although more research is necessary to support this concept.[45-47] Amalgam, which tends to expand and which requires a wedging effect with condensation, is not a good choice.

Prognosis

The overall prognosis depends on the situation but is always questionable at best. The patient is informed of the possible outcomes and the unpredictability of duration of treatment. The fracture may continue to grow to become a split tooth, with devastating consequences, requiring tooth extraction or additional treatments (Figure 7-14). Furthermore, the patient should be informed that cracks may be present in other teeth as well and could manifest in the future.

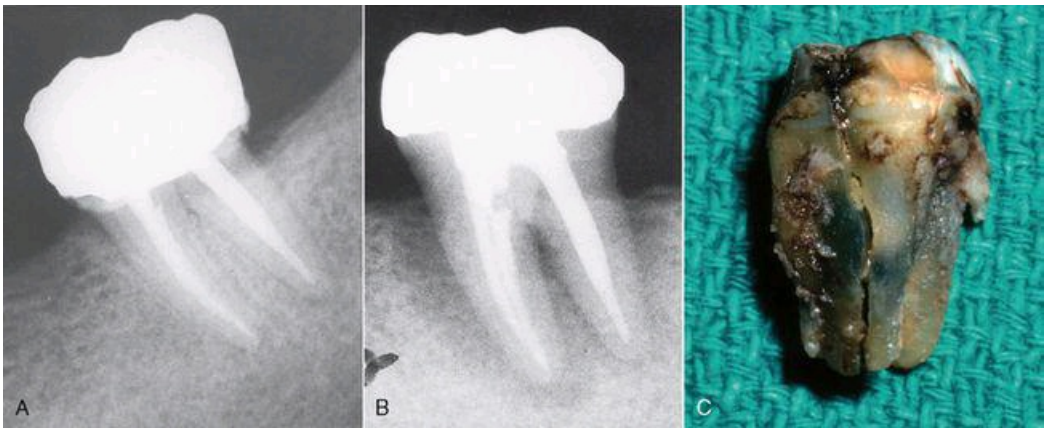

Figure 7-14 Cracked tooth resulting in eventual split tooth. **A,** A cracked tooth was identified, root canal treatment was completed, and a full crown was placed. **B,** After 3½ years the fracture became manifest with extensive bony destruction. **C,** The fracture had grown over time to become a split tooth.

In general, the more centered the origin of the fracture is on the occlusal surface, the poorer the long-term prognosis. These fractures tend to *remain* centered and grow deeper. The result is major damage to the tooth and periodontium. In other words, the cracked tooth may ultimately evolve to a split tooth or develop severe periodontal defects.

Prevention

Generally, patients are encouraged to forego destructive habits such as ice chewing. In addition, most suggestions made earlier for prevention of cusp fractures apply here. The use of deep class I or class II restorations should be minimized, particularly on maxillary premolars (cusp protection may be helpful).[48] Altering the occlusal anatomy or changing occlusal relationships is not useful.

SPLIT TOOTH

A split tooth is the evolution of a cracked tooth. The fracture is now complete and extends to a surface in all areas.[5] The root surface involved is in the middle or apical third. There are no dentin connections; tooth segments are entirely separate (see Figure 7-14, *C*). The split may occur suddenly, but it more likely results from long-term growth of an incomplete fracture (see Table 7-1).

Incidence

As with cracked tooth, the occurrence of split tooth is apparently increasing.[49] Obviously, many factors related to cracked tooth are endemic to split tooth. An assumption is that root canal treatment weakens dentin and renders teeth more susceptible to severe fractures, but this is unlikely.[13,28-30]

Pathogenesis

Causative factors related to cracked tooth also apply to split tooth. Why some cracked tooth fractures continue to grow to a complete split is unknown. Two major causes are probably (1) persistent destructive wedging or displacing forces on existing restorations and (2) new traumatic forces that exceed the elastic limits of the remaining intact dentin.

Clinical Features

Split tooth fractures are primarily mesiodistal fractures that cross both marginal ridges and extend deep to shear onto the root surfaces. The more centered the fracture is occlusally, the greater the tendency to extend apically. These fractures are more devastating. Mobility (or separation) of one or both segments is present. These fractures usually include the pulp. The more centered the fracture, the greater the probability of exposure.

Etiologies

Split tooth has the same causes as cracked tooth. Split tooth may be more common in root canal–treated teeth. However, this is not because the treatment per se weakens the tooth by dehydrating or altering dentin.[50] Rather, the strength of these teeth has already been compromised by caries, restorations, or overextended access preparations.[4]

Diagnosis

Split tooth does not have the same variety of confusing signs, symptoms, and test results as cracked tooth. Generally, split teeth are easier to identify. Damage to periodontium is usually significant and is detected by both patient and dentist.

Subjective Findings

Commonly, the patient reports marked pain on mastication. These teeth tend to be less painful with occlusal centric contacts than with mastication. A periodontal abscess may be present, often resulting in mistaken diagnosis.

Objective Findings

Objective findings are not particularly helpful but should include both pulp and periapical tests.

Radiographic Findings

Findings on radiographs depend partially on pulp status but are more likely to reflect damage to the periodontium. Often there is marked horizontal loss of interproximal or interradicular bone (Figures 7-14, *B,* and 7-15, *A*). The fracture line, which is usually mesiodistal, is not visible.

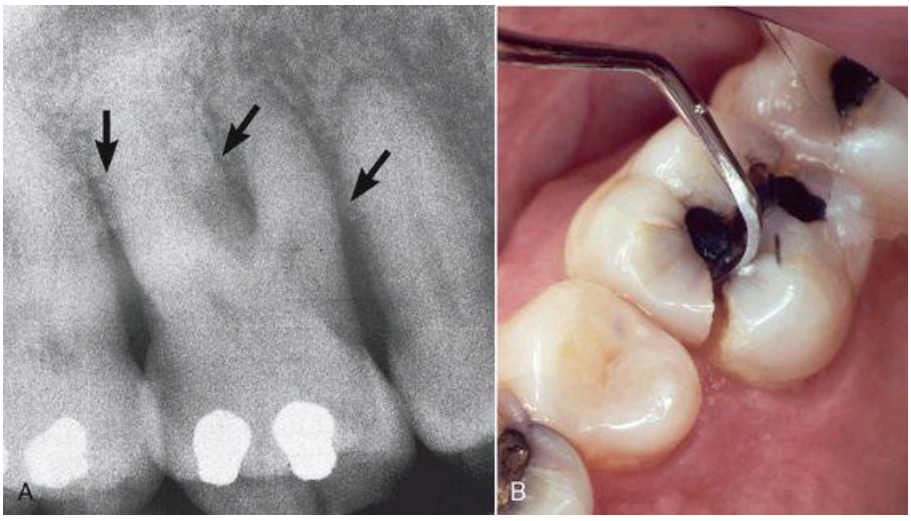

Figure 7-15 Split tooth. **A,** Often split teeth demonstrate marked horizontal and vertical bone loss *(arrows)* interproximally and in the furcation. **B,** This molar shows the definitive sign: separation of the segments on wedging.

Other Findings

The most important consideration is to identify the extent and severity of the fracture, which often requires removal of a restoration. With split tooth, the fracture line is usually readily visible under or adjacent to the restoration; it includes the occlusal surface and both marginal ridges.

Wedging to determine separability of the segments is also important. As with cracked tooth, an instrument is placed in the cavity. Wedging against the walls is done with moderate pressure; the walls are then visualized for separation (Figure 7-15, *B*). The surgical microscope is a very useful aid. A separating movement indicates a through-and-through fracture.

Periodontal probing generally shows deep defects; probings tend to be adjacent to the fracture. Here again, removal of existing restorations is helpful in visualizing interproximal areas.

Treatment

Maintaining an intact tooth is impossible. If the fracture is severe (i.e., deep apically), the tooth must be extracted. If the fracture shears to a root surface that is not too far apical (middle to cervical third of the root), the smaller segment will be very mobile. Then there is a good possibility that the small segment can be removed and the remainder of the tooth salvaged.

Different approaches to maintenance are used, depending on conditions. Some choices are the following:

Remove the fractured segment. Then the type of treatment and restoration are determined. However, the following choice (retention of fractured segment temporarily) is preferred and is generally less complicated.

Retain the fractured segment temporarily (Figure 7-16). First, a rubber dam is applied with a strong rubber dam clamp to isolate and hold the segments together. Root canal treatment is completed (if not already performed), and restoration with a retentive amalcore (onlaying the undermined cusps) or bonded restoration is performed. Then the fractured segment is removed. Granulation tissue proliferates to occupy the space and reattach the periodontium to the root dentin surface. The final restoration usually is the amalcore but may be a full crown with a margin related to the new attachment.

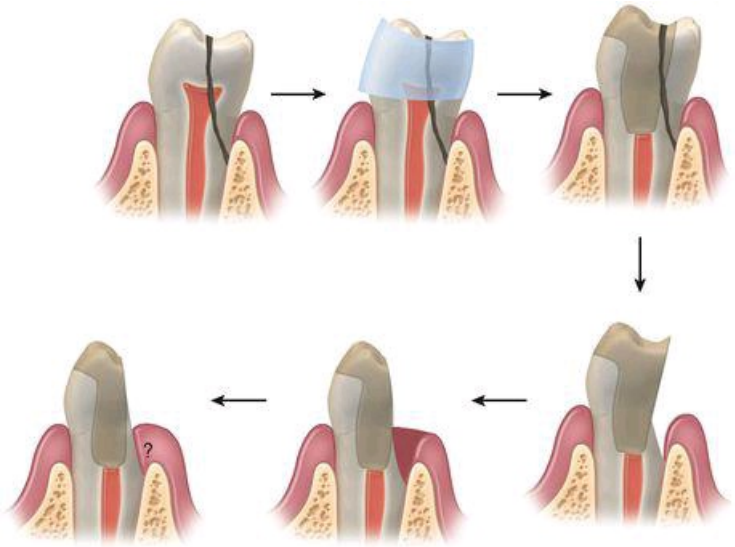

Figure 7-16 A technique for managing certain split teeth and cusp fractures. From *upper left* to *lower left:* Separable segment is held with a matrix or band. Root canal treatment is followed by an amalcore onlay. The fractured segment is removed and the amalgam is contoured. The tissue will heal and usually reattach. The nature of the attachment (connective or epithelial tissue) is unknown. Usually, normal sulcus depth is reestablished.

Remove the fractured segment and perform crown lengthening or orthodontic extrusion. The mobile segment is removed first; root canal treatment is then performed, followed by crown lengthening or orthodontic extrusion and placement of an appropriate restoration. This is not feasible in most situations because the fracture is too deep on the root surface.

Remove the fractured segment and perform no further treatment. This choice is appropriate when root canal treatment has been completed previously and the tooth already restored. All pulp space areas *must* be filled to the margins with permanent restorative material (e.g., amalgam) with no root-canal filling material exposed (Figure 7-17). The defect often granulates in, and reattachment to the fractured dentin surface occurs.

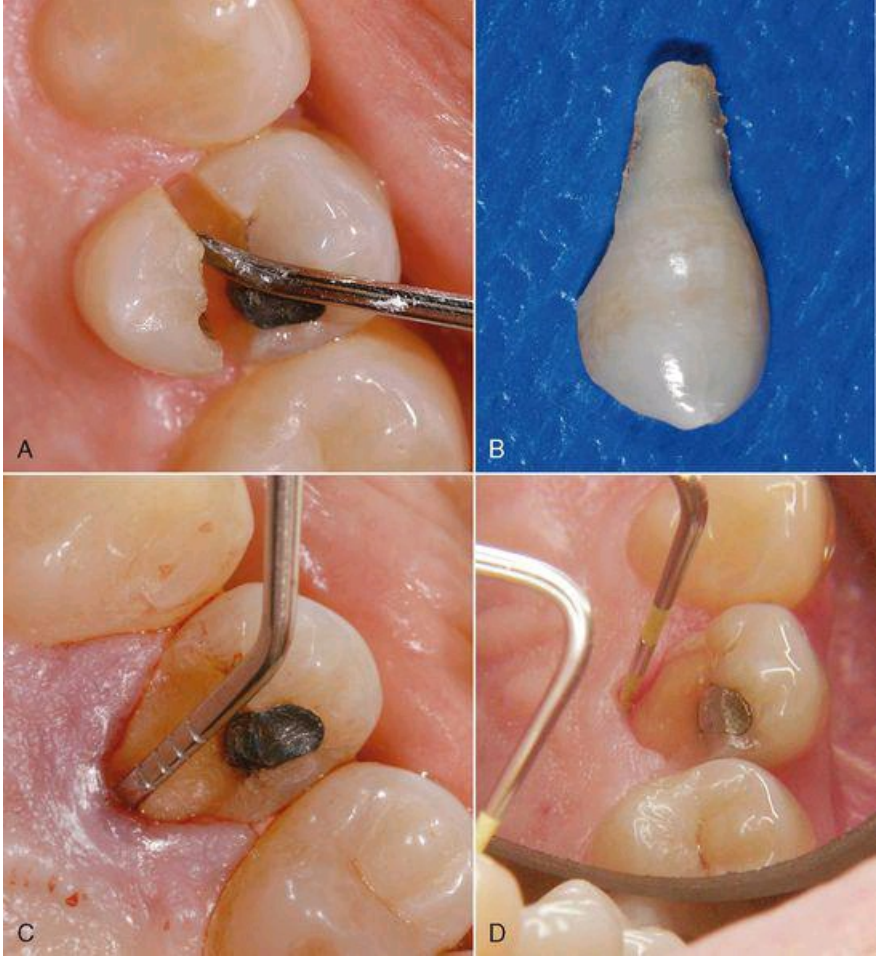

Figure 7-17 A technique for managing fractures. "Cuspidization" of a premolar. **A,** Lingual cusp fractured. **B,** Fracture extended deep subgingival. **C,** With a deep probing defect. **D,** After a brief healing period, the tissue has reattached to form a normal sulcus depth.

If the fracture does not extend to the pulp, the segment is removed; the epithelial and periodontal tissues will usually reattach to the fresh, raw dentin surface (see Figure 7-17).

In summary, treatment may be complex or relatively simple, depending on the situation. Because of the complexity, these patients should be considered for referral to an endodontist for diagnosis and treatment.

Prognosis

As expected, prognosis is variable. Some treatments of split tooth are successful, whereas others are doomed to failure if attempted. When the fracture extends to and surfaces in the middle to cervical third of the root, there is a reasonable chance of successful treatment and restoration. If the fracture surfaces in the middle to apical third, the prognosis is poor. With these deep fractures, usually too much of the pulp space is exposed to the periodontium. Root canal treatment with restoration of this space would result in deep periodontal defects.

Sometimes prediction of success or failure cannot be determined before treatment is completed if the more conservative approach is taken (i.e., if the segment is temporarily held in place during root canal treatment and restoration). After root canal treatment has been completed and the segment has been removed, the dentist may discover that unfortunately the fracture is indeed very deep and the tooth cannot be salvaged. The patient must be informed of all these possibilities before beginning treatment.

Prevention

Generally, preventive measures are similar to those recommended for cracked tooth: eliminating oral habits that damage tooth structure and impose wedging forces. Teeth requiring large, deep access preparations should be protected by an onlay or full crown. Large access preparations also require appropriate cusp protection.[13]

VERTICAL ROOT FRACTURE

Vertical root fracture (VRF) differs from the entities (see Table 7-1) previously described because the treatment plan is easy; however, diagnosis often is tricky and elusive because the VRF mimics other conditions.[51,52] Because treatment invariably consists of either tooth extraction or removal of the fractured root, an error in diagnosis has serious consequences.

Incidence

The overall occurrence is unknown, but VRF is not uncommon.[53] These defects occur more often in teeth that have undergone complex restorative procedures such as root canal treatment and intraradicular post retention.

Pathogenesis

VRFs result from wedging forces within the canal. These excessive forces exceed the binding strength of root dentin, causing fatigue and fracture. Irritants that induce severe inflammation in the adjacent periodontium result from the fracture.[54] Generally, this periodontal destruction and the accompanying findings, signs, and symptoms bring the fracture to the attention of the patient or dentist.

Clinical Features

VRFs occur primarily in the faciolingual plane (see Figure 7-2).[51,53,55-60] They are longitudinal and may either be short or extend the length of the root from apical to cervical (Figure 7-18). The fracture probably begins internally (canal wall) and grows outward to the root surface. In addition, the fracture may begin at the apex or at midroot.[61] Therefore it may be incomplete (see Figure 7-2), extending neither to both facial and lingual root surfaces nor from apical to cervical root surfaces.

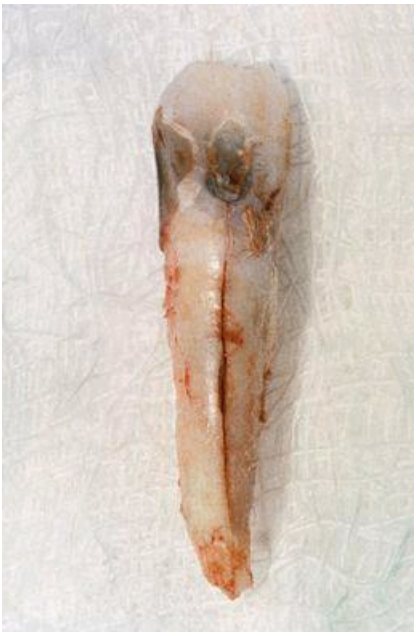

Figure 7-18 Vertical root fracture extends facially and lingually and in this case in an apical to cervical direction.

Although VRFs usually show only mild clinical signs and symptoms, the effects on the periodontium are eventually devastating and irresolvable.

Etiologies

There are two major causes (the only demonstrated ones) of VRFs: (1) post placement (cementation) and (2) condensation during root canal filling.[62] The only reported cases of VRF occurring in nonendodontically treated teeth are in Chinese patients.[63,64] Other causes, such as occlusal forces, wedging of restorations, corrosion and expansion of metallic posts, and expansion of postsurgical retrograde restorations, have been mentioned but not convincingly shown to cause VRF.

Condensation, both lateral and vertical, may cause excessive wedging forces, creating a VRF.[56,60,61,65-71] Intraradicular retentive posts have also been implicated.[62,65,72,73] Two aspects of posts cause wedging forces. Wedging occurs during cementation of posts and also during the seating of tapered posts or with posts that depend on frictional retention.[62,67] Occlusal forces exerted on the post after cementation and restoration may also be a factor but probably a minor one. Post placement (cementation) has been shown to exert a greater wedging force than lateral condensation.[67]

Certain root shapes and sizes are more susceptible to VRF. Roots that are curved and deep facially and lingually but narrow mesially and distally are particularly prone to fracture.[58,61] Examples are mandibular incisors and premolars, maxillary second premolars, mesiobuccal roots of maxillary molars, and mesial and distal roots of mandibular molars. Round, oval, or bulky roots are resistant to fracture, for example, maxillary central incisors, lingual roots of maxillary molars, and maxillary canines.

Susceptibility of any root to fracture is markedly increased by excessive dentin removal during canal instrumentation or post preparation.[56,73,74] An additional factor occurring during condensation is the placement of excessive numbers of accessory cones requiring multiple spreader insertions.[61,66] Also, the insertion of tapered, inflexible condensing instruments into curved canals creates root distortion and the potential for fracture.[68]

Diagnosis

Vertical root fractures become manifest by a variety of signs, symptoms, and other clinical findings. They may mimic other entities such as periodontal disease or failed root canal treatment. This variety of findings often makes VRF a perplexing diagnosis.[53] Interestingly, because VRFs are often mistaken for periodontal lesions or for failed root canal treatment, the dentist may refer these difficult diagnosis patients to the periodontist or endodontist, presumably for periodontal therapy or endodontic retreatment.

Diagnostic findings of VRF were reported in a series of 42 clinical cases in a study performed by Michelich et al.[52] Much of the information that follows is derived from the findings in that study in conjunction with other reports.

Subjective Findings

Symptoms tend to be minimal. Seldom is the VRF painful; it is often asymptomatic or shows mild, insignificant signs and symptoms. Often, some mobility is detectable, but many teeth are stable. Periradicular symptoms (pain on pressure or mastication) are common but mild.

Because many VRFs resemble periodontal lesions, a periodontal-type abscess (either as a presenting sign or in the history) is a common occurrence.[54,65] In fact, this localized swelling is often what brings the patient to the dentist's office.

Objective Tests

Periradicular tests of palpation and percussion are not particularly helpful. Periodontal probing patterns are more diagnostic. Significantly, some teeth with VRFs have normal probing patterns.[52] Most show significant probing depths with narrow or rectangular patterns, which are more typical of endodontic-type lesions.[53,57,65,75] These deep probing depths are not necessarily evident on both the facial and lingual aspects. Overall, probing patterns are not in themselves totally diagnostic, but they are helpful.

Radiographic Findings

Radiographs also show a variety of patterns. At times there are no significant changes.[52] However, when present, bone resorptive patterns tend to be marked, extending from the apex along the lateral surface of the root, and often include angular resorption at the cervical root (Figure 7-19).[60,76,77] However, many of the resorptive patterns related to VRF mimic other entities. The resorptive pattern may extend over the apex and along one root surface, described as a "J-shaped" or "halo" pattern.[60,76,77] Lesions may resemble failed root canal treatment because they have an apical "hanging drop" appearance.[60,76,77] In only a small percentage of teeth is there a visible separation of fractured root segments (Figure 7-20).[52] Interestingly, VRFs may be more readily identified using CT rather than conventional radiography.[35-38]

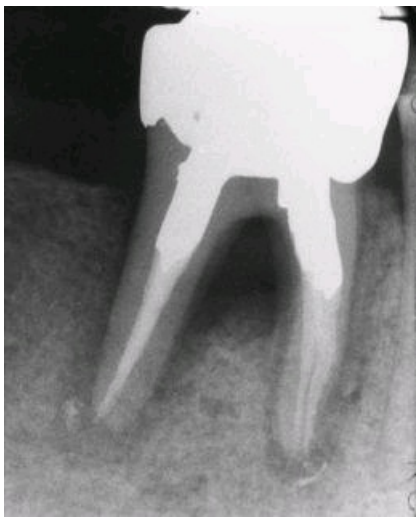

Figure 7-19 Vertical root fracture of the distal root. A common radiographic pattern of bone resorption is seen. The defect extends along the fractured root and into the furcation.

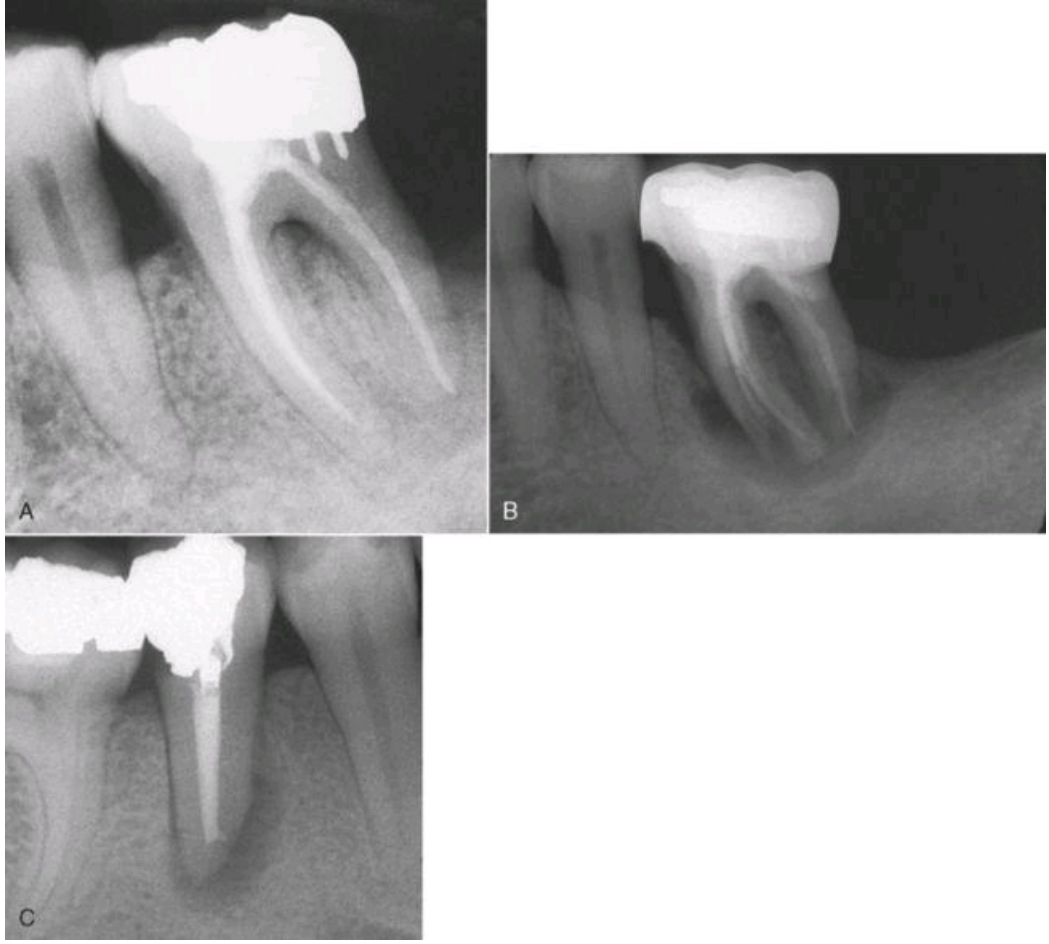

Figure 7-20 Vertical root fracture. **A,** At the time of root canal treatment and restoration. **B,** Several years later the fracture manifests devastating results. Visualizing the fracture on a radiograph is unusual. **C,** The common "J-shape" of bone resorption with a VRF.

The idea that a radiolucent line separating the root-canal filling material from the canal wall is diagnostic has been advocated. However, this radiolucent line may be a radiographic artifact, incomplete root canal filling, an overlying bony pattern, or other radiographic structure that is confused with a fracture. Therefore radiographs are helpful but are not solely diagnostic except in those few instances in which the fracture is obvious.

Dental History

Virtually all teeth with a VRF have had root canal treatment,[9] and many have been restored with cast or prefabricated posts. Conventional tapered wedging posts and cores have higher failure thresholds but potentially result in greater destructive forces involving tooth fracture.[78,79] The newer fiber post and core systems have lower failure thresholds and are more likely to fail by core fracture rather than by tooth fracture.[46,79-83] Posts that are poorly designed (too long or too wide) are a frequent culprit.[8]

Endodontic and restorative treatment may have been done months or years before the fracture. Forces (without fracture) are established at the time of treatment or restoration.[56,64,65,67] These forces are stored in root dentin but may not result in an actual fracture until later. Neither patient nor dentist may relate the fracture to earlier procedures.

Other Findings

Signs, symptoms, and radiographs all give variable findings. However, having a sinus tract and a narrow, isolated periodontal probing defect in association with a tooth that has had root canal treatment, with or without a post placement, is considered to be pathognomonic for the presence of a vertical root fracture.[53,60,84]

Flap reflection is the only reliable diagnostic approach. Surgical exposure of soft tissue and bone overlying the root surface is the best method of identification.[51,52] VRFs have consistent patterns (Figure 7-21). There is usually a "punched-out" bony defect that tends to be oblong and overlies the root surface. This defect may take the form of a dehiscence or fenestration at various root levels. The defect is filled with granulomatous tissue.

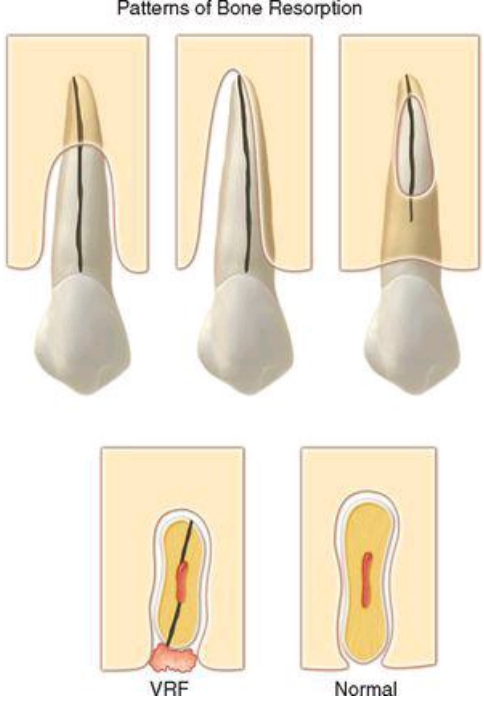

Figure 7-21 Vertical root fracture. After flap reflection and visualization, the pattern of bony changes tends to be consistent with oval or oblong "punched-out" defects filled with granulomatous tissue (VRF). This is differentiated from the normal bony fenestration.

After inflammatory tissue has been removed, the fracture is usually (but not always) visible on the root (Figure 7-22). The operating microscope is useful. If not obvious, the fracture line may be hidden or very small and undeveloped. However, the characteristic punched-out, granulomatous tissue–filled defect is diagnostic of VRF, which should be strongly suspected.[52] Transillumination or staining with dyes are helpful. Also, the root end could be resected and examined under magnification to detect the fracture.

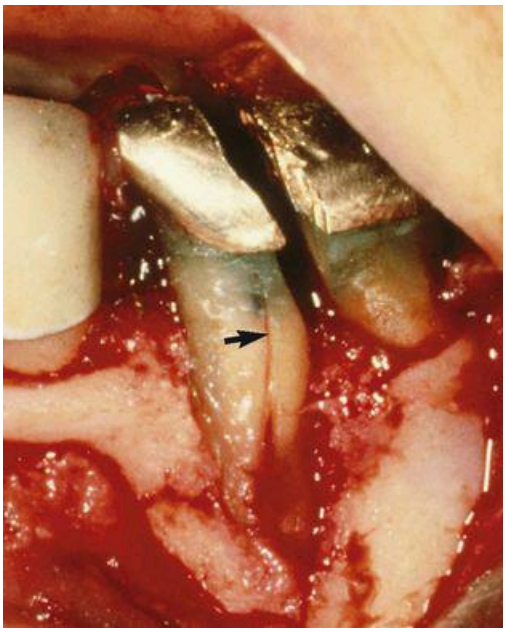

Figure 7-22 Vertical root fracture is usually but not always identified *(arrow)* after flap reflection. Usually (as in this case), the bone has been resorbed, showing a long defect. This molar has been hemisected, and the fractured mesial segment will now be removed.

Fracture Characteristics

Histologic fracture characteristics have been described with VRF after removal.[54] All fractures extended from the canal to at least one root surface but not necessarily to both (Figure 7-23). Usually, fractures extend to facial and lingual surfaces (see Figure 7-23). Similarly, fractures often extend only the partial length of the root, usually to the apex but not always to the cervix.

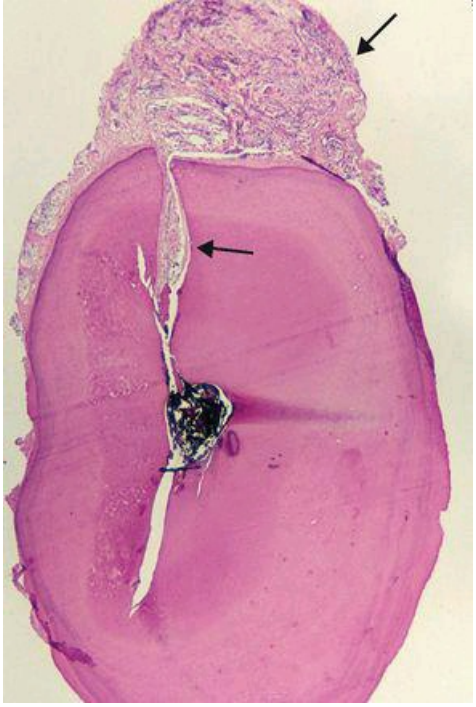

Figure 7-23 Vertical root fracture. This histologic cross section shows a fracture extending to only one surface and to the canal. The fracture space and root surface show inflammatory tissue *(arrows)*.

Many irritants occupy the fracture space and adjacent canal.[54] Fractures harbor bacteria, sealer particles, and amorphous material. Canals adjacent to the fracture often contain necrotic tissue, as well as concentrations of bacteria. Periodontal tissues adjacent to the fracture are chronically inflamed. Occasionally, connective tissue grows into the fracture toward the canal; this is often associated with resorption at the root surface (Figure 7-24).

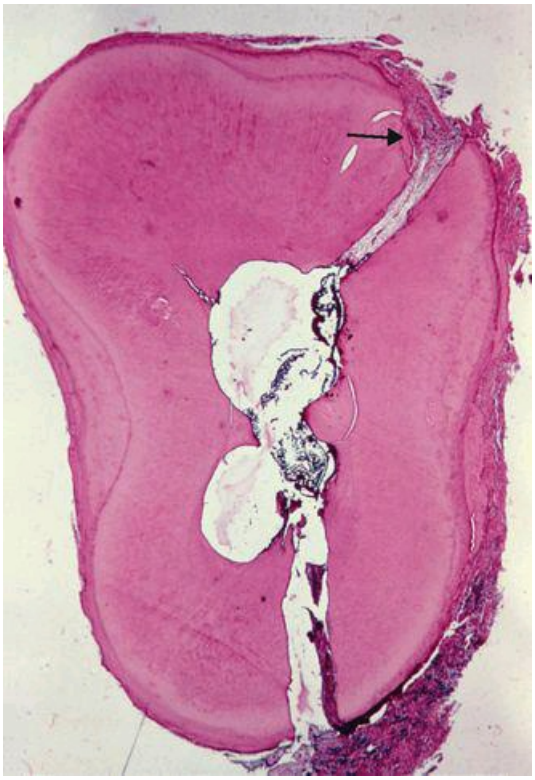

Figure 7-24 Vertical root fracture extends to both surfaces (facial and lingual). The facial surface shows resorption *(arrow)* and ingrowth of connective tissue. The lingual component contains necrotic debris and bacteria.

Thus profound irritants related to the fracture with resulting inflammation at the surface were identified in the study.[54] VRFs resemble a very long apical foramen that communicates with necrotic pulp containing bacteria—thus the hopeless prognosis.

Treatment

As stated earlier, the only predictable treatment is removal of the fractured root. In multirooted teeth, this could be done by root amputation or hemisection (see Figure 7-22).[85]

Other modalities have been suggested in attempts to reduce the fracture or retain the root: placement of calcium hydroxide, ligation of the fractured segments, or cementation of the fractured segments, trying to bind them by adhesive resins, epoxies, or glass ionomer.[86,87] A unique approach is to extract the tooth; repair the fracture with a laser, cement, or bonding agent; and then replant the tooth. Many of these suggested methods are impractical and have not been shown to have long-term effectiveness. Surgical repairs, such as removal of one of the fractured segments or repair with amalgam or resin after surgical exposure and preparation, have also been suggested, but successful results have limited documentation.

Prognosis

At present, prognosis is virtually hopeless for a tooth with a vertically fractured root.

Prevention

Because the causes of VRF are well known, prevention is not difficult. The cardinal rules for safety are to (1) *avoid excessive removal of intraradicular dentin* and (2) *minimize internal wedging forces*. The binding strength of root dentin is considerable but is easily compromised. Treatment and restorative procedures that require minimal dentin preparation should be selected. Canal preparation techniques that overenlarge the canal and overly aggressive instruments, such as nickel titanium files that are more tapered, must be further evaluated with respect to their effect on changing the fracture resistance of teeth.[59,70,88,89]

Condensation of obturating materials should be carefully controlled. More flexible and less tapered finger pluggers or spreaders are preferred because they are safer than stiff, conventional hand-type spreaders.[66,68,90] Posts weaken roots and should not be used unless they are necessary to retain a foundation (see Chapter 16). The post design least likely to cause stress and to fracture dentin is the flexible (including carbon-fiber) or cylindrical (parallel-sided) preformed post,[62,79] although these designs are not suitable in all restorative situations. Cast posts or some of the tapered preformed posts may be necessary. Their shape may exert wedging forces that readily split roots or cause dentin strain, particularly if they lack a stop or ferrule on the root seat.[91-96] Any post used should be as small as possible, have a passive fit, and not lock or grip the root internally with threads.[62] Cementation should be done carefully and slowly; an escape vent for the cement is probably helpful.

Chapter Review Questions available in Appendix B or on the DVD

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Local Anesthesia

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Explain why apprehension and anxiety, fatigue, and tissue inflammation create difficulties in obtaining profound anesthesia.
2. Define pain threshold and the factors affecting pain threshold.
3. Describe patient management techniques that will facilitate obtaining adequate anesthesia.
4. List techniques that are helpful in giving "painless" injections.
5. Describe the "routine" approach to conventional local anesthesia: when and how to anesthetize.

6. Describe circumstances that create difficulties in obtaining profound anesthesia using conventional techniques.

7. Describe when to use supplemental methods of obtaining pulpal anesthesia if standard block or infiltration methods fail.

8. Review techniques of intraosseous, periodontal ligament, and intrapulpal injections.

9. Discuss how to obtain anesthesia for specific pulpal and periradicular pathoses: irreversible pulpitis, symptomatic teeth with pulpal necrosis, asymptomatic teeth with pulpal necrosis, and surgical procedures.

> When a tooth which is loose or painful is to be extracted, the nose of the patient should be rubbed with brown sugar, ivy and green oil; he is advised to hold his breath, a stone is then placed between his teeth, and he is made to close his mouth. The fluid which causes the pain is then seen to flow from the mouth in such quantity as frequently to fill three pots; after having cleansed the nose with pure oil, rinsed the mouth with wine, the tooth is no longer painful, and may easily be extracted.
> SCRIBONIUS, 47 AD

This historical anecdote illustrates Scribonius' description of a method of obtaining "anesthesia." He was convinced that he could perform painless extractions using what was apparently a rather crude technique of pressure anesthesia. Our concern for the patient continues: how are adequate levels of anesthesia attained to keep our patients relatively comfortable during endodontic procedures? Obtaining profound anesthesia for the endodontic patient is difficult and challenging. Many patients recount vivid (and often valid) accounts of painful experiences. Although routine anesthetic techniques are usually effective for restorative dentistry, endodontic procedures present special situations that require additional techniques and special approaches.

FACTORS AFFECTING ENDODONTIC ANESTHESIA

Emotional considerations, as well as tissue changes, impair the effectiveness of local anesthesia.[1] A patient who is psychologically distraught with an inflamed pulp or periapex will have a decreased pain threshold (i.e., less stimulus is required to produce pain).[2]

In addition, the trigeminal nerve, which supplies primary sensory innervation to oral structures, is a complex entity. Knowledge of its more common anatomic features will aid in successful anesthesia.

Apprehension and Anxiety

Many endodontic patients have heard horror stories about root canal treatment. The cause may not be the treatment but the experience of a painful or “infected” tooth. They vividly recall the pain, swelling, and sleepless nights associated with the tooth before treatment. The procedure itself is generally less threatening—a survey of endodontic patients completing therapy indicated that 96% would agree to have future root canal treatment.[3] Therefore because they fear the unknown and have heard unfavorable stories, patients will be apprehensive or anxious. This emotion plays a role in their perceptions and also affects how they react to pain. Many patients effectively mask this apprehension!

Fatigue

Over a course of days, many patients with a toothache have not slept well, not eaten properly, or otherwise not functioned normally. In addition, many are apprehensive or anxious about the appointment. The end result is a patient with a decreased ability to manage stress and less tolerance for pain.

Tissue Inflammation

Inflamed tissues have a decreased threshold of pain perception.[4] This is the “allodynia” phenomenon. In other words, a tissue that is inflamed is much more sensitive and reactive to a mild stimulus.[4] Therefore an inflamed tissue responds painfully to a stimulus that otherwise would be unnoticed or perceived only mildly. Because root canal treatment procedures generally involve inflamed pulpal or periradicular tissues, this phenomenon has obvious importance. A related complication is that inflamed tissues are more difficult to anesthetize.[5]

A good example of the phenomenon of increased sensitivity is sunburn. Exposed tissues that have been burned are irritated and inflamed. The skin has now become quite sensitive (decreased pain threshold) to contact and is painful. This same principle also applies to inflamed pulpal and periradicular tissues.[6]

Previous Unsuccessful Anesthesia

Unfortunately, profound pulpal anesthesia is not always obtained with conventional techniques. Previous difficulty with teeth becoming anesthetized is associated with a likelihood of subsequent unsuccessful anesthesia.[7] These patients are likely to be apprehensive (decreased pain threshold) and generally identify themselves by comments such as “Novocain never seems to work very well on me” or “A lot of shots are always necessary to deaden my teeth.” The practitioner should anticipate difficulties in obtaining anesthesia in such patients. Often, psychologic management and supplemental local anesthesia techniques are required.

INITIAL MANAGEMENT

The early phase of treatment is most important. If the patient is managed properly and anesthetic techniques are done smoothly, the pain threshold elevates. The result is more predictable anesthesia and a less apprehensive, more cooperative patient.

Psychologic Approach

The psychologic approach involves the four Cs: control, communication, concern, and confidence. *Control* is important and is achieved by obtaining and maintaining the upper hand. *Communication* is accomplished by listening and explaining what is to be done and what the patient should expect. *Concern* is shown by verbalizing awareness of the patient's apprehensions. *Confidence* is expressed in body language and in professionalism, giving the patient confidence in the management, diagnostic, and treatment skills of the dentist. Management of the four Cs effectively calms and reassures the patient, thereby raising the pain threshold.

"Painless" Injections

Patients generally have a fear of dental injections. A good practice builder and method of winning confidence and cooperation is to master injection techniques that are nearly painless.[8] Assuredly, patients will inform their friends and family that their dentist does not "hurt when he/she gives me a shot." Although most injections cannot be totally pain-free, there can be a minimum of discomfort. This also relaxes the patient and effectively raises the pain threshold and the tolerance level.

Obtaining Patient Confidence

Patient confidence is critical. Before any injection is given, establishing communication, exhibiting empathy, and informing patients of an awareness of their apprehension, as well as their dental problem, will markedly increase confidence levels.[9] Most importantly, having the patient's confidence will give control of the situation to the dentist; this is a requisite!

Topical Anesthetic

Use of a topical anesthetic is popular as an adjunct to painless injections. Some investigators have shown topical anesthetics to be effective,[10-13] whereas others have not.[14] A particularly important area of the mouth where topical anesthesia has been shown to be effective is in the maxillary anterior region.[13] The most important aspect of using topical anesthesia is not primarily the actual decrease in mucosal sensitivity but rather the demonstrated concern that everything possible is being done to prevent pain. Another aspect is the power of suggestion that the topical anesthetic will reduce the pain of injection.[14] When a topical anesthetic gel is used, a small amount on a cotton-tipped applicator is placed on the dried mucosa for 1 to 2 minutes before the injection.[15]

A relatively new device is an intraoral adhesive, 20% lidocaine patch. When in place for 5 minutes, it was shown to reduce the pain of needlestick.[12,16] However, because of poor adherence to oral mucosa and the extra time needed for application, some authors have not supported the use of the patch.[17]

Solution Warming

A common belief is that an anesthetic solution warmed to or above body temperature is better tolerated and results in less pain during injection. However, in a clinical trial, patients could not differentiate between prewarmed and room temperature anesthetic solutions.[18] Therefore warming anesthetic cartridges is not necessary.

Needle Insertion

Initially, the needle is inserted *gently* into the mucosal tissue.

Small-Gauge Needles

A common misconception is that smaller needles cause less pain, but this is not true for dental needles. Patients cannot differentiate between 25-, 27-, or 30-gauge needles during injections.[19] These sizes also have similar deflection patterns and resistance to breakage.[20,21] As a recommendation, a 27-gauge needle is suitable for most conventional dental injections.

Slow Injection

A slow injection decreases both pressure and patient discomfort.[22] A technique for slow injection is to use a computer-controlled anesthetic delivery system (Figure 8-1). The device delivers 1.4 ml of anesthetic solution over 4 minutes and 45 seconds (slow rate). Most studies on the computer-controlled anesthetic delivery system compared the pain of injection with the delivery system to standard syringe injections,[23-30] generally with favorable results.[25-30] Some showed no difference[23] or even increased pain.[24] However, the system is not painless.[23-30]

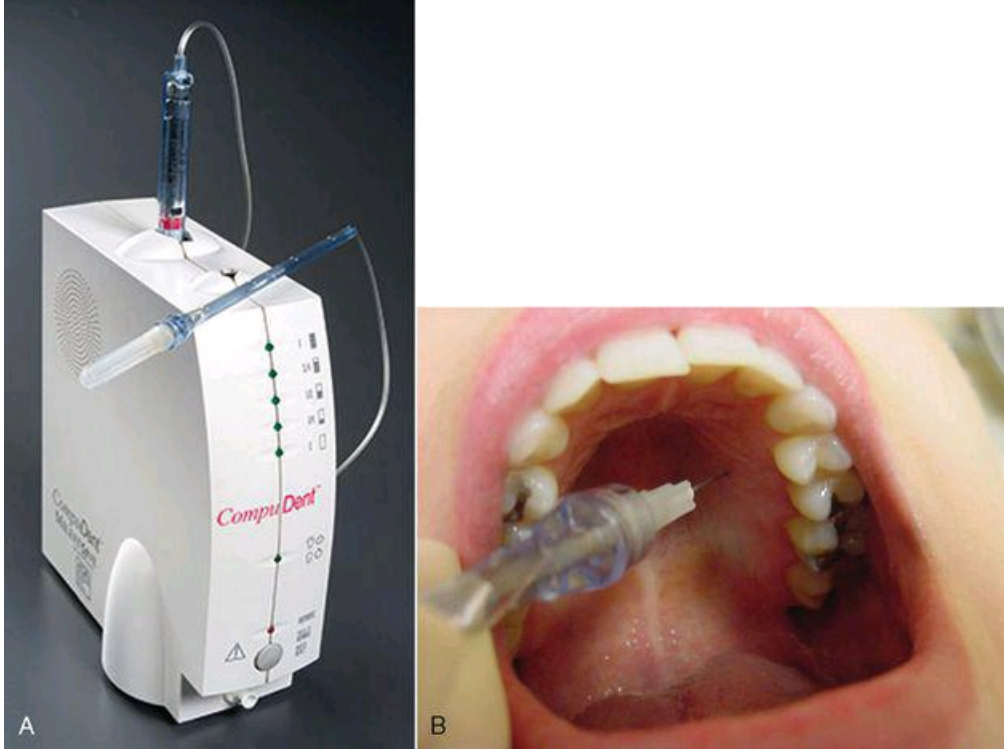

Figure 8-1 A, Computer-controlled injection device. Note the handpiece assembly and microtubing. **B**, The specialized handpiece and needle may be used in most situations, including a palatal approach.

Two-Stage Injection

A two-stage injection consists of initial very slow administration of approximately a quarter-cartridge of anesthetic just under the mucosal surface. After regional numbness, the remainder of the cartridge is deposited to the full depth at the target site. The two-stage injection decreased the pain of needle placement for females in the inferior alveolar nerve block.[31] This injection technique is indicated for apprehensive and anxious patients or pediatric patients but may be used on anyone. It is also effective for any injection including the inferior alveolar nerve block.

Gender Differences in Pain

Women try to avoid pain more than men, accept it less, and fear it more.[32,33] Anxiety may also modulate differences in pain responses between males and females.[33] Apparently, women react differently to pain than men and are more likely to present anesthesia challenges.

When to Anesthetize

Preferably, anesthesia should be given at each appointment. There is a common belief that instruments may be used in canals with necrotic pulps and periradicular lesions painlessly without anesthesia. Occasionally, there may be vital tissue in the apical few millimeters of the canal.[34] This inflamed tissue contains nerves and is sensitive. Not only is this vital tissue contacted during instrumentation, but also pressure is created. These may cause discomfort if the patient is not anesthetized.

There is an antiquated notion that canal length can be determined in a nonanesthetized patient by passing an instrument into a necrotic canal until the patient shows an “eye-blink response.” Unfortunately, patient perceptions and responses are too variable for accuracy. Pain may be felt when the instrument is far short of the apex, or some patients may have no sensation even when the instrument is several millimeters beyond the apex. Not using anesthesia to aid in length determination cannot replace radiographs or an electronic apex locator for accuracy. Another misconception is that after the canals have been cleaned and shaped, it is not necessary to anesthetize the patient at the obturation appointment. Unfortunately, during obturation, pressure is created and small amounts of sealer may be extruded beyond the apex. This may be quite uncomfortable. Many patients (as well as the dentist) are more at ease if regional hard and soft tissue anesthesia is present.

Adjunctive Pharmacologic Therapy

Sedation (intravenous, oral, or inhalation) may enhance local anesthesia, particularly in patients who want to cooperate but are extremely apprehensive. A discussion on agents that control anxiety is included in Chapter 9.

CONVENTIONAL ANESTHESIA

Success of local anesthesia is variable. Two surveys of patients and dentists indicated inadequate anesthesia was common during restorative treatment.[7,35] Overall, of 10 patients treated, 3 may feel pain during the procedure. This is a significant number! Several factors affect anesthesia, such as the type of procedure (endodontic, extraction, restorative, periodontal, and so on), arch location (maxillary or mandibular), anxiety level, and the presence of inflamed tissue.

Many clinical studies have objectively evaluated local anesthetic agents and techniques. A measurement of pulpal anesthesia before beginning a procedure is obtained with an electric pulp tester or with application of carbon dioxide (dry ice) or spray refrigerant. No pulpal response to the stimuli after administration of anesthetic means probable profound anesthesia in asymptomatic teeth with vital pulps.[36,37] Experimental studies that have investigated the use of local anesthesia are discussed in the following sections. Conventional injection techniques are detailed in other textbooks.

MANDIBULAR ANESTHESIA

Anesthetic Agents

The most commonly used agent is 2% lidocaine with 1 : 100,000 epinephrine. This agent is indicated for procedures in this chapter unless specified otherwise.

Lidocaine is a safe and effective drug.[38] Vasoconstrictors are also generally safe. In a few circumstances (patients taking tricyclic antidepressants or nonselective α-adrenergic blocking agents, or with moderate-to-severe cardiovascular disease), there is the potential for problems with vasoconstrictors.[39]

Related Factors

Although the most common method of mandibular anesthesia is the inferior alveolar nerve block, this injection also has the greatest number of failures.[40,41] The expected signs of successful or unsuccessful anesthesia after administering one cartridge of 2% lidocaine with 1 : 100,000 epinephrine are as follows.

Lip Numbness

Numbness usually occurs in 5 to 7 minutes.[41-47] Lip numbness indicates only that the injection blocked the nerves to the soft tissues of the lip, not necessarily that pulpal anesthesia has been obtained.[41-47] If lip numbness is not obtained, the block has been "missed." If this occurs frequently, the injection technique should be reviewed.

Onset of Pulpal Anesthesia

Pulpal anesthesia usually occurs in 10 to 15 minutes.[41-47] In some patients, onset occurs sooner and in others it is delayed.[41-47]

Duration

Duration of pulpal anesthesia in the mandible is very good.[41-48] Therefore, if successful, anesthesia usually (but not always) persists for approximately 2½ hours.[48]

Success

The incidence of successful mandibular pulpal anesthesia tends to be more frequent in molars and premolars and least frequent in anterior teeth.[41-48] Not all patients achieve pulpal anesthesia after what appears to be a clinically successful (lip and chin numb) inferior alveolar nerve block, thus other approaches are required.

Alternative Techniques

Increasing the Volume

Increasing the volume of anesthetic from one to two cartridges does not increase the success rate of pulpal anesthesia with the inferior alveolar nerve block.[41,43,49]

Increasing the Epinephrine Concentration

There is no improvement in anesthesia with a higher concentration (1 : 50,000) of epinephrine in an inferior alveolar nerve block.[50,51]

Alternative Solutions

Alternative solutions to 2% lidocaine with 1 : 100,000 epinephrine are 2% mepivacaine with 1 : 20,000 levonordefrin, 4% prilocaine with 1 : 200,000 epinephrine, or solutions without vasoconstrictors (3% mepivacaine plain and 4% prilocaine plain) for pulpal anesthesia with effects that last at least 1 hour.[44,47] No solutions have proved to be superior for pulpal anesthesia.

Articaine

Articaine was recently introduced in the United States.[52] Basically, this is a safe and effective local anesthetic agent.[52-60] Articaine has a reputation of providing an improved local anesthetic effect.[61] However, clinical trials have failed to detect any superiority of articaine over lidocaine in block mandibular anesthesia.[55,57,60] Infiltration anesthesia in the maxilla has shown an equivalent effect for articaine and prilocaine/lidocaine[56,58] except for one recent study,[62] which showed a prolonged anesthetic duration with articaine.

Articaine, like prilocaine, has the potential to cause neuropathies.[63] The incidence of paresthesia (which involved the lip and/or tongue) associated with articaine and prilocaine was approximately five times that found with either lidocaine or mepivacaine.[63] The incidence of paresthesia is rare, approximately one in 785,000 injections. Fear of paresthesia should not limit the use of articaine.

Alternative Injection Locations

Neither the Gow-Gates[64] nor the Vizarani-Akinosi technique[65] is superior to the standard inferior alveolar nerve block injection.[42,66-71] These techniques are not replacements for the inferior alveolar nerve block but are useful when standard approaches cannot be used (for example, trismus).

Incisive Nerve Block at the Mental Foramen

The incisive nerve block alone is successful in anesthetizing the premolar teeth[46,72] but not the central and lateral incisors.[46]

Infiltration Injections

Labial or lingual infiltration injections alone are not effective for pulpal anesthesia in the mandible.[56,57,73] A combination of labial and lingual infiltrations is effective for lower anterior teeth.[74] Articaine is significantly better (but not very effective) than lidocaine for buccal infiltration of the mandibular first molar.[75] However, articaine only had a 64% success rate.

Long-Acting Anesthetics

Clinical trials of bupivacaine and etidocaine have been conducted in oral surgery, endodontics, and periodontics.[76-79] These agents provide a prolonged analgesic period and are indicated when postoperative pain is anticipated. However, not all patients want prolonged lip numbness.[77] For those patients, analgesics may be prescribed. Compared with lidocaine, bupivacaine has a somewhat slower onset but almost double the duration of pulpal anesthesia in the mandible (approximately 4 hours).[48]

Accuracy of Needle Placement

Accurate anatomic positioning is no guarantee of a successful block.[80,81] Interestingly, even locating the inferior alveolar nerve with ultrasound before the injection did not improve success.[82] The anesthetic solution may not completely diffuse into the nerve trunk to reach and block all nerves even if deposited at the correct site.[83]

Needle Deflection

Needle deflection has been theorized as a cause for failure with the inferior alveolar nerve block.[84-88] However, two recent studies have shown needle bevel (away or toward the mandibular foramen or ramus) does not affect the success of the inferior alveolar nerve block.[89,90]

Accessory Innervation

Anatomic evidence suggests that accessory innervation exists from branches of the mylohyoid nerve.[91,92] An experimental study using a mylohyoid injection lingual and inferior to the retromolar fossa in addition to an inferior alveolar nerve block showed no enhancement of pulpal anesthesia.[93] The contribution of the mylohyoid nerve to pulpal sensitivity is insignificant.

Cross-Innervation

Cross-innervation from the contralateral inferior alveolar nerve has been implicated in failure to achieve anesthesia in anterior teeth after an inferior alveolar injection. Cross-innervation does occur in incisors but is not the primary cause of anesthetic failure.[94]

Pain and Inflammation

Most studies have evaluated anesthesia in the absence of symptoms and inflammation; results differ if these conditions are present.[4,95] Patients who have symptomatic pulpal or periapical pathosis (and/or who are anxious) present additional anesthesia problems.

MAXILLARY ANESTHESIA

Anesthetic Agents

Unless otherwise specified, the conventional solution used is 2% lidocaine with 1 : 100,000 epinephrine.

Related Factors

Anesthesia is more successful in the maxilla than in the mandible. The most common injection for the maxillary teeth is infiltration. Several events can be expected with this technique when one cartridge of anesthetic is used.

Lip Numbness

Lip numbness usually occurs within a few minutes. Lip or cheek numbness does not correspond entirely to the duration of pulpal anesthesia because the pulp does not remain anesthetized as long as these soft tissues.[96,97]

Success

Infiltration results in a high incidence of successful pulpal anesthesia.[97-100]

Onset of Pulpal Anesthesia

Pulpal anesthesia usually occurs in 3 to 5 minutes.[97-100] Occasionally, onset is slower in first molars.[97-100]

Duration

A problem with maxillary infiltration is duration.[97-100] In about a third of patients, pulpal anesthesia of the anterior teeth declines after about 30 minutes, with most losing anesthesia by 60 minutes.[97-100] In premolars and first molars, about a third of patients have no pulpal anesthesia at 45 minutes, with half losing anesthesia by 60 minutes.[97-100] Frequently, additional local anesthetic must be administered, depending on the duration of the procedure and the tooth group affected.

Alternative Techniques

Volumes of Solution

For maxillary infiltrations, increasing the volume (two cartridges instead of one) increases the duration of pulpal anesthesia.[98] A suggestion for anterior teeth and premolars is to give two cartridges initially, or one initially and inject another approximately 30 minutes later. In first molars, administration of two cartridges initially will speed onset and prolong duration.[98]

Alternative Solutions

In maxillary infiltrations, prilocaine, mepivacaine, articaine, and lidocaine (all with vasoconstrictors) act similarly.[56,99,100] Solutions without vasoconstrictors (3% mepivacaine plain and 4% prilocaine plain) provide a short duration of pulpal anesthesia, averaging 15 to 20 minutes.[99,100]

Long-acting anesthetics do not provide prolonged pulpal anesthesia with a maxillary infiltration (as they do in a mandibular block).[96,97]

Other Techniques

The *posterior superior alveolar (PSA) nerve block* anesthetizes the second and third molars and usually the first molar.[101,102] The evidence is that the middle superior alveolar (MSA) nerve does not supply the first molar.[101,102] An additional mesial infiltration injection may occasionally be necessary to anesthetize the first molar. Generally, the PSA block injection is indicated when all molar teeth require anesthesia. When one tooth is treated, infiltrations are preferred.

The *infraorbital block* results in lip numbness but does not predictably anesthetize incisor pulps.[103,104] It usually anesthetizes the premolars, but duration is less than 1 hour.[103,104] Essentially, the infraorbital injection is similar to infiltration over the premolars.

The *second division block* usually anesthetizes pulps of molars and some second premolars but does not predictably anesthetize anterior tooth pulps.[105] The high tuberosity technique is preferred to the greater palatine approach because it is easier and less painful.[105]

The *palatal anterior superior alveolar (P-ASA) nerve block* has been advocated to anesthetize all the maxillary incisors with a single palatal injection into the incisive canal.[106] The injection technique did not provide predictable pulpal anesthesia for the anterior teeth[107] and was often painful.[27]

The *anterior middle superior alveolar (AMSA) nerve block* has been advocated to unilaterally anesthetize the maxillary central and lateral incisors, canines, and first and second premolars with a single palatal injection in the premolar region.[108] The injection technique did not provide predictable pulpal anesthesia for these maxillary teeth[109] and was often painful.[28]

Pain and Inflammation

Again, results will differ from the normal when anesthesia is given to patients with either or both of these conditions or with anxiety.

ANESTHESIA DIFFICULTIES

What follows is a classic scenario. The diagnosis is irreversible pulpitis. The dentist administers the standard block or infiltration. The patient reports classic signs of anesthesia (lip numbness and a dull feeling of the tooth or quadrant). After isolation, access preparation is begun. When the bur is in enamel, the patient feels nothing. Once the bur enters dentin or possibly not until the pulp is exposed, the patient feels sharp pain. Obviously, pulpal anesthesia is not profound and additional anesthetic is required. There are theories as to why this problem occurs, as follows:

1. The anesthetic solution may not penetrate to the sensory nerves that innervate the pulp, especially in the mandible.

2. The central core theory states that nerves on the outside of the nerve bundle supply molar teeth, whereas nerves on the inside supply anterior teeth (Figure 8-2). The anesthetic solution may not diffuse into the nerve trunk to reach all nerves to produce an adequate block, even if deposited at the correct site. The theory may explain the higher experimental failure rates in anterior teeth with the inferior alveolar nerve block.[41-48]

3. Local tissues change because of inflammation. This popular theory states that the lowered pH of inflamed tissue reduces the amount of the base form of the anesthetic available to penetrate the nerve membrane.[15] Consequently, there is less of the ionized form within the nerve to achieve anesthesia. Although this theory may have some validity for regions with swelling, it is an unlikely cause of anesthesia difficulties in the mandible.[110] It does not explain the major problem, which is the mandibular molar with pulpitis that is not anesthetized by an inferior alveolar injection. The injection site is distant from the area of inflammation; changes in tissue pH would be unrelated to the anesthesia problem.

4. Hyperalgesia of nociceptor (pain receptors) is a more plausible explanation. This theory states that the nerves arising in inflamed tissue have altered resting potentials and decreased excitability thresholds. These changes are not restricted to the inflamed pulp itself but affect the entire neuronal membrane, extending to the central nervous system.[5,6] Local anesthetic agents are not sufficient to prevent impulse transmission, owing to these lowered excitability thresholds.[5]

5. Patients in pain often are apprehensive, which lowers their pain threshold. A vicious cycle may be established in which initial apprehension leads to decreased pain threshold, which leads to anesthesia difficulties, which leads to increased apprehension, which results in loss of control and confidence, and so on. Therefore, if this cycle becomes evident, the practitioner should stop treatment immediately and regain control or schedule another appointment or consider referral to an endodontist. Most patients will endure some pain during the initial stages of root canal treatment if they have confidence in the dentist. However, they will not tolerate being hurt repeatedly!

6. The dentist may not allow sufficient time for the anesthetic to diffuse and to block the sensory nerves. Onset may be very slow, particularly with the inferior alveolar block.

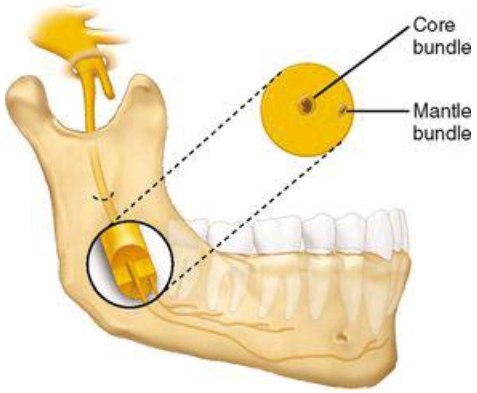

Figure 8-2 Central core theory. The large diameter and density of the bundle may inhibit diffusion of a sufficient quantity of anesthetic to provide profound pulpal anesthesia.

SUPPLEMENTAL ANESTHESIA

Indications

A supplemental injection is used if the standard injection is not effective. It is useful to repeat an initial injection only if the patient is not exhibiting the “classic” signs of soft tissue anesthesia. Generally, if the classic signs are present, reinjection is ineffective. For example, after the inferior alveolar (IA) injection, the patient develops lip, chin, and tongue numbness and quadrant “deadness” of the teeth. However, the patient cannot tolerate pulp or dentin invasion with a bur. To think that reinjection using the IA approach will be successful is wishful thinking; failure the first time is usually followed by failure on the second attempt. The dentist should go directly to a supplemental technique. Three such injections are (1) *the intraosseous* (IO), (2) *the periodontal ligament* (PDL), and (3) *the intrapulpal* (IP). The PDL and IO injections are the preferred approaches, with the IP injection being reserved for special situations.

Anesthetic Agents

With any of the three supplemental techniques, a conventional anesthetic agent is indicated. Therefore 2% lidocaine with 1 : 100,000 epinephrine is used unless an alternative is suggested.

Intraosseous Anesthesia

The IO injection is a supplemental technique that has been shown to be effective through substantial research and clinical usage. It is particularly useful in conjunction with a conventional injection when it is likely that supplemental anesthesia will be necessary, for example, with pulpitis in mandibular posterior teeth.[57,111-115] The IO injection allows placement of a local anesthetic directly into the cancellous bone adjacent to the tooth. There is an IO system with two components (Figure 8-3). One part is a slow-speed handpiece–driven perforator, which drills a small hole through the cortical plate (Figure 8-4). The anesthetic solution is delivered into cancellous bone through a matching 27-gauge ultrashort injector needle (Figure 8-5). Another IO system uses a guide sleeve (Figure 8-6) that remains in the perforation (Figure 8-7). This serves as a guide for the needle and may remain in place throughout the procedure if reinjection is necessary. The perforation may be made in attached gingiva or alveolar mucosa with this system.[114,116] Another innovation is an IO system that combines a slow-speed handpiece with an anesthetic cartridge dispenser system and a rotating needle/drill. No studies have been performed on this system.

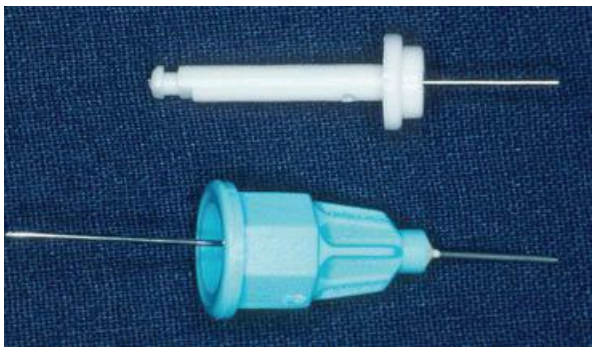

Figure 8-3 Components for intraosseous injection. The perforator *(top)* is a small, sharp, latch-type drill to make an opening through soft tissue and bone. The needle *(bottom)* is short and a small gauge to insert and inject directly through the opening.

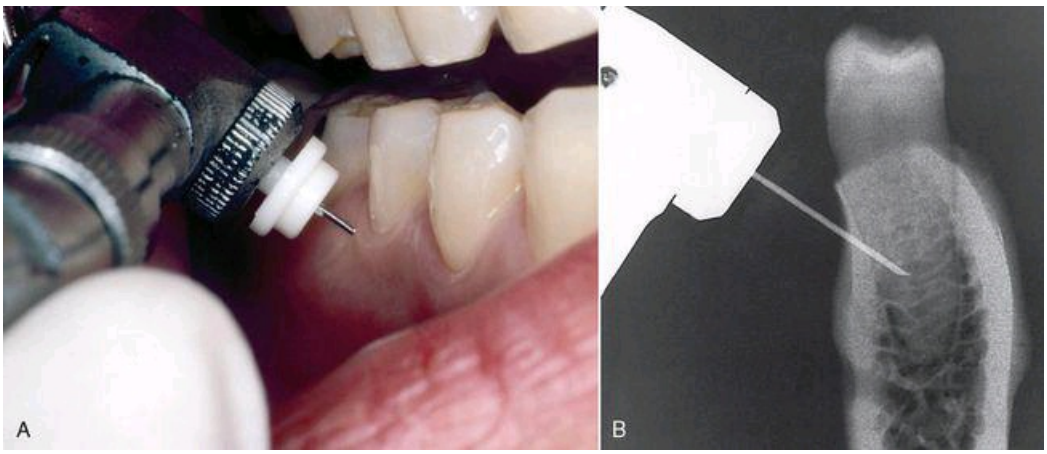

Figure 8-4 Intraosseous injection technique. **A,** Location and angulation of the perforator. **B,** The perforator "breaks through" cortical bone into medullary space.

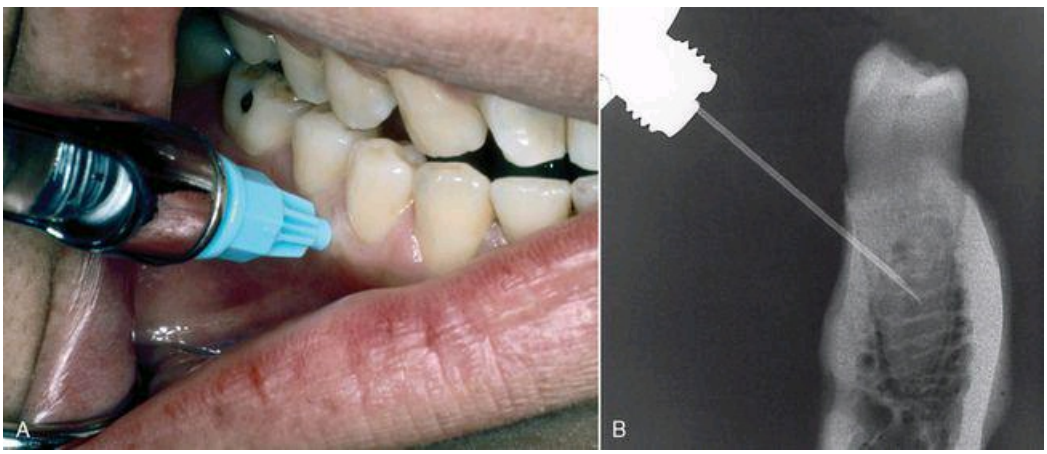

Figure 8-5 A, The needle is inserted directly into the opening. **B,** Anesthetic is injected into medullary bone, where it diffuses widely to block

dental nerves.

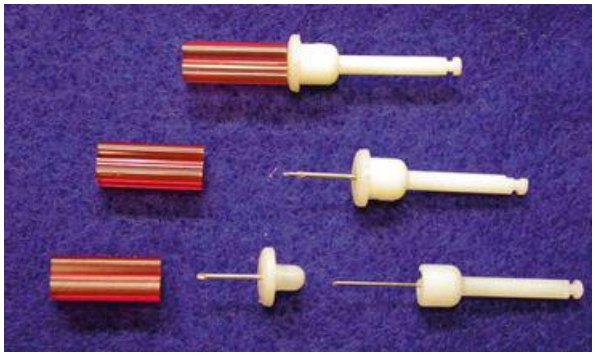

Figure 8-6 Components for another approach to intraosseous injection—the drill and guide sleeve and cover *(top)*. The drill (a special hollow needle) leads the guide sleeve through the cortical plate *(middle)*, in which it is separated and withdrawn *(bottom)*. The remaining guide sleeve is designed to accept a 27-gauge needle to inject the anesthetic solution.

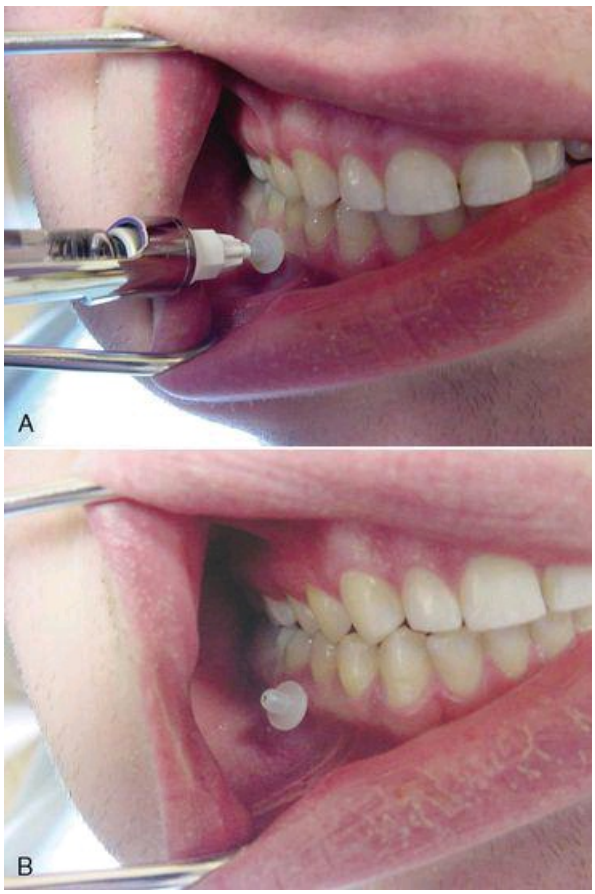

Figure 8-7 A, The tissue and bone have been perforated, and the perforator now serves as a guide sleeve. **B**, The anesthetic needle is in place in the guide sleeve.

Technique

The area of perforation and injection is on a horizontal line of the buccal gingival margins of the adjacent teeth and a vertical line that passes through the interdental papilla distal to the tooth to be injected. A point approximately 2 mm below the intersection of these lines is selected as the perforation site. The soft tissue is first anesthetized by infiltration. The perforator is placed through the gingiva perpendicular to the cortical plate. With the point gently resting against bone, the handpiece is activated at full speed, while pushing the perforator, with light pressure, against bone and then slightly withdrawing the perforator and pushing it again against the bone. This action is continued until a “break through” into the cancellous bone is achieved (taking approximately 2 to 5 seconds).[116]

As the standard syringe is held in a “pen-gripping” fashion, the needle is precisely aligned with and inserted into the

perforation. A full cartridge of anesthetic solution is *slowly* delivered over a 1- to 2-minute time period with light pressure. If back-pressure is encountered, the needle is rotated approximately a quarter turn and deposition is reattempted. If this attempt is unsuccessful, the needle should be removed and checked for blockage. If the needle is not blocked, it is reinserted or the site is opened with a new perforator and the injection is repeated.[116]

Perforator "Breakage"

Rarely, the metal perforator "separates" from the plastic hub. If this occurs, the perforator is easily removed with a hemostat; there are no reports of a perforator breaking into parts.[116-124]

Injection Discomfort

When the IO injection is used as a primary injection, neither perforation, needle insertion, nor solution deposition is painful for most patients.[116,117,125] However, when the IO injection is used as a supplemental injection in a patient with irreversible pulpitis, moderate pain may occur.[112,113,115]

Selection of Perforation Site

Distal perforation and injection to the tooth will result in the best anesthesia.[116-124] An exception would be in second molars where a mesial site is preferred.[116-124] When necessary, a lingual approach may also be successful, although this approach has not been studied.

Anesthetic Agents

For use as a supplemental injection after the IA block in patients without pain, excellent success has been reported for 2% lidocaine with 1 : 100,000 epinephrine, 2% mepivacaine with 1 : 20,000 levonordefrin, and 1.5% etidocaine with 1 : 200,000 epinephrine.[118,121,124] However, because of adverse cardiovascular reactions with the long-acting anesthetics (etidocaine and 0.5% bupivacaine with 1 : 200,000 epinephrine), these agents should not be used.[126] Three percent mepivacaine is successful, but the duration of pulpal anesthesia is shorter.[123]

Onset of Anesthesia

The onset of anesthesia is rapid.[116-124] There is no "waiting period" for anesthesia.

Success

For use as a primary injection, success rates are good[116]; lower success rates are seen with 3% mepivacaine.[119,123] For use as a supplemental injection with irreversible pulpitis, high success rates (around 90%) have been reported.[57,112-115] Three percent mepivacaine has an 80% success rate, which increases to 98% with a second IO injection.[111]

Failure

If the anesthetic solution squirts out of the perforation, there will be failure.[114] Reperforation or choosing another perforation site would then be necessary.

Duration

With a primary IO injection, duration of pulpal anesthesia declines steadily over an hour.[116,119,127] There is an even shorter duration with 3% mepivacaine or 1.5% etidocaine with 1 : 200,000 epinephrine compared with 2% lidocaine with 1 : 100,000 epinephrine.[119,128] With a supplemental IO injection after the IA block in patients without pain, duration of pulpal anesthesia is very good.[118,121] A solution of 3% mepivacaine will result in a shorter anesthetic duration.[123] For irreversible pulpitis, the IO injection should provide anesthesia for the entire debridement appointment.[57,111-115]

Postoperative Problems

With primary and supplemental techniques, the majority of patients report no pain or mild pain.[117-125] Less than 5% will develop exudate and/or localized swellings at the perforation site, possibly from overheating of the bone during perforation.[117-123,125]

Systemic Effects

With both primary and supplemental techniques using anesthetics with a vasoconstrictor (epinephrine or levonordefrin), most patients perceive an increased heart rate.[129-131] When these agents are used, the patient should be informed *before the injection* of this tachycardia to lessen his or her anxiety. No significant heart rate increase occurs with 3% mepivacaine plain.[123,129] The venous plasma levels of lidocaine are the same for an IO injection as for infiltration injection.[130] Therefore the same precautions for the maximum amount of lidocaine given for an infiltration injection would also apply to an IO injection.[130]

Medical Contraindications

Patients with moderate-to-severe cardiovascular disease or those taking tricyclic antidepressants or nonselective α-adrenergic blocking agents should not receive IO injections of solutions containing epinephrine or levonordefrin.[129] Three percent mepivacaine plain is preferred.

Periodontal Ligament Injection

The PDL injection is also a useful technique if a conventional injection is unsuccessful. The PDL (or intraligamental) injection technique has generated considerable research and has spawned the marketing of a variety of special syringes. The technique (regardless of the device used) is clinically effective and is a valuable adjunct.[132-134] It is particularly useful if a rubber dam is in place; the needle may be inserted between the tooth and rubber dam margin.

Technique

The procedure (Figure 8-8) is not difficult but does require practice and familiarity. A standard syringe or pressure syringe is equipped with a 30-gauge ultrashort needle or a 27-or a 25-gauge short needle. The needle is inserted into the mesial gingival sulcus at a 30-degree angle to the long axis of the tooth. The needle is supported by the fingers or hemostat and is positioned with maximum penetration (wedged between root and crestal bone). Heavy pressure is *slowly* applied on the syringe handle for approximately 10 to 20 seconds (conventional syringe), or the trigger is *slowly* squeezed once or twice with resistance (pressure syringe). *Back-pressure is important.* If there is no back-pressure (resistance)—that is, if the anesthetic readily flows out of the sulcus—the needle is repositioned, and the technique repeated until back-pressure is attained. The injection is then repeated on the distal surface. Only a small volume (approximately 0.2 ml) of anesthetic is deposited on each surface.

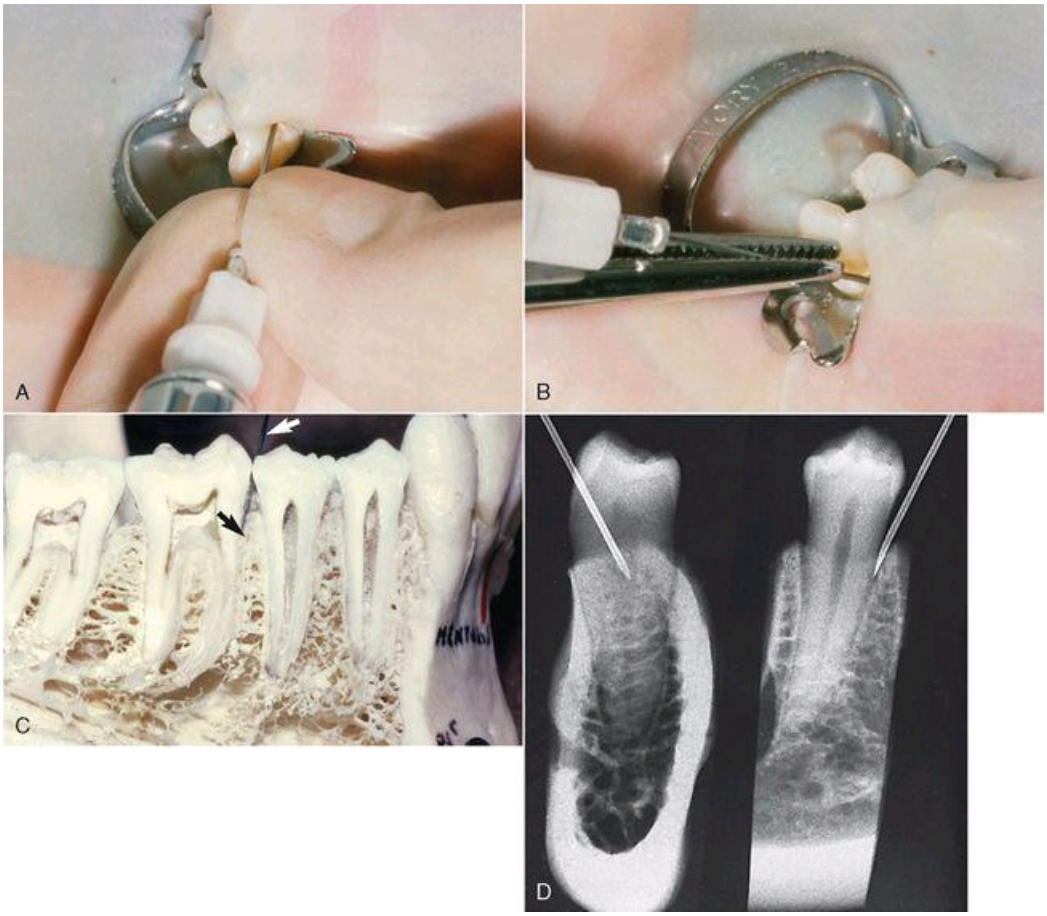

Figure 8-8 Periodontal ligament injection. **A,** Needle insertion using the fingers to prevent needle buckling. **B,** A hemostat may be substituted for fingers to support and direct the needle. The injection may be given with or without the rubber dam in place. **C,** Note the direction and position of the needle *(arrows)*. The tip of the needle will be wedged between the crestal bone and the root surface. **D,** Angle of the needle relative to the long axis of the tooth *(left)*. With approximately a 30-degree orientation, the needle tip will be positioned close to the midline of the root.

Mechanism of Action

The PDL injection forces anesthetic solution through the cribriform plate (Figure 8-9) into the marrow spaces and into the vasculature in and around the tooth (Figures 8-10 and 8-11).[135-137] The primary route is not the PDL; the mechanism of action is not related to direct pressure on the nerves.[138,139]

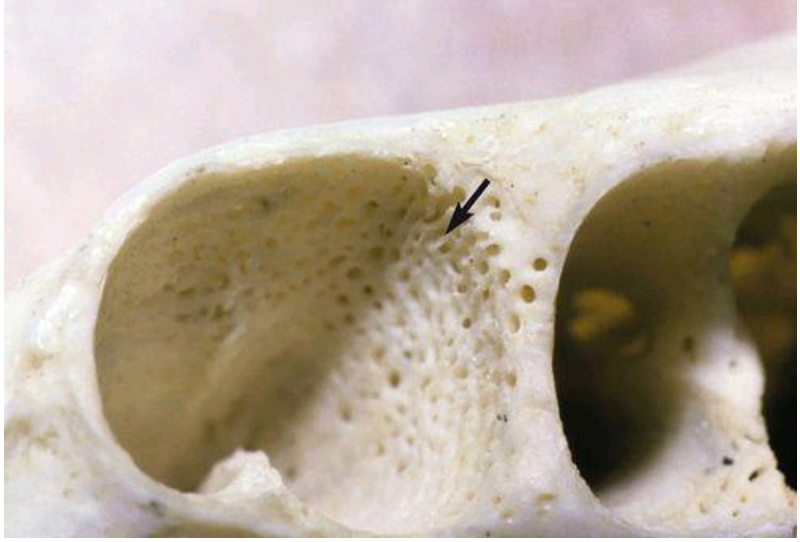

Figure 8-9 An extraction socket of a second molar. The bone of the cribriform plate is very porous, particularly in the cervical region *(arrow)*. During the periodontal ligament injection, this is the region of passage of most anesthetic solution into the medullary space.

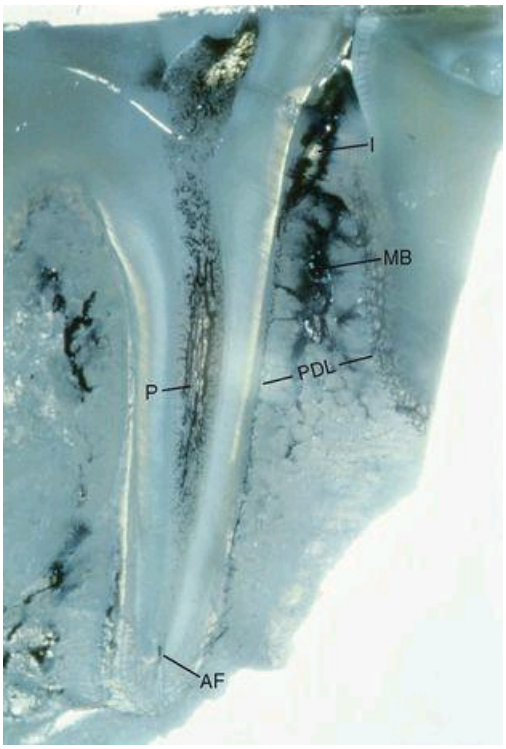

Figure 8-10 A single periodontal ligament injection of carbon dye adjacent to a dog's tooth demonstrates the distribution of dye particles. Particles are concentrated at the injection site *(I)*, as well as in the medullary bone *(MB),* the apical foramen *(AF),* and the pulp *(P)* of the injected tooth. Dye particles have been spread through the periodontal ligament *(PDL)* of both the injected and adjacent teeth.

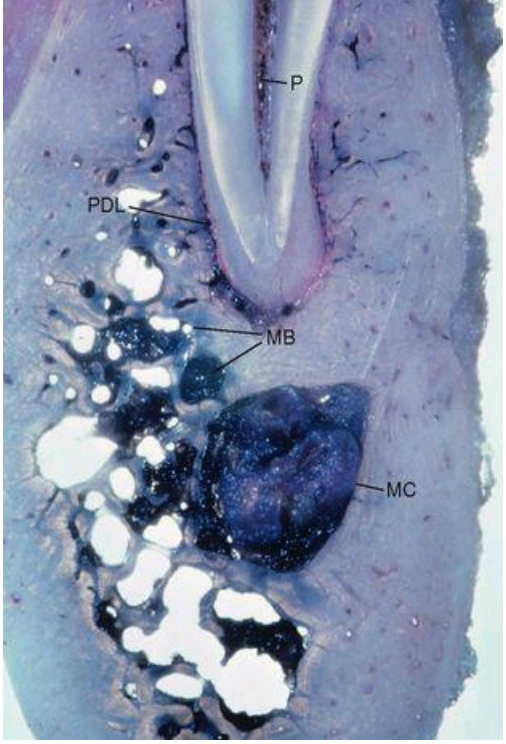

Figure 8-11 A single injection of dye was made in the distal periodontal ligament. This frontal section, including the tooth apex and surrounding structures, shows that dye distributes to the pulp *(P),* periodontal ligament space *(PDL),* medullary bone space *(MB),* and mandibular canal *(MC).* The widespread distribution of solutions from the periodontal ligament injection may anesthetize the adjacent teeth.

Injection Discomfort

When the PDL injection is used as a primary injection, needle insertion and injection are mildly uncomfortable in posterior teeth. In anterior teeth, the PDL injection may be quite painful[140] and patients must be informed of this possibility.

Onset of Anesthesia

Onset of anesthesia is rapid; there is no waiting period to begin the clinical procedure.[132-134,139-141] If anesthesia is still not adequate, reinjection is necessary.

Success

For use as a primary injection, good success rates have been reported for restorative procedures. More difficulty in achieving adequate pain control occurs in extractions and endodontic treatment.[142] Higher success rates have been shown in posterior teeth than in anterior teeth.[140,143] Anesthetic solutions without vasoconstrictors (3% mepivacaine) or with reduced vasoconstrictor concentrations (bupivacaine or etidocaine with 1 : 200,000 epinephrine) are not very effective.[141,144-146] Articaine is equivalent to lidocaine.[59]

For use as a supplemental injection (standard techniques have failed to provide adequate anesthesia), good success rates (83% to 92%) are achieved.[132-134,147] Reinjection increases the success rate.[132,133] The use of a computer-controlled local anesthetic delivery system (see Figure 8-1) for supplemental PDL injections was successful in about half of the patients with irreversible pulpitis.[148]

Duration

The duration of profound pulpal anesthesia (either primary or supplemental) is approximately 10 to 20 minutes.[139-141] The operator must work quickly and be prepared to reinject.

Postoperative Discomfort

When PDL injection is used as a primary technique, postoperative discomfort (mostly mild pain) usually occurs, with a duration

of 14 hours to 3 days.[140,141,146,149,150] There is no difference between articaine and lidocaine.[150] The discomfort is related to damage from needle insertion rather than to the pressure of depositing the solution.[149] Many patients report that their tooth feels "high."[140,141]

Selective Anesthesia

It has been suggested that the PDL injection may be used in the differential diagnosis of poorly localized, painful irreversible pulpitis.[151] However, adjacent teeth are often anesthetized with PDL injection of a single tooth.[139-141] Therefore this injection is *not* useful for differential diagnosis.

Systemic Effects

Although some authors[152] have found that the PDL increases heart rate, human studies have shown that PDL injections do not cause significant changes in heart rate.[150,153]

Other Factors

Different needle gauges (25-, 27-, or 30-gauge) are effective.[132] Special pressure syringes have been marketed (Figure 8-12) but have not proved to be more effective than a standard syringe.[132,133,149]

Figure 8-12 Example of a special syringe used for the periodontal ligament injection. Although these devices are capable of injecting with more pressure, they have not been shown to be superior to the standard syringe.

Safety to the Periodontium

Clinical and animal studies have demonstrated the relative safety of the PDL injection.[139-141,154-159] Minor local damage is limited to the site of needle penetration (Figure 8-13); this subsequently undergoes repair. In rare instances, periodontal infections have occurred.[140,141] Histologic areas of root resorption after PDL injections have also been reported, which also heal with time.[158,159] Damaging effects of injecting into an area of periodontal disease are unlikely.[160]

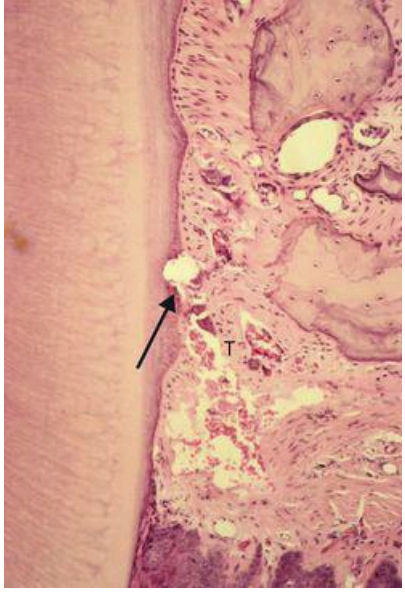

Figure 8-13 The injection site at the time of injection. The needle tract *(T),* which ends in a gouge in cementum *(arrow)*, is apparent in the connective tissue. No tissue changes are evident outside the penetration site, including the more apical tissues.

Safety to the Pulp

Clinical and animal studies have shown no adverse effects on the pulp after PDL injections.[139-141,158,161,162] However, physiologic changes in the pulp do occur, including a rapid and prolonged marked decrease in blood flow caused by epinephrine.[163] This vascular impairment has no demonstrated damaging effect, even in conjunction with restorative procedures.[164] The PDL injection probably would not result in severe pulpal injury, although this has not been studied with extensive (crown) preparations or in teeth with caries.

Safety to Primary Teeth

Minor enamel hypoplasia of succedaneous teeth has been seen after PDL injections in primary teeth.[165] However, this effect was caused by the cytotoxicity of the local anesthetic rather than by the actual injection. Therefore the PDL injection may be used for anesthetizing primary teeth.[138]

Intrapulpal Injection

Indications

On occasion, the IO and PDL injections, even when repeated, do not produce profound anesthesia, and pain persists when the pulp is entered. This is an indication for an IP injection. However, the IP injection should not be used without first administering an IO or PDL injection. The IP injection is very painful without some other form of supplemental anesthesia.

The IP injection may also be useful in injecting into individual canals, when anesthesia is not adequate or "touch-up" anesthesia would be helpful.

Advantages and Disadvantages

Although the IP injection is popular, it has disadvantages, as well as advantages, making it the third supplemental injection of choice. The major drawback is that the needle is inserted directly into a vital and very sensitive pulp; thus the injection may be exquisitely painful. Also, the effects of the injection are unpredictable if it is not given under pressure. Duration of anesthesia once attained is short (5 to 20 minutes). Therefore the bulk of the pulp must be removed quickly and at the correct working length to prevent recurrence of pain during instrumentation. Another disadvantage is that the pulp must be exposed to permit direct injection; often problems with anesthesia occur before pulpal exposure.

The advantage is the predictability of profound anesthesia if the IP injection is given under back-pressure. Onset will be immediate, and no special syringes or needles are required, although different approaches may be necessary to attain the desired back-pressure.

Mechanism of Action

Strong back-pressure has been shown to be the major factor in producing anesthesia.[166,167] Depositing anesthetic passively into the pulp chamber is not adequate; the solution will not diffuse throughout the pulp. Therefore the anesthetic agent is not solely responsible for intrapulpal anesthesia; it also depends on pressure.

Technique

Again, the patient must be informed that a "little extra" anesthetic will ensure comfort and that there will be "a sharp sensation."

One technique creates back-pressure by stoppering the access with a cotton pellet to prevent backflow of anesthetic (Figure 8-14).[167,168] Other stoppers, such as gutta percha, waxes, or pieces of rubber, have been used. If possible, the roof of the pulp chamber should be penetrated by a half-round bur, thus the needle will then fit snugly in the bur hole.

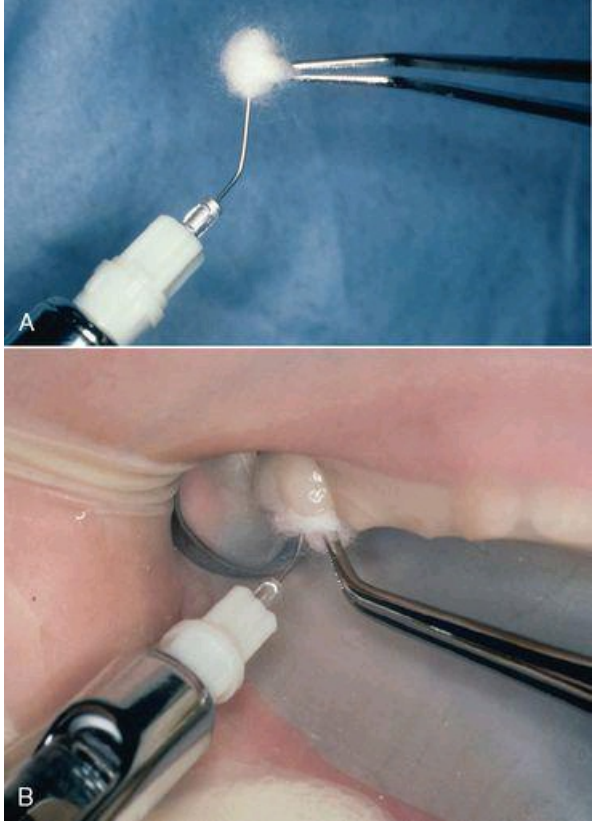

Figure 8-14 Intrapulpal injection technique. **A,** A 45-degree bend is placed on the needle. For stoppering the injection site, a cotton pellet is pulled over the needle and the needle is placed in the opening in the pulp (the patient is forewarned of discomfort!). **B,** The cotton pellet is packed *tightly* and held in the access opening, and the syringe handle is pushed *slowly*. The patient often feels sharp pain with resistance on the syringe handle; this resistance usually indicates successful anesthesia.

Another approach is an injection into each canal after the chamber is unroofed. A standard syringe is usually equipped with a bent short needle. With fingers supporting the needle shaft to prevent buckling, the needle is positioned in the access opening and then moved down the canal, while slowly expressing the anesthetic, to the point of wedging. Maximum pressure is then applied slowly on the syringe handle for 5 to 10 seconds. If there is no back-pressure, anesthetic flows out of the access opening. The needle is then wedged deeper or withdrawn and replaced with a larger gauge needle (or stoppered with a cotton pellet), and the injection is repeated. This may be necessary in each canal.

ANESTHETIC MANAGEMENT OF PULPAL OR PERIRADICULAR PATHOSES

Irreversible Pulpitis

With irreversible pulpitis, the teeth most difficult to anesthetize are the mandibular molars followed by (in order) mandibular and maxillary premolars, maxillary molars, mandibular anterior teeth, and maxillary anterior teeth. The vital inflamed pulp must be invaded and removed. Also, pulpal tissue has a very concentrated sensory nerve supply, particularly in the chamber. These factors, combined with others related to inflammatory effects on sensory nerves and failures occurring with conventional techniques, make anesthetizing patients with painful irreversible pulpitis a challenge.

Different clinical situations present surprises. In some cases, inflamed vital tissue exists only in the apical canals and the tissue in the chamber is necrotic and does not respond to pulp testing. Obviously, in this situation the chamber is entered with no problem, but when the operator attempts to place a file to length, severe pain results. IO or PDL injections are helpful, and an IP injection may be used. However, irreversible pulpitis must be differentiated from a symptomatic apical pathosis because IO, PDL, and IP injections are contraindicated in the latter condition.

General Considerations

Conventional anesthesia using primary techniques is administered. After signs of soft tissue anesthesia occur, the pain abates and the patient relaxes. Frequently, however, on access opening or when the pulp is entered, pain results because not all sensory nerves have been blocked. A useful procedure is to pulp test the tooth with cold or an electric pulp tester before the access is begun.[112,134] If the patient responds, an IO or PDL injection is given. However, no response does not ensure complete anesthesia.[36,112,134] The patient is always informed that the procedure will be immediately discontinued if pain is experienced or if there is a “premonition” of impending pain. Appropriate supplementary injections are then used. Occasionally, all attempts fail, and in this case, it is best to place a temporary restoration and refer the patient to an endodontist.

Mandibular Posterior Teeth

A conventional inferior alveolar injection is administered, usually in conjunction with a long buccal injection. Because of the high failure rate of anesthesia for these teeth, an IO or PDL injection is routinely administered before access is begun.[*] If pain is felt, the IO or PDL injection may be repeated, or an IP injection is given, if the pulp is exposed. Usually, once the pulp is removed, further pain is minimal, owing to the longer duration of mandibular anesthesia.[43,44,48]

Mandibular Anterior Teeth

An inferior alveolar injection is given. If pain is felt, an IO injection is administered. If this is unsuccessful, an IP injection is added.

Maxillary Posterior Teeth

Approaches are the same as those outlined under “General Considerations” *except* that the initial dose is doubled (3.6 ml) for buccal infiltration. The injection site may be a PSA block for molars. Infiltration of 0.5 ml of anesthetic over the palatal apex enhances pulpal anesthesia.[169] If pain is felt during the access, an IO or PDL injection is administered. Seldom is an IP injection needed.

The duration of anesthesia in the maxilla is less than that in the mandible.[97-100,127] Therefore, if pain is experienced during instrumentation, additional primary or supplemental injections are necessary.

Maxillary Anterior Teeth

Anesthetic is administered initially as a labial infiltration and occasionally as a palatal infiltration for the rubber dam retainer. Rarely is an IO injection needed, and the PDL injection also is not very effective.[140] The duration of anesthesia may be less than 1 hour, requiring additional infiltration.[97-100,127]

Symptomatic Pulp Necrosis

This diagnosis indicates pain and/or swelling and therefore periradicular inflammation. Because the pulp is necrotic and apical tissues are inflamed, anesthesia problems are different. These teeth may be painful when manipulated during treatment.

For the mandible, an inferior alveolar nerve block and long buccal injection are administered. For maxillary teeth, if no swelling is present, anesthesia is given with a conventional infiltration or block. If soft tissue swelling is present (cellulitis or abscess), a regional block plus infiltration on either side of the swelling is administered. Access is begun *slowly.* Usually the pulp chamber is entered without discomfort if the tooth is not torqued excessively. File placement and débridement also can be performed without much pain if instruments are used gently.

Occasionally, conventional injections do not provide adequate anesthesia. IO, PDL, or IP injections are *contraindicated*. Although effective for vital pulps, these injections are painful and ineffective with apical pathosis. Rather, the patient should be informed that profound anesthesia is not present owing to inflammation in the bone. As an alternative with maxillary molars, a PSA injection or second division nerve block (high tuberosity injection) may be given. In anterior teeth and premolars, an infraorbital injection is administered to provide some degree of bone and soft tissue anesthesia.

In patients with severe preoperative pain without drainage from the tooth (or when no swelling can be incised), a long-acting anesthetic (such as bupivacaine or etidocaine) may help control postoperative pain in mandibular teeth. However, the duration of analgesia is usually not so long to preclude use of oral analgesics.[48]

Asymptomatic Pulp Necrosis

Asymptomatic teeth are the easiest to anesthetize. Although it may be tempting to proceed without anesthesia, vital sensitive tissue may be encountered in the apical portion of canals.

The conventional injections are usually administered: inferior alveolar nerve block and long buccal injection for mandibular teeth and infiltration (or PSA block) in the maxilla. Usually the patient remains comfortable. Rarely, there may be some sensitivity during canal preparation that requires an IO or PDL injection. IP injection is not indicated because bacteria and debris may be forced periradicularly. In the maxilla an additional infiltration may be necessary during longer procedures.

ANESTHESIA FOR SURGICAL PROCEDURES

Incision for Drainage

Patients tolerate the procedure better when some anesthesia is present before incision and drain placement. However, profound anesthesia is difficult, which should be explained to the patient. In the mandible, inferior alveolar plus long buccal injections (for posterior teeth) or labial infiltration (for anterior teeth) are administered. In the maxilla, infiltration is given at several sites peripheral to the swelling. As an alternative, a PSA or second-division block may suffice for molars and an infraorbital injection for anterior teeth and premolars. For palatal swellings, a small volume of anesthetic is infiltrated over the greater palatine foramen (for posterior teeth) or over the nasopalatine foramen (for anterior teeth). With swelling over either foramen, lateral infiltration is indicated. An adjunctive measure is to spray ethyl chloride on the area just before incision.

Injection directly into a swelling is contraindicated. These inflamed tissues are hyperalgesic and difficult to anesthetize. Traditional beliefs are that the anesthetic solution may be affected by the lower pH and is rendered less effective and that direct injection will “spread the infection,” although neither belief has been proved. Nevertheless, reasons for avoiding injection into a swelling are the pain from the pressure and ineffectiveness. Theoretically, the area of swelling (cellulitis) has an increased blood supply, thus anesthetic is transported quickly into the systemic circulation, diminishing the anesthetic effect. Also, edema and purulence may dilute the solution.

Periradicular Surgery

Additional considerations in periradicular surgery involve anesthesia of both soft tissue and bone. Also, inflammation is usually present. In the mandible the inferior alveolar injection is reasonably effective. Additional infiltration injections in the vestibule are useful to achieve vasoconstriction, particularly in the mandibular anterior region. In the maxilla, infiltration and block injections are generally effective, and larger volumes usually are necessary to provide anesthesia over the surgical field.

If the area of operation is inflamed or the patient is apprehensive, 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 a half when compared to anesthesia for nonsurgical procedures. When reflecting a flap and opening into bone, the anesthetic solution is diluted by bleeding and is removed by irrigation.[170]

As a prophylactic measure, an IO or PDL injection is administered at the site after infiltration and block injections and before surgery. This seems to enhance the depth of anesthesia and may provide better hemostasis.

Use of a long-acting anesthetic has been advocated.[76,77] 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.[79,170] After periradicular surgery, administration of a long-acting anesthetic has been suggested.[15] However, postsurgical pain is usually not severe and can be managed by analgesics.[171]

Chapter Review Questions available in Appendix B or on the DVD

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Endodontic Emergencies and Therapeutics

Richard E. Walton, Karl Keiser

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Identify causes of emergencies as they occur before treatment, between appointments, and after obturation.
2. Recognize what constitutes a true emergency as opposed to urgency.
3. Identify patients who are greater risk for experiencing pain after endodontic procedures.
4. Describe the emotional status of the emergency patient and explain how this complicates diagnosis and treatment.
5. Describe the psychologic and physiologic factors that affect pain perception and pain reaction and how these are managed.

6. Define the flare-up and describe its management.

7. List the factors that relate to greater frequency of interappointment or postobturation flare-ups.

8. Describe and outline a sequential approach to endodontic emergencies:
   a. Determine the source of pain (pulpal or periradicular)
   b. Establish a pulpal and periradicular diagnosis
   c. Identify the etiologic factor of the pathosis
   d. Design an emergency (short-term) treatment plan
   e. Design a long-term treatment plan

9. Outline a system of subjective and objective examinations and radiographic findings to identify the source of pain and the pulpal or periradicular diagnosis.

10. Describe when pretreatment emergencies might occur and how to manage these emergencies.

11. Outline the steps involved in treatment of painful, irreversible pulpitis.

12. Describe the steps involved in treatment of necrotic pulp with acute apical periodontitis.

13. Describe treatment of acute apical abscess and include the indications and procedure for incision and drainage.

14. Describe treatment of acute apical periodontitis after cleaning and shaping (interappointment) or after obturation (posttreatment).

15. Detail pharmacologic supportive therapy (analgesics, anxiolytics, antibiotics, and antiinflammatory agents) used in emergencies and its role in controlling pain and infection.

Endodontic emergencies are a challenge for both diagnosis and management. Knowledge and skill in several aspects of endodontics are required. Failure to apply these will result in serious consequences for the patient. Incorrect diagnosis or incorrect treatment will fail to relieve pain and in fact may aggravate the situation. The clinician must have knowledge of pain mechanisms, patient management, diagnosis, anesthesia, therapeutics, and appropriate treatment measures for both hard and soft tissues.

This chapter discusses approaches to the diagnosis and treatment of various categories of emergencies. It includes a review of etiologic factors and details of a systematic approach to identifying and diagnosing the offending cause. Appropriate treatment, including pharmacotherapy, is described.

Endodontic emergencies are a matter of concern to patients, dentists, and staff. Varying (but not uncommon) frequencies of pain or swelling occur in patients before, during, and after root canal treatment.[1-6] Causes of such emergencies are a combination of irritants that induce severe inflammation in the pulp or periradicular tissues.

Investigations into the role of host factors (age, gender, tooth type, and so on) contributing to the occurrence of endodontic emergencies have been inconclusive; clear cause-and-effect relationships have not been established. Most studies of interappointment pain have shown little or no direct relationship between emergencies and the factors that can be controlled by the operator such as intracanal medication, occlusal reduction, and so on.[1,7] Associated factors are those presented by the patient such as the pulp or periapical diagnosis and the presenting levels of pain. Clearly, higher levels of preoperative pain and the presence of percussion sensitivity are predictive for increased postoperative pain.[8]

The role of bacteria and their by-products in dental emergencies is well established. Bacteria are important causative agents of pathosis of the pulpal and periradicular tissues.[9,10] A mixed flora, including gram-negative anaerobic bacteria, is related to clinical signs and symptoms of periradicular pathosis.[11-13] Bacterial by-products (including collagenase, chondroitinase, hyaluronidase, cysteine and serine proteinases, and lipopolysaccharide) isolated from root canals have also been related to clinical symptoms.[14-16] Receptors for the lipopolysaccharide component of gram-negative bacterial cell walls (endotoxin) have been found on pulpal pain–sensing nerve fibers, or nociceptors. Thus bacteria have the potential to directly cause pulpal and periapical pain.[17]

Irritation of pulp and periradicular tissues results in inflammation and the release of a group of chemical mediators that initiate inflammation. The concentrations of some of these substances in the pulp and periradicular tissues are significantly higher in symptomatic than in asymptomatic lesions.[18,19] Pain results from two factors related to inflammation: (1) *chemical mediators* and (2) *pressure.* Chemical mediators can cause pain directly by activating nociceptors, causing spontaneous pain, or by lowering their response threshold, which causes, for example, a heightened response to normally non–pain-producing thermal stimuli. Inflammatory mediators may also cause pain indirectly by increasing vascular permeability and producing edema. Edema results in increased fluid pressure, which mechanically stimulates pain receptors. Reducing the concentration of chemical mediators and allowing release of pressure from the root canal system and from the periapical region are both effective treatment measures.

DEFINITION

By definition, endodontic emergencies are usually associated with pain and/or swelling and require immediate diagnosis and treatment. These emergencies are caused by pathoses in the pulp or periradicular tissues. Emergencies also include severe traumatic injuries that result in luxation, avulsion, or fractures of the hard tissues. Management of emergencies related to trauma is not included in this chapter (see Chapter 10).

CATEGORIES

Pretreatment Emergency

A pretreatment emergency is a situation in which the patient is seen initially with severe pain and swelling. Problems occur with both diagnosis and treatment.

Interappointment and Postobturation Emergency

The interappointment and postobturation emergency, also referred to as the “flare-up,” occurs after an endodontic appointment. Although this is an upsetting event, it is easier to manage because the offending tooth has already been identified and a diagnosis has been previously established. Also, the clinician has knowledge of the prior procedure and will be better able to correct the problem.

THE CHALLENGE

It is satisfying and rewarding to successfully manage a distraught patient who has an emergency (Figure 9-1). In contrast, it is distressing to have a patient with a flare-up after root canal treatment in a previously asymptomatic tooth. The aim is to increase occurrences in the first category and decrease those in the second.

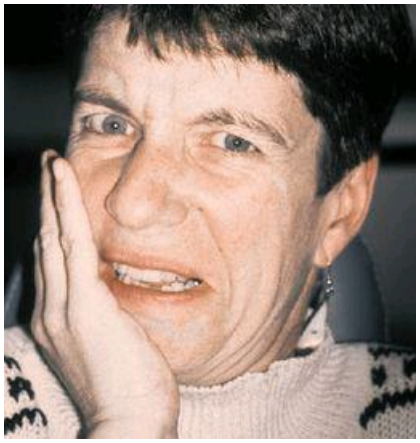

Figure 9-1 Patient is distraught from severe pain of irreversible pulpitis. This patient will be a challenge to diagnose and treat.

Differentiation of Emergency and Urgency

Whether a pretreatment, interappointment, or postobturation problem, it is important to differentiate between a *true emergency* and the less critical *urgency.* A *true emergency* is a condition requiring an unscheduled office visit with diagnosis and treatment. The visit cannot be rescheduled because of the severity of the problem. *Urgency* indicates a less severe problem, thus a visit may be scheduled for mutual convenience of the patient and the dentist. Key questions for the patient (that may be asked by telephone) to determine severity include the following:

1. Does the problem disturb your sleeping, eating, working, concentrating, or other daily activities? (A true emergency disrupts the patient's activities or quality of life.)

2. How long has this problem been bothering you? (A true emergency has rarely been severe for more than a few hours to 2 days.)

3. Have you taken any pain medication? Was the medication ineffective? (Analgesics do not relieve the pain of a true emergency.)

An affirmative answer to these questions requires an immediate office visit for management and constitutes a true emergency. Obviously, the patient's emotional and mental status must also be determined. To some patients, even a minor problem has major proportions and is disruptive.

Development of a System

Because a misdiagnosis will probably result in improper treatment and an exacerbation of the problem, a systematic approach is mandatory. The emotional status of the patient, pressures of time, and stress on dentist and staff should not affect an orderly approach.

PAIN PERCEPTION AND PAIN REACTION

Pain is a complex physiologic and psychologic phenomenon. Pain perception levels are not constant; pain thresholds, as well as reactions to pain, change significantly under various circumstances.[20] Psychologic components of pain perception and pain reaction comprise cognitive, emotional, and symbolic factors. The pain reaction threshold is significantly altered by past experiences and by present anxiety levels and emotional status.[21] (Chapter 5 includes additional details on pain perception and reaction and pain threshold.)

To reduce anxiety and consequently obtain reliable information about the chief complaint and to receive cooperation during treatment, the clinician should: (1) establish and maintain control of the situation, (2) gain the confidence of the patient, (3) provide attention and sympathy, and (4) treat the patient as an important individual.[22] Providing positive written information about pain control during root canal therapy can also reduce the fear associated with an emergency endodontic procedure.[23] By managing these pain components, pain perception and reaction thresholds are raised significantly, greatly facilitating the procedure. Psychologic management of the patient is the most important factor in emergency treatment.

Adjunctive pharmacotherapy may also be required in the management of patient anxiety during emergency treatment. Reducing anxiety at this stage will not only reduce the response to potentially painful stimuli during treatment but also will decrease the tendency of the patient to recall the endodontic procedure as unpleasant.[21] Mild anxiety may be managed with nitrous oxide.[24] However, the apparatus may be a bit cumbersome while making treatment radiographs. Oral benzodiazepines can be very effective in managing more significant anxiety. Triazolam has a fast onset and a relatively short half-life and because of its lipophilic nature can be given sublingually for rapid absorption.[25] Therefore triazolam is quite convenient for sedation in the dental office. One quarter of a milligram (0.25 mg) of oral triazolam has been shown to be as effective as intravenous valium.[26] Of course, patients who have taken or are given an oral sedative in the dental office must have transportation provided. The potential drug interactions with other centrally acting agents must also be considered.

PHYSICAL CONDITION

In addition to the emotional factors that complicate the diagnosis of endodontic emergencies, physical conditions induced as a result of these situations also contribute to the problems. Pain or swelling may limit mouth opening, thereby hampering diagnostic procedures and treatment (Figure 9-2). In addition, hypersensitivity to thermal stimuli or pressure influences diagnosis and treatment. These significant impairments to diagnosis and treatment should make the patient a consideration for referral.

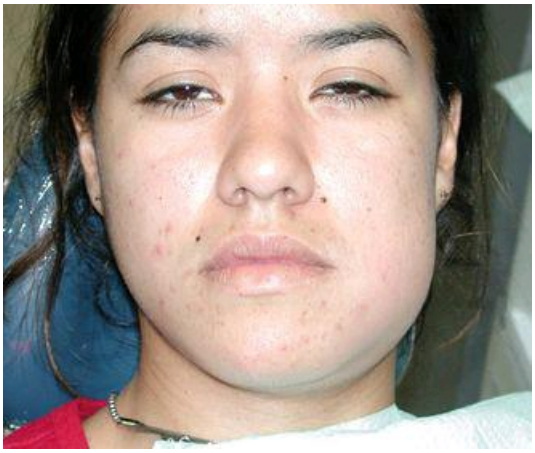

Figure 9-2 Severe mandibular swelling. This patient has limited mouth opening resulting from trismus.

SYSTEM OF DIAGNOSIS

Patients in pain often provide information and responses that are exaggerated and inaccurate. They tend to be confused and apprehensive. It is easy (and tempting) to rush through the diagnosis to institute treatment for a suffering patient. After pertinent information regarding the medical and dental histories is obtained, both subjective questioning and objective examination are performed carefully and completely[27,28] (Box 9-1).

Box 9-1 Diagnosis Sequence

1. Obtain pertinent information about the patient's medical and dental histories.
2. Ask pointed subjective questions about the patient's pain: history, location, severity, duration, character, and eliciting stimuli.
3. Perform an extraoral examination.
4. Perform an intraoral examination.
5. Perform pulp testing as appropriate.
6. Use palpation and percussion sensitivity tests to determine periapical status.
7. Interpret appropriate radiographs.
8. Identify the offending tooth and tissue (pulp or periapex).
9. Establish a pulpal and periapical diagnosis.
10. Design a treatment plan (both emergency and definitive).

A rule of the true emergency is *one tooth is the offender*, that is, the source of pain. In the excitement of the moment, the patient might believe that the severe pain is emanating from more than a single tooth. The clinician may become convinced also, which can lead to overtreatment.

Medical and Dental Histories

Medical and dental histories should be reviewed first. If the patient is the dentist's own, the medical history is briefly reviewed and updated. If the patient is new, a standard, complete history is taken. An important medical complication may be easily overlooked in an emergency situation. Either a short or a complete dental history is taken. This includes recalling dental procedures, the chronology of symptoms, or an earlier relevant comment by a dentist.

Subjective Examination

When the patient is in pain, the subjective examination includes careful questioning and is the most important aspect of diagnosis. Questions relate to the history, location, severity, duration, character, and eliciting stimuli of pain. Questions relating to the cause or stimulus that elicits or relieves the pain help select appropriate objective tests to arrive at a final diagnosis.

Pain that is elicited by thermal stimuli or pain that is referred is likely to originate from the pulp. Pain that occurs on mastication or tooth contact and is well localized is probably apical.

The three important factors constituting the quality and quantity of pain are its *spontaneity, intensity,* and *duration* (see Chapter 5). If the patient reports any of these symptoms (and assuming that the patient is not exaggerating), significant pathosis is likely to be present. Careful questioning provides important information about the source of the pain and whether it is pulpal or periradicular. In fact, a perceptive, clever clinician should be able to arrive at a tentative diagnosis by means of a thorough subjective examination; objective tests and radiographic findings are then used for confirmation. For example, a reported complaint of severe, continuous (lingering) pain when the patient drinks cold beverages and a marked tenderness on mastication indicate irreversible pulpitis and symptomatic apical periodontitis. These stimuli are then repeated in an objective examination to confirm the patient's response.

Again, it is important in identifying the offending tooth to repeat tests that mimic what the patient reports subjectively. In other words, the best test is to repeat the stimulus that reportedly causes the pain. This is especially true for pulpal disease that has not extended to the periradicular tissues (e.g., irreversible pulpitis without symptomatic apical periodontitis). It is often difficult for the patient to localize the pain to a particular tooth because of the paucity of proprioceptive neurons in the dental pulp. As in the previous example, applying cold and pressure should reproduce pain of basically the same type and magnitude as related by the patient. If similar subjective symptoms are not reproduced, this may not be a true emergency, the patient may be "overreporting" (exaggerating the problem), or the pain may be referred from a source other than that perceived by the patient.

In addition, objective tests include extraoral and intraoral examination such as observation for swelling and mirror and explorer examination to note the presence of defective restorations, discolored crowns, recurrent caries, and fractures.

Periradicular tests include: (1) palpation over the apex, (2) digital pressure on or wiggling of teeth (preferred if the patient reports severe pain on mastication), (3) light percussion with the end of the mirror handle, and (4) selective biting on an object such as a cotton swab or Tooth Slooth.

Pulp vitality tests are most useful to reproduce reported pain. Cold, heat, electricity, and direct dentin stimulation also indicate the pulp status (vital or necrotic).

Periodontal Examination

A periodontal examination is always necessary. Probing helps in differentiating endodontic from periodontal disease. For example, a periodontal abscess can simulate an acute apical abscess (Figure 9-3). However, with a localized periodontal abscess, the pulp is usually vital (see Chapter 6). In contrast, an acute apical abscess is related to an unresponsive (necrotic) pulp. These abscesses occasionally communicate with the sulcus and have a deep probing defect. In addition, when the differential diagnosis is difficult, a test cavity may identify the pulp status and isolate the offending tooth. A narrow-walled, isolated probing defect may also indicate a coronal fracture that has extended beyond the level of sulcular attachment or a vertical root fracture (see Chapter 7).

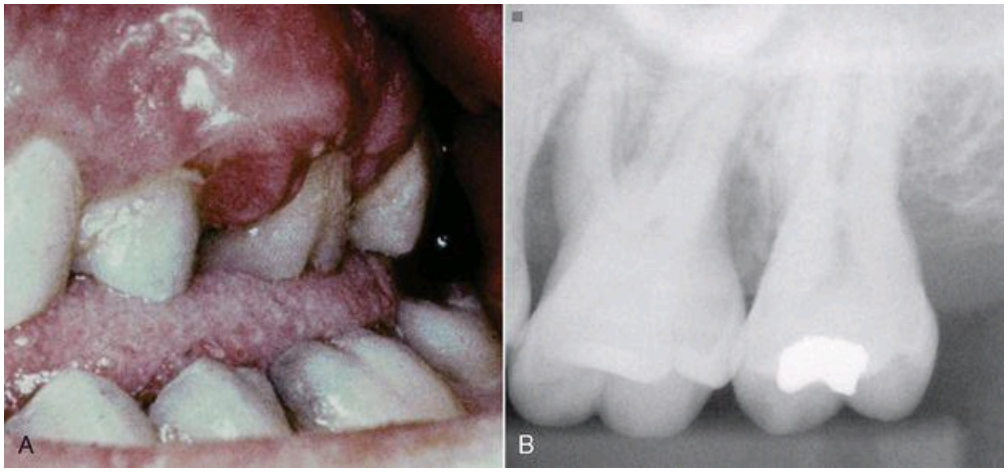

Figure 9-3 A, Periodontal abscess. **B,** Radiographic appearance. Positive responses to pulp testing differentiate this condition from an acute apical abscess.

Radiographic Examination

Radiographs are helpful but have limitations. There is a tendency to rely too much on radiographs, often with unfortunate consequences. Periapical and bitewing radiography may detect the presence of interproximal and recurrent caries, possible pulpal exposure, internal or external resorption, and periradicular pathosis, among other entities, but it is important to understand that diagnosis should not be based on radiographs alone.

Diagnostic Outcome

After carefully working through the previously described sequence, the clinician should have identified the offending tooth and the tissue (pulpal or periradicular) that is the source of pain and should have recorded a pulpal or periapical diagnosis. For many reasons, all or none of these conclusions may be clear. This may not be a true emergency or the problem may be beyond the capability of the generalist and the patient should be referred (Figure 9-4). If the diagnosis is clear, however, treatment planning follows.

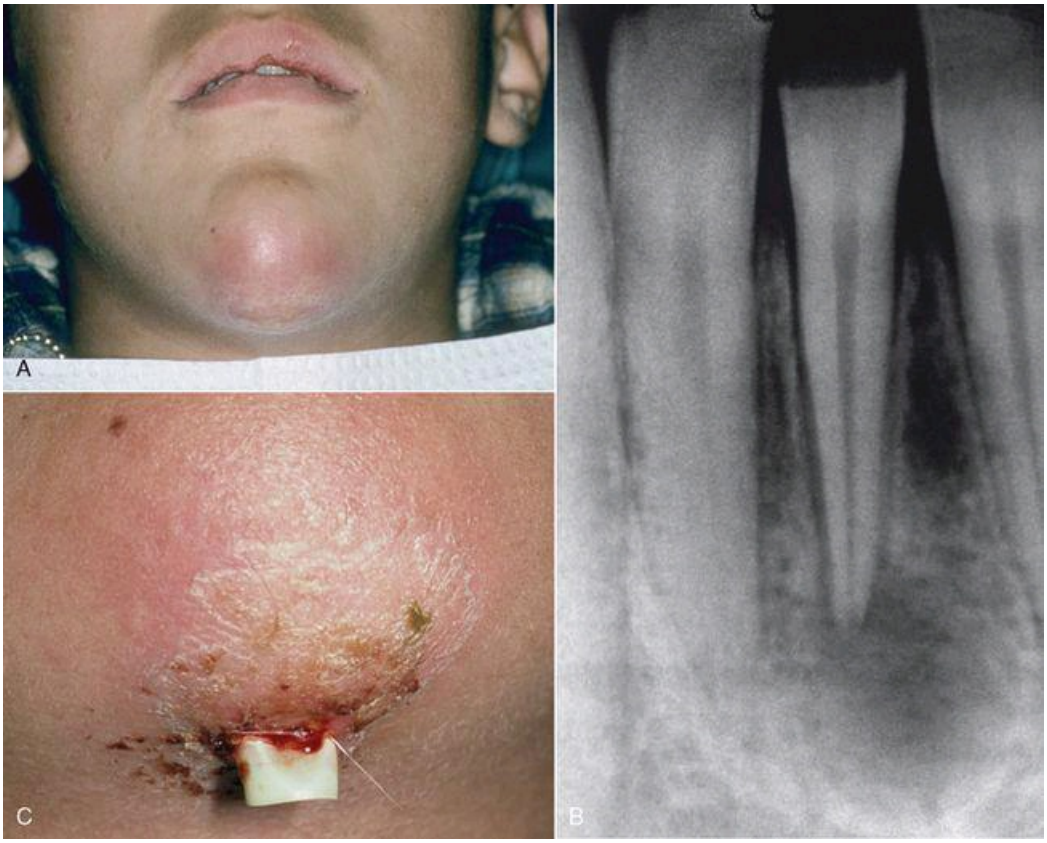

Figure 9-4 This patient has a complex problem and should be considered for referral. **A** and **B**, Submental space swelling resulting from trauma followed by acute apical abscess. **C**, Removal of the necrotic debris from the pulp space was followed by extraoral incision and placement of a drain.

TREATMENT PLANNING

As previously discussed, inflammation and its consequences (i.e., increased tissue pressure and release of chemical mediators in the pulp or periradicular tissues) are the major causes of painful dental emergencies.[29] Therefore reducing the irritant, by releasing pressure or removing the inflamed pulp or periradicular tissue, should be the immediate goal, which usually results in pain relief. Of these, pressure release is the most effective.

PRETREATMENT EMERGENCIES

Pretreatment emergencies require a diagnosis and treatment sequencing. Each of the following steps is important: (1) categorizing the problem, (2) taking a medical history, (3) identifying the source, (4) making the diagnosis, (5) planning the treatment, and (6) treating the patient.

Patient Management

Patient management is always the most critical factor. The frightened patient who is in pain must have confidence that his or her problem is being properly managed.

Profound Anesthesia

Obtaining profound anesthesia of inflamed painful tissue is a challenge. Adequate anesthesia, however, will instill confidence and cooperation and influence the patient's desire to save the offending tooth. Maxillary anesthesia is usually obtained by infiltration or block injections in the buccal and palatal regions. With mandibular teeth, in addition to an inferior alveolar and lingual nerve block, a long buccal injection for soft tissue anesthesia may be helpful. Often (particularly with mandibular molars), access into the dentin or pulp is painful, although all "classic" signs of profound anesthesia are present (such as lip numbness). This pain is presumably a result of sensitized pulpal nociceptors. It is therefore prudent to retest the tooth with a cold stimulus to assess pulpal anesthesia before initiating access. For those cases that still respond after traditional inferior alveolar nerve blocks, periodontal, intrapulpal, or intraosseous injection techniques are indicated.[30] These supplemental injections are often given prophylactically, particularly with painful irreversible pulpitis.[31] Other conditions (e.g., acute apical abscess) require other approaches (see Chapter 9).

Management of Painful Irreversible Pulpitis

Because the pain is the result of inflammation, primarily in the coronal pulp, removal of the inflamed tissue will usually reduce the pain.

Without Symptomatic Apical Periodontitis

Complete cleaning and shaping of the root canals is the preferred treatment, if time permits. With limited time, most pulpal tissue is extirpated with a broach (partial pulpectomy) in single-rooted teeth. In molars, a partial pulpectomy is performed on the largest canals (palatal or distal root). Also, pulpotomy is usually effective in molars when minimal time is available[32,33] (Figure 9-5).

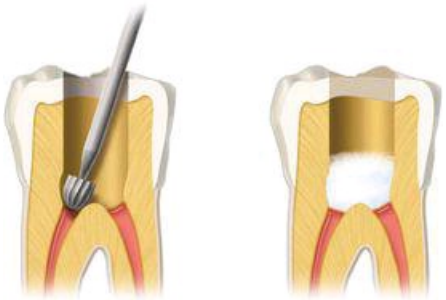

Figure 9-5 Removal of coronal pulp. Pulpotomy and placement of a dry cotton pellet and a temporary filling result in relief of pain from irreversible pulpitis.

An old but still popular idea is that chemical medicaments sealed in chambers help control or prevent additional pain; however, this is not true. A dry cotton pellet alone is as effective in relieving pain as a pellet moistened with camphorated monochlorophenol (CMCP), formocresol, Cresatin, eugenol, or saline.[32] Therefore, after irrigation of the chamber or canals with sodium hypochlorite, a dry cotton pellet is placed and the access is sealed temporarily.

A mild analgesic, such as 400 mg of ibuprofen or 650 to 1000 mg of acetaminophen, is usually sufficient, as the majority of pain relief is provided by the removal of the inflamed tissues[34] (Figure 9-6). *The cause must be removed to effect a cure*.

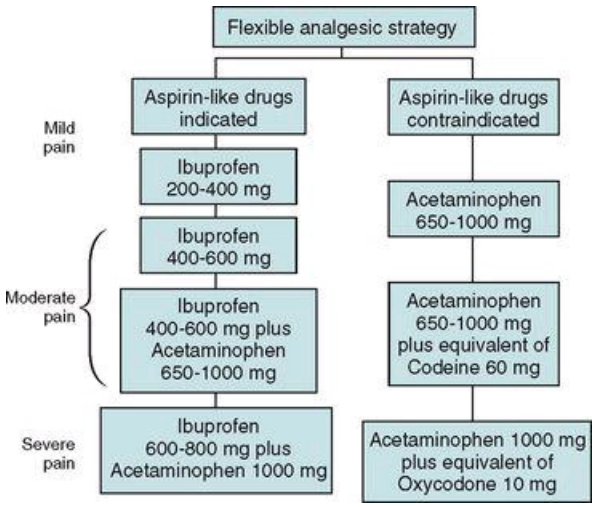

Figure 9-6 Simplified analgesic strategy to guide drug selection based upon patient history and level of present or anticipated posttreatment pain.

The irreversibly inflamed pulp is still vital and immunocompetent, with the ability to resist bacterial infection. Antibiotics, therefore, are definitely not indicated in cases of irreversible pulpitis.[35] Unfortunately, old habits die hard, and some practitioners still prescribe antibiotics inappropriately, as in the case of irreversible pulpitis.[36] This has the potential to result in at least two very serious sequelae: adverse reaction to the antibiotic and increasing antibiotic-resistant microbial strains. The former is a local problem in which injudicious use of an antibiotic could lead to a life-threatening situation for an allergic patient. The latter is a global problem. Resistant microbial strains are emerging faster than pharmaceutical companies are developing new antibiotics, thus it is critical that healthcare providers practice good stewardship with existing antibiotics.[37] Otherwise, in the near future, clinicians may not have a pharmacologic option in treating severe odontogenic infections.

In patients with extreme tenderness on percussion, a partial or total pulpectomy (as previously described) is appropriate. Reducing the occlusion to eliminate contact has been shown to aid in relief of symptoms.[38] Trephination (artificial fistulation) by creating an opening through mucosa and bone is not useful and is contraindicated.[39,40]

Patients who initially present with percussion sensitivity are more likely to experience significant postoperative pain. Therefore a slightly more aggressive pharmacologic may be indicated[8,41,42] (see Figure 9-6). Nonsteroidal antiinflammatory drugs (NSAIDs) are still the primary drugs of choice, given that the pain is of inflammatory origin. For those that can tolerate them, traditional nonselective cyclooxygenase (COX) inhibitors, such as ibuprofen, are as effective as the more selective (and more expensive) COX-2 inhibitors in the control of postoperative dental pain.[43] The patient takes 600 to 800 mg of ibuprofen every 6 hours for the immediate postoperative period. To prevent the build-up of the arachidonic acid metabolites that contribute a large portion of the inflammatory pain stimulus, it is important that the patient take the first dose before the loss of local anesthesia and then take the NSAID "by the clock," rather than "as needed" (PRN). Administering NSAIDs to the patient while in the chair has been shown to reduce initial postoperative pain.[42]

If more severe pain occurs, the patient may take 650 to 1000 mg of acetaminophen in between doses of the ibuprofen. The combination of an NSAID and acetaminophen provides synergistic analgesic effects.[44,45] For the patient that cannot tolerate NSAIDs, a preparation containing acetaminophen and a narcotic may be required. If codeine is selected as the narcotic component, a 60 mg dosage is indicated; 30 mg of codeine typically performs in a manner comparable to placebo in analgesic efficacy trials. Another option is to prescribe an acetaminophen/hydrocodone preparation that contains 500 mg of acetaminophen and 5 mg of hydrocodone per tablet; the patient is instructed to take 2 tablets every 6 hours, thereby receiving the maximum dose of both the acetaminophen and hydrocodone. In patients for whom the sedative effect of hydrocodone is undesirable, 50 to 100 mg of tramadol may be taken every 4 to 6 hours.[46] Box 9-2 provides useful web sites for continually updated drug information, including dosing regimens and drug-drug interactions.

Box 9-2 Useful Web Sites for Continually Updated Drug Information, Including Dosing Regimens and Drug-Drug Interactions

www.RxList.com

www.thomsonhc.com/hcs/librarian

www.epocrates.com

www.fda.gov/

Management of Pulp Necrosis with Apical Pathosis

The pain of pulp necrosis is related to periradicular inflammation, which results from potent irritants in the necrotic tissue in the pulp space. Treatment now is biphasic: (1) remove or reduce the pulp irritants and (2) relieve the apical fluid pressure (when possible). The diagnosis may be symptomatic apical periodontitis (no significant periradicular resorption) or acute apical abscess with or without swelling.

Therefore with pain and pulp necrosis there may be: (1) no swelling, (2) localized swelling, or (3) diffuse, more extensive swelling (cellulitis). Each condition is managed differently. Diffuse swelling is the least common of these three conditions.

Pulp Necrosis without Swelling

In pulp necrosis without swelling, the teeth may contain vital inflamed tissues in the apical canal and have inflamed painful periradicular tissues (symptomatic apical periodontitis).[47] Profound local anesthesia may be a problem, requiring a supplemental injection.

Alternatively, the lesion may have expanded and formed an abscess that is confined to bone. These are often painful, primarily because of fluid pressure in a noncompliant environment. The aim is to reduce the canal irritants and to try to encourage some drainage through the tooth.

After determining the corrected working length, complete canal débridement is the treatment of choice. If time is limited, partial débridement at the estimated working length is performed with light instrumentation with a passive step-back or crown-down technique to remove irritating debris. Canals are not enlarged without knowledge of the working length. During cleaning, canals are flooded and flushed with copious amounts of sodium hypochlorite. Finally, canals are irrigated with the same solution, dried with paper points, filled with calcium hydroxide paste (if the preparation is large enough), and sealed with a dry cotton pellet and a temporary filling.

Some clinicians empirically place a cotton pellet lightly dampened with intracanal chemical medication in the pulp chamber before placing a temporary filling. There is no value to these medicaments. Administering a long-acting anesthetic, reassuring the patient, removing (or reducing) the irritant, and prescribing an analgesic will usually reduce postoperative pain significantly. Patients with pulp necrosis and apical periodontitis without swelling are often in moderate to severe pain because the inflammatory exudate is confined to the cancellous space between cortical plates. For such patients, a maximum dose of an NSAID, such as 800 mg of ibuprofen, may be combined with the maximum dose of acetaminophen (1000 mg). Also, it may be necessary to add a narcotic component to the acetaminophen (see Figure 9-6). For those patients in whom NSAIDs are contraindicated, acetaminophen 1000 mg may be combined with 10 mg of oxycodone.[28] Antibiotics are not indicated.[48] The patient is told that there will still be some pain (the inflamed, sensitive periradicular tissues are still present) and that the pain should subside during the next 2 or 3 days, as the inflammation decreases.

Pulp Necrosis with Localized Swelling

The abscess has now invaded regional soft tissues and at times, there is purulence in the canal. Radiographic findings range from no periapical change (seldom) to a large radiolucency.

Again, treatment is biphasic. *First and most important* is débridement (complete cleaning and shaping if time permits) of the canal or canals. *Second in importance* is drainage. Localized swelling (whether fluctuant or nonfluctuant) should be incised (Figure 9-7). Drainage accomplishes two things: (1) relief of pressure and pain and (2) removal of a very potent irritant—purulence.

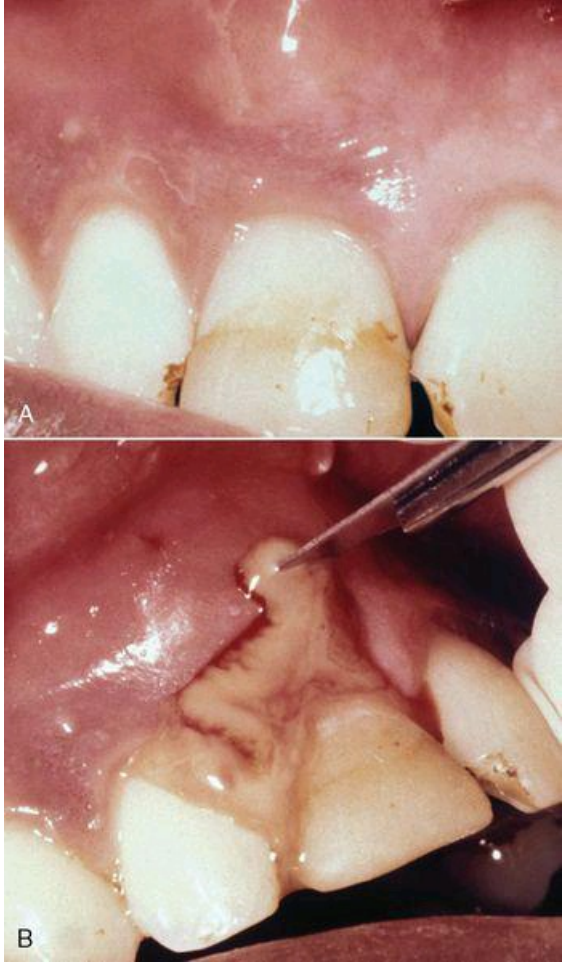

Figure 9-7 A, Localized swelling. **B**, Incision for drainage after cleaning and shaping offending incisor.

(Courtesy Dr. E. Rivera.)

In teeth that drain readily after opening, instrumentation should be confined to the root canal system (Figure 9-8). In patients with a periradicular abscess but no drainage through the canal, penetration of the apical foramen with small files (up to 25) may initiate drainage and release of pressure. This release often does not occur because the abscess cavity does not communicate directly with the apical foramen. Occasionally there may be more than one abscess (Figure 9-9). One communicates with the apex while another separate abscess is found in the vestibule. Because they do not communicate, drainage must occur through both the tooth and a mucosal incision. Often, a drain is placed to permit continued drainage for 1 or 2 days or until débridement is complete. Several designs of drain can be used (Figure 9-10).

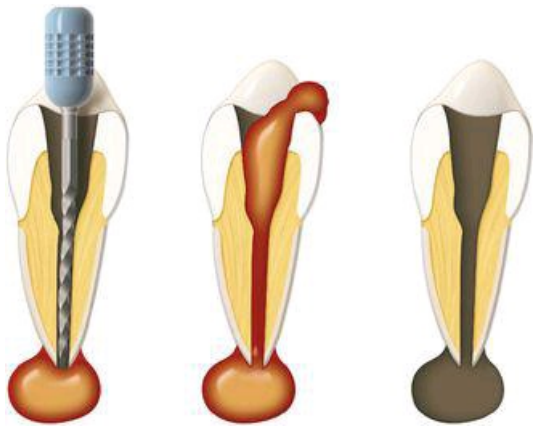

Figure 9-8 After opening into the root canal and establishment of drainage, instrumentation should be confined to the root canal system. Release of purulence removes a potent irritant (pus) and relieves pressure.

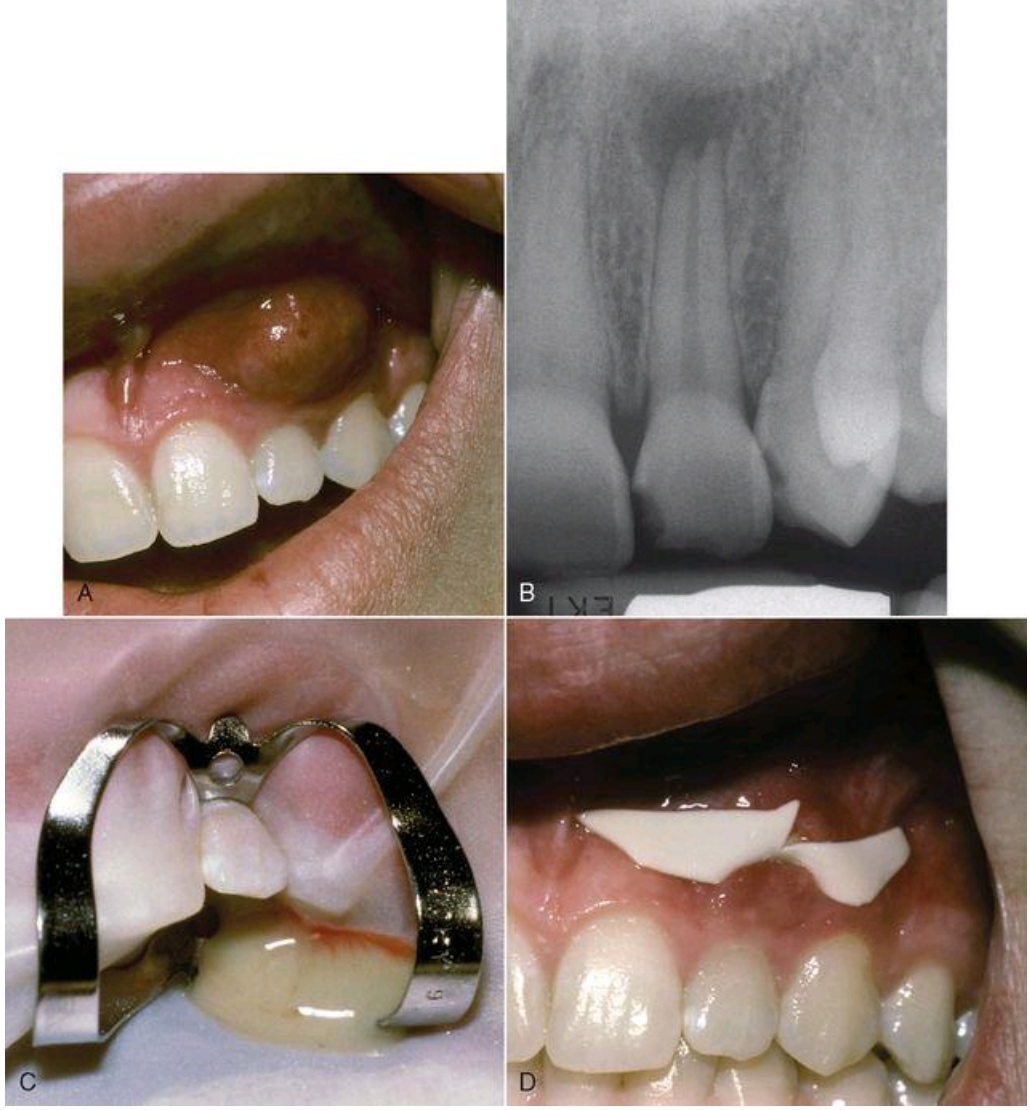

Figure 9-9 **A**, Localized fluctuant abscess as a result of periradicular pathosis after trauma. **B**, Radiographic appearance. **C**, Drainage was spontaneous when the tooth was opened. **D**, Christmas tree–shaped rubber drain placed after soft tissue incision. Antibiotics are unnecessary, but analgesics are indicated.

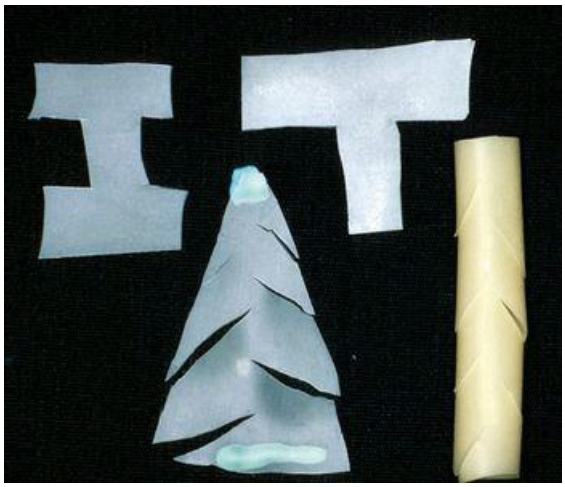

Figure 9-10 Different shapes of rubber drains. From left to right: I drain, Christmas tree drain, T drain, and Penrose drain with oblique cuts. These designs are self-retentive and do not require suturing to the incision margins.

Copious irrigation with sodium hypochlorite is performed throughout instrumentation to reduce amounts of necrotic tissue and bacteria. The canals are then dried with paper points and filled with calcium hydroxide paste. After placement of a dry cotton pellet, the access is sealed temporarily. These teeth should not be left open to drain, although leaving teeth open has been a common procedure. A canal exposed to the oral cavity is a potential home for introduced bacteria, food debris, and even viruses.[49] Occasionally, purulence will continue to fill the canal during the preparation (the so-called weeping canal). If

this occurs, the patient should sit for a time. Usually, the flow will cease and the access may be closed.

These patients seldom have elevated temperatures or other systemic signs. Therefore, in acute apical abscess with localized swelling, the use of systemic antibiotics is not necessary, having been shown to be of no benefit.[48,50,51] An analgesic regimen appropriate for mild-moderate pain should be followed (see Figure 9-6). Relief of pressure is most important in pain control for these patients.

Pulp Necrosis with Diffuse Swelling

Rapidly progressive and spreading swellings, commonly referred to as *cellulitis,* are not localized and may have dissected into the fascial spaces (Figure 9-11). Fortunately, these serious infections seldom occur. Occasionally, there is elevated temperature or other systemic signs indicating a more serious infection. These patients should be referred to a specialist.

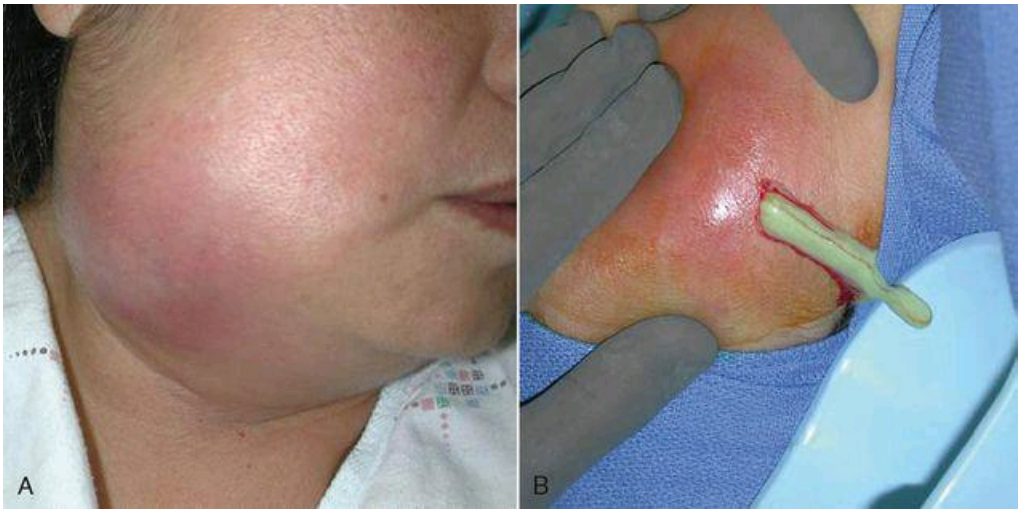

Figure 9-11 A, An acute apical abscess from a mandibular molar has rapidly spread into the buccal and submandibular spaces and is localizing extraorally and subcutaneously, requiring extraoral incision for drainage **(B)**.

Most important is removal of the irritant by canal débridement (cleaning and shaping is completed, if possible) or by extraction. The apical foramen may be gently penetrated with a file to hopefully permit a flow of exudate (see Figure 9-9, *C*), although drainage often does not occur. At this time, swelling may be incised and a rubber drain inserted for 1 or 2 days. Occasionally, the abscess may spread to multiple spaces. This requires hospitalization and aggressive treatment, including intravenous antibiotics, incision, and placement of several drains (Figure 9-12).

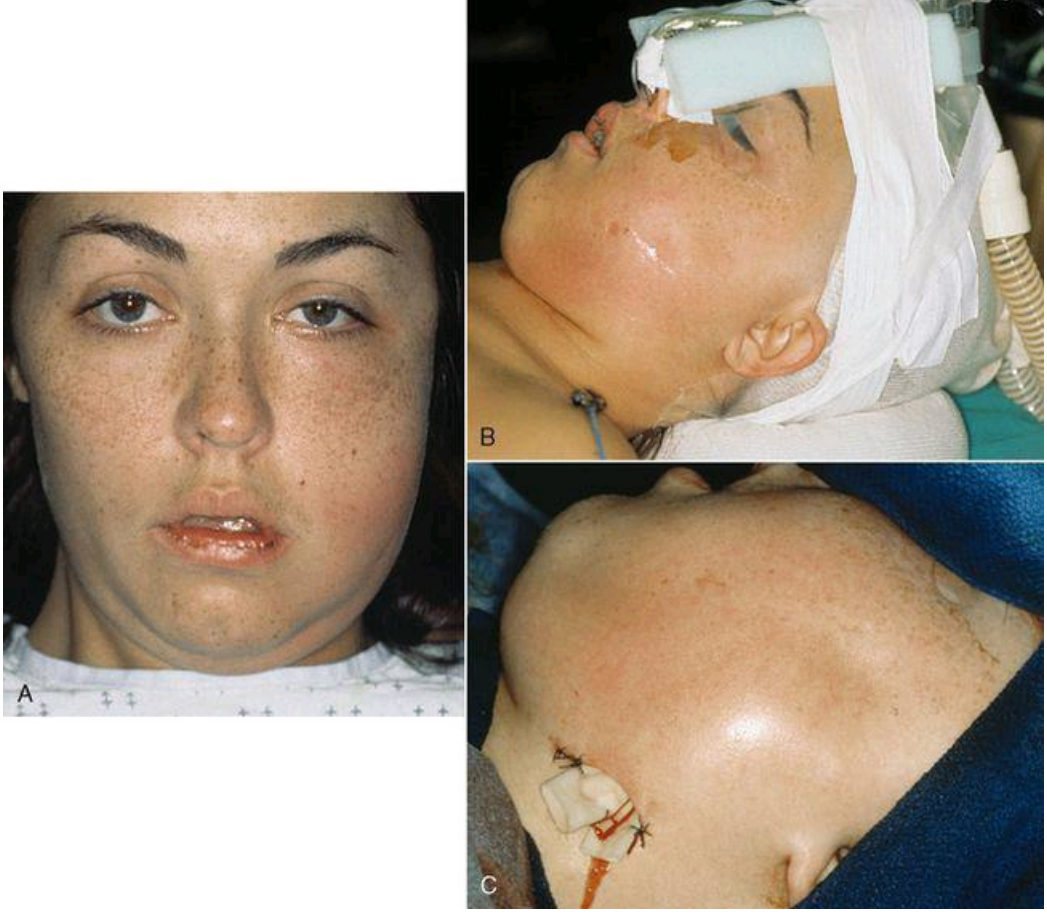

Figure 9-12 Progressively spreading swelling from a flare-up resulting in an acute apical abscess from a mandibular second molar. **A**, The swelling has extended to the infratemporal, submandibular, pharyngeal, and sublingual spaces. This condition compromised the patient's airway, requiring hospitalization for aggressive therapy that included nasal intubation **(B)** and placement of extraoral drains to different involved spaces **(C).** These severe infections are best managed by an oral surgeon.

After placing calcium hydroxide paste and a dry pellet, the access is closed with a temporary filling. Systemic antibiotics are indicated for the diffuse, rapidly spreading swelling. Culturing and molecular identification techniques utilized to study odontogenic abscesses reveal a polymicrobial infection, with anaerobic species predominating.[12,13,52] Antibiotic sensitivity testing confirms that the majority of isolates are susceptible to penicillin VK, which remains a good, inexpensive first choice for those patients who are not allergic.[12,13] Initial therapy should begin with a loading dose of 1000 mg, followed by 500 mg every 6 hours for 7 days. For patients whose symptoms do not improve after adequate local therapy and penicillin VK, 500 mg of metronidazole may be added to the existing penicillin regimen. Another option is to switch to clindamycin for α-lactamase–producing microorganisms.[53]

For patients allergic to penicillin, clindamycin is an alternative,[13] given as a 300 mg loading dose, followed by 150 to 300 mg every 6 hours for 7 days. Because of the reduction in normal gut flora, patients on oral antibiotics occasionally develop signs of colitis as a result of the overgrowth of *Clostridium difficile*. This can occur with nearly all classes of antibiotics, and patients should be warned to watch for the development of watery diarrhea, cramping abdominal pain, and low-grade fever. Patients taking oral contraceptives for whom antibiotics are prescribed should also be warned to use alternative methods during and for 1 week after the course of antibiotic therapy.

Systemic steroids may help to reduce the inflammatory pain; however, they require 24 to 48 hours to be of benefit.[54] Analgesics for moderate to severe pain should be prescribed as was outlined previously.

Speed of recovery (whether the swelling is localized or diffuse) depends primarily on canal débridement and drainage. Because edema (fluid) has spread through the tissues, diffuse swelling decreases slowly, over a period of perhaps 3 to 4 days.

Postoperative Instructions

Patients must be informed of their responsibilities and of what to expect. The pain and swelling will take time to resolve, proper nutrition and adequate intake of fluids are important, and medications must be taken as prescribed. The problem may recur or worsen (flare-up), requiring another emergency visit.[1] Communication after the visit is very important. Calling the patient the day after the appointment has been shown to reduce pain perception and analgesic needs[55] and allows the clinician the opportunity of monitoring the progress of the patient.

INTERAPPOINTMENT EMERGENCIES

The interappointment *flare-up* is a true emergency and is so severe that an unscheduled patient visit and treatment are required. Despite judicious and careful treatment procedures, complications, such as pain, swelling, or both, may occur. Regional temporary paresthesia has even been reported.[56] As with emergencies occurring before root canal therapy, these interappointment emergencies are undesirable and disruptive events and should be resolved quickly. Occasionally, flare-ups are unexpected, although they can often be better predicted according to certain patient presenting factors.

Incidence

The reported incidence of flare-ups in endodontics varies widely because of variations in study parameters.[57] Properly controlled prospective studies show an overall incidence of 1.8% to 3.2%.[1,58,59] Even though the overall occurrence is low, interappointment flare-ups represent such a stressful situation to the patient (most postoperative discomfort is in the mild to moderate range[57]) that it behooves the prudent clinician to consider the likely related factors and try to prepare the patient for the possibility of such an event. It is especially distressing for the patient who had minimal preoperative discomfort to experience pain and swelling after treatment.

Causative Factors

It is difficult to assess causality when reviewing the literature on flare-ups; however, certain risk factors have emerged. These factors generally can be categorized as related to the patient (including pulpal and periapical diagnosis) or to treatment procedures. Patient factors include gender (more flare-ups are reported to occur in females; however, this may represent a greater tendency for females to seek medical care for painful symptoms[60]) and preoperative diagnosis. Flare-ups are quite uncommon in teeth with vital pulps.[1,61] More often, flare-ups occur in teeth with necrotic pulps and especially in those with a periapical diagnosis of symptomatic apical periodontitis or acute apical abscess.[1,7,59,61] The presence of a periradicular radiolucency has also been shown to be a risk factor.[1,59,62,63] However, it is clear that the patient who experiences a flare-up is more likely to have presented with significant preoperative pain and/or swelling.[57]

Treatment factors have also been examined for the potential to create flare-ups. Although it would seem intuitive that certain procedures, such as overinstrumentation, pushing debris out the apex, or completing the endodontic therapy in one visit, may increase the incidence of flare-ups, no definitive treatment risk factors have been identified.[57]

Prevention

Procedures

Use of long-acting anesthetic solutions, complete cleaning and shaping of the root canal system (possibly), analgesics, and psychologic preparation of patients (particularly those with preoperative pain) will decrease interappointment symptoms in the mild to moderate levels.[64] There are, however, no demonstrated treatment or therapeutic measures that will reduce the number of interappointment flare-ups. In other words, no particular relationship of flare-ups to actual treatment procedures has been shown.[1,7]

Verbal Instructions

Most important is the preparation of patients for what to expect after the appointment. They should be told that discomfort ("soreness") is likely and that the discomfort should subside within a day or two. Increases in pain, noticeable swelling, or other adverse signs necessitate a call and sometimes a visit. This explanation reduces the number of calls from unnecessarily concerned patients.

Therapeutic Prophylaxis

A popular preventive approach has been the prescribing of antibiotics in an attempt to minimize postoperative symptoms. This approach has been demonstrated to be not useful and needlessly exposes the patient to expensive, potentially dangerous drugs, as previously described.[65-67] In contrast, certain NSAIDs have been shown to reduce postendodontic treatment pain.[41,42] For the patient at risk for flare-up, 600 to 800 mg of ibuprofen should be given while the patient is in the chair and then taken by the clock for the first 24 to 48 hours postoperatively. Although this will reduce postoperative symptoms, it is uncertain whether this will reduce the incidence of flare-ups.

Diagnosis

The same basic procedure is followed as outlined earlier in this chapter for pretreatment emergencies, although with modifications. The problem has been diagnosed initially, so the operator has an advantage. However, a step-by-step approach to diagnose the existing condition reduces confusion and error; most importantly, it calms a patient who has been shaken by the episode of pain or swelling. After the underlying complications are identified, treatment is initiated.

Treatment of Flare-ups

Reassurance (the "Big R") *is the most important aspect of treatment*. The patient is generally frightened and upset and may even assume that extraction is necessary. The explanation is that the flare-up is neither unusual nor irrevocable and will be managed. Next in importance is restoring the patient's comfort and breaking the pain cycle. For extended anesthesia and analgesia, administration of bupivacaine hydrochloride is recommended.[68]

Interappointment emergencies are divided into patients with an initial diagnosis of a vital or a necrotic pulp and with or without swelling.

Previously Vital Pulps with Complete Débridement

If complete removal of the inflamed vital pulp tissues was accomplished at the first visit, this situation is unlikely to be a true flare-up, and patient reassurance and the prescription of a mild to moderate analgesic (see Figure 9-6) often will suffice. Generally, nothing is to be gained by opening these teeth; the pain will usually regress spontaneously, but it is important to check that the temporary restoration is not in traumatic occlusion. Placing corticosteroids in the canal or giving an intraoral or intramuscular injection of these medications after cleaning and shaping reduces inflammation and somewhat lowers the level of moderate pain.[69-72] Flare-ups, however, have not been shown to be prevented by steroids, whether administered intracanal[62] or systemically.

Previously Vital Pulps with Incomplete Débridement

It is likely that tissue remnants have become inflamed and are now a major irritant. The working length should be rechecked, and the canal(s) should be carefully cleaned with copious irrigation of sodium hypochlorite. A dry cotton pellet is then placed, followed by a temporary filling, and a mild to moderate analgesic is prescribed (see Figure 9-6).

Occasionally, a previously vital pulp (with or without complete débridement) will develop into an acute apical abscess. This will occur some time after the appointment and indicates that pulpal remnants have become necrotic and are invaded by bacteria.

Previously Necrotic Pulps with No Swelling

Occasionally, these teeth develop an acute apical abscess (flare-up) after the appointment.[1] The abscess is confined to bone and can be very painful.

The tooth is opened and the canal is gently recleaned and irrigated with sodium hypochlorite. Drainage should be established if possible (see Figure 9-8). If there is active drainage from the tooth after opening, the canal should be recleaned (or débridement completed) and irrigated with sodium hypochlorite. The rubber dam is left in place after the tooth is opened; the patient is allowed to rest pain-free for at least 30 minutes or until drainage stops. Then, the canals are dried, calcium hydroxide paste is placed, and the access is sealed. The tooth should not be left open. If there is no drainage, the tooth should also be lightly instrumented, gently irrigated, medicated with calcium hydroxide paste, and then closed. The symptoms usually subside but do so more slowly than if drainage was present. Again, patient education and reassurance are critical. A long-acting anesthetic and an analgesic regimen for moderate to severe pain are helpful; antibiotics are not indicated.[48,50]

Previously Necrotic Pulps with Swelling

Previously necrotic pulps with swelling are best managed with incision and drainage (see Figure 9-7). In addition, it is most important that the canals have been débrided. If not, they should be opened and débrided, medicated with calcium hydroxide paste, and closed. Then incision and drainage with placement of a drain are completed. Occasionally but rarely, a flare-up or a presenting acute apical abscess may be serious (cellulitis) or even life-threatening (see Figure 9-12). These situations may require hospitalization and aggressive therapy with the cooperation of an oral surgeon.

Follow-up Care

With flare-ups, the patient should be contacted daily until the symptoms abate. Communication may be made by telephone. Patients with more serious problems or those that are not resolving (many do not and require additional measures) should return to the dentist for treatment as previously described, depending on findings. When symptoms recur or cannot be controlled, these patients should be considered for referral. Ultimate treatment by a specialist may include extra measures, such as apical surgery, or even hospitalization.

POSTOBTURATION EMERGENCIES

True emergencies postobturation are infrequent, although pain at the mild level is common. Therefore active intervention is seldom necessary. Symptoms usually will resolve spontaneously.

Causative Factors

Little is known about the etiologic factors involved in postoperative pain after obturation. Reports of the incidence of postobturation pain vary; however, most show that the pain tends to occur in the first 24 hours.[73,74] There is a correlation between the level of obturation and pain incidence, with overextension associated with the highest incidence of discomfort.[73,75] Postobturation pain also relates to preobturation pain. Levels of pain reported after obturation tend to correlate to levels of pain before the appointment.[73,74]

Information about possible discomfort for the first few days (especially in patients who had higher levels of preoperative pain), reassurance about the availability of emergency services, and administration of analgesics for mild pain (see Figure 9-6) significantly control the patient's anxiety and prevent overreaction. This in turn decreases the incidence of postobturation frantic telephone calls or "emergency" visits. Some patients, however, do develop serious complications and require follow-up treatment.

Retreatment is indicated when prior treatment has been obviously inadequate. Apical surgery is often required when an acute apical abscess develops, and there is uncorrectable, inadequate root canal treatment. If root canal treatment was acceptable, incision and drainage of swelling after obturation (an occasional occurrence) should be performed. Swelling usually resolves without further treatment. At times, the patient reports severe pain, although there is no evidence of acute apical abscess and the root canal treatment has been well done. These patients are treated with reassurance and appropriate analgesics (see Figure 9-6). Again, the symptoms usually subside spontaneously.

Patients with postobturation emergencies that do not respond to therapy should be referred to an endodontist for other treatment modalities such as surgery.

Chapter Review Questions available in Appendix B or on the DVD

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Management of Traumatic Dental Injuries

Leif K. Bakland, Marie Therese Flores

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe the clinical and radiographic features of the following: enamel fractures, crown fractures without pulp exposure, crown fractures with pulp exposure, crown-root fractures, root fractures, tooth luxations (concussion, subluxation, lateral luxation, extrusive luxation, intrusive luxation), avulsions, and alveolar fractures.

2. Describe possible short- and long-term responses of pulp, periradicular tissues, and hard tissues to the injuries listed above.

3. List pertinent information needed when examining patients with dental injuries (from health history, nature of injury, and symptoms).

4. Describe the diagnostic tests and procedures used in examining patients with dental injuries and interpret the findings.

5. Describe appropriate treatment strategies (immediate and long-term) for various types of traumatic injuries.

6. Recognize outcomes of traumatic dental injuries.

7. Recognize pulp space obliteration and describe management considerations.

8. Recognize surface resorption, inflammatory (infection-related) resorption, and replacement (ankylosis-related) resorption, and describe their respective treatment strategies.

9. Describe the differences in treatment strategies for traumatic dental injuries in primary and permanent dentition.

Trauma to teeth involves the dental pulp either directly or indirectly. Consequently, endodontic considerations are important in evaluating and treating dental injuries. The purpose of this chapter is to describe examination procedures, emergency care, treatment options, and possible sequelae in traumatized teeth. Because injuries can also occur to primary teeth, a separate section has been included for these teeth. The recommendations for managing traumatic dental injuries are based on the guidelines published by the International Association of Dental Traumatology, which publishes updated guidelines on its web site: www.iadt-dentaltrauma.org.

Age is an important factor in trauma to teeth. By age 14, about 25% of children will have had an injury involving their permanent teeth.[1,2] The significance of age is of a “good news/bad news” nature. The good news is that pulps in children’s teeth have a better blood supply than those in adults and therefore better repair potential. The bad news is that root development will be interrupted in teeth with damaged pulps, leaving the roots thin and weak. Cervical fractures often occur either spontaneously or from even minor injuries because of thin dentin walls (Figure 10-1). Therefore, when dental injuries occur in children, every effort must be made to preserve pulp vitality.

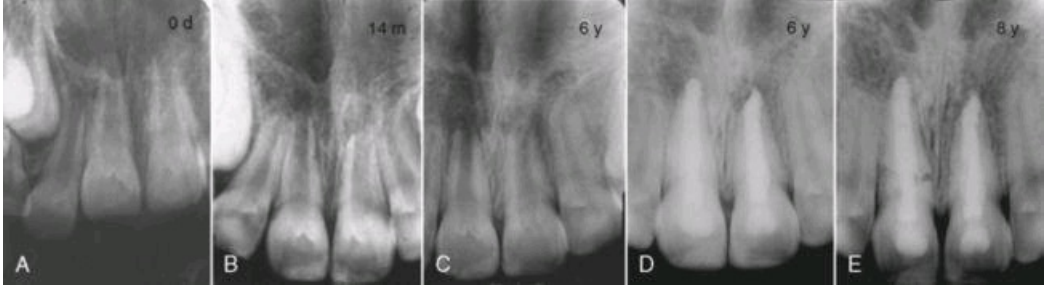

Figure 10-1 Trauma to immature incisors resulting in pulp necrosis leads to interruption of tooth development, leaving the roots with thin, weak walls. Root canal treatment can be performed, but the teeth will be weak and prone to fracture. **A,** Both maxillary-central incisors were intruded in a 7-year-old girl. The teeth were left for spontaneous reposition. **B,** The teeth have spontaneously repositioned at 14-month follow-up control. **C,** The radiograph reveals apical radiolucency and interruption of root formation in both incisors at 6-year follow-up control, indicating pulp necrosis. **D,** Root canal treatment was performed. MTA was used in the apical region and gutta-percha in the rest of the root canal. **E,** After 8 years, spontaneous root fracture at the cervical level occurred in the right central incisor.

Classification of traumatic injuries promotes better communication and dissemination of information. The system used in this chapter is based on Andreasen's modification of the World Health Organization's classification[1,3] (Box 10-1). It is preferable to other classification systems because it is internationally accepted and has a descriptive format based on anatomic and therapeutic considerations.

Box 10-1 Classification of Dental Injuries

Enamel fracture: Involves the enamel only and includes enamel chipping and incomplete fractures or enamel cracks.

Crown fracture without pulp exposure: An uncomplicated fracture involving enamel and dentin with no pulp exposure.

Crown fracture with pulp exposure: A complicated fracture involving enamel and dentin and exposure of the pulp.

Crown-root fracture: Tooth fracture that includes enamel, dentin, and root cementum and may or may not include the pulp.

Root fracture: Fracture of root only involving cementum, dentin, and pulp and also referred to as horizontal root fracture.

Luxation injuries: Tooth luxations include concussion, subluxation, extrusive luxation, lateral luxation, and intrusive luxation.

Avulsion: Complete displacement of a tooth out of its socket.

Fracture of the alveolar process (mandible or maxilla): Fracture or comminution of the alveolar socket or of the alveolar process.

EXAMINATION AND DIAGNOSIS

Examination of a patient with dental injuries should include the following: history (chief complaint, history of present illness, pertinent medical history) and clinical examination. The emphasis in this chapter is on those aspects of the examination that specifically relate to dental trauma.[4]

History

Pertinent information regarding traumatic injuries should be obtained expeditiously by following a system.

Chief Complaint

The chief complaint is simply a statement in the patient's (or parent's or guardian's) own words of the current problem such as "I broke my tooth," or "My tooth feels loose." It may also be unstated, as in a patient with obvious injuries.

History of Present Illness

To obtain the history of the present illness (injury), the dentist can ask a few specific questions, such as the following:

1. *When and how did the injury occur*? The date and time of the accident must be recorded. The record should also include *how* the injury took place: bicycle accident, playground, sports, violence, or other. Such information is useful in the search for avulsed teeth and embedded tooth fragments, assessment of possible contamination, determination of time factor with respect to choice of treatment and healing potential, and filling out accident reports.

2. *Have you had any other injuries to your mouth or teeth in the past*? Individuals may have repeated traumatic injuries if they are accident prone or active in contact sports.[4] Crown or root fractures may have occurred as a result of an earlier injury but are observed at a later time.

3. *What problems are you now having with your tooth or teeth?* Pain, mobility, and occlusal interference are common symptoms. The patient's description of symptoms will help in diagnosis.

Medical History

The patient's medical history is often significant. For example, the patient may have an allergy to prescribed medication, may be taking medications that interact with proposed new medications, or may have a medical condition that affects treatment. Tetanus immunization status should be recorded; a booster may be indicated when there are contaminating injuries such as avulsions and penetrating lip and soft tissue lesions.[5]

Clinical Examination

The lips, oral soft tissues, and facial skeleton should be examined, as well as the teeth and supporting structures.

Soft Tissues

The purpose of the soft tissue evaluation is to determine the extent of tissue damage and to identify and remove foreign objects from wounds. In crown fractures with adjacent soft tissue lacerations, wounds are examined visually and radiographically for tooth fragments. Lips are likely areas for a foreign body impaction. Also, severe lacerations require suturing (Figure 10-2).

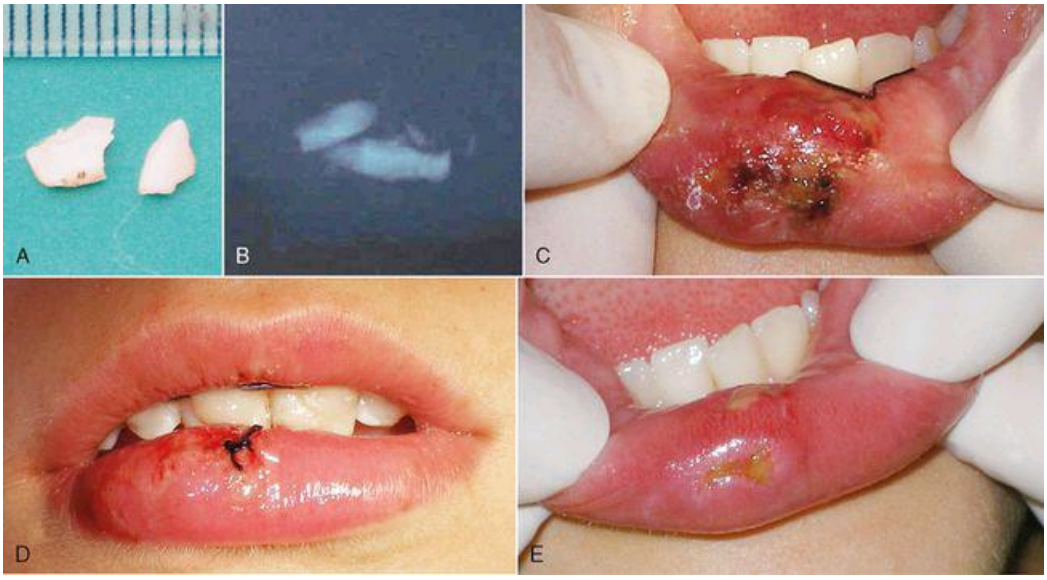

Figure 10-2 Tooth fragment embedded in lip. **A,** Crown fracture involving the maxillary left central incisor. **B,** A radiograph of the lacerated lip showed tooth fragments embedded. These were surgically removed and the lip was sutured **(C,D). E,** One-week follow-up control.

Facial Skeleton

The facial skeleton is evaluated for possible fractures of the jaw or alveolar process. Such fractures, when they involve tooth sockets, may produce pulpal necrosis in teeth associated with fracture lines.[6,7] Alveolar fractures are suspected when several teeth are displaced or move as a unit, when tooth displacement is extensive, when occlusal misalignment is present, or when there is continuous bleeding from gingival tissues.

Teeth and Supporting Tissues

Examination of teeth and supporting tissues should provide information about damage that may have occurred to dental hard tissues, pulps, periodontal ligaments, and bony sockets. The following guidelines provide a method of collecting information systematically.

Mobility

Teeth are examined (gently) for mobility, noting whether adjacent teeth also move when one tooth is moved (indicating alveolar fracture). The degree of horizontal mobility is recorded: 0 for normal mobility, 1 for slight (<1 mm) mobility, 2 for marked (1 to 3 mm) mobility, and 3 for severe (>3 mm) mobility, both horizontally and vertically. If there is no mobility, the teeth are percussed for sounds of ankylosis (metallic sound). Absence of normal mobility may indicate ankylosis or “locking” of the tooth in bone such as with intrusion and lateral luxation.

Displacement

A displaced tooth has been moved from its normal position. If this occurs as a result of a traumatic injury, it is referred to as “luxation.” See the section on Luxation Injuries for descriptions of the various types of displacement.

Periodontal Damage

Injury to the supporting structures of teeth may result in swelling and bleeding involving the periodontal ligament. The involved teeth are sensitive to percussion, even light tapping. Apical displacement with injury to vessels entering the apical foramen may lead to pulp necrosis if the blood supply is compromised.[8]

The use of *percussion* can help identify periodontal injury. This testing procedure must be done gently because traumatized teeth are often exquisitely sensitive to even light tapping. Uninjured teeth should be examined first to enhance the patient's confidence and understanding of the procedures. In addition to testing the tooth or teeth involved in the patient's complaint, several adjacent and opposing teeth also should be included. This testing permits recognition of other dental injuries that the patient may not be aware of and that may not be obvious clinically. If later complications develop involving one of these adjacent or opposing teeth, previous information will help in diagnosis.

Pulpal injury

The ideal pulpal response to injury is complete recovery after a traumatic injury. Two other potential outcomes may occur: calcific metamorphosis in which the pulp tissue is gradually replaced with calcified tissue (and recognized clinically as a yellowing effect on the crown) or pulp necrosis, which can result in external inflammatory (infection-related) root resorption.[9] Rarely, resorption may occur in the pulp space (internal resorption). In any case, a tooth may undergo resorption without any clinical symptoms, emphasizing the need for follow-up controls.

The status of the pulp may be determined by symptoms, history, and clinical tests (see Chapter 5). In particular, two clinical tests deserve consideration because of their applicability to traumatized teeth—the electrical pulp test (EPT) and carbon dioxide (CO_2) ice test. These tests are generally reliable in evaluating and monitoring pulpal status except in teeth with incomplete root development.[2,8] A protocol for EPT and the CO_2 ice test is discussed in the section on Luxation Injuries.

Radiographic Examination

Radiographs can reveal fractures of bone and teeth and the stage of root development. Horizontal root fractures and lateral luxations are often overlooked because the conventional angle may miss irregularities that are not parallel with the x-ray beam. Therefore multiple exposures should be routine for examination of traumatized teeth to ensure complete disclosure and diagnosis of the injury.[2,8]

The film size should be such that it can accommodate two incisors without bending or distorting the image. It is also important to use a film holder whenever possible to achieve standardized radiographic images, especially for subsequent comparisons.

A thorough examination combined with accurate records forms the basis for an appropriate treatment plan. The information gathered also provides content for accident reports that may be requested either immediately or later for legal or insurance purposes.

SPECIFIC INJURIES

Enamel Fractures

Chips and cracks confined to enamel do not in themselves constitute a hazard to the pulp. The prognosis is good; however, the injury that produced the fracture may also have displaced (luxated) the tooth and damaged the blood vessels supplying the pulp. If the tooth is sensitive to percussion or if there are other signs of injury, the recommendations in the section on Luxation Injuries are followed. Grinding and smoothing the rough edges or restoring lost tooth structure may be all that is necessary.

Crown Fractures without Pulp Exposure

Description

Crown fractures that involve enamel and dentin without pulpal exposure (Figure 10-3) are also known as *uncomplicated crown fractures.* Such injuries are usually not associated with severe pain and generally do not require urgent care. The prognosis is good unless there is an accompanying luxation injury, in which case the tooth may be sensitive to percussion.[8] If so, the recommendations as outlined in the section on Luxation Injuries must also be followed.

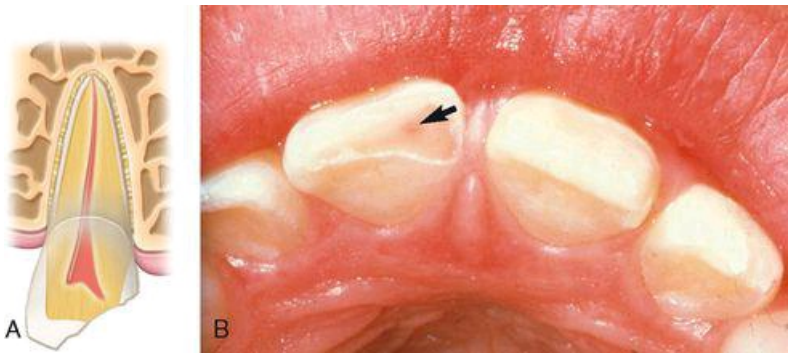

Figure 10-3 Crown fracture without pulp exposure. **A,** The injury results in loss of enamel and dentin without direct exposure to the pulp. **B,** Often a pink spot can be seen *(arrow)*, but if there is no direct exposure of pulp, the fracture is said to be "without pulp involvement."

Treatment

Since the advent of the acid-etch technique, conservative restoration with composite resin of crown-fractured incisors has become possible without endangering the pulp (Figure 10-4). More conservative yet is reattachment of the separated enamel-dentin fragment (Figure 10-5). This reattachment requires a dentin-bonding agent after acid etching the enamel to improve fracture strength of the restored incisor. Clinical experiments, as well as bonding studies, have indicated that reattachment of dentin-enamel crown fragments is an acceptable restorative procedure and does not threaten pulp vitality.[10] Generally, fracture bonding represents an advance in the treatment of anterior fractures. Dental anatomy is restored with normal tooth structure that abrades at a rate identical to that of the adjacent noninjured teeth. Also, pulpal status may be reliably monitored.

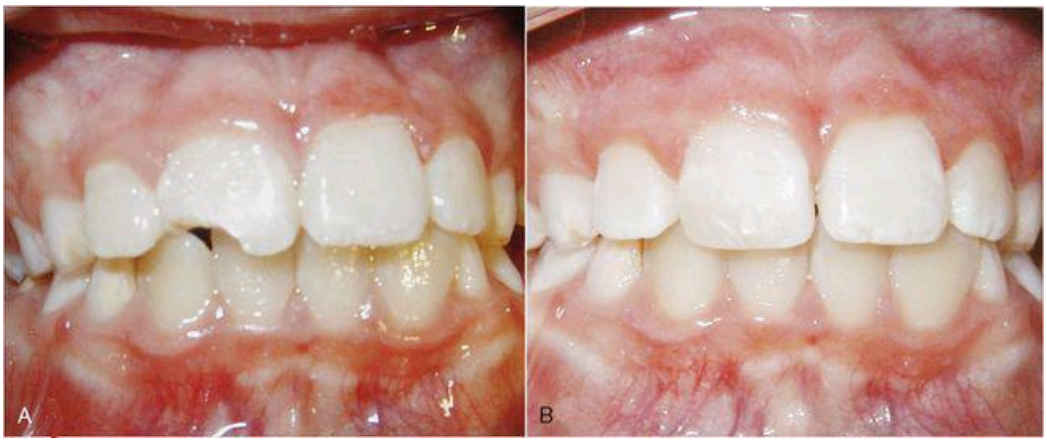

Figure 10-4 A, An uncomplicated crown fracture without pulp exposure in an 8-year-old boy. **B,** Protection of the fracture site is accomplished by repairing the crown with acid-etched bonded composite.

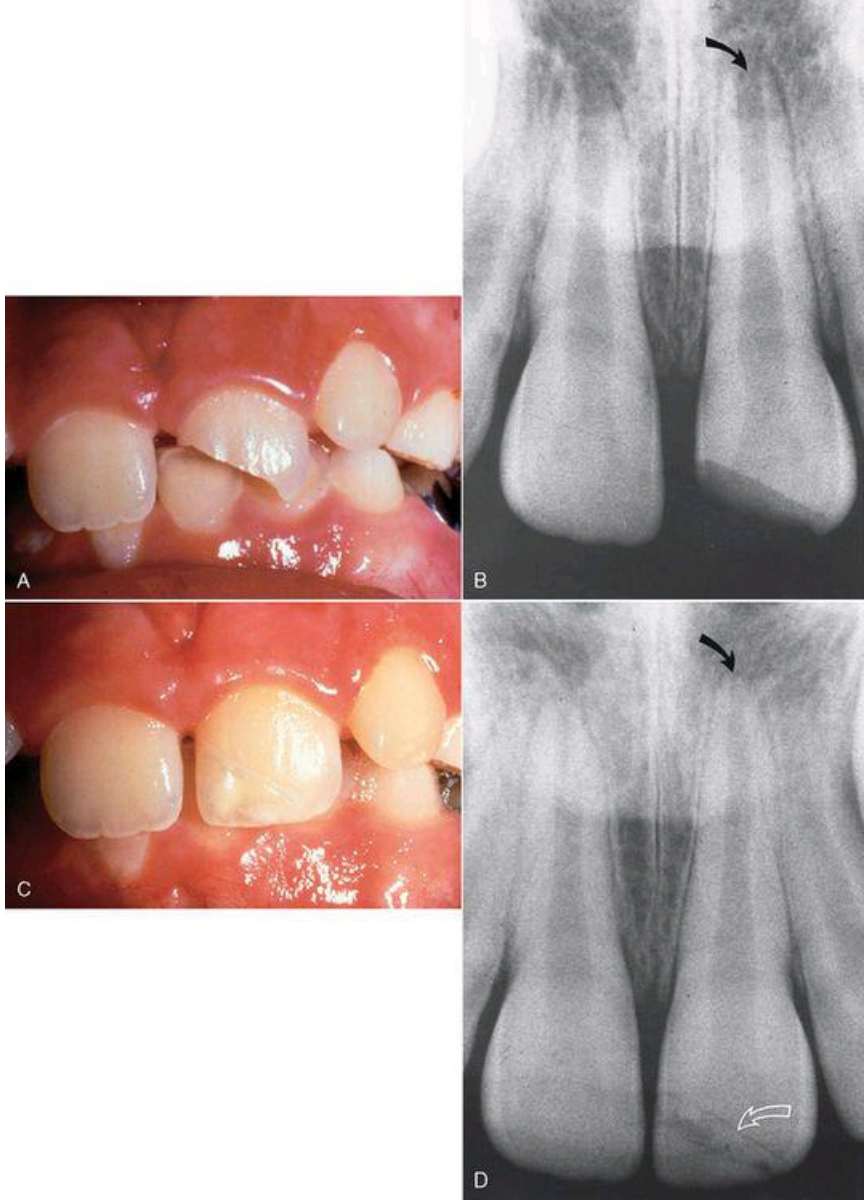

Figure 10-5 A, An 8-year-old patient with a fractured incisor without pulp exposure. **B,** Immature tooth with open apex *(arrow)*. **C,** After the fractured segment was bonded into its original position. **D,** One-year evaluation showing continued root maturation and closing of apex *(black arrow)*. Note the line where the fractured segment was bonded to the remaining tooth *(white arrow).*

(From Bakland LK, Milledge T, Nation W: Calif Dent Assoc J 24:45, 1996.)

Chair time for the restorative procedure is minimal. The use of indirect veneering techniques at a later date to reinforce bonding or to restore the fractured incisor is a conservative approach to improving esthetics and function.[11]

Crown Fractures with Pulp Exposure

Description

Crown fractures with pulp exposure involve the enamel, dentin, and pulp (Figure 10-6) and are also referred to as a *complicated crown fracture.* The pulp is exposed, and therefore the fracture becomes complicated. The extent of fracture, the stage of root development, and the length of time since injury are noted.

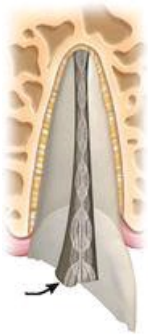

Figure 10-6 Crown fracture that results in exposure of the pulp. Granulation tissue *(arrow)* forms at the pulp wound within 24 hours and may actually proliferate and protrude with time.

Considering the extent of fracture helps to determine pulpal treatment and restorative needs. A small fracture may undergo vital pulp therapy and can be restored by an acid-etched composite restoration. An extensive fracture may require root canal treatment with a post and core-supported crown, depending on the age of the patient.

The stage of root maturation is an important factor in choosing between pulpotomy and pulpectomy. Because immature teeth have thin-walled roots (see Figure 10-1), every effort should be made to preserve the pulp to allow continued root development (Figure 10-7). The best way to this goal is with a *shallow (partial) pulpotomy*, which is described in the next section. Vital pulp therapy followed by an acid-etched composite restoration or reattaching the fractured segment is often feasible in mature teeth also. However, if the extent of tooth loss dictates restoration with a crown, root canal treatment is recommended.[12]

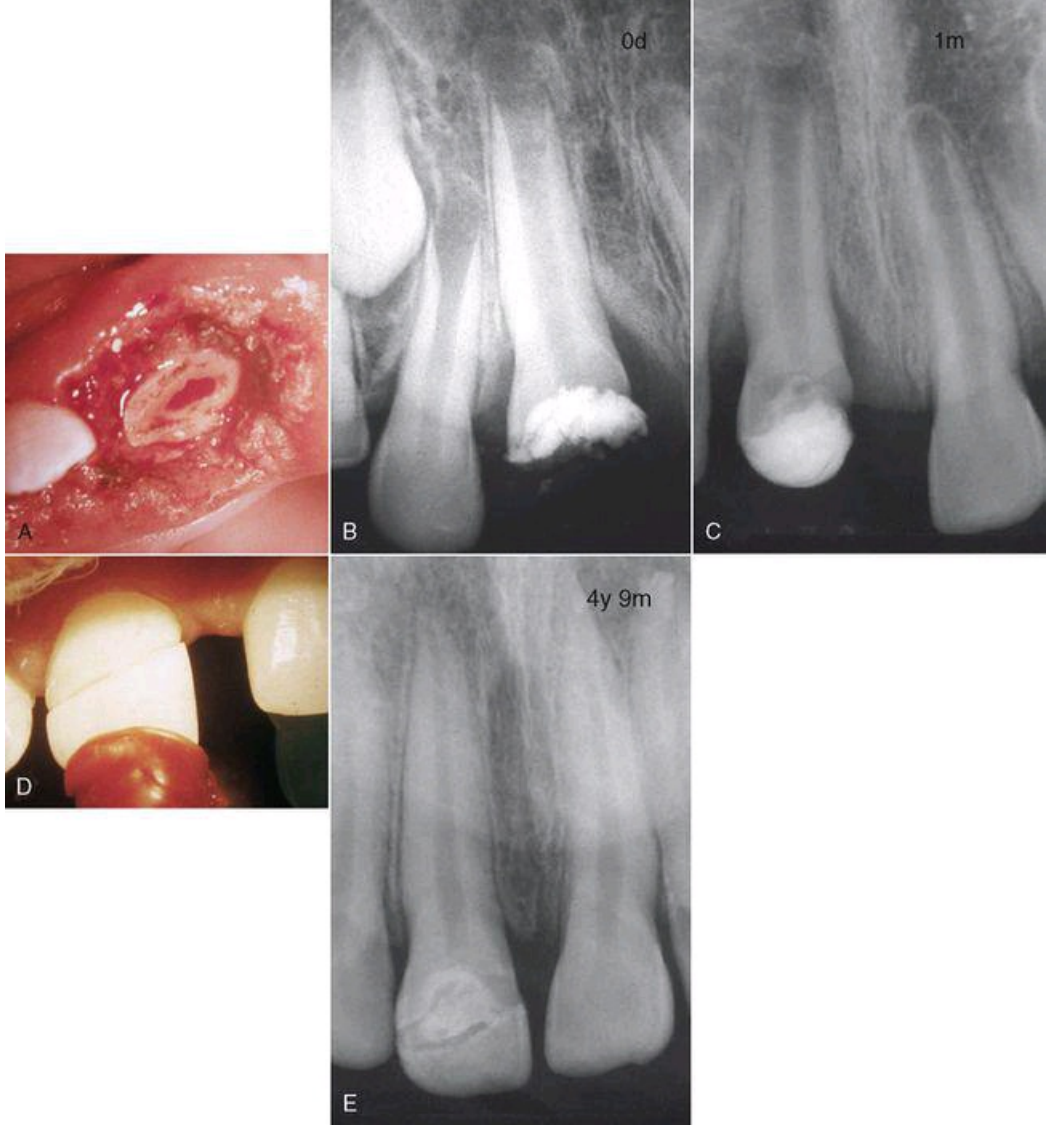

Figure 10-7 Pulpotomy as a conservative treatment for a crown fracture that resulted in exposure of the pulp in a partially erupted immature tooth. **A,** Horizontal mesiodistal chisel fracture in a 9-year-old boy. The coronal tooth fragment was found and stored in saline. **B,** Radiographic view after pulpotomy treatment. The immature incisor has thin-walled roots and open apex. **C,** Continuing root development is seen at one month control. A protective dressing of hard-setting calcium hydroxide covered with glass ionomer and composite was placed as a temporary restoration. **D,** One year later, when the tooth had completed its eruption, the coronal tooth fragment was reattached with acid-etched composite resin. **E,** Complete root formation is observed at 4-year 9-month follow-up control.

The amount of time elapsed between injury and examination may directly affect pulpal health.[13] Generally, the sooner a tooth is treated, the better the prognosis for preserving the pulp. However, as a rule, pulps that have been exposed for less than a week can be treated by pulpotomy. Successful pulpotomy procedures after pulp exposure of several weeks' duration have been reported,[14,15] but the prognosis becomes poorer the longer the pulp is exposed.[13]

Treatment of Crown Fractures

Teeth with crown fractures and exposed pulps can be treated either by pulp capping or pulpotomy (vital pulp therapy) or by root canal therapy before restoration of lost tooth structure. If vital pulp therapy is planned, it is important to perform treatment as soon after the injury as possible.

Vital Pulp Therapy

The main reason for recommending vital pulp therapy in a tooth with an exposed pulp is to preserve the vitality of the pulp. This is particularly important in immature teeth in which continued root development will result in a stronger tooth that is more resistant to fractures than one with thin root walls.

In the past, pulpotomy meant removal of pulp tissue to or below the cervical level. Loss of pulp tissue in that area prevents dentin formation, which results in a weakened tooth that is more prone to fracture. In recent years, a more conservative and shallow pulpotomy has been popularized by Cvek and has sometimes been referred to as the *Cvek technique*.[14] This shallow or partial pulpotomy preserves all the radicular and most of the coronal pulp tissue, allowing more hard tissue to develop in the

root.

The pulp may need to be removed to or below the cervical level when the entire crown of an immature tooth fractures (see Figure 10-7). Pulpotomy then is performed to encourage enough additional root development to allow subsequent crown restoration or post and core construction to support a crown. These situations are relatively uncommon. In recent years, the technique has been modified to use mineral trioxide aggregate (MTA) (ProRoot MTA, Tulsa Dental Products, Tulsa, OK) instead of calcium hydroxide.[16,17]

Case Selection

Both immature and mature teeth that can subsequently be restored with acid-etched composite can be treated with a shallow (partial) pulpotomy (see Figure 10-7). Generally, immature teeth are more likely to be involved for the reasons stated previously.

Technique

The shallow (partial) pulpotomy procedure (Figure 10-8) starts with anesthesia and rubber dam isolation. Exposed dentin is washed with saline or sodium hypochlorite solution. Extruding granulation tissue is removed with a spoon excavator from the pulp wound site. This provides an opportunity to determine more accurately the size and location of the exposure. Next, pulp tissue is removed to a depth of about 2 mm below the exposure. This relatively small amount of pulp removal is the reason for calling this procedure a shallow or partial pulpotomy.

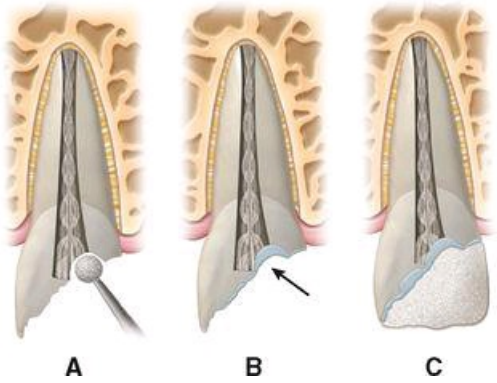

Figure 10-8 Shallow pulpotomy. **A,** Preparation of the pulp wound area with a round diamond stone cooled by a constant water spray. **B,** Protective dressing *(arrow)* of hard-setting calcium hydroxide covered with sealing cement (zinc oxide–eugenol, zinc phosphate, or glass ionomer). **C,** Missing tooth structure restored with acid-etched composite resin bonded to enamel.

The procedure is accomplished using a water-cooled small round diamond (about the size of a No. 4 round bur) in the high-speed handpiece. Gently and gradually, the surface layers of pulp tissue are wiped away, beginning at the exposure site and extending into the pulp to a depth of about 2 mm below the exposure site.

After the pulp is amputated to the desired level, a dentin shelf is created surrounding the pulp wound. The wound is gently washed with sterile saline, and hemostasis can usually be expected within 5 minutes. Then the wound is washed again to remove the clot and is dressed with calcium hydroxide. The remainder of the cavity is carefully sealed with a hard-setting cement such as glass ionomer. When the cement has set, the tooth may be restored with acid-etched composite.

Considering the disintegration of calcium hydroxide liners with time, whenever possible the tooth should be reentered after a period of 6 to 12 months to remove the initial calcium hydroxide layer and replace it with a dentin bonding material. This will prevent microleakage at the site where the initial calcium hydroxide has deteriorated and produced a space between the new dentin bridge and the covering restoration.

If MTA is used in place of calcium hydroxide, it is not necessary to wait for bleeding to stop completely. The material requires moisture for curing and can be placed directly onto the pulp tissue. Care should be followed to reduce the risk of forcing the material into the pulp proper; gently dab small increments of the material onto the pulp using a moist cotton pellet. The pulpotomy space is filled with MTA white powder so that it is completely flush with the fractured dentin surface. The material is then allowed to cure, which may take 4 to 6 hours. During the curing time, it is not necessary to protect the material with a restoration, but the patient must avoid using the tooth. After curing, the tooth may be restored either with a composite resin or by bonding the fractured crown segment back onto the tooth.[17] It is not necessary to reenter the tooth because MTA is stable and does not break down in the manner of calcium hydroxide.

Treatment Evaluation

Treatment is evaluated after 6 months and then yearly. Successful shallow pulpotomy procedures (Box 10-2) may be considered definitive treatment and have a very good long-term success rate.[15,18]

Box 10-2 Criteria for Successful Shallow Pulpotomy

1. The tooth is asymptomatic and functions properly.
2. There is no radiographic evidence of apical periodontitis.
3. There is no indication of root resorption.
4. The tooth responds to pulp testing (if pulp testing is possible).
5. Continued root development and dentin formation are evident radiographically, if the root was immature at the time of treatment. If the pulp becomes necrotic or root formation is arrested, apexification is then necessary.

Root Canal Therapy

Teeth with mature roots may undergo either pulpotomy or root canal therapy. Root canal therapy is usually necessary to accommodate prosthetic requirements. For example, if the crown has fractured in the gingival margin region, root canal treatment will allow post and core and crown placement.

Crown-Root Fractures

Description

These fractures are usually oblique and involve both crown and root. Anterior teeth show the so-called chisel-type fracture, which splits the crown diagonally and extends subgingivally to a root surface (Figure 10-9). They resemble a crown fracture but are more extensive and more serious because they include the root. Another variation is the fracture that shatters the crown (Figure 10-10). The pieces are held in place only by the part of a fractured segment still attached to the periodontal ligament. In all of these fractures, the pulp is usually exposed.

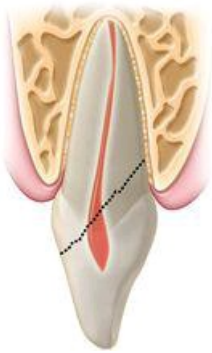

Figure 10-9 Crown-root fracture. Anterior teeth may develop “chisel-type” fractures, which extend below the cementoenamel junction. Because of the nature and location of these fractures, they are difficult to manage.

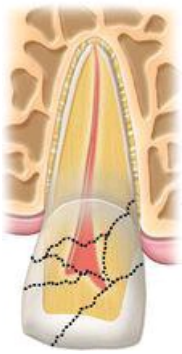

Figure 10-10 Crown-root fracture resulting in a shattered crown with subgingival extension.

In contrast to other traumatic injuries in which posterior teeth are rarely involved, crown-root fractures often include the molars and premolars. Cusp fractures that extend subgingivally are common (Figure 10-11). Diagnostically, however, they may be difficult to identify in the early stages of development. Similarly, vertical fractures along the long axis of roots are difficult to detect and diagnose. Such crown-root fractures are discussed in more detail in Chapter 7.

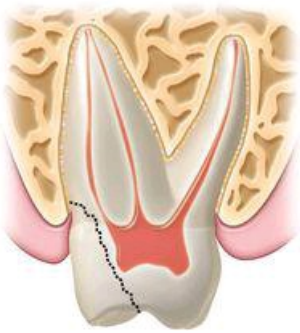

Figure 10-11 Pulp exposure may or may not be present with a crown-root fracture of a posterior tooth.

Crown-root fractures in posterior teeth cannot always be associated with a single traumatic incident, although bicycle or automobile accidents may produce these results at times. The risk is increased with a sharp blow to the chin, causing the jaws

to slam together, and skin abrasions under the chin may be a sign of such an impact. Also, all posterior teeth should be examined using a sharp explorer to detect movement of loose fragments.[1,2]

Examination

Crown-root fractures are complex injuries that are both difficult to evaluate and to treat. Until recently, it was recommended that all loose fragments be removed to evaluate the extent of injury. This may still be necessary in some instances, but with the availability of bonding agents it is now possible to bond loose fragments at least temporarily. The current recommendation is to attempt to bond loose fragments together, particularly if the tooth is immature and still developing.[12] Clinical judgment must be used to decide when to follow one course of action or another.

It is not unusual, in a tooth in which the crown has broken into several pieces, to find that the same shattering effect has extended to the root as well. Additional radiographs at different angles (as described in the section on Root Fractures) may help to identify radicular fracture lines.[2]

Emergency Care

Teeth with crown-root fractures are often painful. Such injuries often require urgent care that may consist of bonding loose tooth fragments but often also includes pulp therapy (Figure 10-12). If the root is immature, pulpotomy (see the section on Crown Fractures with Pulp Exposure in this chapter and also the section on Vital Pulp Therapy in Chapter 2) is preferable to pulpectomy, whereas pulpectomy is the treatment of choice in patients with fully developed teeth. Definitive treatment should be postponed until an overall endodontic and restorative treatment plan is developed.[12]

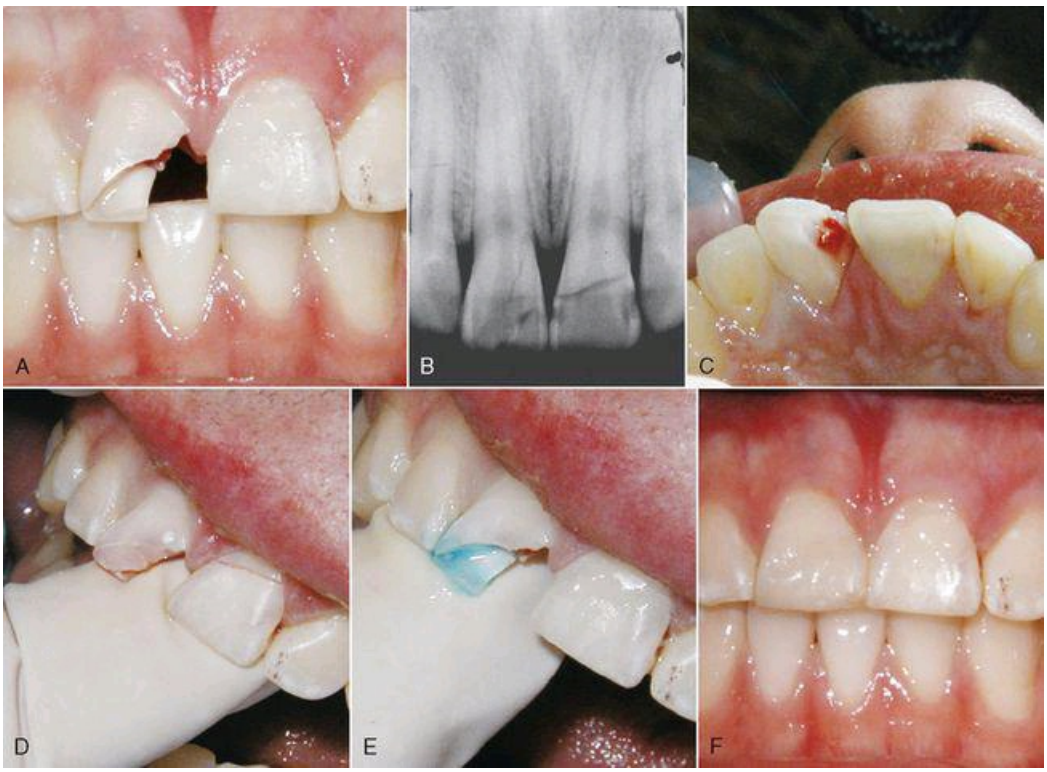

Figure 10-12 Urgent care for crown-root fractures of the maxillary central incisors in a 15-year-old boy. Because of the nature and location of these types of fractures, they are difficult to manage. **A,** The fractured segments of both teeth are mobile and the fractures extend below cementoenamel junction. **B,** There is a mesial oblique fracture line in the right incisor and a horizontal fracture line in the left incisor in the middle third of the crown. **C,** After removal of the loose fragment, which has a subgingival extension, the pulp is exposed. **D,** The fractured segment of the crown is repositioned with digital pressure. **E,** The tooth fragments are bonded as temporary restoration, and pulp therapy should be considered at this time. **F,** Esthetics is reestablished at the emergency visit after composite restorations of both teeth.

(Courtesy Dr. J. Onetto.)

Treatment Planning

Crown-root fractures are often complicated by pulp exposures and extensive loss of tooth structure. In developing a treatment plan, the following questions must be considered:

□ Which is better for this tooth, pulpotomy or pulpectomy?

□ After all loose fragments have been removed, will there be enough tooth structure to support a restoration? Or, if the loose fragments are bonded in an immature tooth, will it last until the alveolus has developed enough for placement of an implant?

□ Is the subgingival fracture below a level at which a restorative margin can be placed, thus necessitating root extrusion or gingivoplasty or alveoplasty?

□ Should the tooth be extracted and replaced with a bridge or implant? Or, if extraction is chosen, can the space be closed orthodontically?

These are but a few of the many questions to be asked not only in cases of crown-root fractures but also in other trauma situations that are very complicated. Because of this complexity, a team approach involving specialists in the areas of pediatric dentistry, endodontics, periodontics, orthodontics, oral and maxillofacial surgery, and prosthodontics is beneficial in developing the treatment plan.

Root Fractures

Description

Fractures of roots (Figure 10-13) have been called intraalveolar root fractures, horizontal root fractures, and transverse root fractures. They do not occur often and may be difficult to detect.[1,2,4,19]

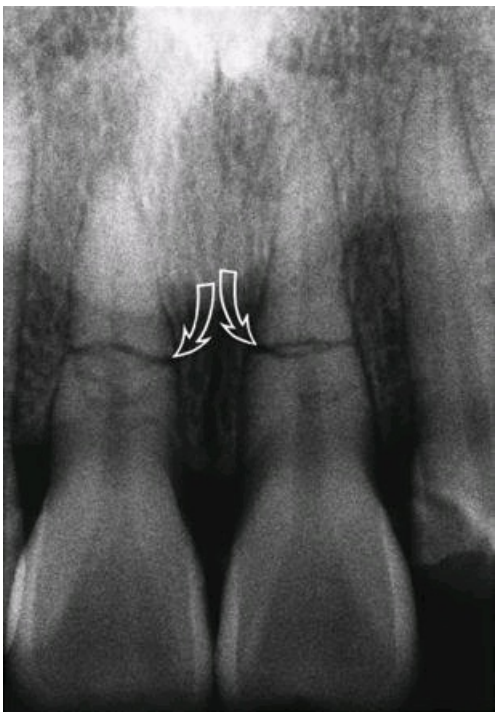

Figure 10-13 Horizontal root fractures. Central incisors are the teeth most often involved. Unless the coronal segments are displaced or mobile, no splinting or other treatment is necessary.

Radiographically, a root fracture is visualized if the x-ray beam passes through the fracture line. Because these fractures often are transverse-to-oblique (involving pulp, dentin, and cementum), they may be missed if the central beam's direction is not parallel or close to parallel to the fracture line. For this reason, a steep vertical angle is included in addition to the normal parallel angle whenever a root fracture is suspected. This additional angle (i.e., a foreshortened view or an occlusal view that is approximately 45 degrees) will detect many fractures, particularly in the apical regions of the roots (Figure 10-14).[19,20]

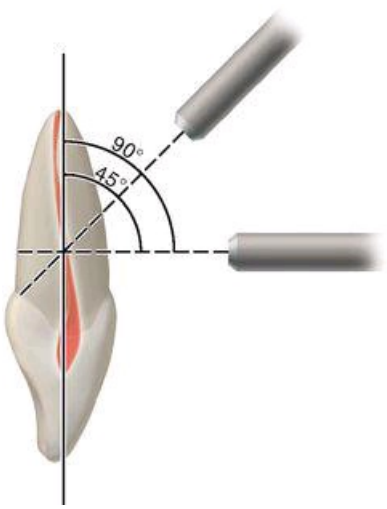

Figure 10-14 Radiographic technique used for suspected root fractures. At least two angulations are made: the conventional (90-degree) view and a steep vertical (45-degree) view. Additional angulations help to detect suspected root fractures by directing the x-ray beam through a diagonal fracture.

Clinically, root fractures may present as mobile or displaced teeth, with pain on biting. Symptoms are generally mild. If

mobility and displacement of the coronal segment are absent or slight, the patient may have no chief complaint and may not seek treatment.[21] Generally, the more cervical the fracture, the more mobility and displacement of the coronal segment and a greater likelihood of pulp necrosis of this segment if not promptly repositioned. Splinting is indicated in cervical and middle third root fractures.[12,22] Root fractures in the apical third usually require no immediate treatment but must be observed long term.[22]

Emergency Care

Initial treatment for root fractures—repositioning and stabilization—should be classed as an acute priority for best results (Figure 10-15). Repositioning of displaced coronal tooth segments is easier if performed soon after the injury, and delayed repositioning may require orthodontic intervention to be able to move the coronal segment into desirable position.

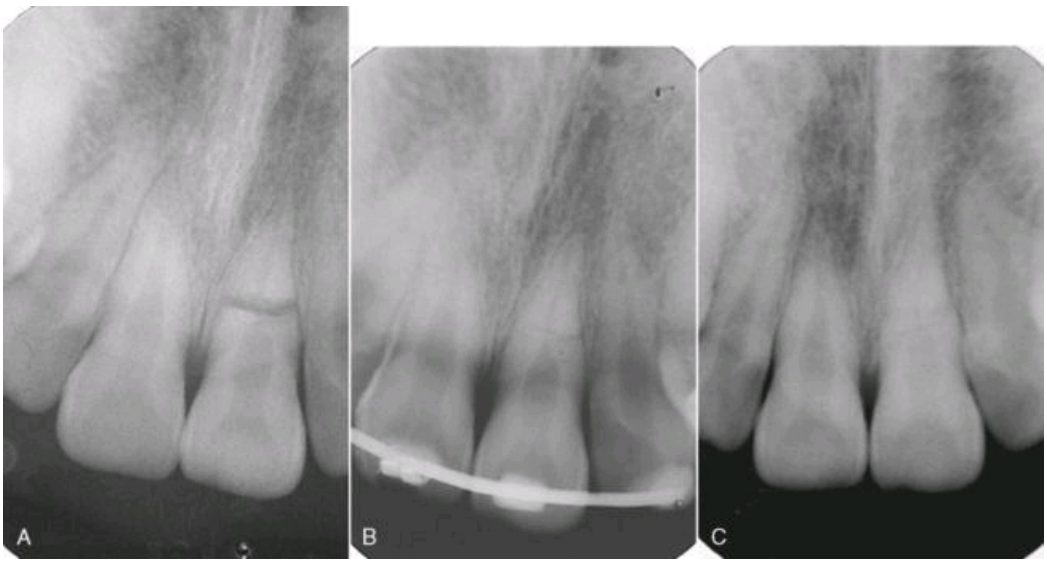

Figure 10-15 Stabilization of a root fracture in the left maxillary central incisor; the coronal segment has been displaced. **A,** A horizontal root fracture is located at the middle third of the root as shown in a steep vertical (45-degree) view x-ray film. **B,** Radiographic examination after splinting reveals tight repositioning of the root fragments. **C,** Later, healing with hard tissue is shown radiographically. The fracture line is slightly visible on the film, and the fragments are in close contact. Internal calcification adjacent to the fracture indicates repair.

After repositioning, the coronal tooth segment must be splinted to allow repair of the periodontal tissues. Four to six weeks of stabilization is usually sufficient, unless the fracture location is close to the crest of the alveolar bone, in which case longer splinting time periods may be advisable.[22] The outcome of the emergency care must be monitored periodically.

Sequelae of Root Fractures

Root fractures are often characterized by development of calcific metamorphosis (radiographic obliteration) in one (usually coronal) or both segments; therefore EPT readings may be very high or absent. Lack of response to EPT by itself, however, in the absence of other evidence of pulp necrosis (bony lesions laterally at the level of the fracture or symptoms of irreversible pulpitis or necrosis) does not indicate a need for root canal treatment. The majority of root fractures heal either spontaneously or after splint therapy (see Figure 10-15).[22-25]

Root Canal Treatment

Root canal treatment is indicated when pathosis is evident, usually due to the development of pulp necrosis in the coronal portion, which subsequently leads to inflammatory lesions adjacent to the fracture lines (Figure 10-16).[25] The endodontic procedure, when necessary, usually is complex, and referral to a specialist should be considered. In contrast to root canal treatment in most other endodontic situations, when it is indicated for teeth with horizontal root fractures, the treatment is usually limited to the root canal in the coronal segment to the fracture line. The pulp in the apical segment usually remains vital.[25-28]

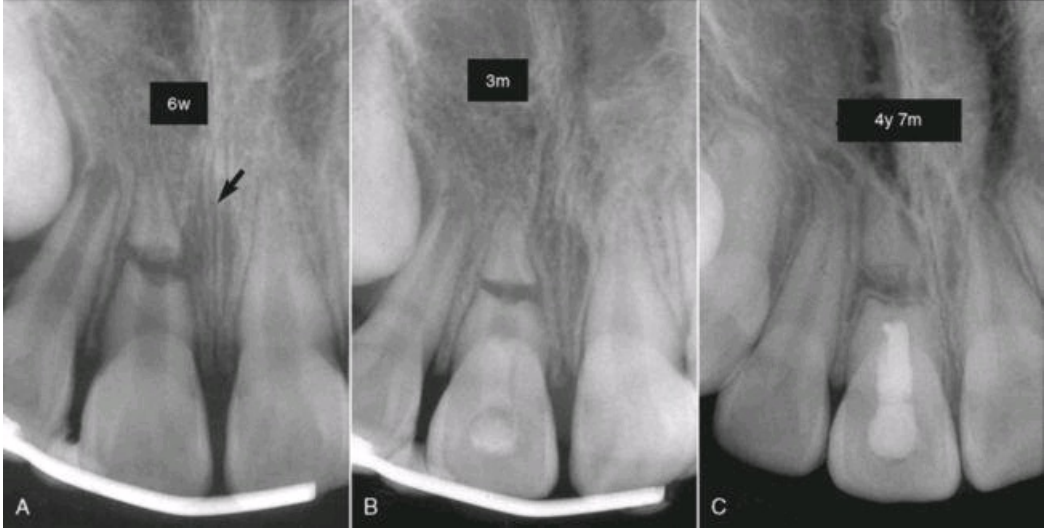

Figure 10-16 Root canal treatment for a root fractured incisor due to development of pulp necrosis in an 8-year-old girl. **A,** Six weeks after repositioning and splinting the right central incisor, an increased space between the fragments and a radiolucency located laterally at the level of the fracture developed. **B,** The root canal of the coronal fragment was treated and filled with calcium hydroxide. The x-ray film at 3 months shows formation of a hard tissue barrier at the apical end of the coronal segment. The coronal root canal was filled with gutta-percha. Note healing of the marginal bone. **C,** A normal periodontal ligament contouring both fragments and interposition of bone is seen after 4 years 7 months.

Luxation Injuries

Description

Luxation injuries involve trauma to the supporting structures of teeth and often affect the neural and vascular supply to the pulp (Figure 10-17). The cause is usually a sudden impact such as a blow or striking a hard object during a fall.[1,2,29] Generally, the more severe the luxation (involving more displacement), the greater the damage to the periodontium and dental pulp. Table 10-1 provides a summary of the typical clinical and radiographic findings associated with different types of luxation injuries.[1]

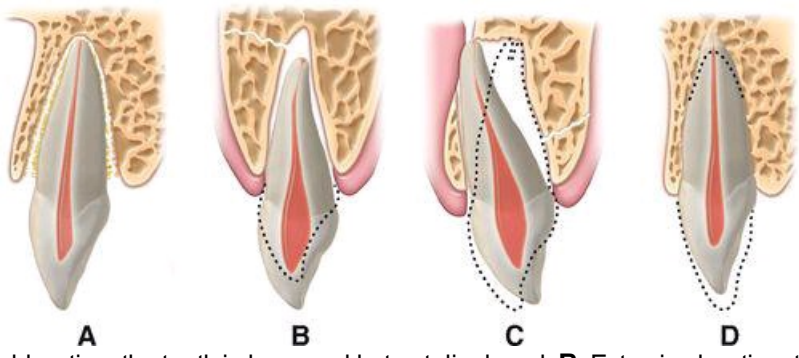

Figure 10-17 Luxation injuries. **A,** Subluxation: the tooth is loosened but not displaced. **B,** Extrusive luxation: the tooth is partially extruded from its socket. Occasionally this is accompanied by an alveolar fracture. **C,** Lateral luxation: the crown is displaced palatally and the root apex labially. **D,** Intrusive luxation: the tooth is displaced apically.

Table 10-1 Clinical Findings with Various Types of Luxation Injuries

Clinical Finding	Concussion	Subluxation	Extrusive Luxation	Lateral Luxation	Intrusive Luxation
Abnormal mobility	–	+	+	– (+)*	– (+)*
Tenderness to percussion	+	+ (–)*	±	– (+)*	– (+)*
Percussion sound	Normal†	Dull	Dull	Metallic	Metallic
Positive response to pulp testing	±	±	– (+)*	– (+)*	– (+)*
Clinical dislocation	–	–	+	+	+
Radiographic dislocation	–	–	+	+	+

From Andreasen JO, Andreasen FM: *Textbook and color atlas of traumatic injuries to the teeth*, ed 3, St Louis, 1994, Mosby.
*A sign in parentheses indicates a finding of rare occurrence.
†Teeth with incomplete root formation and teeth with marginal or periapical inflammatory lesions will also elicit a dull percussion sound.

Concussion

The tooth is sensitive to percussion only. There is no increase in mobility, and the tooth has not been displaced. The pulp may respond normally to testing, and no radiographic changes are found.[1,2,12]

Subluxation

Teeth with subluxation injuries are sensitive to percussion and also have increased mobility. Often sulcular bleeding is present, indicating vessel damage and tearing of the periodontal ligament. The teeth are not displaced, and the pulp may respond normally to testing, sometimes after initially failing to respond. Radiographic findings are unremarkable.[12]

Extrusive Luxation

These teeth have been partially displaced from the socket along the long axis. Extruded teeth have greatly increased mobility, and radiographs show displacement. The pulp usually does not respond to testing.[1,2,12]

Lateral Luxation

The teeth may be displaced lingually, buccally, mesially, or distally (i.e., away from their normal position in a horizontal direction). If the apex has been displaced into the surrounding alveolar bone, the tooth may be quite firm. Percussion sensitivity may or may not be present, with a metallic sound if the tooth is firm, indicating that the root tip has been forced into the alveolar bone.[1,2,12]

Intrusive Luxation

These teeth are forced into their sockets in an axial (apical) direction, at times to the point of being buried and not visible. They have no mobility, resembling ankylosis.[1,2,12,30]

Examination and Diagnosis

The clinical descriptions of the five types of luxation injuries should be sufficient to make the initial diagnosis. Pulpal status must be continually monitored until a definitive diagnosis can be made, which in some cases may require several months or years. The CO_2 ice test and the EPT are used in monitoring pulpal status.[4,31]

Concussion injuries generally respond to pulp testing. Because the injury is less severe, pulpal blood supply is more likely to return to normal. Teeth in the subluxation injury group also tend to retain or recover pulpal responsiveness but less predictably than teeth with concussion injuries. In both cases, an immature tooth with an open apex usually has a good prognosis.

Extrusive, lateral, and intrusive luxation injuries involve displacement of the teeth and therefore more damage to apical vessels and nerves. Pulp responses in teeth with extrusive, lateral, and intrusive luxations are often absent. These pulps often do not recover responsiveness even if the pulp is vital (has blood supply) because sensory nerves may be permanently damaged. Exceptions are immature teeth with wide-open apices in which pulp vitality (responsiveness) is gained or retained even after severe injuries.[8,31]

Monitoring pulpal status requires a schedule of pulp testing and radiographic evaluations for a long enough period of time to permit determination of the outcome with a degree of certainty (may require 2 or more years). Pulpal status is best monitored with pulp testing, radiographic findings, developing symptoms, and observation for crown color changes.[8,9,31]

Pulp Testing

The CO_2 ice test or the EPT is used to test teeth that have been injured; several adjacent and opposing teeth are included in the test. An initial lack of response is not unusual nor is a high reading on the pulp tester. Retesting is done in 4 to 6 weeks, and the results are recorded and compared with the initial responses. If the pulp responds in both instances, the prognosis for pulp survival is good. A pulp response that is absent initially and present at the second visit indicates a probable recovery of vitality, although cases of subsequent reversals have been noted.[1,2] If the pulp fails to respond both times, the prognosis is questionable and the pulp status uncertain. In the absence of other findings indicating pulp necrosis, the tooth is retested in 3 to 4 months. Continued lack of response may indicate pulp necrosis by infarct, but lack of response may not be enough evidence to make a diagnosis of pulp necrosis. The pulp may permanently lose sensory nerve supply but retain its blood supply. After a period of time, the pulp often responds to testing if it recovers.[9,31]

Radiographic Evaluation

The initial radiograph made after the injury will not disclose the pulpal condition. However, it is very important for evaluation of the general injury to the tooth and alveolus and serves as a basis for comparison of subsequent radiographs. These radiographs are taken at the same intervals used for pulp testing. Evidence of external resorption and periradicular bony changes is sought. Resorptive changes, particularly external changes, may occur soon after injury. If no attempt is made to arrest the destructive process, much of the root may be rapidly lost. Inflammatory (infection-related) resorption can be intercepted by timely endodontic intervention.[1,2,9,31]

Periodic radiographs will show if the root of a developing tooth is continuing to grow—a positive sign showing recovery of the pulp (see Figure 10-6). Another finding may be pulp space calcification or obliteration, which is a common finding after luxation injuries in immature teeth.[32] Also called *calcific metamorphosis*, this canal obliteration may be partial or nearly complete (after several years) and does not indicate a need for root canal treatment, except when other signs and symptoms suggest pulp necrosis.[32]

Crown Color Changes

Pulpal injury may cause discoloration, even after only a few days. Initial changes tend to be pink. Subsequently, if the pulp

does not recover and becomes necrotic, there may be a grayish darkening of the crown, often accompanied by a loss in translucency. Also, color changes may take place from calcific metamorphosis of the pulp. Such color changes are likely to be yellow to brown and do not indicate pulp pathosis. Other signs, findings, or symptoms are necessary to diagnose pulp necrosis.[31,32]

Finally, discoloration may be reversed. This usually happens relatively soon after the injury and indicates that the pulp is vital. Because of unpredictable changes associated with traumatized teeth, long-term evaluation is recommended.[31]

Treatment of Luxation Injuries

Luxation injuries, regardless of type, often present diagnostic and treatment complexities that require consultation with specialists. For concussion injuries, no immediate treatment is necessary. The patient should allow the tooth to “rest” (avoid biting) until sensitivity has subsided. Pulpal status is monitored as described. Subluxation may likewise require no treatment unless mobility is moderate; if mobility is graded 2, stabilization may be necessary for a short period of time (1 to 2 weeks).[1,2,12]

Extrusive and lateral luxation injuries require repositioning and splinting. The length of time needed for splinting varies with the severity of injury. Extrusions may need only 2 weeks for splinting, whereas luxations that involve bony fractures need 4 weeks.[1,2,12] Professional judgment will dictate variations from these recommendations. Root canal treatment is indicated for teeth with a diagnosis of irreversible pulpitis or pulp necrosis. Such a diagnosis often requires a combination of signs and symptoms such as discoloration of the crown, lack of pulp response to pulp testing, and a periradicular lesion seen radiographically.[1,2,12]

Treatment of intrusive luxation injuries depends on root maturity.[33,34] If the tooth is incompletely formed with an open apex, it may reposition spontaneously. If it is fully developed, active extrusion will be necessary soon after the injury, either orthodontically or surgically. Root canal treatment is indicated for intruded teeth with the exception of those with immature roots, in which case the pulp may revascularize.[35] The patient must be monitored carefully because complications, such as failure of pulpal healing, will usually be symptomless. If radiographic evidence indicates pulp necrosis (lack of continued root development), root canal treatment should be performed to save the tooth.[12]

Luxated teeth in which the pulps become necrotic are indicated for root canal therapy. Often in luxated teeth, there has been damage to the root cementum. If the pulps become infected, external resorption is stimulated by the presence of bacteria in the pulp space. To arrest any ongoing resorption and to prevent additional resorption, it is important that the root canal treatment includes all efforts to disinfect the root canal system. It has been recommended that calcium hydroxide be placed in the canal for up to 2 weeks to aid in disinfection before filling the root canals.[1,2]

Avulsions

Description

An avulsed tooth is one that has been totally displaced out of its alveolar socket. If the tooth is replanted soon after avulsion (immediate replantation), the periodontal ligament has a good chance of healing. Time out of socket and the storage media used are the most critical factors in successful replantation. It is important to preserve the periodontal ligament cells and the fibers attached to the root surface by keeping the tooth moist and minimizing handling of the root.[1,2,12,36-39]

Treatment

Three situations involving avulsions may occur: (1) someone may telephone for advice about an avulsed tooth, presenting an opportunity for immediate replantation (within minutes); (2) the patient may be brought to the office with a tooth that has been out of the alveolus for *less* than 1 hour, or kept in a suitable storage medium; or (3) the tooth has been out for *more* than 1 hour and not kept in a storage medium.

Immediate Replantation

The prognosis is improved by replantation immediately after avulsion.[36-39] Many individuals—parents, guardians, athletic instructors, and others—are aware of this emergency procedure and can replant on site. Some may ask for advice by phone. The procedure used for immediate replantation is presented in Box 10-3.

Box 10-3 First Aid for Avulsed Teeth

1. Rinse the tooth in cold, running tap water (10 seconds).
2. Do not scrub the tooth.
3. Replace the tooth in the socket using gentle finger pressure.
4. Hold (or have the patient hold) the tooth in position.
5. Seek dental care immediately.

When a patient who has had a tooth replanted at the accident site comes to the dental office, the replantation should be examined both clinically and radiographically. The dentist looks for additional injuries to adjacent or opposing teeth and evaluates the replanted tooth for stability and alignment. The procedure outlined in the next section (with the exception of the replantation step) is followed.

Replantation within 1 Hour of Avulsion—Tooth with a Closed Apex (**Figure 10-18**)

If replantation is not feasible at the place of injury, the injured person should be brought to the dental office and the tooth transported in such a way as to keep it moist.[12] The best transport medium is a commercially available storage-transport medium or physiologic saline (usually neither is available); milk, however, is an excellent alternative.[12] Saliva is acceptable, whereas water is not good for maintaining root-surface cell vitality.[36-39]

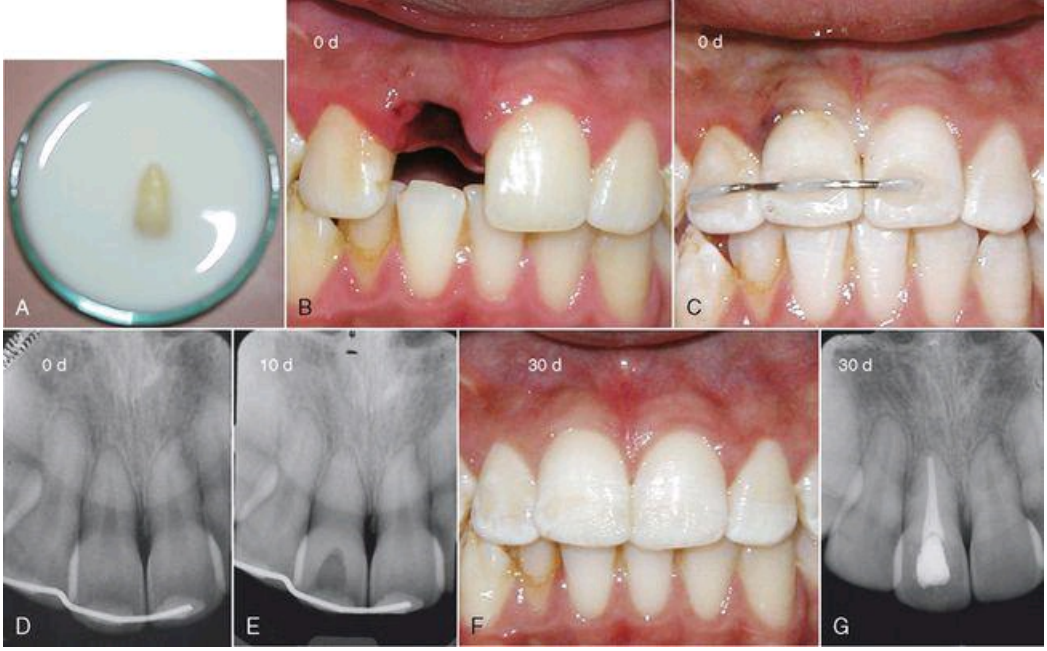

Figure 10-18 Replantation of a tooth with closed apex within 1 hour of avulsion in a 14-year-old boy. **A,** The avulsed central incisor was brought to the dentist in a cup of milk. **B,** Clinical appearance of the avulsion site. **C,** A wire-composite splint is used after repositioning the tooth. **D,** Radiographic examination after splinting. The roots appear short, probably due to history of previous trauma to the anterior teeth. **E,** Calcium hydroxide is placed in the root canal 8 days later and left in place for 2 months. **F-G,** Clinical and radiographic appearance at 2 months follow-up control. Root canal treatment with gutta-percha and sealer has been completed.

When the patient arrives, the following steps are recommended:

1. Place the tooth in a cup of physiologic saline while preparing for replantation.

2. Take radiographs of the area of injury to look for evidence of alveolar fracture.

3. Examine the avulsion site carefully for any loose bone fragments that may be removed. If the alveolus is collapsed, spread it open gently with an instrument.

4. Irrigate the socket gently with saline to remove contaminated coagulum.

5. Grasp with extraction forceps the crown of the tooth to avoid handling the root.

6. Examine the tooth for debris and if present, gently remove it with saline solution from a syringe.

7. Using the forceps, partially insert the tooth into the socket. Gentle finger pressure can be used for complete seating of the tooth, or the patient can bite on a piece of gauze to accomplish the seating.

8. Check for proper alignment and correct any hyperocclusion. Soft tissue lacerations should be tightly sutured, particularly cervically.

9. Stabilize the tooth for 2 weeks with a flexible splint.

10. Antibiotics are recommended for patients with replanted avulsed teeth.[41] In patients 12 years of age and older, doxycycline 100 mg, 2 times per day for 7 days, is the current recommendation. Alternatively, penicillin V 500 mg, 4 times per day for 7 days, can be prescribed. For children under the age of 12, penicillin V 25 to 50 mg/kg of body weight in divided doses every 6 hours for 7 days can be prescribed.[12] A tetanus booster injection is recommended if the last one was administered more than 5 years previously.[5]

11. Supportive care is important. Instruct the patient (and parents) to use a soft diet for up to 2 weeks, to brush with a soft toothbrush after every meal, and to use a chlorhexidine mouth rinse (0.12%) twice a day for a week.

Replantation within 1 Hour of Avulsion—Tooth with Open Apex (Figure 10-19)

When the patient arrives, the following steps are recommended:

1. Place the tooth in a cup of physiologic saline while preparing for replantation.

2. Administer local anesthetic.

3. Examine the alveolar socket, looking for fracture of the socket wall.

4. If available, cover the root surface with minocycline hydrochloride microspheres (Arestin, OroPharma Inc., Warminster, PA) before replanting the tooth.[12]

5. Replant the tooth with slight digital pressure.

6. Suture gingival laceration, especially in the cervical area.

7. Verify normal position of the replanted tooth.

8. Apply a flexible splint for two weeks.

9. For children younger than nine years, penicillin V 25 to 50 mg/kg in divided doses every 6 hours for 7 days can be prescribed. A tetanus booster injection is recommended if the last one was administered more than 5 years previously.

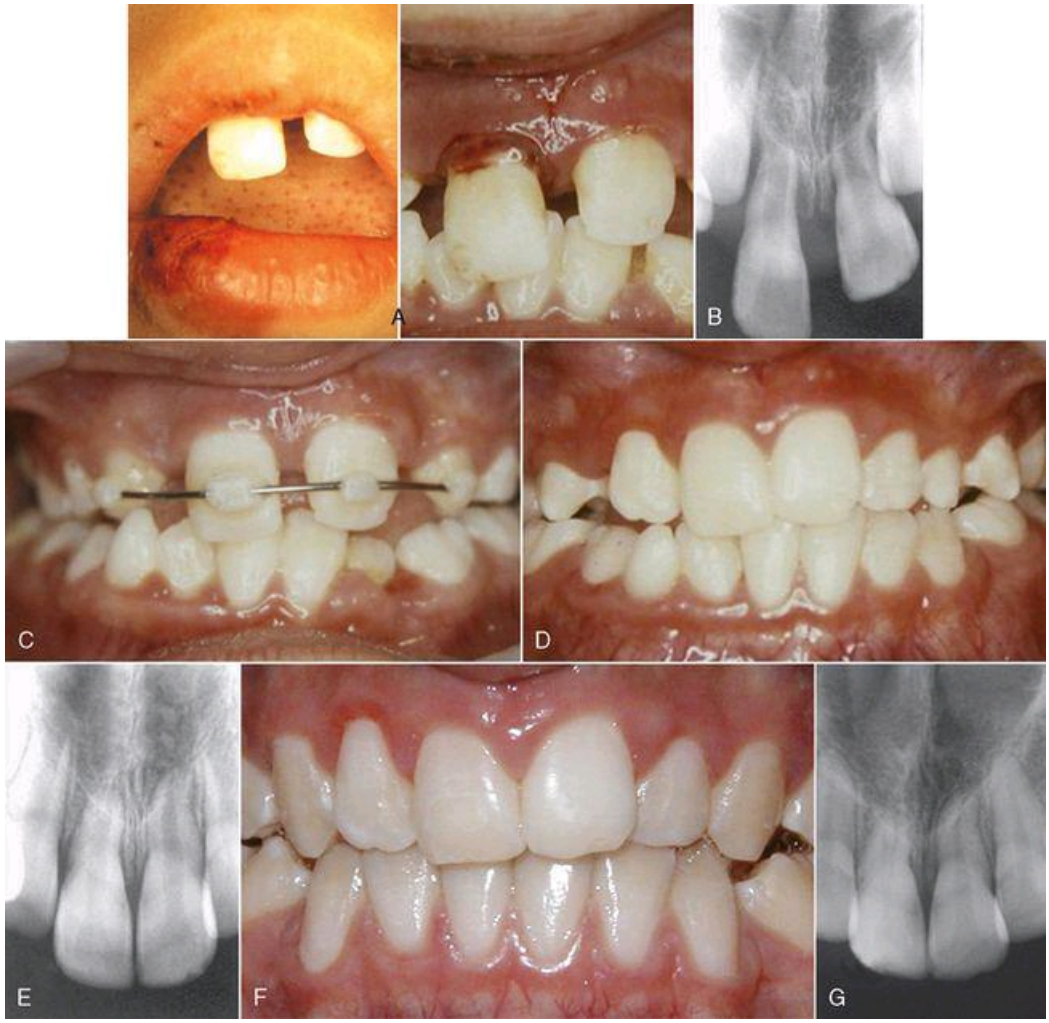

Figure 10-19 Replantation of an immature tooth within 1 hour of avulsion. A 7-year-old boy fell and avulsed the right central incisor, which was found and placed in water during transport to the emergency room. It was replanted (partially) but not splinted. **A,** Clinical appearance of the partially replanted tooth the next day in the dental clinic. **B,** The x-ray film reveals an immature tooth with one third of root formation. **C,** The tooth was repositioned and stabilized with a flexible wire-composite splint. **D-E,** Clinical and radiographic appearance at 1 year follow-up control. Note that the root formation is stunted but the root canal walls have continued to develop due to revascularization of the pulp after replantation. No root canal treatment is indicated. **F-G,** Control after 6 years shows the tooth in normal position and pulp canal obliteration.

Root canal treatment is indicated for mature teeth and should be done optimally after 1 week and before the splint is removed (the splint stabilizes the tooth during the procedure). The exceptions to routine root canal therapy are immature teeth with wide-open apices; they may re-vascularize but must be evaluated at regular intervals of 2, 6, and 12 months after replantation. If subsequent evaluations indicate pulp necrosis, root canal treatment, probably including apexification, is indicated.[12,42]

Replantation More Than 1 Hour After Avulsion—Tooth with Closed Apex

If a tooth has been out of the alveolar socket for more than 1 hour (and not kept moist in a suitable medium), periodontal ligament cells and fibers will not survive, regardless of the stage of root development. Replacement resorption (ankylosis) will probably be the eventual sequela after replantation. Therefore, treatment efforts before replantation include treating the root surface with fluoride to slow the resorptive process.[2,39]

When the patient arrives, the following steps are recommended:

1. Examine the area of tooth avulsion and the radiographs for evidence of alveolar fractures.
2. Remove debris and pieces of soft tissue adhering to the root surface using a dry piece of gauze.
3. Soak the tooth in a 2.4% solution of sodium fluoride (acidulated to pH 5.5) for 5 to 20 minutes.
4. Perform root canal treatment on the tooth while it is held in a fluoride-soaked piece of gauze. Often the procedure can be accomplished from an apical direction if the root is immature.
5. Administer local anesthesia.
6. Suction the alveolar socket carefully to remove the blood clot and irrigate the socket with saline.
7. Replant the tooth gently into the socket, checking for proper alignment and occlusal contact.
8. Splint the tooth for 4 weeks.

Sequelae to Replantation

External root resorption is a frequent occurrence in replanted avulsed teeth. Three types have been identified: surface, inflammatory, and replacement.[36-39,43,44]

Surface Resorption

Surface resorption, also called “repair-related,” is transient and shows as lacunae of resorption in the cementum of replanted teeth. They are not usually visible on radiographs. If resorption does not continue, the lacunae are repaired by deposition of new cementum.

Inflammatory (Infection-Related) Resorption

Inflammatory resorption occurs as a response to the presence of infected necrotic pulp tissue in conjunction with injury to the periodontal ligament. It occurs with replanted teeth (Figure 10-20), as well as with other types of luxation injuries. It is characterized by loss of tooth structure and adjacent alveolar bone. Resorption usually subsides after removal of the necrotic, infected pulp, so the prognosis is good. Root canal treatment is therefore recommended routinely for replanted teeth with closed apexes to prevent the occurrence of inflammatory resorption.

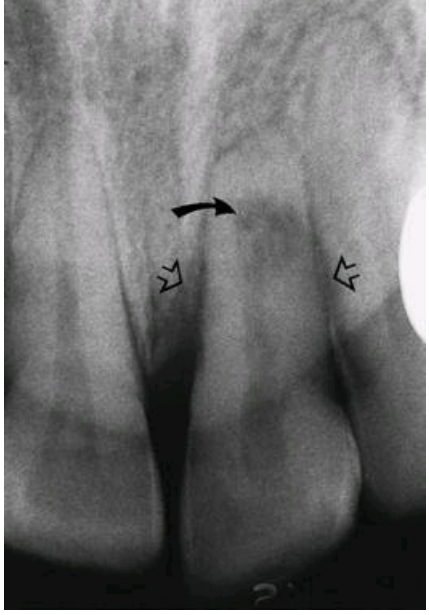

Figure 10-20 Replanted tooth with inflammatory resorption characterized by resorption of tooth structure *(curved arrow)* and a periodontal lesion in the periradicular bone *(open arrows)*. Although the inflammatory resorption appears internal, it is in fact superimposed and is external.

Replacement (Ankylosis-Related) Resorption

In replacement resorption the tooth structure is resorbed and replaced by bone (Figure 10-21), resulting in ankylosis in which bone fuses directly to the root surface. The characteristics of ankylosis are lack of physiologic mobility, failure of the tooth to erupt along with adjacent teeth (leading to infraocclusion in young individuals), and a "solid" metallic sound when percussed. Currently, no known treatment is available for replacement resorption, which tends to be continuous until the root is replaced by bone. In teeth that have had long extraalveolar dry periods, the resorptive process is apparently slowed (but not halted) by immersing the tooth in fluoride before replantation.[12,40,43]

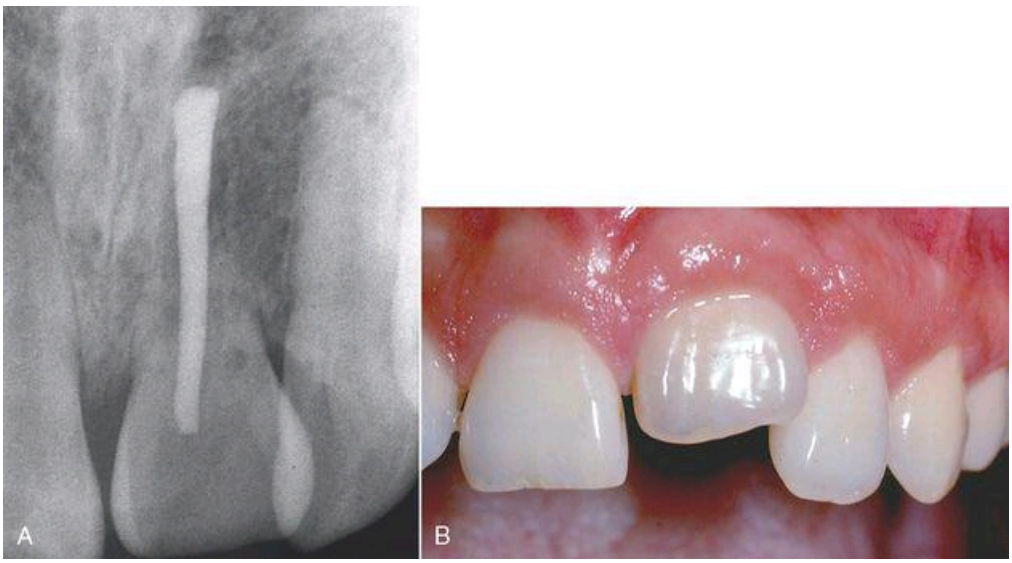

Figure 10-21 Replacement resorption (ankylosis). **A,** The root of this replanted root canal–treated tooth has been almost totally resorbed and has been replaced by bone that fuses to the tooth structure, resulting in ankylosis and resorption. **B,** Tooth is in infraocclusion owing to ankylosis, which prevents normal eruption, as evidenced by continuous eruption of the adjacent teeth.

Root Canal Treatment

When mature avulsed teeth are replanted, they cannot be expected to reestablish pulpal blood supply.[37] Revascularization may occur in immature teeth with wide-open apexes, but it is unpredictable and must be monitored carefully. These teeth must be monitored radiographically over a period of time to watch for evidence of pulp necrosis.

In the mature replanted tooth, root canal treatment is definitely indicated and should ideally be started 7 to 10 days after

replantation. The splint may remain during treatment for stability. The use of calcium hydroxide as an antimicrobial intracanal interappointment medicament may be helpful.[43,45] It is particularly beneficial if the root canal is infected, a condition that would be likely to occur when root canal treatment is delayed more than a few weeks after replantation.

The procedure consists of cleaning and shaping, followed by calcium hydroxide placement for a minimum of 1 to 2 weeks.[45] Obturation is then accomplished with gutta-percha and sealer. Long-term evaluation is necessary to monitor for possible resorption.

Restoration of the coronal access opening, both temporary and permanent, is a key to success. It is important to prevent bacterial leakage into the root canal system. For long-term stability, a dentin-bonding agent with acid-etched composite is indicated.[45]

Alveolar Fractures

Pulp necrosis is often associated with alveolar fractures, which may in turn be associated with other major facial injuries.[6,7] The initial, urgent need is management of the fracture, which is splinting of the segment to the adjacent teeth. Oral and maxillofacial surgeons usually perform this procedure. When the patient is able to have the teeth examined, those in the line of fracture, as well as adjacent teeth, are evaluated. Lack of response to pulp testing, if not reversed within 3 to 6 months, may indicate pulp necrosis, but the presence of other indicators (apical radiolucency or symptoms) is necessary before recommending root canal treatment[46] (Figure 10-22).

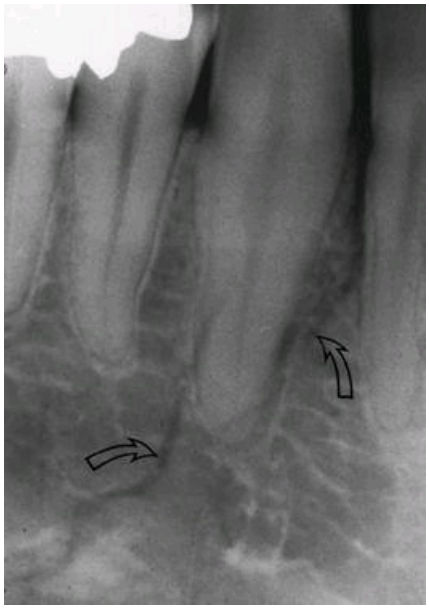

Figure 10-22 Alveolar fracture *(arrows).* Teeth involved in an alveolar fracture should have careful short- and long-term monitoring for pulp responsiveness or other signs of pathosis.

Management of Traumatic Injuries in the Primary Dentition

A traumatic dental injury in a child is always a stressful situation for her or him and the parents or guardians. After clinical and radiographic examination, a careful diagnosis should be made in order to recommend a conservative, biologically based emergency treatment and to avoid taking any risk that may damage the developing permanent successors[12,47,48] (Figure 10-23).

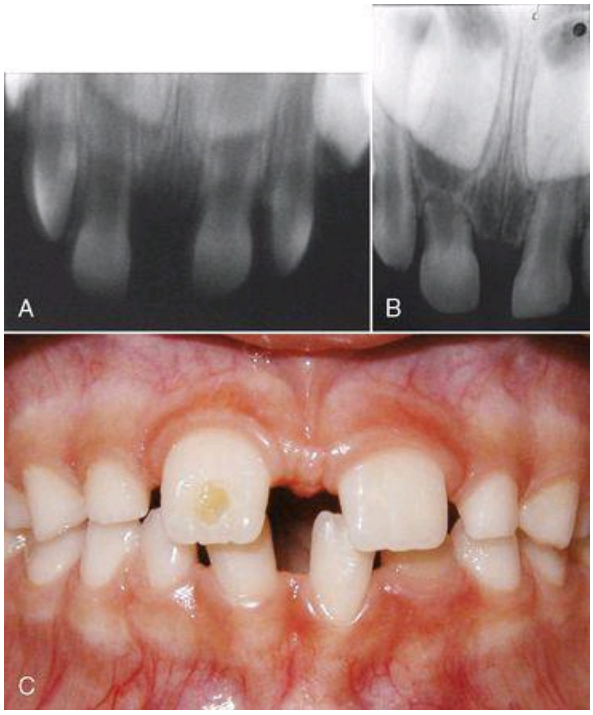

Figure 10-23 Hypoplasia in a permanent incisor following avulsion and subsequent replantation of a primary central incisor. A 1 year-old boy fell and struck his central incisors against a table. The primary right central incisor was avulsed and replanted. The patient followed a 7-day course of amoxicillin. **A,** The x-ray film at the time of injury shows the replanted immature central incisor. **B,** At 3-year 5-month follow-up control, the root has been almost completely resorbed. The contralateral tooth shows complete root formation. **C,** Crown hypoplasia of the right permanent successor at 7-year follow-up control.

Luxation injuries are common in the primary dentition; most of them are left untreated waiting for spontaneous reposition influenced by physiologic forces of the tongue and lips. Injuries requiring emergency care are crown fractures with pulp exposure and those in which displacement occurs, resulting in occlusal interference: root fractures, alveolar fractures, extrusions, and lateral luxations (Figure 10-24). The ability of the dentist to cope with a very young child, which may include the safe use of sedative agents, dealing with the close relationship between the apex of the primary tooth and its developing permanent successor, and the degree of root resorption of the primary tooth are all factors to be considered at the time of selecting an appropriate treatment.[12,47,48]

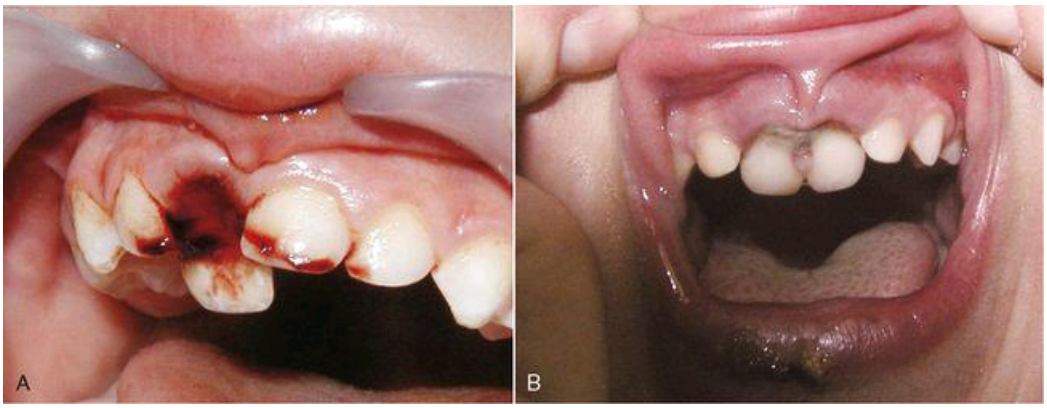

Figure 10-24 Treatment priorities after traumatic injuries in the primary dentition include those that require urgent care to alleviate a child's pain and recover masticatory function. **A,** Severe tooth displacement. **B,** Extrusive luxation of both primary central incisors.

Crown Fractures without Pulp Exposure

Crown fractures become urgent care when very young children have broken teeth resulting in sharp edges. Primary teeth may be restored with glass ionomer or composite, or the fracture sites may be smoothed without restoring them.

Crown Fractures with Pulp Exposure

A crown fracture with pulp exposure is a difficult emergency situation, especially when a very young child is affected. Treatment includes partial pulpotomy with calcium hydroxide, pulpotomy, or extraction, depending on the patient's age and cooperation. If vital pulp therapy is possible, the fractured crown may be restored with composite resins.

Crown-Root Fractures

A crown-root fracture in primary teeth usually exposes the pulp and extraction is indicated.

Root Fractures

Removing the coronal segment and leaving the root apex in situ is the treatment of choice for root fractures with marked coronal displacement. Any attempt to remove the root apex may damage the subjacent permanent tooth bud. Root fractures not accompanied by mobility usually require no treatment unless problems develop subsequently.

Alveolar Fractures

Alveolar fractures are severe injuries that may dictate treatment under general anesthesia. The displaced segment should be repositioned and splinted to adjacent teeth for up to 4 weeks.

Luxation Injuries

Concussion and subluxation injuries require no treatment, other than promoting good oral hygiene to prevent healing complications. Crown color changes are usually the main complaint for seeking treatment. Because it has not been possible to relate discolored teeth to pulpal status,[49-51] persistent gray discoloration of the crown is not considered an indication for root canal treatment,[52] unless a sinus tract or an abscess develops. Discolored primary teeth may return to normal color, probably indicating recovery of the pulp (Figure 10-25). Pulp canal obliteration is common after luxation injuries.[53] This changes the primary crown to a darker yellow color, which is not pathologic and requires no treatment.

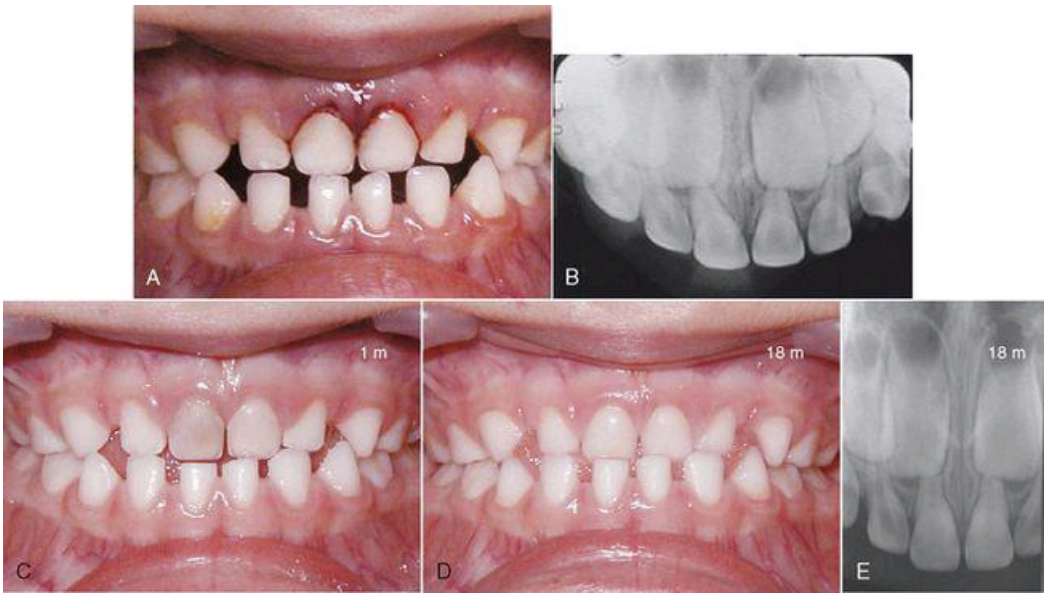

Figure 10-25 Color changes in primary teeth following subluxation injury. A 5-year-old girl fell in the playground and struck her front teeth against the floor. **A,** The girl arrived in the clinic within an hour for clinical examination. Both central incisors were mobile but not displaced. Bleeding from the gingival crevice was observed. Because of minor occlusal interference, slight grinding of the opposite teeth was performed. Oral hygiene instructions were given to the mother recommending the topical use of chlorhexidine and avoiding the use of pacifier. **B,** The x-ray film at the time of injury shows no radiographic changes. **C,** After 1 month, there is no occlusal interference, but gray discoloration is seen in both central incisors. **D,** At 18-month follow-up control, the color of the crowns has returned to normal. **E,** The periapical radiograph shows pulp canal obliteration in both traumatized teeth.

Teeth with lateral and extrusive luxations may be left untreated, may be repositioned if there is occlusal interference, or may be extracted, depending on the severity of injury.

Teeth with intrusive luxation should be carefully evaluated to determine the direction of intrusion. Radiographs provide valuable information to confirm the intruded position of the tooth and its proximity to the permanent successor. If the intruded tooth appears foreshortened on the film, the apex is oriented toward the x-ray cone. Therefore these teeth should present no danger to the permanent successor and may be left to reerupt. If the tooth appears elongated, the apex is oriented toward the permanent successor and may pose a risk to the permanent tooth bud. The tooth should be carefully extracted if it impinges on the permanent successor. The permanent tooth buds should also be evaluated for symmetry.[47]

Avulsions

Replantation of avulsed primary teeth is not recommended because of the risk of damage to the permanent successor (see Figure 10-23).[12] Severe impacts resulting in avulsions may cause damage at the time of injury; therefore treatment that may inflict an additional injury to the succedaneous teeth must be avoided. Furthermore, the parents or guardians should be asked to bring the avulsed tooth to ensure that the tooth is not intruded (Figure 10-26). A radiographic examination will confirm tooth avulsion and will reveal the stage of development of the permanent tooth bud.

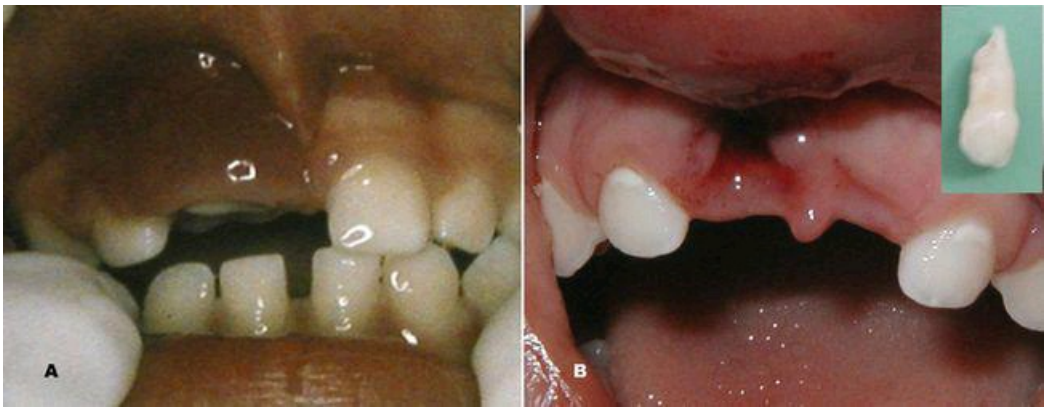

Figure 10-26 Importance of accurate diagnosis after intrusions and avulsions in the primary dentition. **A,** Intrusive luxation. A maxillary right central incisor has been deeply intruded in the alveolar socket, giving the appearance of having been avulsed. **B,** Avulsion. The avulsed tooth was brought into the clinic, confirming that it was not intruded.

Patient Instructions

Parents or guardians should receive information on how to brush their children's teeth after an injury. Careful oral hygiene after each meal, in addition to topical use of chlorhexidine twice a day for 1 week, will keep plaque away. Also, restricted use of pacifiers is indicated.

Chapter Review Questions available in Appendix B or on the DVD

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Endodontic Radiography

Richard E. Walton

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe the importance of radiographs in endodontic diagnosis and treatment.

2. Discuss special applications of radiography to endodontics.

3. Discuss reasons for limiting the number of exposures.

4. Identify normal anatomic features in the maxilla and mandible on radiographs.

5. Describe radiographic characteristics to differentiate between endodontic and nonendodontic (normal and pathologic) radiolucencies and radiopacities.

6. Describe the reasons for varying horizontal and vertical cone angulations on working radiographs to create image shift.

7. Describe how to determine the third dimension on angled radiographs (i.e., faciolingual structures [SLOB] rule).

8. Describe structural elements of the tooth as visualized on both facial and angled projections.

9. Discuss how to detect the presence of and locate undiscovered canals or roots on angled working radiographs.

10. Describe techniques for making “working” radiographs (i.e., film placement and cone alignment with rubber dam in place).

11. Describe specific details of film placement and cone alignment for each tooth on working radiographs.

12. Describe the limitations of rapid processing of working films.

13. Describe the radiographic technique for locating a “calcified” canal.

14. Discuss the limitations of radiographic interpretation.

15. Describe some new technologies and their application to endodontic radiography now and in the future.

16. Describe the technique for extraoral positioning of the film and cone.

> We are sick of the roentgen ray … you can see other people’s bones with the naked eye, and also see through eight inches of solid wood. On the revolting indecency of this there is no need to dwell. But what we seriously put before the attention of the Government … that it will call for legislative restriction of the severest kind. Perhaps the best thing would be for all civilized nations to combine to burn all works on the roentgen rays, to execute all the discoverers, and to corner all the tungstate in the world and whelm it in the middle of the ocean.
> EDITORIAL IN *PALL MALL GAZETTE,* LONDON, 1896

Obviously (and fortunately), the concern expressed by the editorial in this London publication did not become the popular view of radiography. Radiographs are essential; they are a second set of “eyes” for the dentist. This is particularly true in endodontics, in which so many diagnostic and treatment decisions are based on radiographic findings. Because most structures of concern are not visible to the naked eye, there is considerable dependence on radiographs, which are an obvious necessity and a blessing. But they also may be somewhat of a liability from the standpoint of both safety and time, and unfortunately, radiographs are often overinterpreted or underinterpreted.

A radiographic exposure is an irreversible procedure, and therefore only necessary exposures should be made. With the increasing emphasis and justifiable concern for radiation safety, overall radiation exposure must be minimized.[1] However, the amount of radiation dosage to oral and other tissues has been calculated to be very low and cause minimal (but some) risk.[2,3]

Another concern is the time required to make and process individual radiographs—time is money. Therefore, in the interests of both safety and time, only the radiographs necessitated by the procedure should be made.

This chapter discusses radiography as applied to endodontic procedures. Radiography as a discipline in dentistry has become increasingly important with advances in technology and has recently been granted specialty status, thereby replacing endodontics as the youngest dental specialty.[4] Technology has exploded in recent years, with new devices and approaches that require special training and experience. How these new devices and approaches apply to diagnosis and treatment in endodontics is discussed later in this chapter.

IMPORTANCE OF RADIOGRAPHY IN ENDODONTICS

Radiographs perform essential functions in three areas. However, they have limitations that require special approaches. A single radiograph is but a 2-dimensional shadow of a 3-dimensional object. For maximum information, the third dimension must be visualized and interpreted.[5] The three general areas of application are diagnosis, treatment, and recall; each requires its own special approach.

Digital Radiography

Digital radiography is increasingly becoming more common in dentistry. Although there are technical advantages over conventional approaches, the same limitations apply. Overall, digitized radiographs are equivalent to conventional radiographs as to diagnostic interpretation. These factors, as well as other considerations, will be further discussed throughout this chapter.

Diagnosis

Diagnostic radiology involves not only identifying the presence and nature of pathosis but also determining root and pulp anatomy and characterizing and differentiating other normal structures.

Identifying Pathosis

Radiographs must be studied carefully by someone with a working knowledge of the changes that indicate pulpal, periapical, periodontal, or other bony lesions. Many changes are obvious, but some are subtle.

Determining Root and Pulpal Anatomy

Determining the anatomy involves not only identifying and counting the roots and canals but also identifying unusual tooth anatomy, such as dens invaginatus and C-shaped configuration,[6] and determining curvatures, canal relationships, and canal location.[7,8] Identification also includes characterizing the cross-sectional anatomy of individual roots and canals (Figure 11-1).

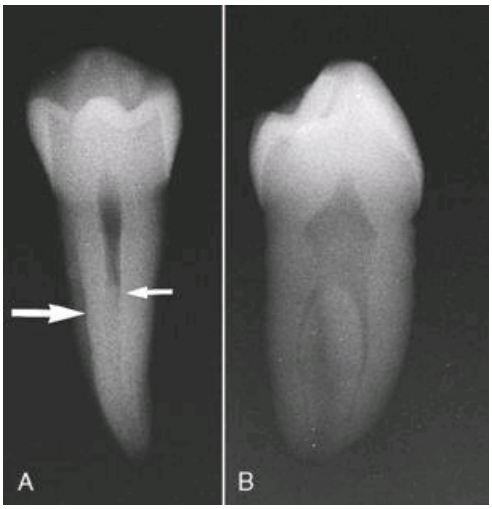

Figure 11-1 A, The facial projection of this premolar gives some limited information about pulp/root morphology. "Fast break" *(small arrow)* usually indicates canal bifurcation. A double root prominence on the mesial surface *(large arrow)* indicates two bulges and a concavity; its absence on the distal surface indicates a flat or convex root surface. **B,** The same premolar from the proximal view. The presence of two definitive canals, each in its own "root bulge," is confirmed.

Characterizing Normal Structures

Numerous radiolucent and radiopaque structures often lie in close proximity. Frequently, these structures are superimposed over and obscure crowns and roots.[9] These must be distinguished and differentiated from pathosis and from dental anatomy.

Treatment

"Working" radiographs are made while the rubber dam is in place, thereby creating problems in film placement and cone positioning. These radiographs are exposed *during* the treatment phase and have special applications.

Determining Working Lengths

Distance from a reference point to the radiographic apex is determined precisely. This establishes the distance from the apex at which the canal is to be prepared and obturated.[10]

Moving Superimposed Structures

Radiopaque anatomic structures often overlie and obscure roots and apexes. By using special cone angulations, these radiopaque structures can be "moved" to give a clear image of the apex.

Locating Canals

Canal location is obviously essential to success. Standard and special techniques allow the practitioner to determine the position of canals not located during access.

Differentiating Canals and Periodontal Ligament Spaces

Canals end in the chamber and at the apex. A periodontal ligament space ends on a surface and in a furcation (molars) and demonstrates an adjacent lamina dura (Figure 11-2).

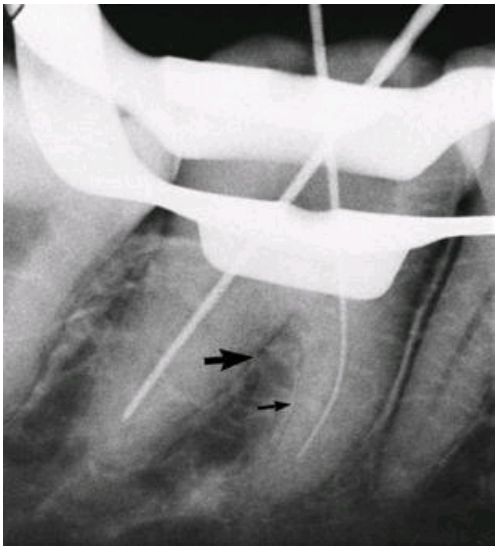

Figure 11-2 This distal angulation shows the root surface outline *(large arrow)* and a periodontal ligament space *(small arrow)* with an adjacent lamina dura. The file is in the mesiobuccal canal (same lingual, opposite buccal [SLOB] rule).

Evaluating Obturation

Length, density, configuration, and the general quality of obturation in each canal are determined.

Recall

Ultimate success is verified at specified intervals of months or years after treatment. Because failures often occur without signs or symptoms, radiographs are essential to evaluate periapical status.[11]

Identifying New Pathosis

The presence and nature of lesions that have arisen after treatment are best detected on radiographs. These lesions may be periapical, periodontal, or nonendodontic. Importantly, such lesions frequently present with no overt signs or symptoms and are detectable only on radiographs (Figure 11-3).

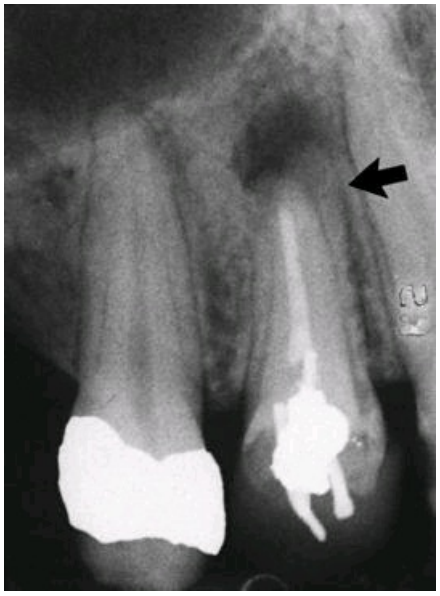

Figure 11-3 Failed root canal treatment because of missed root or canal. This mesial-angled radiograph clearly shows the untreated lingual root *(arrow).*

(Courtesy Dr. L. Wilcox.)

Evaluating Healing

Pretreatment lesions should be resolving or should have resolved. In a successful (healed) treatment, restitution of generally normal structures should be evident on recall radiographs (Figure 11-4).

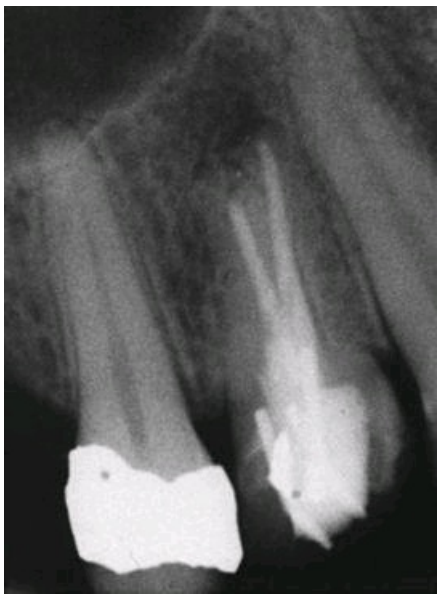

Figure 11-4 Same tooth as shown in Figure 11-3. Recall radiograph after 9 months shows almost complete regeneration of bone, indicating a healing lesion. A permanent restoration must be placed as soon as possible.

(Courtesy Dr. L. Wilcox.)

Special Applications

Radiographs should be used to their maximum advantage. There are alternative techniques that greatly enhance the ability to make an accurate and definitive diagnosis and to control treatment procedures. Although these techniques may be applied to disciplines other than endodontics, the following do apply and are essential to diagnosis and treatment.

Cone-Image Shift

Varying either the vertical or, particularly, the horizontal cone angulation from parallel alters images and enhances interpretation.[5,12] These shifts reveal the third dimension and superimposed structures. Shifts also permit identification and positioning of objects that lie in the faciolingual plane.

Working Films

Working films are essential aids to treatment and are exposed when necessary but with discretion.

RADIOGRAPHIC SEQUENCE

Radiographs are made in a recommended order and number for each procedure. The minimum number are described here, although special situations may require extra exposures.

Number

The number of exposures depends on the situation. For diagnosis in most cases, only a single exposure is necessary. A properly positioned film and cone (usually a parallel projection is best) permits visualization at least 3 to 4 mm beyond the apex. The initial diagnostic film is used primarily to detect pathosis and to provide general information on root and pulp anatomy. Usually, it is not necessary at this time to make additional films for identification of extra canals because this will be accomplished later with shifted working length radiographs.

Frequently, several films are available for study (e.g., if a full mouth survey has been taken). If other films are at hand, each will give a slightly different view of the same tooth (Figure 11-5). *Examine the tooth on each film in which it appears.*

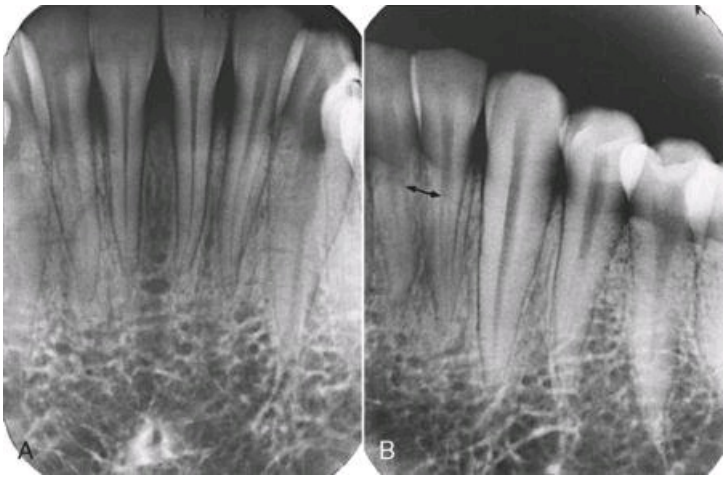

Figure 11-5 A, Facial projection of incisors suggests a single canal and a single root. **B,** Distal (canine) projection gives a different perspective. The canals of the lateral and central incisors are seen to bifurcate in the middle third of the root *(arrow)* and reunite in the apical third.

Angulation

Unquestionably, the most accurate radiographs are made using a paralleling technique.[13] The advantages are (1) less distortion and more clarity and (2) reproducibility of film and cone placement with preliminary and subsequent radiographs. Reproducibility is important when assessing whether changes occurring in the periapex indicate healing or nonhealing. Paralleling devices enhance reproducibility.

There may be special situations in which the paralleling technique is not feasible, such as a low palatal vault, maxillary tori, exceptionally long roots, or an uncooperative or gagging patient, and these may necessitate an alternative technique. A second choice is the modified paralleling technique; the least accurate technique is the bisecting angle.

Working Films

Special situations require special considerations. Although the basic principles of doing everything possible to obtain the best quality radiograph are followed, there are definite limitations in making working films. These require cooperation by the patient if they hold the film in position.

These radiographs are usually neither parallel nor bisecting angle. The technique used is called *modified paralleling.*[14] Essentially, the film is not parallel to the tooth, but the central beam is oriented at right angles to the film surface. In endodontic working films, a further modification is made by varying the horizontal cone angle. Specific details of film and cone placement and film interpretation are discussed later in this chapter.

Working Length

Generally, establishment of working length should require only a single exposure. If a root contains or may contain two superimposed canals, either a mesial or distal angle projection is absolutely necessary; the straight facial view is not particularly helpful.[15] Additional working length films may be required later for confirmation of working lengths to detect the presence or lengths of newly discovered canals (Figure 11-6), or for reexposure if an apex has been cut off in the first radiograph.

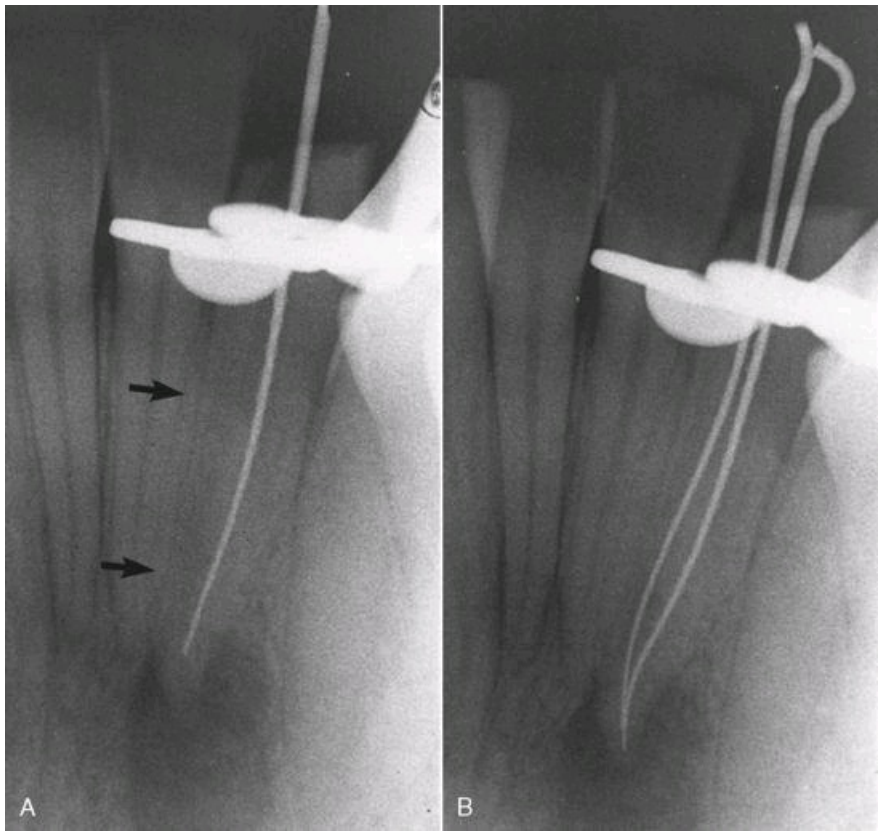

Figure 11-6 Identifying and locating a canal. This incisor was rotated, requiring mesially angled working radiographs. **A,** The file is off-center as indicated by the mesial root surface *(arrows)*. Therefore the file is in the facial canal. **B,** A search to the lingual locates the lingual canal. There is a common canal apically.

Master Cone

The same principles used with working length films apply. With proper technique, only one radiograph is necessary to evaluate the length of the master gutta-percha cone.

Exposure and Film

As with diagnostic films, adequate clarity (and decreased exposure) is achieved by using an E film at intermediate kilovoltage.[16] Clarity is particularly important when trying to visualize the tips of files or small apexes to determine working lengths. The F film, very recently introduced, requires 20% to 25% less exposure than E film. There are no studies yet as to quality and usefulness of this new film type.

Other Considerations

Additional working films are often required. For example, they are useful as aids in locating a canal or in determining the occurrence of procedural accidents (perforations, separated instruments, or ledges). Variations in cone positioning and angulation are made as required.

Obturation

The same basic principles used for diagnostic radiographs apply. At least a parallel projection should be made. It may be desirable to supplement this with an angled film to visualize separate superimposed canals for separate evaluation of each. The exposure factors used for diagnostic radiographs are duplicated for obturation. However, the radiograph gives only a rough indication of obturation length and quality.[17,18]

Recall

The same principles used for diagnostic and obturation radiographs (parallel projection and exposure factors) apply to recall radiographs. There is one exception. If treatment is deemed to be questionable or a failure, additional angled radiographs are often required to search for a previously undetected canal or other abnormality.

EXPOSURE CONSIDERATIONS

Proper x-ray machine settings and careful film processing are important for maximal quality and interpretative diagnostic and working radiographs. Both D (Ultraspeed) and E (Ektaspeed) films have been used and compared. Although D film has been shown to have slightly better contrast, overall suitability is equivalent between the two film types.[19] The newer Ektaspeed Plus film produces an image similar in quality to Ultraspeed film but requires only half the radiation of Ultraspeed.[20]

The optimal setting for maximal contrast between radiopaque and radiolucent structures is 70 kV. Exposure time and milliamperage should be set individually on each machine. Therefore, the preferred film types are E and Ektaspeed Plus to minimize x-irradiation at 70 kV to maximize clarity.

Digital radiographs require much less exposure time than conventional radiographs, which is a definite advantage.

CONE-IMAGE SHIFT

The cone-image shift reveals the third dimension.

Principles

Image Shift

Superimposed Structures

The cone-image shift technique separates and identifies the facial and lingual structures.[5] An example is the mesiobuccal root of a maxillary molar that contains two superimposed canals. The cone shift separates and permits visualization of both canals.

Faciolingual Determination

Principles of relative movement of structures and film orientation are applied to the differentiation of object position (Figures 11-7 and 11-8).

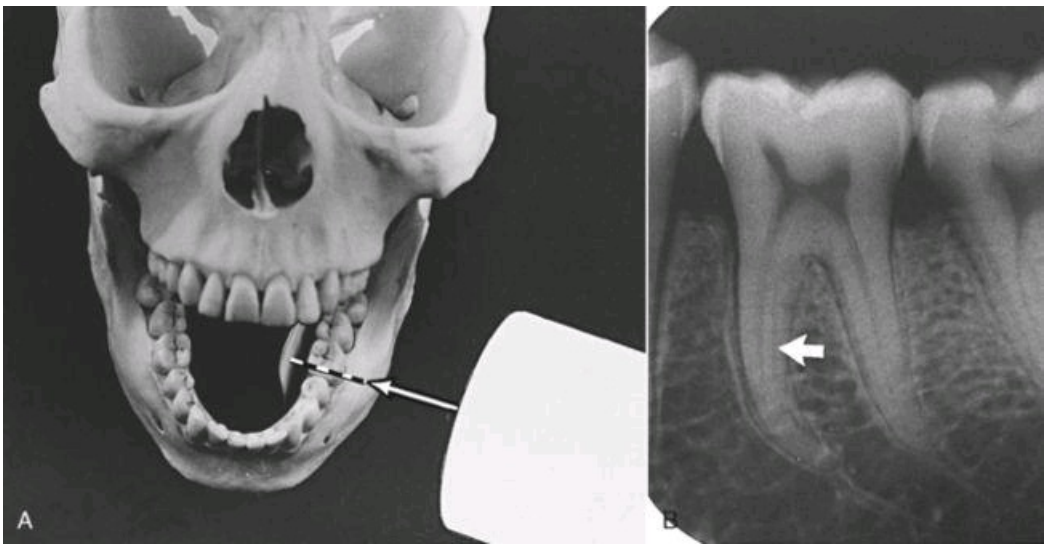

Figure 11-7 A, The film is positioned parallel to the plane of the arch. The cone has the central ray *(arrow)* directed toward the film at right angles. This is the basic cone-film relationship used for horizontal or vertical angulations. **B,** There is a clear outline of the first molar but limited information about superimposed structures (canals that lie in the buccolingual plane). The *arrow* points to a periodontal ligament space adjacent to a superimposed root bulge, not to a second canal.

(From Walton R: Dent Radiogr Photogr 46:51, 1973.)

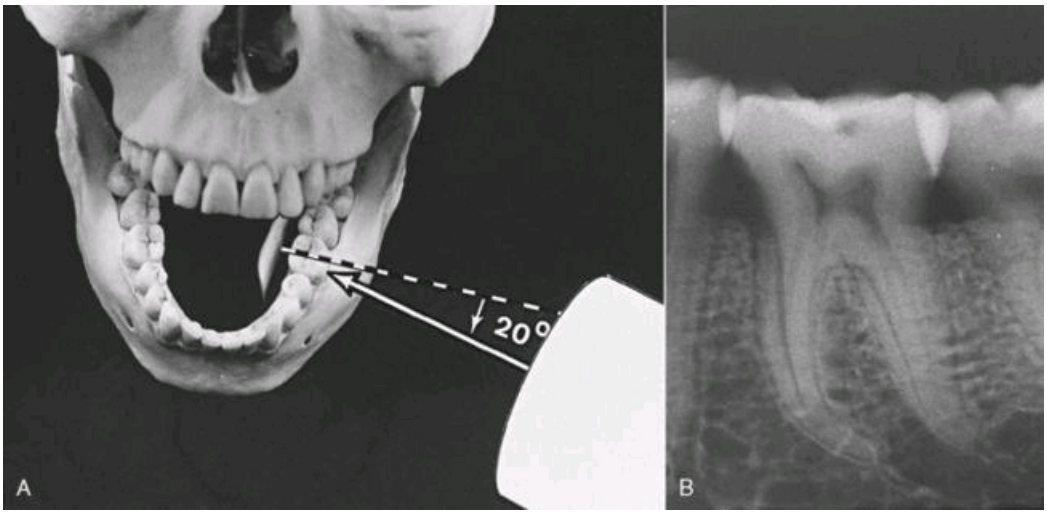

Figure 11-8 A, The horizontal angulation of the cone is 20-degrees mesial from the parallel, right-angle position (mesial projection). **B,** The resultant radiograph demonstrates the morphologic features of the root or canal in the third dimension. For example, two canals are now visible in the distal root of the first molar.

(From Walton R: Dent Radiogr Photogr 46:51, 1973.)

SLOB Rule

As the cone position moves from parallel, whether toward the horizontal or toward the vertical, the objects on the film shift away from the direction of the cone (or in the direction of the central beam). In other words, when two objects and the film are in a fixed position and the radiation source (cone) is moved, images of both objects move in the opposite direction (Figure 11-9). The facial (buccal) object shifts farthest away; the lingual object shifts less. The resulting radiograph shows a lingual object that moved relatively in the same direction as the cone and a buccal object that moved in the opposite direction.[21] This principle is the origin of the acronym SLOB (same lingual, opposite buccal) (Figure 11-10).

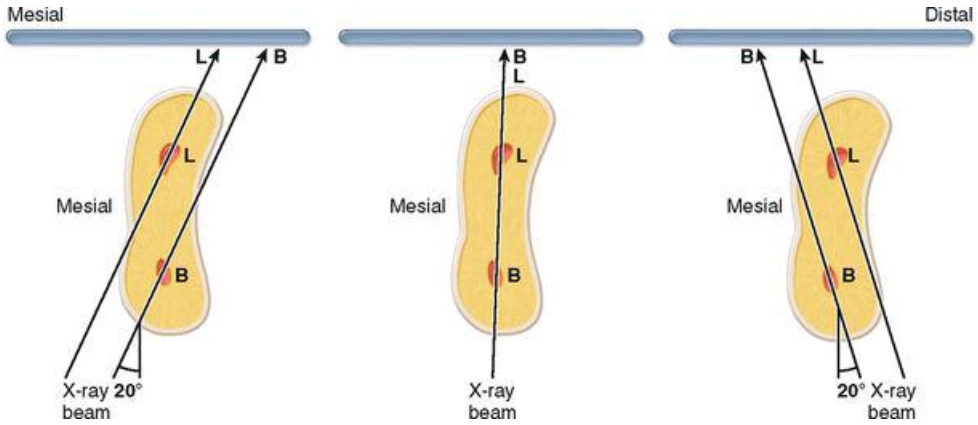

Figure 11-9 Central (x-ray) beam passing directly through a root containing two canals will superimpose the canals on the film. When the cone is shifted to the mesial or distal aspect, the lingual object will move in the same direction as the cone; the buccal object will move in the opposite direction (SLOB rule).

(Courtesy Dr. A. Goerig.)

Figure 11-10 The SLOB rule.

One way to visualize this is to close one eye and hold two fingers directly in front of the open eye so that one finger is superimposed on the other. By moving the head one way and then the other, the position of the fingers relative to each other shifts. The same effect is produced with two superimposed roots (the fingers) and the way in which they move relative to the radiation source (the eye) and the central beam (the line of sight). When the cone-shift technique is used, it is critical to determine what is facial and what is lingual. Otherwise, serious errors may occur.

Indications and Advantages

Separation and Identification of Superimposed Canals

Separation and identification of superimposed canals is necessary in all teeth that may contain two canals lying in a faciolingual plane.

Movement and Identification of Superimposed Structures

Occasionally, a radiopaque object may overlie a root. An example is the zygomatic process, which often obscures the apexes of maxillary molars. Because this dense structure lies facial to the roots, a mesial shift of the cone "pushes" the zygoma distally (Figure 11-11). In addition, a decrease in vertical angulation of the cone "pushes" the zygoma superiorly.

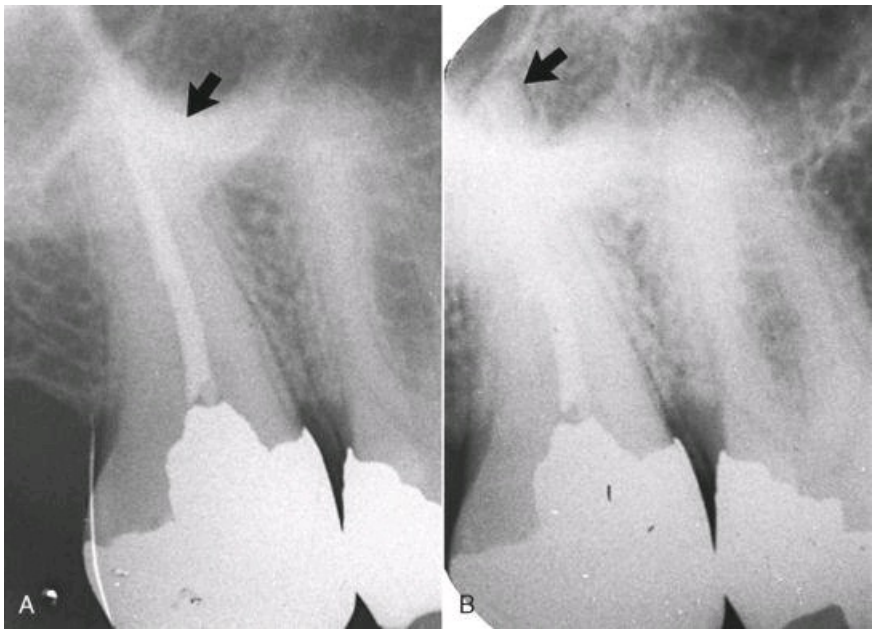

Figure 11-11 A, Malar process of maxillary zygoma *(arrow)* obscures the apex and blocks the view of the obturation. **B,** Slight mesial shift of the cone "pulls" the lingually positioned root apex *(arrow)* to the mesial for visibility.

Determination of Working Length

Individual superimposed canals may be traced from orifice to apex (Figure 11-12).

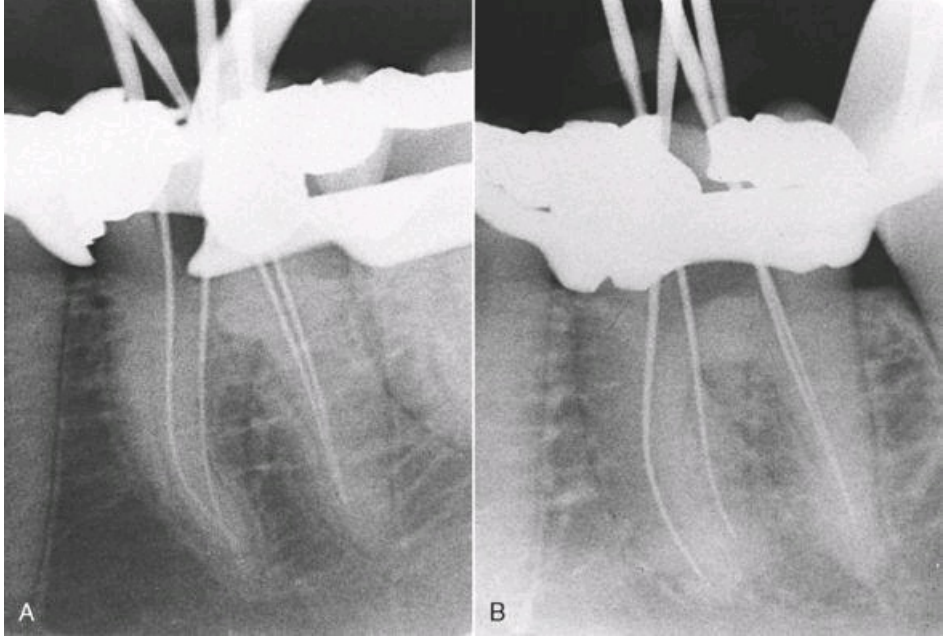

Figure 11-12 A, Mesial projection gives limited information about morphologic features and relationship of four canals. **B,** Correct distal projection for mandibular molars "opens up" roots. Mesial canals are easily visualized for their entire length. The distal canal is a single wide canal because instruments are close and parallel.

Determination of Curvatures

The SLOB rule applies when determining curvatures. Depending on the direction of movement of the curvature relative to the cone, it can be determine whether this curvature is facial or lingual. The severity of the curvature can also be determined.

Determination of Faciolingual Locations

The SLOB principle is applied to locating something on a root surface or within a canal. One example might be the site of a perforation: to which surface does it extend, facial or lingual? Two radiographs at different horizontal angles readily disclose this (Figure 11-13).

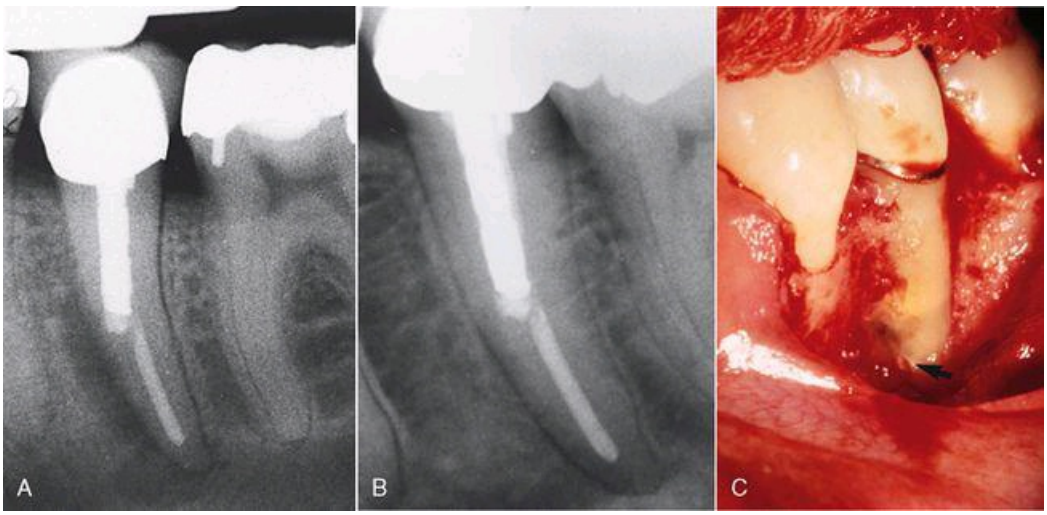

Figure 11-13 A, A perforation of the post preparation is indicated by the mesial lesion, although the perforation is not visible on this facial projection. The perforation will be visible toward the buccal or lingual aspect and revealed on an additional angled radiograph. **B,** The tip of the post has moved slightly distal on this mesial projection, and the perforation is therefore located toward the buccal aspect (SLOB rule). **C,** The perforation site *(arrow).*

Identification of Undiscovered Canals

The SLOB procedure applies during access. An anatomic axiom is that *if a root contains only a single canal, that canal will be positioned close to the center of the root.* If a single canal is discovered initially on access preparation, an instrument is placed in the canal. Then a radiograph *must* be made either mesial or distal because another canal may be present. If the instrument is skewed considerably off center, another canal must be present (Figure 11-14). The location of the missed canal is determined by applying the SLOB rule.

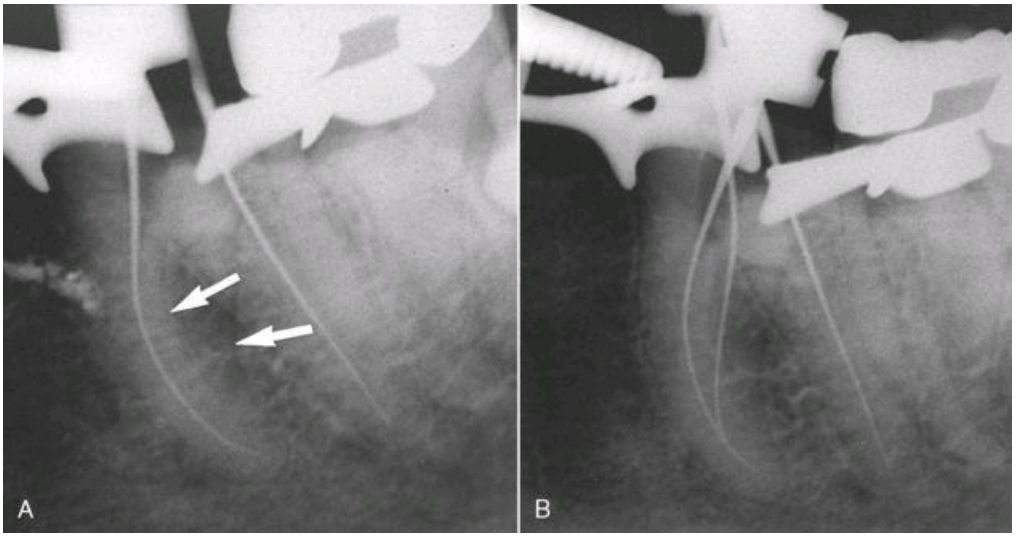

Figure 11-14 Technique for locating a canal missed during access preparation and searching of the chamber. **A,** Distal radiograph with a single file in the mesial root shows the file skewed buccally. Therefore another canal would be located toward the lingual aspect. The vertical radiolucent lines *(arrows)* are periodontal ligament spaces of the mesial root. **B,** A careful search toward the lingual aspect reveals the canal.

Location of "Calcified" Canals

Locating "calcified" canals also applies during access preparation. Another endodontic anatomic axiom is that *a root always contains a canal*. The canal may be very tiny, or it may be difficult or impossible to find or negotiate, but it is present. Also, canals are frequently not visible on radiographs. A single canal will lie in the center of the root. Therefore, when searching for an elusive canal by penetrating progressively deeper with a bur, occasionally two working radiographs must be made. One is made from the straight facial view and the other from either the mesial or distal view. The straight facial radiograph gives the mesiodistal location of the bur penetration; the mesial- or distal-angled radiograph indicates the faciolingual angulation of the bur. The direction is adjusted accordingly toward the center of the root where the canal surely lies (Figure 11-15).

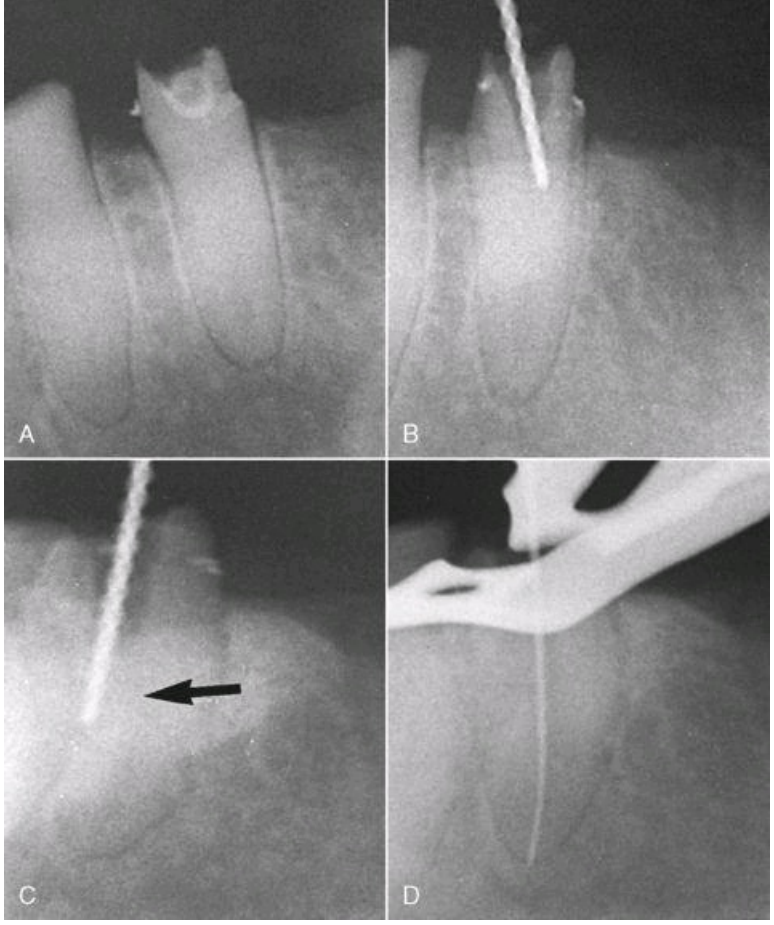

Figure 11-15 Location of a canal that has undergone severe calcific metamorphosis. Initial searching is done without a rubber dam. **A,** A small, receded canal and missing crown make orientation and canal search difficult. **B,** Facial radiograph taken during access shows that preparation is mesial to the canal. (Remember the canal occupies the center of the root.) **C,** Mesial radiograph shows that access is also misdirected to the buccal aspect; the canal will be centered *(arrow).* Therefore the subsequent search must be to the distal and lingual aspects. **D,** On redirecting the bur, the single canal is discovered in the center of the root. Now the rubber dam is placed.

Disadvantages

The cone-image shift has inherent problems and therefore on occasion should not be used or the angulation of the cone should be minimized.

Decreased Clarity

The clearest radiograph with the most definition is a parallel or modified parallel projection.[22] When the central beam changes direction relative to object and film (passing through the object and striking the film at an angle), the object becomes blurred. Distinctions between radiolucent and radiopaque objects show less contrast. This blurred or fuzzy appearance increases as the cone angle increases, and other structures are more likely to be superimposed. Therefore, for maximum clarity, the cone angle should deviate only to the extent necessary to obtain sufficient shift for interpretive purposes.

Superimposition of Structures

Objects that ordinarily have a natural separation on parallel radiographs may, with cone shift, move relative to each other and become superimposed. One example is the roots of a maxillary molar. A parallel radiograph generally shows three separate roots and separate apexes. A mesial-or distal-angled radiograph moves the palatal root over the distobuccal or mesiobuccal root, decreasing the ability to distinguish the apexes clearly (Figure 11-16). Another example is an increase in the vertical angulation of the cone in the maxillary incisor region; this may “pull” the apexes “into” the radiodense anterior nasal spine.

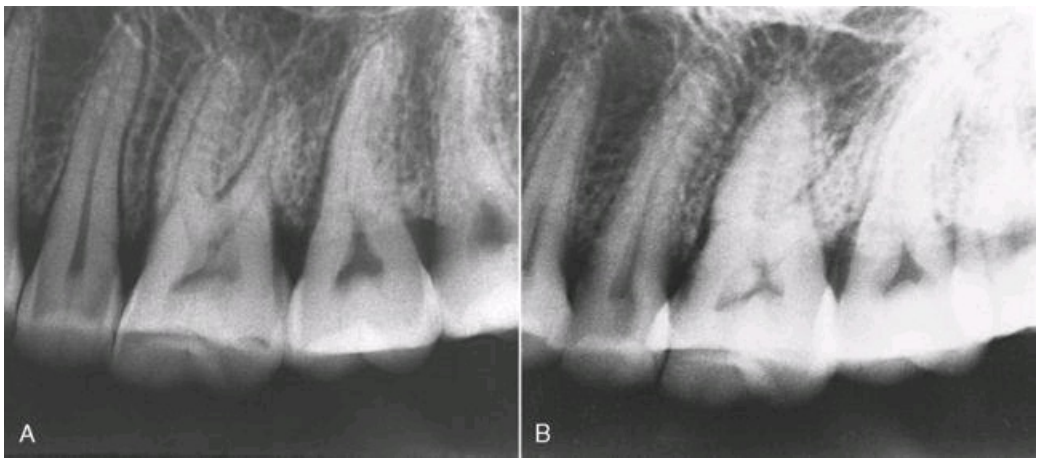

Figure 11-16 A, Facial parallel projection shows maximum clarity on the first molar. **B,** Mesial shift of 30 degrees reduces contrast and the distinction between radiopaque and radiolucent objects. Also, roots are now superimposed, making interpretation more difficult.

ENDODONTIC RADIOGRAPHIC ANATOMY

Interpretation

Radiographs can be termed *the great pretenders*—they often are as misleading as they are helpful.[23,24] There is a definite tendency to try to extract more information from a radiograph than is present. The practitioner must remember that only hard tissues, not soft tissues, are visible.

Limitations

Studies of interpretation of bony lesions have shown that considerable bone must be resorbed before the lesion is clearly visible.[25,26] This, of course, varies with root location and thickness of the overlying cortical bone. In most regions, a periradicular lesion tends to be most evident radiographically if cortical bone has resorbed. However, resorption of only medullary bone may be sufficient for visualization.[27,28] In either case, a periradicular inflammatory lesion must be well developed and fairly extensive before an obvious radiolucency can be seen.

DIFFERENTIAL DIAGNOSIS

Endodontic Pathosis

Radiolucent Lesions

Radiolucent lesions have the following four distinguishing characteristics that aid in differentiating them from nonendodontic pathoses (Figure 11-17):

1. Apical/radicular lamina dura is absent, having been resorbed.

2. A "hanging drop of oil" shape is characteristic of the radiolucency, although this is a generalization because these lesions may have a variety of appearances.

3. The radiolucency "stays" at the apex regardless of cone angulation.

4. A cause of pulpal necrosis is usually (but not always) evident.

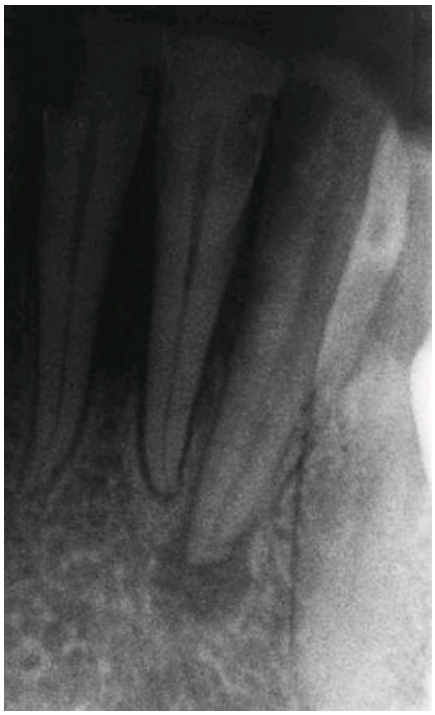

Figure 11-17 Characteristics of apical radiolucency strongly suggest endodontic pathosis. Lamina dura is not present, and the lesion has a "hanging drop of oil" appearance. The cause of pulpal necrosis is also evident.

A common concept is that an endodontic granuloma can be distinguished from a radicular cyst. The supposed differential is that the cyst is outlined radiographically by a "corticated" or radiopaque lamina. It has been demonstrated that this is *not* a reliable indicator.[29]

The ultimate differentiation is not the radiograph but the pulp test. If a developed, sizable radiolucency is an endodontic lesion, it *must* result from a necrotic (hence nonresponsive) pulp.

Radiopaque Lesions

Radiopaque lesions are better known as condensing osteitis. Such lesions have an opaque diffuse appearance, and histologically they represent an increase in trabecular bone.[30] The radiographic pattern is one of diffuse borders and a roughly concentric arrangement around the apex (Figure 11-18). Pulpal necrosis and a radiolucent inflammatory lesion may or may not be present. Frequently, condensing osteitis and apical periodontitis are present together. The pulp is often vital and inflamed.

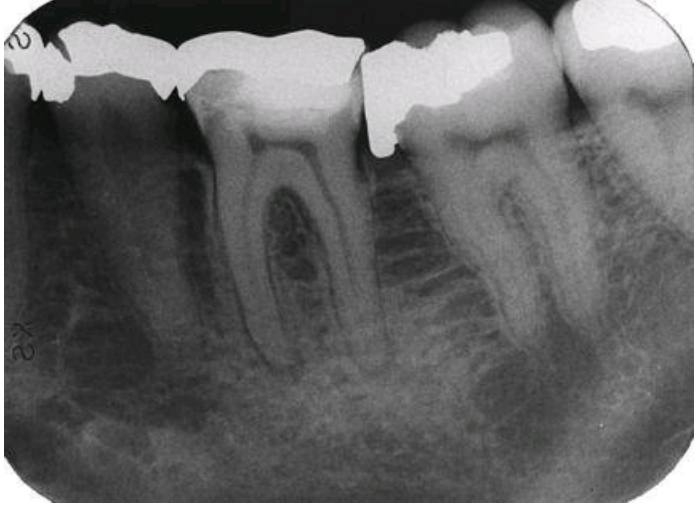

Figure 11-18 Condensing osteitis. There is diffuseness and a concentric arrangement of increased trabeculation around the apex. Close inspection shows a radiolucent lesion at the apexes also.

Nonendodontic Pathosis

Radiolucent Lesions

Radiolucent lesions are varied but infrequent. Bhaskar lists 38 radiolucent lesions of the jaws, 35 of which are nonendodontic and have a variety of configurations and locations,[31] and many are positioned at or close to the apexes and radiographically mimic endodontic pathosis. Again, the pulp test provides the cardinal differentiation—nonendodontic lesions are associated with a responsive tooth.

Radiopaque Lesions

Frequently, interpretive errors are made in identifying radiopaque structures located in the apical region of the mandibular posterior teeth. Unlike condensing osteitis, these are not pathologic and have a more well-defined border and a homogeneous structure. They are not associated with pulpal pathosis (Figure 11-19).

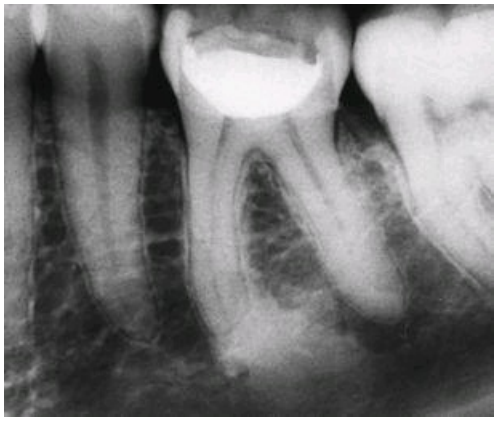

Figure 11-19 Enostosis (or sclerotic bone) is represented by the dense, homogeneous, defined radiopacity. This is not pathosis and is common in the posterior mandible near the apexes, although it may occur in any region. This radiodense area would have appeared on earlier radiographs.

Anatomic Structures

Several anatomic entities are superimposed on or may be confused with endodontic pathosis. Although most radiology courses cover identification of these structures, it is not uncommon for a practitioner to fail to identify these normal structures when there is an existing or suspected endodontic problem. Common sources of confusion are the areas created by sparse trabecular patterns, particularly in the mandible. Another problem area is the apical region of the maxillary anterior teeth. One must remember to look *through* these radiolucencies for an apical lamina dura.

Mandible

The classic example of a radiolucency that may overlie an apex is the mental foramen over a mandibular premolar.[32] This is easily identified by noting movement on angled radiographs and by identifying the lamina dura (Figure 11-20).[33]

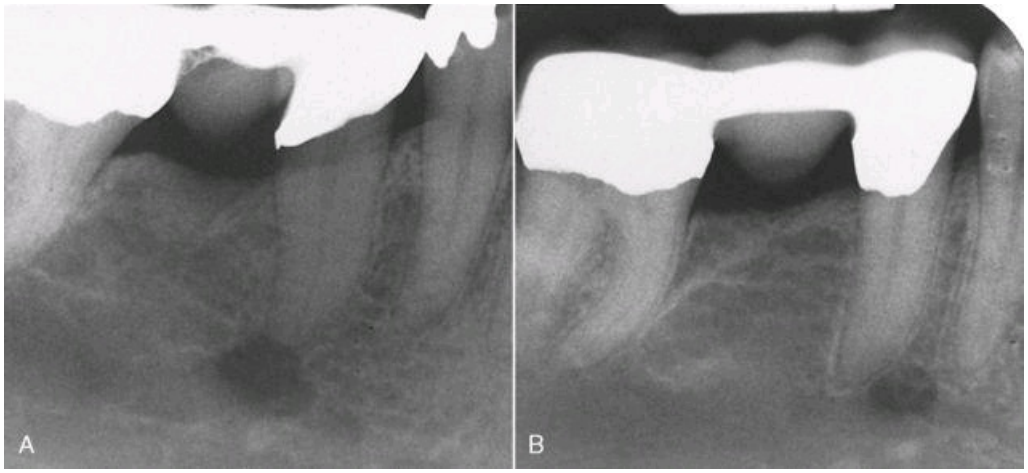

Figure 11-20 A, Radiolucent area over the apex could be mistaken for pathosis. **B,** Pulp testing (vital response) and a more distal angulation show the radiolucency to be a buccally placed (SLOB rule) mental foramen.

Maxilla

The maxilla region contains several structures (both radiolucent and radiopaque) that may be confused with endodontic pathosis. Examples are the maxillary sinus, incisive canals, nasal fossa, zygomatic process, and anterior nasal spine. Again, characteristics of the structure, as well as pulp responsiveness to tests, are important in differentiation.

Bitewing Projections

Although not truly a “special technique,” bitewing projections are often helpful in diagnosis and treatment planning. The relationships of film, cone, and tooth give a more consistent parallel orientation (Figure 11-21).

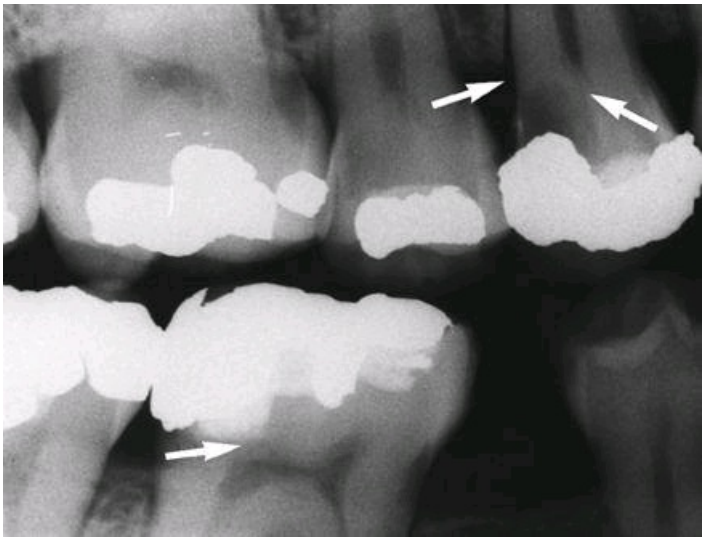

Figure 11-21 Bitewing radiograph shows important features clearly: relationship of bone to gingival extent of caries *(arrows)* and depth of caries and restorations relative to the pulp *(lower arrow)*.

(Courtesy Dr. C. Koloffon.)

Film Selection

Posterior packet film should be used for every projection in all patients except children. The anterior (narrow) films are unnecessary and in fact are frequently not wide enough to pick up an apex on an angled radiograph. Use of wider packet film obviously requires special placement for anterior projections (Figure 11-22). The film type recommended for diagnostic radiographs is E (Ektaspeed) film.[16]

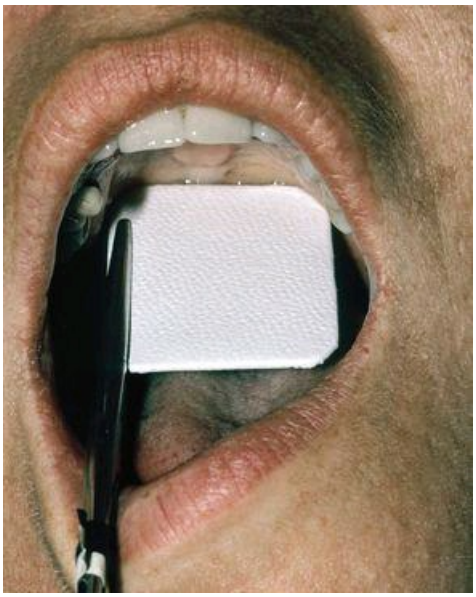

Figure 11-22 Narrow palate requires placement of posterior packet film distally. Note that the superior edge of the film is distal to the tuberosities.

Film Holders

Special adaptations of paralleling devices can be used for endodontic working films.[34] However, with some practice, nothing is more effective than a hemostat for ease and adaptability. The hemostat also is conveniently placed and sterilized in a kit with other instruments. The hemostat handle aligns the cone in both the vertical and horizontal planes (Figure 11-23). Having the patient hold the film with direct finger pressure is discouraged. This is awkward and frequently results in a bent film with a distorted radiographic image (Figure 11-24). *The film surface must remain flat.*

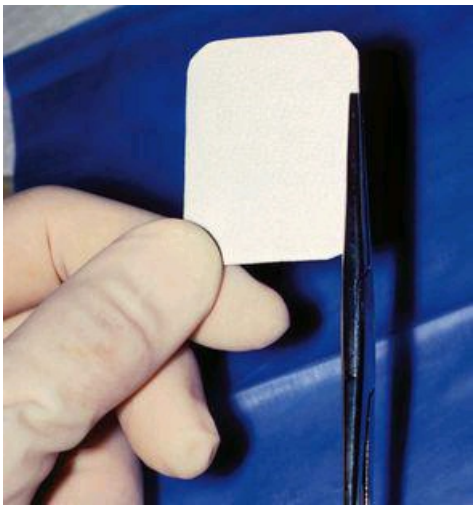

Figure 11-23 A hemostat is used for grasping the film and as a cone positioning and orientation device.

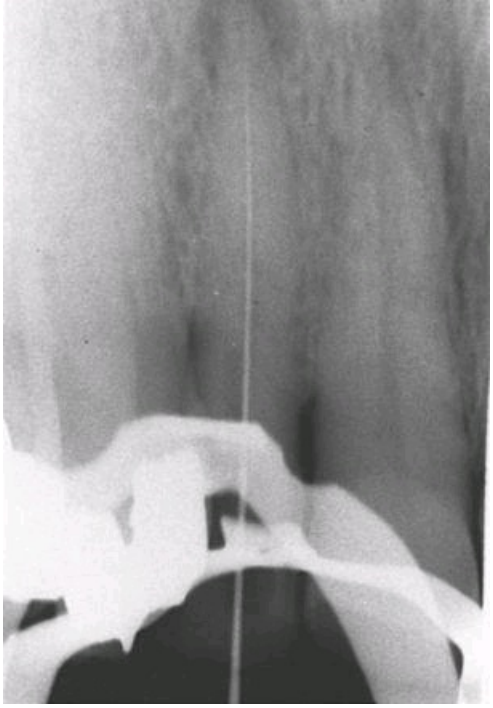

Figure 11-24 Pressure on film often causes bending, producing a distorted image. This bent film “stretches” the apical half of the root, making accurate interpretation and length determination impossible.

The hemostat-held film is placed by the operator. Then the patient holds the hemostat in the same position. The cone is aligned parallel to the hemostat in the frontal plane (vertical angulation) (Figure 11-25) and at 90 degrees to the handle (horizontal plane) (Figure 11-26). Because the handle is at an angle of 90 degrees to the film surface, the central beam strikes the film at the same 90-degree angle. This is the modified parallel technique because the film is often not parallel to the tooth. However, with the modified parallel technique, distortion is minimal and is not significant in working radiographs.[14]

Figure 11-25 Vertical angulation of the cone is set by aligning the long axis of the cone with the end of the hemostat handle.

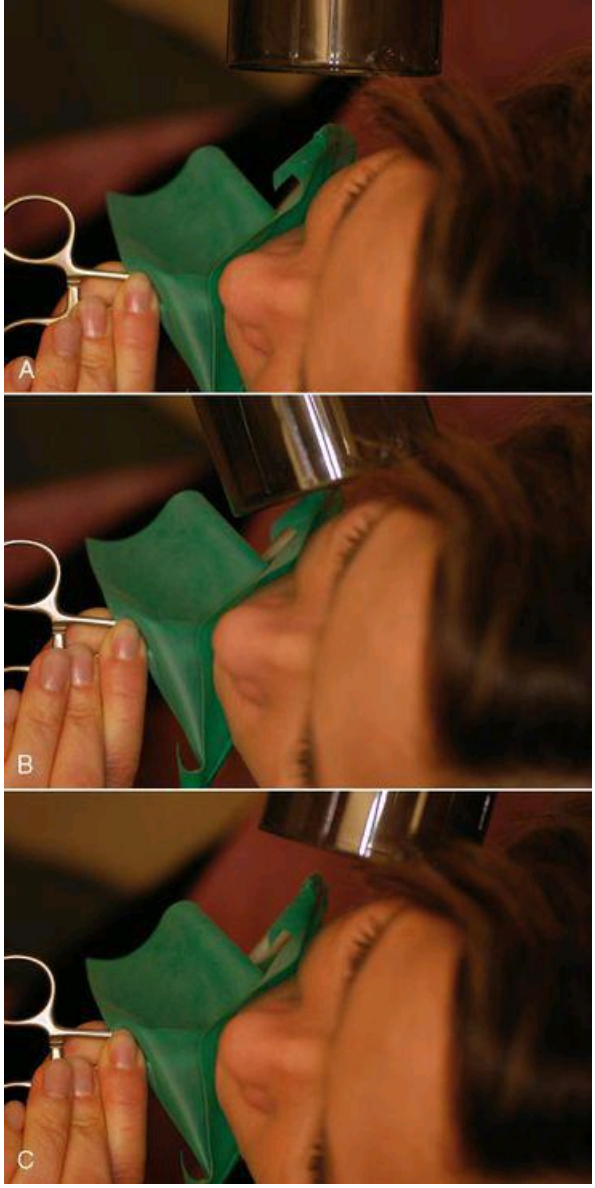

Figure 11-26 Horizontal angulation is determined by looking down from the top of the patient's head. **A,** The position is set by aligning the long axis of the cone (central beam) 90 degrees to the long axis of the hemostat handle. **B,** Mesial and **(C)** distal horizontal angulations are then varied accordingly.

Film Placement

Usually, films are positioned in the standard periapical projection. However, there are exceptions. Because of the film width and the relative narrowness of the arches, maxillary and mandibular anterior projections require film placement farther posteriorly.

In the maxillary posterior region, particularly when imaging molars, the film is placed on the side of the median raphe opposite the teeth to be radiographed, which has the effect of positioning the top of the film in a more superior position relative to the apexes (Figure 11-27).

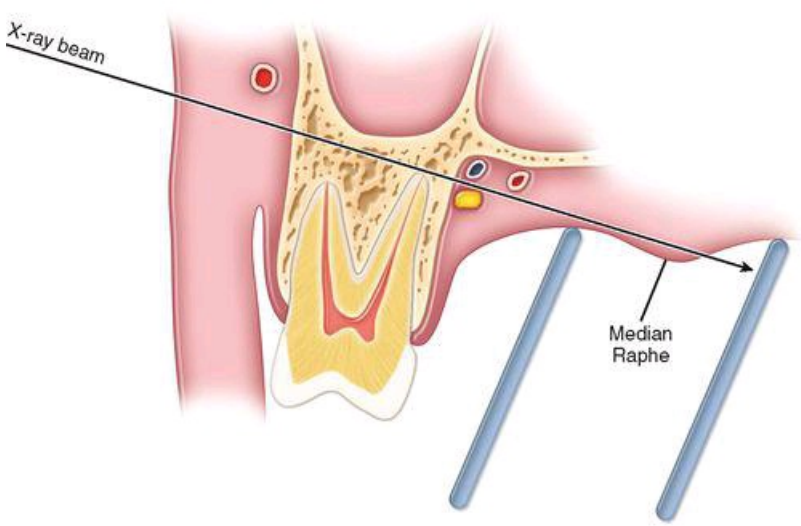

Figure 11-27 Positioning the film on the opposite side of the median raphe has the effect of "pulling" the upper edge of the film more superior relative to the apexes.

In the mandibular posterior region, the film is positioned toward the midline (under the tongue). Also, if the mouth is closed slightly, the mylohyoid muscle relaxes and permits the film to drop inferiorly.

The radiolucent rubber dam frame is not removed during film placement. A lower corner or edge of the rubber dam is released to allow insertion and positioning of hemostat and film (see Figure 11-25).

Cone Alignment

Indicated cone positions (Figures 11-28 and 11-29) (facial, mesial, or distal) are as follows.

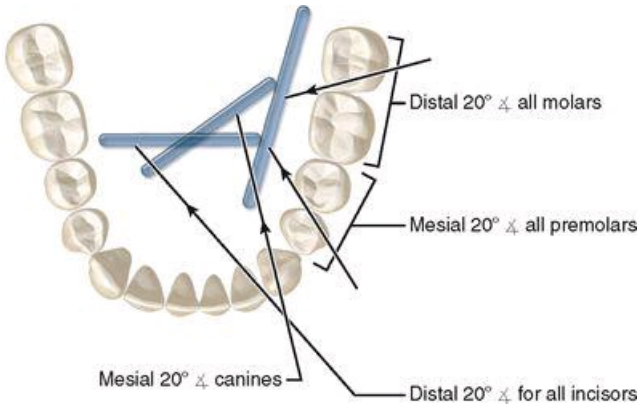

Figure 11-28 Correct film-cone placement on the mandible.

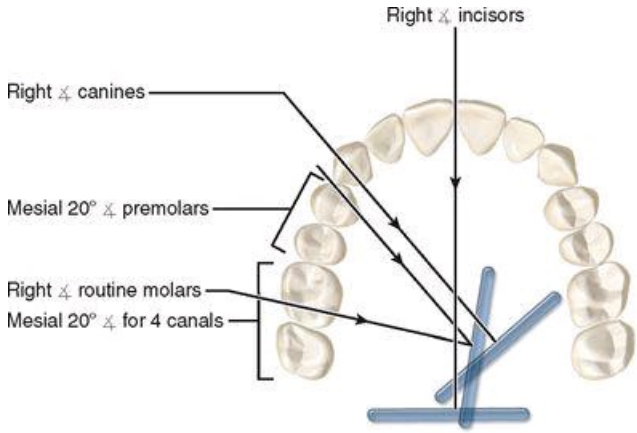

Figure 11-29 Correct film-cone placement on the maxilla.

Facial

Maxillary anterior teeth rarely have more than a single root and a single canal, thus only a facial (straight-on) projection is required. This is also true for maxillary molars unless a second mesiobuccal (mesiolingual) canal is detected and negotiated during access. The straight facial projection provides maximum resolution and clarity (which is difficult at best with maxillary molars).

Mesial

The mesial projection is indicated for maxillary and mandibular premolars and for mandibular canine teeth. A mesial projection is used for maxillary molars with a mesiolingual canal.

Distal

The distal projection is used for mandibular incisors and mandibular molars. The distal is preferred to the mesial projection for mandibular molars because of the relative position of the canals. Generally, the distal angle more effectively “opens up” the mesial root.

To summarize, angled working radiographs are made for maxillary premolars and molars with a mesiolingual canal and for all mandibular teeth. The maxillary projections are mesial and the mandibular projections are as follows: incisor—distal, canine—mesial, premolar—mesial, and molar—distal. An acronym for the cone angles on the mandible is DMMD.

Digital Working Radiographs

The same principles apply as with conventional analogue working films, including similar positioning of both the cone and the image-capturing device. The charged storage phosphor system and the direct digital systems utilize a sensor. The sensor, which is rigid, can be positioned and then held in place with the patient's finger. A preferred approach is to use a simple device that has been developed specifically for digital sensors. It is a tab with a sticky portion to attach to the sensor. An extending tab end can be held with a hemostat. The cone is then aligned using the handle of the hemostat, as previously described.

Rapid Processing

Different techniques and special solutions are available for fast processing (less than 1 minute) of working films and may be beneficial for speed viewing. If these rapid processing techniques are used, films may not retain their quality with time unless they are thoroughly fixed and washed.[35] Therefore, if a film is to be processed rapidly, a double-packet film should be used, with the duplicate processed in the routine manner.

Viewers

There are several types of radiographic viewers, both commercial and adapted. Commercial viewers magnify the image and block out peripheral light (Figure 11-30). This enhances the readability and interpretation of the film.[36] Other techniques or adaptations, such as a standard magnifying glass and small slide viewers, are also useful.

Figure 11-30 Magnifier-viewer blocks out peripheral light for better contrast. Contained within is a lens that magnifies the image four times.

Extra-Oral Film/Cone Positioning

Some patients cannot tolerate radiographs intraorally, usually because of gagging problems. Acceptable diagnostic and working films can be obtained.[37] This requires special positioning of the film and cone (Figure 11-31).

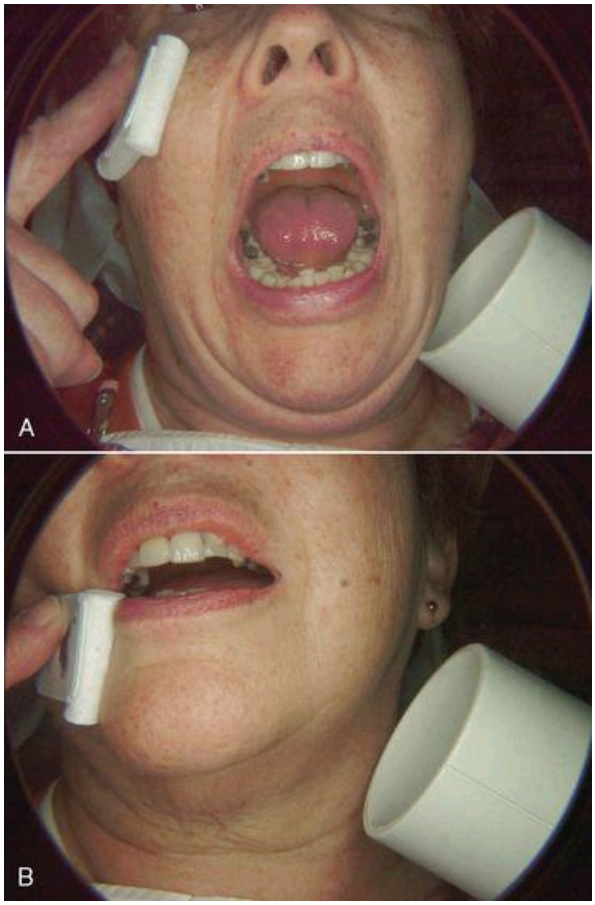

Figure 11-31 Extraoral radiographic technique. Useful for gaggers, this involves placing the film or sensor on the cheek. Increasing the exposure time is usually necessary. **A,** Maxillary posterior. Cone is positioned a negative 45 degrees to the occlusal plane. **B,** Mandibular posterior. Cone is positioned a negative 35 degrees.

NEW TECHNOLOGY

New approaches to radiography have been and are being developed. These approaches are unique, and some will improve existing techniques in addition to decreasing the radiation dose to patients. This new technology includes digital radiography, digital subtraction radiology, and tomography.[38-40]

Digital Radiography

A variety of digital radiographic systems has been compared, although none has been shown to be significantly superior as to image quality.[41] These systems are of considerable interest, offering the advantages of reduced radiation to the patient, increased speed of obtaining the image, ability to be transmitted, computer storage and enhancement, and a system that does not require a darkroom or x-ray processor.[42] However, these systems generally show no superiority to conventional radiographs for diagnosis or for working films.[43-48] Furthermore, computer-image enhancement does not seem to improve diagnostic interpretation significantly.[49]

Micro-Computed Tomography

Particularly intriguing is the potential of micro-computed tomography (micro-CT), with endodontic applications. This technology shows promise because it is very accurate and has the ability to identify morphology not visible on conventional radiographs.[50] Micro-CT also may be useful for treatment procedure evaluations, having shown accuracy for evaluating obturation quality.[51]

Ease of use with alternative systems and cost are factors that preclude routine use of these in the general dental office. Endodontists, however, require many working films and are finding the speed and versatility of digital radiography to be useful. As costs decrease and technology improves, there is no doubt that these or similar devices will come into more common usage by all groups of practitioners.

Chapter Review Questions available in Appendix B or on the DVD

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Endodontic Instruments

Keith V. Krell

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Define a basic set of instruments appropriate for these procedures: diagnosis, emergency treatment, canal preparation, obturation, and bleaching.

2. Describe the general physical properties of endodontic instruments and show how these characteristics are related to their use.

3. Describe the design (longitudinal, cross-sectional, and tip configuration) of the more common canal preparation instruments and their mode of use.

4. Explain the basis for sizing and taper (standardization) of hand-operated instruments.

5. Describe proper use of instruments to prevent breakage within the canal.

6. Recognize visible changes in instruments that will predispose to breakage.

7. Describe techniques used for sterilization and disinfection of instruments.

8. Select appropriate sterilization methods for each instrument type.

9. Identify procedures and chemicals that might cause deterioration of files and how to recognize that deterioration.

10. Describe and differentiate between conventional files and files of alternative designs.

11. Define the differences between stainless steel and nickel titanium intracanal instruments, including both physical properties and usage characteristics.

12. Describe the action and use of rotary instruments for both cleaning and shaping canals.

In considering endodontic instruments, those that are hand-operated (such as files and reamers) are the most important.

However, other specialized instruments, such as explorers and excavators, have been designed to adapt to root canal treatment requirements. Originally, instruments for root canal treatment were few in number and crude in design.[1] The earliest handoperated devices had long handles that were best suited for preparation of anterior teeth. As root canal treatment diversified, smaller “finger” instruments were developed for posterior teeth. In addition to being more adaptable, these provided improved tactile sense for the operator.[2] New designs in endodontic instruments have been introduced and will continue to evolve.

This chapter reviews the types of metals used and important aspects of the physical properties and usage characteristics of different designs of endodontic instruments. A basic armamentarium is described for each procedure, as well as systems for effective sterilization. Detailed information about all aspects of manufacturing and testing of intracanal instruments is beyond the scope and intent of this chapter; however, certain essential facts, as identified in the learning objectives, will be presented to enable effective use (and not abuse) of these instruments. To better illustrate the variety of design and usage in endodontic instruments, many of the instrument types are included on the accompanying video disc.

INSTRUMENTS FOR DIFFERENT PROCEDURES

Examination

A kit for examination and diagnosis includes (1) a front surface mouth mirror; (2) a periodontal probe; (3) an explorer, such as the double-ended No. 5 explorer; (4) the Glick No. 1 instrument; and (5) cotton forceps. These instruments have special endodontic applications (Figure 12-1).

Figure 12-1 Specialized endodontic instruments. *Top,* D16 explorer. *Center,* 31L spoon excavator. *Bottom,* Glick No. 1. The plugger end *(left)* is for heating and removal of gutta-percha; the paddle *(right)* is for placing temporary materials.

Explorers are double-ended instruments with long tapered tines at either a right or an obtuse angle. This design facilitates the location of canal orifices. They are very stiff and should not be inserted into canals or used for condensing gutta-percha. Explorers should never be heated.

The *spoon excavator* is another long-shank instrument. The excavator is used to remove caries, deep temporary cement, or coronal pulp tissue. The endodontic excavator has a right or left orientation similar to that of operative hand excavators. Excavators should not be heated.

The *Glick No. 1* instrument is used for placement of temporary restorations with the paddle end and removal (and then condensation) of excess gutta-percha with the heated plugger end. The rod-shaped plugger is graduated in 5-mm increments.

Emergency

The instruments necessary for emergency treatment are dictated by the diagnosis. For instance, irreversible pulpitis requires pulpotomy or pulpectomy. An acute apical abscess may require incision and drainage.

Basic instrumentation for most emergency procedures includes (1) an examination kit, (2) an anesthetic armamentarium, (3) a canal preparation (cleaning and shaping) system, and (4) occasionally an incision for drainage kit. Incision and drainage instruments include (1) scalpel handle and blade, (2) periosteal elevator, (3) rubber dam drain, (4) needle holder, and (5) irrigating syringe with an 18-gauge needle, sterile saline, and suction tip (Figure 12-2).

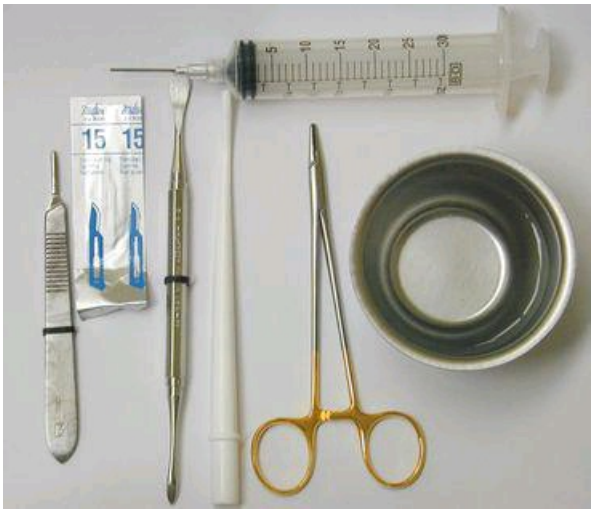

Figure 12-2 Basic emergency kit for an incision for drainage includes (1) scalpel handle and (2) blade, (3) periosteal elevator, (4) suction tip, (5) needle holder, (6) irrigating syringe with an 18-gauge needle, and (7) sterile saline. A rubber dam drain is a frequent addition.

Cleaning and Shaping

Instruments used for access and cleaning and shaping include (1) a 5- to 6-ml Luer-Lok syringe with a 27-gauge needle, (2) locking cotton pliers, (3) rotary instruments (Gates-Glidden drills), (4) a plastic instrument (Glick No. 1) for temporary placement, (5) broaches and files, (6) a lentulo spiral drill, and (7) a millimeter rule.

Nomenclature for Instruments

The nomenclature follows the recommendations of the International Organization for Standardization (ISO):

1. *Hand-operated* include K-type reamers and files, broaches, and Hedstrom-type files.

2. *Engine-driven* are hand types that have a latch that inserts into a slow-speed handpiece. These include rotary (Gates-Glidden and Peeso) engine-driven reamers and files and reciprocating files or reamers.

3. *Ultrasonic* and *sonic* are diverse in design. Some resemble barbed broaches, some resemble files, and others are diamond-coated wires. All insert into a dedicated vibratory handpiece that energizes the instrument. Some are further described in Chapter 15.

4. *Nickel-titanium* is a cross-over design and has been adapted both for hand instruments and rotary applications. Several designs have developed. Both hand- and engine-driven instruments have various configurations. The cross-section of the instruments takes many shapes.

In addition to canal preparation, other hand-operated instruments are adapted for other aspects of root canal treatment such as canal identification and obturation.

PHYSICAL CHARACTERISTICS

To débride a region of the canal space completely, the instrument must contact and plane all walls.[3] Despite continual improvements in design and physical properties, there are still no instruments that totally clean and shape all root canal spaces. Irregular canal spaces do not correspond to and cannot always be well prepared by an instrument with a regular (round) shape. In addition, stainless steel instruments are relatively inflexible, which renders them not particularly adaptable to canal curvatures. Nickel-titanium instruments are more flexible and adapt more readily to fine, curved canals[4] but have no advantage over stainless steel files in irregular canal spaces.[5-7] These incongruences between reality and ideal shape require judicious and skillful use of canal preparation instruments to maximize débridement and to avoid procedural errors.[8]

Instrument Fabrication

A hand-operated reamer or file begins as a round wire that is modified to form a tapered instrument with cutting edges. The instrument is used with a twisting (reaming) or pulling (filing) motion in an attempt to produce clean, smooth, symmetrical canal walls. However, the prepared canal is rarely round when viewed in cross section.

Several factors inherent in the stainless steel wire from which hand-operated instruments are made must be considered. How is adequate flexibility maintained without instrument fatigue? How much abuse can these files endure before fatigue and failure ensue? How does the operator know when the file has been fatigued to a critical point? And finally, how does an operator maintain an efficient cutting edge while at the same time avoid cutting new, nonanatomic canal spaces?

With nickel-titanium files, the same questions are addressed. These instruments have different physical properties and different usage characteristics. Importantly, they can also fatigue and separate when used incorrectly or overused.

Hand-Operated Instruments

Several cross-sectional shapes of files are commercially available (Figure 12-3). Two techniques for manufacturing these instruments have been developed.

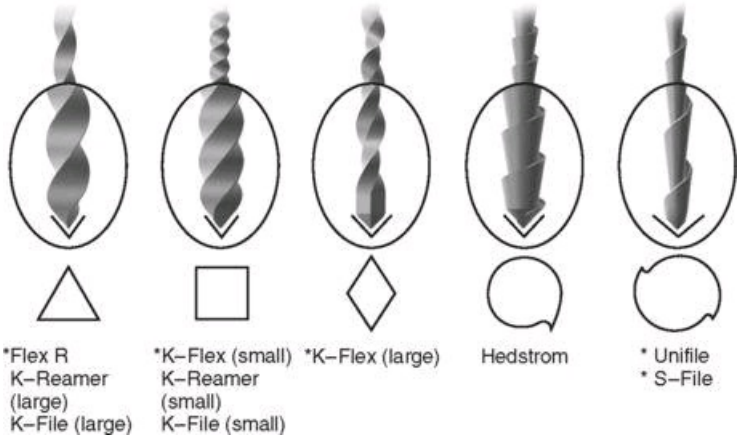

Figure 12-3 Longitudinal and cross-sectional shapes of various hand-operated instruments. (Those marked with an asterisk are brand names.) Note that small sizes of K-reamers, K-files, and K-Flex* have a different shape than the larger sizes.

Machined

One technique involves machining (grinding) the instrument directly on a lathe; an example is the Hedstrom-type file (Figure 12-4, *A*). All nickel-titanium instruments are machined.

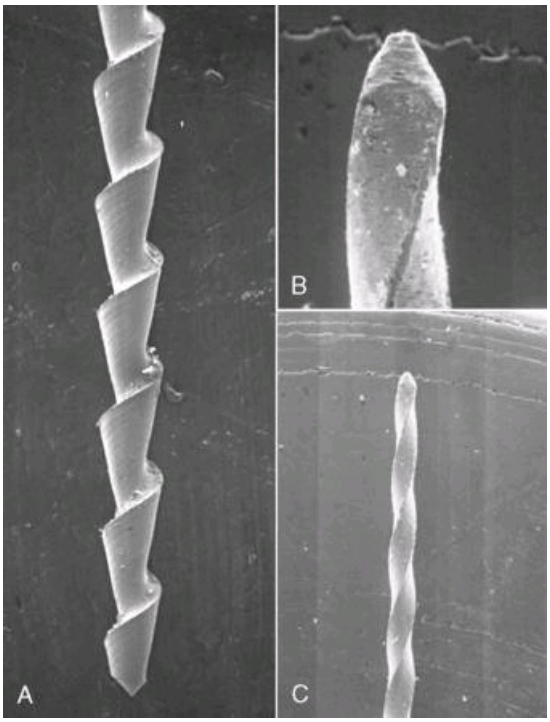

Figure 12-4 A, Hedstrom file, machined by rotating a wire on a lathe. Note the spiral shape. These are efficient cutters (on the pull stroke) but are more susceptible to separation when locked and twisted. **B** and **C,** A machined K-type file. Note that the transition angle at the leading cutting edge of the tip is rounded, rendering it noncutting.

Some manufacturers produce K-type files using the machined (lathe-grinding) process (Figure 12-4, *B* and *C*). This change from the grinding and twisting manufacturing process results in different physical and working properties from the original K-type file.[9,10] For instance, the machined file has less rotational resistance to breakage than a ground-twisted file of the same size.[10]

Ground-Twisted

Another technique consists of first grinding, then twisting. Raw wire is ground into tapered geometric blanks: square, triangular, and rhomboid (Figure 12-5). The blanks are then twisted counterclockwise to produce helical cutting edges. These are K-type files and reamers. K-type files have more twists per millimeter of length than the corresponding size of K-type reamer. Both have a pyramidal tip (75 ± 15 degrees) that is produced by grinding after twisting.

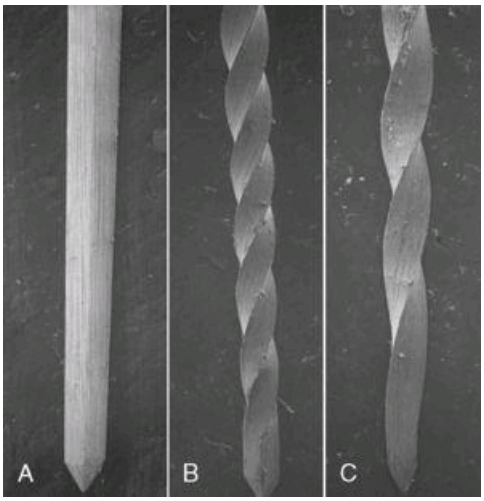

Figure 12-5 Ground-twisted instruments. **A,** A square file blank ground from wire. After twisting counterclockwise, the appearance of a file (more flutes) **(B)** and reamer (fewer flutes) **(C).**

Clinical Use

Besides their configuration, the difference between files and reamers is their intended use. Files are manipulated with a rasping, or push-pull planing, motion. This motion is more efficient when there are many flutes or spirals on the instrument shaft making contact with the canal walls; the flutes are at right angles to the long axis. Reamers are twisted and withdrawn; therefore cutting takes place during rotation. This motion is most efficient with a cutting edge that more closely parallels the instrument shaft. Reamer configuration is created by imparting fewer twists, resulting in increased flute spacing, which tends to prevent clogging of the cutting edge.[11] Files can be both filed and reamed, whereas reamers can only be reamed. Because they are less versatile, reamers are seldom used.

Physical Properties

Materials researchers and manufacturers have certain theories or hypotheses about file properties such as “Flexibility is increased by increasing the length or decreasing the cross-sectional diameter” and “The more acute the cutting angle, the more efficient a blade is in removing a substance.”[12,13] These theories are more applicable to the laboratory than to the clinical situation. Because of bench-top research, certain important limits to specific physical properties have been identified and incorporated into a series of standards for the manufacture of hand-operated instruments. However, clinical behavior may not directly relate to such in vitro testing.[14]

Standards for K-type files and reamers were first published in 1976 as American Dental Association (ADA) specification No. 28.[15] These standards were developed specifically for hand-operated instruments used in the canal. Therefore standards were established for *fracture resistance* by twisting, *stiffness* of the files and reamers, and *corrosion resistance*. In addition to physical characteristics, these standards established *dimensions,* as well as acceptable tolerances in manufacturing.

Flexibility, sharpness, and corrosion resistance are properties related to metal and design. Traditional metals have included stainless or carbon steel. Compared to stainless steel, many carbon steel instruments have been shown to cut somewhat more efficiently[16]; they are similar in other respects. However, carbon steel is little used because it is more susceptible to corrosion by autoclaving and irrigating solutions.[17]

By changing the cross-sectional design from square to triangular or rhomboid and decreasing the number of flutes per millimeter, greater flexibility is gained. Blank nickel-titanium wire is even more flexible. Nickeltitanium alloy possesses a modulus of elasticity that is one-fourth to one-fifth that of stainless steel, allowing a wide range of elastic deformation.[18] An advantage of this increased flexibility is that a file follows the canal curvature with less deformation (transportation) during enlargement. A disadvantage of the increased flexibility is the inability to precurve the file for introduction into canals of posterior teeth when there is a decreased interocclusal opening. Another disadvantage is that cutting efficiency of nickel-titanium files may be reduced with clinical usage compared with stainless steel because of the greater elasticity. Therefore, to best utilize the properties offered with nickel-titanium files, engine-driven configurations have been developed. Several of the present nickel-titanium instruments incorporate a U-shaped groove with a flat land area. When the instrument is rotated, the flutes will plane the canal wall as the land area keeps the instrument centered; this centering is important in fine, curved canals.[4,19]

Standardization

Original (conventional style) K-type files were available in only six sizes with no uniformity among different brands as to taper or tip configuration. ADA specification No. 28 established standards for instrument taper, tip geometry, and size criteria for 19 different sizes of instruments, as well as acceptable tolerance of manufacturing error.[15] Also implemented was a color code for instrument handles for identification and an additional file size (No. 06).[20] In 1982, ADA specification No. 58 standardized and established the minimal physical criteria for Hedstrom-type files.[21] The most recent revisions of ADA specifications No. 28 and No. 58 were published in 1989. These further clarified minimal physical criteria and added newer criteria for plastic handle retention on files.[22]

ADA/ANSI specification No. 95 was updated in 2003 and provides dimension and sizing requirements for "root canal enlargers" such as Gates-Glidden drills and Peeso reamers. It also has added testing procedures and test devices for resistance to fatigue.[23]

ADA/ANSI specification No. 101 was published in 2001 and covers root-canal instruments that are not covered by ADA/ANSI specifications No. 28 and No. 58 such as the .04 taper nickel-titanium rotary instruments. ADA/ANSI specification No. 101 provides definitions for flexible instruments, guided-tip instruments, and the tip portion of these instruments. The dimensional requirements and designations that are inclusive of any taper or shape of instrument are also specified. Finally, the modified twisting and bending data tables that compensate for the effect of tapers other than the standard 0.02 (2%) are given. The document does *not* specify the type of metal to be used by the manufacturer nor the type of material to be used for the handle.[24]

Despite reported specific size and shape requirements, hand-operated instruments do not demonstrate reliable and consistent dimensional standardization.[25]

Lengths

Files and reamers are available in three shaft lengths: 21, 25, and 31 mm. Shorter instruments afford improved operator control and easier access to posterior teeth, to which limited opening impairs access. The 25- and 31-mm instruments are used for longer roots. The 25-mm instruments are the most commonly used instruments during root canal preparation.

Sizing

Dimensions of K-type files and reamers are designated according to the diameters of the instrument at specified positions along its length (as stated in ADA specification No. 28) (Figure 12-6). File tip diameters increase in 0.05-mm increments up to the size 60 file (0.60 mm at the tip), and then by 0.10-mm increments up to size 140. The diameter at the tip of the point is known as D_0. The spiral cutting edge of the instrument must be at least 16 mm long, and the diameter at this point is D_{16}. The file diameter increases at a rate of 0.02 mm per running millimeter of length.

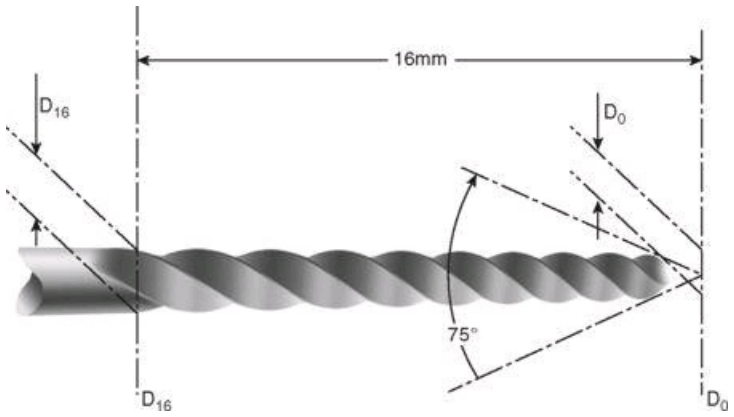

Figure 12-6 The specifications for standardization of files and reamers.

Another "standard" introduced by one of the nickel-titanium rotary series (Profile)is a 29% constant increase in tip diameters between sizes; each file tip increases by a constant percentage rather than the random increases seen in ISO sizing.

The nickel-titanium rotary instruments have other variable tapers of 0.04 and 0.06. For every millimeter of length, the diameter increases by 0.04 or 0.06 mm. These greater tapers make these more aggressive in creating marked flaring preparation. These types of instruments have not been demonstrated to be superior.[26]

Tip Design

Originally, the tip angle of K-type files and reamers was approximately 75 degrees plus or minus 15 degrees. This design was intended to provide cutting efficiency without an excessively sharp transition angle. Newer designs have different tip angles and designs in an attempt to minimize canal alterations. Some machined K-files incorporate a so-called nonaggressive tip or noncutting tip (see Figure 12-4, *B*) to provide less dentin cutting by reducing the sharp tip transition angle. The flexible file type (Flex-R) has a conical noncutting file tip with a tip angle of 70 degrees and a guiding collar angle of 35 degrees. The intent is to guide the file through the curve rather than cutting only the outer canal wall.[27,28]

Another modification in file design is the Lightspeed. Unlike other instruments that have several millimeters of cutting or planing area, this nickel-titanium instrument has a cutting area of about 3 mm. The tip is noncutting and looks similar to a small Gates-Glidden drill. (A further description of the Lightspeed instrument is found in the section on Unique Designs.)

Torsional Limits

Torsional limit is the amount of rotational torque that can be applied to a "locked" instrument to the point of breakage (separation). Obviously, an instrument should have sufficient strength to be rotated and worked vigorously without separating in the canal. Standards for steel hand-preparation instruments were established for rotational limits (point of breakage) at various forces. Smaller instruments (less than size 20) can withstand more rotations without breaking than larger (greater than size 40) instruments.

Machined K-type files have different physical and working properties than ground-twisted files. There is no difference in torsional strength between ground-twisted and machined files. However, machined files are weaker, demonstrating less plastic deformation before failure occurs.[8] Therefore this tendency toward less visible deformation before separation requires more caution with the use of machined files to avoid instrument failure.[9]

Under test conditions, the nickel-titanium files (all are machined) have increased resistance to fracture compared with stainless steel files.[29] Only Lightspeed instruments have incorporated a designated separation point at 18 mm to allow for retrieval of broken instruments. The ANSI/ADA specification No.101 has established new torsional standards for all nickel-titanium and stainless steel rotary instruments.[24] Nickel-titanium metal may have advantages over stainless steel, although these advantages have not been conclusively demonstrated in clinical usage or in clinical trials.[6,8,19]

Color Coding

Color coding of file handles designates size. Color coding of the newer nontraditional instruments varies according to the manufacturer.

Variations

Broaches

Barbed broaches are stainless steel instruments with plastic handles. The tapered-wire broach is barbed by scoring and prying a tag of metal away from the long axis of the wire (Figure 12-7). Barbs entangle and remove canal contents. This instrument should be neither bound in the canal nor aggressively forced around a canal curvature. Either action may cause the barbs to engage the canal wall, preventing the broach from being removed intact or fracturing. Barbed broaches should not be reused. Single-barbed broaches are available in presterilized bubble packaging.

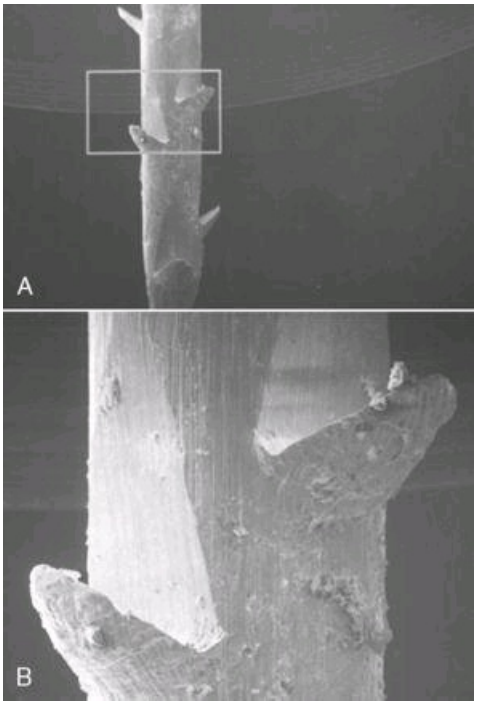

Figure 12-7 Barbed broach showing the barbs pulled away from the instrument shaft.

Lentulo Spiral Drills

Lentulo spiral drills are twisted wire instruments used in the slow-speed handpiece (Figure 12-8). They have been used to spin pastes, sealer, cements, or calcium hydroxide into the canal. They must be used with care to avoid “throwing” quantities of unset material out of the apex. In fact, there is no reason to use lentulo drills other than to place calcium hydroxide paste. The drill must be rotated so that it will not “screw” itself into the canal; it may lock and separate.

Figure 12-8 Lentulo spiral drill is used to spin calcium hydroxide into canals.

Unique Designs

An innovative nickel-titanium instrument, the Lightspeed (Figure 12-9), has been introduced that incorporates three distinct features: a noncutting pilot tip, a small fluted area, and a small flexible shaft. Both hand-operated and engine-driven instruments are available. These instruments tend to stay centered in curved canals if used properly.[4,30,31] Although interesting, these have not been shown to be superior to conventional hand instruments and are prone to separation if overused.[31,32]

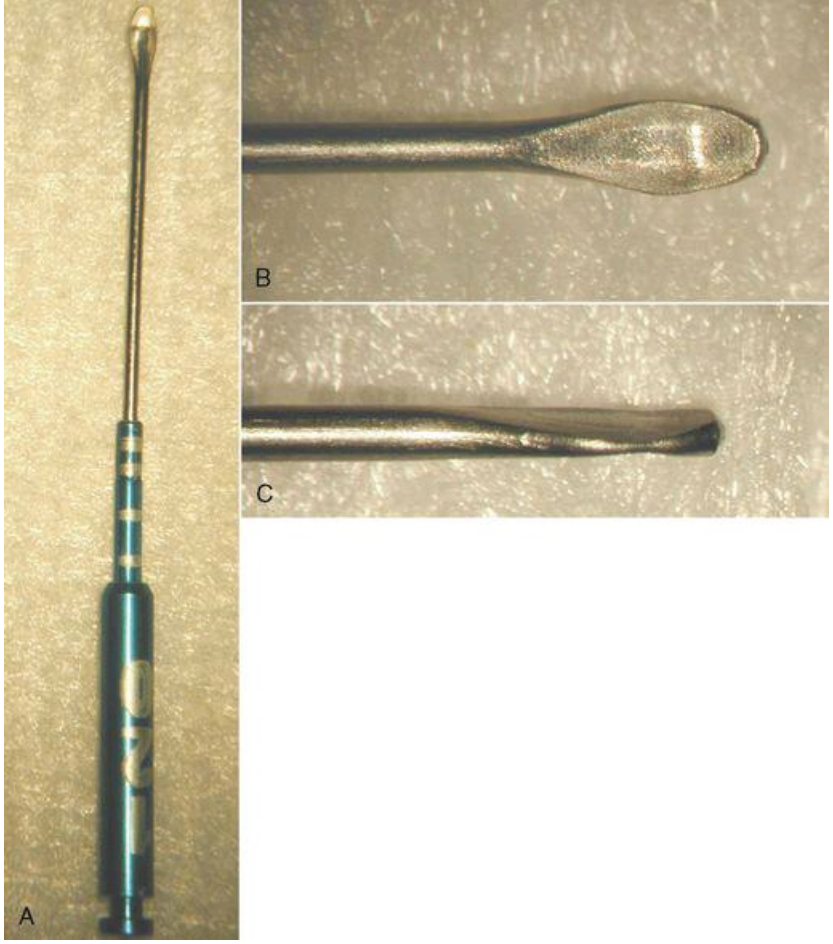

Figure 12-9 A, Innovative file design of the rotary nickel-titanium Lightspeed instrument. The end of the instrument does the work, and the noncutting tip and the small flexible shaft tend to stay centered during preparation of the canal. **B** and **C,** Magnified views of the tip of the Lightspeed instrument.

Rotary Instruments

Some preparation techniques require slow-speed rotary instruments to facilitate preparation, primarily in establishing straight-line access (Figure 12-10), although engine-driven reamers and files for cleaning and shaping are also marketed. Most common are Gates-Glidden drills and Peeso reamers. Table 12-1 shows the comparative ISO sizes of both Gates-Glidden drills and Peeso reamers.

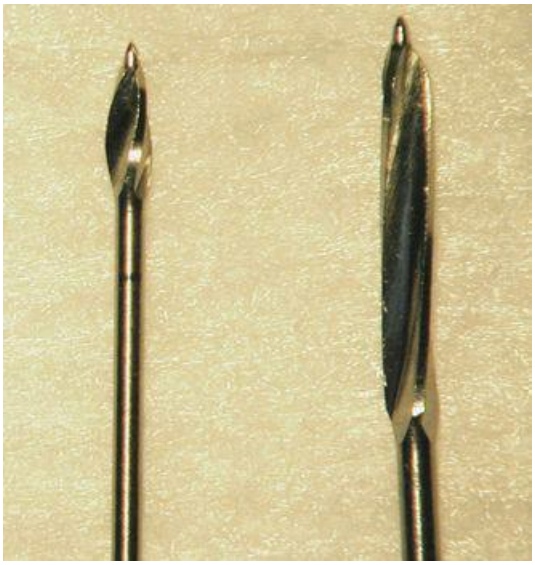

Figure 12-10 *Left,* Gates-Glidden drill. Note the noncutting tip and the elliptical shape. *Right,* Peeso reamer. Note the noncutting "safe" tip and parallel sides. These are stiffer and more aggressive than the Gates-Glidden drill. Both are used for straight-line access preparation.

Table 12-1 Rotary Flaring Instruments

Size	Gates-Glidden Drills	Peeso Reamers
No. 1	0.4 mm	0.7 mm
No. 2	0.6 mm	0.9 mm
No. 3	0.8 mm	1.1 mm

No. 4	1.0 mm	1.3 mm
No. 5	1.2 mm	1.5 mm
No. 6	1.4 mm	1.7 mm

Gates-Glidden drills and Peeso reamers are made from either carbon or stainless steel. Carbon steel has inferior properties, particularly less strength, and therefore should not be used. Corrosion is not a problem with stainless steel, but dulling occurs with sterilization procedures and repeated usage.

Gates-Glidden Drills

Gates-Glidden drills are elliptically (flame) shaped burs with a latch attachment. Gates-Glidden drills are used to open the orifice. They also achieve straight-line access by removing the dentin shelf and rapidly flaring the coronal and middle third of the canal.

Gates-Glidden drills are designed to break high in the shank region. This design allows easier removal of the broken instrument from a tooth; fracture near the cutting head may block a canal. ANSI/ADA specification No. 95 established torsional limits of rotary intracanal instruments, including Gates-Glidden drills, and they usually fracture where intended.[33-35] Importantly, these drills must be continuously rotated. If they stop, the head may lock in the canal, with torsional failure and fracture. Gates-Glidden drills are available in 15- and 19-mm lengths. The shorter instruments are helpful in posterior teeth, where access to the canal orifice is limited.

Peeso Reamers

Peeso reamers are also used as adjunctive devices in canal preparation. They are basically similar to Gates-Glidden drills but have parallel cutting sides rather than an elliptical shape. These instruments are available with or without safe tips. Peeso reamers have been suggested as a means of improving straight-line access, although they are less flexible and less well controlled than Gates-Glidden drills.[35] Both types are aggressive and can rapidly overenlarge the canal.

Engine-Driven Reamers

Engine-driven reamers are used for cleaning and shaping. The traditional types are made of stainless steel. Because of their relative stiffness, they are difficult to control and generally create irregular, poorly débrided preparations, particularly in curved canals. These aggressive instruments also generate large amounts of debris, which packs apically. Their use is not recommended.

Engine-Driven Nickel-Titanium Files

Engine-driven nickel-titanium files allow greater control in small, curved canals. These instruments do not have a cutting end and have less tendency to transport the apical preparation.[36,37] The files are available in a variety of shapes and designs (Figure 12-11). Utilization of these instruments by dental students in technique laboratories has demonstrated fewer preparation errors than with use of stainless steel hand instruments.[38-40]

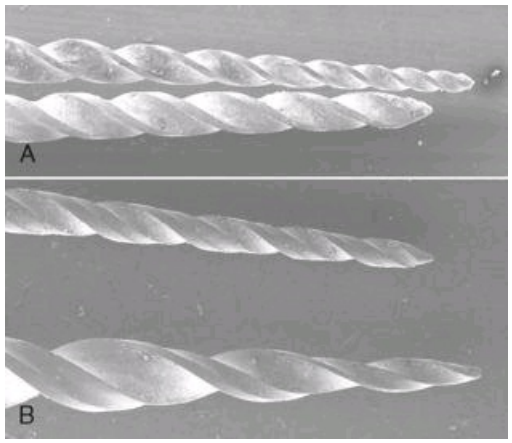

Figure 12-11 Examples of nickel-titanium rotary files of varying design. **A,** 0.04 and greater taper (GT) titanium files (ProFile). **B,** Quantec files. Note the aggressive taper of the file on the bottom.

INTRACANAL USAGE

Broaches

Removal of pulp requires a broach that will not bind and yet is large enough to ensnare the tissue. Binding should be minimized because of possible breakage.

Reamers and Files

Two types of motion are common in root canal preparation: reaming and filing[41] (Figure 12-12). Reaming consists of rotating the instrument clockwise and scribing an arc from one cutting edge to the next. For example, a triangular reamer has three 60-degree cutting edges and therefore requires 120 degrees of rotation (one-third of a turn), whereas a file with a 90-degree angle necessitates only a quarter turn before withdrawal. Rhomboidal files require a 180-degree turn to make an edge-to-edge arc.

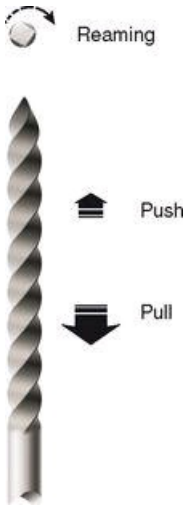

Figure 12-12 Filing and reaming motions. Tooth structure is removed primarily on the pull stroke with filing and on rotation with reaming.

Filing requires a series of repetitive motions. First, the instrument is advanced to its full length into the canal space using a passive “twiddling” (teasing without planing) motion. Next, the file is rotated (a quarter turn or more) and then withdrawn from the canal space while the tip is pushed firmly against a canal wall, much as a paintbrush is applied to a wall when painting. The twiddling, reaming, and withdrawal motions are repeated with the file tip pushed against a different portion of the canal wall on each outstroke until all walls have been planed (circumferential filing).

Hedstrom-type files and files with a similar design (S or U) are used only with a filing motion because they have less torsional resistance to breakage. In other words, Hedstrom-type files are more prone to separation because of the decreased cross-sectional diameter of equivalent-sized instruments. Also, their design does not facilitate reaming.

Avoidance of Instrument Separation

Separation of hand files in the canal is prevented by regularly inspecting the instrument for defects (Figure 12-13) such as (1) unwinding of the flutes (twisting clockwise and opening of the flutes), (2) roll-up of the flutes (excessive continued clockwise twisting after unwinding), (3) tip distortion (the tip has been bent excessively), and (4) corrosion. If an instrument exhibits *any* signs of wear, it should be discarded immediately. Prevention is the key to avoiding untimely instrument separation.

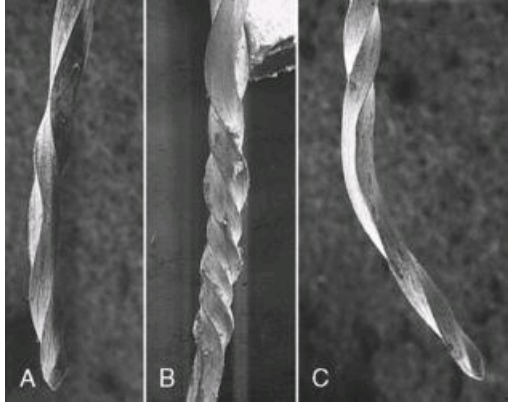

Figure 12-13 Defects created during instrumentation. Each file shown must be discarded because of possible breakage. **A,** Unwinding of the flutes. **B,** "Roll-up" of the flutes. **C,** Unwound and bent instrument.

Rotary Instruments

All of the engine-driven nickel-titanium files rely on rotational motion only and therefore have a reaming action.

Avoidance of Instrument Separation

The manufacturer of the nickel-titanium 0.04- and 0.06-mm taper rotary files suggests using them in high-torque slow-speed handpieces that rotate at 150 to 300 rpm. The number of canals that can be prepared with a nickeltitanium instrument varies from 4 to 16, depending on the size and curvature of the canals and pressure used with the files. When the canal is smaller and more curved, there is more wear and tear on the instrument. All manufacturers suggest discarding the files if any deformation occurs. Studies have suggested that lower speeds reduce the likelihood of instrument fracture.[42-44] Figure 12-14 shows instrument fatigue and "roll-up," which may result in breakage of nickel-titanium instruments after use in canals. Preflaring of canals has been shown to allow more uses of rotary instruments before separation compared with the crown-down technique by itself.[44] The Lightspeed nickel-titanium instruments are also used in high-torque slow-speed handpieces at 750 to 2000 rpm. Within this limit, faster rotational speed does not appear to increase the likelihood of fracture.[45] The manufacturer suggests discarding each file after 10 uses.

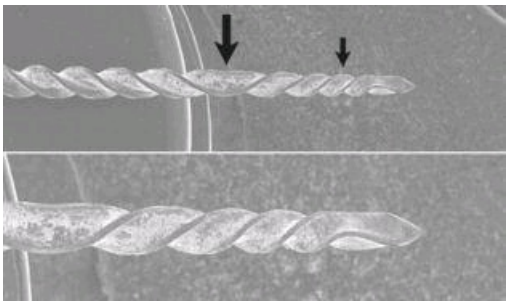

Figure 12-14 Overused rotary nickel-titanium files exhibiting "roll-up" *(small arrow)*, and "unwinding" *(large arrow)*.

INSTRUMENTS FOR OBTURATION

Instruments used for obturation include (1) spreaders or pluggers, (2) Glick No. 1 for heat transfer and temporary placement, (3) locking cotton pliers, and (4) 5/7 plugger or pluggers used for vertical condensation.

Several filling techniques are available. The instruments used for the two most practiced techniques, lateral and vertical condensation, are described here.

Lateral Condensation

The instruments used for lateral condensation are spreaders and small pluggers (Figure 12-15). They are used for condensing and adapting gutta-percha and creating space for accessory cones. They are either handled, with a shank attached to a metal handle, or finger-type, with only a plastic handle (Figure 12-16). The handled instruments are stiff because they are generally made of annealed stainless steel. Finger spreaders and pluggers are not annealed and therefore are dead soft, giving them more flexibility. Handled instruments do not negotiate curved canals. Finger spreaders and pluggers are best suited for obturating curved canals.

Figure 12-15 Fine finger spreader *(left)* and fine finger plugger *(right)*. Both are used similarly for lateral condensation. Spreaders are pointed, and pluggers are flat at the tip.

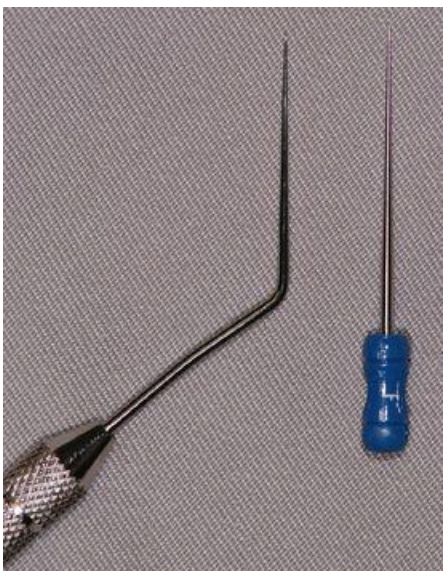

Figure 12-16 D11 handled spreader *(left)* and a fine finger spreader *(right)*. Both are designed for lateral condensation. The finger spreader (or plugger) is more versatile and safer.

Finger spreaders and pluggers have different tips (see Figure 12-15). Pluggers are flat, whereas spreaders are pointed. Finger spreaders and pluggers behave similarly and are used interchangeably in lateral condensation.

The taper of spreaders varies among instruments. Highly tapered spreaders increase in diameter at a greater rate than do standardized instruments, which increase 0.02 mm per mm of length. The greater the taper the more the canal space must be enlarged or flared to facilitate spreader penetration.

Both stainless steel and nickel-titanium spreaders are available. The obvious advantage of nickel-titanium spreaders over stainless steel spreaders is greater spreader penetration in highly curved canals.[46] Nickel-titanium spreaders also create less

stress in curved canals compared with stainless steel spreaders.[47] The disadvantage of nickel-titanium spreaders is the inability to precurve them for patients with limited interocclusal space.

Vertical Condensation

In this obturation technique the filling material is alternately softened (with heat) and then vertically compacted with pluggers. The softened gutta-percha filling material is pushed into the interstices of the canal, but this technique offers less apical control of the material than lateral condensation.

Vertical condensation instruments can be divided into two categories: those that are heated to transfer heat to the gutta-percha and those that condense the gutta-percha (pluggers) (Figure 12-17). Heat transfer instruments have handles, as do most pluggers. Finger pluggers may be used in lieu of handled instruments in small curved canals.

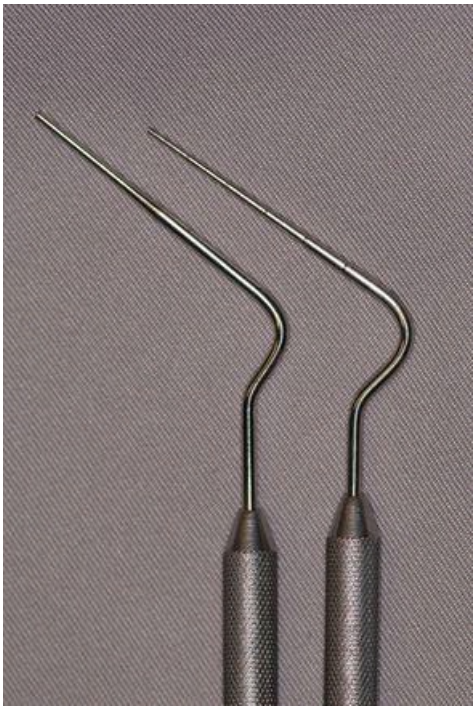

Figure 12-17 Heat transfer *(left)* and condenser (heater-plugger) *(right)* for vertical condensation of gutta-percha.

INSTRUMENTS FOR BLEACHING

Bleaching usually is done with the instruments included with the cleaning and shaping tray with the addition of the paste ingredients and a plastic instrument (Figure 12-18). A walking bleach technique requires the plastic instrument to place the sodium perborate mixture in the chamber and then to temporize.

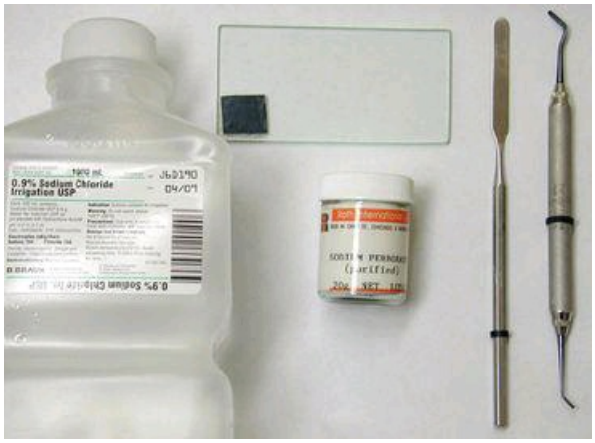

Figure 12-18 For internal bleaching. Sterile saline for mixing with sodium perborate. Spatula for mixing and plastic instrument for paste and temporary placement.

STERILIZATION AND DISINFECTION

Endodontic instruments are contaminated with blood, soft and hard tissue remnants, and bacteria and bacterial byproducts. Thus they must be cleaned often and disinfected during the procedure and then sterilized. Also, because the instruments may be contaminated when new, they must be sterilized before initial use. Different sterilization techniques are available.

Small kits, such as those used for examination, may conveniently be bagged, sterilized, and stored in the package until needed. Larger kits for treatment may be more rapidly and easily handled in cassettes for sterilization and storage.

Sterilization

Cold- or heat-labile instruments, such as some rubber dam frames, may be immersed for a sufficiently long period of time in solutions such as glutaraldehyde. Generally, 24 hours are required to achieve cold sterilization. Immersion may be effective for disinfection but will fail to kill all organisms. Because this method of sterilization is not presently verifiable with biologic indicators, it is the least desirable and should be reserved for instruments that cannot withstand heat.

Pressure Sterilization

A common method of sterilizing all files and other endodontic hand instruments is the steam or chemical autoclave. Instruments that have been wrapped in gauze should be autoclaved for 20 minutes at 121° C and 15 psi.[48] This will kill all bacteria, spores, and viruses. Various sodium nitrite dips will retard rust. These “milk baths” help spare the carbon steel Gates-Glidden drills.

Pressure sterilizers using a chemical rather than water have the advantage of causing less rusting. However, both steam and chemical autoclaving will dull the edges of all cutting instruments because of expansion with heat and contraction with cooling, resulting in permanent edge deformation.

Dry Heat Sterilization

Dry heat is superior for sterilizing sharp-edged instruments, such as scissors, to best preserve their cutting edge. The time cycle for dry heat sterilization is temperature dependent. After the temperature reaches 160° C, the instruments should be left undisturbed for 60 minutes. The disadvantage of this method is the substantial time required, both for sterilization and subsequently for cooling. If the temperature falls below 160° C, the full 60-minute heat cycle must be repeated.[48]

Disinfection

Surface disinfection during canal débridement is accomplished by using a banker's sponge soaked in 70% isopropyl alcohol or proprietary quaternary ammonium solutions. Files can be thrust briskly in and out of this sponge to dislodge debris and contact the disinfectant. This procedure cleans but does not sterilize the instrument.

Chapter Review Questions available in Appendix B or on the DVD

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CHAPTER 13

Internal Anatomy

Richard E. Walton, Frank J. Vertucci

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter and the Appendix, the student should be able to:

1. Recognize errors that may cause difficulties or failures in root canal treatment because of lack of knowledge of pulp anatomy.

2. List ways that help to determine the type of pulp canal system.

3. Draw common shapes of roots in cross-section and common canal configurations in these roots.

4. Describe the most common root and pulp anatomy of each tooth.

5. List the average length, number of roots, and most common root curvatures for each tooth type.

6. Characterize the more frequent variations in root and pulp anatomy of each tooth.

7. Explain why standard periapical radiographs do not present the complete picture of root and pulp anatomy.

8. Draw a representative example of the most common internal and external anatomy of each tooth and root in the following planes: (1) sagittal section of mesiodistal and faciolingual planes and (2) cross-section through the cervical, middle, and apical thirds.

9. Suggest methods for determining whether roots and canals are curved, as well as the severity of the curvature.

10. State the tenet of the relationship of shape of pulp system to root anatomy.

11. List each tooth and the root(s) that require a search for more than one canal.

12. List and recognize the significance of iatrogenic or pathologic factors that may cause alterations in pulp anatomy.
13. Define the pulp space and list and describe its major components.
14. Describe variations in the pulp system in the apical third, including the apical foramen region.
15. Describe how to determine clinically the distance from the occlusal-incisal surface to the roof of the chamber.
16. Discuss location, morphology, frequency, and importance of accessory (lateral) canals.
17. Describe relationships between anatomic apex, radiographic apex, and actual location of the apical foramen.
18. Describe common variations in pulp anatomy resulting from developmental abnormalities and state their significance.
19. Describe why many root curvatures are not apparent on standard radiographs.

In terms of success of treatment, knowledge of pulp anatomy cannot be overstated. As a cause of treatment failures, lack of a working knowledge of pulp anatomy ranks second only to errors in diagnosis and treatment planning. It is critical to know the normal or usual configuration of the pulp and to be aware of variations. Special techniques are required to determine the internal anatomy of the tooth under treatment.

Knowledge of pulp anatomy must be three-dimensional. The pulp cavity must be mentally visualized both longitudinally (from coronal aspect to apical foramen) and in cross-section. In addition to general morphologic features, irregularities and "hidden" regions of pulp are present within each canal. To clean and shape the pulp system maximally, intracanal instruments must reach as many of these regions as possible to plane the walls to loosen tissue and tissue remmants.[1] Lack of attention to this important principle may lead to treatment failure.

METHODS OF DETERMINING PULP ANATOMY

Textbook Knowledge

Gaining knowledge of anatomy from textbooks is the most important and most useful method of learning. Common and frequent variations *must* be memorized for each tooth. This means having a working knowledge of the number of roots, number of canals per root and their location, longitudinal and cross-sectional shapes, most frequent curvatures (particularly in the faciolingual plane), and root outlines in all dimensions.[2-6] It is useful to know the approximate percentage of each. Anatomic features are diagrammed in the Appendix.

Radiographic Evidence

Certainly, radiographs are useful, but they are somewhat overrated for determining pulp anatomy, particularly conventional periapical films.[7] The standard parallel facial projection gives just two dimensions; a common error is to examine only this view, overlooking the important third dimension (Figure 13-1). In addition, radiographs tend to make the canals look relatively uniform in shape and tapered. In fact, the aberrations often found are generally not visible (Figure 13-2).

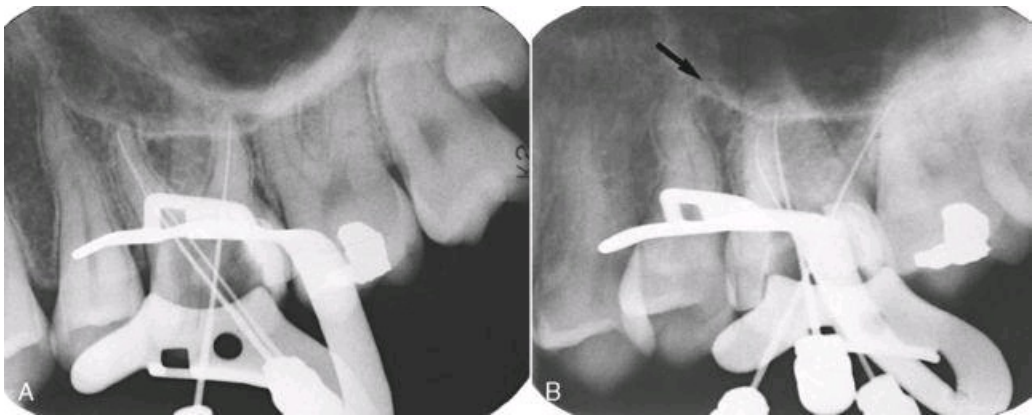

Figure 13-1 A, Facial projection. Both the second premolar and the first molar appear to have fairly straight buccal roots and an uncomplicated anatomy. **B,** Mesial angled projection. The more proximal view shows severe "bayonet" dilaceration of the second premolar with marked buccal curve in the apex *(arrow)*. Sharp curves of molar roots and two definitive canals in the mesiobuccal root are now evident. Both are difficult problems to treat.

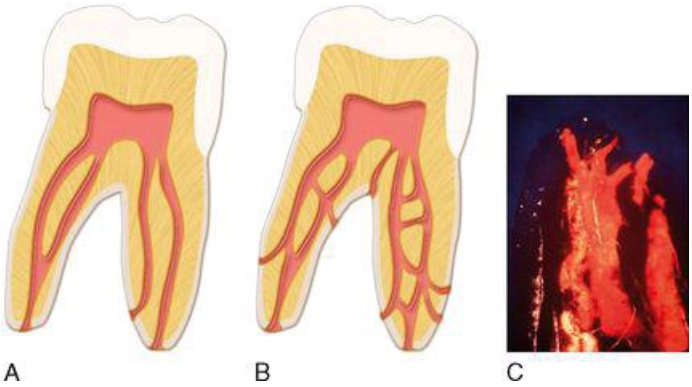

Figure 13-2 A, The impression of pulp anatomy received from viewing a radiograph. **B,** The reality: internal anatomy actually has many aberrations and intercommunications. **C,** Complex root canal system with multiple apical foramina is noted in the mesiobuccal root of a maxillary molar after dye injection. Note the multiple canals in apical region to form a delta.

*(**A** and **B**, Courtesy Dr. A. Goerig; **C**, Courtesy Dr. J. West.)*

Standard projections indicate general anatomic features. Special radiographic techniques disclose missed canals and determine curvatures.[8-10] These techniques are discussed in detail in Chapter 11.

Exploration

Additional determinations of pulp anatomy are made during access preparation and when searching for canals. These methods also have limitations because canals often are neither readily apparent nor easily discovered with instruments.[11]

GENERAL CONSIDERATIONS

A basic tenet in pulp root anatomy is *the shape of the pulp system reflects the surface outline of the crown and root.*[12,13] In other words, because the pulp tends to form the surrounding dentin uniformly on opposite walls, the pulp is generally a miniature version of the tooth and conforms to the tooth surface.[14]

Although root shape in cross-section is variable, there are seven general configurations: *round*, *oval*, *long oval*, *bowling pin*, *kidney bean*, *ribbon*, and *hourglass* (Figure 13-3). Shape and location of canals are governed by root shape (in cross-section). Different shapes may appear at any level in a single root. For example, a root may be hourglass-shaped in cross-section at the cervical third, taper to a deep oval in the middle third, and blend to oval in the apical third. The number and shape of canals in each level will vary accordingly.[15] It is important to note that a canal is seldom round at any level. To assume that it is may result in improper canal preparation.

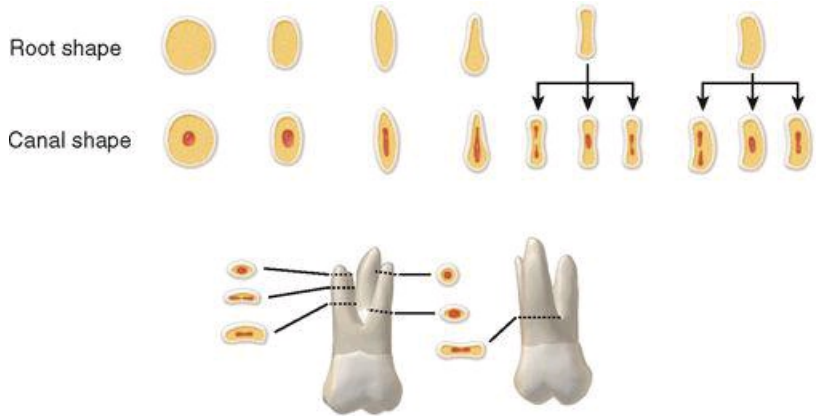

Figure 13-3 Common variations in root or pulp cross-sectional anatomy. Note that the pulp outline tends to reflect the root outline. Deep concave roots have a greater variety of pulp anatomies.

Knowledge of the more common variations in internal anatomy is an aid but does not give the final answer. This ultimately is determined during treatment. Root canals take various pathways to the apex. The pulp canal system is complex; canals may branch, divide, and rejoin. Canal systems have been categorized into four basic types.[16] However, Vertucci et al[17] utilized precise techniques and found a complex canal system and identified eight pulp space configurations. Other investigators[18-21] have carefully studied canal morphologies in different tooth groups, as well as examining gender and racial variations. The findings have consistently verified the earlier Vertucci et al[17] results.

Both gender and ethnic origin should be considered during the preoperative evaluation stage. Specific types of canal morphology occur in different racial groups. For example, African-Americans have a higher number of mandibular premolars with extra canals. These patients had more than one canal in 33% of first premolars and 8% of second premolars in contrast to Caucasians, who had multiple canals in 14% of first premolars and 3% of second premolars.[22]

Identification of Canals and Roots

Differentiation and identification of canal orifices are facilitated by following certain procedures and by identifying anatomic features (Box 13-1). Obviously, to clean, shape, and obturate a canal, it must first be located.[24] In roots that *may* contain two canals, a basic rule is to *assume that the root contains two canals until proved otherwise.* Rather than memorize roots that often contain two canals, it is easier to remember those few that are unlikely to have two canals. Maxillary teeth contain some roots that rarely have two canals: anterior roots, premolars with two or three roots, and distobuccal and lingual roots of molars. All other maxillary roots and all mandibular roots require a careful search for two (or possibly more) canals.

Box 13-1 The Laws of Location of Canal Orifices

The actual number and relationship of the canals are determined during treatment. The pulp chamber floor and walls provide a guide to determining the morphology and location of canal orifices.[23]

LAWS OF CANAL ORIFICES

1. *Symmetry 1:* Canal orifices are equidistant from a line drawn in a mesiodistal direction through the pulp-chamber floor (exception: maxillary molars).

2. *Symmetry 2:* Canal orifices lie on a line perpendicular to a line drawn in a mesiodistal direction across the center of the floor of the pulp chamber (exception: maxillary molars).

3. *Orifices' location 1:* Canal orifices are located at the junction of the walls and the floor.

4. *Orifices' location 2:* Canal orifices are located at the line angles in the floor-wall junction.

5. *Orifices' location 3:* Canal orifices are located at the terminus of the root development fusion lines.

6. *Color change:* Pulp chamber floor color is always darker than the walls.

ALTERATIONS IN INTERNAL ANATOMY

Again, initial pulp shape reflects root shape. However, because pulp and dentin react to their environment, changes in shape occur with increasing tooth age and in response to irritation.

Age

Although dentin formation tends to occur with age on all surfaces, it occurs predominately in certain areas. For example, in molars, the roof and floor of the chamber show more dentin formation, eventually making the chamber almost disclike in configuration (Figure 13-4). Treatment implications (difficulty in locating chamber and canals) are obvious.

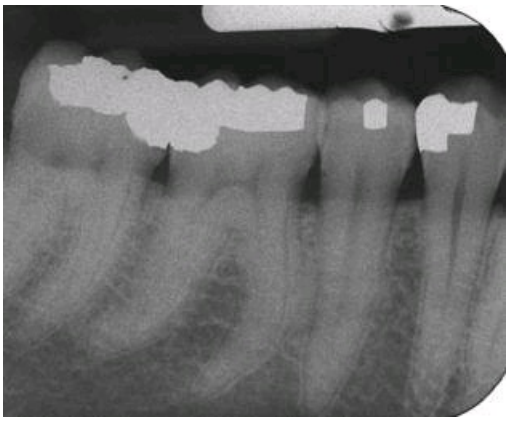

Figure 13-4 Note the disclike configuration of the pulp chamber in the first molar because of the predominance of dentin formation in the roof and floor of the chamber. These chambers are difficult to locate during access preparation.

Irritants

Anything that exposes dentin to the oral cavity can potentially stimulate increased dentin formation at the base of tubules in the underlying pulp.[14] Causes of such dentin exposure include caries, periodontal disease, abrasion, erosion, attrition, cavity preparations, root planing, and cusp fractures (Figure 13-5). Vital pulp therapy, such as pulpotomy, pulp capping, or placement of irritating materials in a deep cavity, may cause an increase in dentin formation, occlusion, calcific metamorphosis, resorption, or other unusual configurations in the chamber or canals. These tertiary (irregular secondary) dentin formations tend to occur directly under the involved tubules.

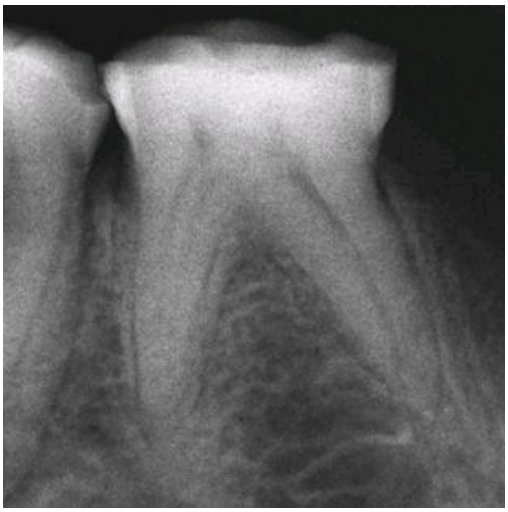

Figure 13-5 Severe attrition has resulted in tertiary dentin formation on the roof and floor and flattening of the chamber.

It is imperative that the clinician study radiographs and visually examine the tooth being treated to identify factors that may cause alterations in anatomy. Failure to do so may result in serious errors, lost time, and inadequate treatment.

Calcifications

Calcifications take two basic forms within the pulp: pulp stones (denticles) and diffuse calcifications. Although pulp stones are usually found in the chamber and diffuse calcifications within the radicular pulp, the reverse may also occur. These calcifications may form either normally or in response to irritation. Pulp stones are often seen on radiographs[25]; diffuse calcifications are visible only histologically.

Pulp stones in the chamber may reach considerable size and can markedly alter the internal chamber anatomy (Figure 13-6). Although they do not totally block a canal orifice, pulp stones often make the process of locating an orifice challenging. These large pulp stones may be attached or free and are often removed during access preparation. Although pulp stones are not common in canals, if present, they are usually attached or embedded in the canal wall in the apical region. Rarely do they form a barrier to instrument passage.

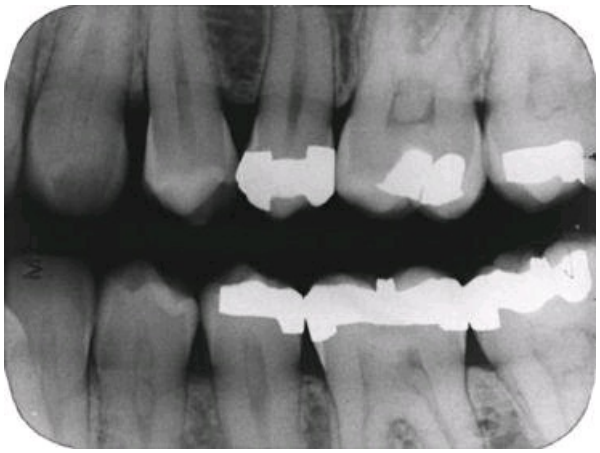

Figure 13-6 Calcifications (pulp stones [or denticles]) are visualized in the chambers. Their discrete appearance surrounded by radiolucent spaces shows these calcifications to be natural and not formed in response to irritation.

(Courtesy Dr. T. Gound.)

Such resorptions are uncommon and when present are usually not extensive. They also are a response to irritation that is sufficient to cause inflammation. Most resorptions are small and are not detectable on radiographs or during canal preparation. When visible radiographically, they are usually extensive and often perforate. Internal resorptions usually create operative difficulties (Figure 13-7).

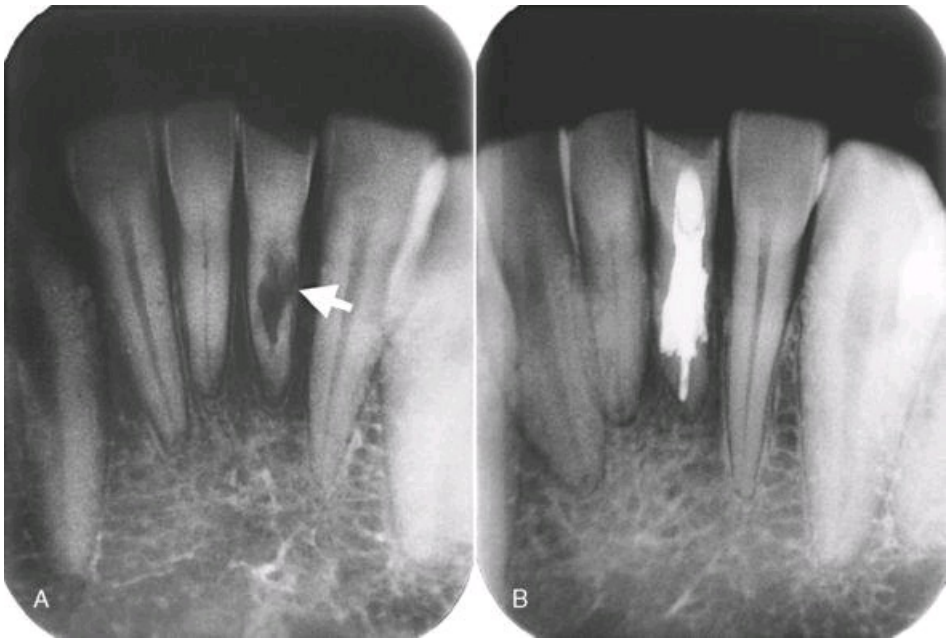

Figure 13-7 A, Extensive internal resorption defect *(arrow)*. **B,** Four years after treatment. Special cleaning, shaping, and obturating techniques (lateral condensation plus thermoplasticized) were required, resulting in successful treatment.

COMPONENTS OF THE PULP SYSTEM

The pulp cavity is divided into a coronal (the pulp chamber) and a radicular portion (the root canal). Other features include *pulp horns*, *canal orifices*, *accessory (lateral) canals*, and the *apical foramen* (Figure 13-8). The internal anatomy of these pulp components is altered by secondary dentin or cementum formation.

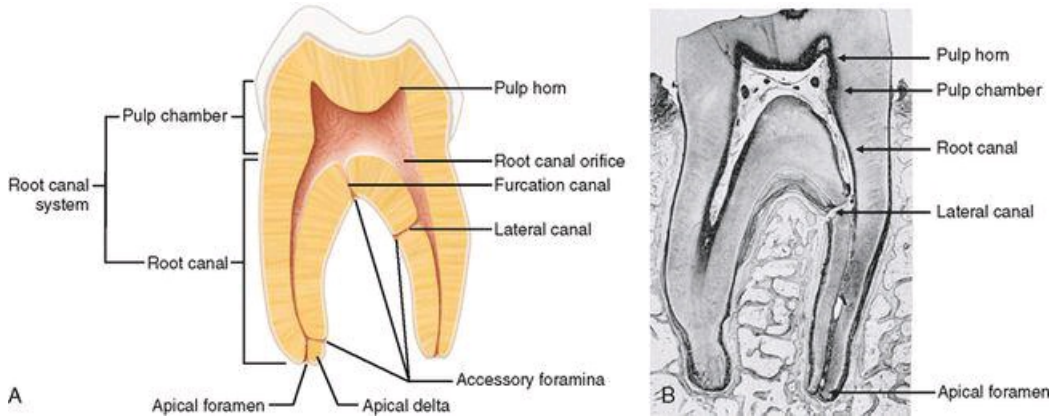

Figure 13-8 A and **B,** Major anatomic components of the pulp cavity.

*(**A**, From Cohen S, Hargreaves K, Keiser K: Pathways of the pulp, ed 9, Mosby, 2006, St. Louis.)*

Pulp Horns

Pulp horns represent what the dentist does not want to locate during restorative procedures but does want to locate during access preparation. Although they may vary in height and location, a single pulp horn tends to be associated with each cusp in a posterior tooth, and mesial and distal horns tend to be in incisors. Generally, the occlusal extent of the pulp horns corresponds to the height of contour in a younger tooth but because of continued dentin formation lies closer to the cervical margin in an older tooth.

During access preparation, the height and location of pulp horns may be more accurately determined by measuring from occlusal surface or cusp tip to pulp horn or chamber roof using a bur and a handpiece (Figure 13-9).

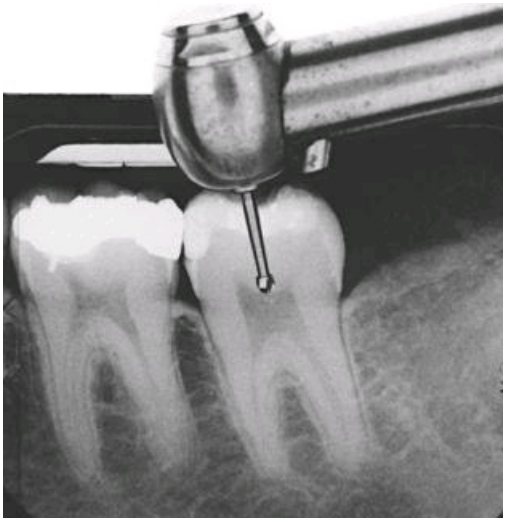

Figure 13-9 A technique for determining the distance from the occlusal surface to the roof of the pulp chamber. This is an obvious benefit during access preparation to prevent perforation.

Pulp Chamber

The pulp chamber occupies the center of the crown and trunk of the root. Again, its shape, in both longitudinal and cross-sectional dimensions, depends on the shape of the crown and trunk; this configuration varies with tooth age and irritation.[12] In mature molars, the roof of the chamber is approximately at the level of the cementoenamel junction.[26]

Root canals extend the length of the root, beginning as a funneled orifice and exiting as the apical foramen. Most canals are curved, often in a faciolingual direction.[27,28] Therefore a curved canal is often undetectable on facial projection radiographs. As a result, the uninitiated or uninformed clinician may assume that a canal is straight and may overenlarge what is in reality a facial or lingual curvature, resulting in ledging or perforation. *The operator should always assume that a canal is curved.*

Canal shape varies with root shape and size, degree of curvature, and the age and condition of the tooth (see Figures 13-2 and 13-3). As a rule, when two canals occur in a root, they tend to be more oval. In the deep faciolingual root with mesial or distal (or both) concavities (hourglass or kidney bean shape), a single canal may have a bowling pin, kidney bean, hourglass, or ribbon shape. Regardless of the shape in the cervical third, in the apical curvature the root (and canal) tends to become more oval but may be somewhat flattened.[29] A canal that is oval in the cervical one-third usually is oval or ribbon the apical few millimeters.

The shape and number of canals in a root reflect the faciolingual depth and shape of the root at each level (Figure 13-10); the deeper the root, the more likely that there are two separate, definitive canals. If the root tapers toward the apical third, there is a greater likelihood that the canals will converge to exit as a single canal.

Figure 13-10 Maxillary first premolar roots. A common finding is a cross-section through buccal *(B)* and lingual *(L)* roots that shows a concavity on the lingual surface and an irregular canal shape in the middle third of the buccal root.

(Courtesy Dr. A. Tamse.)

Irregularities and aberrations are common and particularly true in posterior teeth. Such aberrations include hills and valleys in canal walls, intercanal communications (isthmuses between two canals),[30] cul-de-sacs, fins, and other variations. Again, these aberrations are usually neither accessible to instruments or irrigants nor are they consistently obturated.

The chamber tends to occupy the center of the crown; a canal occupies the center of the root. When there are two canals in a root, each will often occupy the center of its own root “bulge.”

Accessory Canals

Accessory (or lateral) canals are lateral branches of the main canal that form a communication between the pulp and periodontium. They contain connective tissue and vessels and may be located at any level from furcation[31-33] to apex but tend to be more common in the apical third and in posterior teeth.[34] In other words, the more apical and the farther posterior the tooth, the more likely that accessory canals will be present. The relationship of accessory canals to pulp health and disease, as well as to treatment, is debatable.[35] They do not supply collateral circulation and therefore contribute little to pulp function and probably represent an anomaly that occurred during root formation.

These canals do form an exit for irritants from the pulp space to the lateral periodontium. They probably cannot be débrided during cleaning and shaping[1] but are occasionally filled with obturating materials during canal filling (Figure 13-11). Débriding and obturating lateral canals are not important for success of root canal treatment.[22,36]

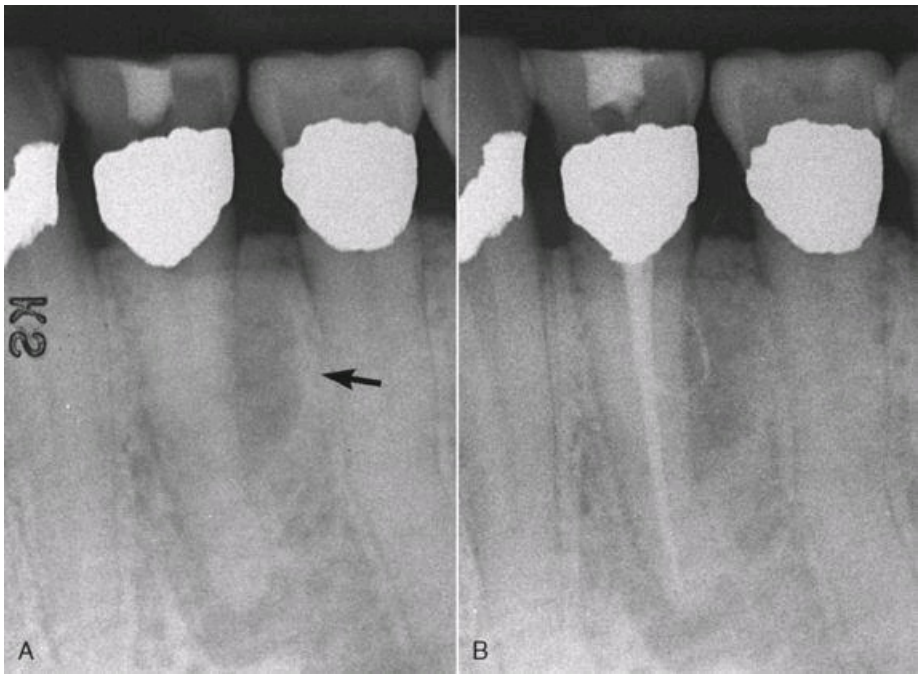

Figure 13-11 A, Resorptive bony lesion *(arrow)* usually indicates an accessory canal (not visible) that is a pathway for irritants. **B,** The accessory canal is now obvious after obturation.

Apical Region

Development

The apex is the root terminus. It is relatively straight in the young mature tooth but tends to curve more distally with time. This curvature results from continued apical-distal apposition of cementum in response to continued mesial-occlusal eruption. Alterations in the apical region may also result from resorption and irregular cementum apposition. Thus apical anatomy tends to be nonuniform and unpredictable.[37-41]

Apical Foramen

The apical foramen varies in size and configuration with maturity. Before maturation, the apical foramen is open. With time and deposition of dentin and cementum, it becomes smaller and funneled. Significantly, the foramen usually does not exit at the true (anatomic) root apex[42,43] but is offset approximately 0.5 mm and seldom more than 1.0 mm from the true apex. The degree of deviation is unpredictable and may vary considerably from the average, particularly in the older tooth that has undergone cementum apposition (Figure 13-12). For this reason, root canal preparation and obturation end short of the anatomic root apex (Figure 13-13) as seen in the radiograph.[44] Usually, the apical foramen is not visible radiographically. The clinician relies on averages or on electronic measuring devices to determine the extent of canal preparation and obturation.[37,42]

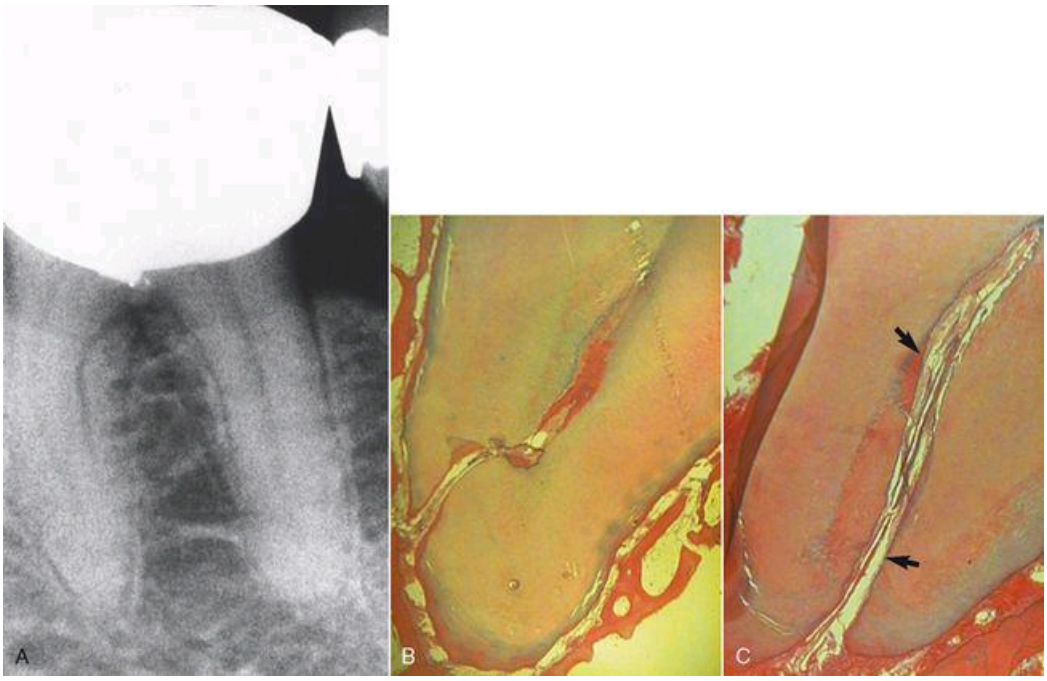

Figure 13-12 Variations in apical canal anatomy. **A,** The radiograph often does not demonstrate the size, shape, or curvature of canals apically. **B,** Mesial root apex showing abrupt curve and apical foramen exiting on the mesial well short of the anatomic apex. **C,** Distal root apex showing uniform canal with no constriction and variable levels *(arrows)* of cementodentinal junctions; these variabilities are common.

(Cadaver specimen courtesy Dr. D. Holtzmann.)

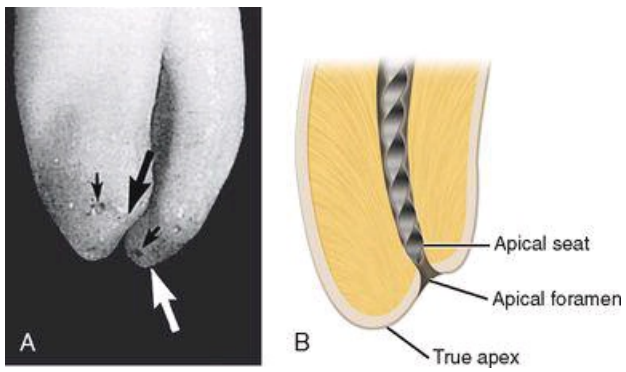

Figure 13-13 A, The apical foramina *(small arrows)* do not correspond to the true anatomic apex *(large arrows).* **B,** In most situations the apical

terminus or seat of the preparation will vary from the apical foramen and radiographic apex.

(A, Courtesy Dr. D. Melton.)

Variations in Anatomy

The only consistent aspect of the apex region is its inconsistency.[39,45] The canal may take twists and turns, divide into several canals to form a delta with ramifications on the apical root surface, or exhibit irregularities in the canal wall (Figure 13-14). Generally, these aberrations are neither detectable nor predictably negotiable and are neither well débrided nor obturated.

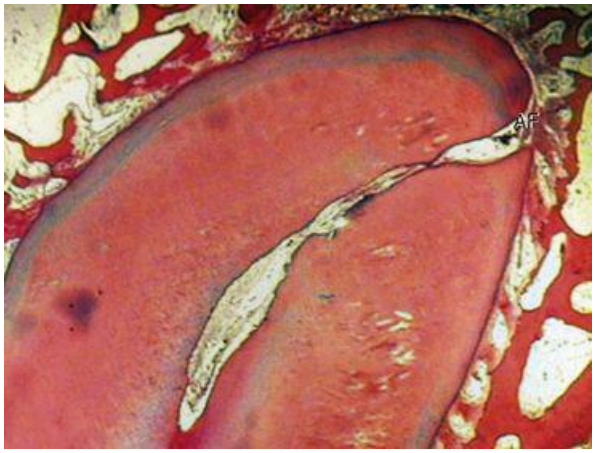

Figure 13-14 The apical region of the canal and apical foramen *(AF)* are often very irregular.

A common concept is that canals round out in this apical region. This is not always true. Canals are frequently a long oval or even ribbon-shaped apically.[46] These nonround canals cannot be enlarged to a round shape without perforating or weakening the roots.[46]

Apical Constriction

The presence of an apical constriction is unpredictable. Frequently there is no apical constriction. It has been proposed that the cementodentinal junction forms the apical constriction; however, this concept is incorrect. In fact, the junction is difficult to determine clinically with accuracy,[42] and the intracanal extent of cementum is variable. If an apical constriction is present, it is not visible on a radiograph and usually is not detectable with tactile sense using a file, even by the most skilled practitioner.

VARIATIONS OF ROOT AND PULP ANATOMY

Representative examples of the tooth groups are diagrammed in the Appendix, where both cross-sectional and longitudinal aspects are outlined. In addition, the pulp anatomy of each is shown in relation to the design of the access preparation.

Occasionally, teeth vary significantly in root or, more likely, in pulp anatomy. Such variations and abnormalities are most common in the maxillary lateral incisors, maxillary[47-49] and mandibular premolars,[50] and maxillary molars.[51,52] Unusual root morphology tends to be bilateral.[53]

Dens Invaginatus (Dens in Dente)

Dens invaginatus, which is most common in maxillary lateral incisors,[54] results from an infolding of the enamel organ during proliferation and is an error in morphodifferentiation (Figure 13-15). It often results in an early pulp-oral cavity communication requiring root canal treatment.[55] Dens invaginatus shows varying degrees of severity and complexity.[56,57] The more severe cases should be referred to a specialist because special treatment, such as surgery, is frequently required. The prognosis of any treatment often is questionable. The invagination is usually visible on radiographs; however, it is often small and obscure. The lingual pit on maxillary anterior teeth represents a minor form of dens invaginatus.

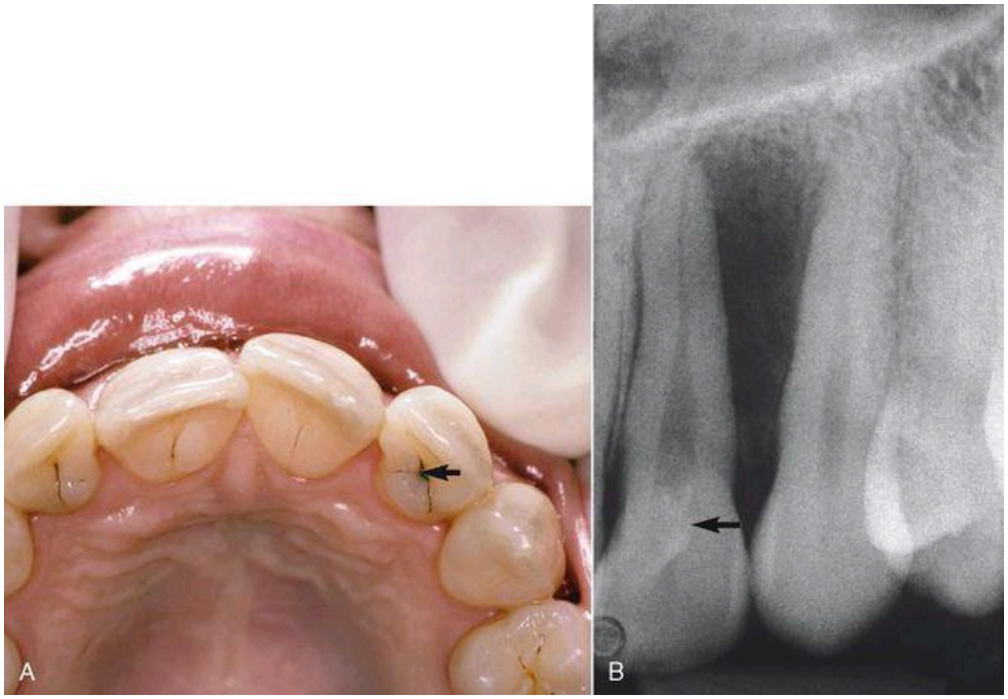

Figure 13-15 Dens invaginatus. **A,** The invagination is visible on the lingual surface *(arrow)* of this abnormally shaped incisor. **B,** The invagination is lined internally by enamel *(arrow).* By communicating with the pulp, the exposure resulted in pulp necrosis and apical pathosis. These situations are difficult to treat.

(Courtesy Dr W. Johnson.)

Dens Evaginatus

A variation of dens invaginatus,[58] dens evaginatus is most common in mandibular premolars and in individuals with Asian ancestry, as well as Native Americans and Hispanics. Clinically, dens evaginatus initially appears as a small tubercle "bulge" on the occlusal surface, but it may not be obvious radiographically (Figure 13-16). These tubercles often contain an extension of the pulp. When these fragile tubercles fracture off, the pulp is exposed and will become necrotic, requiring apexification. There are different treatment measures to prevent this accidental exposure of the pulp. One method, before the tubercle fractures, is to remove the tubercle with a bur and then cap, followed by a good sealing restoration with amalgam.[59]

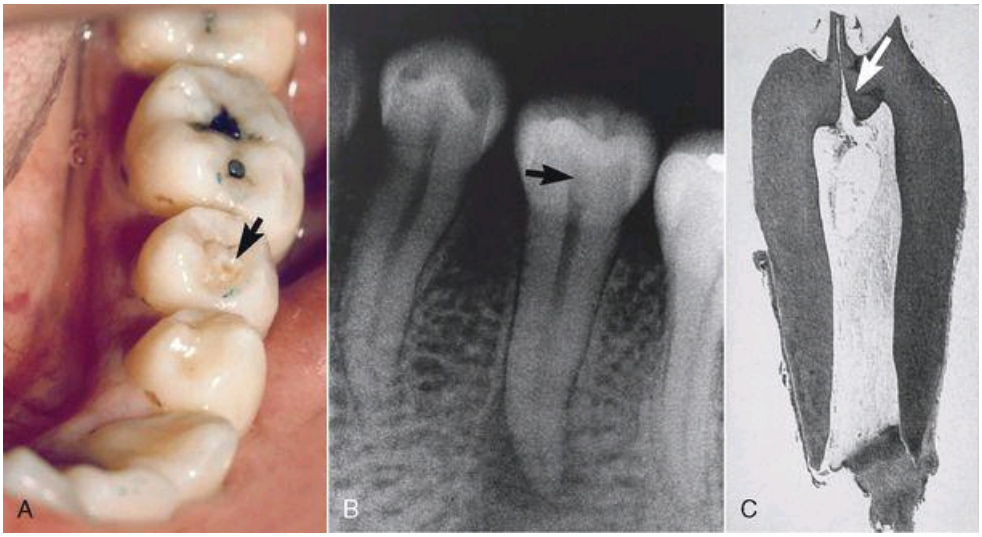

Figure 13-16 Dens evaginatus. **A,** The second premolars show two stages: the arrow indicates the tubercle on the occlusal surface. **B,** The tubercle has fractured *(arrow),* exposing the pulp. **C,** Histologic section demonstrates the extension of the pulp into the evagination tubercle *(arrow).*

*(**A** and **B**, Courtesy Dr. W. Johnson.)*

High Pulp Horns

Occasionally, a pulp horn extends far into a cusp region, resulting in premature exposure by caries or accidental exposure during cavity preparation. These high pulp horns are often not visible on radiographs. This is most common in the mesiobuccal of first molars.

Lingual Groove

Usually found in maxillary lateral incisors, a lingual groove appears as a surface infolding of dentin oriented from the cervical toward the apical direction (Figure 13-17) .[60] Frequently, this results in a deep narrow periodontal defect that occasionally communicates with the pulp, causing an endodontic/periodontal problem (Figure 13-18). Treatment is difficult and unpredictable, so prognosis is poor. Usually these teeth require extraction.

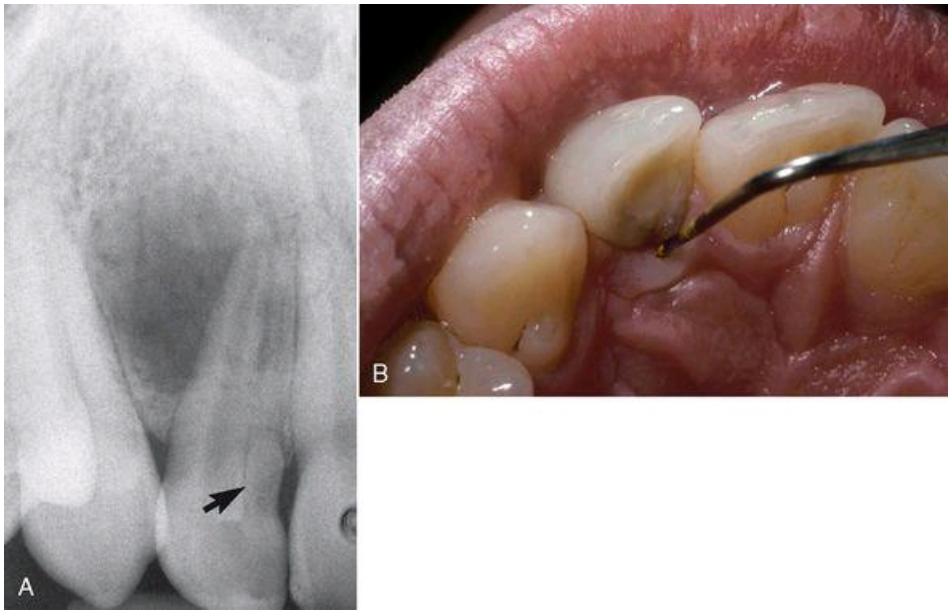

Figure 13-17 Lingual groove, which is an apparent infolding during root and crown formation. **A,** The groove is faintly visible on the periapical radiograph *(arrow).* **B,** The groove is often detected on the surface with probing and is usually untreatable.

(Courtesy Dr. K. Baumgartner.)

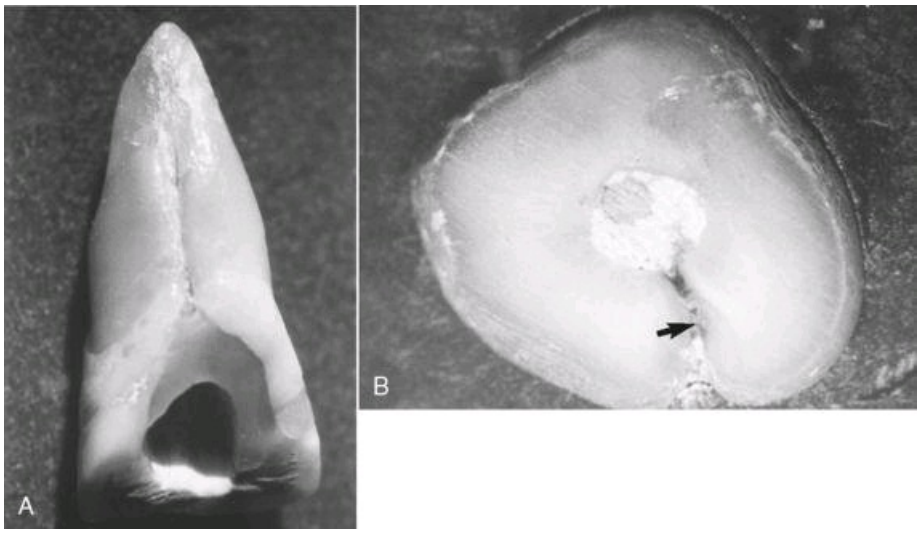

Figure 13-18 A, Lingual groove defect proved untreatable periodontally and endodontically. **B,** Cross-section shows that the groove invagination *(arrow)* communicates with the pulp.

Dilaceration

By definition, dilaceration is a severe or complex root curvature (see Figure 13-1). During root formation, structures, such as the cortical bone of the maxillary sinus or the mandibular canal or nasal fossa, may deflect the epithelial diaphragm, resulting in a severe curvature. Many of these curvatures are found in a faciolingual plane and are not obvious on standard radiographic projections.

Other Variations

Many other pulp and root anomalies may occur.[61-65] Some occur in association with genetic disorders,[66] such as variations in the number of canals or roots (Figure 13-19). Teeth with unusual chamber and root canal configurations have an impact on treatment.[67,68] The astute clinician will be alert to these possibilities and will study radiographs and occlusal anatomy carefully. A common abnormality is the **C**-shaped canal (see Appendix and Figure 13-20). This usually occurs in mandibular second molars and is more common in Asian individuals.[69] Because of the complex internal anatomy, prognosis of root canal treatment is questionable because of difficulty in adequate débridement and obturation.[70] Additional treatment measures may be required, and patients with such teeth should be considered for referral.

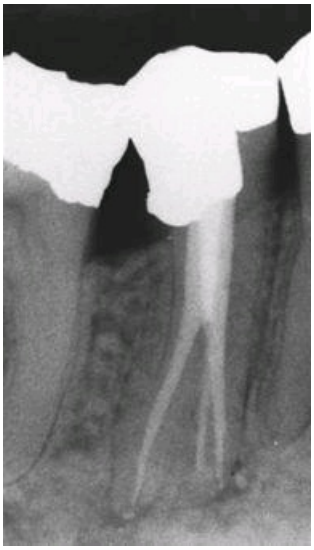

Figure 13-19 Premolar with three canals—a challenge to treat.

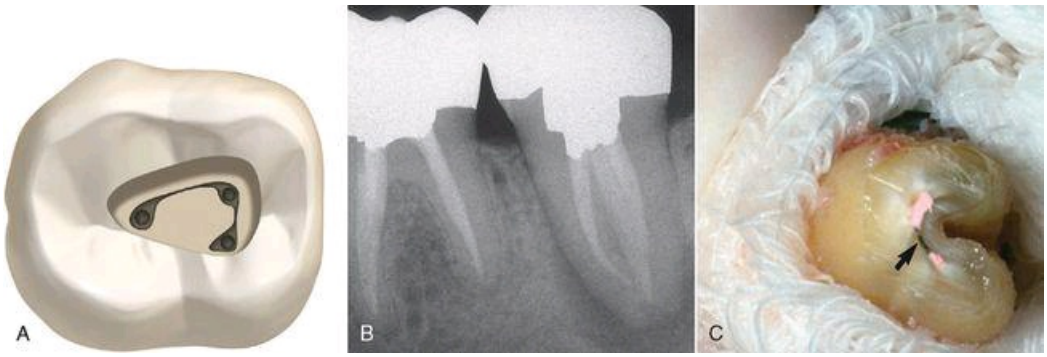

Figure 13-20 A, C-shaped pulp chamber. The C-space may be continuous throughout the length of the root but is variable anatomically. More commonly, three separate canal orifices may be found within the groove. **B,** C-shaped molar shows failed treatment. **C**, After extraction and resection of the apical one-third, the undébrided and unobturated groove is evident *(arrow).*

Another unusual chamber and root morphology is the occasional three-rooted maxillary premolar (see Appendix and Figure 13-21). Another departure from the usual is the distolingual third root on mandibular first molars (see Appendix and Figure 13-22). These can occur in any race and also may be a challenge to treat.[71]

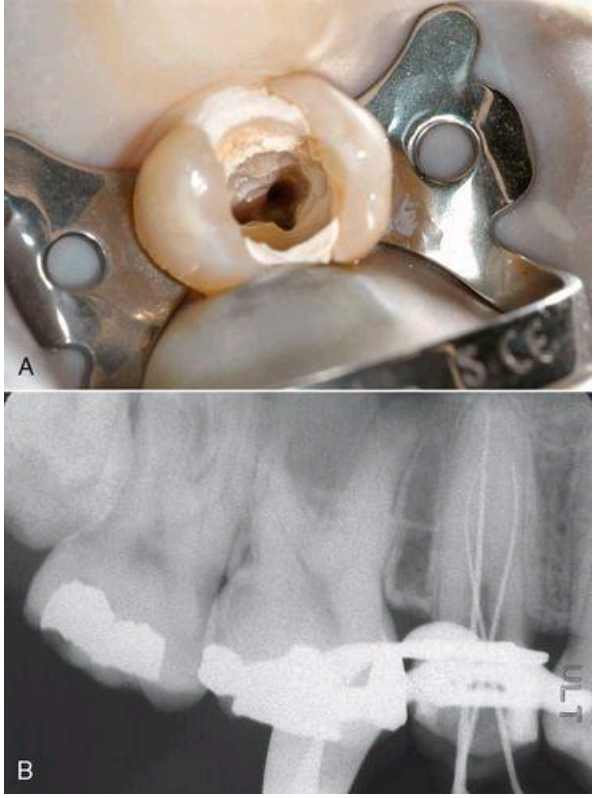

Figure 13-21 Three-rooted/three-canaled premolar. **A,** The access shows three distinct orifices. **B**, The radiographic appearance is similar to a maxillary molar.

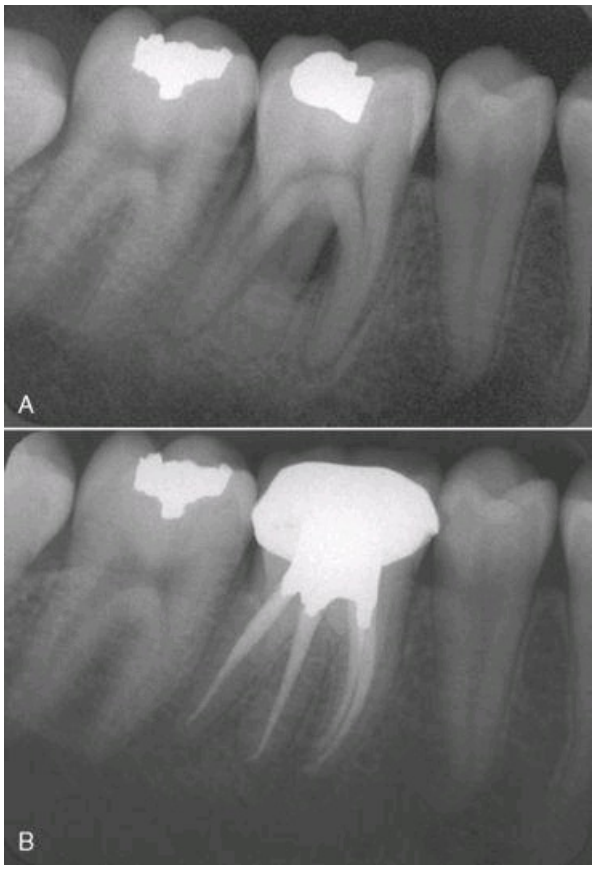

Figure 13-22 Three-rooted mandibular molar. **A,** Pulp necrosis has caused apical and furcal bony lesions. **B,** Several months after treatment. The resorptive lesions have resolved.

(Courtesy Dr. A. Law.)

New technologies, using computer reconstruction of microtomography, are able to provide 3-dimensional morphologic information on tooth and pulp anatomy.[13,72] Unfortunately, this technology is not yet practical for routine use in dentistry.

Chapter Review Questions available in Appendix B or on the DVD

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Isolation, Endodontic Access, and Length Determination

William T. Johnson, Anne E. Williamson

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe the reasons for rubber dam isolation during endodontic procedures.
2. List a rubber dam clamp selection for anterior, premolar, and molar teeth.
3. Identify those clamps that have several applications. Which two are “universal”?
4. Describe techniques for application of clamp/rubber dam in single-tooth isolation.
5. Describe techniques to stop salivary or hemorrhagic “seepage” into the operative field.
6. Recognize situations in which special isolation approaches are necessary.
7. Describe techniques utilized in special isolation situations.
9. Identify patients with difficult isolation situations who should be considered for referral.
10. Identify major objectives of access preparation in both anterior and posterior teeth.

11. Describe why straight-line access is critical.

12. Explain the importance of pulp horn removal in anterior teeth.

13. Relate reasons and indications for removing caries or restorations during access preparation.

14. Explain the reason and technique for removing the dentin shelf in anterior and posterior teeth.

15. Describe the procedure, burs used, and sequence of operations to start and complete access preparations on various teeth.

16. Identify common errors for specific teeth that may occur during access preparation.

17. Recognize when these errors occur and know how to correct them (if correctable).

18. Describe techniques for locating difficult-to-find chambers or canals.

19. Demonstrate the step-by-step technique for obtaining estimated and final working lengths.

20. Account for conditions under which working length (distance from radiographic apex) varies.

21. Describe how to designate and maintain (and create, when necessary) a stable reference point.

22. Describe electronic apex locators'how they function and when they are useful.

Refer to the Appendix:

23. Diagram the portions of the tooth that must be removed to attain straight-line access to the canals. Illustrate this on sagittal sections of both anterior and posterior teeth.

24. Diagram the outline form of the access preparation for all teeth.

25. Show the location of each canal orifice relative to the occlusal or lingual surface.

Chapters 14, 15, and 17 deal with the technical aspects of nonsurgical root canal treatment. Areas presented include isolation, access, length determination, cleaning and shaping, and obturation. A number of instruments and techniques are advocated for treatment procedures. These chapters introduce concepts and principles that must be met for successful treatment. These building blocks of treatment are based on the best available evidence and provide a basis on how to incorporate more complex and alternative techniques.

ISOLATION

Rubber Dam Application

Application of the rubber dam for isolation during endodontic treatment has many distinct advantages and is mandatory for legal considerations.[1] The use of the rubber dam in the United States is considered the standard of care; thus expert testimony is not required in cases when patients swallow or aspirate instruments or materials because juries are considered competent to determine negligence.

The rubber dam provides protection for the patient,[2] and it creates an aseptic environment that enhances vision, retracts tissues, and makes treatment more efficient. Soft tissues are protected from laceration by rotary instruments, chemical agents, and medicaments. Irrigating solutions are confined to the operating field, and most important, rubber dam isolation protects the patient from swallowing or aspirating instruments and materials (Figure 14-1).[3] An additional advantage is that the dentist and auxiliary employees are also protected.[4] The risk from aerosols is minimized,[5,6] and the dam provides a barrier against the patient's saliva and oral bacteria. Application of the rubber dam may also decrease the potential for transmission of systemic diseases such as acquired human immunodeficiency syndrome (AIDS), hepatitis, and tuberculosis.[4]

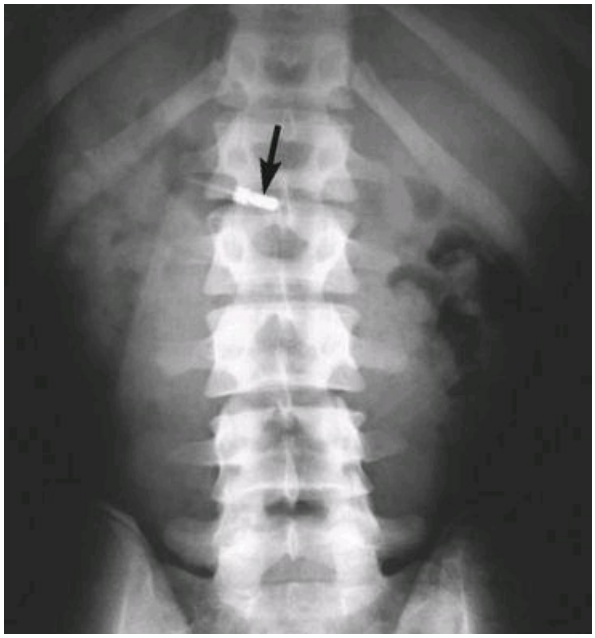

Figure 14-1 Note a file *(arrow)* that a patient swallowed during endodontic treatment.

The rubber dam is manufactured from latex; however, nonlatex rubber dam material is available for patients with latex allergy (Figure 14-2). The rubber dam can be obtained in a variety of colors that provide contrast to the tooth. The thickness also varies (light, medium, heavy, and extra heavy). A medium-weight dam is recommended because a lightweight dam is easily torn during the application process. Also, the medium material fits better at the gingival margin and provides good retraction.

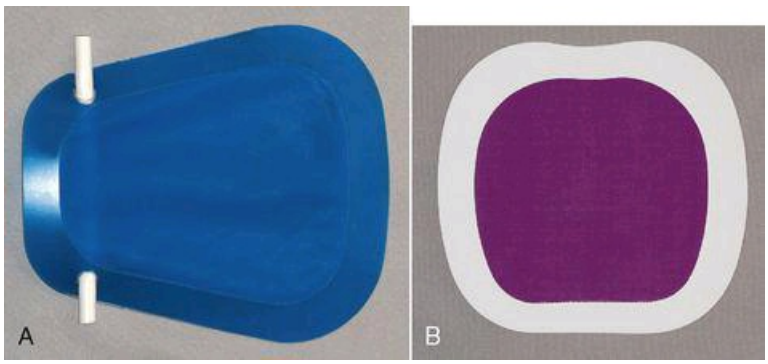

Figure 14-2 A and **B,** Disposable rubber dam systems.

The design of the rubber dam frames is also variable. For endodontics, plastic frames are recommended; they are radiolucent and do not require complete removal during exposure of interim films such as the working length and master cone radiographs/digital images.

Rubber Dam Retainers

Rubber dam clamps are manufactured to fit the various tooth groups. During routine treatment, metal clamps are adequate; however, they may damage tooth structure[7] or existing restorations. Some have serrated edges to enhance retention when minimal coronal tooth structure remains. Plastic clamps are manufactured and have the advantage of being radiolucent. This is an advantage in difficult cases where the pulp chamber and canal cannot be located. Metal clamps often must be removed when exposing a radiograph for orientation purposes. When using a plastic clamp, the rubber dam can remain in place. The plastic clamps are less likely to damage tooth structure or existing restorations.[8]

Types

Different styles and shapes are available for specific situations. The following selection is recommended: anterior teeth: Ivory No. 9 or No. 212; premolars: Nos. 0 and 2; and molars: Nos. 14, 14A, and 56. The clamps that will manage most isolation situations during root canal treatment are shown in Figure 14-3. The use of winged clamps permits the application of the rubber dam as a single unit during single-tooth isolation (Figure 14-4).[9]

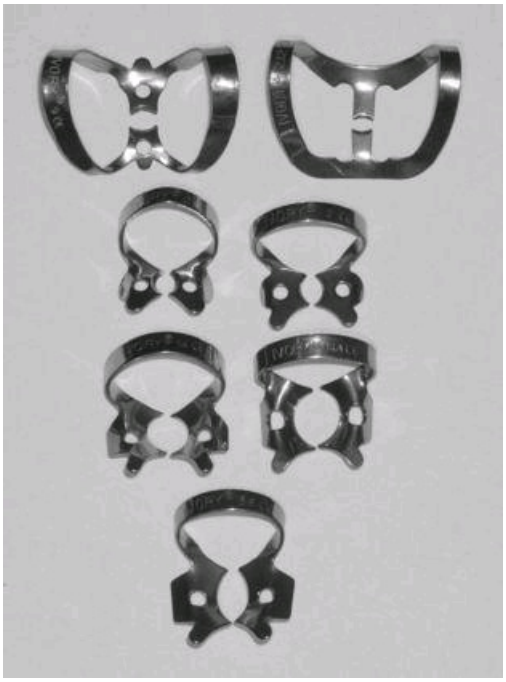

Figure 14-3 The retainers in the first row (Nos. 9 and 12) are designed for anterior teeth but are useful for premolars. The two clamps in the second row (Nos. 0 and 2) are for premolars and anterior teeth. The third row clamps (Nos. 14 and 4A) are for molars. Nos. 14 and 14A are deeper reaching than the No. 56. The bottom row clamp (No. 56) is more universal and is used for most molars.

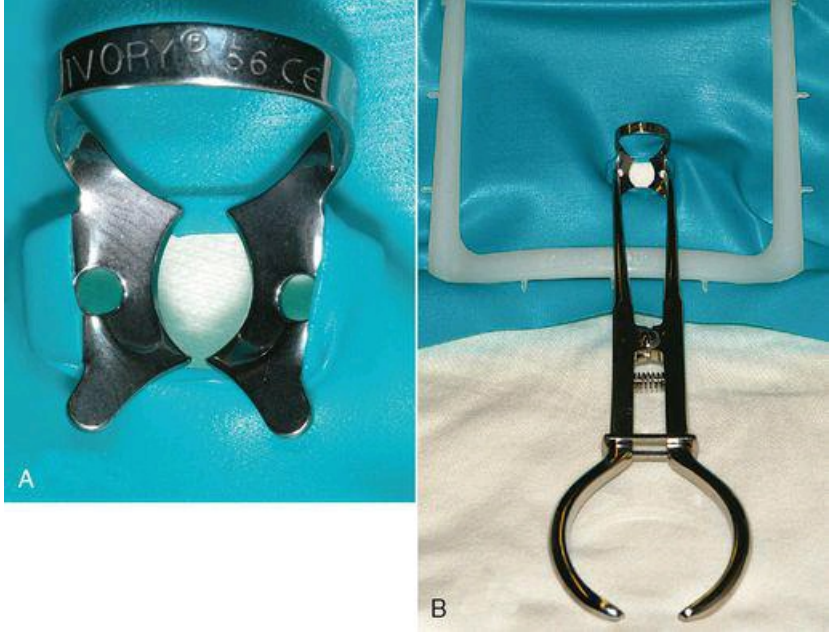

Figure 14-4 A, Placement of the rubber dam as a single unit requires the use of a winged clamp. A hole is punched in the rubber dam and then stretched over the wings of the appropriate clamp. **B,** The rubber dam is attached to a plastic radiolucent frame, and the rubber dam forceps is then used to carry the unit to the tooth.

Universal Clamp Designs

Two designs (see Figure 14-3), the "butterfly" Ivory No. 9 and the Ivory No. 56, are suitable for most isolations. The butterfly design (No. 9) has small beaks, is deep reaching, and can be applied to most anterior and premolar teeth. The No. 56 clamp will isolate most molars.

With teeth that are smaller, reduced in crown preparation, or abnormally shaped, a clamp (Nos. 0, 9, or 14) with smaller radius beaks is necessary. Small radius beaks can be positioned farther apically on the root, which will stretch the dam cervically in the interproximal space.

Additional Designs

Clamps that may be most useful when little coronal tooth structure remains have beaks that are inclined apically. These are termed *deep-reaching* clamps. Clamps with serrated edges are also available for cases with minimal coronal structure. These clamps should not be placed on porcelain surfaces because damage may occur.[7]

For stability, the clamp selected must have four-point contact between the tooth and beaks. Failure to have a stable clamp may result in damage to the gingival attachment and coronal structure[7,10] or be dislodged. Clamps may also be modified by grinding to adapt to unusual situations.[11]

Placement of the rubber dam as a single unit is fast and efficient. Once in place the dam is flossed through the contacts, and the facial and lingual portions of the dam are flipped under the wings.

Identification of the tooth requiring treatment is usually routine. However, in cases where there are no caries or restorations present, the operator may clamp the *wrong* tooth. This can be avoided by marking the tooth before rubber dam application or by beginning the access after placement of a throat pack without the rubber dam in place.

Preparation for Rubber Dam Placement

Before initiating treatment, degree of difficulty in obtaining adequate isolation must be assessed. Often, teeth requiring root canal treatment have large restorations, caries, or minimal remaining tooth structure that may present complications during isolation and access. Adequate isolation requires that caries, defective restorations, and restorations with leaking margins be removed before treatment. This preparation ensures an aseptic field of operation, allows assessment of tooth restorability, and permits temporization between visits.

Once the treatment plan is finalized, it may be necessary to perform ancillary procedures to allow for placement of the rubber dam.[12,13]

Isolation of Teeth with Inadequate Coronal Structure

Ligation, the use of deep-reaching clamps, bonding, and clamping the gingiva are the major methods of isolating teeth without adequate coronal tooth structure. Surgical management may also be required (Figure 14-5).

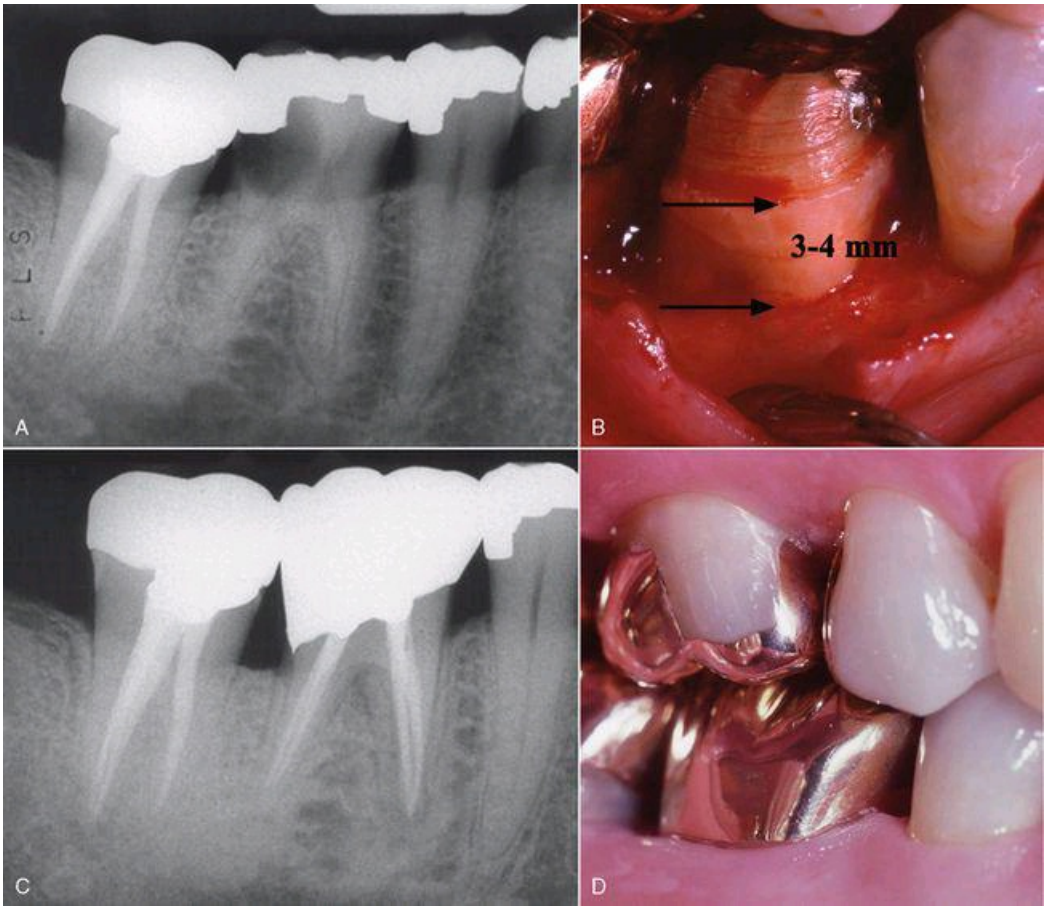

Figure 14-5 A, The first molar exhibits extensive caries on the distal extending to the crestal bone. **B,** A full-thickness mucoperiosteal flap and osseous reduction after caries excavation and preparation for a provisional crown, then 3 to 4 mm of tooth structure coronal to the osseous crest restores the biologic width. **C,** Root canal treatment and placement of the crown. **D,** The definitive restoration.

Ligation

Inadequate coronal structure is not always the cause for lack of retention. In young patients the tooth may not have erupted to the point that the cervical area is available for clamp retention. In these cases, ligation with floss or the use of interproximal rubber Wedjets is indicated (see Figure 14-15, *D*). Another approach is multiple tooth isolation.

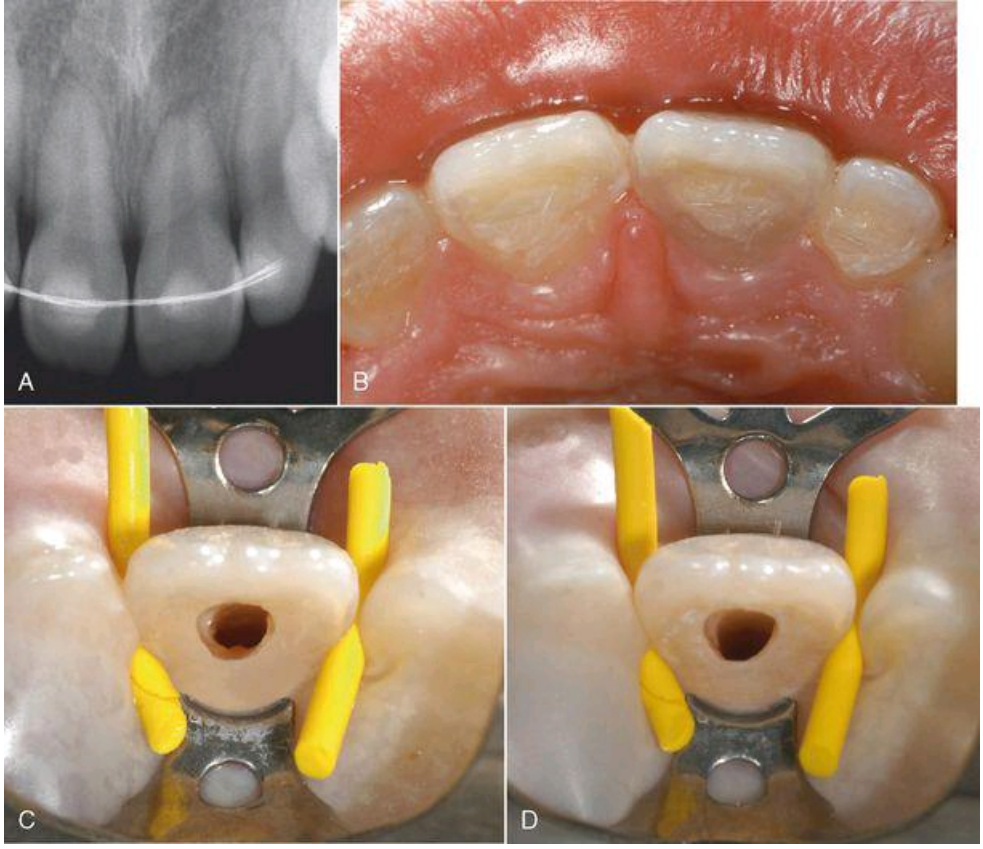

Figure 14-15 A maxillary left central incisor exhibiting pulp necrosis. **A**, A large pulp space with pulp horns that requires a triangular access outline. **B,** Lingual surface after removal of the orthodontic retaining wire. Note No. 9 is slightly discolored. **C,** The initial triangular access form exposing the chamber. Note the lingual shelf has not been removed to expose the lingual wall. **D,** Removal of the lingual shelf and the access complete.

Deep-Reaching Clamps

When the loss of tooth structure extends below the gingival tissues but there is adequate structure above the crestal bone, a deep-reaching clamp is indicated. It may be necessary to use a caulking material around the clamp to provide an adequate seal (Figure 14-6). Another option is using an anterior retainer regardless of the tooth type.

Figure 14-6 A, Caulking and putty materials are available to prevent leakage after rubber dam application. **B,** Application of the caulk. **C,** The sealed dam.

Bonding

When there is missing tooth structure, including the natural height of contour, retention can be increased by bonding resin on the facial and lingual surfaces of the remaining tooth structure.[14] The clamp is placed apical to the resin undercut. After treatment the resin is easily removed. This technique is preferred over the more invasive technique of cutting horizontal grooves in the facial and lingual surfaces for the prongs of the clamp.

Clamping the Gingiva

When the loss of tooth structure extends below the gingival tissues or below the crestal bone, clamping the gingival tissues is an option. This produces minimal damage and the tissues heal. Postoperative discomfort is minimal (see Figure 14-16, *D*).

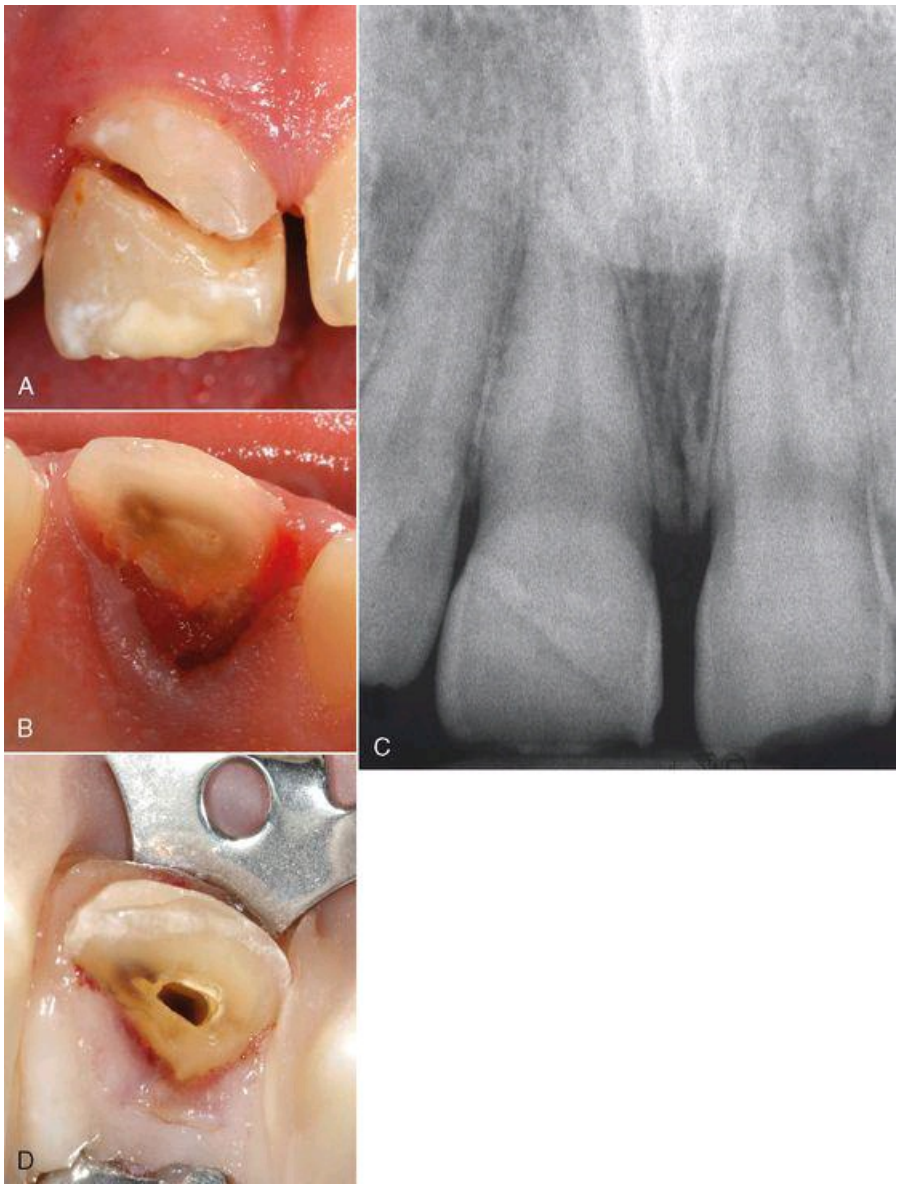

Figure 14-16 A crown root fracture. **A,** Initial presentation demonstrating fragment separation. **B,** The lingual surface with the segment removed. **C,** Preoperative radiograph. **D,** The extent of the fracture subgingival requires a unique approach to isolation. Note that a premolar clamp is placed on the gingival tissues for isolation.

Replacement of Coronal Structure

Temporary Restorations

When there is missing tooth structure but adequate retention, missing structure can be restored with reinforced intermediate restorative material (IRM) containing zinc oxide–eugenol, glass ionomers, or resins. These materials provide an adequate coronal seal and are stable until the definitive restoration is placed. Bonded materials provide a better seal with improved strength and esthetics, but placement is more time consuming.

Coronal Buildups

Coronal buildups replace missing structure when the tooth exhibits inadequate retention for a temporary.[15] They are rarely required and are time consuming. Materials include amalgam and resins. Special retention is required, and anatomic landmarks are often lost.

Band Placement

Placement of orthodontic bands is a better option if a buildup is being considered. They are available in various sizes and are appropriately contoured. A band can be cemented, and the missing tooth structure replaced with IRM (see Figure 14-39). During the placement procedure, it is important to protect the canals and pulp chamber.

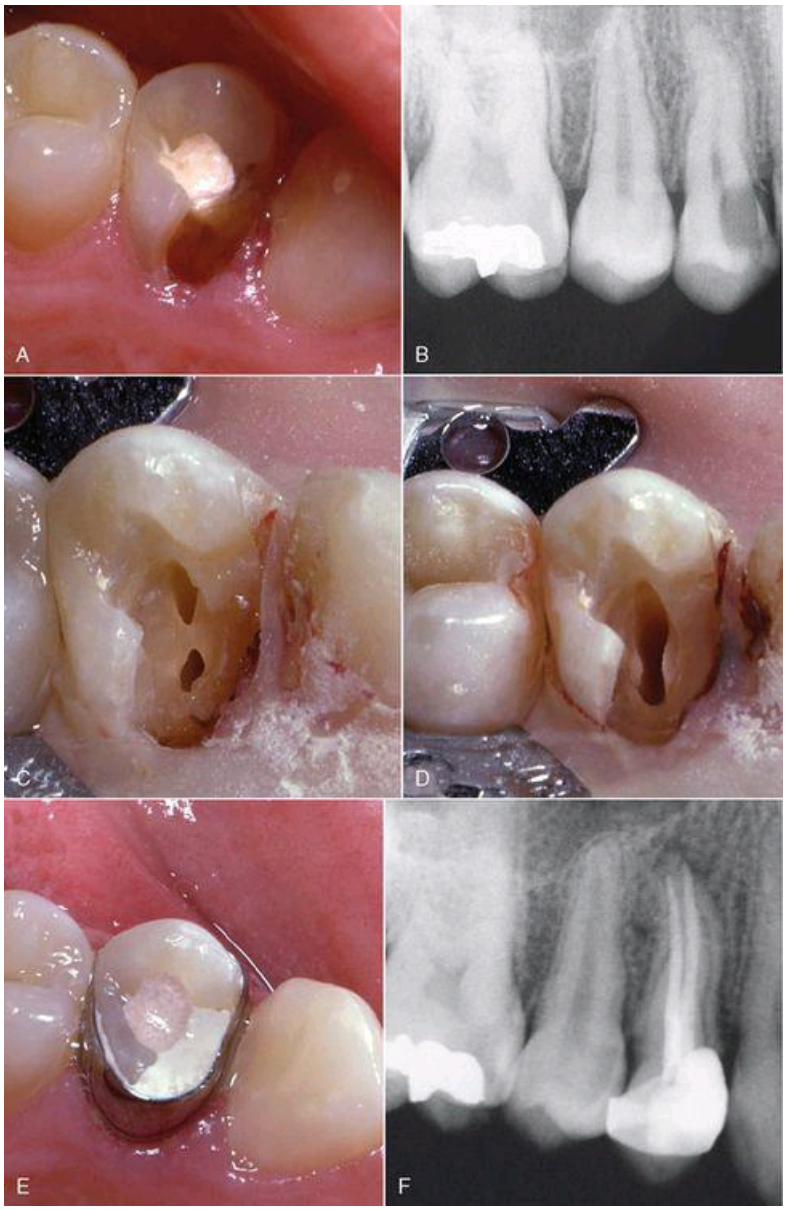

Figure 14-39 A, Access made through gross mesial caries. **B,** Caries at the level of the crestal bone. **C,** Caries removal provides an aseptic operating field and assessment of restorability. Note that the previous access failed to de-roof the chamber. **D,** Appropriate access reveals a

ribbon-shaped pulp chamber. **E,** An orthodontic band placed to provide isolation. **F,** Postobturation.

Provisional Crowns

Placement of temporary crowns is an option; however, they decrease visibility, result in the loss of anatomic landmarks, and may change the orientation for access and canal location. Often, temporary crowns are displaced during treatment by the rubber dam clamp. In general, when provisional crowns are placed, they should be removed during root canal treatment and replaced after the procedure to increase visibility, provide adequate orientation, and maintain the remaining tooth structure.

Corrective Surgery

Gingivectomy

Gingivectomy before root canal treatment is an option when the loss of tooth structure extends below the gingival tissues but there is adequate structure above the crestal bone. It can also be used when the gingival tissues have grown into a carious defect. In general the biologic width requires a minimum of 3.0 mm of sound tooth structure above the crestal bone: 1.0 mm for the epithelial attachment, 1.0 mm for the connective tissue attachment, and 1.0 mm for the margin of the final restoration.[16] If there is less than the required 3.0 mm of tooth structure, crown lengthening should be considered. Gingivectomy removes excessive gingival tissue and exposes the coronal root structure; however, osseous reduction is not possible. Electrosurgery is an option to the traditional scalpel because it provides a bloodless operating site. The small surgical electrodes make it easy to reach difficult areas, and the coagulating property provides hemostasis and enhances vision.[17,18] Care must be taken not to contact the alveolar bone because irreversible damage and necrosis will occur.[19,20]

Crown Lengthening

Crown lengthening is a procedure requiring an intrasulcular incision and flap reflection before osseous recontouring.[21-23] Enough osseous tissue should be removed to provide at least 3.0 to 4.0 mm of sound tooth structure coronal to the crestal bone (see Figure 14-5).[24-26] An additional factor to consider is the ability to complete the root canal treatment adequately. Performing the surgical procedure and then discovering that the endodontic treatment cannot be accomplished is not in the best interest of the patient. For this reason the procedures are often performed concurrently.

Orthodontic Extrusion

Orthodontic extrusion (forced eruption) is indicated when there is inadequate tooth structure for isolation and subsequent restoration, and crown lengthening is contraindicated.[20] Criteria to consider during the treatment planning process include the resulting crown-root ratio after extrusion, as well as esthetics (narrowing of the root form). The tooth must be extruded so that the biologic width is restored (3.0 mm of root coronal to the crestal bone). The minimum crown-root ratio after the extrusion is a 1 : 1 ratio.

Rubber Dam Placement

Placement as a Unit

Placement of rubber dam, clamp, and frame as a unit is preferred (see Figure 14-4). This is most efficient and is applicable in most cases. A traditional dam and frame can be used or proprietary disposable systems are available (see Figure 14-2).

1. The dam is placed on the frame so that it is stretched tightly across the top and bottom but has slack horizontally in the middle.

2. The hole is punched in the dam, and then the clamp wings are attached to the dam.

3. The dam, frame, and clamp are placed as a unit to engage the tooth near the gingival margin.

4. The dam is released apically off the clamp wings to allow the dam to constrict around the tooth neck. The dam is then flossed through the contacts.

Placement of Clamp Followed by the Dam and Then the Frame

Placement of a clamp followed by the dam and frame is seldom used but may be necessary when an unobstructed view is required while the clamp is positioned. The clamp is first placed on the tooth and secured. The rubber dam is then stretched over the clamp, and the frame affixed.[4]

Placement of the Rubber Dam and Frame and Then the Clamp

The preferred method for applying a butterfly clamp that does not have wings (No. 212) is to place the dam and frame and then the clamp. Better visualization is possible when the hole is stretched over the tooth and gingiva first by the dental assistant, and the clamp is then placed. The No. 212 clamp has narrow beaks and is often used in situations where wing clamps are unstable or cannot be retained.

Rubber Dam Leakage

Several proprietary products are available for placement around the rubber dam at the tooth-dam interface should leakage occur (see Figure 14-6). These caulk-like materials are easily applied and removed after treatment and are especially useful for isolation of an abutment for a fixed partial denture or for a tooth that is undergoing active orthodontic treatment.

The caulk can be placed on the gingival tissues before dam placement or at the dam-tooth interface after isolation. Both the caulking and putty materials adhere to wet surfaces, with the putty having a stiffer consistency.

Disinfection of the Operating Field

Various methods and techniques have been used to disinfect the tooth, clamp, and surrounding rubber dam after placement. These include alcohol, quaternary ammonium compounds, sodium hypochlorite, organic iodine, mercuric salts, chlorhexidine, and hydrogen peroxide. An effective technique is as follows: (1) plaque is removed by rubber cup and pumice; (2) the rubber dam is placed; (3) the tooth surface, clamp, and surrounding rubber dam are scrubbed with 30% hydrogen peroxide; and (4) the surfaces are swabbed with 5% tincture of iodine or with sodium hypochlorite.[27]

ACCESS OPENINGS

Endodontic access openings are based on the anatomy and morphology of each individual tooth group. In general, the pulp chamber morphology dictates the design of the access preparation. The internal anatomy is projected onto the external surface. The major objectives of the access openings include (1) locating all canals, (2) unimpeded straight-line access of the instruments in the canals to the apical one third or the first curve (if present), (3) removal of the chamber roof and all coronal pulp tissue, and (4) conservation of tooth structure.

General Principles

The general principles for endodontic access are outline form, convenience form, caries removal, and toilet of the cavity.

Outline form is the recommended shape for access of a normal tooth with radiographic evidence of a pulp chamber and canal space. The outline form assures the correct shape and location and provides straight-line access to the apical portion of the canal or to the first curvature. The access preparation must remove tooth structure that will impede the cleaning and shaping of the canal or canals. The outline form is a projection of the internal tooth anatomy onto the external root structure. The form can change with time. As an example, in anterior teeth with mesial and distal pulp horns the access is triangular. In older individuals with chamber calcification, the pulp horns are absent, so the access is ovoid.

Convenience form allows modification of the ideal outline form to facilitate unstrained instrument placement and manipulation. As an example, the use of nickel-titanium rotary instruments requires straight-line access. An access might be modified to permit placement and manipulation of the nickel-titanium instruments. Another example is a premolar exhibiting three roots. The outline form might be made more triangular to facilitate canal location.

Caries removal is essential for several reasons. First, removing caries permits the development of an aseptic environment before entering the pulp chamber and radicular space. Second, it allows assessment of restorability before treatment. Third, it provides sound tooth structure so that an adequate provisional restoration can be placed. Unsupported tooth structure is removed to ensure a coronal seal during and after treatment so that the reference point for length determination is not lost should fracture occur.

Toilet of the cavity involves preventing materials and objects from entering the chamber and canal space. A common error is entering the pulp chamber before the coronal structure or restorative materials are adequately prepared. As a result, these materials enter the canal space and may block the apical portion of the canal.

Canal Morphologies

Five major canal morphologies have been identified (Figure 14-7).[28] They include round, ribbon or figure eight, ovoid, bowling pin, kidney bean, and **C**-shape. With the exception of the round morphologic shape, each presents unique problems for adequate cleaning and shaping.

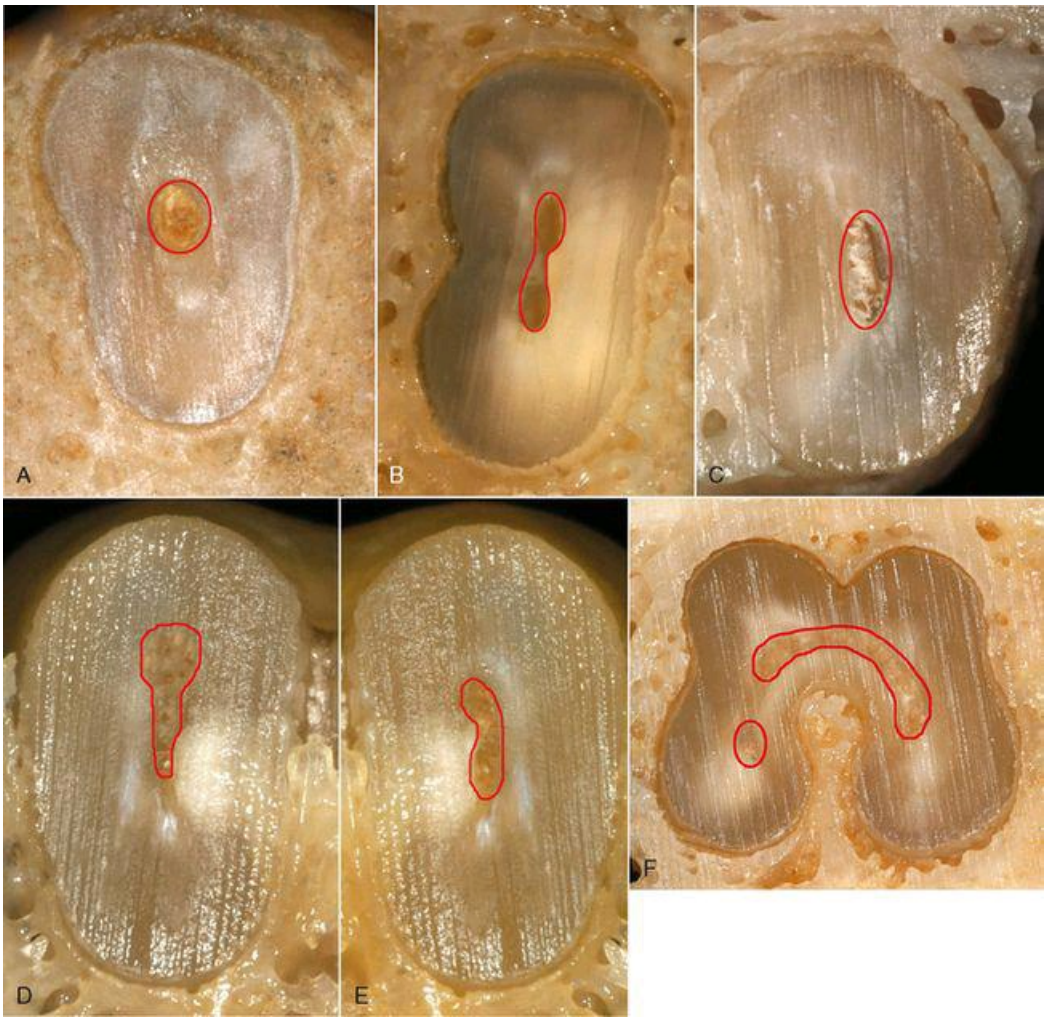

Figure 14-7 Common canal morphologies. **A,** Round. **B,** Ribbon-shaped (hourglass). **C,** Ovoid. **D,** Bowling pin. **E,** Kidney bean-shaped. **F,** C-shaped.

In difficult cases the access can be prepared without the rubber dam in place. This provides visualization of the tooth shape, orientation, and position in the dental arch. When the canal or chamber is located, the rubber dam is applied. *Caution:* Until the rubber dam is in place, broaches and files cannot be used (see Figure 14-1).

Care must be taken to prevent tooth structure or restorative materials from entering the radicular portion of the root if additional expansion of the access is necessary after the chamber is exposed. When an access is to be enlarged or restorative materials removed after chamber exposure, the radicular space must be protected. The canal orifice and chamber floor can be blocked by placing gutta-percha temporary stopping. The material is heated and then compacted with a plugger. Removal of the temporary stopping is with heat (preferred) or solvents after completion of the access preparation.

Before beginning the access the preoperative radiographs should be assessed to determine the degree of case difficulty. In more complex cases, an angled film/digital image will be useful in determining the canal morphology. Bite-wing radiographs can give a more accurate image of the anatomy of the pulp chamber in posterior teeth.

At this stage the estimated depth of access is calculated. This is a measurement from the midlingual surface of anterior teeth and the occlusal surface of posterior teeth to the coronal portion of the pulp chamber. Calculated in millimeters, this information is then transferred to the access bur and provides information on the depth necessary to expose the pulp. When the estimated depth of access is reached and the pulp has not been encountered, the access depth and orientation must be reevaluated. A parallel radiograph exposed with the rubber dam removed helps in determining the depth and orientation, so the incidence of perforations and unnecessary removal of tooth structure can be avoided (see Figure 14-33).

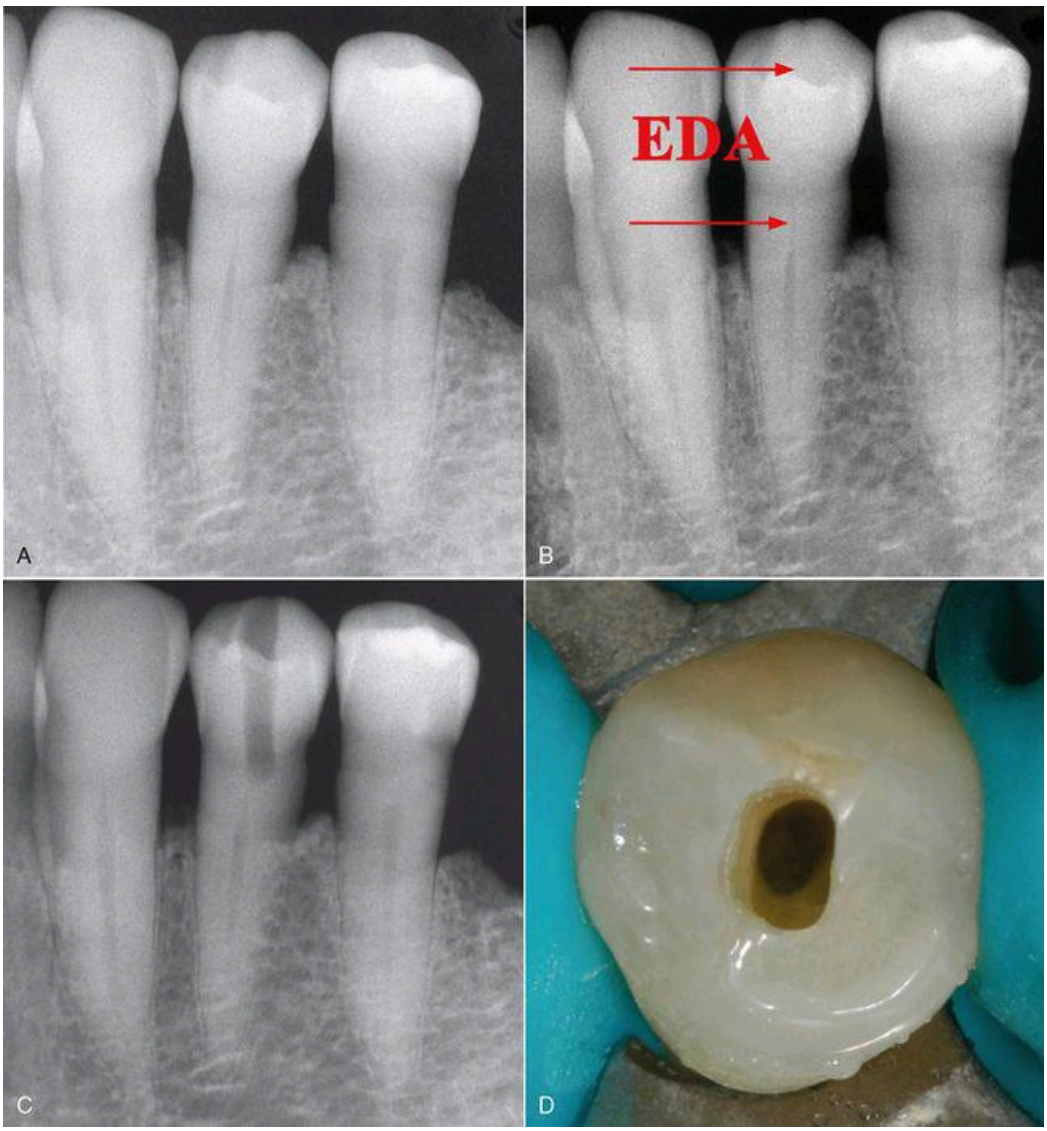

Figure 14-33 A, Mandibular right first premolar. Note the receded pulp space. **B,** Calculation of the estimated depth of access. **C,** The estimated depth of access is reached, and the canal is not located. The rubber dam is removed and a straight-on parallel radiograph exposed. The film/digital image indicates the canal is located mesial to the opening. **D,** The completed access.

Access openings are best accomplished using a fissure bur in the high-speed handpiece. A number of special burs are also available for access. No single bur type is superior. With knowledge of anatomy and morphology and the appropriate clinical skills and judgment, bur selection is a personal choice (Figures 14-8 and 14-9). Regardless of the high-speed bur

chosen, the bur is placed in the chamber and removed while rotating. High-speed burs are not used in the canals. Violating these principles can result in breakage (Figure 14-10).

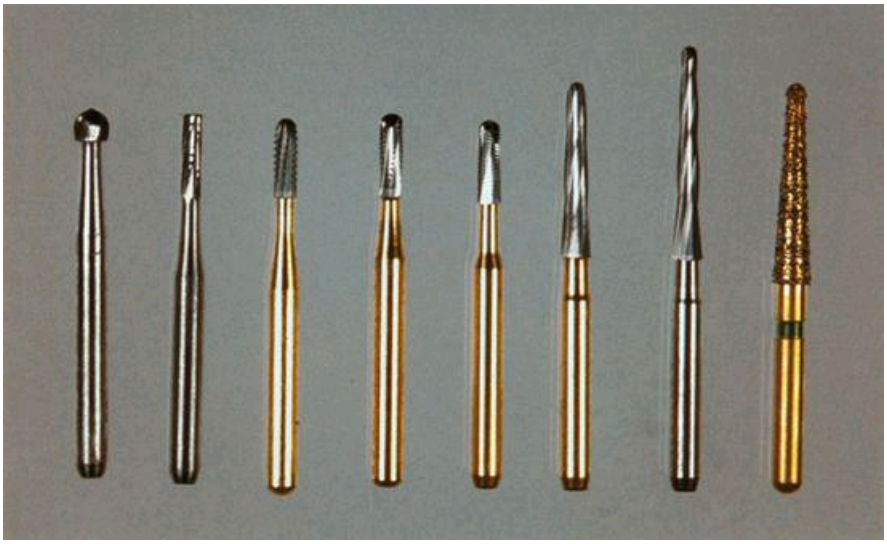

Figure 14-8 Examples of access burs. *Left to right,* No. 4 round carbide, No. 557 carbide, Great White, Beaver bur, Transmetal, Multipurpose bur, Endo Z bur, and Endo Access bur.

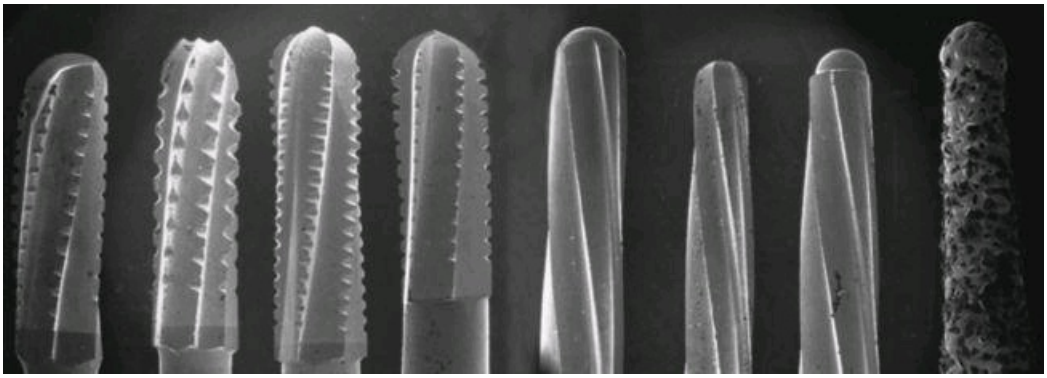

Figure 14-9 Magnified image *(left to right)* of the Great White, Beaver bur, Transmetal, H34L, 269GK, Multipurpose bur, Endo Z bur, Endo Access bur.

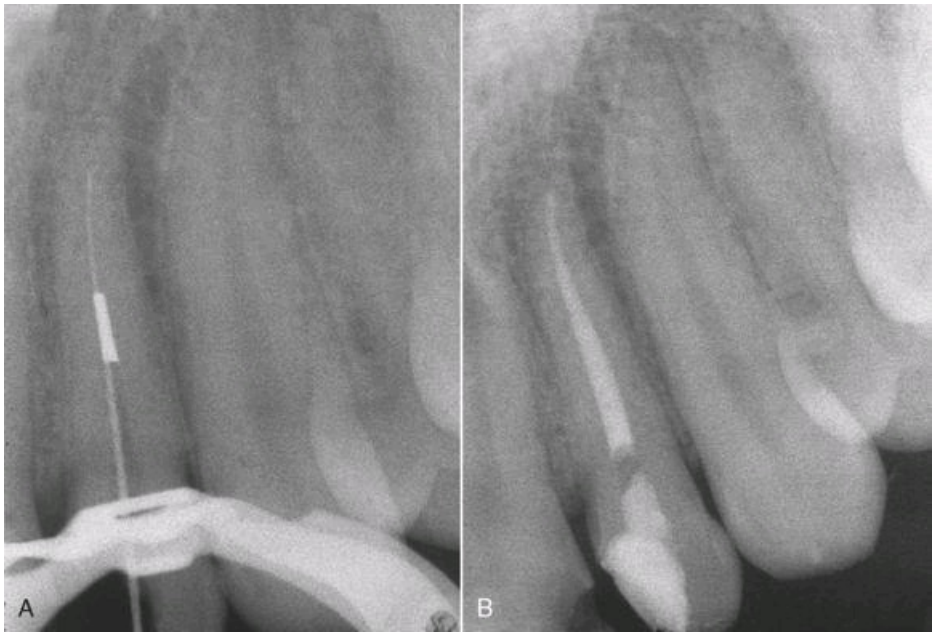

Figure 14-10 A, Fractured fissure bur and working length file bypassing the obstruction. **B,** After the bur was removed with files and ultrasonics.

Visualization of the internal anatomy is enhanced during access by using a fiberoptic handpiece and microscopy.[29] Illumination is a key factor. A sharp endodontic explorer can be used for detection of the canal orifice or to aggressively dislodge calcifications. When a canal is located, a small file or pathfinding instrument (.06, .08, or .10 stainless steel file) is used to explore the canal and determine patency close to the apical foramen. Care should be exercised during this process to prevent forcing tissue apically, which might result in canal blockage (Figure 14-11). This procedure is performed in the presence of irrigant or lubricant.

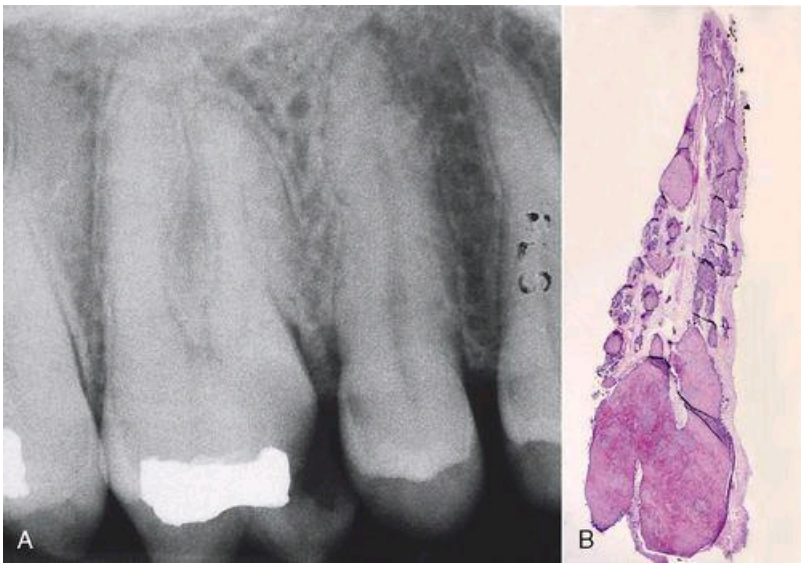

Figure 14-11 A, Maxillary first molar exhibits extensive mesial caries. **B,** Histologic section of pulp tissue from the palatal canal reveals extensive calcification. Early canal exploration should be done with small files to prevent forcing the tissue and calcification apically and blocking the canal.

Often, in an attempt to preserve tooth structure, the access openings are constricted and underprepared. This creates problems with locating canals and gaining straight-line access. Removal of restorative materials is often warranted, knowing that following treatment they will be replaced. Removal enhances visibility and may reveal undetected canals, caries, or coronal fractures. When difficulties occur with calcifications or extensive restorations, the operator can become disoriented with respect to canal morphology. The discovery of one canal can serve as a reference in locating the remaining canals. A file can be inserted and a radiograph exposed to reveal which canal has been located.

Complex restorations, such as crowns and fixed partial dentures, may have changed the coronal landmarks used in canal location. A tipped tooth might be “uprighted” or a rotated tooth “realigned.” Loss of orientation can result in the incorrect identification of a canal, and searching for the other canals in the wrong direction results in excessive removal of tooth structure, perforation, or the failure to locate and debride all canals.

Access through crowns with extensive foundations may make visibility difficult. Class V restorations may have induced coronal calcification or could have been placed directly into the pulp space or the canals. In some instances, it may be best to remove restorative materials that interfere with visualization before initiating root canal treatment.

A modification of the armamentarium for teeth restored with crowns has been advocated for all-ceramic crowns. The initial outline and penetration through the restorative material are made with a round diamond bur in the high-speed handpiece with water coolant. After penetration into dentin, a fissure bur can be used. In teeth with porcelain-fused-to-metal restorations, a metal cutting bur is recommended. When possible, the access should remain in metal to reduce the potential for fracture in the porcelain. Evidence indicates that with a water coolant and careful instrumentation, diamond and carbide burs are equally effective.[30]

In summary, aids in canal location include knowledge of pulp anatomy and morphology; parallel straight-on and angled radiographs/digital images; a sharp endodontic explorer; interim radiographs/digital images; long-shanked, slow-speed burs (Figure 14-12); ultrasonic instruments for troughing; dye staining; irrigation; transillumination; and enhanced vision with loupes or microscopy.[31]

Figure 14-12 Mueller burs exhibit a round cutting head attached to a long shank. The long shank is not designed to drill deep into the root but to extend the head of the slow-speed handpiece away from the tooth and permit better visibility.

ACCESS OPENINGS AND CANAL LOCATION*

Maxillary Central and Lateral Incisors

The maxillary central incisor has one root and one canal.[32] In young individuals, the prominent pulp horns present require a triangular-outline form to ensure tissue and obturation materials are removed, which might cause coronal discoloration (Figure 14-13). While the canal is centered in the root at the cementoenamel junction (CEJ) and when viewing the tooth from a mesial to distal orientation, it is evident that the crown is not directly in line with the long axis of the root (Figure 14-14). For this reason the establishment of the outline form and initial penetration into enamel are made with the bur perpendicular to the lingual surface of the tooth. This outline form is made in the middle-third of the lingual surface (Figures 14-15 and 14-16). After penetration to the depth of 2 to 3 mm, the bur is reoriented to coincide with the long axis and lingual orientation of the root. This reduces the risk of a lateral perforation through the facial surface. An additional common error is the failure to remove the lingual shelf (Figure 14-15, *C*), which will result in inadequate access to the entire canal. The canal is located by using a sharp endodontic explorer. In cases where calcification has occurred, long-shanked burs in a slow-speed handpiece can be used (see Figures 14-12 and 14-24, *D*). These burs move the head of the handpiece away from the tooth and enhance the ability to see exactly where the bur is placed in the tooth.

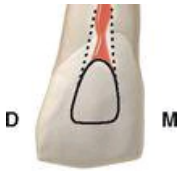

Figure 14-13 A triangular outline form for access of the maxillary central incisor.

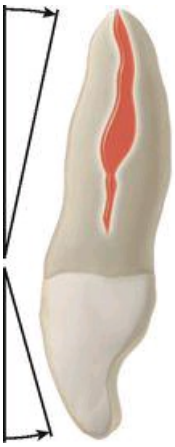

Figure 14-14 Note the lingual inclination of the root in relation to the crown. Also the pattern of calcification occurs from the coronal portion of the pulp apically.

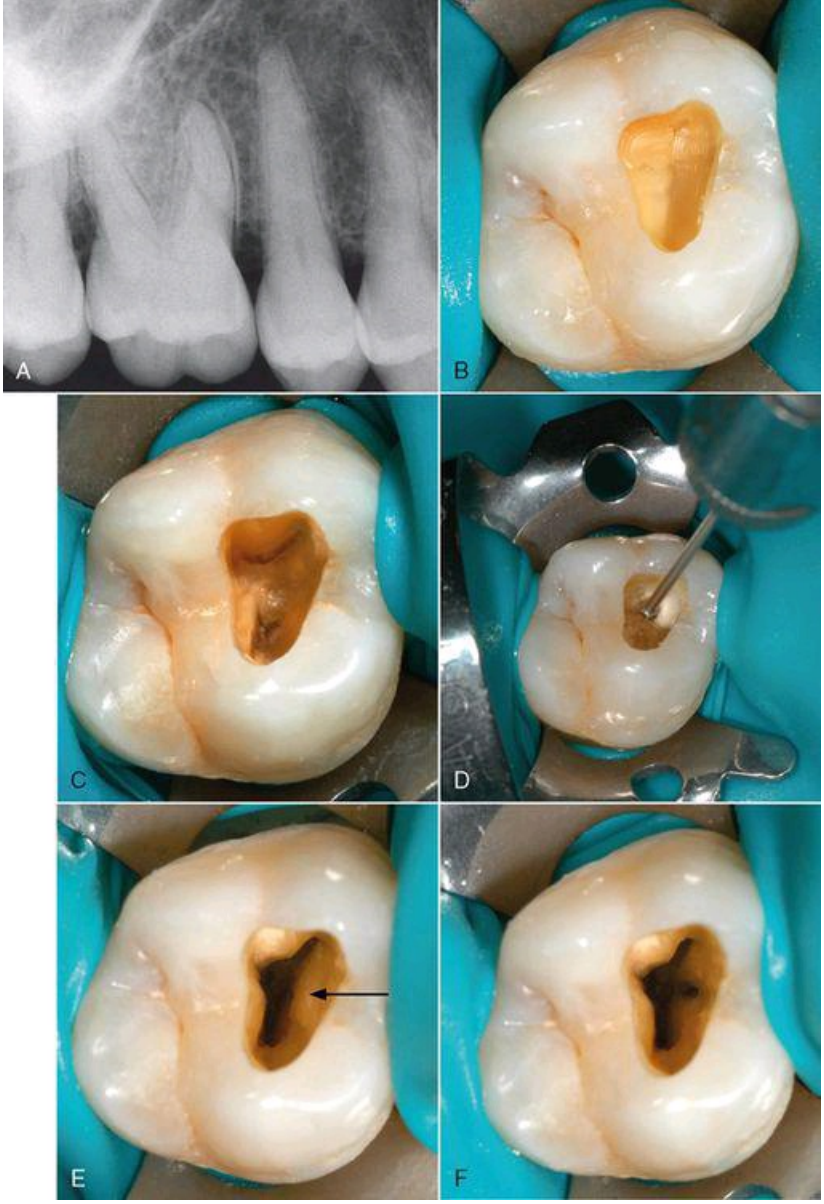

Figure 14-24 A, Maxillary left first molar. Note the calcification in the chamber. **B,** The outline form established and dentin removed apically in layers. **C,** Exposure of the pulp horns. **D,** The use of a Mueller bur to completely unroof the chamber. Note the visibility and ability for precise removal of dentin. **E,** The completed access. The mesiobuccal canal is evident under the mesiobuccal cusp tip, the distobuccal canal is found opposite the buccal groove and slightly lingual to the main mesiobuccal canal, and the palatal canal is located under the mesiolingual cusp tip. Note the identification of the mesiolingual canal *(arrow)*. **F,** Removal of the dentinal cornice that covers the mesiolingual canal to reveal the canal orifice. See Appendix A, Pulpal Anatomy and Access Preparations, for colorized illustrations that depict the size, shape, and location of the pulp space within each tooth.

Access for the maxillary lateral incisor is similar to that for the central incisor. A triangular access is indicated in young patients with pulp horns (Figure 14-17), and as the pulp horns recede, the outline form becomes ovoid (Figure 14-18).

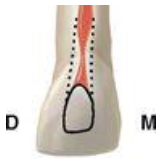

Figure 14-17 Triangular outline form of the maxillary lateral incisor.

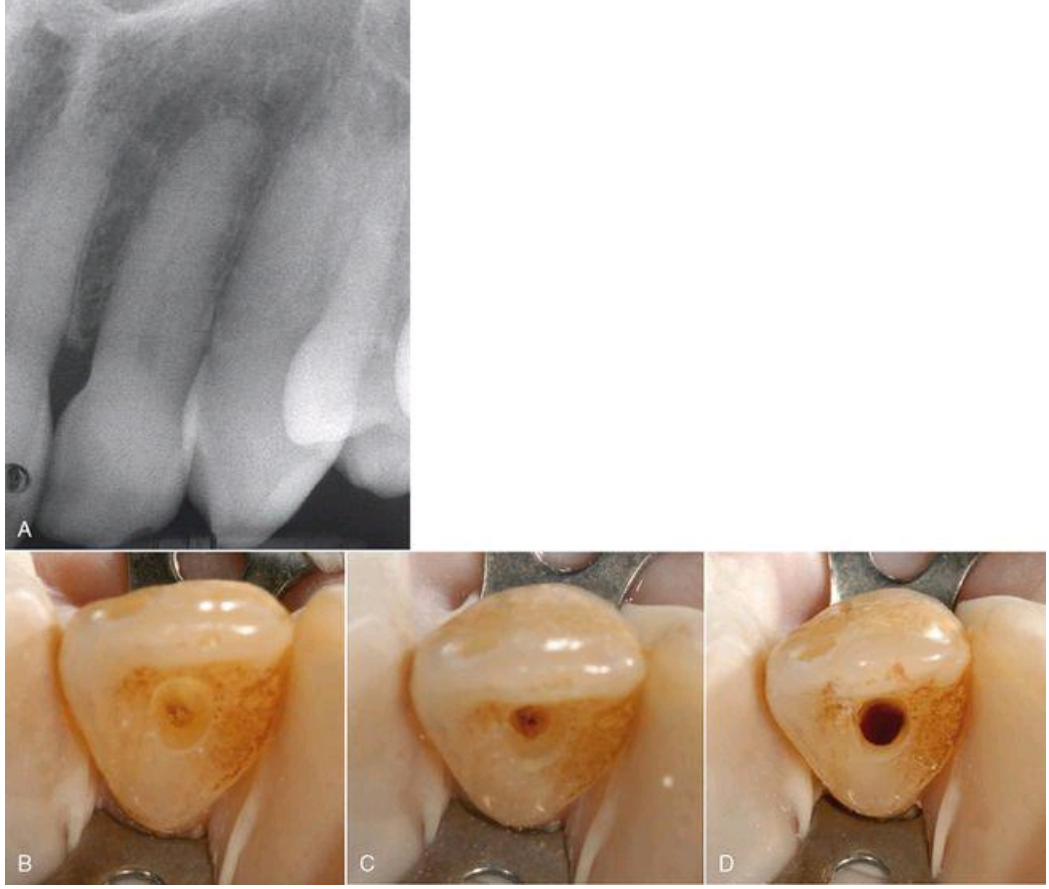

Figure 14-18 A, Lateral incisor with a receded pulp chamber. **B,** Initial ovoid outline form is initiated. **C,** Coronal calcification indicated by the color change. **D,** The completed access.

Dens invaginatus or dens en dente is a common developmental defect in the maxillary lateral incisor that results in pulp necrosis.[33,34] Additionally, a lingual groove may be found in maxillary lateral incisors, as evidenced by a narrow probing defect. These developmental defects complicate treatment and affect prognosis.

Maxillary Canines

Maxillary canines exhibit one canal in a single root. Generally, pulp horns are absent so the outline form is ovoid in the middle third of the lingual surface (Figures 14-19 and 14-20). As attrition occurs, the chamber appears to move more incisally because of the loss of structure. In cross-section the pulp will be wide in a faciolingual direction when compared to the mesiodistal dimension.

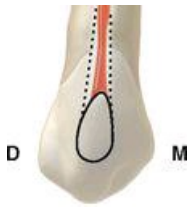

Figure 14-19 The outline form for the maxillary canine.

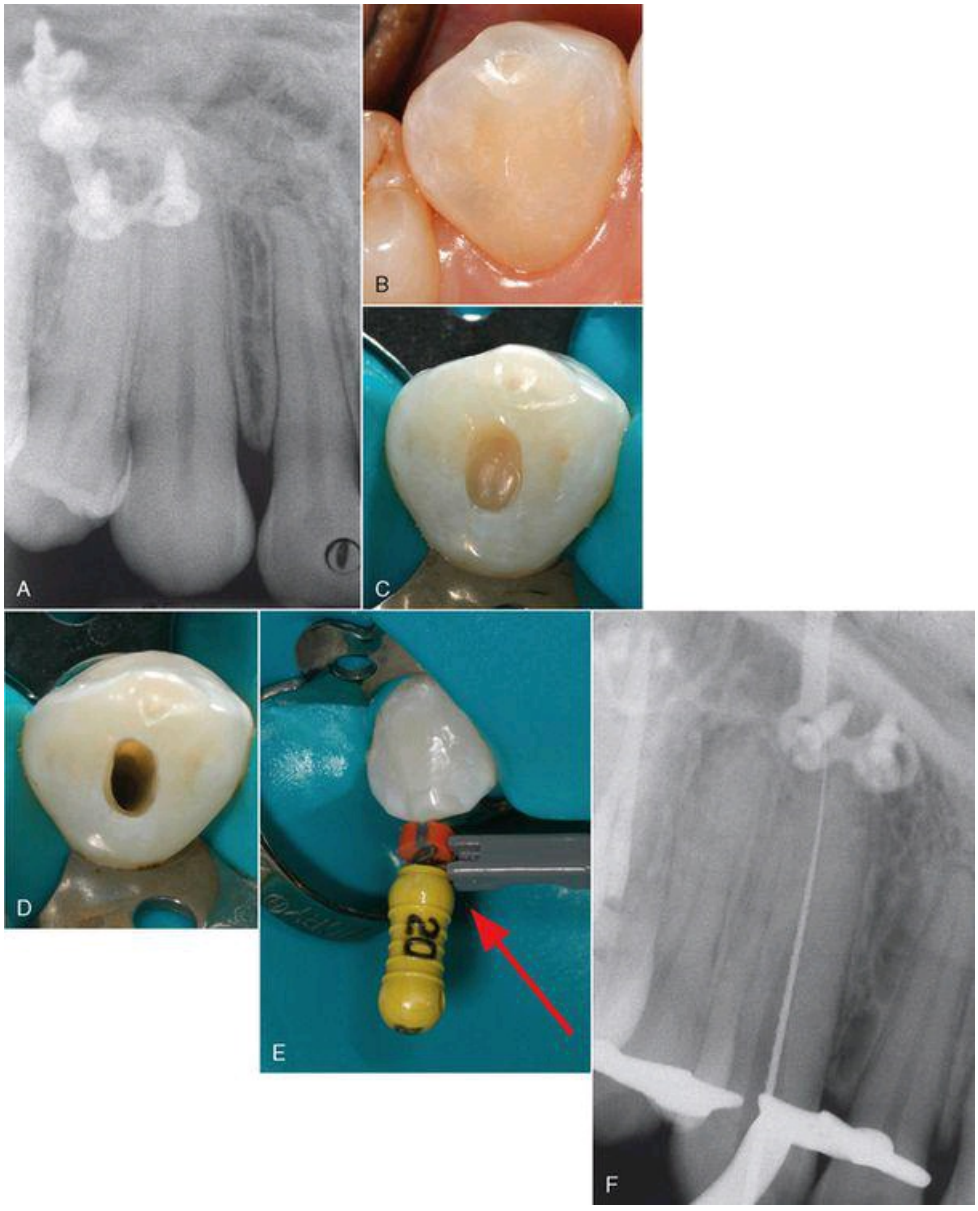

Figure 14-20 A, The apex is obscured by the screws placed during a maxillary surgical advancement. **B,** Lingual surface. **C,** Initial access outline into dentin and **D** is finalized. **E,** Apex locator *(arrow)*. **F,** Working length.

Maxillary Premolars

The maxillary first and second premolars exhibit a similar coronal structure so the outline form is similar for both teeth, is centered in the crown, and exhibits an ovoid shape in the faciolingual direction (Figures 14-21 and 14-22). An important anatomic consideration with these teeth is the mesial concavity at the CEJ. This is an area in which a lateral perforation is likely to occur. When two canals are present, the canal orifices are located under the buccal and lingual cusp tips equal distance from a line drawn through the center of the chamber in a mesial to distal direction. The cross-sectional morphology exhibits a kidney bean– or ribbon-shaped configuration. In rare instances when three canals are present, the outline form is triangular with the base to the facial and the apex toward the lingual.

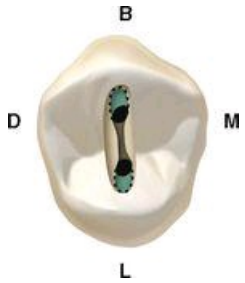

Figure 14-21 Ovoid outline form for the maxillary premolars.

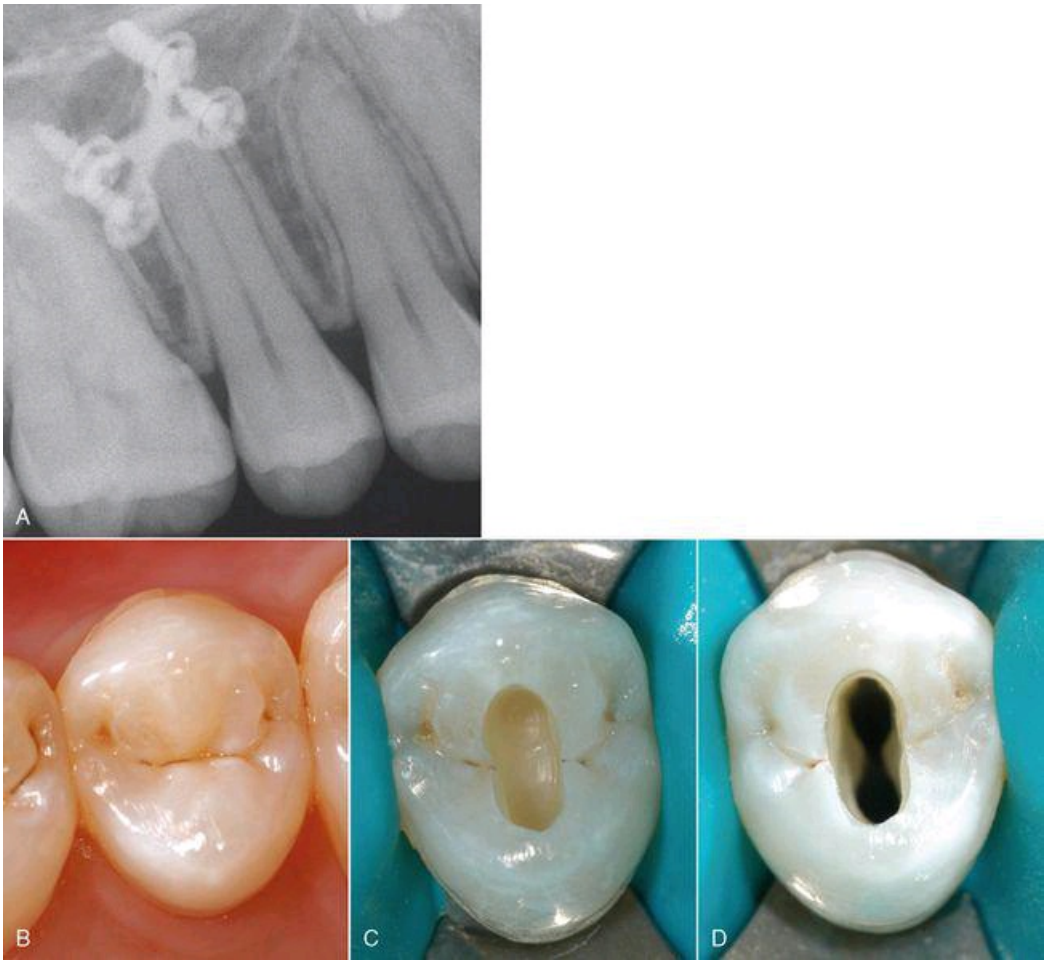

Figure 14-22 A, Note the obstructed view of the apical region. **B,** Maxillary right second premolar. **C,** The initial outline form prepared into dentin. **D,** The chamber and canals are accessed.

Maxillary Molars

The maxillary first and second molars have similar access outline forms. The outline form is triangular and located in the mesial half of the tooth, with the base to the facial and the apex toward the lingual (Figures 14-23 and 14-24). The transverse or oblique ridge is left mostly intact. The external references for canal location serve as a guide in developing the outline form. The mesiobuccal canal orifice lies slightly distal to the mesiobuccal cusp tip. The distobuccal canal orifice lies distal and slightly lingual to the main mesiobuccal canal and is in line with the buccal groove. The lingual or palatal canal orifice generally exhibits the largest orifice and lies slightly distal to the mesiolingual cusp tip. The mesiobuccal root is very broad in a buccolingual direction, thus a small second canal is common.[35-39] The mesiolingual canal orifice (commonly referred to as the *MB_2 canal*) is located lingual to the main mesiobuccal canal (MB_1 canal) from 1 to 3 mm and is slightly mesial to a line drawn from the mesiobuccal to the lingual or palatal canal. The initial movement of the canal from the chamber is often not toward the apex but laterally toward the mesial (Figure 14-25). Removal of the coronal dentin (cornice) in this area permits exposure of the canal as it begins to move apically and facilitates negotiation (Figures 14-26 and 14-27; see Figures 14-24 and 14-25).[40] In addition, the operating microscope is a valuable aid.[29,38]

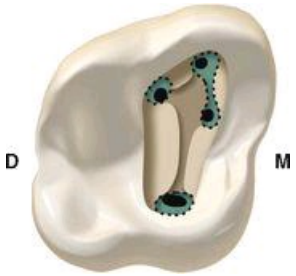

Figure 14-23 Triangular outline form for access of the maxillary molar.

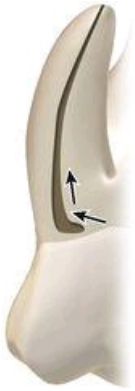

Figure 14-25 The mesiolingual canal as it leaves the pulp chamber. Canals that are not negotiable but detected by an explorer may move laterally before proceeding apically.

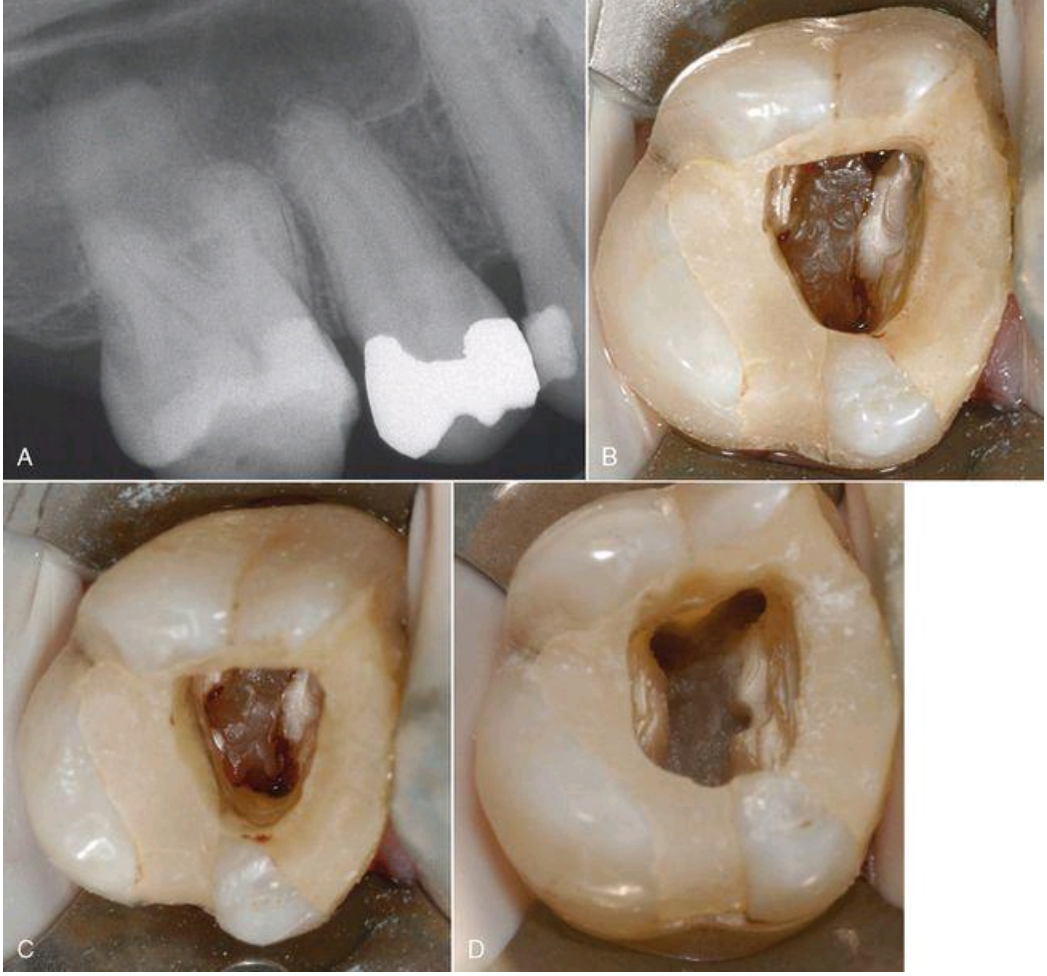

Figure 14-26 A, Maxillary left first molar exhibiting calcification. **B** and **C,** Initial access and identification of a pulp stone. Color and a thin line surrounding the periphery identify the hemorrhage. **D,** The pulp chamber with the stone removed. See Appendix A, Pulpal Anatomy and Access Preparations, for colorized illustrations that depict the size, shape, and location of the pulp space within each tooth.

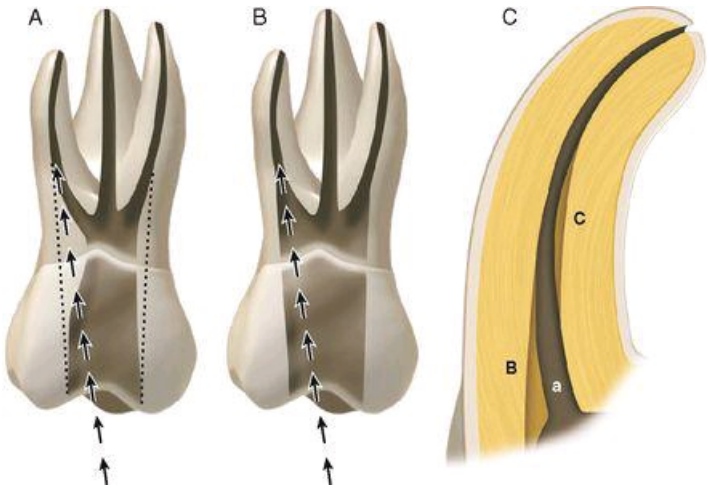

Figure 14-27 A, The dashed lines show where dentin must be removed to achieve straight-line access. **B,** The access completed. **C,** The original canal *(a)* is modified by Gates-Glidden burs by removing tooth structure at *B* and *C*.

Mandibular Central and Lateral Incisors

The mandibular incisors are narrow in the mesiodistal dimension and broad faciolingually. There may be one canal with an ovoid or ribbon-shaped configuration or there can be two canals. When there are two canals, the facial canal is easier to locate and is generally straighter than the lingual canal, which is often shielded by a lingual shelf. Since the tooth is often tipped facially, the lingual canal is difficult to locate and perforations primarily occur on the facial surface.

The narrow mesiodistal dimension of these teeth makes access and canal location difficult. In young patients with mesiodistal pulp horns the outline form is triangular with the base incisally and the apex gingivally. As the pulp recedes over time and the pulp horns disappear, the shape becomes more ovoid. The access is positioned in the middle-thirds of the lingual surface (Figures 14-28 and 14-29). Because of the small size of these teeth and the presence of mesiodistal concavities, access must be precisely positioned. The initial outline form is established into dentin with the bur perpendicular to the lingual surface. When a depth of 2 to 3 mm is reached, the orientation of the bur is reoriented along the long axis of the root. Because the percentage of teeth with two canals is reported to be 25% to 40%,[41,42] the lingual surface of the chamber and canal must be diligently explored with a small precurved stainless steel file. A Gates-Glidden drill is used on the lingual to remove the dentin shelf.

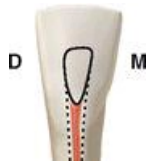

Figure 14-28 Lingual outline form for the mandibular incisor.

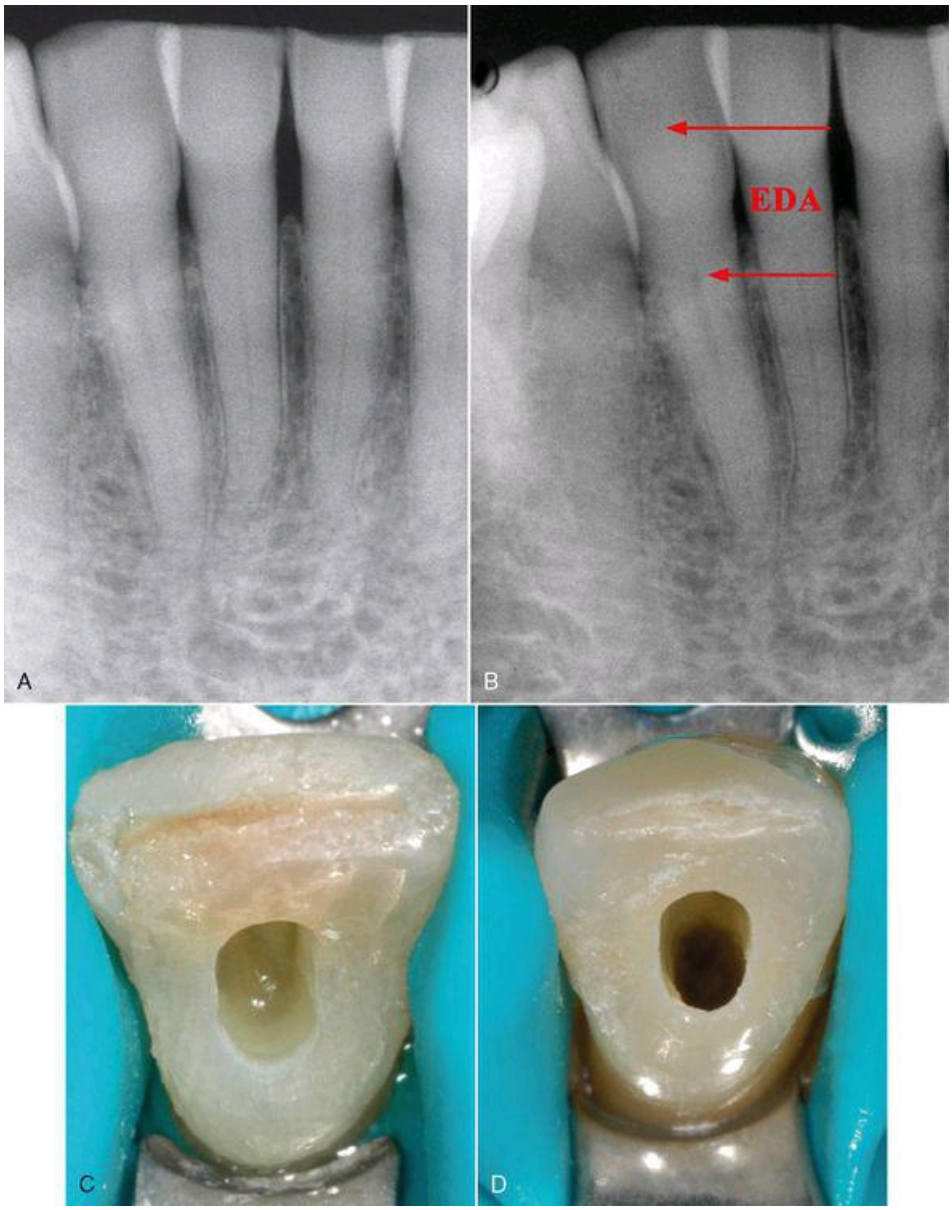

Figure 14-29 A, Mandibular lateral incisor. **B,** Calculation of the estimated depth of access from the middle of the lingual surface to the coronal extent of the pulp. **C,** The initial outline form is more oval due to the receded chamber. **D,** The completed access.

In cases of attrition, the access moves toward the incisal surface. With the use of nickel-titanium rotary instruments, straight-line access is imperative. A more incisal approach on the lingual or facial surface is justified.[43] A modification of the access for the incisors is a facial approach.[44] This provides better visibility and can be employed when there is crowding or when the canal is receded below the CEJ.

Mandibular Canines

The mandibular canines usually exhibit a long slender crown when compared to the maxillary canine, which is shorter and wider in a mesiodistal direction. The tooth may exhibit one or two roots. The root is broad in a faciolingual dimension and therefore may contain two canals. The outline form is ovoid and positioned in the middle-third of the crown on the lingual surface (Figures 14-30 and 14-31). On access opening into the chamber, the lingual surface should be explored for the presence of a lingual canal. As attrition occurs, the access will need to be more incisal, and in severe cases, it may actually include the incisal edge of the tooth.

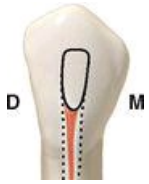

Figure 14-30 Lingual ovoid outline form for the mandibular canine.

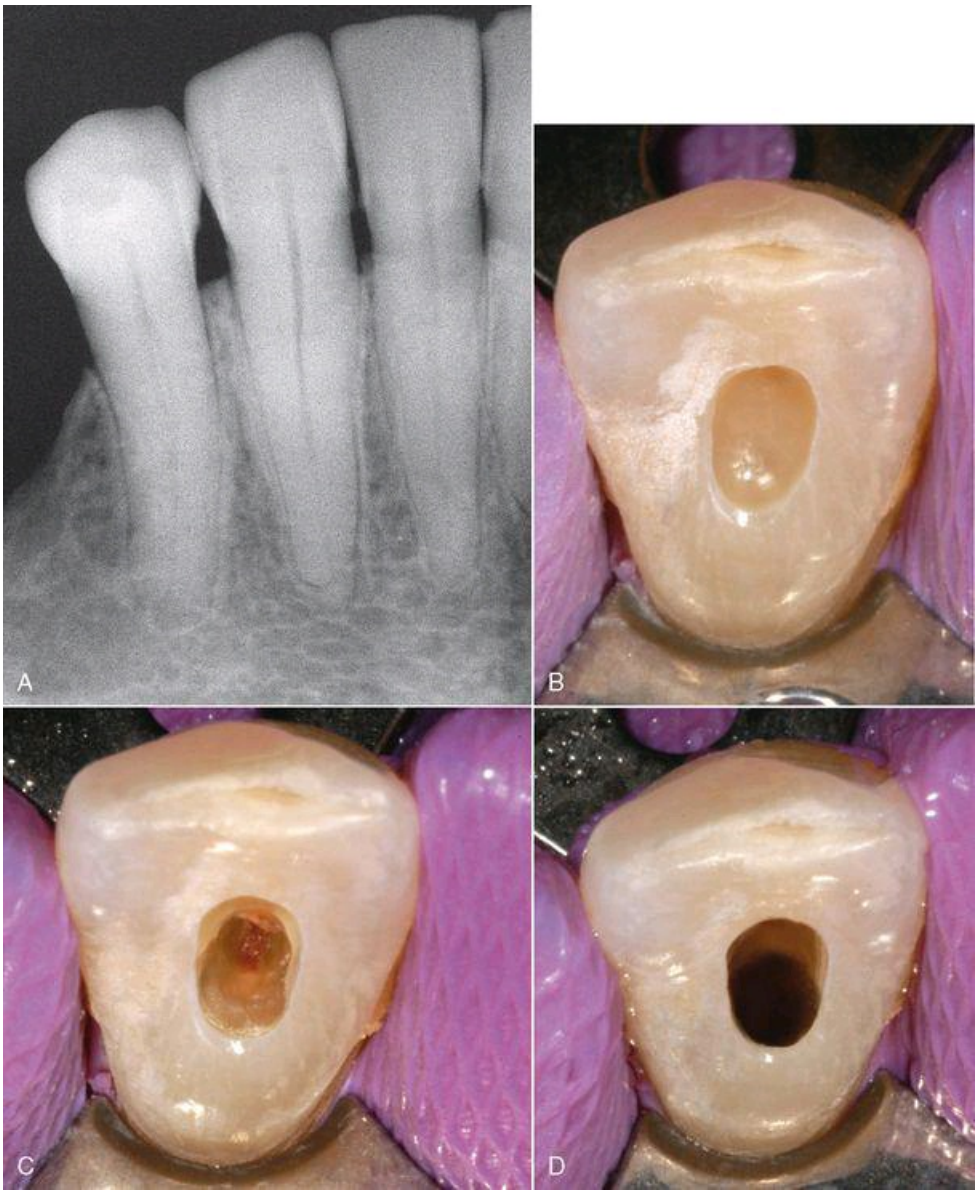

Figure 14-31 A, Mandibular canine. **B,** The initial outline form is established into dentin. **C,** Exposure of the coronal pulp. **D,** The completed access opening.

Mandibular Premolars

The mandibular premolars appear to be easy teeth to treat, but the anatomy may be complex. One, two, or three roots are possible, and canals often divide deep within the root in these complex morphologic configurations. The crown of the first premolar exhibits a prominent buccal cusp and a vestigial lingual cusp. In addition, there is a lingual constriction. Mesiodistal projections reveal that the chamber and canal orifice are positioned buccally. The access is therefore ovoid in a buccolingual dimension and positioned buccal to the central groove (Figures 14-32 and 14-33). It extends just short of the buccal cusp tip. The mandibular second premolar has a prominent buccal cusp, but the lingual cusp can be more prominent than with the first premolar. There is also a lingual constriction, so the outline form is ovoid from buccal to lingual and positioned centrally (Figure 14-34).

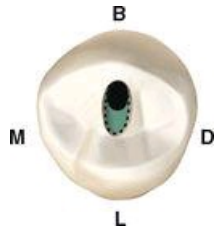

Figure 14-32 Ovoid outline form for the mandibular first premolar. Note the access is buccal to the central groove.

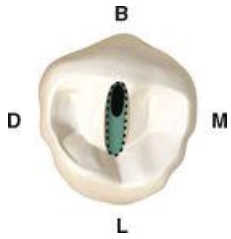

Figure 14-34 Ovoid outline form for the mandibular second premolar.

Mandibular Molars

The mandibular molars are similar in anatomic configuration; however, there are subtle differences. The most common mandibular first molar configuration is two canals in the mesial root, although three have been reported,[45] and one canal in the distal root. The presence of a second canal in the distal root is 30% to 35% (Figure 14-35).[46,47] The roots often exhibit a kidney bean shape in cross-section with the concavity in the furcal region. The most common configuration for the mandibular second molar is two canals in the mesial root and one canal in the distal root. The incidence of four canals is low.[46]

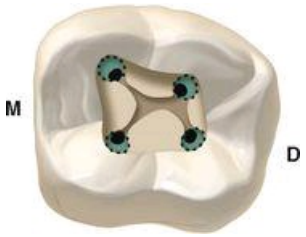

Figure 14-35 Rectangular outline form for the mandibular first molar. Note the mesiobuccal canal is located under the mesiobuccal cusp and the mesiolingual canal lies centrally in relation to the crown and slightly to the distal of the mesiobuccal canal. The distolingual canal is located centrally, and the distobuccal canal lies more to the buccal and mesial to the main canal.

The coronal reference points for canal location in the mandibular molar roots are influenced by the position of the crown on the root and by the lingual tipping of these teeth in the arch (Figure 14-36). The mesiobuccal canal orifice is located slightly distal to the mesiobuccal cusp tip. The mesiolingual canal orifice is located in the area of the central groove area and slightly distal when compared to the mesiobuccal canal. The distal canal is located near the intersection of the buccal, lingual, and central grooves. When a distobuccal canal is present, the orifice can be found buccal to the main distal canal and often is slightly more mesial. The mandibular first molar may even exhibit a distinct separate extra distal root. Because of these anatomic relationships, the access outline form is rectangular or trapezoidal and positioned in the mesiobuccal portion of the crown (Figure 14-37).

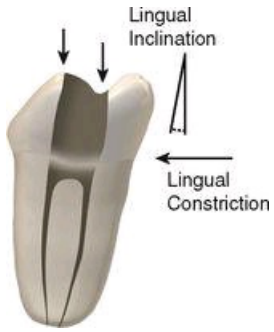

Figure 14-36 Proximal view of a mandibular molar demonstrating the lingual inclination in the dental arch and a lingual constriction of the crown at the cementoenamel junction. Note that the mesiobuccal and mesiolingual canals are uniformly spaced within the root. However, with coronal access, external reference points for canal location are the mesiobuccal cusp tip and the central groove as it crosses the mesial marginal ridge.

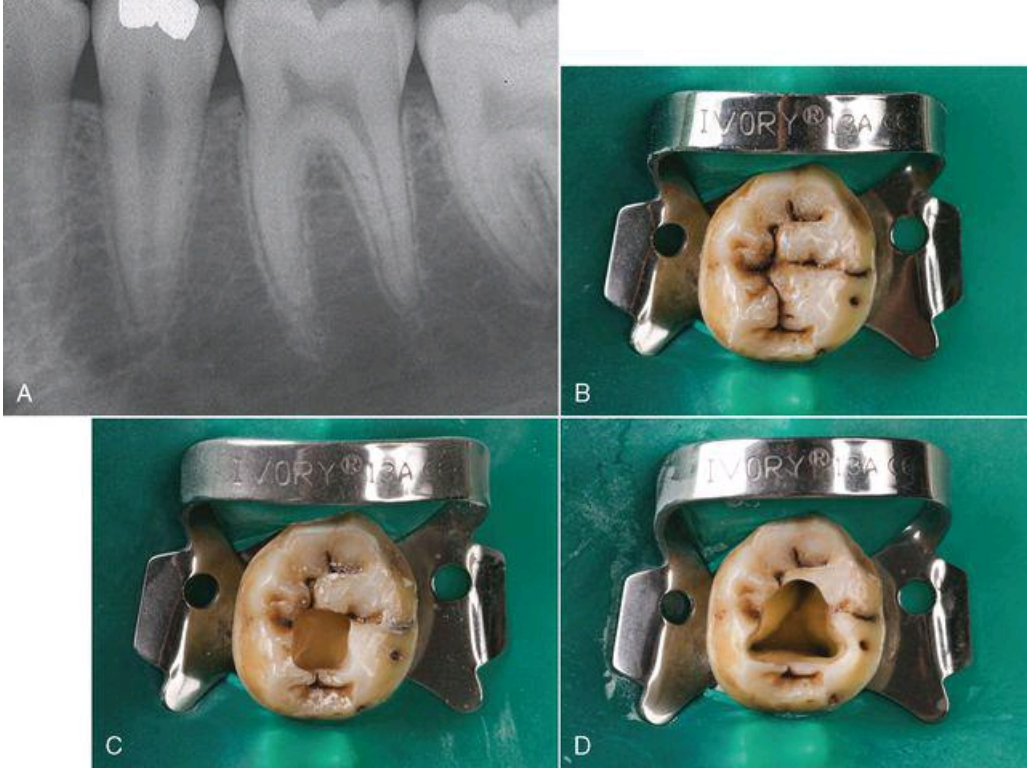

Figure 14-37 A, The preoperative radiograph of a mandibular first molar. **B,** The preoperative occlusal anatomy. **C,** The initial access outline form. **D,** The completed access cavity demonstrating the two mesial canals and the single distal canal. See Appendix A, Pulpal Anatomy and Access Preparations, for colorized illustrations that depict the size, shape, and location of the pulp space within each tooth.

During the access preparation, the cervical bulge that overlies the canal orifices of the mesiobuccal and mesiolingual canals is removed (Figure 14-38), which permits straight-line access to the first curve or apical portion of the root by decreasing the emergence profile. This also enhances entry into the canals.

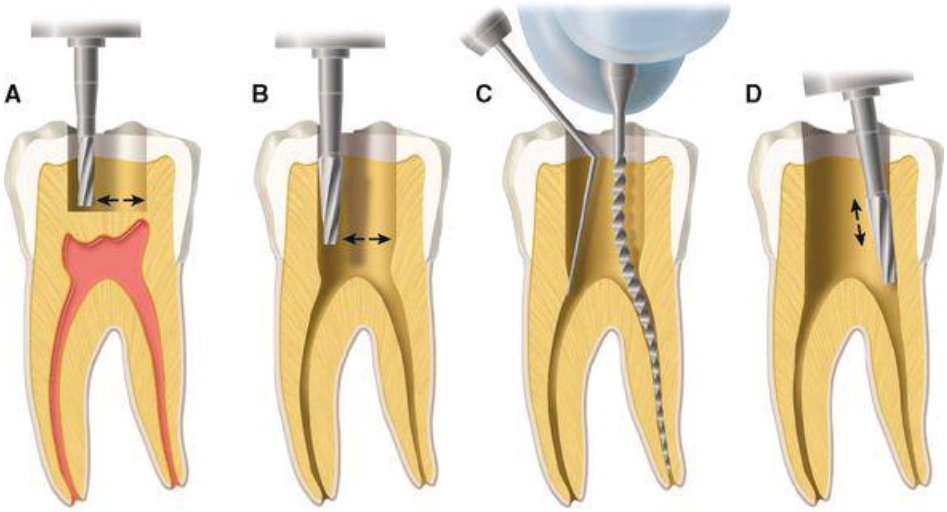

Figure 14-38 Basic steps in access preparation. **A,** The access cavity is outlined deep into dentin and close to the estimated depth of access with the high-speed handpiece. **B,** Penetration and unroofing are achieved by fissure high-speed bur or slow-speed latch-type burs. Other bur configurations are acceptable. **C,** Canal orifices are located and identified with an endodontic explorer. Small files are used to negotiate to the estimated working length. The dentin shelf that overlies and obscures the orifices is removed.

ERRORS IN ACCESS

Inadequate Preparation

Errors in access preparations are varied (Figure 14-39, *C*). A common error is inadequate preparation, which has several significant consequences. Direct effects are decreased access and visibility, which prevents locating the canals. The ability to remove the coronal pulp tissue and subsequent obturation materials is limited, and straight-line access cannot be achieved. Inadequate straight-line access can indirectly lead to errors during the cleaning and shaping. When files are deflected by coronal interferences, procedural errors, such as loss of working length, apical transportation, ledging, and apical perforation, are likely in curved canals. A No. 25 file or above has a straightening force that overcomes the confining resistance of the dentin wall. The file cuts on the outer surface apical to the curvature and the inner wall coronal to the curve. Adequate straight-line access decreases the canal curvature and reduces the coronal interferences, allowing the instrument to work more freely in the canal.

Excessive Removal of Tooth Structure

The excessive removal of tooth structure has direct consequences and unlike inadequate preparation is irreversible and cannot be corrected. A minimum consequence is weakening the tooth and subsequent coronal fracture. Evidence indicates that appropriate access and strategic removal of tooth structure that does not involve the marginal ridges will not significantly weaken the remaining coronal structure.[48] The marginal ridges provide the faciolingual strength to the crown[49]; access openings do not require removal of tooth structure in this area.[50]

The ultimate result of removing excessive tooth structure is perforation. Perforations in single-rooted teeth are located on the lateral surface. In multirooted teeth, perforations may be lateral or furcal (see Chapter 18).

LENGTH DETERMINATION

Radiographic

The working length is defined as the distance from a predetermined coronal reference point (usually the incisal edge in anterior teeth and a cusp tip in posterior teeth) to the point that the cleaning and shaping, and obturation should terminate. The reference point must be stable so fracture does not occur between visits. Unsupported cusps that are weakened by caries or restorations should be reduced. The point of termination is empirical, and based on anatomic studies, it should be 1.0 mm from the radiographic apex.[51,52] This accounts for the deviation of the foramen from the apex, and the distance from the major diameter of the foramen to the area where a dentinal matrix can be established apically.

Before access an estimated working length is calculated by measuring the total length of the tooth on the diagnostic parallel radiograph or digital image. In cases where the canal is curved, the canal length can be measured by placing a file that has been curved to duplicate the canal morphology against the film. The stop can be adjusted to coincide with the reference point, while the file tip is aligned with the radiographic apex. After adjustment of the stop, the file is straightened and the length measured. From a practical perspective, a calculation to the nearest 0.5 mm should be made. Then 2.0 millimeters are subtracted to account for the foramen distance (1.0 mm) and radiographic image distortion/magnification (1.0 mm).[53] This provides a safety factor so instruments are not placed beyond the apex. Violation of the apex may result in inoculation of the periapical tissues with necrotic tissue, debris, and bacteria[54] and lead to extrusion of materials during obturation[55,56] and a decreased prognosis.[57]

After access preparation, a small file is used to explore the canal and establish patency to the estimated working length. The largest file to bind is then inserted to this estimated length because a file that is loose in the canal may be displaced during film exposure or forced beyond the apex if the patient bites down inadvertently. Millimeter markings on the file shaft or rubber stops on the instrument shaft are used for length control. A sterile millimeter ruler or measuring device can be used to adjust the stops on the file. To ensure accurate measurement and length control during canal preparation, the stop must physically contact the coronal reference point. To obtain an accurate measurement, the minimum size of the working length should be a No. 20. With files smaller than No. 20, it is difficult to interpret the location of the file tip on the working length film or digital image. In multirooted teeth, files are placed in all canals before exposing the film.

Angled films are necessary to separate superimposed files and structures (Figure 14-40),[58] to provide an efficient method of determining the working length, and to reduce radiation to the patient. It is imperative that the rubber dam be left in place during working length determination to ensure an aseptic environment and to protect the patient from swallowing or aspirating instruments. The film can be held with a hemostat or a positioning device (Figure 14-41).

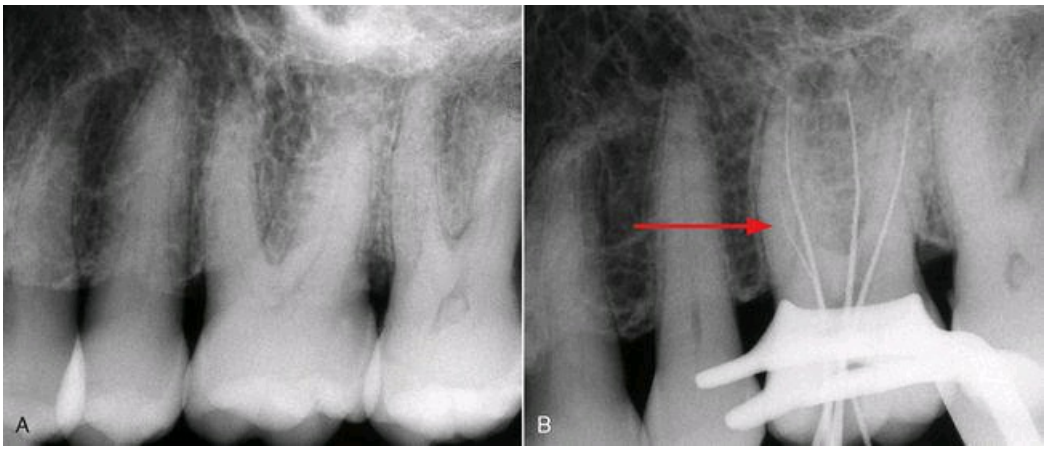

Figure 14-40 A, Parallel preoperative radiograph. **B,** The mesial working length film is made correctly. The apices and file tips are clearly visible. Note the mesiolingual canal *(arrow).*

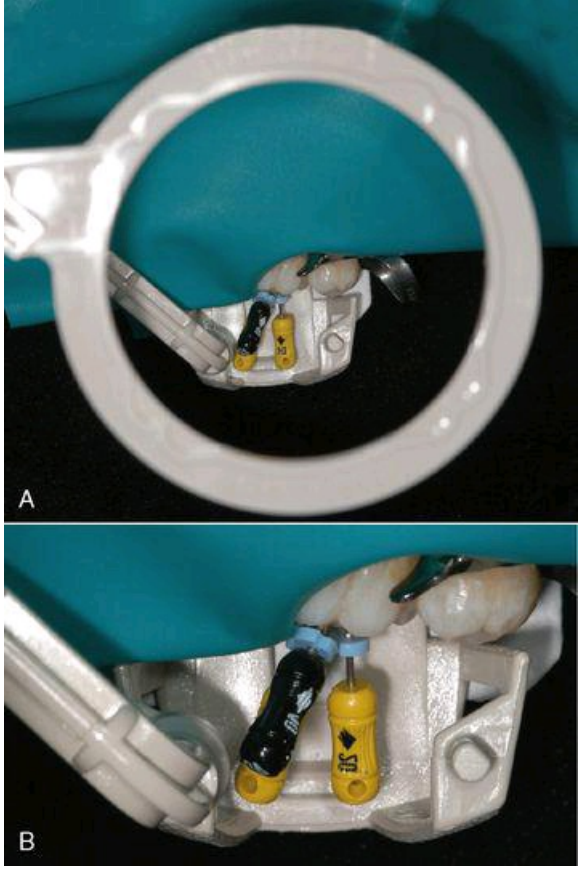

Figure 14-41 A, Positioning device to hold working films. The ring assists in cone alignment. **B,** A close-up view of the device in position.

A modified paralleling technique is used to position the film and the cone and has been shown to be superior to the bisecting-angle technique.[59,60] With the modified paralleling technique, the film is positioned by using a hemostat approximately parallel to the long axis of the tooth. The cone is then positioned so the central beam will strike the film at a 90-degree angle (Figure 14-42). Although this technique is reliable, it is not foolproof.[61]

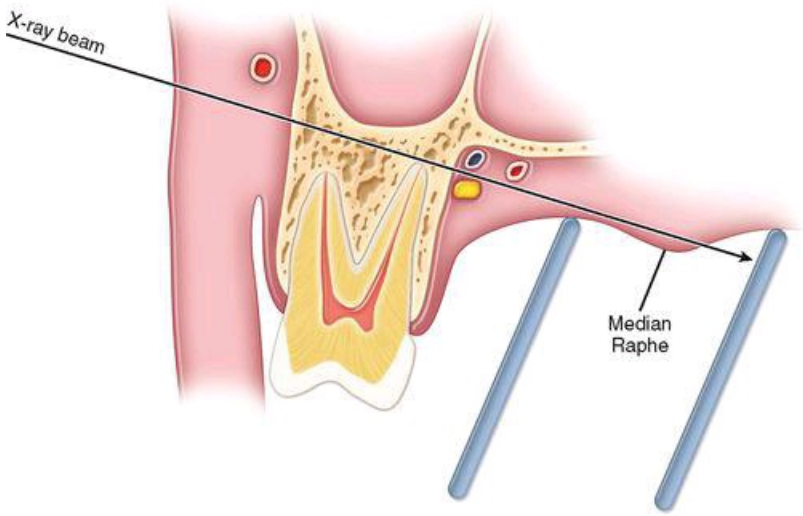

Figure 14-42 Proper positioning of the radiograph when making a working length radiograph. To capture the palatal root the film should be placed on the opposite side of the midline.

Other clinical factors should be considered in establishing the corrected working length. These include tactile sensation,[62] the patient's response, and hemorrhage. The use of tactile sensation may be valuable in large tapering canals; however, in small cylindrical canals, the rate of taper of the files may exceed the rate of taper of the canal and binding occurs coronally, giving the false sense of the constriction. Preflaring the canal before length determination increases the tactile sensation significantly when compared to unflared canals.[63]

After the film or sensor exposure, the corrected working length is calculated. The distance from the file tip to the radiographic apex is determined. When the distance is greater than 1.0 mm, a calculation (adding of length or subtracting) is made so the file tip will be positioned 1.0 mm from the radiographic apex. When the correction is greater than 3.0 mm, it is advisable to make a second working length radiograph with the file placed at the adjusted length.

With angled radiographs, the canal determination is based on the buccal object or SLOB rule (same lingual, opposite buccal) (see Chapter 11).[64,65] Because maxillary anterior teeth have only one canal, no angle is necessary. Mesial angles are recommended for premolars and maxillary molars (Figure 14-43). Distal angulation is recommended for the mandibular incisors and molars (Figure 14-44). For maxillary posterior teeth, the film should be placed on the opposite side of the midline to facilitate capturing the palatal roots on the film (see Figure 14-42).

Figure 14-43 Separation of the mesiobuccal and mesiolingual canals achieved by varying the horizontal angle. With maxillary molars maximum separation occurs with a mesial cone angulation because of the mesial location of the mesiolingual canal in relation to the mesiobuccal canal.

Figure 14-44 Separation of the mesiobuccal and the mesiolingual canals achieved by varying the horizontal angle. With mandibular molars, maximum separation occurs with a distal orientation because of the mesial location of the mesiobuccal canal in relation to the mesiolingual canal.

Electronic Apex Locators

Apex locators are also used in determining length.[66,67] Contemporary apex locators are based on the principle that the flow of higher frequencies of alternating current is facilitated in a biologic environment when compared to lower frequencies. Passing two differing frequencies through the canal results in the higher frequency impeding the lower frequency (Figure 14-45). The impedance values that change relative to each other are measured and converted to length information. At the apex, the impedance values are at their maximum differences. Unlike previous models the impedance apex locator operates accurately in the presence of electrolytes.[68] Apex locators are helpful in length determination but must be confirmed with radiographs. Films/digital images aid in confirming the appropriate length and can identify missed canals. When the file is not centered in the root, a second canal is likely to be present.

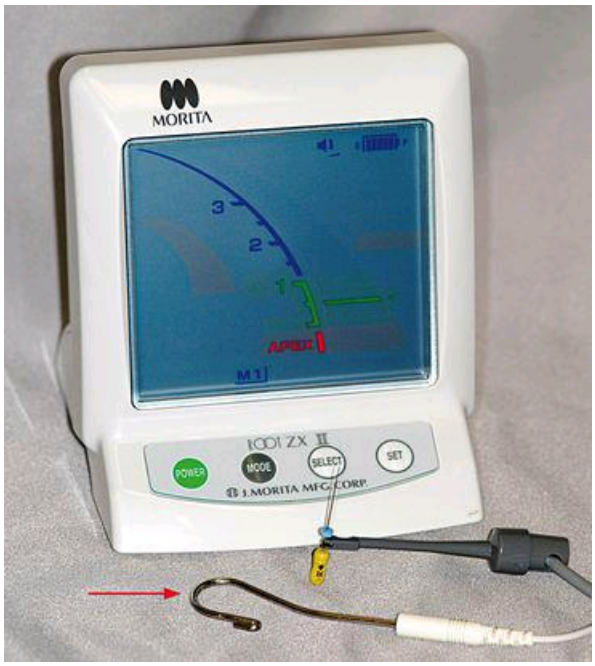

Figure 14-45 An impedance apex locator. Note the lip clip *(arrow)*.

An apex locator is very helpful in patients with structures or objects that obstruct visualization of the apex, patients that have a gag reflex and cannot tolerate films, and patients with medical problems that prohibit the holding of a film or sensor.

The use of apex locators and electric pulp testers in patients with cardiac pacemakers has been questioned.[69-73] In a recent study involving 27 patients with either implanted cardiac pacemakers or cardioverter/defibrillators, 2 impedance apex locators and 1 electric pulp tester did not interfere with the functioning of any of the cardiac devices.[74]

Chapter Review Questions available in Appendix B or on the DVD

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* See Appendix A, Pulpal Anatomy and Access Preparations, for colorized illustrations that depict the size, shape, and location of the pulp space within each tooth.

Cleaning and Shaping

William T. Johnson, W. Craig Noblett

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. State reasons and describe situations for enlarging the cervical portion of the canal before performing straight-line access.
2. Define how to determine the appropriate size of the master apical file.
3. Describe objectives for both cleaning and shaping and explain how to determine when these have been achieved.
4. Diagram “perfect” shapes of flared (step-back) and standardized preparations and draw these both in longitudinal and cross-sectional diagrams.
5. Diagram probable actual shapes of flared (step-back) and standardized preparations in curved canals.
6. Describe techniques for shaping canals that have irregular shapes such as round, oval, hourglass, bowling pin, kidney bean, or ribbon.
7. Describe techniques (step-back or crown-down) for standardized and flaring preparations.
8. Distinguish between apical stop, apical seat, and open apex and discuss how to manage obturation in each.
9. Describe the technique of removing the pulp.
10. Characterize the difficulties of preparation in the presence of anatomic aberrations that make complete débridement difficult.
11. List properties of the “ideal” irrigant and identify which irrigant meets most of these criteria.
12. Describe the needles and techniques that provide the maximal irrigant effect.
13. Discuss the properties and role of chelating and decalcifying agents.
14. Explain how to minimize preparation errors in small curved canals.
15. Describe techniques for negotiating severely curved, “blocked,” or constricted canals.
16. Describe in general the principles of application of ultrasonic devices for cleaning and shaping.
17. Evaluate in general alternative means of cleaning and shaping and list advantages and disadvantages of each.
18. Discuss nickel-titanium hand and rotary instruments and how the physical properties of this metal affect cleaning and shaping.
19. Discuss the properties and role of intracanal, interappointment medicaments.
20. List the principal temporary filling materials and describe techniques for their placement and removal.
21. Describe temporization of extensively damaged teeth.
22. Outline techniques and materials used for long-term temporization.

Successful root canal treatment is based on establishing an accurate diagnosis and developing an appropriate treatment plan; applying knowledge of tooth anatomy and morphology (shape); and performing the débridement, disinfection, and obturation of the entire root canal system. Initially, emphasis was on obturation and sealing the radicular space. However, no technique or material provides a seal that is completely impervious to moisture either from the apical or coronal areas. Early prognosis studies indicated failures were attributed to incomplete obturation.[1] This proved fallacious as obturation only reflects the adequacy of the cleaning and shaping. Canals that are poorly obturated are often incompletely cleaned and shaped. Adequate cleaning and shaping and establishing a coronal seal are the essential elements for successful treatment, with obturation being less important for short-term success.[2] Elimination (or significant reduction) of the inflamed or necrotic pulp tissue and microorganisms are the most critical factors. The role of obturation in long-term success has not been established but may be significant in preventing recontamination either from the coronal or apical areas. Sealing the canal space after cleaning and shaping will entomb any remaining organisms[3] and, with the coronal seal, prevent recontamination of the canal and periradicular tissues.

PRINCIPLES OF CLEANING

Nonsurgical root canal treatment is a predictable method of retaining a tooth that otherwise would require extraction. Success of root canal treatment in a tooth with a vital pulp is higher than that of a tooth that is necrotic with periradicular pathosis. The difference is the persistent irritation of necrotic tissue remnants and the inability to remove the microorganisms and their by-products. The most significant factors affecting this process are tooth anatomy and morphology and the instruments and irrigants available for treatment. Instruments must *contact and plane the canal walls* to débride the canal (Figures 15-1 to 15-3). Morphologic factors include lateral (see Figure 15-2) and accessory canals, canal curvatures, canal wall irregularities, fins, cul-de-sacs (see Figure 15-1), and isthmuses. These aberrations make total débridement virtually impossible. Therefore, the objective of cleaning is to reduce the irritants, not totally eliminate them.

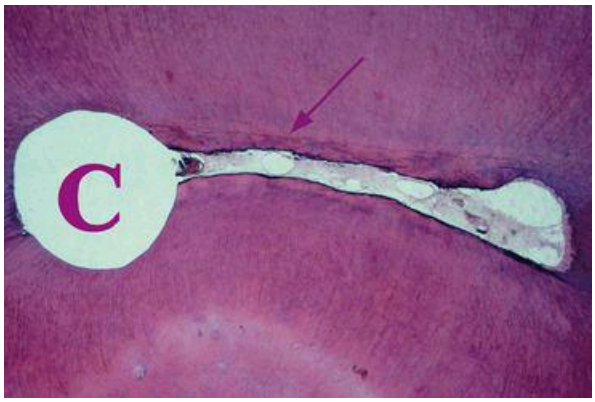


Figure 15-1 Cross-section through a root showing the main canal *(C)* and a fin *(arrow)* and associated cul-de-sac after cleaning and shaping using files and sodium hypochlorite. Note the tissue remnants in the fin.

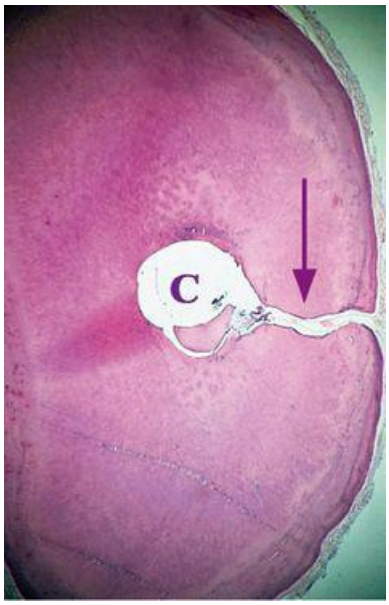


Figure 15-2 The main canal *(C)* has a lateral canal *(arrow)* extending to the root surface. After cleaning and shaping with sodium hypochlorite irrigation, tissue remains in the lateral canal.

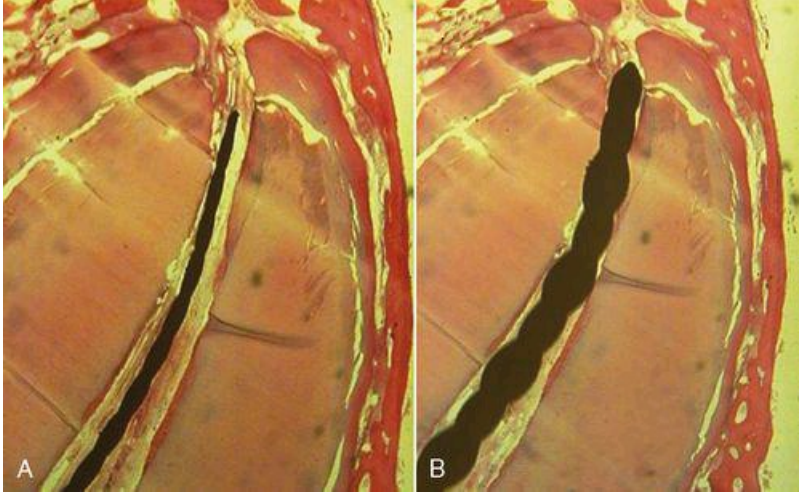


Figure 15-3 A, No. 15 file in the apical canal space. Note the size is inadequate for planing the walls. **B,** No. 40 file more closely approximates the canal morphology.

(Courtesy Dr. Randy Madsen.)

Currently, there are no reliable methods to assess cleaning. The presence of clean dentinal shavings, the color of the irrigant, and canal enlargement three file sizes beyond the first instrument to bind have been used to assess the adequacy of cleaning; however, these do not correlate well with débridement. Obtaining *glassy smooth walls* is a preferred indicator.[4] The properly prepared canals should feel smooth in all dimensions when the tip of a small file is pushed against the canal walls. This indicates that files have had contact and planed all accessible canal walls, thereby maximizing débridement (recognizing that total débridement usually does not occur).

PRINCIPLES OF SHAPING

The purpose of shaping is to facilitate cleaning and provide space for placing the obturating materials. The main objective of shaping is to maintain or develop a continuously tapering funnel from the canal orifice to the apex. This decreases procedural errors when enlarging apically. The degree of enlargement is often dictated by the method of obturation. For lateral compaction of gutta–percha, the canal should be enlarged sufficiently to permit placement of the spreader to within 1 to 2 mm of the corrected working length. There is a correlation between the depth of spreader penetration and the quality of the apical seal.[5] For warm vertical compaction techniques, the coronal enlargement must permit the placement of the pluggers to within 3 to 5 mm of the corrected working length.[6]

As dentin is removed from the canal walls, the root is weakened.[7] The degree of shaping is determined by the preoperative root dimension, the obturation technique, and the restorative treatment plan. Narrow thin roots, such as the mandibular incisors, cannot be enlarged to the same degree as more bulky roots such as the maxillary central incisors. Post placement is also a determining factor in the amount of coronal dentin removal.

APICAL CANAL PREPARATION

Termination of Cleaning and Shaping

Although the concept of cleaning and shaping the root canal space is simple, there are areas where consensus does not exist. The first is the extent of the apical preparation.

Early studies identified the dentinocemental junction as the area where the pulp ends and the periodontal ligament begins. Unfortunately, this is a histologic landmark and the position (which is irregular within the canal) cannot be determined clinically.

Traditionally, the apical point of termination has been 1 mm from the radiographic apex. In a classic study, it was noted the apical portion of the canal consisted of the major diameter of the foramen and the minor diameter of the constriction (Figure 15-4).[8] The apical constriction is defined as the narrowest portion of the canal, and the average distance from the foramen to the constriction was found to be 0.5 mm. One study found the classic apical constriction to be present in only 46% of the teeth and when present, varied in shape and in relation to the apical foramen.[9] Variations from the classic appearance consist of the tapering constriction, the multiple constriction, and the parallel constriction.[9] Based on the variations in apical morphology, the term *apical constriction* is a misnomer. To complicate the issue, the foramen is seldom at the anatomic apex.

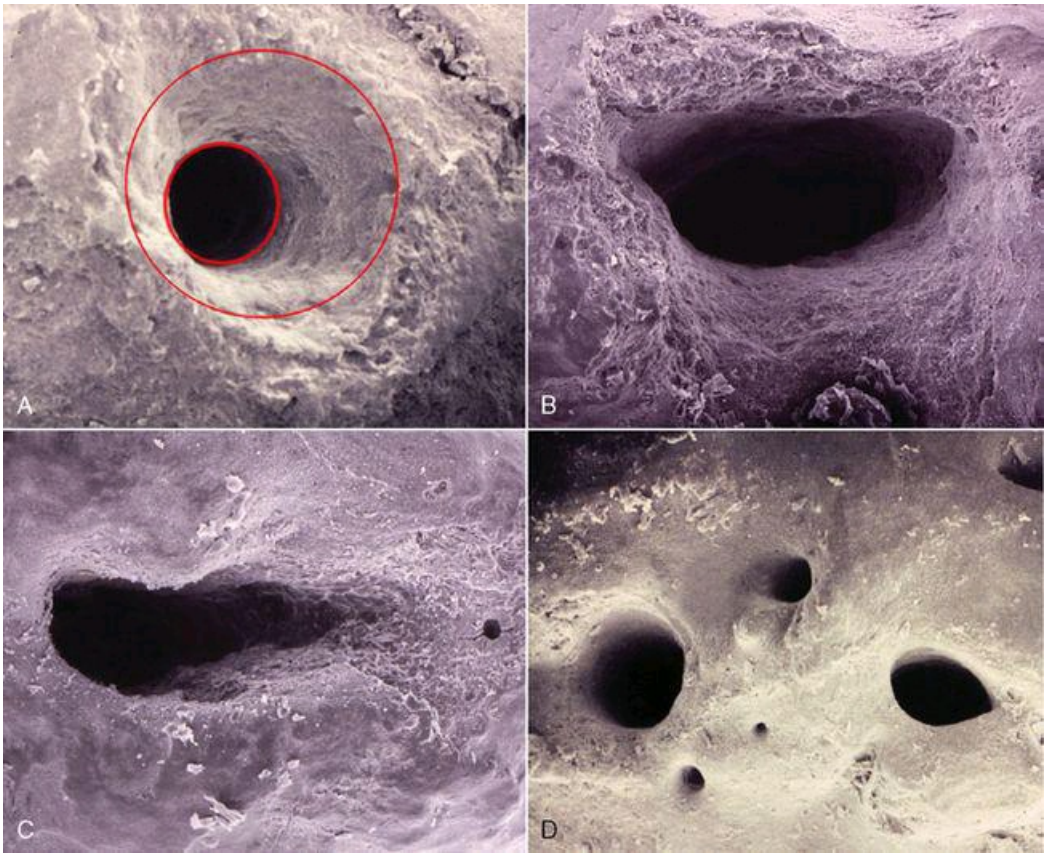


Figure 15-4 A, The classic apical anatomy consisting of the major diameter of the foramen and the minor diameter of the constriction. **B,** An irregular ovoid apical canal shape and external resorption. **C,** A bowling pin apical morphology and an accessory canal. **D,** Multiple apical foramina.

Apical anatomy has also been shown to be quite variable (see Figure 15-4). A recent study found no typical pattern for foraminal openings and that no foramen coincided with the apex of the root.[10] The foramen to apex distance can range from 0.20 to 3.8 mm.[10]

It has also been noted that the foramen to constriction distance increases with age,[8] and root resorption may destroy the classic anatomical constriction. Resorption is common with pulp necrosis and apical bone resorption and can result in loss of the constriction.[11] Therefore root resorption is an additional factor to consider in length determination.

In a recent prospective study evaluating prognosis, significant adverse factors influencing success and failure were perforation, preoperative periradicular disease, and incorrect length of the root canal filling.[12] The authors speculated that canals filled more than 2.0 mm short harbored necrotic tissue, bacteria, and irritants that when retreated could be cleaned and sealed.[12] A meta-analysis evaluation of success and failure indicated a better success rate when the obturation was confined to the canal space.[13] A review of a number of prognosis studies confirms that extrusion of materials decreases success.[14] With pulp necrosis, better success was achieved when the procedures terminated at or within 2 mm of the radiographic apex. Obturation shorter than 2 mm from the apex or past the apex resulted in a decreased success rate. In teeth with vital inflamed pulp tissue, termination between 1 and 3 mm was acceptable.

Whereas the guideline of 1 to 3 mm from the radiographic apex remains rational, the point of apical termination of the preparation and obturation remains empirical. The need to compact the gutta-percha and sealer against the apical dentin

matrix (constriction of the canal) is important for success. The decision of where to terminate the preparation is based on knowledge of apical anatomy, tactile sensation, radiographic interpretation, apex locators, apical bleeding, and the patient's response. To prevent extrusion, the cleaning and shaping procedures should be confined to the radicular space. Canals filled to the radiographic apex are actually slightly overextended.[10]

Degree of Apical Enlargement

Generalizations can be made regarding tooth anatomy and morphology, although each tooth is unique. Length of canal preparation is often emphasized with little consideration given to important factors such as canal diameter and shape. Because morphology is variable, there is no standardized apical canal size. Traditionally, preparation techniques were determined by the desire to limit procedural errors and by the method of obturation. Small apical preparation limits canal transportation and apical "zipping" but decreases the efficacy of the cleaning procedure. It appears that with traditional hand files, apical transportation occurs in most curved canals enlarged beyond a No. 25 stainless steel file.[15] The criteria for cleaning and shaping should be based on the ability to adequately remove the tissue, necrotic debris, and bacteria and not on a specific obturation technique.

Irrigants are unable to reach the apical portion of the root if the canal is not enlarged to a No. 35 or 40 file.[16-18] The larger preparation sizes have been shown to provide adequate irrigation and debris removal and significantly decrease the number of microorganisms.[19-22] Thus there appears to be a relationship between increasing the size of the apical preparation and canal cleanliness[23] and bacterial reduction.[24,25] Instrumentation techniques that advocate minimal apical preparation may be ineffective at achieving the goal of cleaning and disinfecting the root canal space.[23,26]

Bacteria can penetrate the tubules of dentin. These intratubular organisms are protected from endodontic instruments, the action of irrigants, and intracanal medicaments. Dentin removal appears to be the primary method for decreasing their numbers. In addition, it may not be possible to remove bacteria that are deep in the tubules, regardless of the technique. There is a correlation between the number of organisms present and the depth of tubular penetration[27]; in teeth with apical periodontitis, bacteria may penetrate the tubules to the periphery of the root.[28,29]

Elimination of Etiology

The development of nickel-titanium instruments has dramatically changed the techniques of cleaning and shaping. The primary advantage to using these flexible instruments is related to shaping. Neither hand instruments nor rotary files have been shown to completely débride the canal.[30-32] Mechanical enlargement of the canal space dramatically decreases the presence of microorganisms present in the canal[33] but cannot render the canal sterile.[19] To improve the mechanical preparation techniques, antimicrobial irrigants have been recommended.[34] There is no consensus on the most appropriate irrigant or concentration of solution, although sodium hypochlorite (NaOCl) is the most widely used irrigant.

Common irrigants include NaOCl and chlorhexidine.[35-39] Unfortunately, solutions designed to kill bacteria are often toxic for the host cells,[40-43] therefore extrusion beyond the canal space is to be avoided.[44,45] A major factor related to effectiveness is the volume. Increasing the volume produces cleaner preparations.[46]

Apical Patency

Apical patency is a technique whereby small hand files are frequently inserted to or slightly beyond the apical foramen during canal preparation. This has been advocated during cleaning and shaping procedures to ensure working length is not lost and that the apical portion of the root is not packed with tissue, dentin debris, and bacteria (Figure 15-5). With apical patency, concerns regarding extrusion of dentinal debris, bacteria, and irrigants have been raised.[47] Seeding the periradicular tissues with microorganisms may occur.[48] Studies evaluating treatment failure have noted bacteria outside the radicular space,[49,50] and bacteria have been shown to exist as plaques or biofilms on the external root structure.[51]

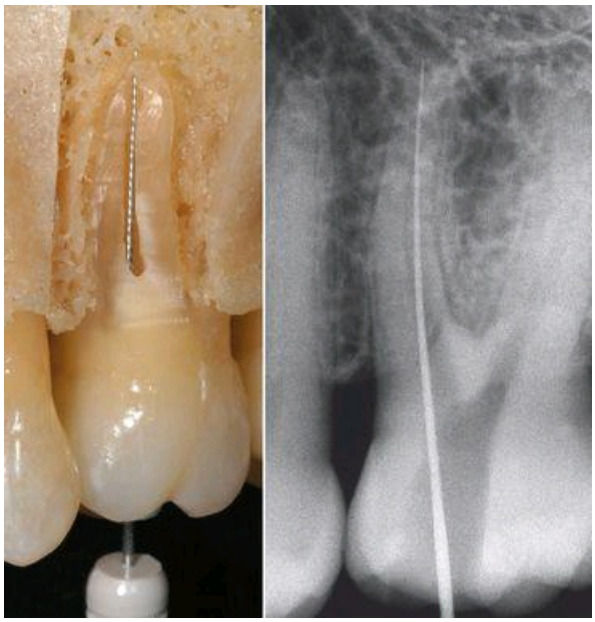


Figure 15-5 A small file (No. 10 or 15) is placed beyond the radiographic apex to maintain patency of the foramen. Note the tip extends beyond the apical foramen.

The apical patency concept also has been advocated to facilitate apical preparation. Extending the file beyond the apex increases the diameter of the canal at working length consistent with the instrument taper. The value of maintaining patency to prevent transportation is questionable,[52] and maintaining patency does not result in bacterial reduction when compared to *not* maintaining it.[53] Small files are not effective in débridement (see Figure 15-3). Therefore, the use of apical patency is not biologically rational.

PRETREATMENT EVALUATION

Before treatment, each case should be evaluated for degree of difficulty. Normal anatomy and anatomic variations are determined, as well as variations in canal morphology (shape).

A parallel preoperative radiograph or image is assessed. The longer a root, the more difficult it is to treat. Apically, a narrow curved root is susceptible to perforation; in multirooted teeth a narrow area midroot could lead to a lateral stripping perforation. The degree and location of curvature are determined. Canals are seldom straight, and curvatures in a faciolingual direction will not be visible on the radiograph. Sharp curvatures or dilacerations are more difficult to manage than a continuous gentle curve. Roots with an S-shape or bayonet configuration are difficult to treat. Calcifications will also complicate treatment. Calcification generally occurs in a coronal to apical direction (see Figure 14-14). A large tapering canal may become more cylindrical with irritation or age, and this presents problems when tapered instruments are used in the coronal third.

Resorption also will complicate treatment. With internal resorption, it is difficult to pass instruments through the coronal portion of the canal and the resorptive defect and into the apical portion. Also, files will not remove tissue, necrotic debris, and bacteria from this inaccessible area. External resorptions may perforate the canal space and present problems with hemostasis and isolation. Restorations may obstruct access and visibility, as well as change the orientation of the crown in relation to the root.

PRINCIPLES OF CLEANING AND SHAPING TECHNIQUES

Cleaning and shaping are separate and distinct concepts but are performed concurrently. The criteria of canal preparation include developing a continuously tapered funnel, maintaining the original shape of the canal, maintaining the apical foramen in its original position, keeping the apical opening as small as possible, and developing glassy smooth walls.[6] The cleaning and shaping procedures are designed to maintain an apical matrix for compacting the obturating material regardless of the obturation technique.[6]

Knowledge of a variety of techniques and instruments for treatment of the myriad variations in canal anatomy is required. There is no consensus on which technique or instrument design or type is clinically superior.[30]

Nickel-titanium files have been incorporated into endodontics because of their flexibility and resistance to cyclic fatigue.[54] The resistance to cyclic fatigue permits these instruments to be used in a rotary handpiece, which gives them an advantage over stainless steel files. Nickel-titanium files are manufactured in both hand and rotary versions and have been demonstrated to produce superior shaping when compared to stainless steel hand instruments.[55,56]

The instruments are designed with increased taper when compared to .02 mm standardized stainless steel files. Common tapers are .04, .06, .08, .10, and .12 mm, and the tip diameters may or may not conform to the traditional manufacturing specifications. The file systems can vary the taper while maintaining the same tip diameter or they can employ varied tapers with ISO standardized tip diameters. They may incorporate cutting or noncutting tips.

In general, the nickel-titanium rotary instruments are not indicated in S-shaped canals, canals that join within a single root (type II configuration), canals with severe dilacerations, canals in which ledge formation is present, and very large canals where they fail to contact the canal walls. Straight-line access to the canal is essential, and the instruments should be used passively.

Instrument fracture can occur as a result of torsional forces or cyclic fatigue. Torsional forces develop because of frictional resistance; therefore, as the surface area increases along the flutes, the greater the friction and the more potential for fracture. Torsional forces may produce an unraveling of the flutes before fracture, and inspection of the instruments after each use is critical. Torsional stress can be reduced by limiting file contact, using a crown-down preparation technique, and lubrication. Cyclic fatigue occurs as the file rotates in a curved canal.[57] At the point of curvature the molecules on the outer surface of the file are under tension while the molecules on the inner surface of the instrument are compressed. As the instrument rotates, the areas of tension and compression alternate and eventual fracture occurs. There is no visible evidence that fracture is imminent. Therefore it is advised that the use of nickel-titanium instruments be monitored[58] and limited to one to five cases. For difficult or calcified or severely curved canals, it is recommended the instruments be used only once.

Ultrasonics

Ultrasonics are used for cleaning and shaping, removal of materials from the canal, removal of posts and silver cones, thermoplastic obturation, and root-end preparation during surgery.

The main advantage to cleaning and shaping with ultrasonics is acoustic microstreaming,[59] which is described as complex steady-state streaming patterns in vortex-like motions or eddy flows that are formed close to the instrument. Agitation of the irrigant with an ultrasonically activated file after completion of cleaning and shaping has the benefit of increasing the effectiveness of the solution.[60-63]

Initially, it was proposed that ultrasonics could clean the canal without procedural errors, such as apical transportation, and remove the smear layer.[64,65] However, later studies failed to confirm these results.[66-68]

IRRIGANTS

The ideal properties for an endodontic irrigant are listed in Box 15-1.[69] Currently, no solution meets all the requirements outlined.

Box 15-1 Properties of an Ideal Irrigant

- Organic tissue solvent
- Inorganic tissue solvent
- Antimicrobial action
- Nontoxic
- Low surface tension
- Lubricant

Irrigation does not completely débride the canal. NaOCl will not remove tissue from areas that are not touched by files (see Figures 15-1 and 15-2).[70] In fact, no techniques appear able to completely clean the root canal space.[22,71-73] Frequent irrigation is necessary to flush and remove the debris generated by the mechanical action of the instruments.

Sodium Hypochlorite

The most common irrigant is NaOCl, which is also known as household bleach. Advantages to NaOCl include the mechanical flushing of debris from the canal, the ability of the solution to dissolve vital[74] and necrotic tissue,[75] the antimicrobial action of the solution,[32] and the lubricating action.[76] In addition, it is inexpensive and readily available.

Free chlorine in NaOCl dissolves necrotic tissue by breaking down proteins into amino acids. There is no proven appropriate concentration of NaOCl, but concentrations ranging from 0.5% to 5.25% have been recommended. A common concentration is 2.5%, which decreases the potential for toxicity yet still maintains some tissue dissolving and antimicrobial activity.[77,78] Because the action of the irrigant is related to the amount of free chlorine, decreasing the concentration can be compensated by increasing the volume. Warming the solution can also increase the effectiveness of the solution.[79,80] However, NaOCl has limitations to tissue dissolution in the canal, because of limited contact with tissues in all areas of the canal.

Because of toxicity, extrusion is to be avoided.[41,45,81] The irrigating needle must be placed *loosely* in the canal (Figure 15-6). Insertion to binding and slight withdrawal minimizes the potential for possible extrusion and a "sodium hypochlorite accident" (Figure 15-7). Special care should be exercised when irrigating a canal with an open apex. To control the depth of insertion the needle is bent slightly at the appropriate length or a rubber stopper is placed on the needle.

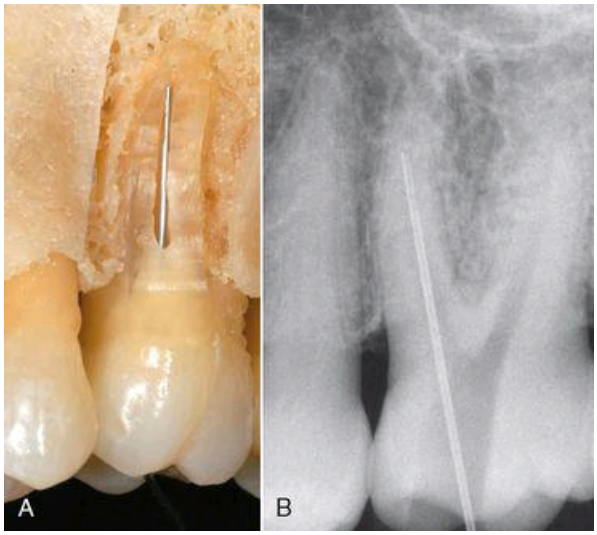


Figure 15-6 For effective irrigation the needle must be placed in the apical one-third of the root and must not bind.

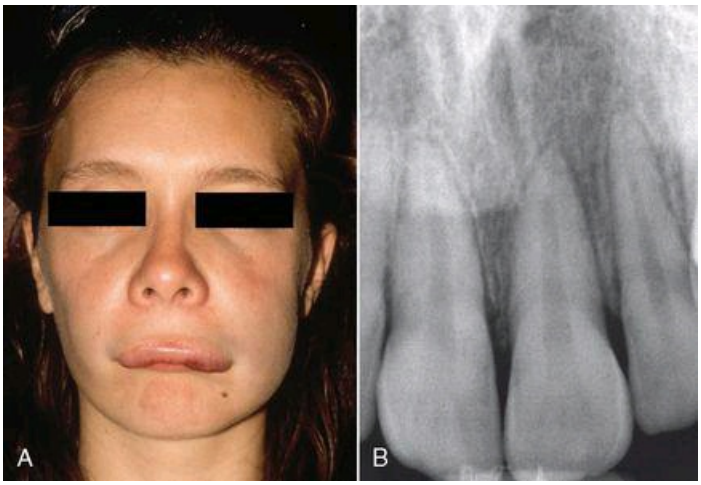


Figure 15-7 A sodium hypochlorite accident during treatment of the maxillary left central incisor. Extensive edema occurred in the upper lip and was accompanied by severe pain.

The irrigant does not move apically more than 1 mm beyond the irrigation tip, so deep placement with small-gauge needles enhances irrigation (see Figure 15-6).[82] Unfortunately, the small bore can easily clog, so aspiration of air after each use is recommended. During rinsing, the needle is moved up and down constantly to produce agitation and prevent binding or wedging of the needle.

Chlorhexidine

Chlorhexidine possesses a broad spectrum of antimicrobial activity, provides a sustained action,[81,83] and has little toxicity.[84-87] Two percent chlorhexidine has similar antimicrobial action as 5.25% NaOCl[84] and is more effective against *Enterococcus faecalis*.[81] NaOCl and chlorhexidine are synergistic in their ability to eliminate microorganisms.[85] A disadvantage of chlorhexidine is its inability to dissolve necrotic tissue and remove the smear layer.

LUBRICANTS

Lubricants facilitate file manipulation during cleaning and shaping. They are an aid in initial canal negotiation, especially in small constricted canals without taper. They reduce torsional forces on the instruments and decrease the potential for fracture.

Glycerin is a mild alcohol that is inexpensive, nontoxic, aseptic, and somewhat soluble. A small amount can be placed along the shaft of the file or deposited in the canal orifice. Counterclockwise rotation of the file carries the material apically. The file can then be worked to length using a watch winding or “twiddling” motion.

Paste lubricants can incorporate chelators. One advantage to paste lubricants is that they can suspend dentinal debris and prevent apical compaction. One proprietary product consists of glycol, urea peroxide, and ethylenediaminetetraacetic acid (EDTA) in a special water-soluble base. It has been demonstrated to exhibit an antimicrobial action.[88] Another type is composed of 19% EDTA in a water-soluble viscous solution.

A disadvantage to these EDTA compounds appears to be the deactivation of NaOCl by reducing the available chlorine[89] and potential toxicity.[90] The addition of EDTA to the lubricants has not proved to be effective.[91] In general, files remove dentin faster than the chelators can soften the canal walls. Aqueous solutions, such as NaOCl, should be used instead of paste lubricants when using nickel-titanium rotary techniques to reduce torque.[76]

SMEAR LAYER

During the cleaning and shaping, organic pulpal materials and inorganic dentinal debris accumulate on the radicular canal wall, producing an amorphous, irregular smear layer (Figure 15-8).[69] With pulp necrosis, the smear layer may be contaminated with bacteria and their metabolic by-products. The smear layer is superficial, with a thickness of 1 to 5 µm, and debris can be packed into the dentinal tubules in varying distances.[92]

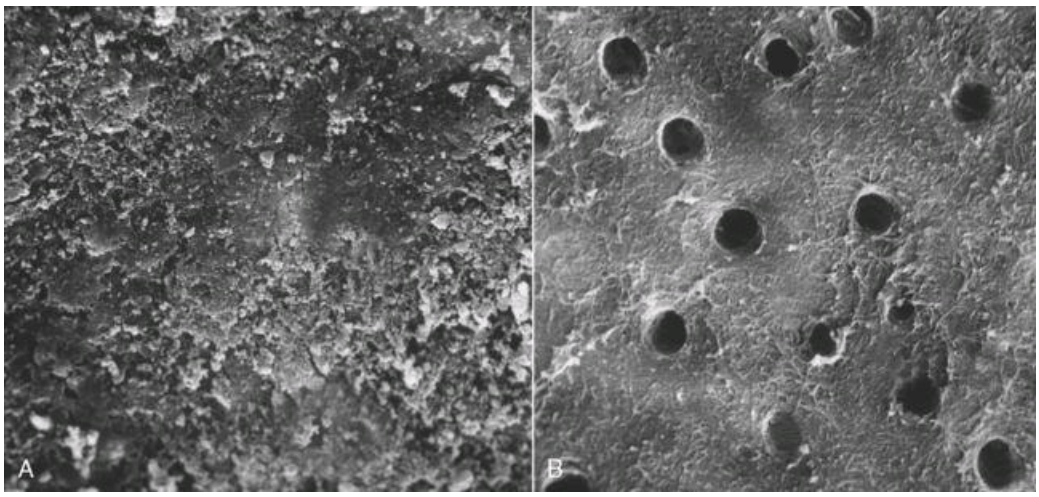


Figure 15-8 A, A canal wall with the smear layer present. **B,** The smear layer removed with 17% EDTA.

There is not a consensus on removing the smear layer before obturation.[69,93,94] The advantages and disadvantages of the smear layer removal remain controversial; however, evidence generally supports removing the smear layer prior to obturation.[69,95] The organic debris present in the smear layer might constitute substrate for bacterial growth, and it has been suggested that the smear layer prohibits sealer contact with the canal wall, which permits leakage. In addition, viable microorganisms in the dentinal tubules may use the smear layer as a substrate for sustained growth. When the smear layer is not removed, it may slowly disintegrate with leaking obturation materials, or it may be removed by acids and enzymes that are produced by viable bacteria left in the tubules or that enter via coronal leakage.[96] The presence of a smear layer may also interfere with the action and effectiveness of root canal irrigants and interappointment disinfectants.[37]

With smear layer removal, filling materials adapt better to the canal wall.[97,98] Removal of the smear layer also enhances the adhesion of sealers to dentin and tubular penetration[97-100] and permits the penetration of all sealers to varying depths.[101] Removal of the smear layer reduces both coronal and apical leakage.[102,103]

DECALCIFYING AGENTS

EDTA

Removal of the smear layer is accomplished with acids or other chelating agents such as ethylenediamine tetraacetic acid (EDTA)[104] after cleaning and shaping. Irrigation with 17% EDTA for 1 minute followed by a final rinse with NaOCl[105] is a recommended method. Chelators remove the inorganic components and leave the organic tissue elements intact. NaOCl is then necessary for removal of the remaining organic components. Citric acid has also been shown to be an effective method for removing the smear layer,[106,107] as has tetracycline.[108,109]

Demineralization results in removal of the smear layer and plugs and enlargement of the tubules.[110,111] The action is most effective in the coronal and middle thirds of the canal and reduced apically.[104,112] Reduced activity may be a reflection of canal size[62] or anatomic variations such as irregular or sclerotic tubules.[113] The variable structure of the apical dentin presents a challenge during endodontic obturation with adhesive materials.

The recommended time for removal of the smear layer with EDTA is 1 minute.[104,114,115] The small particles of the smear layer are primarily inorganic with a high surface to mass ratio, which facilitates removal by acids and chelators. EDTA exposure over 10 minutes causes excessive removal of both peritubular and intratubular dentin.[116]

MTAD

An alternative method for removing the smear layer employs a mixture of a tetracycline isomer, an acid, and a detergent (MTAD) as a final rinse to remove the smear layer.[117] The effectiveness of MTAD to completely remove the smear layer is enhanced when low concentrations of NaOCl are used as an intracanal irrigant before the use of MTAD.[118] A 1.3% concentration is recommended. MTAD may be superior to NaOCl in antimicrobial action.[119,120] MTAD has been shown to be effective in killing *E. faecalis*, an organism commonly found in failing treatments, and may prove beneficial during retreatment. It is biocompatible,[121] does not alter the physical properties of the dentin,[121] and enhances bond strength.[122]

TECHNIQUES OF PREPARATION

Regardless of the technique used in cleaning and shaping, procedural errors can occur. These include loss of working length, apical transportation, apical perforation, instrument fracture, and stripping perforations.

Loss of working length has several causes, including failure to have an adequate reference point from which the corrected working length is determined, packing tissue and debris in the apical portion of the canal, ledge formation, and inaccurate measurements of files.

Apical transportation and zipping occur when the restoring force of the file exceeds the threshold for cutting dentin in a cylindrical nontapering curved canal (Figures 15-9 and 15-10).[123] When this apical transportation continues with larger and larger files, a "teardrop" shape develops and *apical perforation* can occur on the lateral root surface (see Figure 15-9). Transportation in curved canals begins with a No. 25 file.[15] Enlargement of curved canals at the corrected working length beyond a No. 25 file can be done only when an adequate coronal flare is developed.

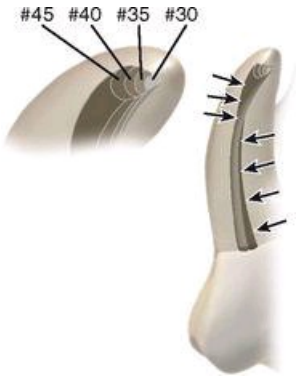


Figure 15-9 Procedural errors of canal transportation, zipping and strip perforation, occur during standardized preparation when files remove dentin from the outer canal wall apical to the curve and from the inner wall coronal to the curve. This is related to the restoring force (stiffness) of the files. Note in the apical portion the transportation takes the shape of a tear drop as the larger files are used.

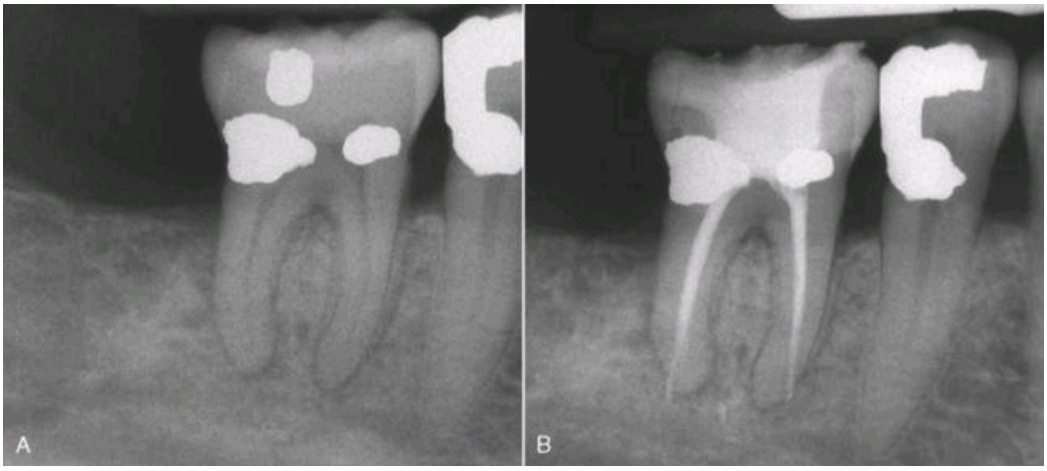


Figure 15-10 The canals have been transported, and there are apical perforations.

Instrument fracture occurs with torsional and cyclic fatigue. Locking the flutes of a file in the canal wall while continuing to rotate the coronal portion of the instrument is an example of torsional fatigue (Figure 15-11). Cyclic fatigue results when strain develops in the metal.

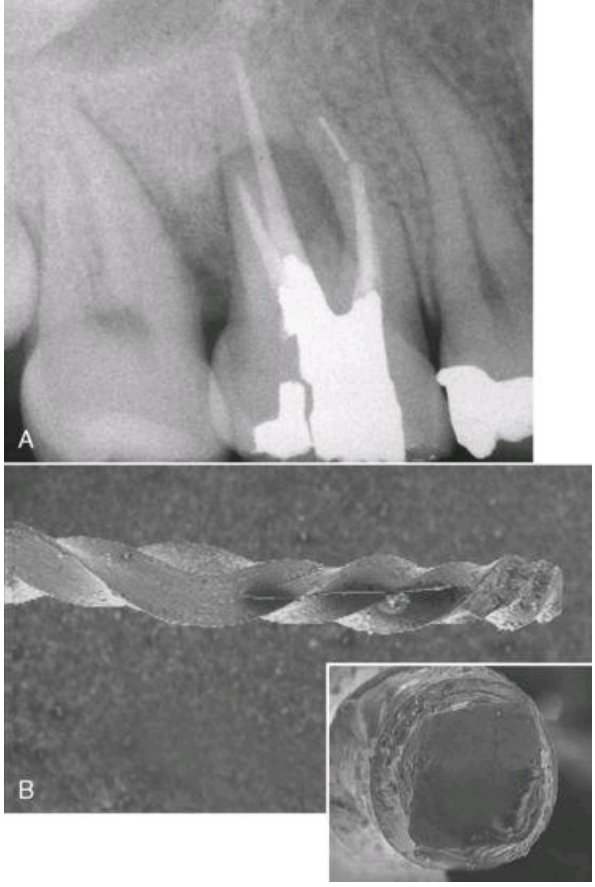


Figure 15-11 A, No. 35 file fractured in the mesiobuccal canal. **B,** Scanning electron microscope examination reveals torsional fatigue at the point of fracture. Note the tightening of the flutes near the fracture and the unwinding of the flutes along the shaft.

Stripping perforations occur in the furcal region of curved roots and frequently in the mesial roots of maxillary and mandibular molars (Figures 15-12 and 15-13). The canal in this area is not always centered in the root, and before preparation, the average distance to the furcal wall (danger zone) is less than the distance to the bulky outer wall (safety zone). An additional problem factor is the furcal concavity of the root.

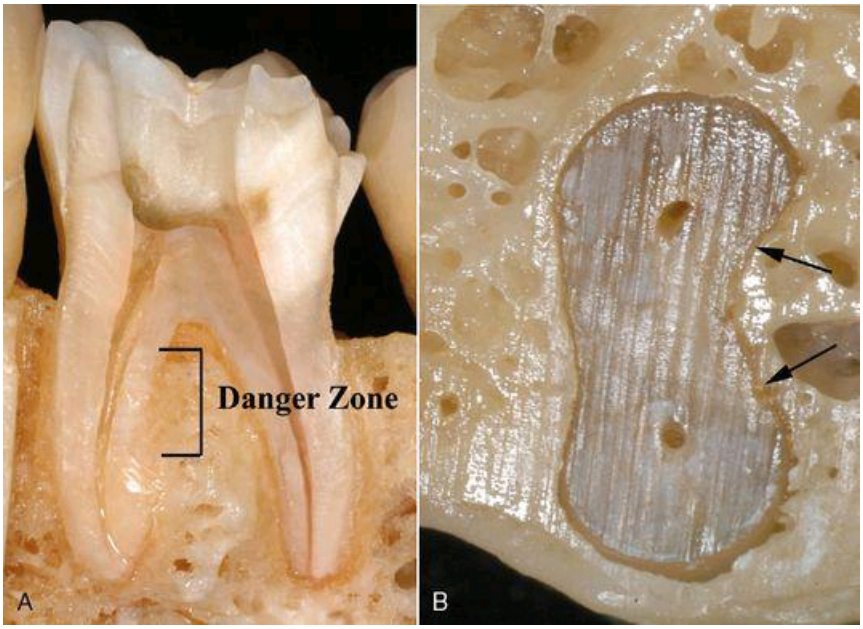


Figure 15-12 A, The furcal region of molars at the level of the curvature (danger zone) is a common site for stripping perforation. **B,** Note the concavity *(arrows)* in the furcation area of this mandibular molar.

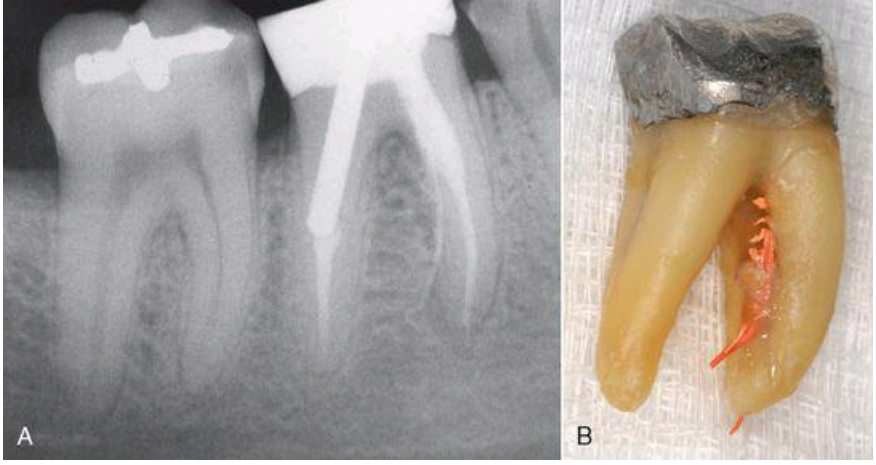


Figure 15-13 Straight-line access can result in stripping perforations in the furcal areas of molars. **A,** The use of large Gates-Glidden drills and overpreparation has resulted in the stripping perforation. **B,** Note that the perforation is in the concavity of the furcation.

Watch Winding

Watch winding (“twiddling”) is reciprocating back and forth (clockwise/counterclockwise) rotation of the instrument in an arch and is used to negotiate canals and to work files to place. Light apical pressure is applied to move the file deeper into the canal.

Reaming

Reaming is defined as the clockwise cutting rotation of the file. Generally, the instruments are placed into the canal until binding is encountered. The instrument is then rotated clockwise 180 to 360 degrees to plane the walls and enlarge the canal space.

Filing

Filing is defined as placing the file into the canal and pressing it laterally while withdrawing it along the path of insertion to scrape (plane) the wall. There is very little rotation on the outward cutting stroke. The scraping or rasping action removes the tissue and cuts superficial dentin from the canal wall. A modification is the turn-pull technique. This involves placing the file to the point of binding, rotating the instrument 90 degrees and pulling the instrument along the canal wall.

Circumferential Filing

Circumferential filing is used for canals that are larger and or not round. The file is placed into the canal and withdrawn in a directional manner sequentially against the mesial, distal, buccal, and lingual walls.

Standardized Preparation

After 1961, instruments were manufactured with a standard formula. Clinicians utilized a preparation technique of sequentially enlarging the canal space with smaller to larger instruments at the corrected working length.[124] In theory, this created a standardized preparation of uniform taper. Unfortunately this does not occur. This technique was adequate for preparing the apical portion of canals that were relatively straight and tapered; however, in cylindrical and small curved canals, procedural errors were identified with the technique.[125]

Step-Back Technique

The step-back technique reduces procedural errors and improves débridement.[70,125] After coronal flaring, the apical canal diameter is determined with the master apical file (MAF: the initial file that binds slightly at the corrected working length); the succeeding larger files are shortened by 0.5- or 1-mm increments from the previous file length (Figure 15-14 and 15-15). This step-back process creates a flared, tapering preparation while reducing procedural errors. The step-back preparation is superior to standardized serial filing and reaming techniques in débridement and maintaining the canal shape.[70] The step-back filing technique results in more pulpal walls being planed when compared to reaming or filing.

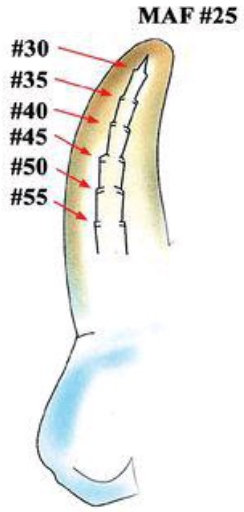
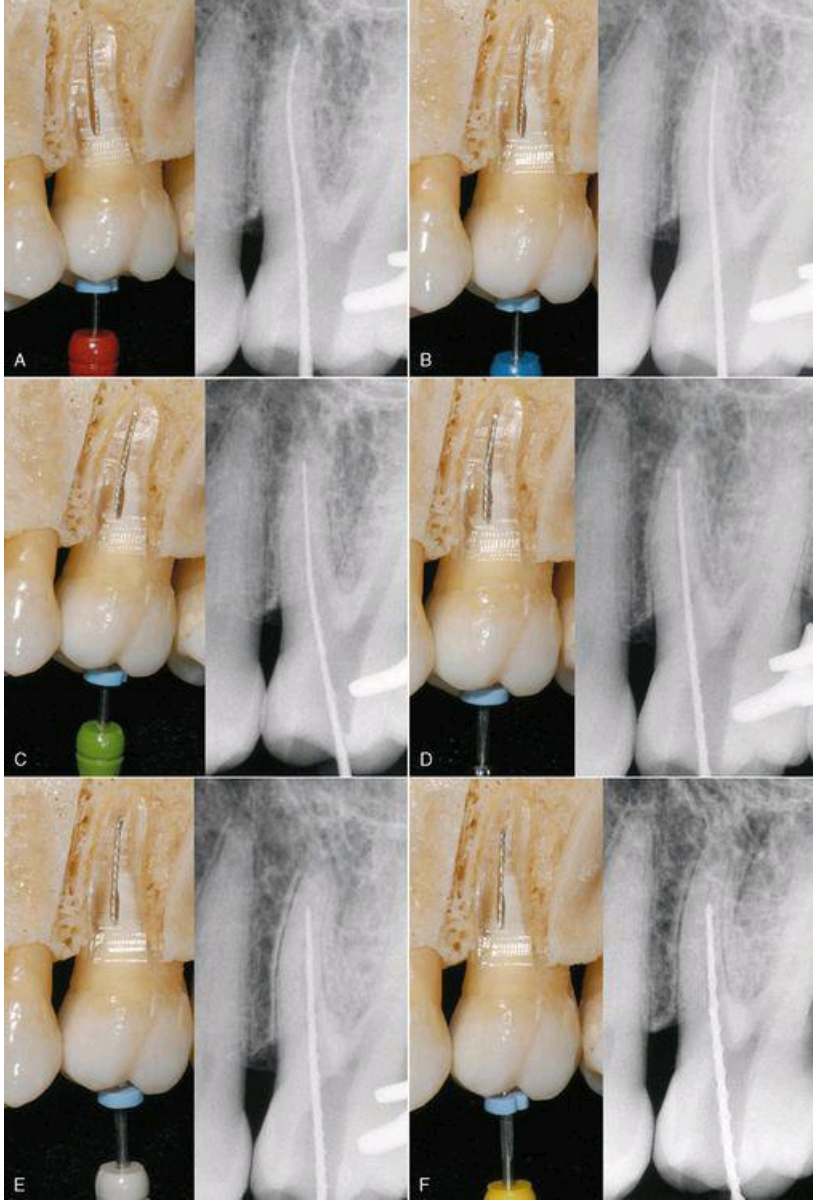


Figure 15-14 The step-back preparation is designed to provide a tapering preparation. The process begins with one file size larger than the master apical file with incremental shortening of either 0.5 or 1 mm.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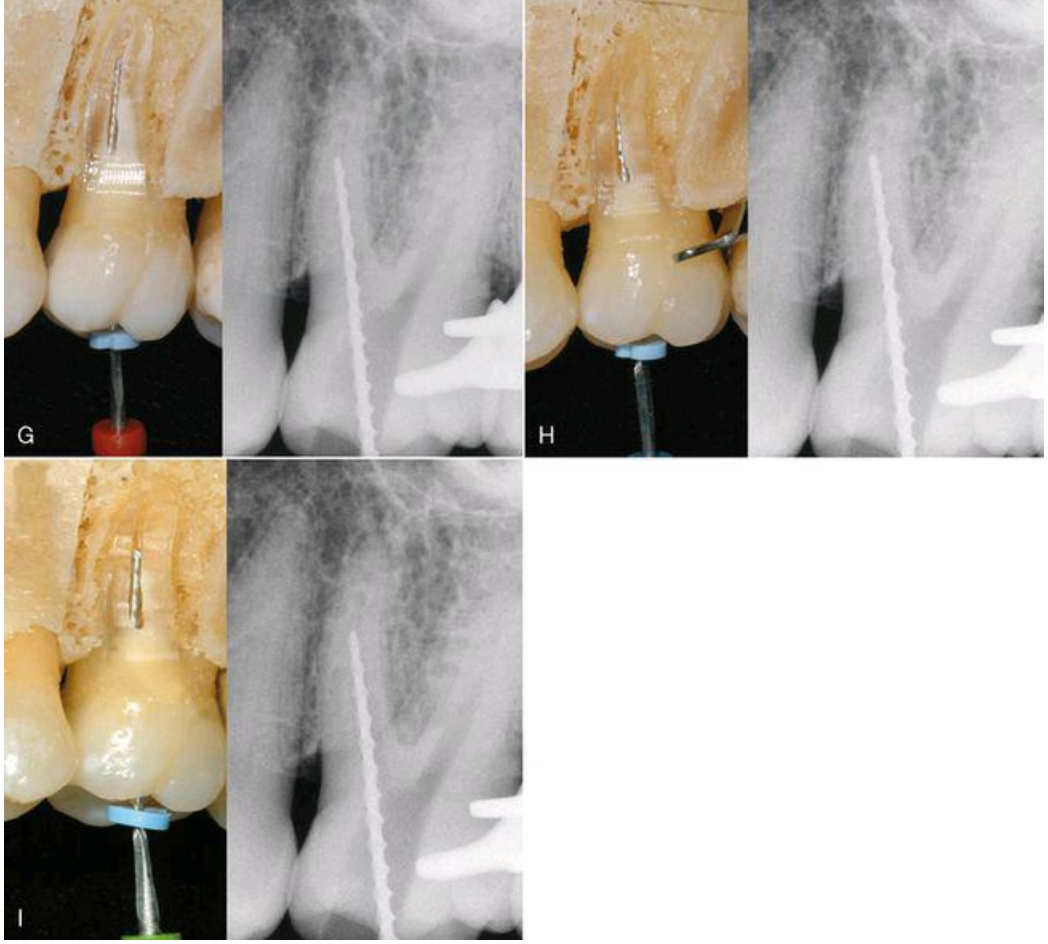

Figure 15-15 An example of step-back preparation in a moderately curved canal. **A,** The No. 25 master apical file at the corrected working length of 21 mm. **B,** The step-back process begins with the No. 30 file at 20.5 mm. **C,** No. 35 file at 20 mm. **D,** No. 40 file at 19.5 mm. **E,** No. 45 file at 19 mm. **F,** No. 50 file at 18.5 mm. **G,** No. 55 file at 18 mm. **H,** No. 60 file at 17.5 mm. **I,** No. 70 file at 17 mm.

Step-Down Technique

The step-down technique is advocated for cleaning and shaping procedures as it removes coronal interferences and provides coronal taper. Originally advocated for hand-file preparation,[126] the step-down technique has been incorporated into those techniques employing nickel-titanium files. With the pulp chamber filled with irrigant or lubricant, the canal is explored with a small instrument to assess patency and morphology (curvature). The working length can be established at this time. The coronal one third of the canal is then flared with Gates-Glidden drills or rotary files of greater taper (.06, .08, or .10). A large file (such as No. 70) is then placed in the canal, and a watch-winding motion is used until resistance is encountered.[126] The process is repeated with sequentially smaller files until the apical portion of the canal is reached. The working length can be determined if this was not accomplished initially. The apical portion of the canal can now be prepared by enlarging the canal at the corrected working length. Apical taper is accomplished using a step-back technique.

Passive Step-Back Technique

The passive step-back technique is a modification of the incremental step-back technique.[6,127] After the apical diameter of the canal has been determined, the next higher instrument is inserted until it *first* makes contact (binding point). It is then rotated one-half turn and removed (Figure 15-16). The process is repeated with larger and larger instruments being placed to their binding point. This entire instrument sequence is then repeated. With each sequence, the instruments drop deeper into the canal, creating a tapered preparation. This technique permits the canal morphology to dictate the preparation shape. The technique does not require arbitrary rigid incremental reductions and the forcing of files into canals that cannot accommodate them. Advantages to the technique include knowledge of canal morphology, removal of debris and minor canal obstructions, and a gradual passive, slight enlargement of the canal in an apical to coronal direction.

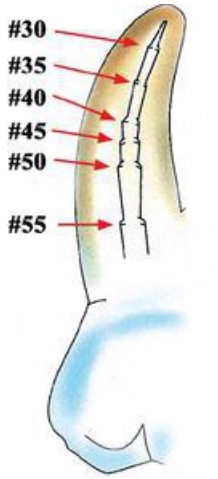

Figure 15-16 Passive step-back. Smaller to larger files are inserted to their initial point of binding and then rotated 180 to 360 degrees and withdrawn. This process creates slight taper and coronal space and permits larger instruments to reach the apical one third.

Anticurvature Filing

Anticurvature filing is advocated during coronal flaring procedures to preserve the furcal wall in the treatment of molars (Figure 15-17). Canals are often not centered in mesial roots of maxillary and mandibular molars; instead, they are located closer to the furcation. Stripping perforations can occur in these teeth during overly aggressive enlargement of the canal space. Stripping perforations occur primarily during use of the Gates-Glidden drills (Table 15-1). To prevent this procedural error, the Gates-Glidden drills should be confined to the canal space coronal to the root curvature and used in a step-back manner (Figures 15-18 and 15-19). The Gates-Glidden drills can also be used directionally in an anticurvature fashion to selectively remove dentin from the bulky wall (safety zone) toward the line angle, protecting the inner or furcal wall (danger zone) coronal to the curve (see Figure 15-17). Although this can be accomplished with the use of hand files, it appears that directional forces with Gates-Glidden drills are not beneficial.[128]

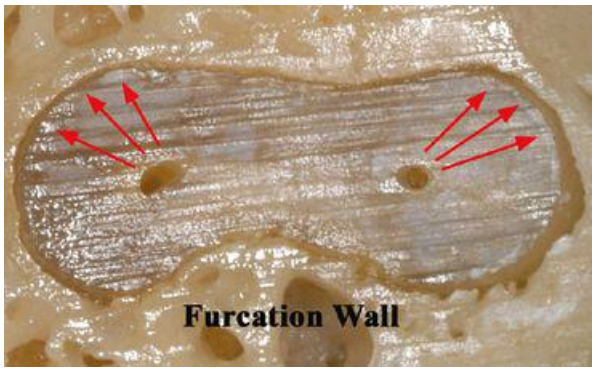

Figure 15-17 The anticurvature filing technique. Instruments are directed away from the furcal “danger zone” toward the line angles (safety zone) where the bulk of dentin is greater.

Table 15-1 The Diameter of Rotary Flaring Instruments

Size (No.)	Gates-Glidden Drills (mm)	Peeso Reamers (mm)
1	0.5	0.7
2	0.7	0.9
3	0.9	1.1
4	1.1	1.3
5	1.3	1.5
6	1.5	1.7

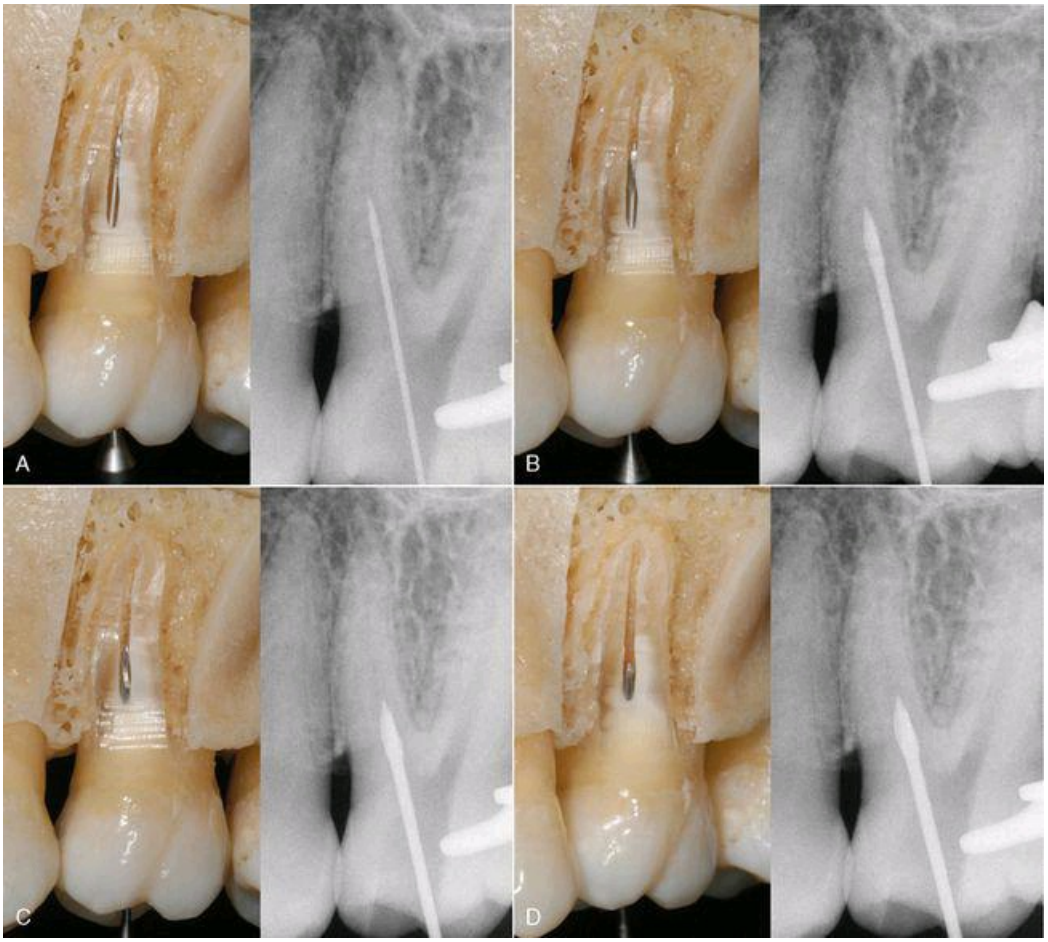

Figure 15-18 Straight-line access in a maxillary left first molar with Gates-Glidden drills used in a slow-speed handpiece using a step-back technique. **A,** The No. 1 Gates is used until resistance. **B,** This is followed by the No. 2, which should not go past the first curvature. **C,** The No. 3 Gates is used 3 to 4 mm into the canal. **D,** Followed by the No. 4 instrument.

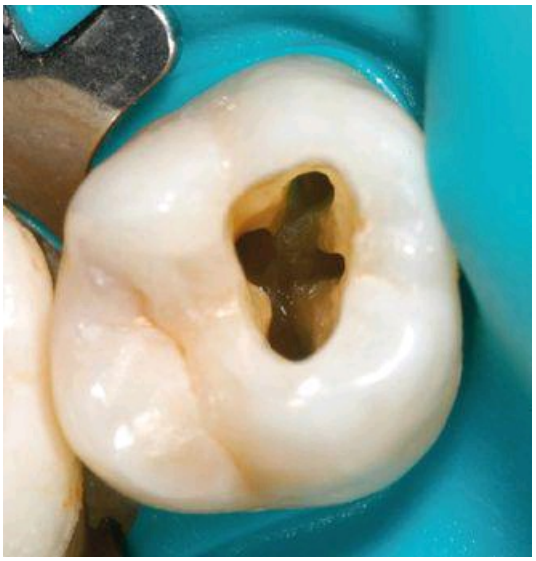

Figure 15-19 A maxillary first molar following straight-line access with the Gates-Glidden drills.

Balanced Force Technique

The balanced force technique recognizes the fact that instruments are guided by the canal walls when rotated.[129] Because the files will cut in both a clockwise and counterclockwise rotation, the balanced force concept of instrumentation consists of placing the file to length and then a clockwise rotation (less than 180 degrees) engages dentin. This is followed by a counterclockwise rotation (at least 120 degrees) with apical pressure to cut and enlarge the canal. The degree of apical pressure varies from light pressure with small instruments to heavy pressure with large instruments. The clockwise rotation pulls the instrument into the canal in an apical direction. The counterclockwise cutting rotation forces the file in a coronal direction while cutting circumferentially. After the cutting rotation, the file is repositioned and the process is repeated until the corrected working length is reached. At this point, a final clockwise rotation is employed to evacuate the debris.

Nickel-Titanium Rotary Preparation

Nickel-titanium rotary preparation utilizes a crown-down approach. The specific technique is based on the instrument system selected. One instrument sequence uses nickel-titanium files with a constant taper and variable ISO tip sizes (Figure 15-20). With this technique, a .06 taper is selected. Initially a size .06/45 file is used until resistance, followed by the .06/40, .06/35, .06/30, .06/25, and .06/20. In a second technique, nickel-titanium files with a constant tip diameter are used. The initial file is a .10/20 instrument, the second a .08/20, the third a .06/20, and the fourth a .04/20 (Figure 15-21). For larger canals, a sequence of files using ISO standardized tip sizes of 30 or 40 might be selected. Using the crown-down approach creates coronal flare and reduces the contact area of the file, so torsional forces are reduced.

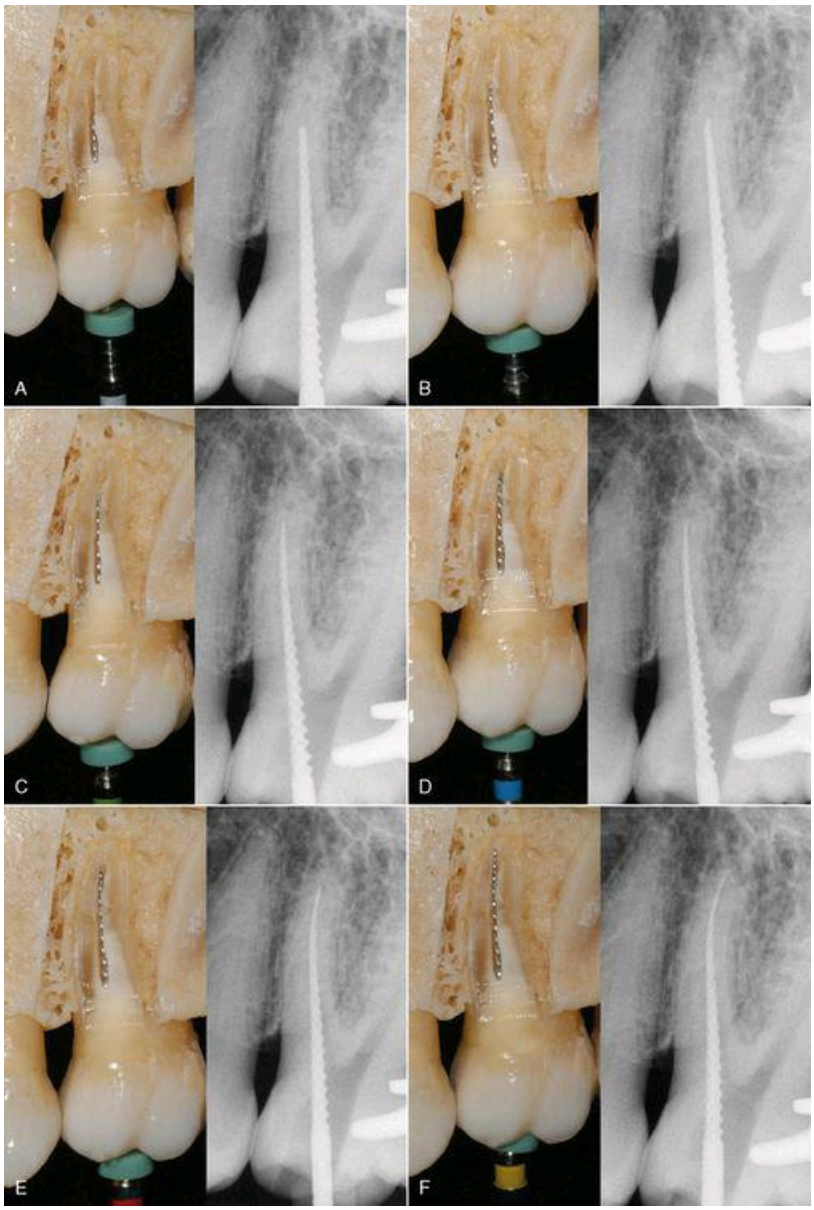

Figure 15-20 The mesiobuccal canal is prepared using nickel-titanium rotary files using a crown-down technique. In this sequence, each instrument exhibits the same .06 taper with varied ISO standardized tip diameters. Instruments were used to resistance. **A,** The process begins with a .06/45 file to resistance at 16.0 mm. **B,** Followed by a .06/40 instrument at 17.0 mm. **C,** The .06/35 file is used to 18.0 mm. **D,** The .06/30 at 19.0 mm. **E,** The .06/25 at 20.0 mm. **F,** The .06/20 file is to the corrected working length of 21.0 mm.

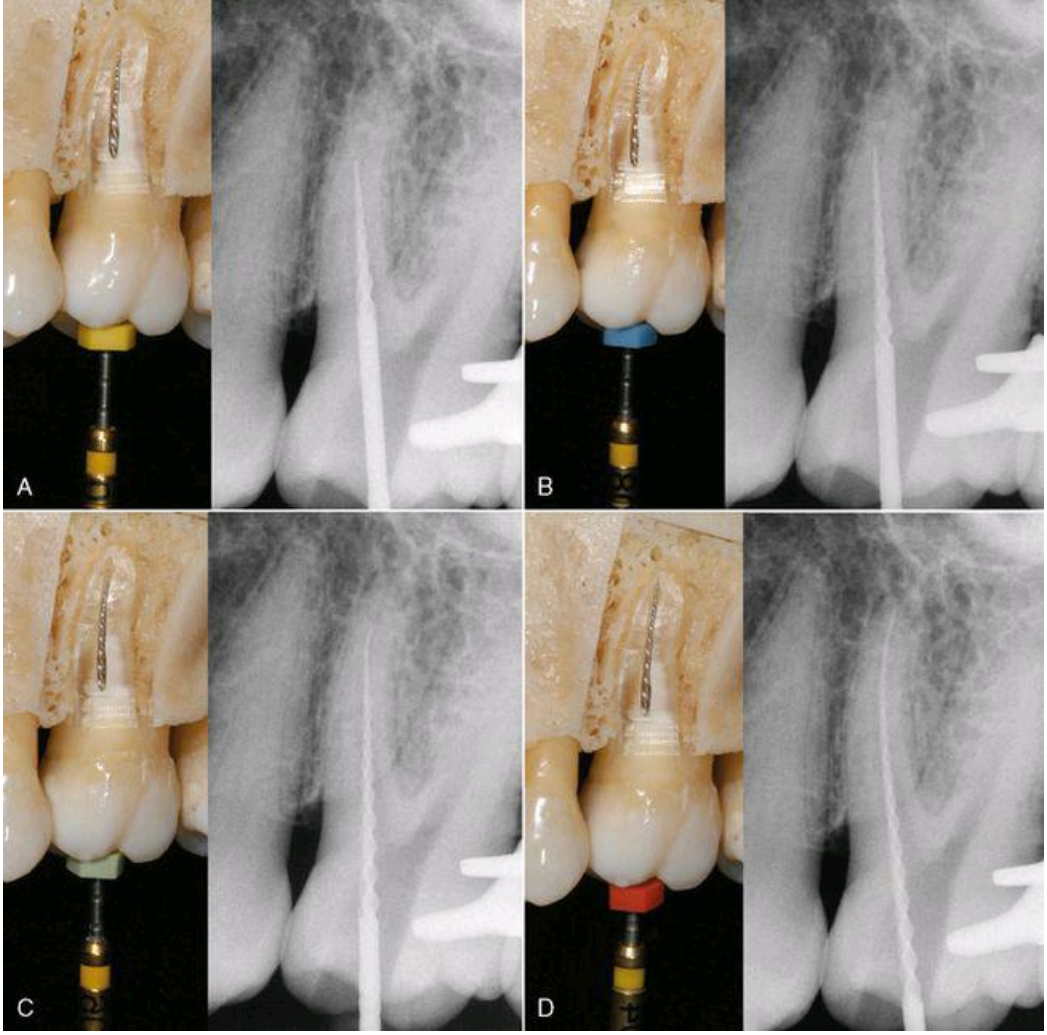

Figure 15-21 Nickel-titanium rotary files with a standardized ISO tip diameter and variable tapered files can be used in canal preparation. In this sequence, the instruments have a standardized tip diameter of .20 mm. **A,** Initially a 1.0/.20 file is used. **B,** This is followed by .08/.20. **C,** The third instrument is a .06/.20. **D,** The final instrument is a .04/.20 file to the corrected working length of 21 mm.

Final Apical Enlargement and Apical Clearing

Apical clearing enhances the preparation of the apical canal, improves débridement, and produces a more definite apical stop in preparation for obturation.[130] Apical clearing is generally performed when there is an apical stop and the MAF is less than a No. 40 file. If the apical configuration is open or a seat, apical clearing might make the opening larger and potentiate the possibility of extrusion of the obturation materials. Apical clearing consists of two distinct steps: final apical enlargement and dry reaming.

Final apical enlargement is performed after the canal has been cleaned and shaped. It involves enlargement of the apical preparation three to five sizes beyond the MAF (Figure 15-22). The degree of enlargement depends on the canal size and root curvature. In a small curved canal, enlargement may only be three sizes to decrease the potential for transportation. In a straight canal, it can be larger without producing a procedural error. Because the prepared canal exhibits taper, the small files at the corrected working length can be used to enlarge the canal without transportation. Final apical enlargement is performed with the irrigant and employs a reaming action at the corrected working length. The last file used becomes the final apical file (FAF). Because the file is only contacting the apical portion of the canal, the technique will result in a less irregular apical preparation. The canal is then irrigated. The smear layer is removed with a decalcifying agent, and the canal dried with paper points.

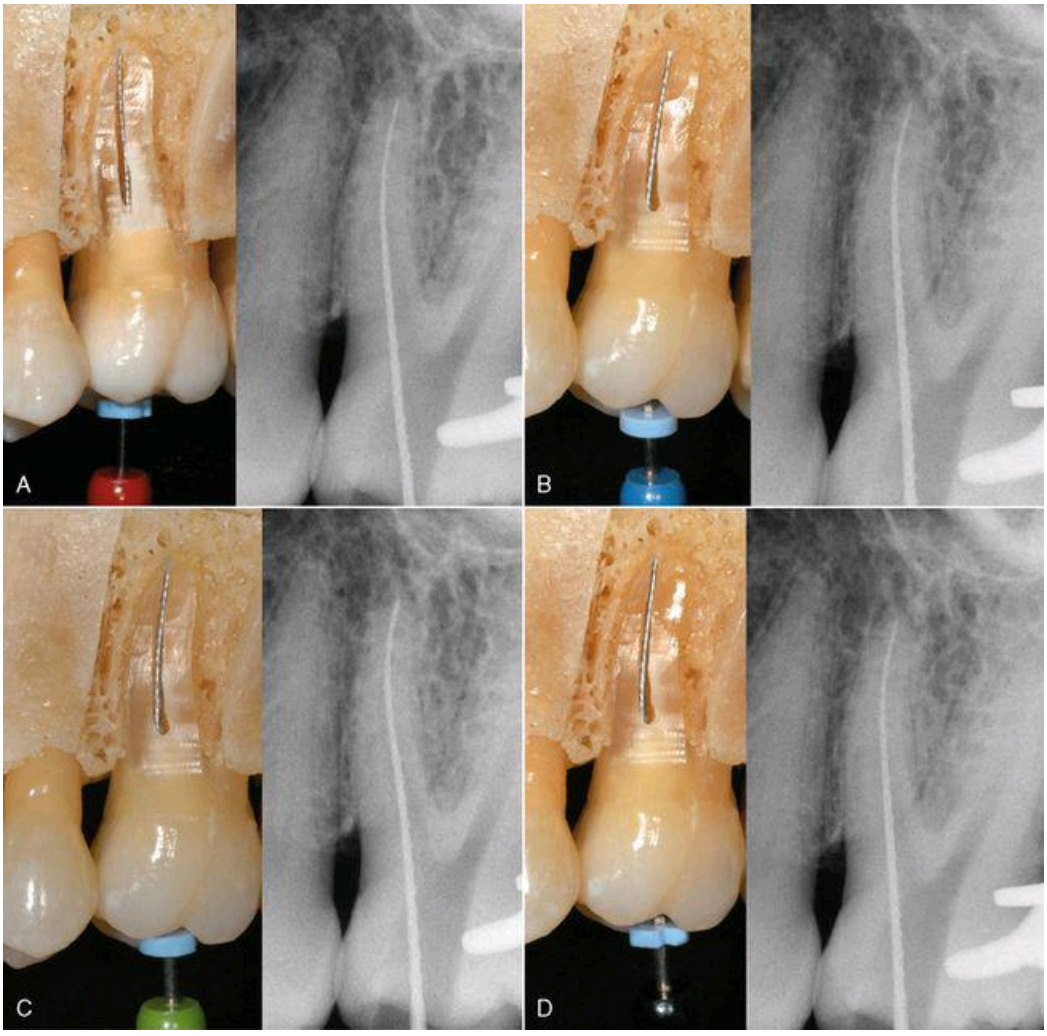

Figure 15-22 Final apical enlargement **A,** The master apical file No. 25 at the corrected working length of 21.0 mm. **B,** Enlargement with a No. 30 file to the corrected working length of 21.0 mm. **C,** Further enlargement with a No. 35 file. **D,** Final enlargement to a No. 40 file. The final instrument used becomes the final apical file.

After drying the canals, dry reaming with the FAF is performed. Dry reaming removes dentin chips or debris packed apically during drying. The FAF (or the MAF in cases where apical enlargement was not performed) is placed to the corrected working length and rotated clockwise in a reaming action.

Recapitulation

Recapitulation is important regardless of the technique selected (Figure 15-23) and is accomplished by taking a small file to the corrected working length to loosen accumulated debris and then flushing it with 1 to 2 ml of irrigant. Recapitulation is performed between each successive enlarging instrument regardless of the cleaning and shaping technique.

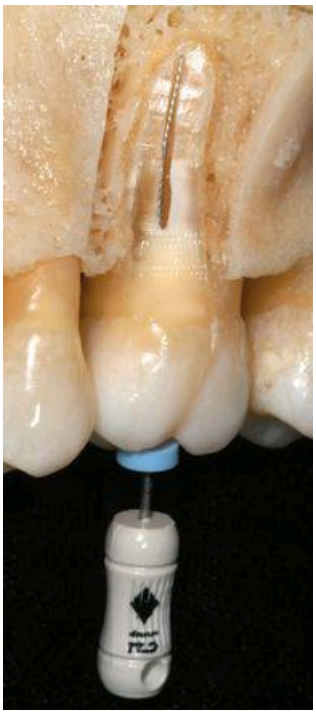

Figure 15-23 Recapitulation is accomplished between each instrument by reaming with the master apical file or a smaller instrument, minimizing packing of debris and loss of length.

Combination Technique

The combination technique combines coronal flaring, nickel-titanium rotary preparation, and the passive step-back technique (Box 15-2). After access, the canal is explored with a No. 10 or 15 file. If the canal is patent to the estimated working length, a working length radiograph can be obtained and the corrected working length established (see Figure 14-40). To ensure an accurate length determination, a No. 20 file or larger should be used (see Figures 14-40 and 14-41). If a No. 20 file will not go to the estimated working length, passive step-back instrumentation can be performed by inserting successively larger files to the point of binding and reaming. This removes coronal interferences and creates greater coronal taper, which permits larger files access to the apical portion of the root.

Box 15-2 The Combination Technique Steps

Canal negotiation

Working length determination

Straight-line access

Master apical file determination

Rotary preparation of the middle one-third of the root

Apical step-back preparation

Apical clearing

After establishing the working length, Gates-Glidden drills are used for straight-line access (see Figure 15-18). A No. 2 Gates is used first followed by the No. 3 and No. 4. In very narrow canals, a No. 1 Gates may be needed. It is important to remember the size of the Gates-Glidden drills. If the canal orifice cannot accommodate a No. 70 file, a passive step-back technique should be performed to provide adequate initial coronal space. To prevent stripping perforations, the Gates should not be placed apical to canal curvatures. Generally, Gates Nos. 2 to 4 provide adequate coronal enlargement and preserve root structure. Nickel-titanium rotary instruments with greater tapers can also be used for this step (.06, .08, and .10 tapers are common). The Gates-Glidden drills can be used in either a crown-down or step-back sequence. After use, the Gates-Glidden drill should be removed from the handpiece to prevent injury to the clinician, assistant, or patient (Figure 15-24).

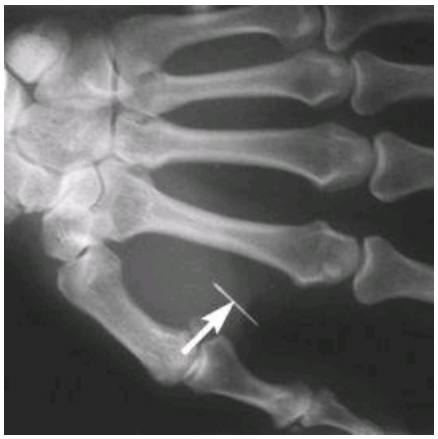

Figure 15-24 After use, the Gates-Glidden drills should be removed from the handpiece to prevent injury. This No. 3 drill *(arrow)* was accidentally driven into the palm of the dentist.

Master Apical File

Emphasis has traditionally been placed on determining the canal length with little consideration of the canal diameter in the apical portion of the root. Because every canal is unique in its morphology, the apical canal diameter must be assessed. The size of the apical portion of the canal is determined by placing successively larger instruments to the corrected working length until slight binding is encountered (Figure 15-25). Often, the next larger instrument will not go to the corrected working length. If it does go to length, a subjective estimation of the apical diameter must be made depending on the degree of binding. This file will be the MAF (initial file to bind). It is defined as the largest file to bind slightly at the corrected working length after straight-line access. This provides an estimate of the canal diameter before cleaning and shaping and is the point where the step-back preparation begins.

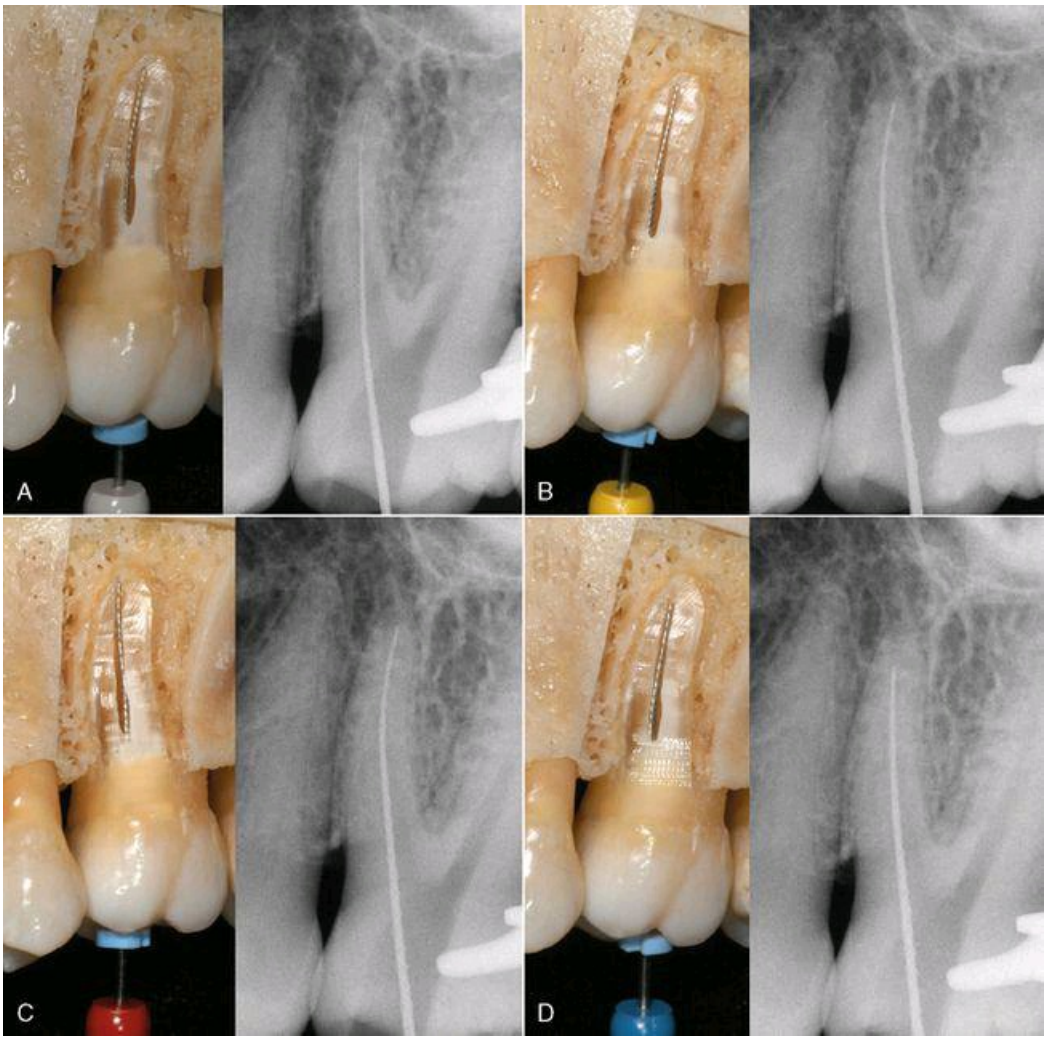

Figure 15-25 After straight-line access in this maxillary molar, the master apical file is determined by successively placing small to larger files to the corrected working length. **A,** No. 15 stainless steel file is placed to 21.0 mm without resistance. **B,** No. 20 is placed to 21.0 mm without resistance. **C,** The No. 25 file reaches 21 mm with slight binding. **D,** No. 30 file is then placed and does not go the corrected working length, indicating the initial canal size in the apical portion of the canal is No. 25.

Nickel-Titanium Rotary

Once the MAF is identified, the middle to apical portion of the canal is prepared using nickel-titanium rotary instruments (see Figures 15-20 and 15-21). Rotary files are used with a crown-down approach to within 3 mm of the corrected working length. Adequate coronal taper is established when the .06/45 goes to within 3 mm of the corrected working length. Using the crown-down approach creates coronal taper and reduces the contact area of the file so torsional forces are reduced.

Step-Back Apical Preparation

When the body of the canal has been shaped, the apical portion is prepared using standardized stainless steel or nickel-titanium hand files in a step-back process (see Figure 15-15). The first instrument selected for this portion of the shaping process is one size larger than the MAF (initial file to bind slightly). Larger files are successively shortened by standardized increments of .05 or 1.0 mm. Generally, sequentially stepping back to a No. 70 will produce adequate flare and blend the apical and middle thirds of the canal.

Apical Clearing

With a flared preparation from the orifice to the corrected working length, the apical portion of the canal is enlarged. With a tapered preparation, the canal can be enlarged with a reaming action because the canal walls will keep the instrument centered (see Figure 15-22).

General Considerations: A Review

The following principles and concepts should be applied regardless of the instruments or technique selected:

1. Initial canal exploration is always performed with smaller files to gauge canal size, shape, and configuration.

2. Files are always manipulated in a canal filled with an irrigant or lubricant present.

3. Copious irrigation is used between each instrument in the canal.

4. Coronal preflaring (passive step-back technique) with hand instruments will facilitate placing larger working length files (either hand or rotary) and will reduce procedural errors such as loss of working length and canal transportation.

5. Apical canal enlargement is gradual, using sequentially larger files from apical to coronal, regardless of flaring technique.

6. Debris is loosened and dentin is removed from all walls on the outstroke (circumferential filing) or with a rotating (reaming) action at or close to working length.

7. Instrument binding or dentin removal on insertion should be avoided. Files are teased to length using a watch-winding or "twiddling" action. This is a back-and-forth rotating motion of the files (approximately a quarter turn) between the thumb and forefinger, continually working the file apically. Careful file insertion (twiddling) followed by planing on the outstroke will help to avoid apical packing of debris and minimize extrusion of debris into the periradicular tissues.

8. Reaming is defined as the clockwise rotation of the file. Generally, the instruments are placed into the canal until binding is encountered. The instrument is then rotated clockwise 180 to 360 degrees to cut and plane the walls. When withdrawn, the instrument tip is pushed alternately against all walls. The pushing motion is analogous to the action of a paintbrush. Overall, this is a *turn and pull.*

9. Filing is defined as placing the file into the canal and withdrawing it along the path of insertion to scrape the wall. There is very little rotation on the outward cutting stroke. The scraping or rasping action removes the tissue and cuts superficial dentin from the canal wall.

10. Turn-pull filing involves placing the file into the canal until binding. The instrument is then rotated to engage the dentin and withdrawn with lateral pressure against the canal walls.

11. Circumferential filing is used for canals that exhibit cross-sectional shapes that are not round. The file is placed into the canal and withdrawn in a directional manner against the mesial, distal, buccal, and lingual walls.

12. Regardless of the technique, after each insertion the file is removed and the flutes are cleaned of debris; the file can then be reinserted into the canal to plane the next wall. Debris is removed from the file by wiping it with an alcohol-soaked gauze or a cotton roll.[131]

13. The canal is effectively cleaned *only* where the files actually contact and plane the walls. Inaccessible regions are poorly débrided.

14. Recapitulation is done to loosen debris by rotating the MAF or a smaller size at the corrected working length followed by irrigation to mechanically remove the material. During recapitulation, the canal walls are not planed and the canal should not be enlarged.

15. Small, long, curved, and round canals are the most difficult and tedious to enlarge. They require extra caution during preparation because they are the most prone to loss of length and transportation.

16. Overenlargement of curved canals by files attempting to straighten themselves will to lead to procedural errors (see Figure 15-9).

17. Overpreparation of canal walls toward the furcation may result in a stripping perforation in the danger zone where root dentin is thinner (see Figure 15-13).

18. It is neither desirable nor necessary to try to remove created steps or other slight irregularities created during canal preparation.

19. Instruments, irrigants, debris, and obturating materials should be contained within the canal. These are all known physical or chemical irritants that will induce periradicular inflammation and may delay or compromise healing.

20. Creation of an apical stop may be impossible if the apical foramen is already very large. An apical taper (seat) is attempted but with care. Overusing large files aggravates the problem by creating an even larger apical opening.

20. Forcing or locking (binding) files into dentin produces unwanted torsional force. This tends to untwist or wrap-up or will weaken and break the instrument.

CRITERIA FOR EVALUATING CLEANING AND SHAPING

After cleaning and shaping procedures, the canal should exhibit “glassy smooth” walls and there should be no evidence of dentin filings, debris, or irrigant in the canal. This is determined by pressing the MAF against each wall in an outward stroke.

Shaping is evaluated by assessing the canal taper and identifying the apical configuration. For obturation with lateral compaction, the finger spreader should go loosely to within 1 mm of the corrected working length. For warm vertical compaction, the plugger should reach to within 5 mm of the corrected working length (Figure 15-26).

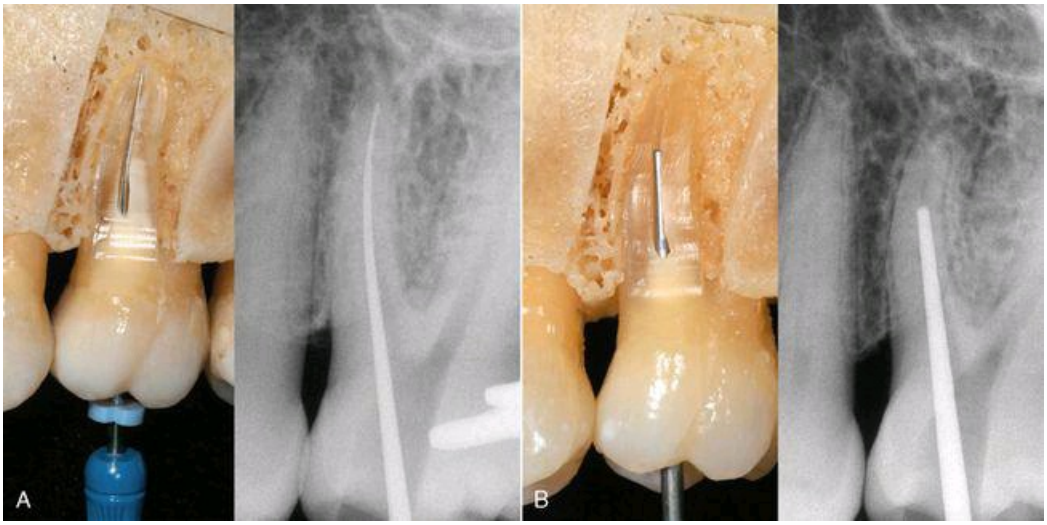

Figure 15-26 The coronal taper is assessed using the spreader or plugger depth of penetration. **A,** With lateral compaction, a finger spreader should fit loosely 1.0 mm from the corrected working length with space adjacent to the spreader. **B,** For warm vertical compaction, the plugger should go to within 5.0 mm of the corrected working length.

The apical configuration is identified as an apical stop, apical seat, or open. This is accomplished by placing the MAF to the corrected working. If the MAF goes past the corrected working length, the apical configuration is open. If the MAF stops at the corrected working length, a file one or two sizes smaller is placed to the corrected working length. If this file stops, the apical configuration is a stop. When the smaller file goes past the corrected working length, the apical configuration is a seat.

INTRACANAL MEDICAMENTS

Intracanal medicaments have a long history of use as interim appointment dressings. They have been employed for the following three purposes: (1) to reduce interappointment pain, (2) to decrease the bacterial count and prevent regrowth, and (3) to render the canal contents inert. Some common agents are listed in Box 15-3.

Box 15-3 Groupings of Commonly Used Intracanal Medicaments

PHENOLICS
- Eugenol
- Camphorated monoparachlorophenol (CMCP)
- Parachlorophenol (PCP)
- Camphorated parachlorophenol (CPC)
- Metacresylacetate (Cresatin)
- Cresol
- Creosote (beechwood)
- Thymol

ALDEHYDES
- Formocresol
- Glutaraldehyde

HALIDES
- Sodium hypochlorite (NaOCl)
- Iodine-potassium iodide

STEROIDS

CALCIUM HYDROXIDE

ANTIBIOTICS

COMBINATIONS

From Walton R: *Dent Clin North Am* 28:783, 1984.

Phenols and Aldehydes

The majority of the medicaments exhibit nonspecific action and can destroy host tissues, as well as microbes.[132-134] Historically, it was thought that these agents were effective, although their use was based on opinion and empiricism. The phenols and aldehydes are toxic, and the aldehydes are fixative agents.[135,136] When placed in the radicular space, they have access to the periradicular tissues and the systemic circulation.[137,138] Research has demonstrated that their clinical use is not justified.[139-143] Clinical studies assessing the ability of these agents to prevent or control interappointment pain indicate that they are not effective.[144-147]

Calcium Hydroxide

One intracanal agent that is effective in inhibiting microbial growth in canals is calcium hydroxide.[148] Calcium hydroxide has antimicrobial activity that is a result of the alkaline pH, and it may aid in dissolving necrotic tissue remnants and bacteria and their by-products.[149-151] Interappointment calcium hydroxide in the canal demonstrates no pain-reduction effects.[152] Calcium hydroxide has been recommended for use in teeth with necrotic pulp tissue and bacterial contamination. It probably has little benefit with vital pulps. Calcium hydroxide can be placed as a dry powder; as a powder mixed with a liquid such as local anesthetic solution, saline, water, or glycerin to form a thick paste; or as a proprietary paste supplied in a syringe (Figure 15-27). A lentulo spiral is effective and efficient for placement.[153-155] Spinning the paste into the canal by rotating a file counterclockwise and using an injection technique is not as effective. It is important to place the material deeply and densely for maximum effectiveness. To accomplish this, straight-line access with Gates-Glidden drills or nickel-titanium rotary files should be performed and the apical portion of the canal prepared to a No. 25 file or greater. Removal after placement is difficult,[156] and this is especially true in the apical portion of the root.

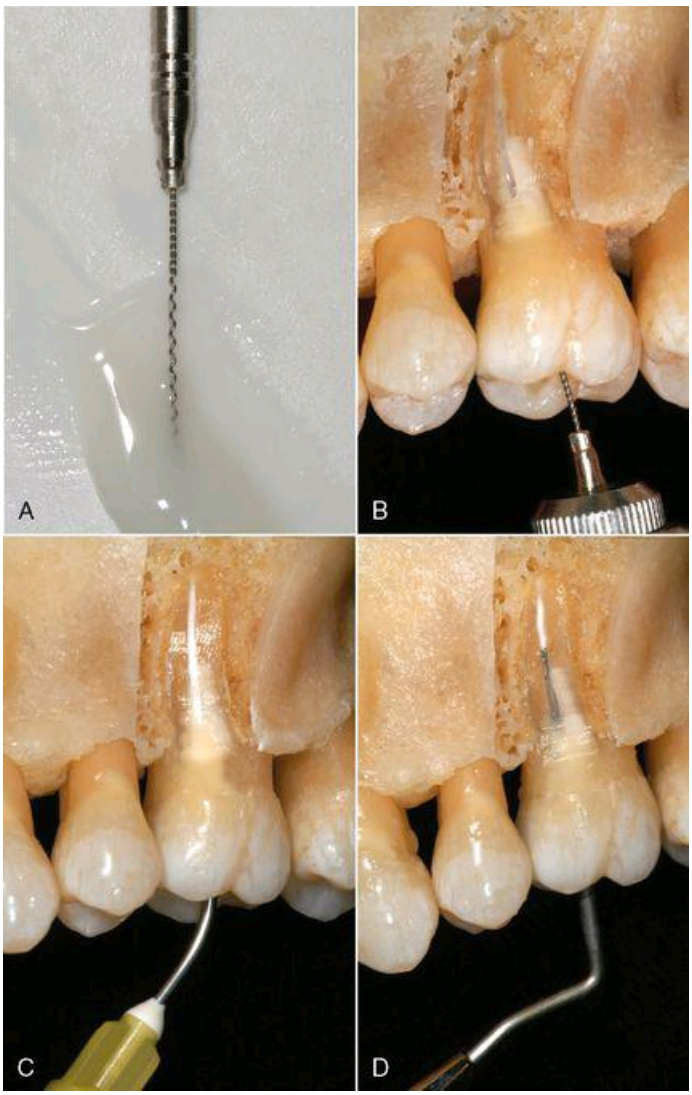

Figure 15-27 Calcium hydroxide placement. **A,** Calcium hydroxide mixed with glycerin to form a thick paste. **B,** Placement with a lentulo spiral. **C,** Injection of a proprietary paste. **D,** Compaction of calcium hydroxide powder with a plugger.

Corticosteroids

Corticosteroids are antiinflammatory agents that have been advocated for decreasing postoperative pain by suppressing inflammation. The use of corticosteroids as intracanal medicaments may decrease lower-level postoperative pain in certain situations[157]; however, evidence also suggests that they may be ineffective, particularly with greater pain levels.[147] Cases of irreversible pulpitis and cases in which the patient is experiencing acute apical periodontitis are examples where steroid use might be beneficial.[157-159]

Chlorhexidine

Chlorhexidine has recently been advocated as an intracanal medicament.[160,161] A 2% gel is recommended. It can be used alone in gel form or mixed with calcium hydroxide. When used with calcium hydroxide, the antimicrobial activity is greater than when calcium hydroxide is mixed with saline,[162] and periradicular healing is enhanced.[163] Its major disadvantages are that it does not affect the smear layer and it is a fixative.

TEMPORARY RESTORATIONS*

Root canal treatment may involve multiple visits. Also, unless it is limited to a routine access cavity, the final restoration is usually not completed in the same appointment as the root canal treatment. A temporary restoration, normally for 1 to 4 weeks, is then required. In special situations when definitive restoration must be deferred, the temporary must last for several months.

Objectives of Temporization

The temporary restoration must do the following:

1. Seal coronally, preventing ingress of oral fluids and bacteria and egress of intracanal medicaments.
2. Enhance isolation during treatment procedures.
3. Protect tooth structure until the final restoration is placed.
4. Allow ease of placement and removal.
5. Satisfy esthetics but always as a secondary consid-eration to providing a seal.

These objectives depend on the intended duration of use. Thus different materials are required, depending on time, occlusal load and wear, complexity of access, and loss of tooth structure.

Routine Access Cavities

Many access cavities involve only one surface and are surrounded by dentin walls or by porcelain or metal (if the restoration is retained). The temporary must last from several days to several weeks. Numerous types are available, including premixed cements that set on contact with moisture (Cavit); reinforced zinc oxide–eugenol cements, such as intermediate restorative material (IRM); glass ionomer cements (GIC); and specially formulated light-polymerized composite materials, such as temporary endodontic restorative material (TERM).[164] Ease of use and good sealing ability make Cavit an excellent routine material, but low strength and rapid occlusal wear limit its use to short-term sealing of simple access cavities. IRM and TERM provide improved wear resistance, although their sealing ability is probably marginally less than that of Cavit.[165,166] More durable restorative materials, especially glass ionomer cements, tend to provide the best seal. A double seal of GIC over Cavit will provide a durable and effective barrier to microbial leakage. It is not known whether experimental leakage differences based on bacterial leakage or dye penetration are significant clinically, especially if thermocycling and occlusal loading are not part of the testing procedure.[167] Clinically, 4 mm of Cavit provided an effective seal against bacterial penetration for 3 weeks.[168] Most critical are the thickness and placement of the material.

Techniques of Placement

The quality of the coronal seal depends on the thickness of the material, how it is compacted into the cavity, and the extent of contact with sound tooth structure or restoration. A minimum depth of 3 to 4 mm is required around the periphery, preferably 4 mm or more to allow for wear. In anterior teeth, the access is oblique to the tooth surface; care must be taken to ensure that the material is at least 3 mm thick in the cingulum area.

Cavit (or a similar material) is placed as follows. Chamber and cavity walls should be dry. Cavit can be placed directly over the obturated canal orifices, or more commonly, a thin layer of cotton is placed over the canal orifices to prevent canal blockage[169] (Figure 15-28). Care must be taken not to incorporate cotton fibers into the restorative material, which can promote rapid leakage.[170] Cavit is packed into the access opening with a plastic instrument in increments from the bottom up and pressed against the cavity walls and into undercuts (Figure 15-29). Excess is removed, and the surface smoothed with moist cotton. The patient should avoid chewing on the tooth for at least an hour.

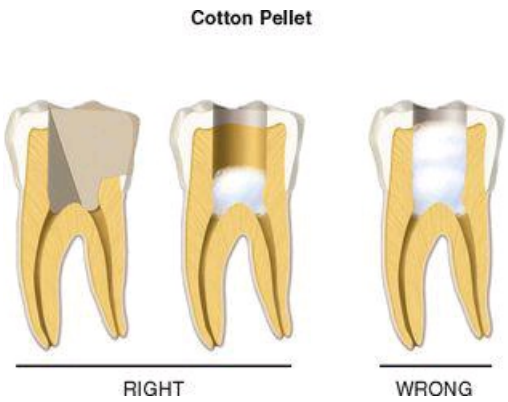

Figure 15-28 Techniques for temporization. The left two are correct techniques. Either minimal space is occupied by cotton or no cotton pellet is used, particularly if the proximal is to be restored. The right diagram is incorrect. Most of the chamber is packed with cotton, which leaves inadequate space and strength for the material (3 to 4 mm are required), and cotton fibers may promote bacterial leakage.

(Courtesy Drs. L. Wilcox and H. Messer.)

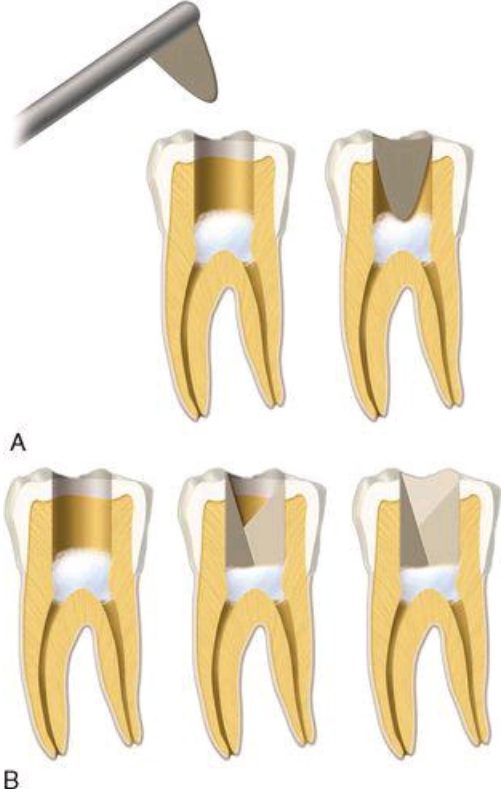

Figure 15-29 Techniques for placing temporary material. **A,** Single large "blob" placed in the access opening will not seal the walls. **B,** The incremental technique, which adds successive layers pressing each against the chamber walls, is correct.

(Courtesy Drs. L. Wilcox and H. Messer.)

Subsequent removal using a high-speed bur requires care to avoid damage to the access opening. Alternatively, an ultrasonic tip can be used.

Extensive Coronal Breakdown

Teeth without marginal ridges or with undermined cusps require a stronger filling material (high-strength GIC), taking care to ensure an adequate thickness and good marginal adaptation proximally. The temporary filling material should extend well into the pulp chamber deep to the proximal margin to ensure a marginal seal. Reducing the height of undermined cusps well out of occlusion reduces the risk of fracture. For severely broken-down teeth, a cusp-onlay amalgam or a well-fitting orthodontic band cemented onto the tooth (restored with glass ionomer cement) provides a durable temporary restoration and strengthens the tooth against fracture.[171] At the next appointment, access is prepared through the restoration.

Provisional Post Crowns

The use of a provisional crown with an incorporated resin post may be required, particularly when a cast post and core is being fabricated for a visible tooth with little remaining coronal tooth structure. However, the use of such a provisional crown retained with a post (preformed aluminum post, safety pin wire, paper clip, or a sectioned large endodontic file) has inherent problems. Using the canal space for a provisional post precludes use of an intracanal medicament, and the coronal seal depends entirely on the cement. The coronal seal is generally inadequate, with a loosely fitting and mobile provisional post and crown.[172] However, in spite of these potential difficulties, such provisional restorations may be required while cast posts and cores are being fabricated. Because of the potential problems, it is prudent to cement the definitive post as soon as possible.

When such a provisional crown-post combination is being used, the post should fit the canal snugly (not binding) and extend apically 4 to 5 mm short of working length and coronally to within 2 to 3 mm of the incisal edge. A polycarbonate shell is trimmed to a good fit; autopolymerizing material then is added to the inside of the shell to mold to the root face and attach to the post. Following contouring and occlusal adjustment, provisional luting cement (Temp Bond or similar cement) is placed on the coronal 3 to 4 mm of the post and root face, and the unit is cemented into place. A provisional removable partial overdenture is a useful alternative; access remains excellent, and there is little chance of disturbing the coronal seal between appointments.

Long-Term Temporary Restorations

Few indications exist to justify delaying the final restoration, and endodontic procedures (other than trauma management) rarely require prolonged treatment. If a temporary restoration has to last more than a few weeks, then a durable material, such as amalgam, GIC, or acid-etch composite, should be used. The pulp chamber is filled with Cavit to provide a good coronal seal and covered with a sufficient thickness of the restorative material to ensure strength and wear resistance. Subsequent access to the canal space is readily achieved without damage to remaining tooth structure because the layer of Cavit can be easily removed.

Chapter Review Questions available in Appendix B or on the DVD

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* Courtesy of Dr. Harold Messer.

Preparation for Restoration

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe the main factors involved in the survival of root-filled teeth.
2. Summarize factors contributing to loss of tooth strength and describe the structural importance of remaining tooth tissue.
3. Explain the importance of coronal seal and how it is achieved.
4. Describe requirements of an adequate restoration.
5. Outline postoperative risks to the unrestored tooth.
6. Discuss the rationale for immediate restoration.
7. Identify restorative options before commencing root canal treatment.
8. Discuss advantages and disadvantages of direct and indirect restorations.
9. Outline indications for post placement in anterior and posterior teeth.
10. Describe common post systems and advantages and disadvantages of each.
11. Describe core materials and their placement.
12. Describe techniques for restoring an access opening through an existing restoration.

Restorability should be confirmed before root canal treatment is begun. Options for restoration are also considered then, although the final choice is often made as treatment progresses. The options for restoration need careful consideration because more endodontically treated teeth are lost as a result of restorative factors than as a result of failure of the root canal

treatment itself.[1,2] In most cases, restoration is straightforward, but the choice must be based on sound principles if the tooth is to be retained long term as a functional unit. This chapter considers principles of restoration rather than detailed techniques, which are beyond the scope of this textbook.

LONGEVITY OF ROOT-FILLED TEETH AND CAUSES OF TOOTH LOSS

Root-filled teeth are expected to function effectively for a prolonged period. Numerous studies investigating the survival of root-filled teeth have documented that at most 1% to 2% are lost per year.[1,3-5] A very large study of 1.4 million cases reported that only 2.6% of teeth were lost after 6 years.[5] A smaller but more detailed study found that root-filled teeth were 3 times as likely to be extracted as their vital, contralateral counterparts in the same patient, but 89% of root-filled teeth were retained after 8 years.[6]

Clearly, many factors contribute to loss of endodontically treated teeth. Endodontic problems (development or persistence of a periapical lesion, with accompanying signs and symptoms) are generally amenable to further management rather than extraction. Most teeth are lost for periodontal or restorative reasons (including caries).[1,2] Many restorative variables influence the success rate achieved in clinical practice, and much lower survival rates can be expected with inadequate restoration.[7-9] When root canal–treated teeth are restored with crowns as opposed to no crown, the tooth survival rate is enhanced.[10,11] A well-placed amalgam with adequate cuspal protection is also a durable restoration. Most posterior teeth requiring extraction are lost because of unrestorable crown fracture, resulting from inadequate restoration that does not protect weakened cusps (Figure 16-1).[4,7,8,11] A literature review of complications associated with posts and cores found an average complication rate of 10% in clinical studies, with post loosening and root fracture being the most common complications.[12]

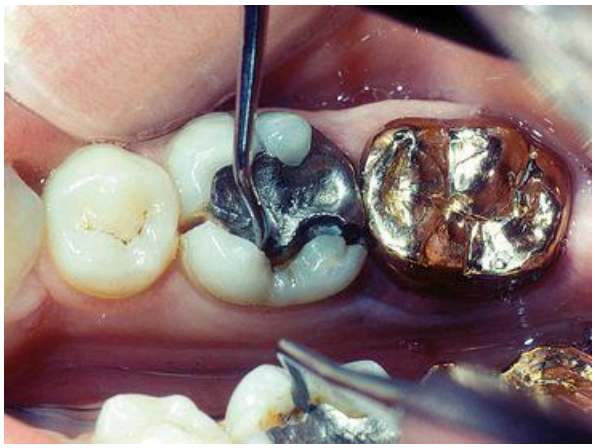

Figure 16-1 Crown root fracture (split tooth) of a root canal–treated tooth restored with amalgam but lacking protection of undermined, weakened cusps.

(Courtesy Dr. H. Colman.)

STRUCTURAL AND BIOMECHANICAL CONSIDERATIONS

Teeth function in a challenging environment, with heavy occlusal forces and repeated loading at a frequency of more than one million cycles per year, over many years of clinical life. Caries, restorative procedures, and occlusal wear add to the risk of serious damage to teeth during normal function, and root-filled teeth are at greater risk than intact teeth.

Restorative failures commonly include cusp fractures or some form of coronal fracture (split tooth) (see Figure 16-1).[2] It is important to understand the basis for this fracture susceptibility when designing restorations. Access preparation superimposed on an extensively carious or restored tooth leads to further loss of tooth structure. Unsupported cusps (particularly those without an adjacent intact marginal ridge) associated with a large access opening are more prone to fracture. Excessive preparation of the canal space for a post further weakens the root and introduces significant stress areas.[13]

Structural Changes in Dentin

The clinical perception persists that root canal–treated teeth become brittle, supposedly losing resilience as the moisture content of dentin declines after pulp loss. This perception has only limited experimental support. Few studies have compared physical properties of endodontically treated versus untreated human teeth with vital pulps. The moisture content of endodontically treated teeth was not reduced, even after 10 years.[14] Also, comparing vital and root-filled teeth revealed only very minor differences in strength, toughness, and hardness of dentin.[15,16] Thus susceptibility to fracture cannot be attributed only to structural changes in dentin after loss of pulp vitality. The loss of dentinal fluid, which may play a role in stress distribution and stress relief, could contribute to changes in the response of root-filled teeth to occlusal stresses.[17]

Loss of Tooth Structure

Teeth are measurably weakened even by occlusal cavity preparation; greater loss of tooth structure further compromises strength. Loss of one or both marginal ridges is a major contributor to reduced cuspal stiffness (strength), which predisposes to fracture (Figure 16-2).[18,19] Access has only a minor effect on decreasing cuspal strength when the access cavity is surrounded by solid walls of dentin. In a tooth already seriously compromised by caries, trauma, or large restorations, the access cavity is more significant, particularly if the adjacent marginal ridge has been lost.[20] Excessive coronal flaring results in greater susceptibility to cusp fractures.[21]

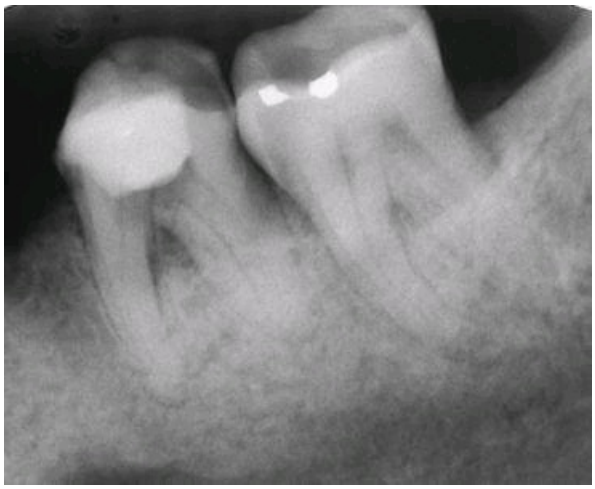

Figure 16-2 Teeth requiring root canal treatment have commonly been structurally compromised by caries and restorative procedures.

Biomechanical Factors

The occlusal loads to which teeth are subjected during normal function generate large stresses in teeth, which are capable of causing cusp fracture and even vertical root fracture in intact vital teeth.[22] Cuspal flexure (movement under loading) will weaken premolars and molars over time.[19,20] As cavity preparations become larger and deeper, unsupported cusps become weaker and show more deflection under occlusal loads. Greater cuspal flexure leads to cyclic opening of margins between the tooth and the restorative material. Fatigue is also a factor; cusps become progressively weaker with repetitive flexing. Thus the restoration must be designed to minimize cuspal flexure to protect against fracture and marginal leakage.

Requirements for an Adequate Restoration

Based on these concepts, the definitive restoration must (1) protect remaining tooth structure, (2) minimize cuspal flexure, (3) provide a coronal seal, and (4) satisfy function and esthetics. Care must be taken to ensure that esthetic demands do not lead to the weakening of teeth by excessive removal of remaining tooth structure.

CORONAL SEAL

Eliminating bacteria from the canal space and preventing reentry are crucial to healing. The coronal seal is an essential component of bacterial control, both during and after treatment. The restoration (both temporary and definitive) provides the coronal seal.

Coronal leakage is a major cause of failure.[23,24] Even a well-obturated canal with appropriate use of sealer cement does not provide an enduring barrier to bacterial penetration.[25,26] Exposure of obturating materials to oral fluids through a lost restoration, marginal discrepancy, or recurrent caries leads eventually to sealer disintegration and bacterial contamination of the canal system, with subsequent apical pathosis.

The restoration must provide the coronal seal either as a separate step (e.g., placing a barrier over canal orifices)[27,28] or more commonly as an integral part of the restoration by virtue of its marginal sealing ability. Posts (particularly prefabricated posts) and cores do not create a seal until the crown is placed.[13,29] Lack of an intact sealing restoration is an important factor in the persistent or developing periapical lesion. Fortunately, this is usually correctable by endodontic and restorative retreatment. Another concern is inadequate temporary seal, both during treatment and after treatment is complete but before definitive restoration. There is not sufficient information to know how much exposure time to oral fluids mandates retreatment because the time required to develop substantive leakage has varied between 3 and 90 days in experimental studies.[25,26,30] In a clinical study in which root canal treatment was of high quality (maximal obturation), periapical lesions did not develop even several years after the coronal seal was lost.[31] Because the quality of obturation is often uncertain, a commonly accepted guideline is 2 to 3 months.[23,30]

RESTORATION TIMING

The unrestored, temporized, root canal–treated tooth without definitive restoration is a candidate for problems. Unless there are specific reasons for delay, definitive restoration is completed as soon as practical.[23,30,33] The tooth is at its weakest after access and remains so until it is appropriately restored. The temporary will not provide complete protection against occlusal forces even when the tooth is out of occlusion or is splinted with an orthodontic band. Fracture during or soon after treatment is all too common (Figure 16-3). Most temporary restorative materials have low wear and fracture resistance; substantial occlusal wear or material fracture may occur within weeks. It is unnecessary to wait for radiographic evidence of healing before the final restoration is placed. Even in many cases with a guarded prognosis, prompt definitive restoration may improve the prognosis because of the better protection.

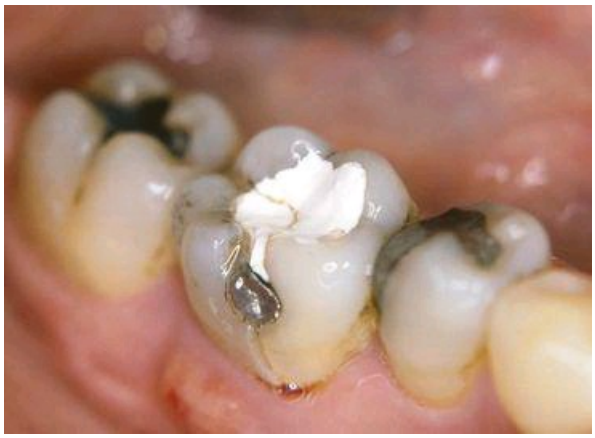

Figure 16-3 Unrestorable fracture during root canal treatment. The lack of cuspal protection combined with deep anatomic grooves led to fracture within days of endodontic access.

Almost the only reason to delay the definitive restoration is a questionable prognosis where failure would lead to extraction. The prognosis for procedural problems, such as perforation, has been so greatly improved with newer repair materials, such as mineral trioxide aggregate (MTA), that few endodontic indications justify a delay. With a guarded prognosis, the rationale for postponing definitive restoration is based on the nature of further management if failure occurs. Decision making for managing failure is covered in Chapter 19. Retreatment is usually possible through the restoration; if correction requires surgery, there is no reason to delay restoration.

If definitive restoration is delayed, the temporary must last as long as necessary (up to a year). It must protect, seal, and meet functional and esthetic demands. A good long-term posterior provisional restoration is an amalcore that encompasses (onlays) weakened cusps, thus providing functional and sealing protection. The definitive crown preparation can be completed later without removing the core (Figure 16-4). Comparable anterior restorations are more challenging because of esthetic factors and difficulties with coronal seal. A provisional post may be susceptible to flexion and loosening, thereby compromising an adequate seal.[29] It is preferable to place a definitive post and core immediately (which provides a better coronal seal) when a provisional crown is indicated.[34]

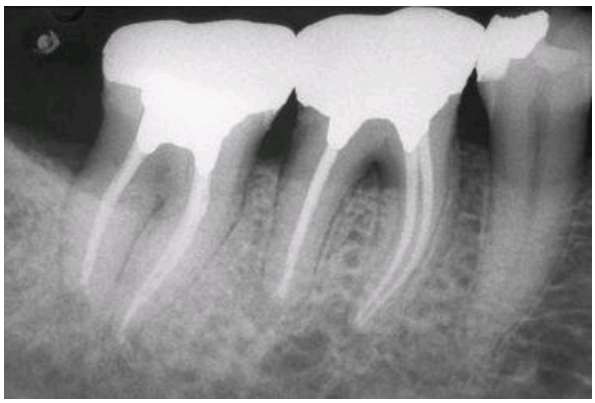

Figure 16-4 Chamber and canal orifices retain an amalgam core, taking advantage of natural undercuts. The teeth can be prepared for crowns without removing the amalcore, or the amalgams may be definitive restorations if the cusps are adequately protected.

(Courtesy Dr. P. Parashos.)

RESTORATION DESIGN

Principles and Concepts

Three practical principles for function and durability are the following:

1. *Conservation of tooth structure.* Most teeth requiring treatment are already compromised structurally; further dentin removal should be minimized.[35] On the other hand, cuspal reduction and crown placement may be necessary to encompass remaining tooth structure, thereby preventing fracture and preserving the tooth. Routinely decoronating and rebuilding an endodontically treated tooth is neither desirable nor in keeping with contemporary science.

2. *Retention.* The coronal restoration is retained by the core and remaining dentin. If the core requires retention, then the pulp chamber and root canal system can be used, with provision of a post. A post weakens and may perforate the root and should be placed only when needed to retain the core.[13,35]

3. *Protection of remaining tooth structure.* In posterior teeth, this applies to protecting weakened cusps by minimizing flexure and fracture. The restoration is designed to transmit functional loads through the tooth to the suspensory apparatus.

Planning the Definitive Restoration

The choice of definitive restoration can be decided only after caries and any existing restoration are removed and the access is prepared. Visualizing the restorative preparation in advance ensures that structural requirements of the core will be preserved. Increasingly, esthetic demands are leading to the use of tooth-colored materials for both direct and indirect restorations. These materials may have less favorable functional properties than metallic restorations and require greater removal of tooth structure. Care must be taken to ensure that structural strength is not compromised.

For anterior teeth, the choice of final restoration is somewhat limited. Wherever possible, restoration of the access (e.g., acid-etch composite) is used; this is sufficient for teeth that are otherwise largely intact. For more extensively damaged teeth (trauma, large proximal restorations), complete coronal coverage supported by a post/core is indicated. The choices for premolars and molars are more varied.

Direct Restorations

Restorations inserted directly into the prepared cavity (amalgam or composite) may be conservative, but a requirement is that the restoration protects against coronal fracture. Indications include the following:

1. Minimal tooth structure has been lost before and during root canal treatment. A conventional access cavity in a tooth with intact marginal ridges is restored without further preparation.

2. Prognosis is uncertain, requiring a durable semipermanent restoration.

3. Ease of placement and cost.

Many posterior teeth may be restored with amalgam if it is esthetically acceptable and if unsupported cusps are adequately protected.[36] A conventional class II amalgam will not achieve this and ordinarily should not be used.[37] At a minimum, cusps adjacent to a lost marginal ridge should be onlayed with sufficient thickness of amalgam (at least 3 to 4 mm) to resist occlusal forces. The amalgam should extend into the pulp chamber and canal orifices to aid retention. The amalgam may subsequently serve as a core for indirect cast restoration if indicated (see Figure 16-4). Bonded amalgams have also been used, but their clinical performance in root-filled teeth has not been documented, and bond failure is likely to be catastrophic for otherwise unsupported cusps.

There can be substantial esthetic deficits when amalgam is used in visible teeth, and the need for a tooth-colored restoration has led to expanded use of bonded composite resin restorations. The use of bonding continues to escalate as materials and techniques improve, and good results have been reported in a long-term prospective clinical study of resin composite restorations (Figure 16-5).[38] Recurrent proximal caries remains a concern.

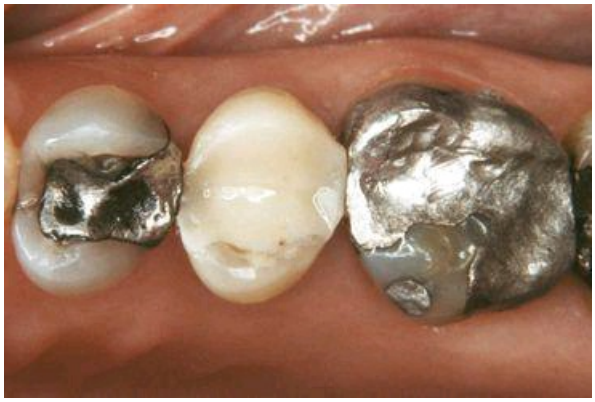

Figure 16-5 Resin composite restorations are more esthetic than amalgam and are increasingly used when cost is a concern. The procedure is technique sensitive and recurrent proximal caries needs to be monitored carefully.

Indirect Restorations

Cast restorations (onlays and three-quarter and full crowns) provide the greatest occlusal protection and are optimal if there is extensive loss of tooth structure. The attractiveness of onlays is that the cavity design usually requires little additional tooth tissue removal other than for the cusp onlays (Figure 16-6). The access cavity should be sealed with an amalcore or glass ionomer, which forms a base for the casting. The strength of gold allows conservative tooth reduction and a reverse bevel for effective cusp reinforcement.

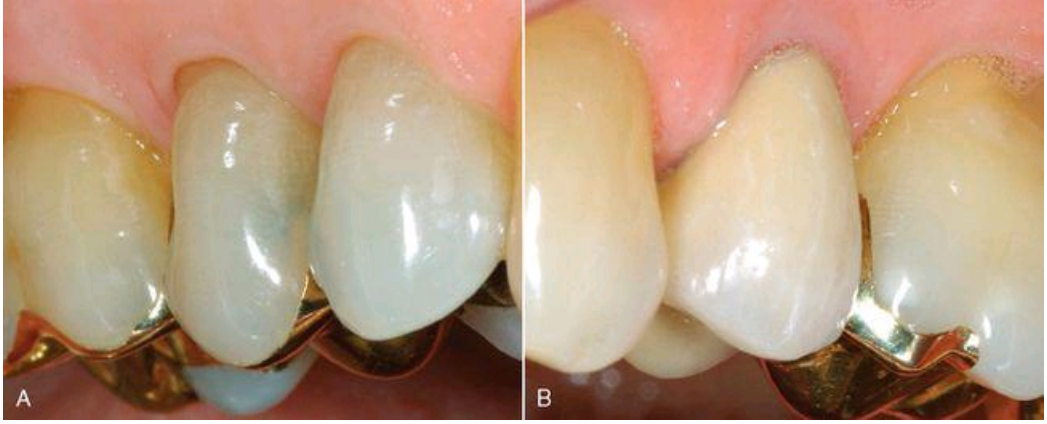

Figure 16-6 A, Cast gold onlay incorporates a reverse bevel for additional protection against cuspal flexure, but may not satisfy esthetic requirements for many patients. **B,** A full crown is esthetically more acceptable but involves greater tooth reduction than an onlay.

A full crown is a reliable, strong restoration that protects against crown-root fracture.[7,35,39] Root canal treatment coupled with tooth reduction may result in the removal of substantial coronal tooth structure, necessitating placement of a core and occasionally a post to retain the core. To plan the core shape, there must be complete exposure of the finish line. Gingival retraction cord and electrosurgery are useful for exposure and also prevent undersized cores being made because of incomplete viewing of the finish line. Complete crowns should be used only when there is insufficient coronal tooth structure present for a more conservative restoration or if functional or parafunctional stresses require the splinting effect of complete coronal coverage.

PREPARATION OF CANAL SPACE AND TOOTH

Post Selection

A post retains the core; the need for a post is dictated by the amount of remaining coronal tooth structure. A major disadvantage is that posts do not reinforce the tooth but further weaken it by additional removal of dentin and by creating stresses that predispose to root fracture.[9,13,35,40,41] A post system should be selected that fits the requirements of the tooth and restoration. The tooth and restoration should not be prepared and adapted to the post system; rather, the post system and preparation design should be selected as appropriate to the situation.

The debate on how the post should interact with the tooth under load is whether the post material should be similar in stiffness to root dentin (carbon fiber, quartz fiber), somewhat stiffer (titanium and gold), or much stiffer (stainless steel and cobalt-chromium alloys).[42] Stiffer posts may lead to tooth fracture, whereas the more flexible posts deform with the tooth and tend to fail without fracturing the tooth.[43] Bonding between the post and the tooth may allow the restored tooth to deform more with loading, compared with the conventional approach of the indirect restoration being much more rigid than the tooth. Long-term clinical trials will determine how posts should interact with teeth and what degree of stiffness functions best. Carbon fiber and quartz fiber posts appear to be advantageous in relation to root fracture potential. Fewer fractures have been recorded in laboratory studies compared to metal posts.[44,45] However, these types of posts have been less retentive than metal posts in laboratory tests,[46] indicating the need for optimal retentive post length. Clinical results provide favorable data regarding longevity, post retention, and low frequency of fracture (Figure 16-7).[47,48]

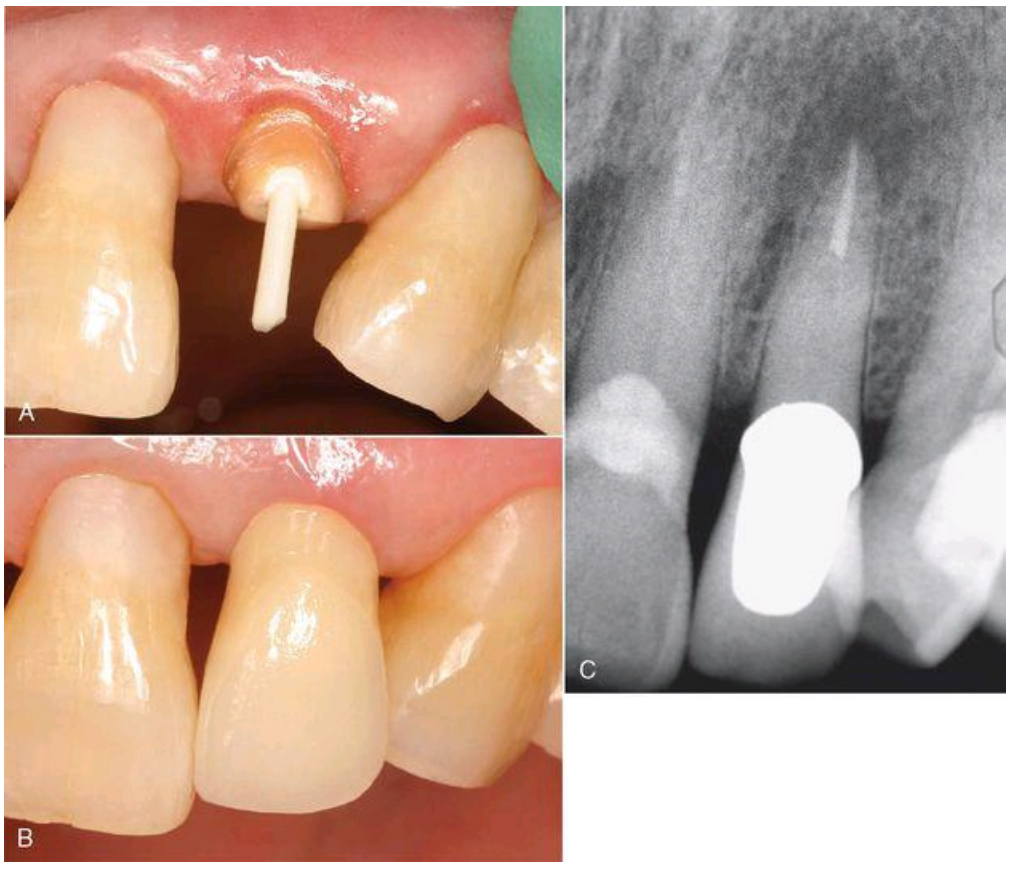

Figure 16-7 A and **B,** Carbon fiber posts are increasingly used for anterior teeth, with excellent esthetic results. **C,** The post is radiolucent.

It has been determined in the laboratory that threaded posts create stress in the root of a tooth.[41,49] Additionally, a meta-analysis of clinical studies indicates the survival rate for threaded posts is 81%, whereas it is 91% for cemented posts.[50]

Post Space Preparation

When a post is required for core retention, the minimum post space (length, diameter, and taper) should be prepared consistent with that need. Preparation consists of removing gutta-percha to the required length, followed by enlargement and shaping to receive the post (Figure 16-8). Caution is required; removing excess gutta-percha results in a defective apical seal.[51,52] Also, excessive dentin removal seriously weakens the root, which may predispose to root fracture. A perforation may occur if the cutting instrument deviates from the canal or if the preparation is too large or extends beyond the straight portion of the canal. Radiographs may be deceptive as a guide to root curvature and diameter by disguising root concavities and faciolingual curves.[53] As a general rule, post diameter should be minimal, particularly apically, and not more than one third of the root diameter.[54] Tapered post preparation prevents the risk of creating a step at the apical post space, which may predispose to wedging and root fracture.

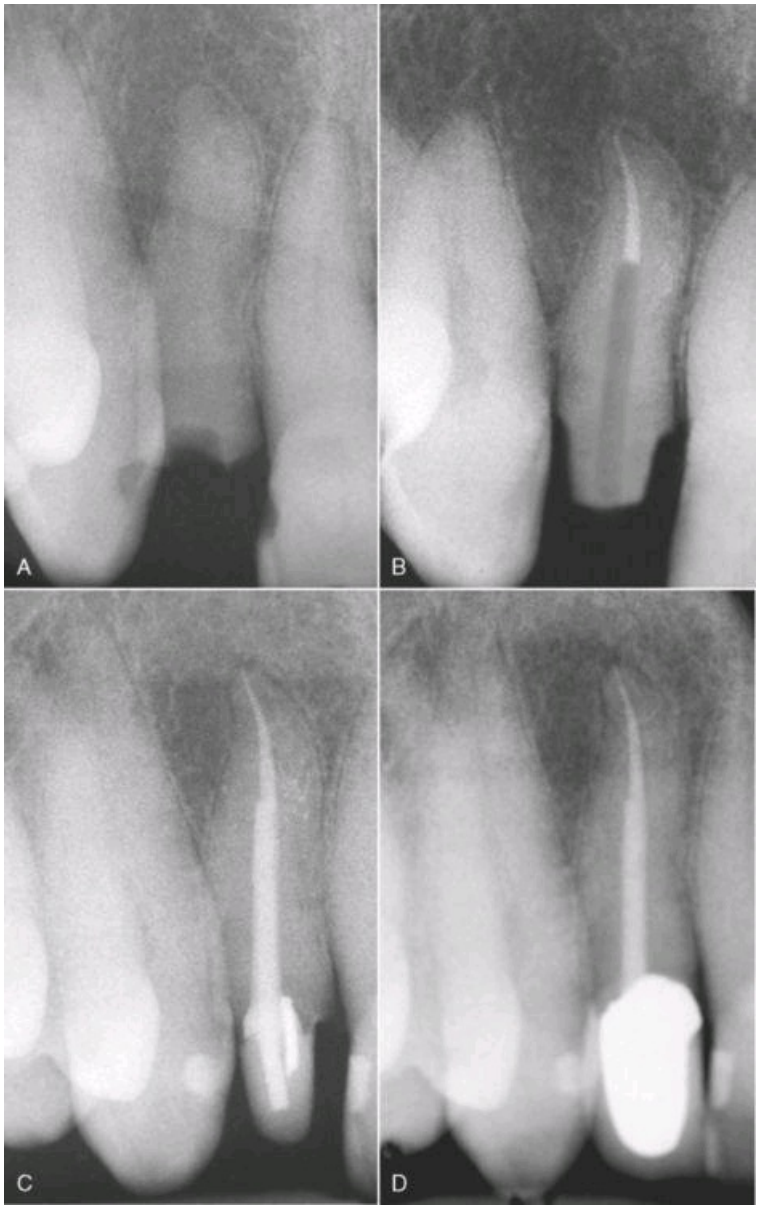

Figure 16-8 A and **B,** Post space preparation showing adequate retention of gutta-percha to provide an enduring apical seal. **C,** The post (here a Parapost) is fitted so that no gap occurs between the post and remaining gutta-percha, and a resin composite core is built up. **D,** The crown in place.

Removal of Gutta-Percha

Whenever possible, gutta-percha is removed immediately after obturation to ensure the most predictable apical seal.[55] At this stage, the dentist is most familiar with the canal features, including shape, length, size, and curvature. Depending on the obturation technique, the canal may be filled only to the desired length or gutta-percha may be removed to length using a hot instrument. The remaining gutta-percha is then vertically condensed in the apical canal before the sealer has set. The obturation radiograph will confirm that sufficient gutta-percha remains (4 to 5 mm). Studies have shown that canal leakage

occurs when only 2 or 3 mm of gutta-percha is retained apically; however, little or no leakage occurs when there is 4 mm or more.[55,56] Therefore 4 mm of gutta-percha provides an appropriate apical seal. However, because of variation in the angulation of clinical radiographs, it may be prudent to retain 5 mm or more of radiographic gutta-percha.

Gutta-percha removal at a subsequent appointment is satisfactory.[55-57] A safe procedure is the use of a heated instrument. Gutta-percha is removed in increments to the desired length, using a heat carrier or heated plugger. Any instrument that penetrates to the desired depth can be used, as long as it has sufficient heat capacity. Alternative means of removal include solvents and mechanical. Problems with solvents, such as chloroform, xylene, or eucalyptol, include messiness and unpredictable depth of penetration. Use of rotary instruments, especially Peeso reamers, requires caution because of their tendency to diverge and perforate or at least seriously damage the root. They may also "grab" and displace the apical gutta-percha. Specially designed rotary nickel-titanium instruments rotating at low speeds may be more effective, but have little evidence to recommend their use.

Finishing the Post Space

The post space is then further refined. If gutta-percha has been adequately removed, use of rotary instruments for final canal shaping should not be a problem. The bulk of the canal preparation has been achieved by the endodontic treatment, requiring only small refinements. Parallel-sided drills generate significant temperature increase; final shaping can be performed using hand manipulation of the cutting instrument because only small amounts of tooth need to be removed.

Ferrule

The use of cervical ferrule (circumferential band of metal) that encompasses tooth structure helps prevent tooth fracture. Ferrules formed by the crown extending cervically so it engages tooth structure apical to the core are effective in helping teeth resist fracture, whereas ferrules created by the core encompassing tooth structure are generally not effective.[58-61] Crown ferrules that encompass more than 1 mm of tooth structure are the most effective in helping teeth resist fracture.[59] Ferrules that encompass 2 mm of tooth structure around the entire circumference of a tooth produce greater fracture resistance than ferrules that engage only part of the tooth circumference.[60,61]

RETENTION AND CORE SYSTEMS

Anterior Teeth

These teeth must withstand lateral forces from mandibular excursive movements, which if transmitted via a post, tend to split the root. Consideration should be given to the occlusal scheme. Where possible, the excursive load should be limited, with more force being borne by adjacent, more structurally sound teeth. Either a prefabricated post with direct core buildup or a cast post and core will function effectively.

A prefabricated post with direct core buildup can be used for an anterior tooth (Figures 16-8 and 16-9). If used, a preformed post should be passive to minimize wedging forces. Also, passive fit facilitates removing the post if retreatment is necessary. Screw-retained posts are contraindicated'they predispose to vertical root fracture and are difficult to remove.

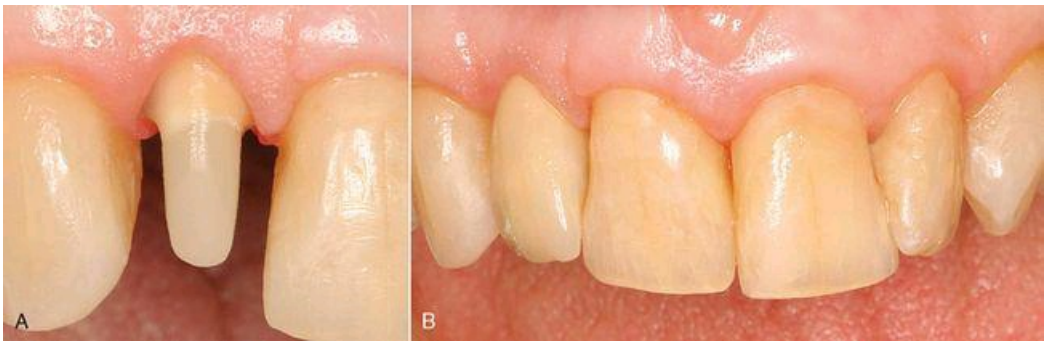

Figure 16-9 A, Resin composite core buildup, with a ferrule incorporated into the preparation. **B,** Full crown as the definitive restoration.

A cast metal post and core is fabricated as a single unit. The post portion provides unit strength and retention, and the core cannot separate from the post. Custom fitting of the post and core permits minimal dentin removal both from the canal space and coronally, plus maximum ferrule effect without crown lengthening.[60,61] Importantly, core shape should conform to the remaining coronal tooth structure, rather than machining the tooth for a standard core or technique.

Posterior Teeth

Premolars with substantial loss of coronal structure, particularly maxillary premolars, are best restored with a cast post and core (Figure 16-10). Narrow mesiodistal root width and substantial developmental root depressions coupled with tapered roots may result in excessive removal of root structure when preparing the tooth for a prefabricated post. Additionally, the mesiodistal thinness of the tooth may not permit adequate core thickness in association with a prefabricated post. Minimal enlargement during post space preparation is essential to preserve sufficient dentin thickness.[54] In maxillary premolars with two roots, only the palatal canal should be used for the post; the buccal furcation groove and narrow root preclude use of the buccal root.[54,62] A small, short (2 to 3 mm) post in the buccal canal provides some retention and antirotation.

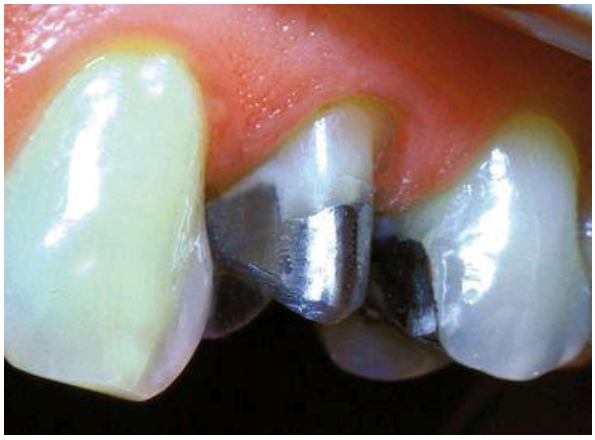

Figure 16-10 A cast post and core provides the best foundation for restoring maxillary premolars.

Molars, which have larger pulp chambers, permit direct core options; the volume of the core is greater and the chamber shape provides retention. Most molars are restored with the direct core only, without a post. However, with minimal remaining coronal tooth structure and a small pulp chamber, a post may be placed in one canal for additional retention. Posts are rarely necessary in molars and should be considered only when essentially no coronal tooth structure remains.

The longest and straightest canal is preferred for the post, usually the palatal canal of maxillary molars and the distal canal of mandibular molars.[53] Other canals are narrower and more curved and are in weaker roots with surface concavities. These should be used only (and cautiously) if other factors preclude placement in the larger canals. Core retention is supplemented by extending the core material 1 to 2 mm into the remaining canal orifices.

A wide variety of passively seated prefabricated posts is available. Parallel-sided posts provide greater retention than tapered posts and do not wedge. They require more post space preparation; matching post size to canal size is important to minimize dentin removal. The post need not contact dentin throughout its length to achieve adequate retention (this is important in distal canals of mandibular molars, which are often broad cervically). Threaded screw posts should not be used.

Molar core design is simple and placement of the core requires little removal of tooth structure. A coronoradicular core of amalgam (amalcore) condensed into the chamber and slightly into canal orifices is preferred and provides a passive, strong core (see Figure 16-4).[63] With fast-setting amalgam, the crown may be prepared at the same visit, although preparation is easier when the material is fully set. A widely used alternative is resin composite, with comparable fracture resistance to amalgam and more favorable fracture patterns if failure occurs.[40,64] However, composite resin has the advantage of allowing immediate crown preparation.[40] Glass ionomer cements do not have sufficient shear strength.

Pins

There is no need for retentive pins. The stresses and microfractures generated in dentin and the risk of perforation by pins outweigh any potential gain in retention of the restoration. Pins have been suggested for antirotation of the post/core, but this is best achieved by other means such as a slightly out-of-round preparation.

RESTORING ACCESS THROUGH AN EXISTING RESTORATION

Occasionally, pulps undergo irreversible pulpitis or necrosis after placement of a crown, requiring root canal treatment (Figure 16-11).[9,12,65] Access through the restoration, with subsequent definitive repair of the opening, is often preferred.

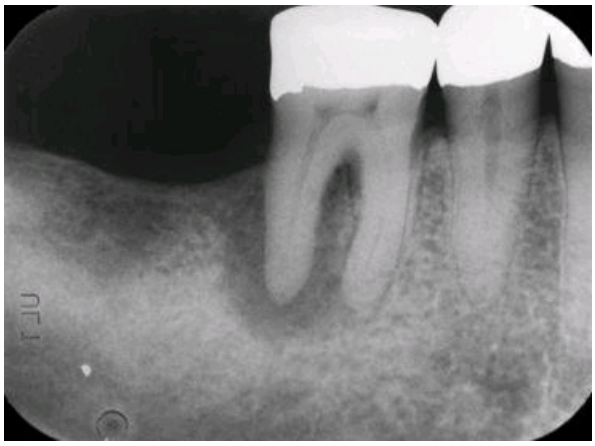

Figure 16-11 Pulp necrosis several years after preparation and placement of a full-coverage restoration. Root canal treatment is now required through the crown, with attendant risks of perforation or loss of retention.

For the restoration to remain functional, three conditions must be met: (1) the interface between the restoration and the repair material must provide a good coronal seal; (2) retention of the restoration must not be compromised; and (3) the final core structure must support the restoration against functional or minor traumatic stresses. Access, particularly if overextended, may leave only a thin shell of dentin, especially in anterior teeth and premolars. Retention then depends almost entirely on the repair material. Fortunately, the chamber and canal are available to create a core that provides adequate retention and support in most instances. Placement of a dowel or post through an access or through an existing restoration adds no support and little retention and is rarely indicated.

The repair material should have high compressive and shear strength. In most situations, amalgam is preferred. It improves retention, maintains and even improves its seal with time, and is easily condensed into the entire chamber and access cavity as a single unit. Amalgam also functions well in anterior teeth with metal-ceramic restorations. Composite resins are usually the material of choice in tooth-colored crowns.[40] Glass ionomer cements and glass cermets do not have the requisite shear strength.

Chapter Review Questions available in Appendix B or on the DVD

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Obturation

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Recognize the clinical criteria that determine when to obturate.
2. List the criteria for the ideal obturating material.
3. Describe the purpose of obturation and the reasons why inadequate obturation may result in treatment failure.
4. Identify core obturating materials most commonly used and list their constituents and physical properties.
5. Describe the advantages and disadvantages of each core material.
6. Discuss the indications and contraindications for obturating with each core material.

7. Differentiate between “standardized,” “conventional,” and “tapered” sizes of gutta-percha cones and discuss when each is indicated.

8. Define and differentiate between lateral and vertical compaction and suggest where each is indicated.

9. Describe the lateral compaction technique.

10. Discuss the significance of depth of spreader penetration during lateral compaction.

11. Describe the vertical compaction technique.

12. Describe briefly other techniques used for obturation, including thermoplasticization, thermocompaction, paste injection, core carrier systems, and sectional obturation.

13. Describe the custom cone (chloroform-softened) technique and discuss when it is indicated.

14. Describe the preparation of the canal for obturation.

15. Review the techniques for final drying and apical clearing.

16. Discuss the technique for fitting the master cone.

17. List criteria for the ideal sealer.

18. Describe a technique for mixing and placing sealer.

19. Discuss the technique for removing excess sealer and obturating material from the chamber and why this process is necessary.

20. Discuss the clinical and radiographic criteria for evaluating the quality of obturation.

OBJECTIVES OF OBTURATION

The obturation phase of root canal treatment receives a great deal of attention. Historically, obturation has been accorded the role of the most critical step and the cause of most treatment failures. An early and often quoted report[1] stated that most treatment failures could be attributed to inadequate obturation. Such retrospective surveys have major limitations. This study consisted of radiographic assessment of healing at various periods of time after root canal treatment.[1] The observed failures were correlated with apparently poorly obturated canals (as evaluated on radiographs). The fallacy in this reasoning is evident; just because two events are associated does not prove cause and effect.

In other words, although canals in these failed treatments may not have demonstrated dense fills, other factors may have caused irritation of the periapical tissues and failure. These include (1) loss of or inadequate coronal seal, (2) inadequate débridement and disinfection, (3) missed canals, (4) vertical root fractures, (5) significant periodontal disease, (6) coronal fractures, (7) poor aseptic technique, and (8) procedural errors such as loss of length, ledging, zipping, and perforations.

Significantly, a periapical lesion may heal after débridement without obturation. Although this is not an acceptable treatment option (an unobturated canal would result in long-term treatment failure), it does demonstrate an important concept: *what is removed from the root canal system is more important than what is inserted in the system.* Obturation is important, but it is not the most significant factor for success.

The objective of obturation is to create a complete seal along the length of the root canal system from the coronal opening to the apical termination. The importance of establishing and maintaining a *coronal* seal has been overlooked; the quality of the coronal seal is at least as important as the apical seal in long-term success.[2]

POTENTIAL CAUSES OF FAILURE

Most treatment failures related to deficiencies in obturation are long-term failures. A low volume of irritant or slow release of irritant into periapical tissues produces damage that is not apparent in the short term. *Persistence or development of periapical pathosis may not be evident for months or even years after treatment.* Therefore recall evaluation to assess the response to treatment is important. Obturation-related failures occur in different ways.

Apical Seal

Irritating Remnants in Canals

Bacteria, tissue debris, and other irritants are usually not totally removed during cleaning and shaping (see Chapter 15). These constitute a potential source of irritation that may lead to failure. It is likely (and there is evidence) that sealing in these irritants during obturation may prevent their escape into the surrounding tissues. Obviously, this seal must remain intact indefinitely because this reservoir of irritants persists forever. Interestingly, some bacteria sealed in the canal may lose viability, probably because of lack of substrate.[3] Possibly, other bacteria remain dormant, waiting for the introduction of substrate to proliferate and create havoc. Even dead bacteria or their remnants can be irritating or antigenic and cause inflammation.

Coronal Seal

Irritants from Oral Cavity

A coronal seal is extremely important. If the myriad of irritants in the oral cavity gain access to periapical tissues, they may cause inflammation and treatment failure. Irritants include substances in saliva such as microorganisms, food, chemicals, or other agents that pass through the mouth.

If coronal gutta-percha with sealer obturation is exposed to saliva, dissolution of sealer and leakage over a relatively short period of time may occur.[4-6] This results in leakage of bacteria, toxins, and chemicals into and around the gutta-percha.[7] The consequences of sealer loss are obvious; communication from the oral cavity to the periapex or periodontium will eventually be complete via a lateral canal or apical foramen.

It is not possible to determine clinically whether communication from the oral cavity to the periapex has been established. Therefore it is unwise to restore a tooth with a canal that may contain saliva, bacteria, food debris, or other irritants. Coronal exposure of the obturating material for more than a short period of time through loss of restoration, recurrent caries, or open margins requires retreatment. The time of exposure requiring retreatment is undetermined but probably depends on varied factors such as quality of obturation, length of canal(s), and surface area of exposure.

Restoration

Both design and placement of the final restoration are critical. This aspect of treatment is an integral part of obturation. The restoration acts as a protector of tooth structure and is the primary coronal seal, whether temporary or final. These factors are discussed in detail in Chapter 16.

Lateral Seal

Although not as critical as the apical and coronal seals, establishment of a seal in the inner middle aspect of the canal is also important. Lateral canals are occasionally found in these regions; they constitute a potential communication for irritants from the canal to the lateral periodontium (Figure 17-1).

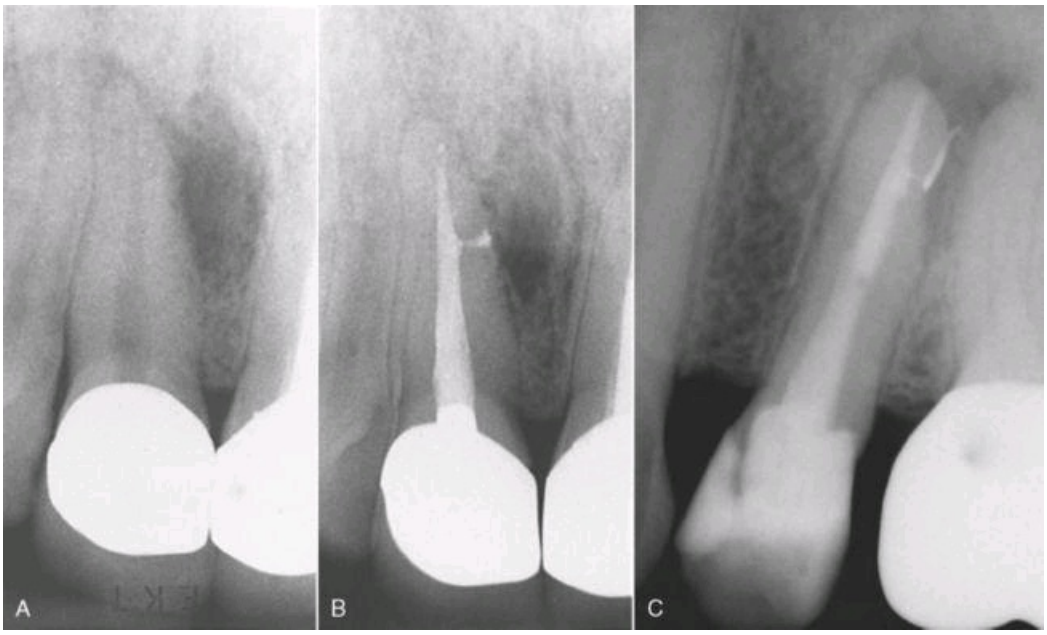


Figure 17-1 A, Pulp necrosis with apical and lateral radiolucent lesions. **B**, On obturation, a lateral canal was detected communicating with periodontium. This lesion should heal after removal of necrotic pulp tissue in the main canal and then obturation. **C**, Completed obturation showing lateral canal with extrusion of sealer along the periodontium. The lesion should heal within 6 months to 1 year.

*(**C**, Courtesy Dr. J. Fransen.)*

Length of Obturation

Extent of obturation relative to the apex is also important. Ideally, obturating materials should remain within the canal.

Overfill

Overfills are undesirable. Prognosis studies consistently show that failures increase with time when the primary obturating material has been extruded.[8,9] Histologic examination of periapical tissues after overfilling typically shows increased inflammation with delayed or impaired healing.[10] Patients experience more postobturation discomfort after overfills. Two other problems with overfills are irritation from the material itself and an inadequate apical seal.

Obturating Materials

Whether the obturating material is core or sealer, both are irritants to a greater or lesser degree.[11] Gutta-percha cores, as well as sealers in particular, are toxic when they are in contact with tissues. Sealers invoke a foreign body response and inflammation.[12,13] Gutta-percha is mildly toxic initially.

Lack of Apical Seal Secondary to Overfill

Lack of apical seal may be even more important than irritation from the materials. Gutta-percha, like amalgam, requires a matrix to compact against. Imagine trying to compact and form amalgam into a class II preparation without a metal matrix. The same is true of gutta-percha and sealer. Absence of an apical matrix or barrier may prevent sufficient lateral and vertical compaction, resulting in inadequate obturation.

A tapered apical preparation with no core materials and a small amount of sealer passing out of the foramen is not a significant problem. The taper helps form an adequate matrix for gutta-percha compaction, and irritation from the sealer should resolve. However, when there is gross overfill of both primary obturating materials and sealer, persistent inflammation[10] and failure often ensue (Figure 17-2).

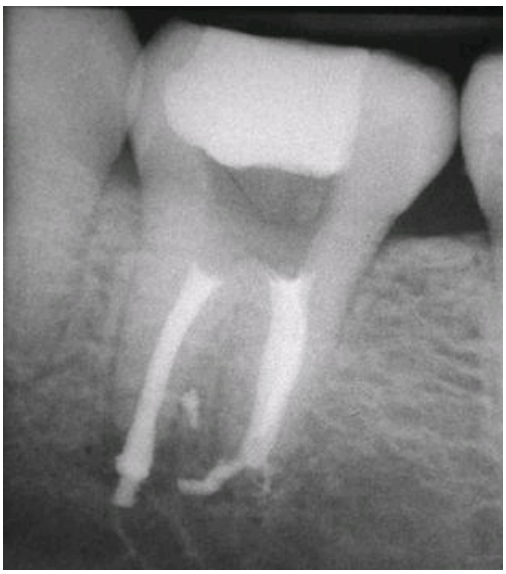


Figure 17-2 Overfill of both mesial and distal canals. Lack of apical resistance and retention form (no apical matrix) permitted the extrusion of the gutta-percha/sealer mass.

Underfill

Underfill results when both preparation and obturation are short of the desired working length or when the obturation does not extend to the prepared length. Either instance (or failure to treat a canal) may contribute to treatment failure, particularly long-term (Figure 17-3).

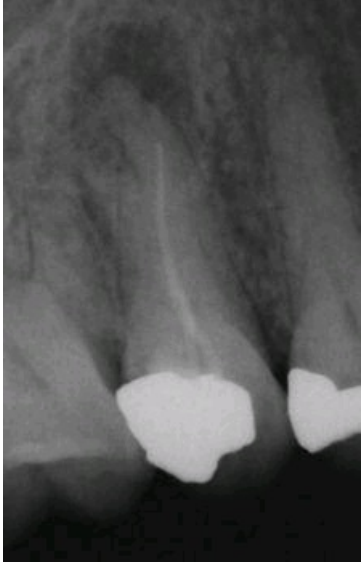


Figure 17-3 Failure caused by operative errors. The buccal canal is underprepared (inadequate débridement) and incompletely obturated (short fill); the palatal canal is neither instrumented nor filled.

The “optimal” preparation/obturation length for a necrotic pulp is 0.5 to 1 mm short of the radiographic apex (Figure 17-4).[13] For a vital pulp, the length is 0 to 2 mm short. Preparation or obturation short of these lengths may leave existing or potential irritants in the apical canal. Periapical inflammation may develop over an extended period of time, depending on the volume of irritants or the balance established between irritants and the immune system.

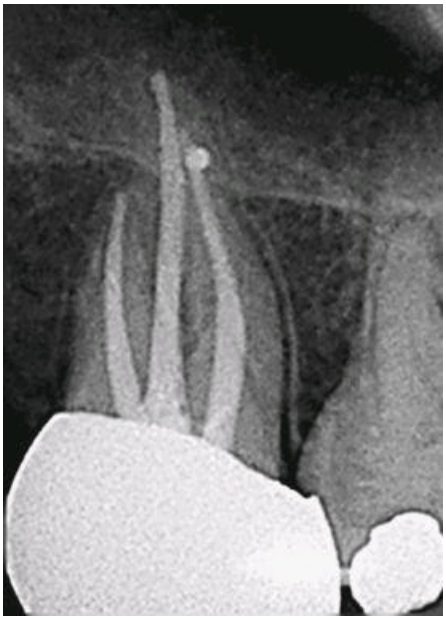


Figure 17-4 Continuously tapering canal preparations and obturations to the desired lengths. The canals were laterally compacted with gutta-percha and sealer; the fills are homogeneous with no voids.

Compared with overfill, underfill is less of a problem, as indicated by prognosis and histologic studies. Therefore the directive is: *if there is going to be an error, err on the short side* and try to confine everything to the canal space.

Lateral Canals

The role of lateral (accessory) canals in root canal treatment has been a subject of debate. These canals connect pulp space and periodontium. Irritants in the root canal system, such as bacteria and necrotic debris, may leak into the lateral periodontium and initiate inflammation (see Figure 17-1).

Histologic examination of roots after débridement shows that lateral canals are rarely if ever débrided.[14] There is no significant difference in the ability of various obturation techniques to fill the main canal. However, certain techniques tend to force materials into the lateral canals.[15]

When the main canal space is adequately débrided and obturated, lateral lesions adjacent to lateral canals heal as readily as periapical lesions. This occurs whether obturating material has or has not been expressed into the lateral canal.

The conclusion is that obturation of lateral canals is inconsequential to the outcome of most root canal treatments despite proponents of certain techniques that claim to fill lateral canals.[16]

Vertical Root Fractures

Vertical root fracture is a devastating occurrence that usually requires removal of the tooth or fractured root. Signs and symptoms, as well as radiographic findings, show that bone loss and soft tissue lesions are common.[17] Lateral forces exerted during obturation or post placement are major etiologic factors owing to their wedging action.[18-22] The pathogenesis, findings, and prevention of vertical fractures are discussed further in Chapter 7.

TIMING OF OBTURATION

When questions arise, such as “When is treatment to be completed? Is it time to obturate?,” the following factors are considered: signs and symptoms, pulp and periapical status, and difficulty of procedure. Combinations of these factors affect decisions made about the number of appointments and timing of obturation.

Patient Symptoms

In general, if the patient presents with severe symptoms and the diagnosis is symptomatic (acute) apical periodontitis or abscess, obturation is contraindicated. These are emergency situations, thus it is preferable to manage the immediate problem and delay definitive treatment. Even an acute apical abscess may be treated in a single appointment.[23] However, this is not good treatment. If the patient continues to have problems, management is more difficult if the canal is filled.

Painful irreversible pulpitis is a different situation. Because the inflamed pulp (which is the pain source) is to be removed, obturation may be completed at the same appointment. However, treatment of these problems requires caution because of difficulties in management of a patient in pain.

Pulp and Periapical Status

Vital Pulp

Regardless of the inflammatory status of the pulp and if time permits, the procedure may be completed in a single visit.

Necrotic Pulp

Without significant symptoms, obturation may be completed during the same appointment as canal preparation. Pulp necrosis with asymptomatic apical periodontitis or chronic apical abscess, or condensing osteitis *alone* is not necessarily a contraindication to single-appointment treatment at least as related to postobturation symptoms.

There may be an advantage, however, to multiple appointments related to healing of apical pathosis. Recent studies indicate the benefits of treating these patients in two visits.[24,25] Placement of an intracanal antimicrobial dressing, such as calcium hydroxide, reduces bacteria and reduces inflammation somewhat. Calcium hydroxide in the canal for 7 days can effectively inhibit bacteria.[26] However, a recent prognosis study comparing singlevisit versus two-visit with intracanal calcium hydroxide treatment did not demonstrate differences in long-term prognosis.[27] At present, there are no definitive conclusions about when single- or multiple-visit procedures are indicated in which situations.

One situation that contraindicates single-visit care is the presence and persistence of exudation in the canal during preparation. The potential for posttreatment exacerbation is increased if the periapical lesion is productive and generates continual suppuration. If the canal is sealed, pressure and corresponding tissue destruction may proceed rapidly. In these cases, canal preparation is completed, followed by calcium hydroxide placement. A dry cotton pellet is placed over the calcium hydroxide and the access is sealed with a temporary restoration. Generally, exudation will be diminished and controllable at a subsequent appointment; obturation may then be completed.

Degree of Difficulty

Complex cases are time consuming and are better managed in multiple appointments.

Culture Results

Few practitioners today rely on culturing canal contents to indicate timing of completion of treatment. Although the evidence is not clear about the value of cultures as an aid in increasing success in root canal treatment, culture results are an indicator of long-term prognosis.[28] Some believe that persistent positive cultures may indicate a poorly débrided canal, missed canals, or resistant strains of bacteria; however, these conclusions have not been proved and are debatable. However, proponents recommend that at least one negative culture be obtained before obturation, which requires more than one appointment. Currently, this approach is seldom used.

Number of Appointments

The decision about the number of appointments needed usually occurs during initial treatment planning. The decision to schedule another appointment, when made *during* an appointment, reflects a change of circumstances such as the patient or dentist is tired or has lost patience.

CORE OBTURATING MATERIALS

Primary obturating materials are usually solid or semisolid (paste or softened form). They comprise the bulk of material that will fill the canal space and may or may not be used with a sealer. However, a sealer is essential with all core obturating materials, although sealers behave differently with different obturating materials and techniques.[29]

These materials may be introduced into the canals in different forms and may be manipulated by different means once inside. Imaginations (and marketing) run rampant, resulting in a variety of materials and techniques. However, a small number of widely accepted and taught materials and techniques are used for obturation. These are discussed in some detail, and alternatives are discussed as well but in less detail. Whatever the material, there are desirable properties that must be considered (Box 17-1).[30]

Box 17-1 Desirable Properties of Obturating Materials

Grossman suggested that the ideal obturant should do the following[30]:

- □ Be easily introduced into the canal.
- □ Seal the canal laterally, as well as apically.
- □ Not shrink after being inserted.
- □ Be impervious to moisture.
- □ Be bactericidal or at least discourage bacterial growth.
- □ Be radiopaque.
- □ Not stain tooth structure.
- □ Not irritate periapical tissues or affect tooth structure.
- □ Be sterile or easily sterilized.
- □ Be easily removed from the root canal.

At this time, no material satisfies all these criteria.

Solid Materials

Solids have major advantages over semisolids (pastes). Although various materials have been tried, the only one universally accepted currently is gutta-percha as the primary material. This has withstood the test of time and research and is by far the most commonly used material.[31]

Synthetic resin–based core materials are new to the endodontic marketplace and are discussed later in the chapter. A major advantage of these solid cores over semisolid paste types is the ability to control length, as well as the reasonable ability to adapt to irregularities and to create an adequate seal.

Gutta-Percha

Composition

The primary ingredient of a gutta-percha cone is zinc oxide (±75%). Gutta-percha accounts for approximately 20% and gives the cone its unique properties such as plasticity. The remaining ingredients are binders, opaquers, and coloring agents.

Shapes

Gutta-percha cones are available in two basic shapes: the “standardized” and the “conventional” (Figures 17-5 and 17-6). Standardized cones are designed to have the same size and taper as the corresponding endodontic instruments (i.e., a No. 40 cone should correspond to a No. 40 file.

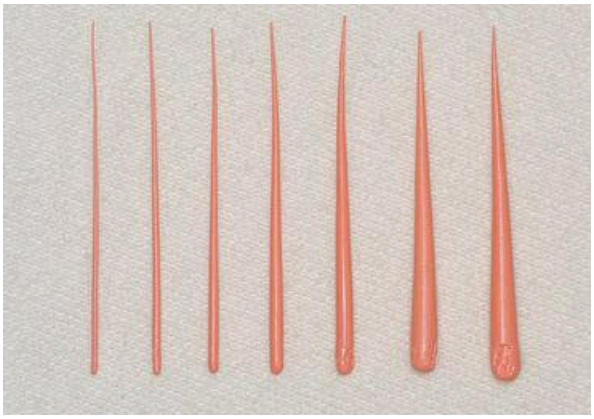


Figure 17-5 Conventional gutta-percha cones; extra fine, fine fine, fine, medium fine, medium, large, and extra large.

(From Cohen S, Hargreaves K: Pathways of the pulp, ed 9, St. Louis, 2006, Mosby.)

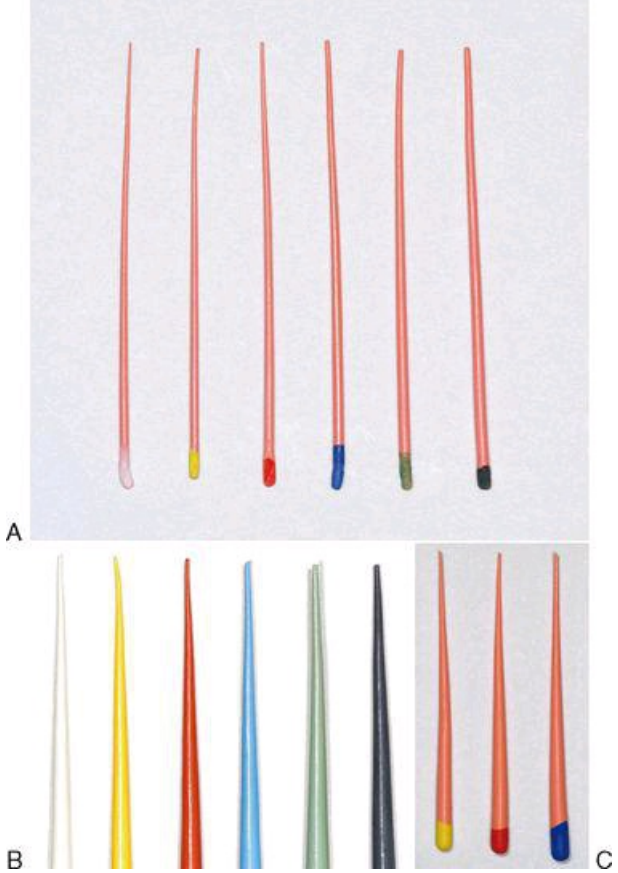


Figure 17-6 A, Standardized cone Nos. 15 to 40. **B,** Standardized cones No. 0.06, taper sizes No. 15 to 40. **C,** Standardized cones Protaper S1, S2, S3.

(From Cohen S, Hargreaves K: Pathways of the pulp, ed 9, St. Louis, 2006, Mosby.)

Interestingly, there is no uniformity in gutta-percha sizing. For example, the contents of a box or vial of No. 40 standardized gutta-percha cones varies in size from No. 35 to No. 45 and has inconsistent tips and shapes.[32] This lack of uniformity is not critical, however; canal shape after preparation is also variable.

Conventional cones use a different sizing system. The tip of the cone has one size and the body of the cone another, and they are available in various combinations. For example, a fine tip end-medium body would be referred to as a fine-medium cone. Generally, conventional cones have a smaller tip with a relatively wider body compared with standardized cones.

Gutta-percha master cones with varying tapers tend to be selected according to the method of canal preparation or to match the master apical fill size and corresponding taper. This practice is becoming more widespread, especially since the introduction of rotary instrumentation using variably tapered instruments.

Advantages

Gutta-percha has withstood the test of time: it was introduced as an obturating material more than 160 years ago. It is the standard to which other obturating materials are compared. First, because of plasticity, gutta-percha adapts with compaction to irregularities in prepared canals. Second, it is relatively easy to manage and manipulate despite some complex obturation techniques. Third, gutta-percha is easy to remove from the canal, either partially to allow post placement or totally for retreatment. Finally, gutta-percha has relatively little toxicity, being nearly inert over time when in contact with connective tissue.[11] Another advantage of gutta-percha is that it tends to be self-sterilizing because it will not support bacterial growth. If there is a possibility of cones being contaminated, they are predictably sterilized by immersion in 1% (or greater) sodium hypochlorite for 1 minute.[33]

Sealability

Regardless of the technique used (compaction or plasticization), studies have consistently shown that gutta-percha without sealer will not seal.[34,35] Disadvantages of gutta-percha are a lack of adhesion to dentin and a slight elasticity, which causes a rebound and pulling away from the canal walls. Warmed gutta-percha shrinks during cooling. Gutta-percha mixed with solvents, such as chloroform or eucalyptol, shrinks markedly with evaporation of the solvent.[36] It was believed that a sealer fills and seals the spaces between the gutta-percha cones and between the gutta-percha and the canal wall. However, it has been shown that sealer does not predictably fill these spaces and coat the wall under gutta-percha.[37] Exactly how sealer contributes to the seal is unknown. Also, sealability of gutta-percha is generally better if added to the canal in increments.

Methods of Placement

As stated earlier, placement methods are varied and imaginative. Most popular is lateral compaction, followed by vertical compaction.[38] Other techniques involve either chemical or physical alteration of the gutta-percha in an attempt to render the material more plastic or more adaptable.

Another variation is a system that includes a solid core (carrier) surrounded by a cone of gutta-percha. The carrier may be stainless steel or titanium but more typically is plastic. After preparation, the carrier and gutta-percha are warmed and placed in the canal as a unit.

Other devices have been introduced that involve warming to plasticize and inject gutta-percha. These will be discussed in more detail later in this chapter.

Resin

As a potential replacement for gutta-percha, synthetic polyester resin–based polymers are emerging as promising obturation materials (Figure 17-7).[39,40] The core material is polycaprolactone with fillers of bioactive glass and other components and is used with a dual cured Bis-GMA resin sealer and self-etching primer. This combination was an attempt to form a single entity or "monoblock" in the root canal system. The material has been shown to be noncytotoxic, biocompatible, and nonmutagenic and has been approved for endodontic use by the Food and Drug Administration (FDA). Earlier research showed this material to be more resistant to leakage than gutta-percha obturation.[40] More recent evidence indicates no difference.[41,42] The resin cores, available in conventional and standardized cones, have similar handling properties as gutta-percha and can be removed by solvents and heat in case retreatment is indicated. Pellets are also available for use in thermoplastic injection techniques. As yet, there are no controlled clinical trials with long-term evaluation to demonstrate how this system compares to gutta-percha as an obturating material.

Figure 17-7 Resin-based obturation system contains primer, sealer, and cones. The cones resemble gutta-percha and can be placed using lateral or warm vertical compaction. Pellets are available for thermoplastic injection.

(From Cohen S, Hargreaves K: Pathways of the pulp, ed 9, St Louis, Mosby, 2006.)

Silver Points

Silver points, which are pure silver, were designed to correspond to the last file size used in preparation and to presumably fill the canal precisely in all dimensions. Because of the complexity of shape of the root canals, this is fallacious. It is impossible to predictably prepare canals to a uniform size and shape.[43]

Although the short-term sealability success of silver points seemed comparable to that of gutta-percha, silver points are a poor long-term choice as a routine obturating material.[44,45] Their major problems relate to nonadaptability (Figure 17-8) and possible toxicity from corrosion.[46] Also, because of their tight frictional fit and hardness, silver cones are difficult to remove totally (retreatment) or partially (post space preparation).[47] Also, if silver cones are contacted with a bur, their seal may be broken.

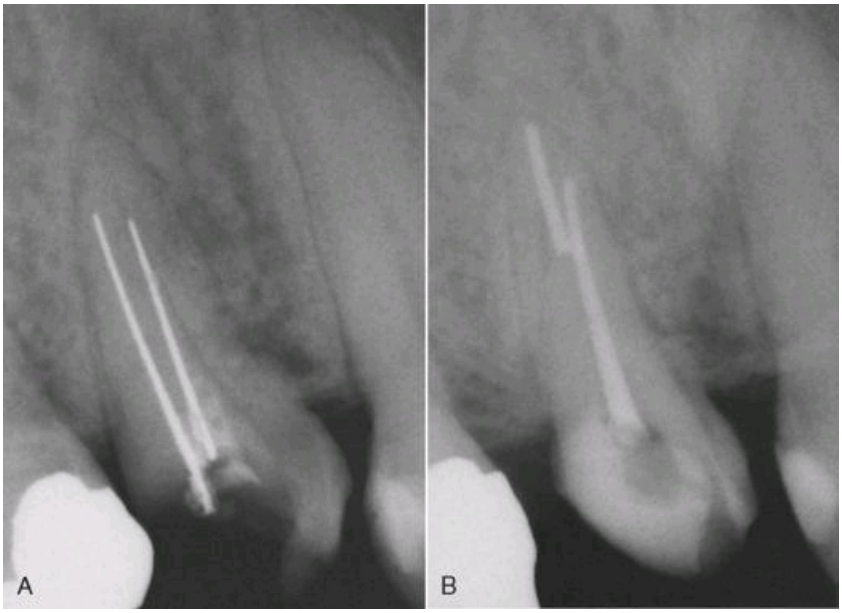


Figure 17-8 A, Obturation with silver points. Retreatment necessary due to loss of coronal restoration, short obturation, and inadequate débridement. **B,** Retreatment and obturation using vertical compaction of gutta-percha and sealer. Post space was provided for the final restoration.

(Courtesy Dr. T. Remmers.)

In summary, *silver cones are no longer recommended* as an obturating material.

Pastes (Semisolids)

Why not develop a paste or cement that can be mixed in a liquid or putty form, inject the material to length, fill the entire canal, and then allow the material to set? This would be fast, the paste would fill the entire canal space, and obturation would be much simpler. In addition, this method would permit use of a material that would adhere to dentin and create an absolute seal.

Although the concept is appealing, there are significant practical difficulties. However, it certainly has been attempted, and work on developing such a material continues. The major disadvantages of paste materials are lack of length control, unpredictability, shrinkage, and toxicity of ingredients.

Types

Zinc Oxide and Eugenol

Zinc oxide and eugenol may be mixed pure (no additives) to intermediate thickness. Other formulations combine zinc oxide–eugenol (ZnOE) with various additives. The types known as N2 or RC2B are most common. These are derivations of Sargenti's formula and contain opaquers, metallic oxides (lead) or chlorides (mercuric), steroids (at times), plasticizers, paraformaldehyde, and various other ingredients. Claims of antimicrobial properties, biologic therapeutic activity, and superiority are made for these paste formulations. No proof exists that they contribute any beneficial aspects to obturation. In fact, most of these additives are quite toxic.[48] In 1998, the American Association of Endodontists issued a position statement on the use of paraformaldehyde-containing endodontic filling materials. Use of these materials is below the standard of care.

Plastics

It has been suggested that a resin-based sealer, such as AH26 and Diaket, be used as the sole obturating material. These sealers have the same disadvantages as pastes and therefore have not attained popular use.

Techniques of Placement

Various approaches and instruments have been devised or modified for insertion of pastes or sealers. Two popular methods are injection and placement with a lentulo spiral.

Injection is accomplished using a syringe-type device with a barrel and special needles.[49] The paste is mixed and placed in the barrel, a screw handle is inserted and twisted, and the paste is extruded through the special needlelike tips. The needles are placed deep in the canal, and the paste is expressed as the needles are slowly backed out of the canal. Advocates claim that this method completely fills the canal from the apical portion to the canal orifice.

Instrument placement is done with lentulo spiral drills. The paste is mixed, the drill is coated, and then it is placed and spun deep in the canal. As with the syringe device, the canal is supposedly filled with paste as the drill is slowly withdrawn.

Both techniques are more attractive in theory than in fact. Neither technique has demonstrated an ability to seal effectively or to fill the root canal system. Because of lack of length control, both injection and placement by lentulo spiral drill have major deficiencies and are contraindicated.

Advantages and Disadvantages of Pastes

The *advantages* are obvious: paste techniques are fast and relativity easy to use and involve the use of a single material. The equipment needed, at least with the lentulo spiral technique, is relatively simple, comprising only a limited assortment of special drills.

The *disadvantages* outweigh the advantages by far. First, the universal problem with any nonsolid core material is length control. It is difficult to avoid overfills or underfills (Figure 17-9). Theoretically, radiographs should be made many times during obturation to assess length and density as the material is being injected or placed. Obviously, this is time consuming and subjects the patient to needless radiation.

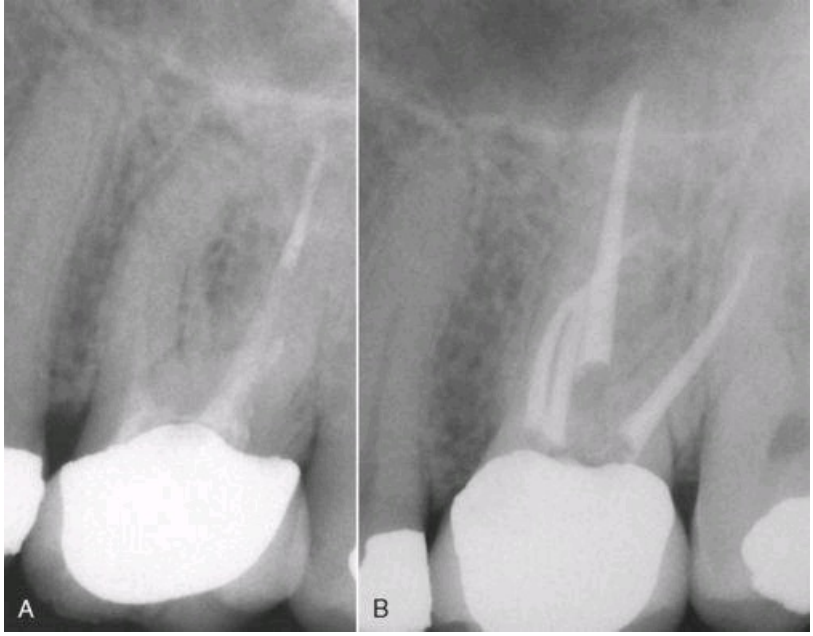


Figure 17-9 A, Inadequate preparation, paste fills, and evidence of periapical disease **B,** Tooth was retreated. A second mesiobuccal canal was located, instrumented, and obturated. All canals were obturated using warm vertical compaction using a resin core and sealer.

Another major disadvantage is sealability. These techniques seal inconsistently: sometimes well, other times poorly.[50] This unpredictability may be related to three factors: (1) large voids or discrepancies within the material or adjacent to the walls; (2) shrinkage of ZnOE on setting, which leaves a space for microleakage; and (3) solubility of pastes in tissue or oral fluids. In addition, injection devices are difficult to clean and maintain.

SEALERS

A basic concept is that sealer is more important than the core obturating material. Sealer accomplishes the objective of providing a fluid-tight seal; the core occupies space, serving as a vehicle for the sealer.[51] Sealer must be used in conjunction with the obturating material, regardless of the technique or material used. This makes the physical properties and placement of the sealer important.

Desirable Properties

Grossman outlined the criteria for an ideal sealer.[51] None of the sealers currently available possesses all these ideal properties, but some have more than others. His criteria are as follows.

Tissue Tolerance

The sealer and its components should cause neither tissue destruction nor cell death. All commonly used sealers show a degree of toxicity.[11] This toxicity is greatest when the sealer is unset but tends to diminish after setting and with time.[52]

No Shrinkage with Setting

Sealer should remain dimensionally stable or even expand slightly on setting.

Slow Setting Time

Sealer should provide adequate working time for placement and manipulation of obturating material, then set reasonably soon after obturation is complete. It is desirable to have sealer unset if post space is made immediately.

Adhesiveness

Adhesiveness is a most desirable property. A truly adhesive material would form an absolute bond between the core material and dentin, closing off any spaces. ZnOE-based sealers have no adhesion; plastics have some.

Radiopacity

Sealer should be readily visible on radiographs. However, the more radiopaque the sealer, the more it obscures voids in the obturation. Some clinicians prefer a highly radiopaque sealer to mask discrepancies.

Absence of Staining

Remnants should not cause future staining of the crown. Currently, all tested sealers, particularly ZnOE-based sealers or those containing heavy metals, stain dentin.[53,54]

Solubility in Solvent

Occasionally, post space or retreatment may be necessary days, months, or years after obturation. The sealer should be soluble in a solvent. Different sealers have different degrees of solubility in different solvents and with varying mechanical techniques.[55]

Insolubility to Oral and Tissue Fluids

Sealer should not disintegrate when in contact with tissue fluids. Sealers are somewhat soluble, particularly when in contact with oral fluids.[5]

Bacteriostatic Properties

Although a bactericidal sealer would seem to be desirable, a substance that kills bacteria will also be toxic to host tissues. At a minimum, the sealer should not encourage bacterial growth.[56]

Creation of a Seal

Creating a seal is obviously an important physical property. The material must create and maintain a seal apically, laterally, and coronally.

Types

Generally, the four major types of sealers are ZnOE-based, plastics, glass ionomer, and those containing calcium hydroxide. Other variations and compounds have been proposed or are marketed as sealers, but these should be considered experimental.

Certainly, the standard sealer with which all others are compared is the Grossman formulation, which has withstood the test of time and usage, although some plastics (resins) now widely used have many desirable properties. Calcium hydroxide and glass ionomer types are newer and have interesting properties but also significant drawbacks.

Zinc Oxide–Eugenol-Based Sealers

The major advantage of ZnOE-based sealer types is their long history of successful usage. Obviously, their positive qualities outweigh their negative aspects (staining, very slow setting time, nonadhesion, and solubility).

Grossman's Formulation

Grossman's formula is as follows:

Powder: zinc oxide (body), 42 parts; stabellite resin (setting time and consistency), 27 parts; bismuth subcarbonate, 15 parts; barium sulfate (radiopacity), 15 parts; sodium borate, 1 part

Liquid: eugenol

Most ZnOE sealers in use and available today are variations of this original formula. A problem with this formulation is the very slow setting time, more than 2 months, as studied in a usage test.[57]

Other Types

ZnOE forms the base for other sealers, some of which have been used more commonly than others. These will not be discussed further here.

Plastics

Plastics are much less commonly used and accepted, at least in the United States. Some have very desirable properties, however.

Epoxy

Epoxy is available in a powder-liquid formula (AH26). Its properties include antimicrobial action, adhesion, long working time, ease of mixing, and very good sealability. Its disadvantages are staining, relative insolubility in solvents, some toxicity when unset, and some solubility to oral fluids. A recently introduced variation (AH Plus) has similar physical properties but better biocompatibility because it releases less formaldehyde and a supposed decrease in dentin staining by elimination of silver from the formula.[58]

Other Plastics

Other plastics are primarily of the methylmethacrylate type and are not commonly used.

Calcium Hydroxide

Calcium hydroxide sealers have been introduced in which the calcium hydroxide is incorporated in a ZnOE or plastic base. These sealers supposedly have biologic properties that stimulate a calcific barrier at the apex; however, these properties have not been conclusively demonstrated in clinical or experimental use. Calcium hydroxide sealers show antimicrobial properties and adequate short-term sealability.[59] Questions have been raised about their long-term stability (greater solubility) and tissue toxicity. Until further experimental and clinical data are available, these sealers have no demonstrated advantages and are not recommended.

Glass Ionomer

Endodontic formulations of glass ionomer have been introduced recently. This material has the advantage of bonding to dentin, seems to provide an adequate apical and coronal seal, and is biocompatible.[60,61] However, its hardness and insolubility make retreatment and post space preparation more difficult.[62] Glass ionomer impregnated gutta-percha used with a glass ionomer sealer will be discussed later in the chapter.

Others

Various luting agents and basing and restorative materials have been tried and tested as endodontic sealers.[63] Examples are zinc phosphate cement, composite, and polycarboxylate cement. These materials have not proved satisfactory.

Mixing

ZnOE sealer types should be mixed carefully to a thick consistency. They should string approximately 2 to 3 inches. The thicker the mix, the better are the properties of the sealer, particularly in regard to stability, superiority of seal, and diminished toxicity.[64] Epoxy resins are mixed to a much thinner consistency.

Placement

Various techniques have been advocated for placement of sealer, which is done before insertion of the core material. The sealer may be placed with paper points, files, ultrasonic activation of files, or special drills (lentulo); as a coating on the primary cone; or by injecting with special syringes. Although different methods have shown varying degrees of effectiveness in sealer application, no technique has proved superior.[65-67] In fact, sealers may not completely cover the interface between gutta-percha and canal wall after obturation.[37]

A simple and effective technique is to coat the walls by picking up sealer on the final apical file or a one-size smaller file (Figure 17-10). The file is teased to length and spun counterclockwise, which has the effect of carrying the sealer apically and coating the walls. Flooding the canal with sealer is neither necessary nor desirable.

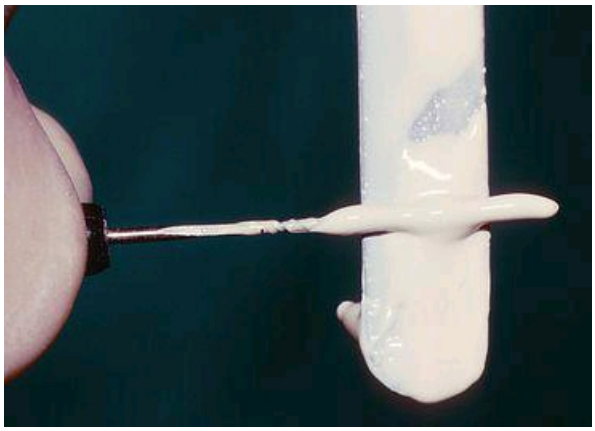


Figure 17-10 An easy, effective method of sealer application. The file covered with sealer will be inserted and spun counterclockwise to coat the canal walls.

Sealer is not placed in all canals at once unless it has a long working time. Removing sealer that has set is difficult. The Grossman formulations and epoxy resins are slow setting and may be placed in all canals.[57]

OBTURATION TECHNIQUES WITH GUTTA-PERCHA

Different approaches are available, depending on the size of the prepared canal, the final shape of the preparation, and irregularities within the canal. The overriding factor is operator preference.[68]

Selection of Technique

The two traditional techniques are lateral and vertical compaction of gutta-percha; sealability is similar in both.[38,69] Again, the choice is dictated primarily by preference and custom, although there may be special situations indicating a particular use of each technique. Both must be used with a sealer.

More recent approaches have been introduced that depend on warming and softening formulations of gutta-percha with special devices and instruments and then placing the gutta-percha incrementally. Many of these techniques and devices are heavily marketed and promoted and will be discussed later in the chapter.

Other methods are also used and most involve alteration of the entire gutta-percha cone with a solvent such as chloroform or eucalyptol. These are technique sensitive and therefore are not widely used or taught in the United States. They are not discussed in this textbook; details are found in other published sources.

A variation of lateral compaction is the solvent-softened (or custom-fitted tip) technique, which is outlined later in this chapter.

Lateral Compaction

Lateral compaction is the most popular technique of obturation, both in practice and as taught in most institutions.[70] Therefore this technique is described in detail.

Indications

Lateral compaction of gutta-percha may be used in most situations. Exceptions are severely curved or abnormally shaped canals or those with gross irregularities such as internal resorption. However, lateral compaction may be combined with other obturation approaches.[71] In general, if the situation is not amenable to lateral (or vertical, if that is the usual approach) compaction, it is too difficult for the general practitioner and the patient should be referred to an endodontist.

Advantages

Lateral compaction is relatively uncomplicated, requires a simple armamentarium, and seals and obturates as well as any other technique in conventional situations.[38,72] A major advantage of lateral compaction over most other techniques is length control. With an apical stop and with careful use of the spreader, the length of the gutta-percha filling is managed well. Additional advantages include ease of retreatment, adaptation to the canal walls, positive dimensional stability, and the ability to prepare post space.[73]

Disadvantages

A disadvantage of lateral compaction is that the resultant obturation is a series of sealer-welded cones and thus not a homogeneous mass. There are no other major disadvantages to lateral compaction other than difficulties in obturating severely curved canals, an open apex, and canals with internal resorptive defects.

Technique

Although there are variations, a workable and acceptable technique is presented here. Variations of lateral compaction are described in other textbooks and manuals.

Spreader or Plugger Selection

Selection and try-in should be performed during the cleaning and shaping of the canal. Finger spreaders or pluggers are preferred over standard (long-handled) spreaders because of better tactile sensations, improved apical seal, better instrument control (Figure 17-11), and reduced dentin stress during obturation.[19,73] Use of these finger instruments likely decreases the incidence of vertical root fractures during obturation. Finger spreaders or pluggers also can be inserted more deeply than standard hand spreaders (Figure 17-12).

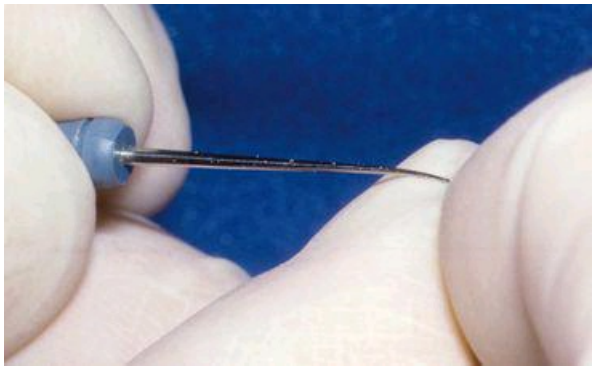


Figure 17-11 Finger spreaders may be precurved to improve negotiation in curved canals.

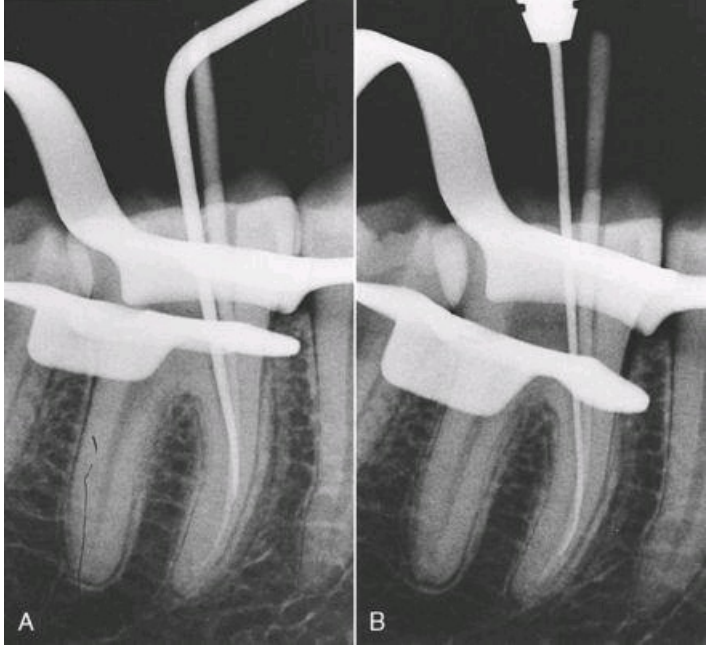


Figure 17-12 Comparison of hand spreader with finger pluggers or spreaders. **A,** The stiff, more tapered hand spreader will not negotiate the curve. **B,** The smaller, more flexible finger spreader permits deeper penetration and produces a superior apical seal.

Nickel-titanium finger spreaders have been introduced recently. Because of their flexibility, these spreaders seem to produce less wedging force while penetrating deeper.[74] Their advantage may be less tendency to produce vertical root fractures. These spreaders do behave differently because of their flexibility and require practice for efficient use.

Master Cone Selection

Either a standardized or a conventionally shaped, fine gutta-percha cone may be adapted as a master cone. Apical preparations that are very irregular in shape, are larger than a No. 50 file, have no apical stop, or have an apical seat greater than a No. 40 file should have the custom solvent-softened cone (described later).

Large standardized cones (No. 50 and above) are used in the custom cone technique in canals larger than a No. 50 file. Conventional cones are cut and adapted to canals less than a No. 50 file.

Fitting the Master Cone

Apical clearing (when indicated) is important before the master cone is fitted (see Chapter 15). After apical clearing is completed, the steps are as follows:

1. Because the master cone fits only in the apical portion of the apically cleared, flared canal, the amount of resistance shown to removal is slight (Figure 17-13). A slight frictional fit is acceptable; the so-called tugback is unnecessary.[75] However, there should be a definite stop when the cone fits into place. The cone is fitted to or within 0.5 mm of working length.

2. A cone may be too small as indicated by a buckling in the apical few millimeters (Figure 17-14, *A*). A larger sized apical end is made by cutting 1-mm segments off the master cone until the slight fit is obtained (Figure 17-14, *B*). Frequently, the cone cannot be inserted quite to working length. This is acceptable only if (a) the apical area has been cleared of debris and (b) the spreader penetrates to within 1 mm of the prepared length. A cleared apical area and deep spreader penetration will usually push the gutta-percha and sealer apically to fill the remaining 1 mm (Figure 17-15).[76]

3. The master cone is removed by grasping it at the reference point, and the length is verified by measuring it on a ruler, then corrected if necessary.

4. Master cone length (not lateral fit) is evaluated radiographically. Again, the cone should be no more than 1.0 mm short of the prepared length. The traditional close radiographic fit of the master cone in the apical third is unrelated to the quality of the final seal.[75]

5. If the master cone length is not within 1 mm of the prepared canal length, either (a) dry reaming is repeated to be certain there is no debris or (b) another, smaller cone is adapted.

6. A gutta-percha cone that extends beyond the apical foramen shows the lack of an apical stop. This requires a custom

solvent-softened cone or selection and modification of a shorter, larger cone or reinstrumentation of the canal to create apical resistance form.

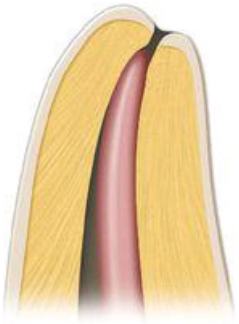
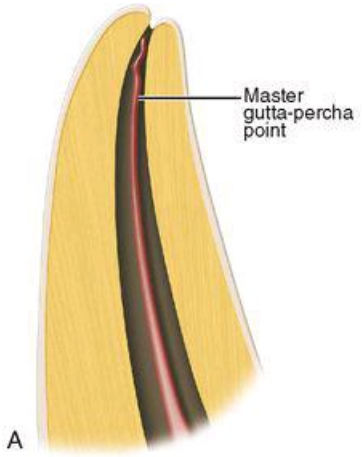


Figure 17-13 The master cone needs only a slight frictional fit in the very apical region. This permits deep spreader penetration between the gutta-percha and the canal wall.



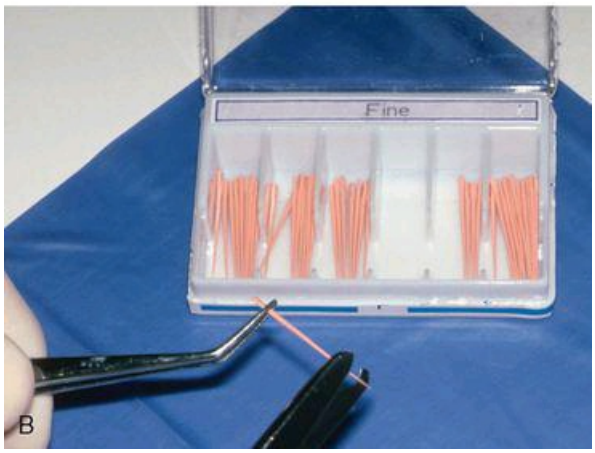


Figure 17-14 A, A cone that appears buckled on the radiograph or on removal is much too small. **B,** A larger cone should be selected or clipped to form a larger size at the tip.

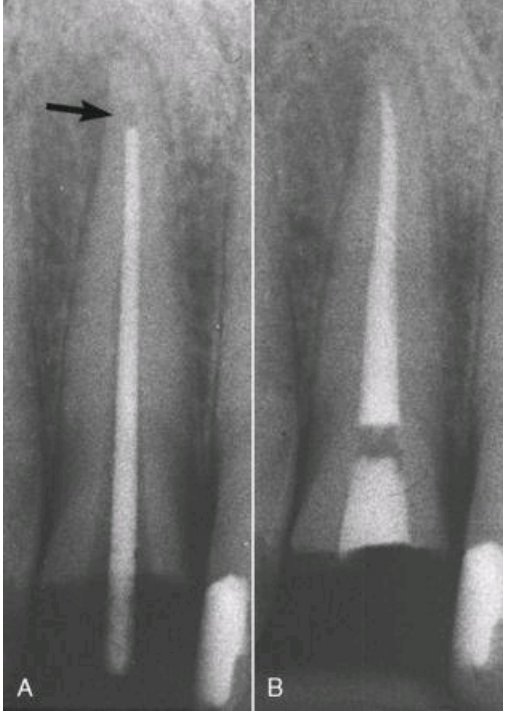


Figure 17-15 A, The master cone need not extend along the entire length if the preparation *(arrow)* has been apically cleared. **B,** Deep spreader penetration then often pushes the gutta-percha and sealer apically to fill the prepared space.

(Courtesy Dr. J. Parsons.)

Steps in Obturation

Although there are many combinations of obturating instruments and different types of gutta-percha, a suggested combination for routine situations is a fine finger plugger and fine accessory cone (Figure 17-16). There is no precise correlation between size of accessory point and size of finger spreader.[77] Specific steps are as follows (Figures 17-17 to 17-19):

1. Sealer is mixed and applied to canal walls.

2. The master cone (without sealer coating) is inserted slowly to allow air and excess cement to escape.

3. Before the spreader is inserted and removed, an accessory cone is picked up with locking pliers at the measured length, ready to be inserted.

4. The measured spreader is inserted between the master cone and the canal wall using firm (apical only) pressure (5 to 7 pounds as for amalgam condensation) to within 1 to 2 mm from working length. Spreader taper is the mechanical force that laterally compacts gutta-percha, creating a space for an additional accessory cone.

5. The spreader is freed for removal by back-and-forth rotation around its axis. The spreader is removed, and the measured accessory (fine) gutta-percha cone is immediately inserted into the space created.

6. (Optional) A radiograph may be made after one or two cones are placed. If there are length problems, the cones are retrieved. A new master cone is fit at a corrected length.

7. This procedure is repeated until the spreader can no longer be inserted beyond the apical third of the canal (approximately three to seven accessory cones depending on canal shape). The last insertion is an accessory cone, not the spreader! The spreader need not be stepped all the way out of the canal, adding accessory cones. Obturation may be evaluated with a radiograph at this time.

8. Excess gutta-percha is seared off with a hot instrument (Glick No. 1, a heated plugger or a battery-controlled heating device) (Figure 17-20). This is done approximately 1 mm apical to the gingival margin in anterior teeth and 1 mm apical to the canal orifice in posterior teeth.

9. The cervical portion of the warm gutta-percha is vertically compacted firmly using the Glick No. 1 or a No. 5-7 heater-plugger.

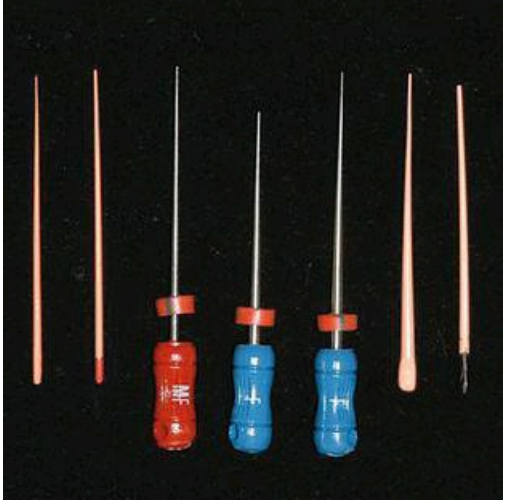


Figure 17-16 An assortment of finger spreaders and gutta-percha cones. From *left to right* are standardized gutta-percha points, followed by both 21 mm and 25 mm conventional finger spreaders with instrument stops on their shafts, followed by conventional gutta-percha points.

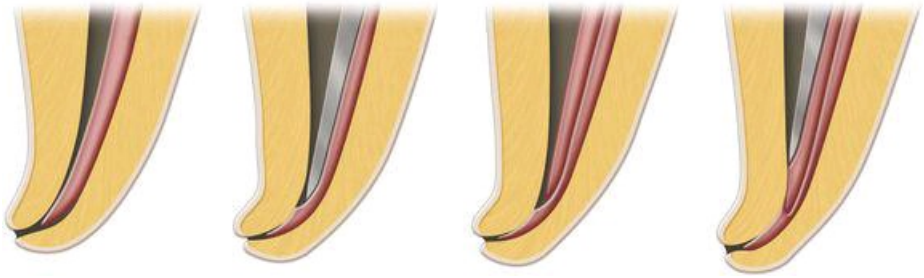


Figure 17-17 The steps of lateral compaction. **A,** The master cone is fitted. **B,** A finger spreader or plugger is inserted, ideally to 1 to 2 mm of the prepared length. **C,** The spreader is rotated and removed, and an accessory cone is placed in the space created. **D,** The process is repeated.

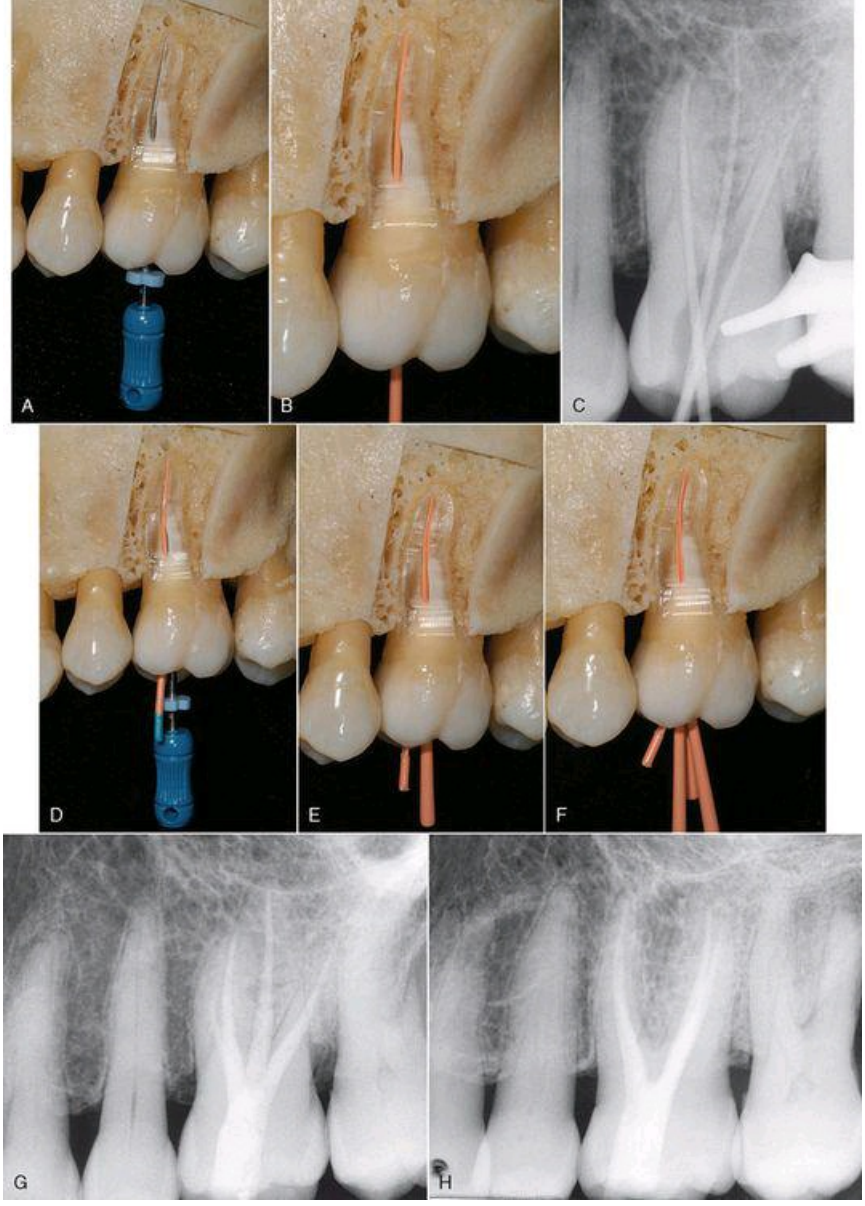
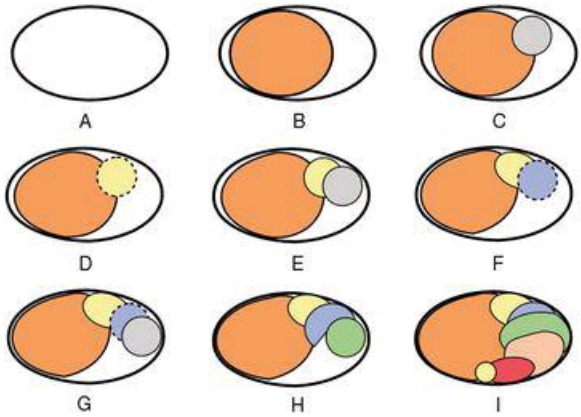


Figure 17-18 Lateral compaction. A finger spreader is first inserted to check proper depth of penetration. Standardized gutta-percha points are placed and verified radiographically. Once sealer is placed and the cone is to length, the spreader is inserted along the side of the cemented cone (in the mesiobuccal canal in this example). An accessory cone is placed in the space created by the spreader. The process is repeated (i.e., reinsertion of spreader followed by placement of another accessory cone) until the spreader does not penetrate beyond the middle third of the canal. The cones are removed at the orifice with heat and then the coronal mass is vertically compacted. The remaining canals are obturated in the same manner. The final radiograph demonstrates four canals properly obturated.

(Courtesy Dr. W. Johnson.)



A. Prepared canal
B. Master cone inserted
C. Spreader placed
D. Placement of accessory cone (shown as dashed circle)
E-H. Continuation of lateral compaction
I. Completion of obturation

Figure 17-19 Schematic of the steps of lateral compaction. Each insertion of the spreader to its most apical extent will laterally compact the gutta-percha cone toward the opposing wall. At the completion of the compaction, the canal will be obturated with a series of cones that have been cold-welded together with sealer.

(Courtesy Dr. J. Schweitzer.)

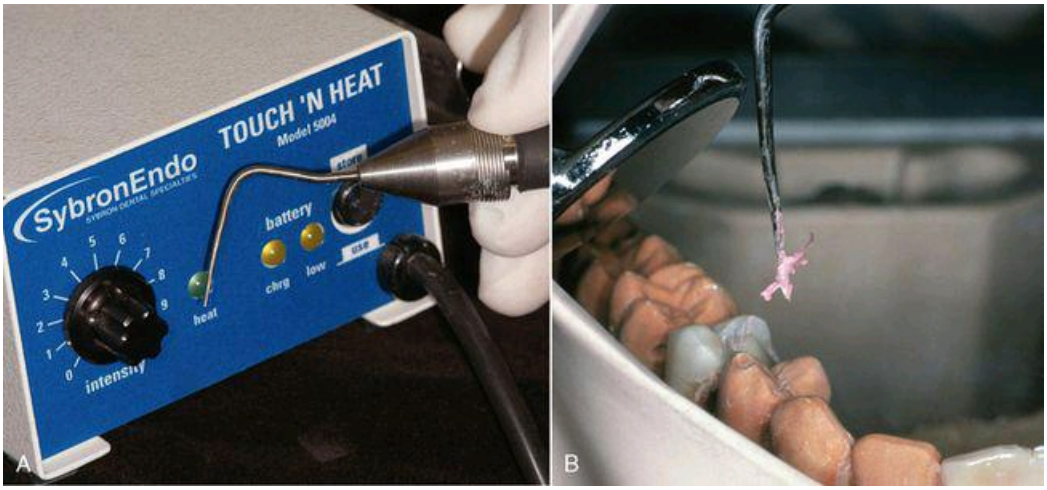


Figure 17-20 A, Convenient battery-controlled heating device holds an assortment of tips. **B,** The tip is rapidly heated for removal of excess gutta-percha from the chamber or from the canal when creating a post space.

Ultrasonic Compaction

A variation is lateral compaction with ultrasonic activation of the spreader. With this technique the spreader is placed next to the master cone and activated without a water coolant. Apical pressure is exerted, and the spreader is inserted to a predetermined length. Advantages are that the ultrasonic action may spread the sealer, the friction of the spreader may thermoplasticize the gutta-percha, and the force required to place the spreader is less.[78]

Suggestion

If two or more canals are obturated, compaction is performed separately in each. Each canal is completed and the excess removed before the next is begun.

Finishing Touches

The procedure is completed as follows:

1. The chamber is cleaned thoroughly with cotton pellets soaked in alcohol or chloroform; unset sealers are soluble in these solutions. Remnants of gutta-percha or sealer (in particular) may cause future discoloration (Figure 17-21).

2. A temporary or permanent restoration is placed. Appropriate temporization or restoration (semi permanent or permanent) is discussed in Chapter 15.

3. A radiograph is made with the restoration in place and the clamp removed.

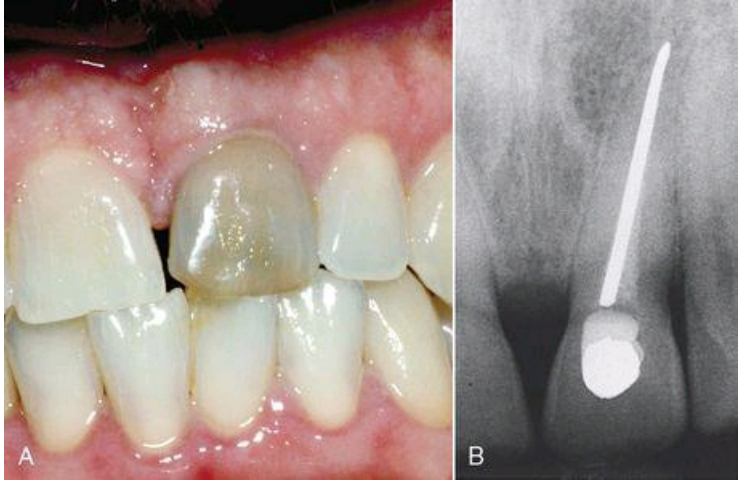


Figure 17-21 Often discoloration is caused by improper technique and is preventable. **A,** Too-frequent unfortunate occurrence: gradual discoloration after root canal treatment. **B,** Causes include sealer remnants and a silver point extending into the chamber and amalgam restoring the lingual access. This tooth will be difficult to bleach because the stains are from metallic ions.

Correcting Obturation Problems

Occasionally, voids or length problems will be apparent on the radiograph taken during or after obturation. These should be corrected *now,* before the sealer sets.

For voids, gutta-percha is removed with hot pluggers until the spreader can be reinserted just beyond the void or discrepancy. Then, a fresh mix of sealer is prepared. Lateral compaction is performed as described previously; sealer is added back to the canal by coating each accessory cone.

An advantage of making an obturation verification radiograph before the excess gutta-percha is seared off is that the entire mass can usually be removed by grasping the cones with the fingers. Fitting a new master cone and reobturation is then possible.

If the excess gutta-percha has been seared off, an overfill can sometimes be corrected before the sealer sets by removing all gutta-percha with files or broaches. When extruded beyond the apex, the overfilled gutta-percha is difficult to recover through the canal, particularly after the sealer sets. Extruded sealer can only be retrieved surgically.

Obturating materials extruded beyond the apex are irritants and affect healing, but generally they do not completely prevent resolution unless there is gross overfill of core material. ZnOE-based sealers often absorb from periapical tissues over time.[79] These situations should not be treated surgically unless failure to heal is evident on recall examination.

Solvent-Softened Custom Cones

Different solvents have been proposed and tested. The two that have proved useful clinically and are used most often are chloroform and halothane; however, concerns about toxicity have been expressed. Some concerns about chloroform are unfounded because recent evaluations show that, if used judiciously, chloroform is safe for retreatment and for the formation of custom cones.[80,81] The technique described here uses chloroform; halothane is used in a similar manner.

An impression of the apical 3 or 4 mm of the canal is made in the gutta-percha master cone. It is basically a "cone within a cone" because only the cone surface is softened and then shaped. The objective is to fit the cone closely into the apical portion to try to create a better seal but primarily to prevent extrusion of gutta-percha beyond the apex. In fact, solvent softening does not result ultimately in a better apical seal.[82,83]

Indications

The two indications are (I) an apical stop is lacking or (2) a stop is present but the apical portion of the canal is very large or irregular.

Technique

The steps are as follows:

1. The master cone selected is usually a larger standardized cone that, when inserted, stops 2 to 4 mm short of the working length.

2. The master cone tip (apical 3 to 4 mm) is softened by dipping it in chloroform for 1 to 2 seconds (Figure 17-22). Halothane dipping is done for 3 to 4 seconds.

3. The cone is tamped apically in the canal several times. Then, the cone is grasped at the reference point, removed, and measured. Softening and tamping are repeated until the cone goes to working length. The cone is marked or bent for orientation; it must be replaced exactly in the same position during obturation.

4. The cone is removed, and the solvent is allowed to evaporate. The cone should not be left in the canal for any length of time while soft; the softening will continue and the tip may separate when the master cone is removed. The cone tip should show an impression of the apical preparation (Figure 17-23).

5. The cone is replaced and a confirmatory radiograph is made. The cone need not extend to working length but may be slightly shorter, up to 1 mm.

6. Sealer is mixed thick. The canal walls are not coated, only the apical third of the master cone. The cone is carefully inserted to length without wiping off all the sealer on the walls.

7. The standard lateral compaction procedure follows with spreader insertion, rotation, removal, accessory cone placement, and so on. More sealer is added by coating each accessory cone before placement.

8. A radiograph may be made to evaluate the obturation before the excess is seared off. The mass can be pulled out and reobturation done if necessary.

9. Post space may be prepared immediately after obturation (Figure 17-24).

Figure 17-22 The softened custom cone technique. The apical portion (3 to 4 mm) is dipped in chloroform for 1 to 2 seconds and then tamped in the canal.

Figure 17-23 After the softened cone has been tamped into the canal and removed, it should show an impression of the apical region.

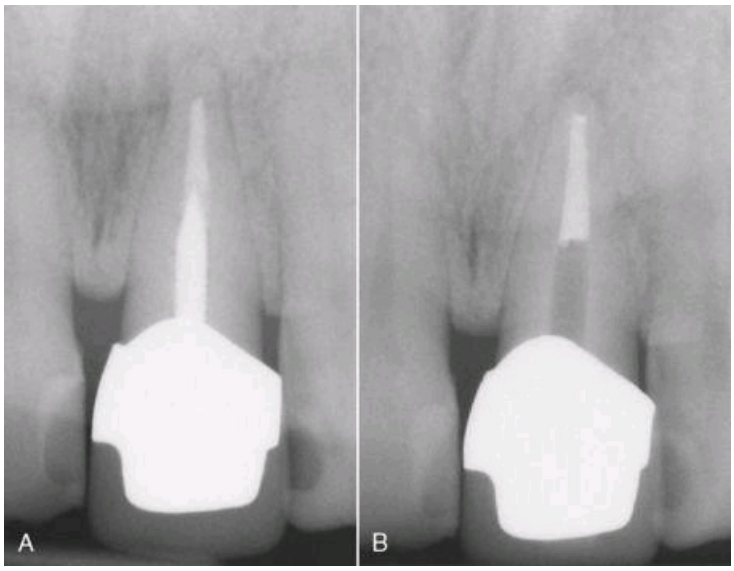


Figure 17-24 A, Retreatment required because of persistent apical periodontitis. **B**, Following post and gutta-percha removal and canal instrumentation, the apical 3 mm of the canal was impressed and obturated using a custom-formed gutta-percha cone. This was caused by the irregular, resorptive nature of the apical portion of the canal. Post space was prepared following obturation.

(Courtesy Dr. T. Remmers.)

Vertical Compaction

Vertical compaction is also an effective technique; studies show its sealability is comparable to that of lateral compaction.[38] Although vertical compaction is not widely taught in dental schools, the technique is becoming more popular. With the introduction of new devices and techniques, the warm vertical compaction techni-que is somewhat more user friendly and is less time consuming.

Indications

In general, vertical compaction can be used in the same situations as lateral compaction. It is preferred in a few circumstances, such as with internal resorption and with root end induction.

Advantages and Disadvantages

The principal advantage of vertical over lateral compaction is the ability to adapt the warmed and softened gutta-percha to the irregular root canal system.[84,85] Disadvantages include difficulty of length control, a more complicated procedure, and a larger assortment of required instruments.[38] Also, a somewhat larger canal preparation is necessary to allow manipulation of the instruments.

Technique

The warm vertical compaction technique requires a heat source and various sized pluggers for compaction of the thermoplasticized gutta-percha. Schilder pluggers begin at 0.4 mm in diameter and increase by 0.1 mm for each of the successive instruments, with 1.1 mm the largest instrument. Pluggers are also available in ISO standardized sizes.

The technique consists of fitting a gutta-percha cone with a taper similar to the canal, short of the apex, and applying heat using a flame-heated carrier. The gutta-percha is softened by the heat and becomes plastic. Pluggers are then placed in the canal with apical pressure to produce a hydraulic force that moves the gutta-percha apically, against the canal walls, and into canal irregularities such as accessory canals. Gutta-percha is then added in small increments, and each increment of gutta-percha is heated and softened and packed vertically until the entire canal is filled. Detailed descriptions of the technique appear elsewhere.[86]

Other Warm Vertical Approaches

A recent modification of the warm vertical compaction technique is termed the *continuous wave of condensation.* Prerequisites for this technique are a tapering canal preparation, a constricted apical preparation, and an accurate cone fit. The technique is often used after preparation with nickel-titanium rotary files of greater taper. The heat source is an electric device that supplies heat to a plugger on demand (Figure 17-25). Pluggers are available in nonstandardized sizes that match the nonstandardized gutta-percha cones or in standardized sizes that match files of greater taper (Figure 17-25, *C*). In addition, two hand pluggers of differing diameters are used to sustain and compact the gutta-percha apically.

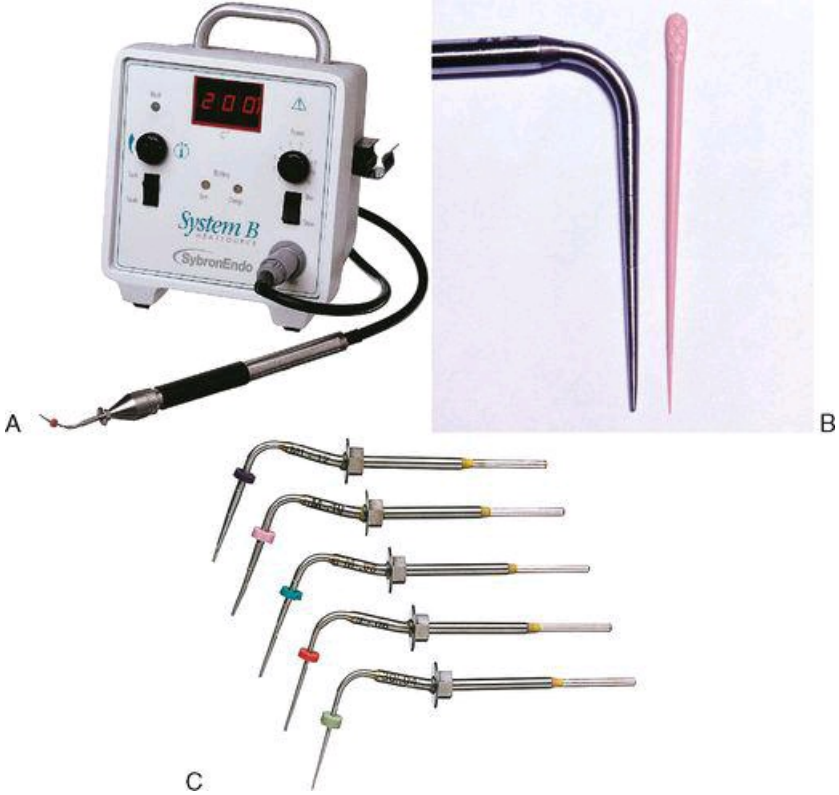


Figure 17-25 A specialized heating device. **A**, Controlled current causes rapid heating of the plugger, which then softens a prefit gutta-percha cone in the canal. **B,** "Continuous wave of condensation" plugger is designed to size-match the rotary file used to prepare the canal. Pluggers are also approximately matched to nonstandardized gutta-percha in an attempt to obturate the apical portion of a canal with a single cone. **C**, A variety of different size and taper pluggers are available.

(Courtesy SybronEndo, Orange, CA.)

Heat is applied at a prescribed temperature (200° C) for a short period of time as determined by the operator. By applying a constant source of heat to a prefitted gutta-percha cone, hydraulic pressure can be applied in one continuous motion. As the plugger moves apically, the fit becomes more precise and the hydraulic pressure is increased, forcing the gutta-percha into canal irregularities. Details of the continuous wave of condensation technique are available in other publications.[87]

There are inherent risks. When thermoplasticization or any technique that physically alters gutta-percha is used, there is the potential for extrusion into the periapical tissues (Figure 17-26), as well as possible damage to the periodontal ligament and supporting alveolar bone from heat. An increase of 10° C above body temperature appears to be a critical threshold for damaging osseous tissues. Flame-heated carriers reach high temperatures and pose the greatest threat of damage to the periodontal structures.[88,89] When used properly, the injectable gutta-percha technique and the continuous wave condensation technique appear to produce temperature changes that are below the critical threshold.[90,91]

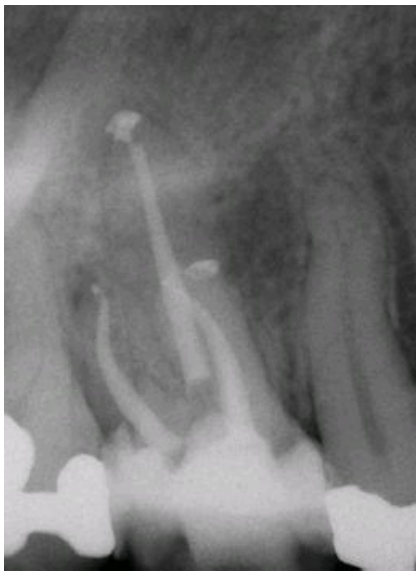


Figure 17-26 Completed obturation using continuous wave of condensation with gutta-percha and sealer. This technique, as with any warm obturation method, tends to apically extrude sealer. The sealer usually absorbs with time.

(Courtesy Dr. A. Hsiao.)

Sectional Obturation

A recent innovation is a technique that uses special devices and involves a two-phased sectional approach (Figure 17-27). A small apical segment of gutta-percha is placed ("downpacked") and followed by a backfilling of gutta-percha. This technique seems relatively fast and may prove useful but requires more investigation.[92] Details of the technique appear elsewhere.[93]

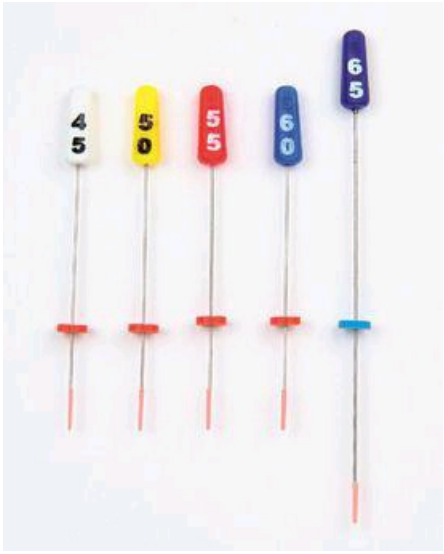


Figure 17-27 Carriers with apical plugs of gutta-percha attached. Once inserted into a canal prepared using specially designed rotary files, the carrier is rotated in a counterclockwise direction to separate the gutta-percha from the carrier. The canal is then backfilled using lateral or warm compaction. Carriers are also available with resin plugs.

(Courtesy Lightspeed Technology, Inc, San Antonio, TX.)

Thermoplasticized Injection

With this technique, specially formulated gutta-percha is warmed and then injected into the prepared canal with a device (Figure 17-28) that works like a caulking gun. When used in conjunction with a sealer, thermoplasticized injection provides an adequate seal.[94] This technique is useful in special situations (Figure 17-29). However, lack of length control and shrinkage on cooling are disadvantages.

Figure 17-28 A thermoplasticizing device. A high-heat gun softens gutta-percha into an injectable plastic mass.

(Courtesy Obtura Spartan, Fenton, MO.)

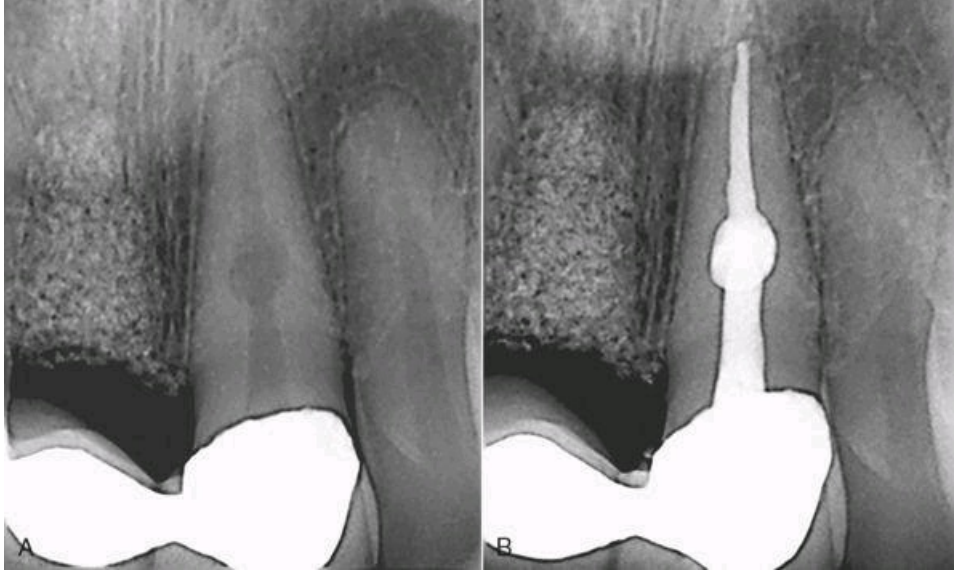


Figure 17-29 Internal resorption. **A,** Once the canal is cleaned and shaped, lateral compaction would not be the recommended technique to fill this defect. **B,** Thermoplastic injection of resin core obturating material was vertically compacted into the resorptive defect.

Solvent Techniques

Solvent techniques involve the total or partial dissolution of gutta-percha in solvents, primarily chloroform or eucalyptol. These have names such as chloropercha, eucapercha, diffusion technique, or chloroform resin. Often, these techniques are not used in conjunction with a standard sealer but depend on softened gutta-percha to adapt closely. The problem is that gutta-percha shrinks away from the walls as the solvents evaporate. Extensive leakage is generally seen with these techniques,[36] and some have a poorer long-term prognosis.[95]

Carrier-Based Systems

Carrier-based systems use a plastic central carrier coated with gutta-percha. The carrier is flexible yet it provides rigidity for the overlying gutta-percha. The obturators are tapered and standardized so the carriers correspond to the size of instruments. After canal preparation, the canal is dried and lightly coated with sealer. The appropriate size obturator is heated in a special oven and firmly placed to working length. The carrier is then sectioned 1 to 2 mm above the orifice to the canal. These carrier/gutta-percha systems are equivalent to conventional gutta-percha obturation with apical sealing but may not consistently create a coronal seal (Figure 17-30).[96-98]

Figure 17-30 Thermafil is an example of a carrier-based system. Obturators are designed to correspond to ISO standardized file sizes and are heated in a specially designed oven.

Advantages of the technique include ease of placement and the potential for the plasticized gutta-percha to flow into canal irregularities.[96] Disadvantages include a tendency for extrusion of material periapically and difficulties in removing carrier and gutta-percha during retreatment.[97,98]

A new flowable gutta-percha obturation system has been recently introduced in the marketplace (Figure 17-31).[99] It is composed of a mixture of finely ground gutta-percha, silicone-based sealer, and silver particles. After trituration, the material is injected into a canal before the placement of a gutta-percha master cone. No compaction is necessary, and the material purportedly self-cures in 30 minutes and expands slightly on setting. No heating is required with this system, and retreatment can be performed using conventional techniques. Clinically validated research of this material has been minimal.

Figure 17-31 GuttaFlow is a cold flowable injection system that combines a silicone-based matrix with finely ground gutta-percha. It is used in conjunction with a master gutta-percha point without the need for compaction.

(Courtesy Coltene/Whaledent, Cuyahoga Falls, OH.)

Another recent development is glass ionomer–impregnated gutta-percha (Figure 17-32).[100] The chemical bond between glass ionomers and dentin has been established.[101] However, the lack of an adequate bond to the core obturation material has been a major drawback for previous glass ionomer—based sealers. This material apparently addresses this by the incorporation of glass ionomer particles into the gutta-percha cone, followed by a 2 µm glass ionomer coating. It is claimed but not conclusively demonstrated that these glass ionomer particles encourage a true bond to form between the glass ionomer—based sealer and obturation cone. As with the polyester resin–based systems discussed previously, this bonding to dentin wall and obturation core by the sealer is referred to as a *monoblock.* Since the working time of the glass ionomer—based sealer is insufficient, only a single cone obturation technique is recommended. Research evaluating the efficacy of this system is not yet available. MTA can be used as an alternative filling material to gutta-percha.

Figure 17-32 Glass ionomer–coated gutta-percha points **(A)** are used in conjunction with a glass ionomer sealer **(B)** to attempt to create a monoblock within the canal system.

These new obturation systems are interesting and have potential. However, there is lack of research verification of their clinical effectiveness.

EVALUATION OF OBTURATION

Surprisingly, evaluation of obturation is difficult. The only means of immediate assessment is radiographic, which is imprecise at best. However, radiographic evaluation has been the standard and at least provides some criteria with which to judge obturation quality.

Symptoms

The presence of symptoms for a few days after obturation is common and probably is unrelated to an inadequate seal. This reflects a different phenomenon, which is likely tissue irritation from the procedure.

Radiographic Criteria

Good obturation (fluid-tight seal) cannot be seen on a radiograph. Only fairly gross discrepancies are visible, and these voids or deficiencies may or may not relate to lack of seal and may result in long-term failure.[102] The evaluative criteria as determined by studying the obturation radiograph (Figure 17-33) are as follows.

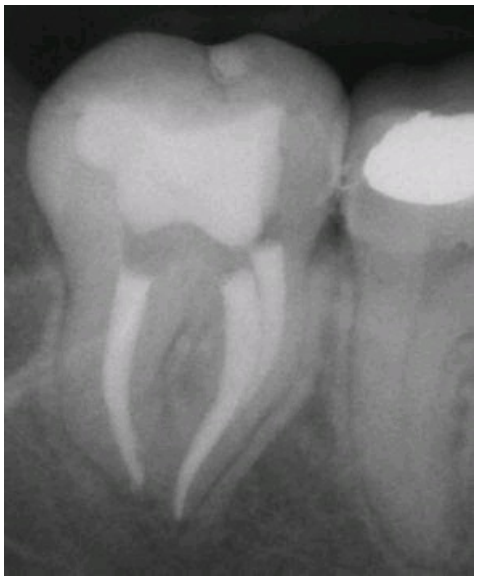


Figure 17-33 Bayonet-shaped canals. Obturation was with a combination of techniques and materials. The result is good quality: no voids, uniform density, and the obturation reflects the taper created during canal preparation. Resin core and sealer were vertically compacted using the continuous wave technique followed by backfilling with injectable thermoplasticized resin.

(Courtesy Dr. T. Remmers.)

Radiolucencies

Voids within the body or at the interface of obturating material and dentin wall represent incomplete obturation.

Density

Material should be of uniform density from coronal to apical aspects. The coronal region (and large canals) are more radiopaque than the apical region because of differences in mass of material. The margins of gutta-percha should be sharp and distinct, with no fuzziness, indicating close adaptation.

Length

The material should extend to the prepared length and be removed apical to the gingival margin (anterior teeth) and orifices (posterior teeth).

Taper

The gutta-percha should reflect the canal shape (i.e., it should be tapered from coronal to apical regions). Taper need not be uniform but should be consistent. Ideally, the apical region should taper nearly to a point unless the canal in this region was not small before preparation.

Restoration

Whether permanent or temporary, the restoration should be contacting enough dentin surface to ensure a coronal seal.

Chapter Review Questions available in Appendix B or on the DVD

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Procedural Accidents

Mahmoud Torabinejad, Ronald R. Lemon

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Recognize procedural accidents and describe the causes, prevention, and treatment of the following:
   a. Pulp chamber perforation during access preparation
   b. Ledging
   c. Dental materials or dentin shavings obstructing the canal
   d. Coronal or radicular perforation
   e. Separated instrument
   f. Obturation short of prepared working length
   g. Obturation materials expressed beyond apex
   h. Incomplete obturation
   i. Vertical root fracture
   j. Post space preparation mishaps

Like other complex disciplines of dentistry, an operator may encounter unwanted or unforeseen circumstances during root canal therapy that can affect the prognosis. These mishaps are collectively termed *procedural accidents.* However, fear of procedural accidents should not deter a practitioner from performing root canal treatment if proper case selection and competency issues are observed.

Knowledge of the etiologic factors involved in procedural accidents is essential for their prevention. In addition, methods of recognition and treatment, as well as the effects of such accidents on prognosis, must be learned. Most problems can be avoided by adhering to the basic principles of diagnosis, case selection, treatment planning, access preparation, cleaning and shaping, obturation, and post space preparation.

Examples of procedural accidents include swallowed or aspirated endodontic instruments, crown or root perforation, ledge formation, separated instruments, underfilled or overfilled canals, and vertically fractured roots. A good practitioner uses his or her knowledge, dexterity, intuition, patience, and awareness of his or her own limitations to minimize these accidents. When an accident occurs during root canal treatment, the patient should be informed about (1) the incident, (2) procedures necessary for correction, (3) alternative treatment modalities, and (4) the effect of this accident on prognosis. Proper medical-legal documentation is mandatory. A successful operator learns from past experiences and applies them to future challenges. In addition, the practitioner who knows his or her limitations will recognize potentially difficult cases and will refer the patient to an endodontist. The beneficiary will be the patient, who will receive the best care.

This chapter discusses the causes, prevention, and treatment of various types of procedural accidents that may occur at different phases of root canal treatment. The effect of these accidents on short- and long-term prognosis will be also described.

PERFORATIONS DURING ACCESS PREPARATION

The prime objective of an access cavity is to provide an unobstructed or straight-line pathway to the apical foramen. Accidents, such as excess removal of tooth structure or perforation, may occur during attempts to locate canals. Failure to achieve straight-line access is often the main etiologic factor for other types of intracanal accidents.

Causes

Despite anatomic variations in the configuration of various teeth, the pulp chamber, in most cases, is located in the *center* of the anatomic crown. The pulp system is located in the long axis of the tooth. Lack of attention to the degree of axial inclination of a tooth in relation to adjacent teeth and to alveolar bone may result in either gouging or perforation of the crown or the root at various levels (Figure 18-1). After establishing the proper access outline form, failure to direct the bur parallel to the long axis of a tooth will cause gouging or perforation of the root. This problem often occurs when the dentist must use the reflected image from an intraoral mirror to make the access preparation. In these situations, the natural tendency is to direct the bur away from the long axis of the root to improve vision through the mirror. Failure to check the orientation of the access opening during preparation may result in a perforation. The dentist should stop periodically to review the bur-tooth relationship. Aids for evaluating progress include transillumination, magnification, and radiographs.

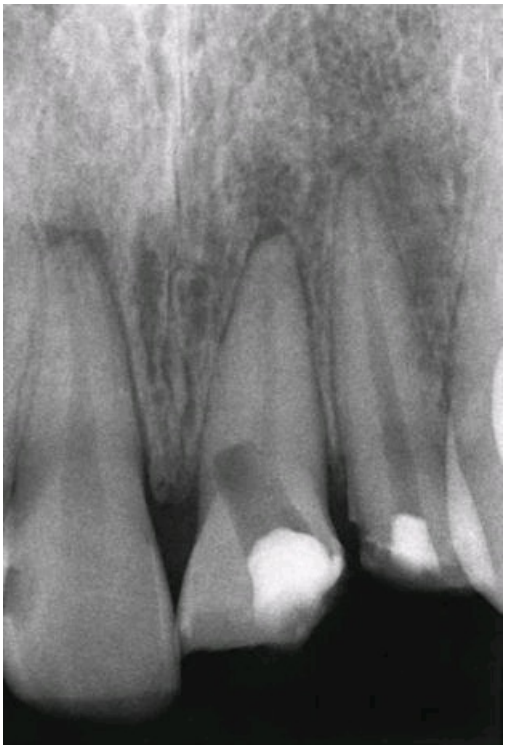

Figure 18-1 A misdirected bur created severe gouging and near-perforation during an otherwise routine access cavity preparation.

Searching for the pulp chamber or orifices of canals through an underprepared access cavity may also result in accidents (Figure 18-2). Failing to recognize when the bur passes through a small or flattened (disklike) pulp chamber in a multirooted tooth may also result in gouging or perforation of the furcation (Figure 18-3).

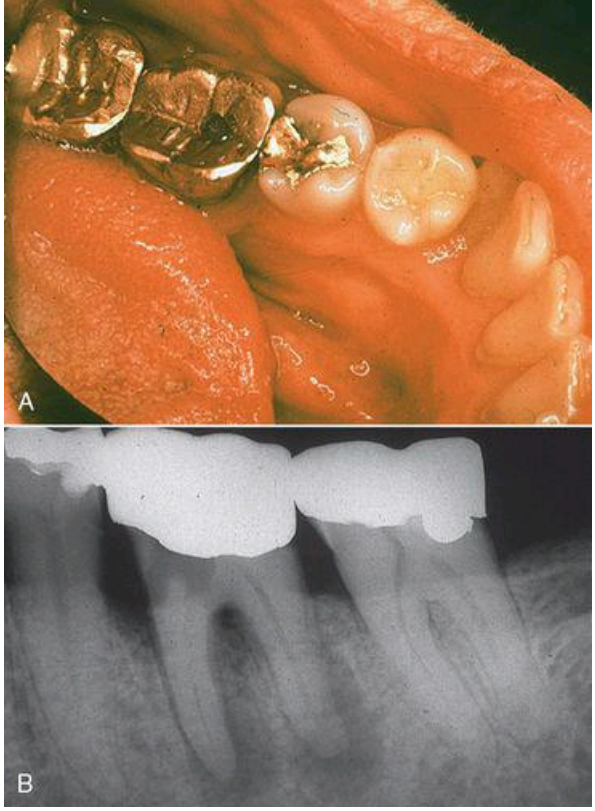

Figure 18-2 A, Inadequate access cavities not only result in compromised preparation and obturation but also may cause procedural accidents such as chamber perforation, canal ledging, and **(B)** root perforation.

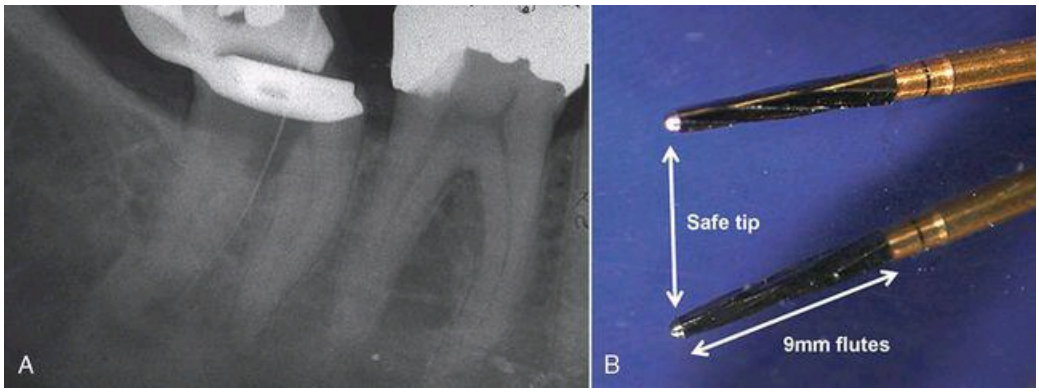

Figure 18-3 A, Failure to recognize when the bur passes through the roof of the pulp chamber in a calcified pulp chamber may result in gouging or perforation of the furcation. The use of apex locators and angled radiographs is necessary for early perforation detection. Early detection reduces damage and improves repair. **B,** Use of a "safe-ended" access bur will prevent perforation of the chamber floor.

A cast crown often is not aligned in the long axis of the tooth; directing the bur along the misaligned casting may result in a coronal or radicular perforation.

Prevention

Clinical Examination

Thorough knowledge of tooth morphology, including both surface and internal anatomy and their relationships, is mandatory to prevent pulp chamber perforations. Next, location and angulation of the tooth must be related to adjacent teeth and alveolar bone to avoid a misaligned access preparation. In addition, radiographs of teeth from different angles provide information about the size and extent of the pulp chamber and the presence of internal changes such as calcification or resorption. *The radiograph is a 2-dimensional projection of a 3-dimensional object*. Varying the horizontal exposure angle will provide at least a distorted view of the third dimension and may be helpful in supplying additional anatomic information.

Operative Procedures

Use of a rubber dam during root canal treatment is usually indicated.[1-3] However, in situations in which problems are anticipated in locating pulp chambers (e.g., tilted teeth, misoriented castings, or calcified chambers), initiating access without a rubber dam is preferred[4] because it allows better crown-root alignment. However, when access is made without rubber dam placement, no intracanal instruments, such as files, reamers, or broaches, should be used unless they are secured by a piece of floss[4] and a throat pack is placed. Constricted chambers or canals must be sought patiently, with small amounts of dentin removed at a time.

Failure to recognize when the bur passes through the roof of the pulp chamber if the chamber is calcified may result in gouging or perforation of the furcation. After penetration of the roof of the chamber, using a “safe-ended” access bur, such as the Endo Z (Dentsply/Maillefer, Tulsa, OK) or a pulp shaper bur (Dentsply/Tulsa Dental, Tulsa, OK), will prevent perforation of the chamber floor (see Figure 18-3, *B*).

The use of apex locators and angled radiographs is necessary for early perforation detection. Early detection reduces damage caused by continued treatment (irrigation, cleaning and shaping) and improves the prognosis for nonsurgical repair.

Another useful method of providing isolation and also visualizing the crown-root alignment is the use of a “split” dam (Figure 18-4). This dam can be applied in the anterior region without a rubber dam clamp (see Chapter 15) or in posterior regions by quadrant isolation if a distal tooth can be clamped. Also, elimination of the metal clamp from the field of operation allows radiographic orientation of coronal access preparation.

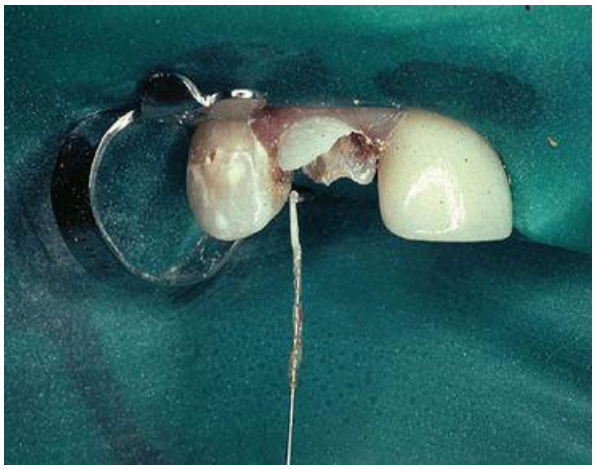

Figure 18-4 Rubber dam can be applied in the anterior region without placing the clamp on the tooth that is undergoing root canal therapy or in posterior regions by quadrant isolation if a distal tooth can be clamped.

To orient the access, a bur may be placed in the preparation hole (secured with cotton pellets) and then radiographed (Figure 18-5). This provides information about depth of access in relation to canal location. Remember, a single canal is located in the center of the root. A direct facial radiograph will show the mesiodistal relationship; a mesial- or distal-angled film will show the faciolingual location. This procedure is helpful for locating small canals.

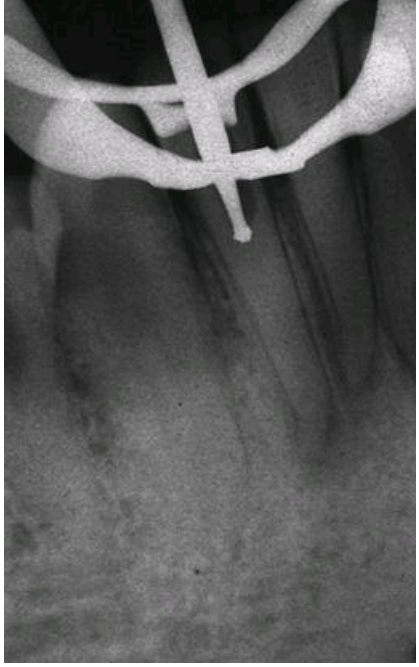

Figure 18-5 A small bur is placed during access preparation when orientation is a problem. This provides information about such data as angulation and depth of bur penetration.

Use of a fiberoptic light during access preparation may assist in locating canals. This strong light illuminates the cavity when the beam is directed through the access opening (reflected light) and illuminates the pulp chamber floor (transmitted light). In the latter case, a canal orifice appears as a dark spot. Using magnifying glasses or an operative microscope[5-7] will also aid in locating a small orifice. Magnification loupes (2.5 or greater) are useful especially when combined with transillumination. The ultimate aid in canal location is the operating microscope. Patients with problems requiring significant magnification for canal location should be referred to an endodontist who has this specialized equipment.

Perforation into the periodontal ligament (PDL) or bone usually (but not always) results in immediate and continuous hemorrhage. The canal or chamber is difficult to dry, and placement of a paper point or cotton pellet may increase or renew the bleeding. Bone is relatively avascular compared with soft tissue. Mechanical perforation may occasionally initially produce only hemorrhage equal to that of pulp tissue.

Perforations must be recognized early to avoid subsequent damage to the periodontal tissues with intracanal instruments and irrigants. Early signs of perforation may include one or more of the following: (1) sudden pain during the working length determination when local anesthesia was adequate during access preparation; (2) sudden appearance of hemorrhage; (3) burning pain or a bad taste during irrigation with sodium hypochlorite; or (4) other signs, including a radiographically malpositioned file (see Figure 18-3) or a PDL reading from an apex locator that is far short of the working length on an initial file entry.

Unusually severe postoperative pain may result from cleaning and shaping procedures performed through an undetected perforation. At a subsequent appointment, the perforation site will be hemorrhagic because of the inflammation of the surrounding tissues. The overall prognosis of the tooth must be evaluated with respect to the strategic value of the tooth, the location and size of the defect, and the potential for repair.

Perforation into the PDL at any location will have a negative effect on long-term prognosis. The dentist must inform the patient of the questionable prognosis[1] and closely monitor the long-term periodontal response to any treatment. In addition, the patient must know what signs or symptoms indicate failure and if failure occurs, what the subsequent treatment will be.

Perforations during access cavity preparation present a variety of problems. When a perforation occurs or is strongly suspected, the patient should be considered for referral to an endodontist. In general, a specialist is better equipped to manage these patients (Figure 18-6). Also, after long-term evaluation, other procedures, such as surgery, may be necessary if future failure occurs.

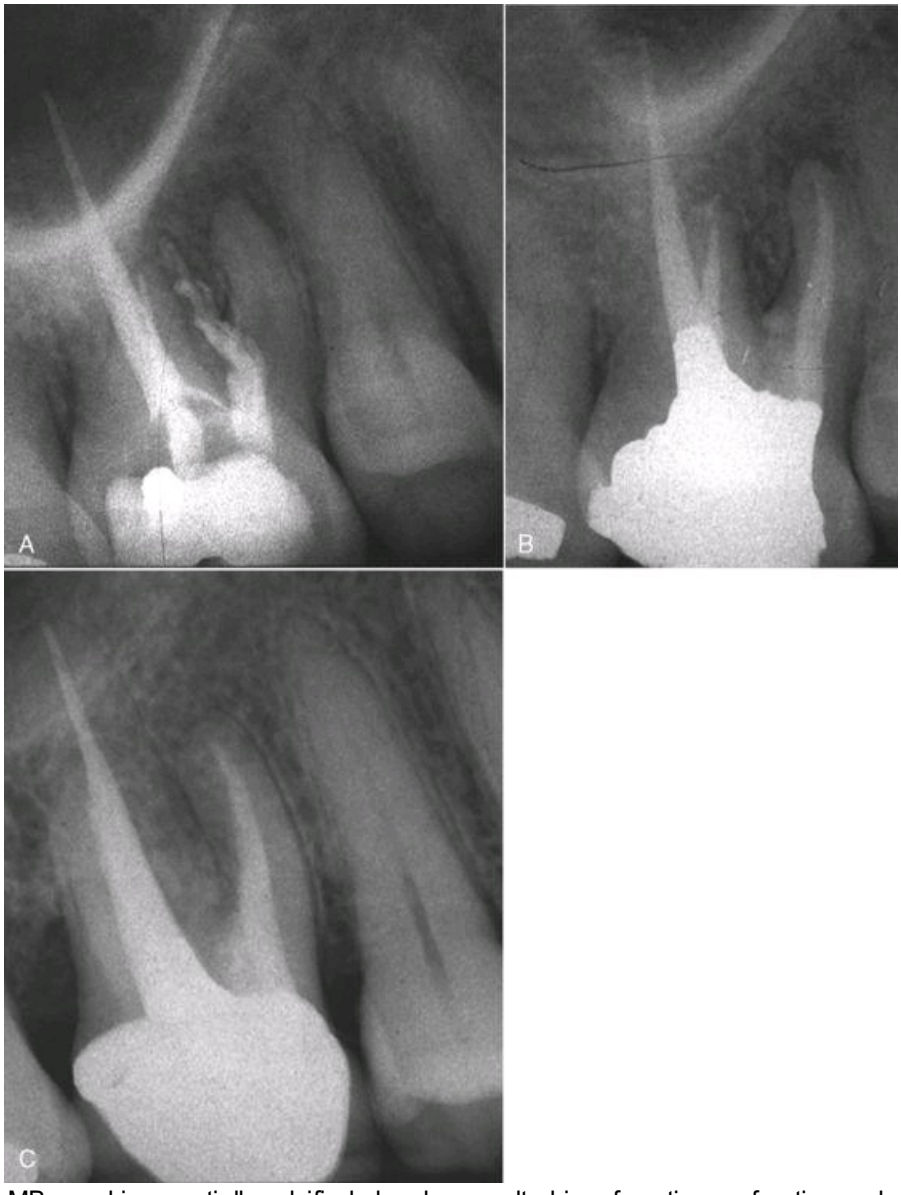

Figure 18-6 A, A search for the MB canal in a partially calcified chamber resulted in a furcation perforation and extrusion of filling materials into the periapical tissues. An apex locator reading or an angled radiograph would have detected this type of error. **B,** The initial treatment was redone

and the perforation was sealed with MTA. **C,** Radiograph 3 years later shows no evidence of pathosis in the repaired area.

(Courtesy Dr. George Bogen.)

Lateral Root Perforation

The location and size of the perforation during access are important factors in a lateral perforation. If the defect is located at or above the height of crestal bone, the prognosis for perforation repair is favorable.[8,9] These defects can be easily "exteriorized" and repaired with standard restorative material such as amalgam, glass ionomer, or composite. Periodontal curettage or a flap procedure is occasionally required to place, remove, or smooth excess repair material. In some cases, the best repair is placement of a full crown with the margin extended apically to cover the defect.

Teeth with perforations below the crestal bone in the coronal third of the root generally have the poorest prognosis. Attachment often recedes and a periodontal pocket forms, with attachment loss extending apically to at least the depth of the defect. The treatment goal is to position the apical portion of the defect above crestal bone. Orthodontic root extrusion is generally the procedure of choice for teeth in the esthetic zone.[10-12] Crown lengthening may be considered when the esthetic result will not be compromised or when adjacent teeth require surgical periodontal therapy. Internal repair of these perforations by mineral trioxide aggregate (MTA) has been shown to provide an excellent seal as compared to other materials.[13]

Furcation Perforation

A perforation of the furcation is generally one of two types: the "direct" or the "stripping" type. Each is created and managed differently and the prognoses vary. The *direct perforation* usually occurs during a search for a canal orifice. It is more of a "punched-out" defect into the furcation with a bur and is usually accessible, may be small, and may have walls. This type of perforation should be immediately (if possible) repaired with MTA (Figure 18-7). If proper conditions exist (dryness), glass ionomer or composite can be used to seal the defect. Prognosis is usually good if the defect is sealed immediately.

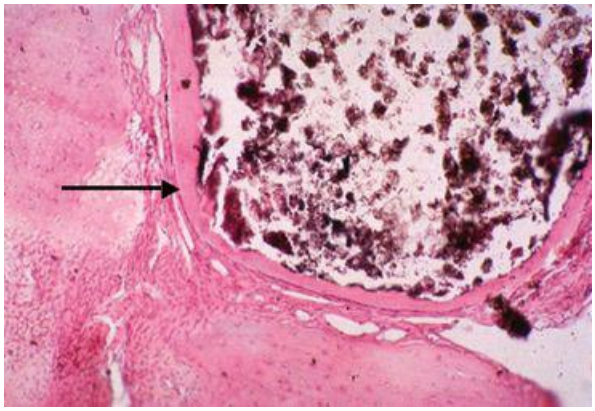

Figure 18-7 Immediate repair of a perforation in the furcation of a dog premolar with MTA results in the formation of cementum *(arrow)* adjacent to the material.

A *stripping perforation* involves the furcation side of the coronal root surface and results from excessive flaring with files or drills. Whereas direct perforations are usually accessible and therefore can be repaired nonsurgically, stripping perforations are generally inaccessible, requiring more elaborate approaches. The usual consequences of untreated stripping perforations are inflammation followed by development of a periodontal pocket. Long-term failure results from leakage of the repair material, which produces periodontal breakdown with attachment loss. Skillful use of MTA has significantly improved the prognosis of nonsurgical repair of stripping perforations compared with other repair materials (Figure 18-8).

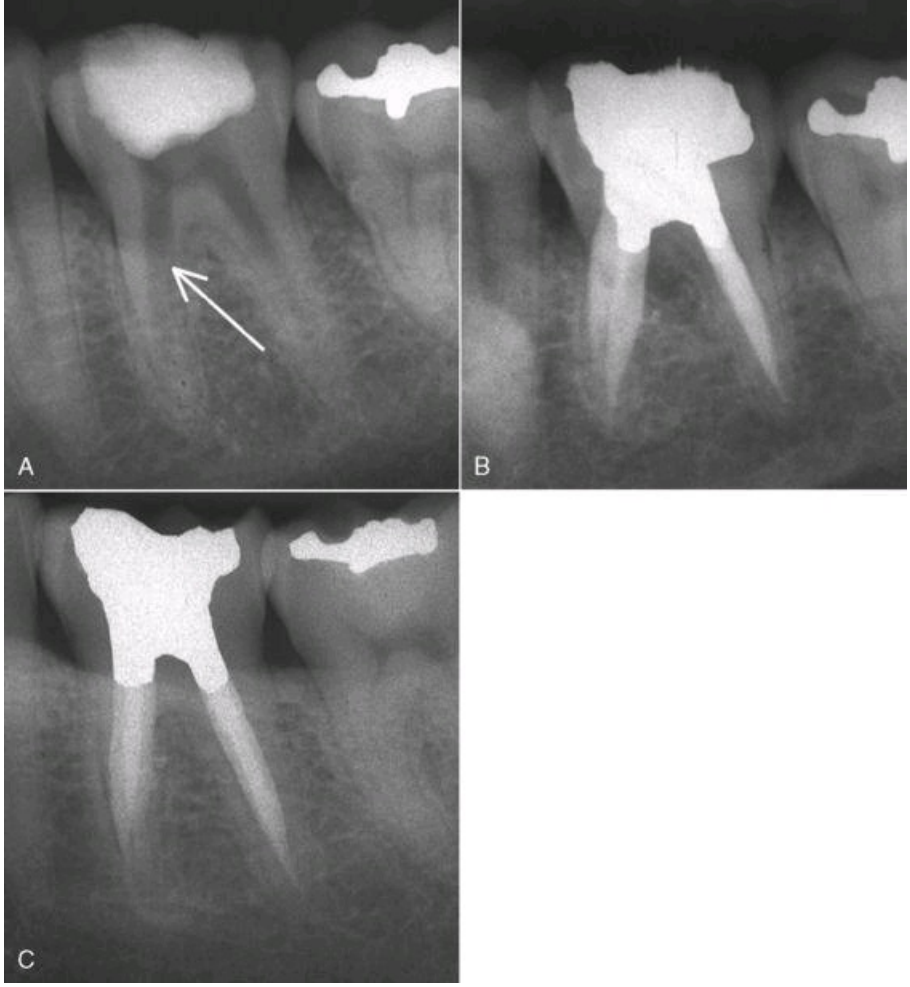

Figure 18-8 A, Radiograph shows stripping perforation *(arrow)* in the mesiobuccal root of the first mandibular molar. **B,** The mesial roots were filled with MTA and the distal root with gutta-percha and root canal sealer. **C,** A radiograph taken 1 year later shows no periradicular pathosis.

Nonsurgical Treatment

If feasible, nonsurgical repair (Figure 18-9) of furcation perforations is preferred over surgical intervention.[4] Traditionally, materials, such as amalgam, gutta-percha, zinc oxide–eugenol, Cavit, calcium hydroxide, freeze-dried bone, and indium foil, have been used clinically and experimentally to seal these defects.[14-23] Repair is difficult because of potential problems with visibility, hemorrhage control, and management and sealing ability of the repair materials. In general, perforations occurring during access preparation should be sealed immediately, but the patency of the canals must be protected. Immediate repair of the perforations with MTA offers the best results for perforation repair.[22-27]

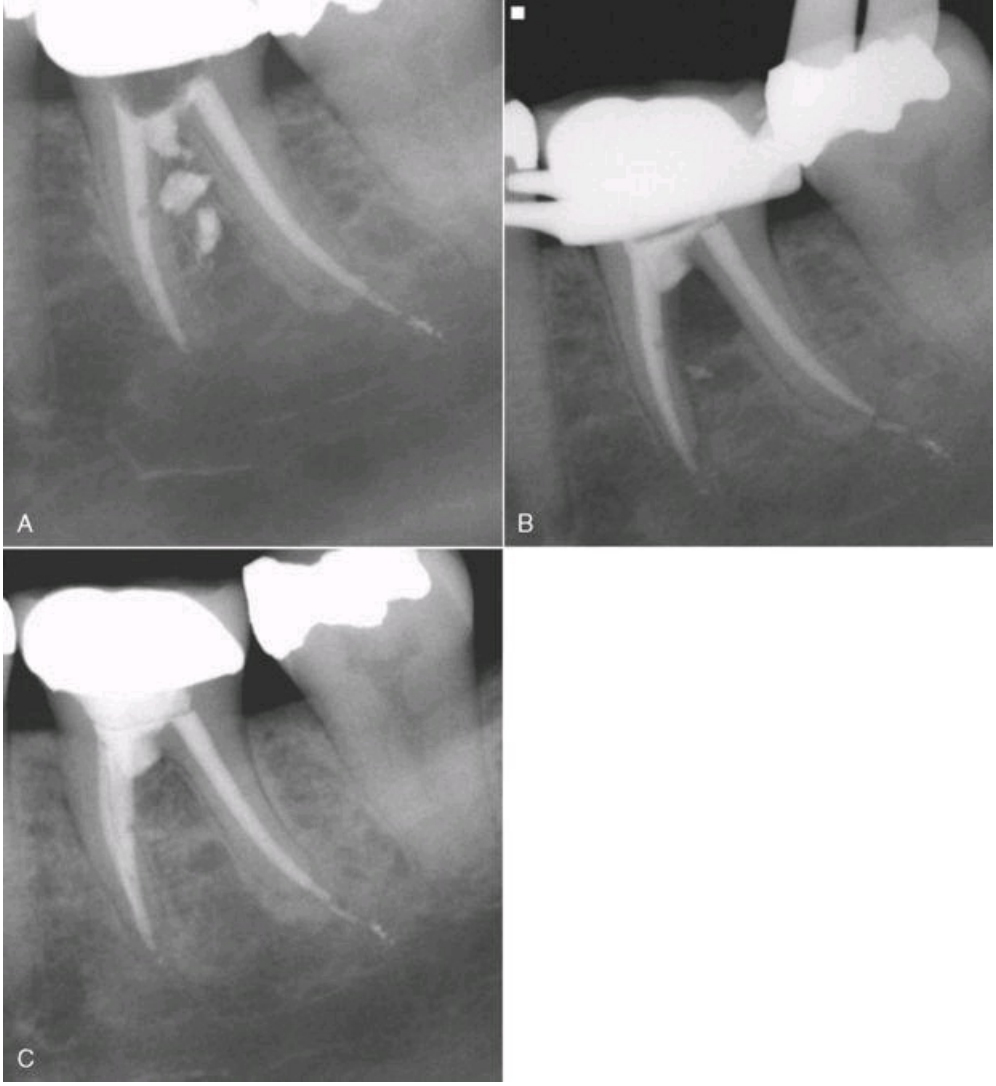

Figure 18-9 A, Periapical radiograph shows presence of a furcation perforation in the first mandibular molar. **B,** The root canal was retreated and the perforation was repaired with MTA. **C,** Radiograph taken 12 months later shows no evidence of furcal pathosis.

(Courtesy Dr. Kenneth Zucker.)

Surgical Treatment

Surgery requires more complex restorative procedures and more demanding oral hygiene from the patient.[9] Surgical alternatives are hemisection, bicuspidization, root amputation, and intentional replantation. Teeth with divergent roots and bone levels that allow preparation of adequate crown margins are suitable for either hemisection or bicuspidization. Intentional replantation (Figure 18-10) is indicated when the defect is inaccessible or when multiple problems exist, such as a perforation combined with a separated instrument, or when the prognosis with other surgical procedures is poor. Dentist and patient must recognize that the prognosis for treatment of surgically altered teeth is guarded because of the increased technical difficulty associated with restorative procedures and demanding oral hygiene requirements. The remaining roots are prone to caries, periodontal disease, and vertical root fracture. Treatment planning options, including extraction, should be discussed with the patient when the prognosis is poor.

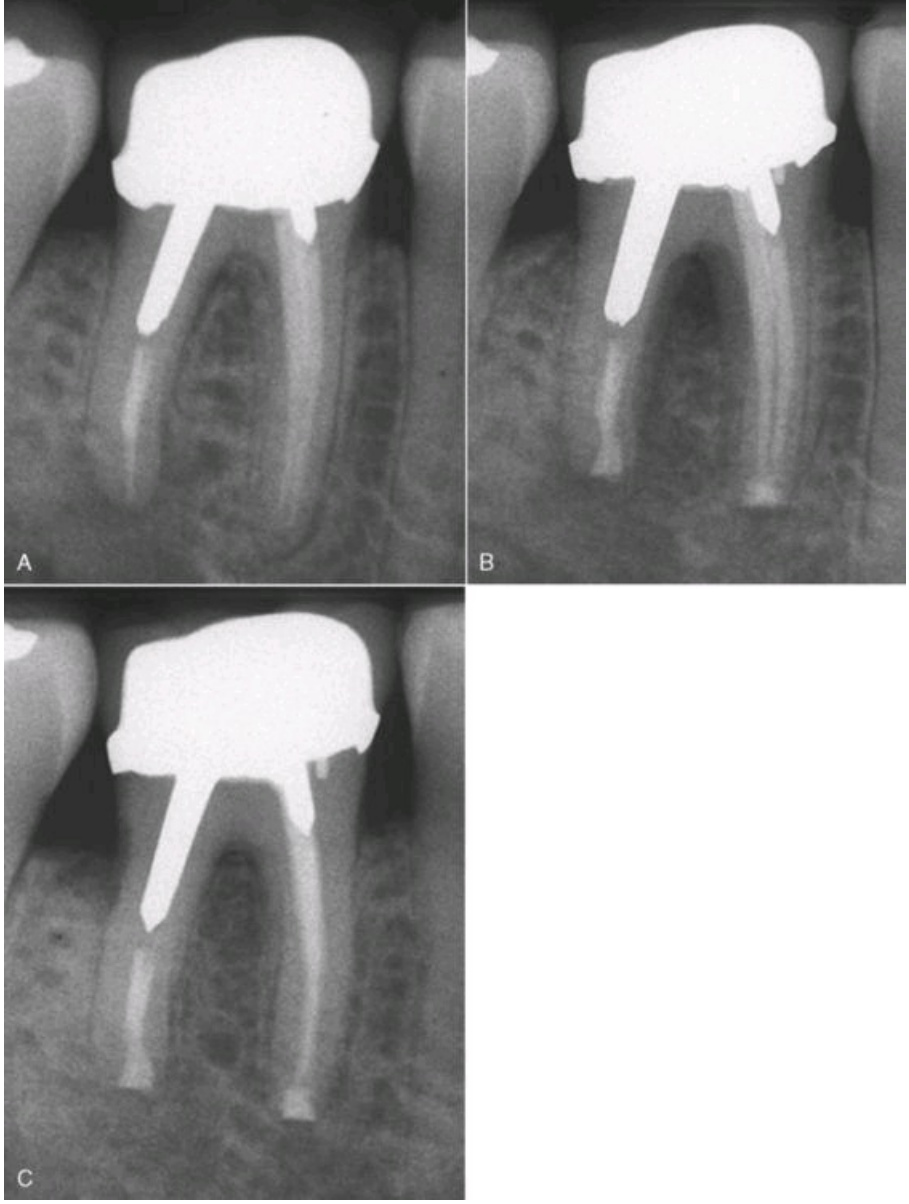

Figure 18-10 A, Postoperative radiograph 6 years after endodontic treatment. The patient is percussion sensitive, and periapical lesions are present. A 5-mm periodontal pocket exists on the distolingual root. A fracture is suspected, and extract-replant was performed for diagnostic reasons. The tooth was extracted, and no fracture was seen. **B,** The tooth is replanted after retrofilling with MTA. Cause of failure was probably leakage. **C,** Radiograph 1 year later showed osseous repair. The periodontal pocket healed.

Prognosis

Factors affecting the long-term prognosis of teeth after perforation repair include the location of the defect in relation to crestal bone, the length of the root trunk, the accessibility for repair, the size of the defect, the presence or absence of a periodontal communication to the defect, the time lapse between perforation and repair, the sealing ability of restorative material, and subjective factors such as the technical competence of the dentist and the attitude and oral hygiene of the patient.[4] Early recognition and repair will improve the prognosis by minimizing damage to the periodontal tissues by bacteria, files, and irrigants. Additionally, a small perforation (less than 1 mm) causes less tissue destruction and is more amenable to repair than a larger perforation. Electronic apex locators or angled radiographs with files in place aid in early detection.

An unrecognized or untreated perforation in the furcation usually results in a periodontal defect that communicates through the gingival sulcus within weeks or sometimes days. A preexisting periodontal communication caused by perforation worsens the prognosis; therefore the time between perforation and repair should be as short as possible.[22,28] Immediate sealing of the defect reduces the incidence of periodontal breakdown. To best determine the long-term prognosis, the dentist must monitor the patient's symptoms, radiographic changes, and most importantly, periodontal status. Radiographs and periodontal probing during recall examination are the best measures of success or failure of the repair procedure.

ACCIDENTS DURING CLEANING AND SHAPING

The most common procedural accidents during cleaning and shaping of the root canal system are ledge formation, artificial canal creation, root perforation, instrument separation, and extrusion of irrigating solution periapically. Correction of these accidents is usually difficult, and the patient should be referred to an endodontist.

Ledge Formation

By definition, a ledge has been created when the working length can no longer be negotiated and the original patency of the canal is lost. The major causes of ledge formation include (1) inadequate straight-line access into the canal, (2) inadequate irrigation or lubrication, (3) excessive enlargement of a curved canal with files, and (4) packing debris in the apical portion of the canal.

Prevention of a Ledge

Preoperative Evaluation

Prevention of ledging begins with examination of the preoperative radiograph for curvatures, length, and initial size.

Curvatures

Most important is the coronal third of the root canal. Severe coronal curvature predisposes the apical canal to ledging. Straight-line access to the orifice of the canal can be achieved during access preparation, but accessibility to the apical third of the canal is achieved only with coronal flaring. Severe apical curvatures require a proper sequence of cleaning and shaping procedures to maintain patency (see Chapter 16).

Length

Longer canals are more prone to ledging than shorter canals. Careful attention to maintaining patency is required to prevent ledging.

Initial Size

Smaller-diameter canals are more easily ledged than larger-diameter canals.

In summary, *the canals most prone to ledging are small, curved, and long.* Radiographs are 2-dimensional and cannot provide accurate information about the actual shape and curvature of the root canal system. All root canals have some degree of curvature, including faciolingual curves, which may not be apparent on straight facial exposures.

Technical Procedures

Determination of working length in the cleaning and shaping process is a continuation of the access preparation. Optimum straight-line access to the apical third is not achieved until cleaning and shaping have been completed. An accurate working length measurement is a requirement because cleaning and shaping short of the ideal length is a prelude to ledge formation. Frequent recapitulation and irrigation, along with the use of lubricants, are mandatory. Sodium hypochlorite may be used initially for hemorrhage control and removal of debris. However, this agent alone may not be adequate to provide maximum lubrication.

Silicone, glycerine, and wax-based lubricants are commercially available for canal lubrication. Because these materials are viscous, they are carried into the apical regions of the canal with the file. Enhanced lubrication permits easier file insertion, reduces stress to the file, and assists with removal of debris. The lubricant is easily removed with sodium hypochlorite irrigation. Flexible files (nickel-titanium) reduce the chances for ledge formation.

A one-eighth to one-fourth reaming motion with the files should be used in the apical third. A filing motion directed away from the furcation is used to form the funnel shape of the canal and reduce the coronal curvature. Each file must be worked until it is loose before a larger size is used.

Canals with a severe coronal curvature require a passive step-back cleaning and shaping technique (see Chapter 16). A No. 15 file is used at working length. With maximum irrigation or lubrication, the canal is passively and progressively flared in a step-back fashion. The No. 15 file is recapitulated many times to maintain patency. This preflaring technique reduces the coronal curvature and enlarges the canal. Better control of the files is gained for enlarging and cleaning the apical third of the canal as the last step (see section on Apical Clearing in Chapter 16). Using this technique, the chances of ledge formation are reduced. Rotary files with increased taper will blend and join the shape into a tapering funnel.

Management of a Ledge

Once created, a ledge is difficult to correct. An initial attempt should be made to bypass the ledge with a No. 10 steel file to regain working length. The file tip (2 to 3 mm) is sharply bent and worked in the canal in the direction of the canal curvature. Lubricants are helpful. A "picking" motion is used to attempt to feel the catch of the original canal space, which is slightly short of the apical extent of the ledge. If the original canal is located, the file is then worked with a reaming motion and occasionally an up-and-down movement to maintain the space and remove debris (Figure 18-11), although this may be only partially successful. Once a ledge is created, even if it is initially bypassed, instruments and obturating materials tend to be continually directed into the ledge.

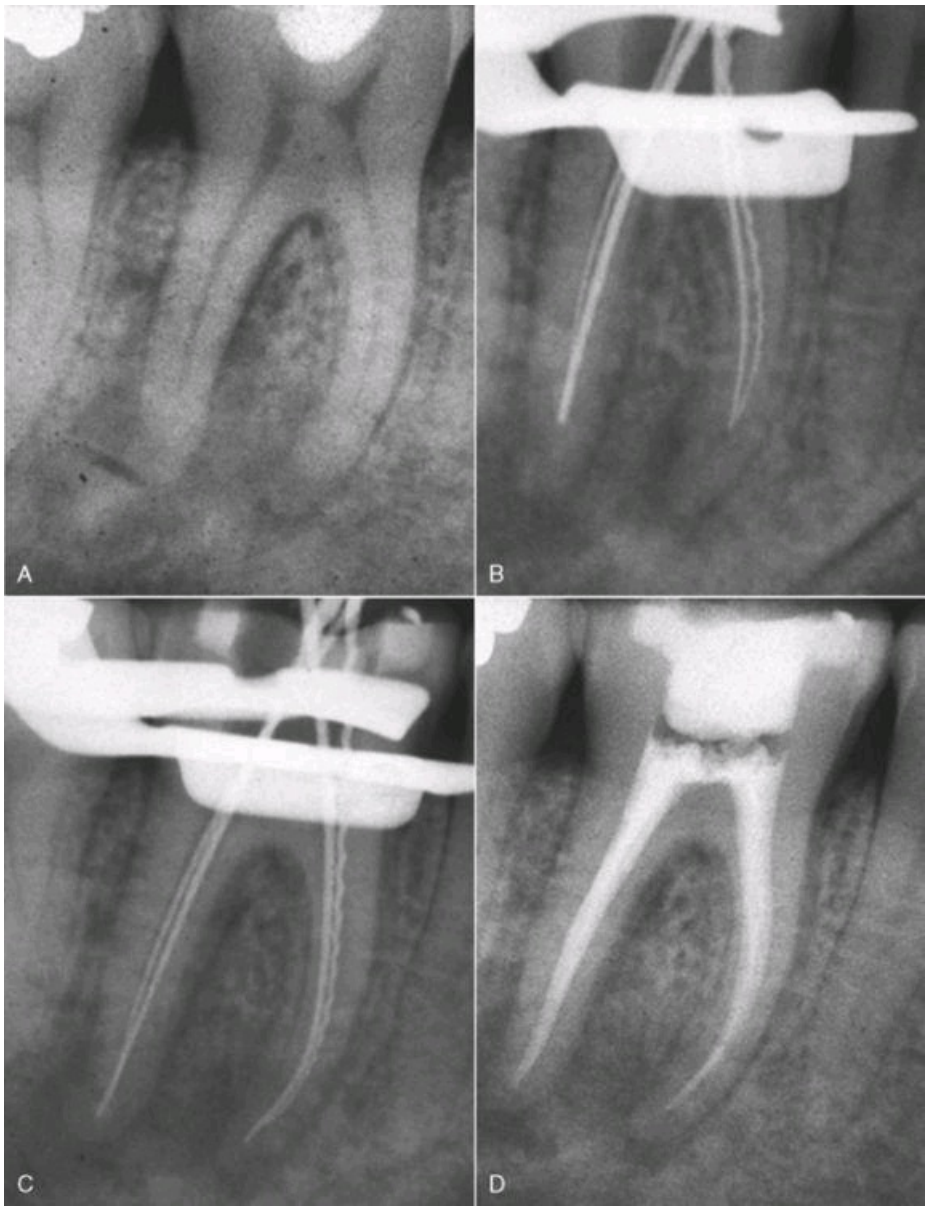

Figure 18-11 A, Preoperative radiograph. **B,** Ledges have been formed in the mesial and distal canals with steel files. Ledges can be bypassed only with small, curved steel files. **C,** Ledges are bypassed, and proper length established. **D,** A final radiograph shows complete obturation of root canals.

If the original canal cannot be located by this method, cleaning and shaping of the existing canal space is completed at the new working length. At times, flaring of the canal may allow the ledge to be bypassed by providing improved access to the apical canal. Small, curved files are used in the manner previously described in a final attempt to bypass the ledge. If this is successful, the apical canal space must be sequentially cleaned and flared to an appropriate size. Complete removal or reduction of the ledge facilitates obturation.

Prognosis

Failure of root canal treatment associated with ledging depends on the amount of debris left in the uninstrumented and unfilled portion of the canal. The amount depends on when ledge formation occurred during the cleaning and shaping process. In general, short and cleaned apical ledges have good prognoses. The patient must be informed of the prognosis, the importance of the recall examination, and what signs indicate failure. Future appearance of clinical symptoms or radiographic evidence of

failure may require referral for apical surgery or retreatment.

Creating an Artificial Canal

Cause and Prevention

Deviation from the original pathway of the root canal system and creation of an artificial canal cause an exaggerated ledge; it is initiated by the factors that cause ledge formation. Therefore the recommendations for preventing ledge formation should be followed to avoid creating artificial canals. The unfortunate sequence is as follows. A ledge is created and the proper working length is lost. The operator, eager to regain that length, “bores” apically with each file, thus creating an artificial canal. Used persistently, the file eventually perforates the root surface. Aggressive use of steel files is the most common cause of this problem.

Management

Negotiating the original canal that has an exaggerated ledge is normally very difficult. Rarely can the original canal be located, renegotiated, and prepared. To obturate, the dentist should determine whether a perforation exists. Methods include apex locator readings, hemorrhage on paper points while drying, and radiographs with a file in position. If confirmed, the working length is adjusted, an apical stop is created at the adjusted length with larger files, and obturation is begun. If there is no perforation, the canal is obturated with a warm or softened gutta-percha technique in conjunction with a root canal sealer. If there is a perforation, the defect should be repaired internally or surgically (see section on Root Perforations).

Prognosis

Prognosis depends on the ability of the operator to renegotiate the original canal and the remaining uninstrumented and unfilled portion of the main canal. Unless a perforation exists, teeth in which the original canal can be renegotiated and obturated have a prognosis similar to those without procedure complications. In contrast, when a large portion of the main canal is uninstrumented and unobturated, a poorer prognosis exists, and the tooth must be examined periodically. Failure usually means surgery will be required to resect the uninstrumented and unobturated root canal.

Root Perforations

Roots may be perforated at different levels during cleaning and shaping. Location (apical, middle, or cervical) of the perforation and the stage of treatment affect prognosis.[17,28] The periodontal response to the injury is affected by the level and size of the perforation. Perforation in the early stages of cleaning and shaping affects prognosis significantly.

Apical Perforations

Apical perforations occur through the apical foramen (overinstrumentation) or through the body of the root (perforated new canal).

Etiology and Indicators

Instrumentation of the canal beyond the apical constriction results in perforation. Incorrect working length or inability to maintain proper working length causes "zipping"[4] or "blowing out" of the apical foramen. The appearance of fresh hemorrhage in the canal or on instruments, pain during canal preparation in a previously asymptomatic tooth, and sudden loss of the apical stop are indicators of foramen perforation. Extension of the largest (final) file beyond the radiographic apex is also a sign. An electronic apex locator may also confirm this procedural accident.

Prevention

To prevent apical perforation, proper working lengths must be established and maintained throughout the procedure. In curved canals, the flexibility of files with respect to size must be considered. Cleaning and shaping procedures straighten the canal somewhat and effectively decrease the working length by as much as 1 to 2 mm, requiring compensation. To prevent apical perforation, the working length should be verified with an apex locator after completion of cleaning and shaping steps.

Treatment

Treatment includes establishing a new working length, creating an apical seat (taper), and obturating the canal to its new length. Depending on the size and location of the apical foramen, a new working length 1 to 2 mm short of the point of perforation should be established. The canal is then cleaned, shaped, and obturated to the new working length. The master cone must have a positive apical stop at the working length before obturation. Placement of MTA as an apical barrier can prevent extrusion of obturation materials.

Prognosis

Success of treatment depends primarily on the size and shape of the defect. An open apex or reverse funnel is difficult to seal and also allows extrusion of the filling materials. In addition, the feasibility of repairing the perforation surgically may influence the final outcome.

Lateral (Midroot) Perforations

Etiology and Indicators

As discussed earlier, inability to maintain canal curvature is the major cause of ledge formation. Negotiation of ledged canals is not always possible, and misdirected pressure and force applied to a file may result in formation of an artificial canal and eventually in an apical or midroot perforation. To avoid these perforations, the same factors mentioned earlier for prevention of ledge formation should be considered: (1) degree of canal curvature and size and (2) inflexibility of the larger files, especially stainless steel files.

Indicators of lateral perforation are similar to those of apical perforation (i.e., fresh hemorrhage in the root canal or sudden pain and deviation of instruments from their original course). Penetration of the instrument out of the root radiographically (or as indicated by an apex locator) is the ultimate indicator.

Treatment

The optimal goal is to clean, shape, and obturate the entire root canal system of the affected tooth. After the perforation is confirmed, the steps discussed previously for bypassing of ledged canals are followed. If attempts to negotiate the apical portion of the canal are unsuccessful, the operator should concentrate on cleaning, shaping, and obturating the coronal segment of the canal. A new working length confined to the root is established, and the canal is then cleaned, shaped, and obturated to the new working length. A low concentration (0.5%) of sodium hypochlorite or saline should be used for irrigation in a perforated canal. Extrusion of concentrated irrigant into the surrounding periodontal tissues would produce severe inflammation.

Prognosis

Success depends partially on the remaining amount of undébrided and unobturated canal. Obturation is difficult because of lack of a stop (matrix), and gutta-percha tends to be extruded during condensation. Teeth with perforations close to the apex after complete or partial débridement of the canal have a better prognosis than those with perforations that occur earlier. In addition to the length of uncleaned and unfilled portions of the canal, size and surgical accessibility of perforations are important. In general, small perforations are easier to seal than large ones. Because of surgical accessibility, perforations toward the facial aspect are more easily repaired and therefore these teeth have a better prognosis than those with perforations in other areas.

On recall, both radiographic and periodontal examinations for signs and symptoms are performed. Failure generally requires surgery or other approaches. These approaches depend on the severity of perforation, the strategic importance of the tooth, and the location and accessibility of the perforation. Corrective techniques include repair of the perforation site, root resection to the level of the perforation, root amputation, hemisection, replantation, and extraction.

Coronal Root Perforations

Etiology and Indicators

Coronal root perforations occur during access preparation as the operator attempts to locate canal orifices or during flaring procedures with files, Gates-Glidden drills, or Peeso reamers. Using the methods described earlier in this chapter can minimize perforations during access preparation. Removal of restorations when possible, use of fiberoptic lights for illumination, magnification, and cautious exploration for calcified canals can prevent most problems during access preparation. Careful flaring (step-back) and conservative use of flaring instruments are required during cleaning and shaping procedures.

Treatment and Prognosis

Repair of a stripping perforation in the coronal third of the root has the poorest long-term prognosis of any type of perforation.[9] The defect is usually inaccessible for adequate repair. An attempt should be made to seal the defect internally, even though the prognosis is guarded. Patency of the canal system must be maintained during the repair process. Referral of the patient to a specialist is recommended.

Separated Instruments

Etiology

Limited flexibility and strength of intracanal instruments combined with improper use may result in an intracanal instrument separation. Any instrument may break'steel, nickel-titanium, hand, or rotary. Overuse or excessive force applied to files is the main cause of separation. Manufacturing defects in files are rare.

Recognition

Removal of a shortened file with a blunt tip from a canal and subsequent loss of patency to the original length are the main clues for the presence of a separated instrument. A radiograph is *essential* for confirmation. It is *imperative* that the patient be informed of the accident and its effect on prognosis.[1] As with other procedural accidents, detailed documentation is also necessary for medical-legal considerations.

Prevention

Recognition of the physical properties and stress limitations of files is critical. Continual lubrication with either irrigating solution or lubricants is required. Each instrument is examined before use. If an unwound or twisted file is rotated and viewed, reflections from the chairside light will magnify fluting distortions (Figure 18-12). Small files must be replaced often. To minimize binding, each file size is worked in the canal until it is very loose before the next file size is used.[29] Nickel-titanium files usually do not show visual signs of fatigue similar to the "untwisting" of steel files. Many factors may affect the fatiguing of nickel-titanium files,[30] and they should be discarded before visual signs of untwisting are seen. Preflaring of preparations using passive step-back before the use of rotary instruments reduces the rates of separation of 0.04 taper nickel-titanium rotary instruments.[31]

Figure 18-12 Each steel file should be inspected for fluting distortion before use in the canal. Only untwisted files will show a shiny spot *(arrow).* This file must be discarded. Nickel-titanium files will not show this distortion and must be discarded after three to six uses.

Treatment

There are basically three approaches: (1) attempt to remove the instrument,[32] (2) attempt to bypass it, or (3) prepare and obturate to the segment. Initial treatment is similar to that discussed earlier for a ledge. Using a small file and following the guidelines described for negotiating a ledge, the operator should attempt to bypass the separated instrument. After bypassing the separated instrument, ultrasonic files,[33] broaches, or Hedstrom files are used to remove the segment (Figure 18-13). If removal of the separated piece is unsuccessful, then the canal is cleaned, shaped, and obturated to its new working length. If

the instrument cannot be bypassed, preparation and obturation should be performed to the coronal level of the fragment.

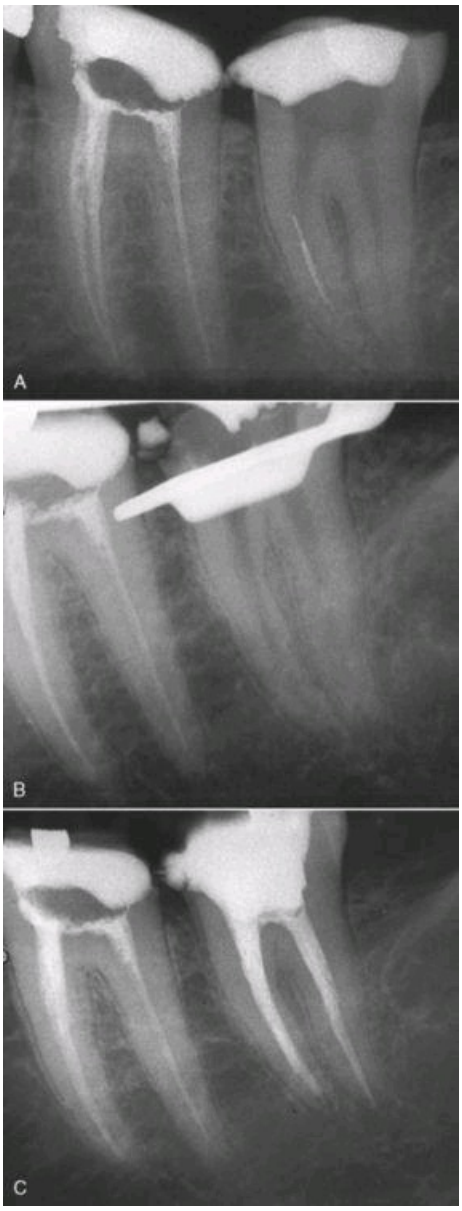

Figure 18-13 A, A file is separated in the mesiobuccal canal of the second mandibular molar. **B,** The separated instrument is bypassed and removed. **C,** Both canals are cleaned, shaped, and obturated. Prognosis is good.

Prognosis

Prognosis depends on how much undébrided and unobturated canal apical to and including the instrument remains. The prognosis is best when separation of a large instrument occurs in the later stages of preparation close to the working length. Prognosis is poorer for teeth with undébrided canals in which a small instrument is separated short of the apex or beyond the apical foramen early in preparation. For medical-legal reasons, the patient must be informed (with documentation in the record) of an instrument separation. Despite the concern of both patient and dentist,[34] clinical reports indicate that the prognosis in most procedures involving broken instruments that are managed properly is favorable.[35]

If the patient remains symptomatic or there is a subsequent failure, the tooth can be treated surgically. Accessible roots are resected with placement of a root-end filling material (Figure 18-14). Accessibility of the root apex for surgical intervention is critical to the final outcome.

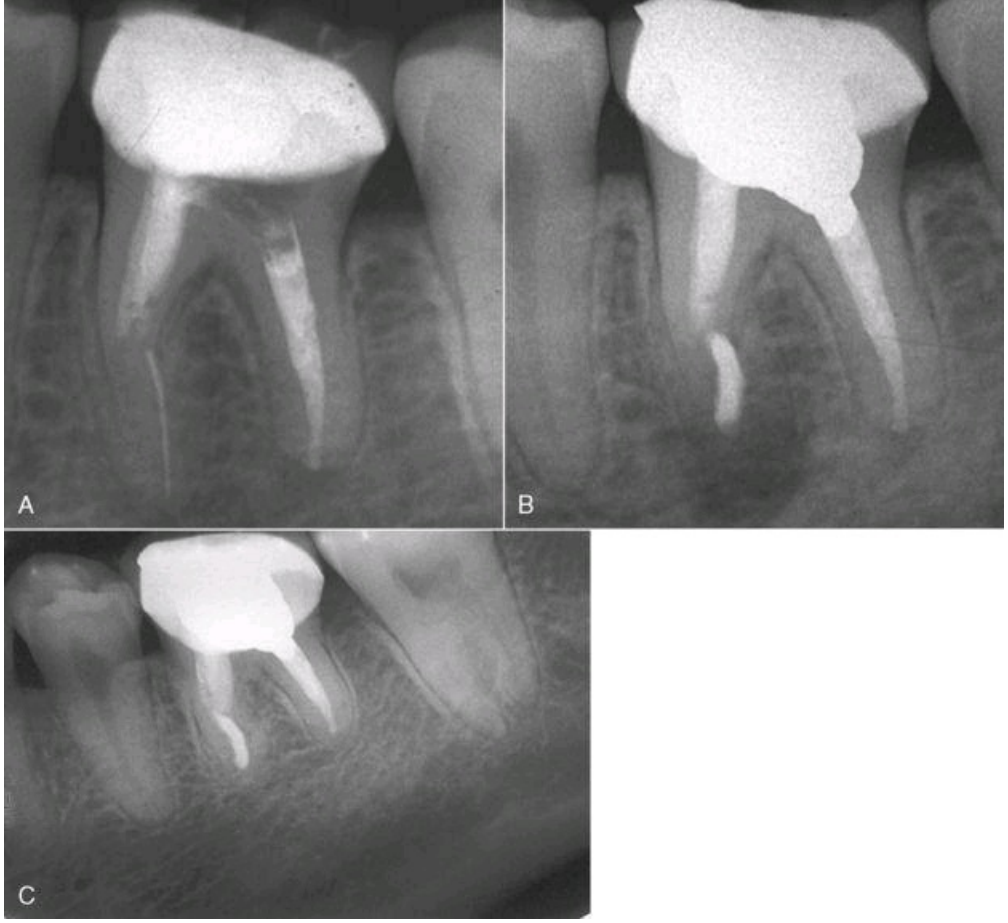

Figure 18-14 A, Nickel-titanium file was broken inside the mesiobuccal canal of the mandibular first molar. **B,** Because of patient discomfort, the segment was removed surgically and MTA was used as root-end filling material. **C,** A periapical radiograph 32 months later shows complete healing.

Other Accidents

Aspiration or Ingestion

Aspiration or ingestion of instruments is a serious event but is easily avoided with proper precautions. *Use of the rubber dam is the standard of care to prevent such ingestion or aspiration* and subsequent lawsuits.[1]

The disappearance of an instrument that has slipped from the dentist's fingers followed by violent coughing or gagging by the patient and radiographic confirmation of a file in the alimentary tract or airway are the chief signs. These patients require immediate referral to a medical service for appropriate diagnosis and treatment. According to a survey by Grossman,[36] 87% of these instruments are swallowed and the rest are aspirated. Surgical removal is required for some swallowed (Figure 18-15) and nearly all aspirated instruments.

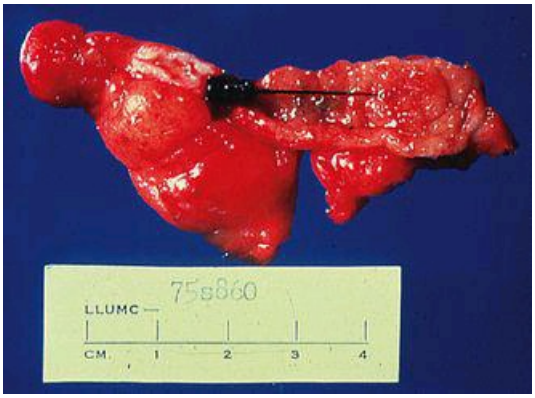

Figure 18-15 A swallowed broach caused removal of a patient's appendix and a subsequent lawsuit against a dentist who did not use a rubber dam during root canal therapy.

(Courtesy Dr. L. Thompsen.)

Extrusion of Irrigant

Wedging of a needle in the canal[37,38] (or particularly out of a perforation) with forceful expression of irrigant (usually sodium hypochlorite [NaOCl]) causes penetration of irrigants into the periradicular tissues and inflammation and discomfort for patients. Extrusion of NaOCl into the periapical tissues can cause a life-threatening emergency.[39] Loose placement of irrigation needles and careful irrigation with light pressure or use of a perforated needle[40] precludes forcing the irrigating solution into the periradicular tissues. Sudden prolonged and sharp pain during irrigation followed by rapid diffuse swelling (the "sodium hypochlorite accident") usually indicates penetration of solution into the periradicular tissues. The acute episode will subside spontaneously with time (Figure 18-16).

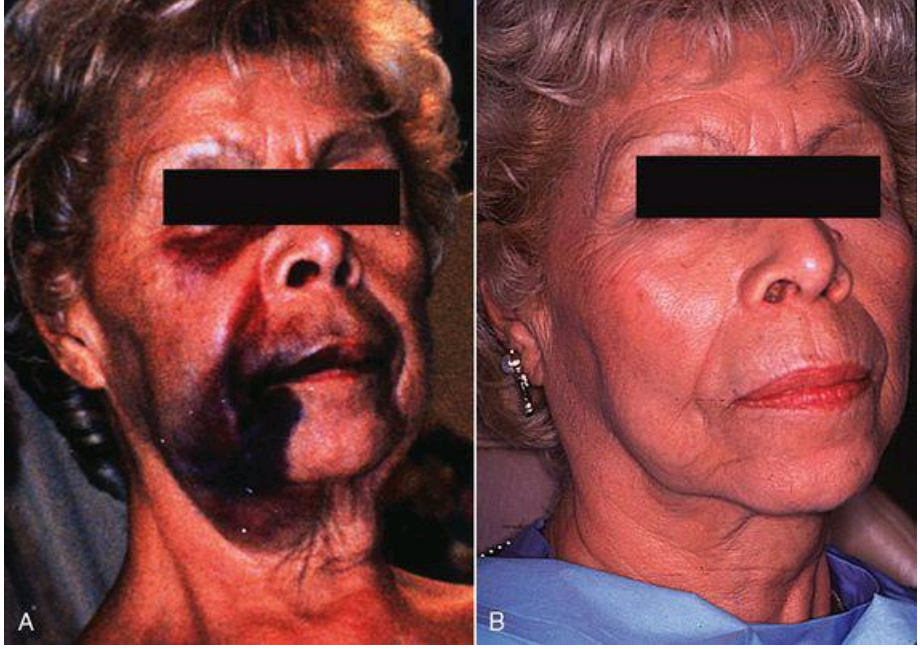

Figure 18-16 A, NaOCl was inadvertently expressed through an apical perforation in a maxillary cuspid during irrigation. Hemorrhagic reaction was rapid and diffuse. **B,** No treatment was necessary; the swelling and hematoma disappeared within a few weeks.

(Courtesy Dr. James Stick.)

Initially, there is no reason to prescribe antibiotics or attempt surgical drainage. Treatment is palliative. Analgesics are prescribed, and the patient is reassured. Because the outcome is so dramatic, evaluation is performed frequently to follow progress.

ACCIDENTS DURING OBTURATION

Appropriate cleaning and shaping are the keys to preventing obturation problems because these accidents usually result from improper canal preparation. In general, adequately prepared canals are obturated without mishap. *The quality of obturation reflects canal preparation*. However, problems do occur.

Underfilling

Etiology

Some causes of underfilling include a natural barrier in the canal, a ledge created during preparation, insufficient flaring, a poorly adapted master cone, and inadequate condensation pressure. Bypassing (if possible) any natural or artificial barrier to create a smooth funnel is one key to avoiding an underfill. The advent of nickel-titanium rotary files of increased taper has greatly improved the predictability of proper funnel and taper.

Treatment and Prognosis

Removal of underfilled gutta-percha and retreatment is preferred. Forcing gutta-percha apically by increased spreader or plugger pressure can fracture the root. If lateral condensation is the method of obturation, the master cone should be marked to indicate the working length. If displacement of the master cone during condensation is suspected, a radiograph is made *before* excess gutta-percha is removed. Removal can then be accomplished by pulling the cones in the reverse order of placement. Removal of gutta-percha in canals obturated with lateral condensation is easier than removal with other obturation techniques. However, warm gutta-percha techniques allow better obturation of irregularities within the canal.

Overfilling

Extruded obturation material causes tissue damage and inflammation. Postoperative discomfort (mastication sensitivity) usually lasts for a few days.

Etiology

Overfilling is usually the consequence of overinstrumentation through the apical constriction or lack of proper taper in prepared canals. When the apex is open naturally or its constriction is removed during cleaning and shaping, there is no matrix against which to condense; uncontrolled condensation forces extrusion of materials (Figure 18-17). Other causes include inflammatory resorption and incomplete development of the root.

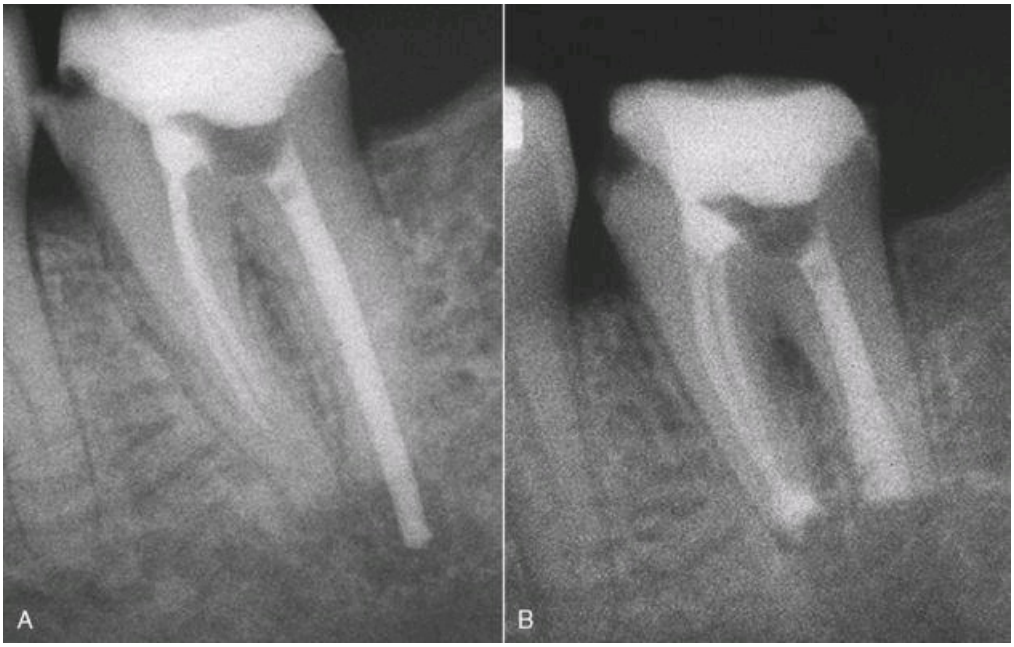

Figure 18-17 A, Lack of proper length measurements resulted in overfilling of the distal root and underfilling of the mesial root. The patient remained percussion sensitive. **B,** Surgical curettage, apical root resection, and root end filling with MTA were necessary to correct the technical deficiencies.

Prevention

To avoid overfilling, guidelines for preventing apical foramen perforation should be followed. Tapered preparation with an apical "matrix" usually prevents overfill. The largest file and master cone at working length should have a positive stop. A customized master cone may be fabricated by briefly applying solvent on the tip. If overfilling is suspected, a radiograph should be made before excess gutta-percha is removed. As with underfilling, the gutta-percha mass may be removed if the sealer has not set.

Treatment and Prognosis

When signs or symptoms of endodontic failure appear, apical surgery may be required to remove the material from apical tissues and place root-end filling material. Long-term prognosis is dictated by the quality of the apical seal, the amount and biocompatibility of extruded material, host response, and toxicity and sealing ability of the root-end filling material.

Vertical Root Fracture

Complete vertical root fracture causes untreatable failure. Aspects of vertical root fracture are described in more detail in Chapter 8.

Etiology

Causative factors include root canal treatment procedures and associated factors such as post placement. The main cause of vertical root fracture is post cementation, and the second in importance is overzealous application of condensation forces to obturate an underprepared or overprepared canal.[41]

Prevention

As related to root canal treatment procedures, the best means of preventing vertical root fractures are appropriate canal preparation and use of balanced pressure during obturation. A major reason for flaring canals is to provide space for condensation instruments. Finger spreaders produce less stress and distortion of the root than their hand counterparts.[42-44]

Indicators

Long-standing vertical root fractures are often associated with a narrow periodontal pocket or sinus tract stoma, as well as a lateral radiolucency (Figure 18-18) extending to the apical portion of the vertical fracture.[45] To confirm the diagnosis, a vertical fracture must be visualized. Exploratory surgery or removal of the restoration is usually necessary to visualize this mishap.

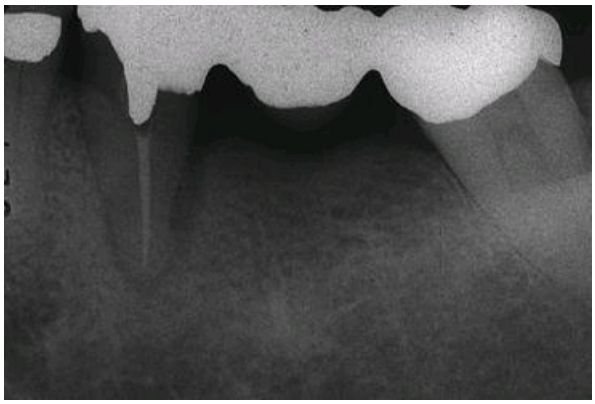

Figure 18-18 A, A "tear-drop" lateral radiolucency and a narrow probing defect extend to the apex of a tooth with vertical fracture.

Prognosis and Treatment

Complete vertical root fracture predicts the poorest prognosis of any procedural accident. Treatment is removal of the involved root in multirooted teeth and extraction of single-rooted teeth.

ACCIDENTS DURING POST SPACE PREPARATION

To prevent root perforation, gutta-percha may be removed to the desired level with heated pluggers or electronic heating devices, such as the “Touch N Heat” (SybronEndo, Orange, CA). This “pilot” post space provides a path of least resistance for sizing drills. Attempting to remove gutta-percha with a drill only can result in perforation. When a canal is prepared to receive a post, drills should be used sequentially, starting with a size that fits passively to the desired level. Miscalculation and incorrect preparation may result in perforation at any level. Knowledge of root anatomy is necessary for determining the size and depth of posts.

Indicators

The indicators of perforations and vertical root fractures are somewhat similar. Appearance of fresh blood during post space preparation is an indication for the presence of a root perforation. The presence of a sinus tract stoma or probing defects extending to the base of a post is often a sign of root fracture or perforation. Radiographs often show a lateral radiolucency along the root or perforation site.

Treatment and Prognosis

Prognosis for teeth with vertical root fractures resulting from post space preparation and post insertion is similar to that for teeth with fractures developing during obturation. The involved root (or tooth) is hopeless and must be removed. As outlined earlier, the prognosis of teeth with root perforation during post space preparation depends on the root size, location relative to epithelial attachment, and accessibility for repair. Management of the post perforation generally is surgical if the post cannot be removed. If the post can be removed, nonsurgical repair is preferred (Figure 18-19). Teeth with small root perforations that are located in the apical region and are accessible for surgical repair have a better prognosis than those that have large perforations, are close to the gingival sulcus, or are inaccessible. Because of the complexity in diagnosis, surgical techniques, and follow-up evaluation, patients with post perforations should be referred to an endodontist for evaluation and treatment.

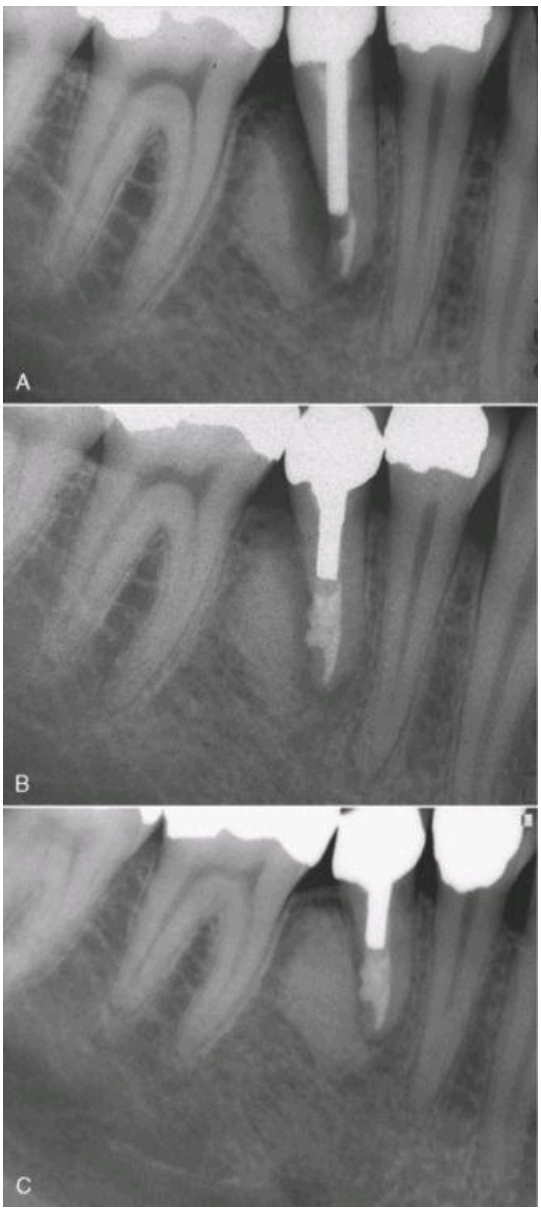

Figure 18-19 A, Lateral root perforation is evident in a patient who has had a previous root canal therapy. **B,** After removal of the post and retreatment of previous therapy, the perforation was repaired with MTA. **C,** Postoperative radiograph taken 5 years later shows absence of any periradicular pathosis.

(Courtesy Dr. N. Chivian.)

Chapter Review Questions available in Appendix B or on the DVD

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Nonsurgical Retreatment

Gary R. Hartwell

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. State the causes for nonhealing after initial nonsurgical endodontic treatment.
2. Discuss the indications and contraindications for retreatment.
3. Discuss the issues that must be considered in retreatment case selection.
4. Identify the treatment options when considering retreatment.
5. Communicate the risks and benefits of treatment options to the patient.
6. Describe the basic materials and techniques used for retreatment.
7. Discuss post-treatment complications, restorative options, and follow-up care.
8. Discuss the prognosis of retreatment.

CAUSES FOR NONHEALING OF INITIAL NONSURGICAL ENDODONTIC TREATMENT

Initial root canal therapy may not result in healing for a number of reasons. The most frequent reasons are the failure to eliminate microorganisms that were present at the time of initial treatment or the reintroduction of microorganisms into the root canal system after the initial treatment was completed.[1,2] Microorganisms may invade the obturated canal space after treatment, primarily as a result of coronal microleakage.[3-5] The primary reason microorganisms persist in the root canal system after initial treatment is the failure to detect or to treat all of the present root canal systems.[3,5-7] Other causes may include inadequate cleansing and shaping of the root canal system, inadequate obturation, and root canal calcifications.[3,5,6]

Healing may also be compromised by procedural accidents that occur during initial treatment. These misadventures may result in root perforations and canal obstructions as a result of ledge formation, canal transportation, or separation of instruments.[3,7]

TREATMENT OPTIONS

If nonhealing occurs, the treatment options include nonsurgical retreatment, root-end surgery, extraction, and, in selected cases, intentional replantation. In cases of nonhealing where the tooth is deemed to be restorable, the patient desires to retain his or her natural dentition, and further endodontic treatment is feasible, nonsurgical retreatment should be the first treatment option considered. The retreatment option affords the practitioner the opportunity to eliminate any microorganisms that may have been left behind during the initial treatment and those microorganisms that may have entered the root canal system after the initial treatment. The surgical retreatment approach should only be the first choice if there are obstructions within the root canal system that make nonsurgical retreatment impossible. The surgical approach will not allow the practitioner to eliminate those microorganisms that cannot be reached with surgical instruments nor will it allow the treatment of the microorganisms with intracanal medicaments. Intentional replantation would be a treatment option only if the nonsurgical and periradicular surgical approaches were not feasible and the patient wanted to retain the tooth by any means possible. Extraction would be the treatment choice if none of the other three options were feasible or if the patient did not desire to have any further treatment procedures performed on the tooth.[3,5,6]

Because of the complexity of these cases, nonsurgical retreatment is usually performed by an endodontist. General dentists may also perform these procedures if they have acquired the appropriate training and experience and have the proper equipment available.[6] When a potential retreatment case presents to the dental office, the general dentist must be fully aware of the risks and benefits of each treatment option that will be offered to the patient. The dentist must also be able to assess the complexity of the case and determine whether he or she can perform the necessary treatment or if referral is the appropriate treatment. The primary goal of this chapter is to provide a basic understanding of nonsurgical retreatment.

INDICATIONS FOR NONSURGICAL RETREATMENT

A patient presents with a tooth that has had root canal therapy, with a history of painful symptoms that either have not changed or have worsened since the initial treatment was performed. The pain may be spontaneous but most often is initiated by chewing or biting pressure. There may also have been a history of episodes of facial swelling or swelling in the mucobuccal fold. The patient may also present with an apical lesion that was not present at the time of initial treatment or the enlargement of a preexisting apical radiolucency (Figure 19-1). Clinical findings may include swelling, percussion and palpation sensitivity, the presence of a draining sinus tract (Figure 19-2), defective or missing coronal restorations, or active recurrent caries. The radiograph (Figure 19-3) may also reveal the presence of one or more untreated root canal systems, poorly obturated root canals, canals not prepared or not obturated to an appropriate length, and recurrent caries that were not noted clinically. All of these are indications of nonhealing associated with the previous treatment. Any one of the clinical symptoms or radiographic findings could indicate that nonhealing has occurred; all three elements do not have to be present to indicate nonhealing.

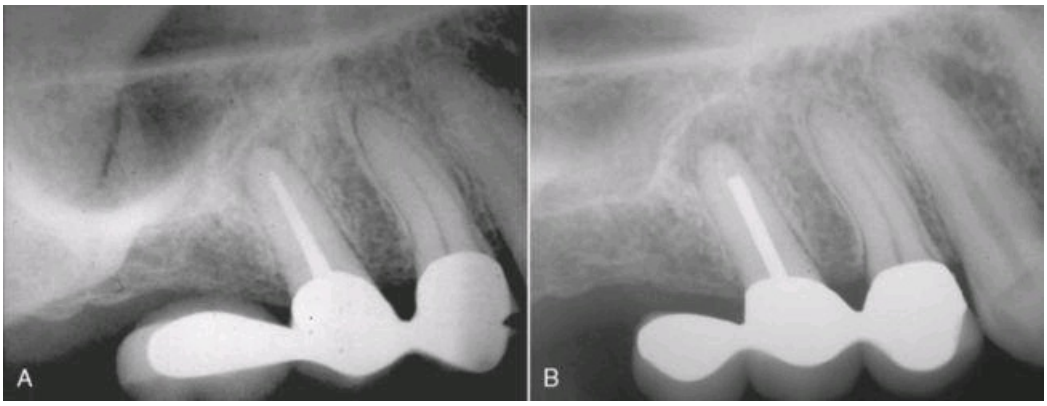


Figure 19-1 A, Root canal obturation. **B,** One year follow-up radiograph shows that periapical lesion has increased in size. A well-fitting fixed prosthesis is in place with a post cemented with resin cement and almost as long as the entire root length. The restoring dentist had removed all the gutta-percha obturation material during post space preparation.

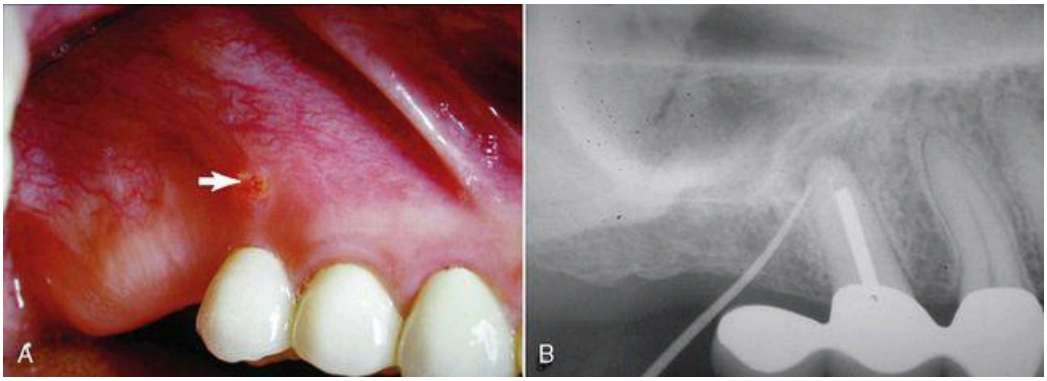


Figure 19-2 A, Presence of a sinus tract opening *(arrow)* adjacent to No. 4. **B,** Sinus tract traced with a gutta-percha point to the apex of No. 4.

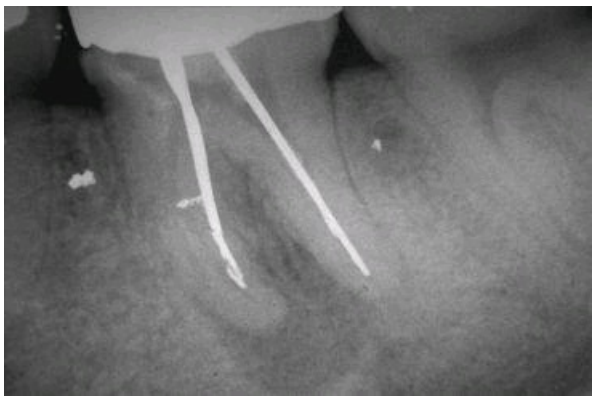


Figure 19-3 Recurrent caries present beneath mesial crown margin. One mesial canal was not treated. The root canals are poorly obturated and not prepared to appropriate sizes and lengths.

Retreatment would be the treatment of choice if it were determined that the procedure is feasible because the tooth is restorable, periodontally sound, and the root canal systems are accessible from a nonsurgical approach. All caries must be excavated, and any defective restorations must be removed. The amount of remaining coronal tooth structure and the tooth's strategic value in the overall dental treatment plan must be assessed to determine restorability. The health of the soft tissue, bony support of the tooth, and crown-to-root ratio must be assessed by periodontal probing, mobility tests, and periapical and bitewing radiographs.[6-8]

The patient must exhibit good oral hygiene habits, have the motivation to retain his or her natural dentition, and be able to financially afford the retreatment and restorative procedures required to restore the tooth to health and function.[9] The patient must also understand that the retreatment procedure will be more complex and time consuming than the original treatment. Any patient concerns with regard to the risks, benefits, and cost of the treatment must be addressed before initiating retreatment.

The chairside staff must be familiar with the procedure to assist the dentist in expediting treatment in a highly professional and expedient manner. The office must have the necessary ultrasonic units with the appropriate tips, solvents, rotary handpieces, endodontic files, and enhanced lighting and visualization equipment to accomplish the retreatment procedure. The dentist must have the experience and level of training to perform the retreatment procedure at the highest skill level and in the most professional and efficient manner.[6] However, there are reasons why retreatment, particularly when the case is complex, is best done by the endodontist.

CONTRAINDICATIONS FOR NONSURGICAL RETREATMENT

Most teeth being considered for nonsurgical retreatment will require removal of previous restorative materials, thus restorability after retreatment is a major consideration. If recurrent caries is also present, the remaining amount of tooth structure will be further compromised. If after caries and restorations are removed, the tooth is deemed to be nonrestorable, then retreatment is contraindicated. If the crown-to-root ratio is compromised and excessive mobility is present as a result of chronic periodontal disease, then retreatment is contraindicated. If the patient lacks the motivation to retain his or her permanent dentition or is incapable of maintaining good oral hygiene because of physical limitations, then retreatment should not be attempted.[3,7,8]

In addition, if the dentist lacks the training and skill level to accomplish the retreatment procedure, the appropriate treatment would be referral to someone with the skills to successfully manage the case. The same applies to a dental office where the staff is not familiar with the procedure and appropriate equipment is not available to manage the case. In these situations, the patient's well being and treatment must not be compromised.[6]

Furthermore, there are several tooth-related factors that may be contraindications for nonsurgical endodontic retreatment. These include situations where the root canal space may no longer be accessible as a result of root canal calcifications or the presence of large, well-fitting post and core restorations (see Figure 19-1, *B*).[3,7] These cases would require periradicular endodontic surgical treatment (Figure 19-4) to attempt to resolve the nonhealing lesion if the tooth is to be retained. In cases of separated instruments that cannot be bypassed or retrieved that result in a nonhealing situation, apical surgery is the choice rather than attempting another nonsurgical retreatment. The same applies to ledges that cannot be bypassed, perforation defects not amenable to nonsurgical repair, and root canals that have been transported and the original root canal space cannot be negotiated[3,7] (see Chapters 18 and 20). External resorptive root defects on previously treated cases are best addressed surgically rather than nonsurgically.[6] Nonhealing cases with vertical root fractures will require either extraction in the case of a single-rooted tooth or possibly root amputation or hemisection on a multirooted tooth that is to be retained.[8] Although this is not a complete list, these are the most common contraindications.

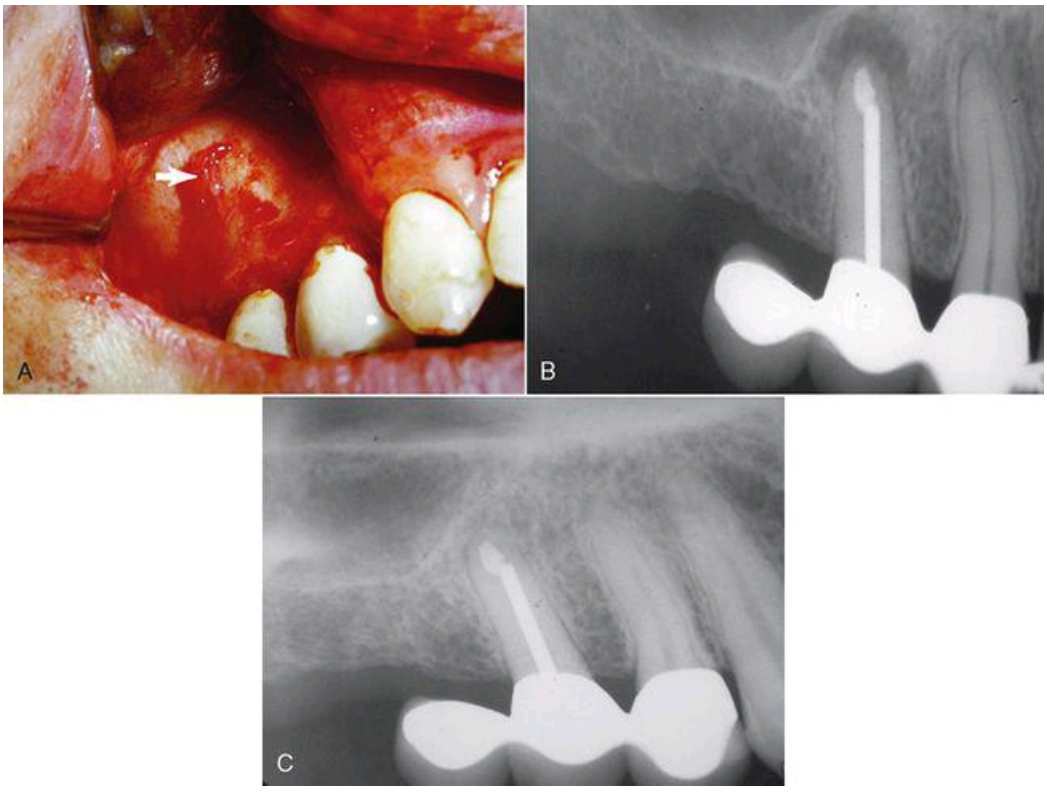


Figure 19-4 A, Surgical procedure performed to remove the periapical lesion and to seal the unfilled apical portion of the root canal. On flap reflection, the periapical lesion is found to have eroded through the buccal cortical bone *(arrow)*. **B,** Time of root-end filling procedure. **C,** One year after the surgery shows that the periapical region has almost completely healed.

RISKS AND BENEFITS OF RETREATMENT

As part of informed consent, the risks and benefits of each treatment option must be presented to the patient. For nonsurgical root canal retreatment procedures, the risks include fracture of the crown or root of the tooth; thinning, weakening, or perforation of the root canal wall; creation of ledges in the canal wall; separation of the instruments used during the retreatment procedure; and loosening with the need for replacement of a well-fitting fabricated crown.[6,7,10] Any one of these risks alone or in combination with the others may necessitate tooth extraction. The benefits of retreatment include retention of the patient's natural tooth structure, which can be utilized to restore the tooth to form and function, decreasing the need for a more extensive and possibly more expensive prosthetic replacement if the tooth was to be extracted.

PROCEDURES FOR RETREATMENT

Criteria for Restoration Removal or Retention

Retreatment procedures can be expedited if the coronal restorations are removed. This allows more favorable access for the removal of the post and core restorations and the root canal obturation materials present in the root canals.[6,8] If there is evidence of coronal microleakage, all defective restorations and any recurrent caries must be removed to assess the restorability.[5,7,8] In cases where the teeth have been restored with amalgam or composite, the entire restoration should be removed and consideration should be given for a new restoration that will provide cuspal protection. If the tooth has previously been restored with a full coverage crown, the crown should be removed if there is evidence of a poor marginal seal or if recurrent caries is present. In some cases, crown removal may be necessary to facilitate the removal of core materials from the pulp chamber area and posts from the root canal system.[6-8] In situations where a cosmetic crown is present, the crown margins are intact, and no recurrent caries is present, an attempt may be made to perform the retreatment through an access opening in the crown.[6,8] Before the treatment through a crown is initiated, the patient must be informed that the crown may loosen or the cosmetic portion of the crown may fracture during the treatment procedure. If either of these occurs, a new crown will likely have to be constructed. If the retreatment is successful and the crown is retained, the access opening must then be restored with a core and permanent restorative material.

Removal of Canal Obstructions

There are four major types of canal obstructions that can prevent the successful negotiation of the entire root canal system in attempting to perform nonsurgical retreatment. Failure to remove these obstructions most likely will result in nonhealing of the nonsurgical retreatment. If nonhealing occurs and the tooth is to be retained, apical surgery would be the treatment of choice. Two other treatment options would be intentional replantation or extraction. The four major obstructions are (1) post and core restorations, (2) root canal calcifications, (3) ledges in the root canal wall, and (4) separated root canal instruments.[3,6,7] The risks and benefits of attempting to remove or bypass these obstructions must be assessed very carefully by the practitioner and must be fully explained to the patient, so she or he will be able to make an educated treatment decision.[11]

Post and Core Removal

Many retreatments require removal of a post and core. As with any of the retreatment techniques, several factors will influence the successful removal of the post and core. The factors include the practitioner's level of training and experience, availability of the best technology, the length and diameter of the post, the design of the post, and the cementing agent used to secure the post in the root canal system.[7,8,10] All methods used to remove posts may jeopardize the remaining tooth structure.[10] Long, well-fitted posts (see Figure 19-1, *B*) may be difficult or impossible to remove. Removal of these long posts may be contraindicated if the dentin walls are very thin or there is a strong possibility that root fracture or perforation may occur during their removal.[8,10] Posts are usually best removed by the endodontist.

The first step in post removal is to section and remove the core material in such a manner as to retain the length of the post that extrudes coronally out of the root canal. The core material may be removed with carbide burs, diamond burs, or specially designed transmetal burs.[7,8,10] Any visible cement around the post should be removed with a very fine ultrasonic tip.[7,12] Ultrasonic energy is then delivered circumferentially to different locations around the exposed portion of the post for no longer than 15 seconds in any one position.[7,12-15] The threshold temperature for heat-induced bone necrosis is 10° C maintained for 1 minute. It was noted that when an ultrasonic tip, without water coolant, was placed into contact with a post for 30 seconds or longer, temperatures greater than 10° C were generated on the external root surface just below the level of the cementoenamel junction.[15] When posts have been placed with zinc oxyphosphate cement, it can take anywhere from 1½ to 20 minutes to loosen the post from the cement.[7,10,13-17] One study found that using two ultrasonic tips on a post at the same time decreased the amount of time required to loosen a post.[17]

A hemostat or small-tipped forceps may then be used to grasp the end of the loosened post to remove it from the root canal. If the post is a screw type, a hemostat may be used to unscrew it from the root canal. If the loose post cannot be removed with either of these methods, specially designed devices may be required for removal of the post.[7,10,18,19] If these devices are used without attempting to first loosen the post, there is a much greater risk of fracturing the tooth or of having to remove excessive amounts of tooth structure to achieve post removal.[7,10,18] When removing posts, there does not appear to be any greater risk of developing cracks in the root when using the ultrasonic method versus other post removal systems.[19] Posts placed with a bonding agent are more difficult if not impossible to remove with any of the post removal methods.[8] Once the post is removed, the cementing material adherent to the root canal wall can be removed with a combination of solvents, hand or rotary endodontic files, and ultrasonic tips.[7,8,10]

If one of the special post removal kits (Figure 19-5) is required to remove the post, the following basic procedure can be followed.[7,10,18] A diamond or transmetal bur is used to reduce the size of the coronal portion of the post. A trepan bur is then used, and the post is tapped so that the matching size extractor will firmly engage the coronal portion of the post. The extractor is then engaged with special pliers included in the kit (Figure 19-6). Rubber washers are placed into contact with the remaining tooth structure, and the pliers are engaged so that the tooth acts as the fulcrum. The rubber washers act as a cushion to reduce pressure and to reduce the chances of fracturing the tooth structure. This technique has been found to be effective and relatively safe.[19]

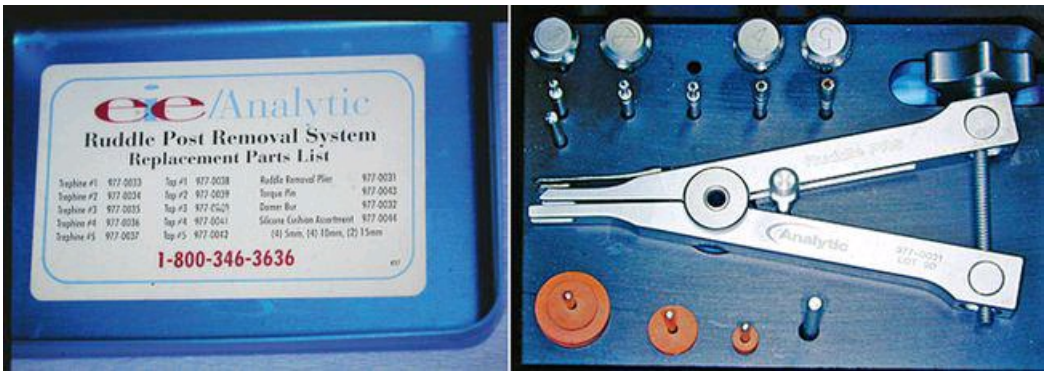


Figure 19-5 Post removal system.

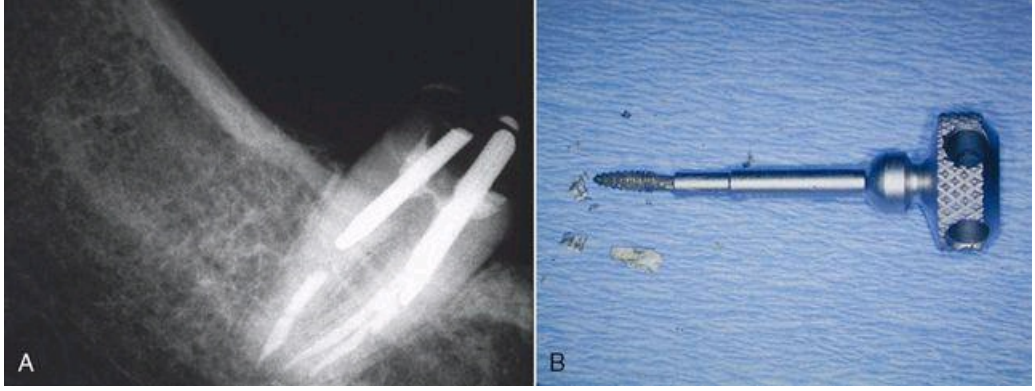


Figure 19-6 A, Posts must be removed before retreatment. **B,** Post removed from the distal canal with the post removal system extractor.

Removal of Calcifications

To remove root canal calcifications, the practitioner needs to be able to visualize the calcified area. This is feasible with the enhanced illumination and magnification provided by the operating microscope. Any obstructions blocking access to the calcified area must be removed first. Once the calcified area is visualized, a combination of stiff hand files, chelating agents, and ultrasonic tips is used to attempt to remove the calcified barrier and allow access to the portion of the root canal apical to the calcification. The use of the ultrasonic tips will be restricted to the straight portion of the canal. A small bend can be prepared in the stiff hand files, and these can then be used in conjunction with the chelating agent in the curved portion of the canal. If the calcified canal obstruction is removed, then the root canal can be enlarged in a crown-down fashion, with a combination of Gates-Glidden burs and hand files or one of the many tapered nickel-titanium rotary file systems. If the calcification cannot be removed, then one of the surgical options or extraction must be considered, particularly if a periradicular lesion is associated with that root.

Removal of Ledges

Ledges are generally created during the cleansing and shaping portion of root canal therapy (see Chapter 18). These obstructions occur more frequently with the canal preparation techniques that use only hand stainless steel files. With the advent of hand and rotary nickel-titanium instruments, there are fewer ledges created because the nickel-titanium files will stay centered in curved canals. Stainless steel files have memory and will attempt to straighten out in a curved canal, with the result being transportation, often with the formation of a ledge or perforation in the outside wall of the curve. If a ledge is present in a retreatment case, all of the obstructions coronal to the ledge must be removed, the coronal portion of the canal opened up in a crown-down manner, and the ledge visualized. The goal is to bypass the ledge with a stiff, curved hand file; once the file is beyond the ledge, use it in a circumferential filing motion to remove the obstruction. One must proceed from small-size files to larger-sized files until the ledge is removed. Once the obstruction is removed, the apical portion of the canal can be cleansed and shaped with any instrumentation technique. If the ledge cannot be removed or bypassed, then the same surgical or extraction treatment options exist as were outlined for the calcified canal obstruction cases.

Removal of Instrument Fragments

Factors that will affect the successful removal of instrument fragments from the root canal system include the skill and experience of the practitioner, the size of the instrument, the length of the fragment, and the location of the fragment within the root canal system.[7,20,21] A smaller size instrument may be bypassed or ground away with ultrasonics or a bur.[8,22] It may also be removed by engaging the fragment with a braided file technique or with special kits or devices designed for removal of the instrument fragment.[7,10,20-26] The longer the instrument fragment, the more likely successful removal can be accomplished.[8,20] The braided file technique or fragment removal kits and devices work very well for removing longer fragments.[7,20-22,26]

If the canal is curved and the fragment is present in a position coronal to the curvature, the chance of successful removal is greatly increased. The chance of successful removal of a fragment located in a position apical to the curvature is very poor (Figure 19-7, *A*). Nonsurgical attempts to remove the fragment in the latter situation frequently result in transportation of the original root canal system, perforation of the root, or fragmentation of additional instruments (Figure 19-7, *B*) during the attempted removal process.[20,23] The thickness of the remaining dentin walls must also be taken into consideration.[7,21] If the prognosis for removal is poor, a periradicular surgical approach may be the treatment of choice (Figure 19-8).[10]

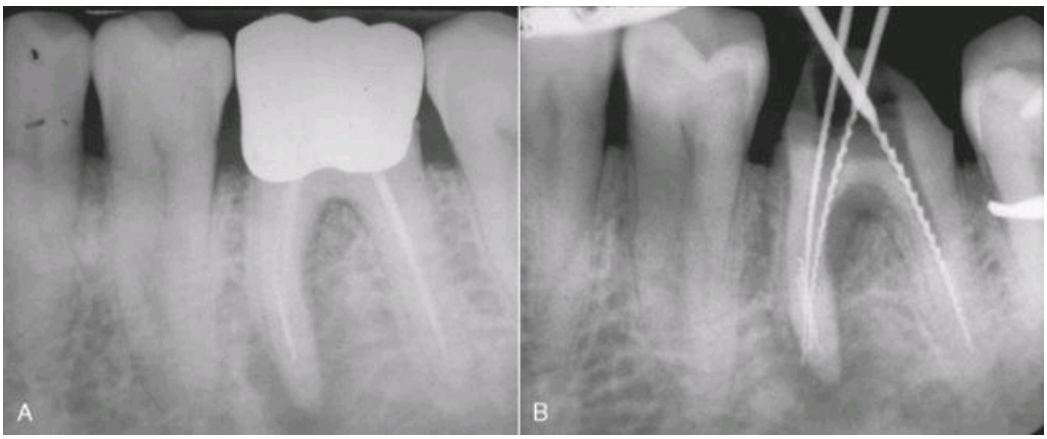


Figure 19-7 A, Instrument fragment in one of the mesial canals just apical to the root curvature. **B,** Attempts to remove the instrument fragment resulted in transportation of the original root canal system, perforation of the root surface, and fragmentation of a second instrument.

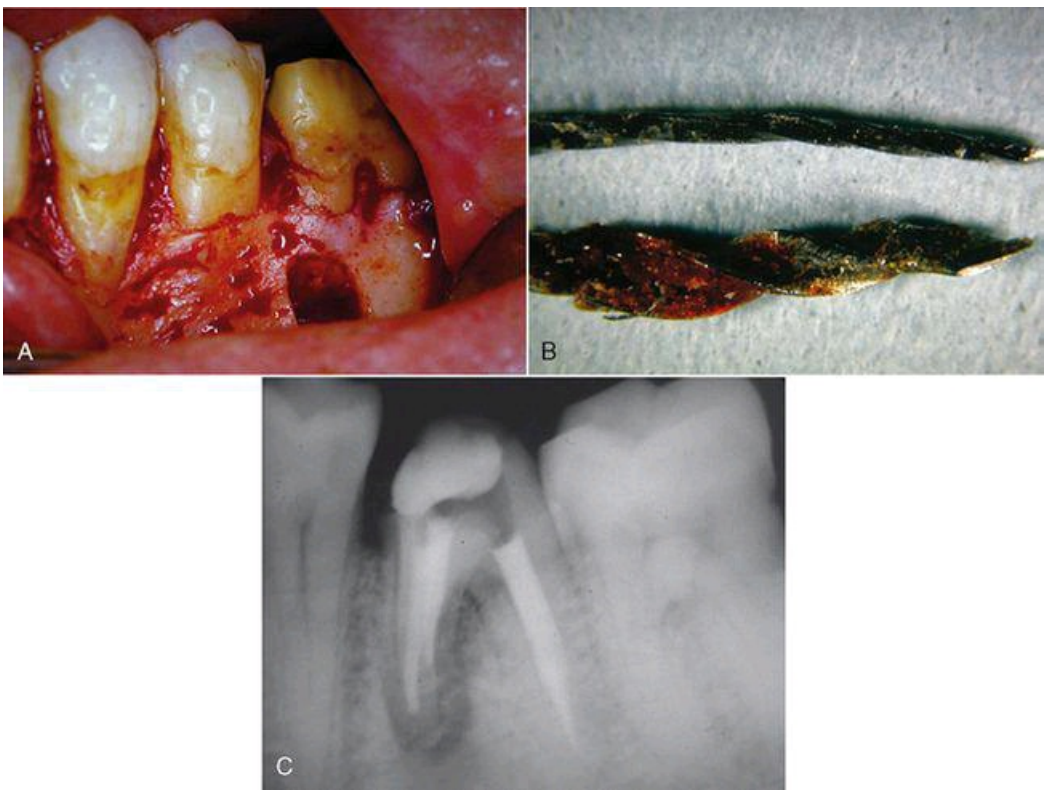


Figure 19-8 A, Surgical flap reflected and mesial root resected. **B,** Two broken file fragments removed from the mesial root. **C,** Postsurgical radiograph to confirm the removal of the instrument fragments.

The first step for successful removal is to gain access to the coronal portion of the fragment so that it may be visualized.

The use of an operating microscope is almost essential for viewing the fragment. The access may be accomplished using one or a combination of small long-neck burs, Gates-Glidden burs, hand files, or ultrasonic tips.[7,20,21,23,24] For a small fragment, a small-diameter hand file is inserted into the canal and an attempt is made to bypass and engage the fragment for its removal.[7,8,20,22-24] Using two or three small files to engage the broken fragment at several locations and then twisting the files in a braided manner may also result in fragment removal when the braided files are pulled in a coronal direction.[21] In some instances, a very-small-diameter ultrasonic tip may be successful in dislodging the fragment, or in slowly breaking the fragment into smaller pieces that can then be removed when the canal is irrigated.[7,10,20,22,23] If the small fragment cannot be removed or bypassed, periradicular surgery may be required to successfully treat the case.

In situations where longer and larger-size fragments are present in the straight portion of the canal, bypassing the segment may be impossible. After the top of the fragment is visualized, a staging platform[21,23,24,27] is created adjacent to the coronal extent of the fragment. This may be accomplished with modified Gates-Glidden burs,[23,24,27] modified burs,[21] or modified rotary files.[27] The platform facilitates the use of a small diameter of modified ultrasonic tip to create space around the coronal end of the segment. The fragment can be then be engaged and removed with the braided file technique or with one of the special instrument fragment removal kits.[*] Due to the large diameters of the trephine burs and tubes used with these kits, the walls of small-diameter root canals can be easily perforated.[‡] Several microtube removal methods have been advocated for use in smaller root canals. One system uses a microtube with cyanoacrylate glue to engage and remove fragments.[7,10] Other systems use microtubes with fine-wire “lassos,” files, or screw wedges (present inside the tubes) to engage the fractured segment.[7,10,22,26]

If the fragment cannot be removed but can be bypassed, then the root canal system apical to the fragment can be cleansed and shaped if it is negotiable. The instrumentation of the apical segment should be accomplished with hand instruments because they are less likely to bind to the retained portion of the rotary files. If the rotary file binds, the result will be an additional broken file segment that will further hinder the successful nonsurgical treatment of the tooth. If the fragment is successfully removed, then the apical portion of the canal can be prepared by either hand or rotary file systems.

Removal of Gutta-Percha

Because gutta-percha is the most frequently used root canal obturation material, it is also the material that most frequently needs to be removed during retreatment. Removal of this material has been done with hand and rotary files, heat, ultrasonics, solvents, and combinations of all of these.

Hedstrom hand files or regular hand reamers[22,29] are the choice when the root canal is poorly sealed with gutta-percha (Figure 19-9) and space either exists or can easily be created between the gutta-percha and the root canal wall. A Hedstrom file or hand reamer is placed into the space and rotated in a clockwise manner until the gutta-percha is engaged. Force is then exerted in a coronal direction with the file/reamer until gutta-percha is removed in one piece (Figure 19-10). Any remaining gutta-percha and sealer are then removed, and the canal is instrumented in a crown-down fashion to avoid pushing debris apically and out of the apical foramen.

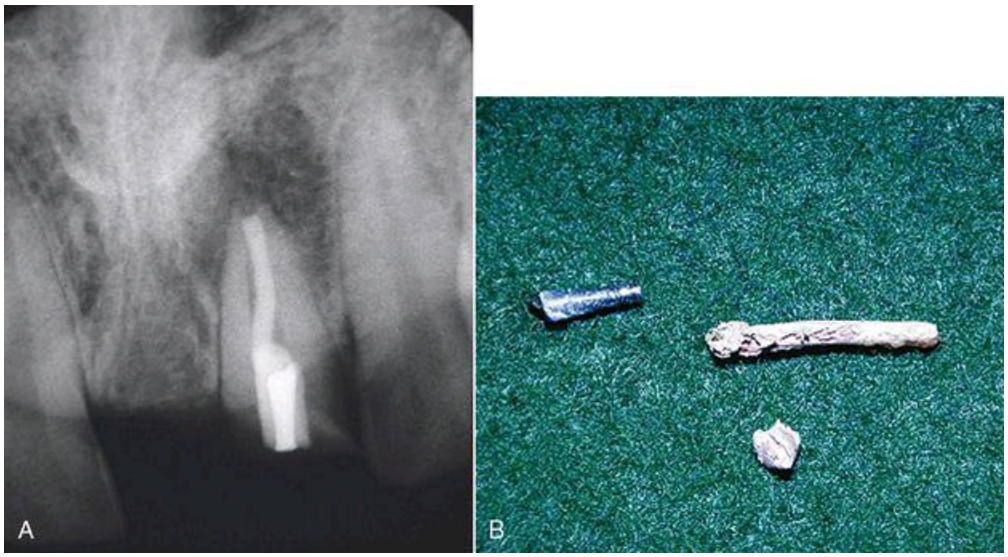


Figure 19-9 A, Poor obturation with gutta-percha. **B,** Gutta-percha and amalgam fragment easily removed from the root canal system.

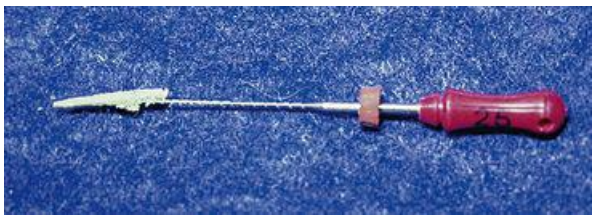


Figure 19-10 Gutta-percha engaged with a Hedstrom file and removed in one piece.

In teeth where the gutta-percha is more closely adapted to the root canal wall, removal can be more of a challenge. Heated pluggers may be used to remove the gutta-percha in these cases.[22,30] Hand pluggers can be heated over an open flame and inserted into the mass of gutta-percha. Gutta-percha dissipates the heat and the plugger cools very rapidly, so the plugger will only advance a short distance into the material. In some instances, the plugger will remove the entire gutta-percha mass, but, more frequently, only small sections of the gutta-percha are removed. This technique is very time-consuming, and as the apical portion of the canal is approached, there is danger of pushing the softened gutta-percha out of the apical foramen. A more predictable method is to use a thermostatically heated plugger that will not cool while being inserted into the gutta-percha.[29] The plugger used with one of these devices can be inserted into the gutta-percha until the plugger tip is located in the apical portion of the canal. When the plugger is allowed to cool while in contact with the gutta-percha as it is removed, there is a much greater chance that the entire mass of gutta-percha will be removed with the plugger. There is also less chance of softening the apical mass of gutta-percha and extruding it into the periapical tissues using this method. Once the majority of gutta-percha is removed, cleansing and shaping of the canal can proceed in a crown-down fashion.

The removal of gutta-percha with sonic[30] and ultrasonic tips[22,31,32] is similar to the method described for the thermostatically controlled heated pluggers. The sonic or ultrasonic energy delivered to the tips softens the gutta-percha, but actual removal of the softened gutta-percha with these sonic and ultrasonic devices is problematic.[22] This method is more likely to aid in the removal of the gutta-percha when used in combination with hand or rotary files.[32] The ultrasonic tip softens the gutta-percha, and the actual removal is then accomplished with the files. These devices are better suited for removal of the remaining adherent root canal sealer from the root canal walls after the gutta-percha has been removed.[33]

The quickest method for removing gutta-percha is with Gates-Glidden burs or rotary files.[29,34] The rotary instruments

recommended for this purpose include Gates-Glidden burs,[22,29,35] special gutta-percha removal burs,[22,29] and various types of rotary files.[8,22,35-38] The speed at which these rotary instruments are used varies from 300 rpm for the RaCe[35] (Brasseler USA, Savannah, GA) and 0.04 ProFile[34] (Dentsply Tulsa Dental, Tulsa, OK) rotary files (Figure 19-11) to 1300 rpm for the 0.06 ProFiles[37] and Liberator[38] (Miltex, York, PA) rotary files.

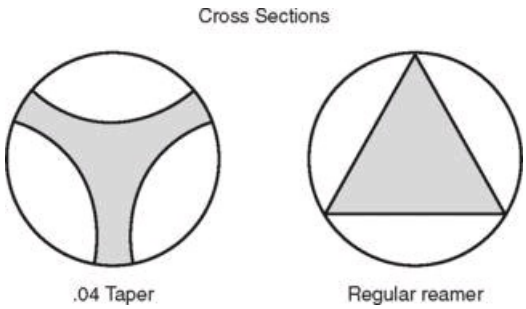


Figure 19-11 The deep flutes on the 0.04 rotary file become filled with gutta-percha as the file rotates and advances apically into the root canal. The flute design of the rotary files allows the gutta-percha and sealer to move coronally and out of the canal rather than being forced apically. Regular files and reamers are not as efficient in the removal of gutta-percha from the canal.

Once a purchase point is created in the most coronal aspect of the gutta-percha, the selected bur or file at the appropriate speed will work its way apically into the gutta-percha mass. An advantage of this technique is that the gutta-percha is removed in a coronal direction as the bur or file is advanced apically. Even though this is an efficient method for removing gutta-percha, its use is limited to roots with straight canals. Once the bulk of gutta-percha is removed, the crown-down cleansing and shaping of the canal can be completed. This method has also been found to be very effective in the removal of the new synthetic polymer-based root canal obturating material.[35,37,38]

A variety of solvents have also been used to soften the gutta-percha and aid in its removal from the root canal.[*] A drop of the selected solvent is placed into contact with the exposed gutta-percha, and once the material begins to soften, a hand or rotary file is used in the manner described to remove the gutta-percha from the canal. Of all the solvents, chloroform works the fastest and is the most efficient in softening the gutta-percha.[22,34,37] Halothane reacts in a similar fashion but more slowly and has been found to be an acceptable alternative to chloroform.[32,42] Methylchloroform has also been found to be a good alternative to chloroform because it is less toxic, not carcinogenic, and more effective than xylene and eucalyptol for softening gutta-percha.[22] Other solvents that have been tried but have been found to be *less* efficient in softening gutta-percha include xylene, eucalyptus oil, orange oil, carbon disulfide, and benzene.[22,39,40]

Because of the reported toxicity and potential carcinogenicity of chloroform, the alternative solvents noted have been used, but the results with regard to their ability to soften gutta-percha have been mixed.[22,32,39,40] One study determined the residual volume of chloroform, xylene, and halothane expressed through the apical foramen during retreatment of teeth obturated with gutta-percha.[40] They found that the volume of each solvent that was expressed beyond the apical foramen was several orders of magnitude below the permissible toxic dose and would result in a negligible health risk to patients.

Solvents are just adjuncts to the removal of the gutta-percha. Hand or rotary files must be used to complete the removal of the material. Then the canal preparation can proceed with any acceptable step-back preparation technique.

Removal of Carrier-Based Gutta-Percha Obturators

Canals may also be obturated with gutta-percha coated onto a central core of metal or plastic. Removal of these carrier-based obturators utilizes a combination of the techniques used to remove gutta-percha, silver cones, and posts. The first step in removal is to soften the gutta-percha on the surface of the carrier with a solvent[43-46] or a heat source[47,48] so that a pathway can be created for an instrument that will engage and remove the carrier. The solvents are the same as those described in the gutta-percha removal section of this chapter. The heat source could be an endodontic heat carrier or plugger heated over an open flame or a thermostatically controlled heat source.[47] There are concerns that the high temperature achieved could potentially damage the periodontal ligament if the tip is left in contact with the dentin wall for too long of a period of time.[48] It has also been suggested that the frictional heat generated by rotary tapered files (run at speeds of 1500 to 2500 rpm) will soften the gutta-percha.

Once the pathway is created, one or more hand files (Figure 19-12) can be used to engage and remove the carrier.[43,45-47] Another suggestion is to engage and remove the carrier with a large taper rotary file. These techniques work very well when dealing with the removal of plastic carriers but not as well for the removal of metal carriers. The flutes on the files have more difficulty engaging the surface of the metal carriers. A braided file technique, using more than one hand file, works best for removal of these carriers (Figure 19-13). Once the gutta-percha and carrier are successfully removed, the canal preparation can then proceed normally.

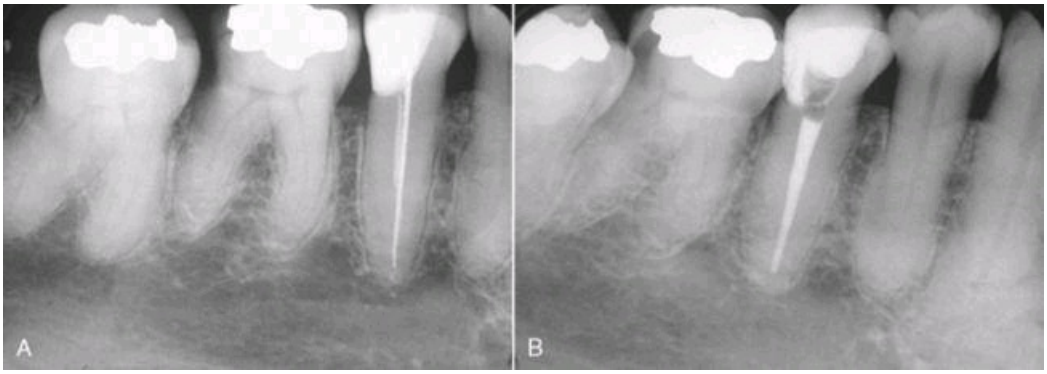


Figure 19-12 A, Symptomatic after carrier-based gutta-percha obturation. **B,** Carrier and gutta-percha removed, root canal prepared, and obturated with a combination warm vertical and backfill gutta-percha technique.

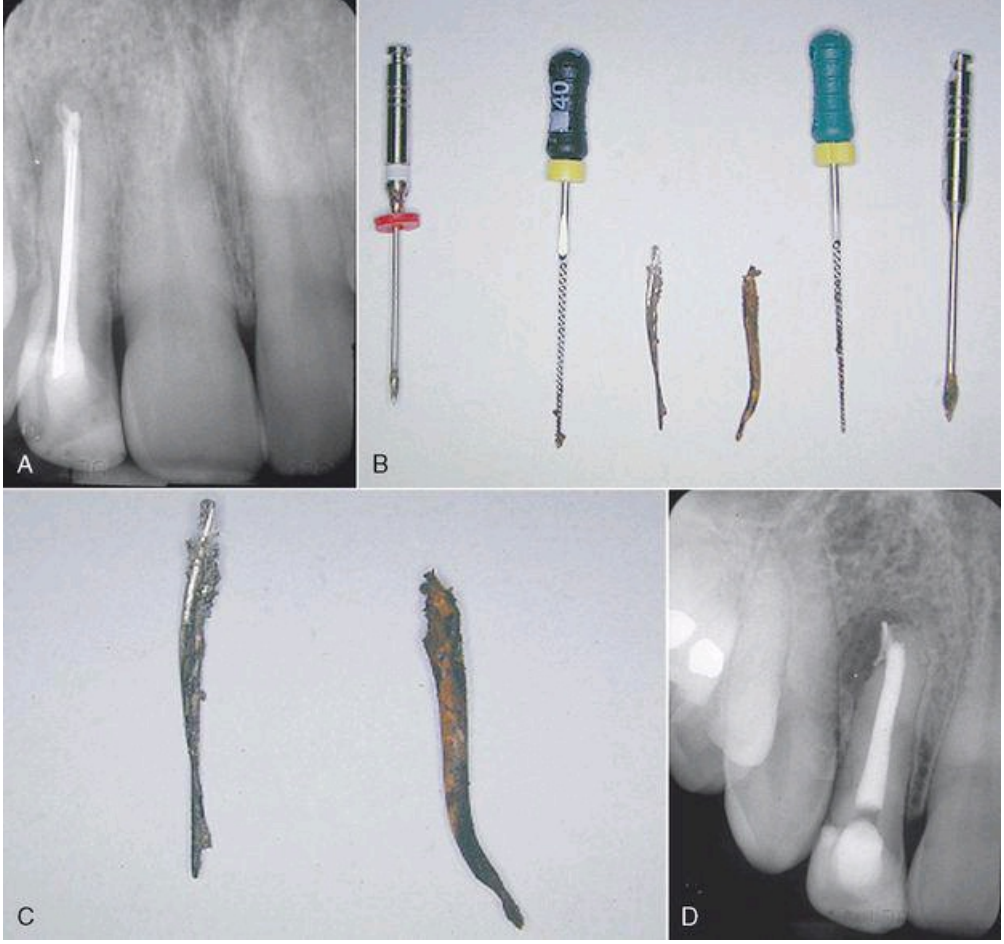
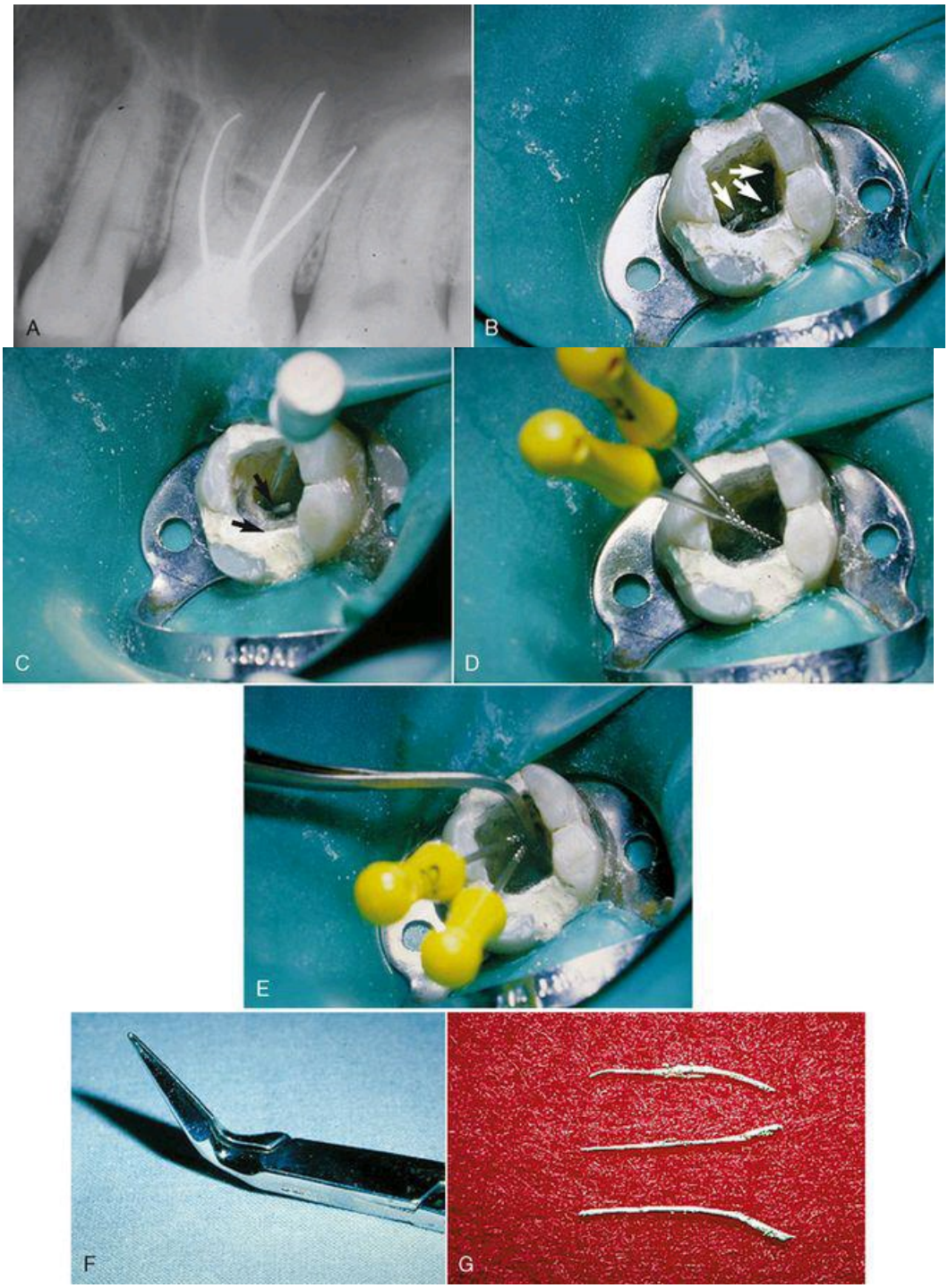


Figure 19-13 A, Symptomatic tooth with metal carrier gutta-percha obturation. **B,** Gates-Glidden burs and K-files used to remove the metal carrier and gutta-percha. **C,** The gutta-percha coating separated from the metal carrier on removal from the root canal. **D,** Root canal system prepared and obturated with a combination warm vertical and backfill gutta-percha technique.

Removal of Silver Cones

The key to success in the removal of silver cones (Figure 19-14, *A*) is to retain as much of the coronal extent of the cone as possible.[8] If the coronal end of the cone is embedded in the base or restorative material, then these materials must be removed very carefully so as to not remove the coronal portion of the cone.[8,49,50] Once the portion of the cone that is in the pulp chamber is exposed (Figure 19-14, *B*), then solvents, ultrasonic tips, and hand files are used to create space (Figure 19-14, *C*) between the root canal wall and silver cone.[22,50,51] The ultrasonic tip should not engage the silver cone because the metal is so soft that the ultrasonic tip will easily cut through the cone. If an attempt is made to break the seal between the silver cone and the root canal cement with ultrasonics, one or two files should be braided around the cone (Figure 19-14, *D*) and the ultrasonic energy delivered to the files (Figure 19-14, *E*). The vibratory energy will loosen the silver cone from the sealer in many cases, thus facilitating removal of the cone.[10,49]



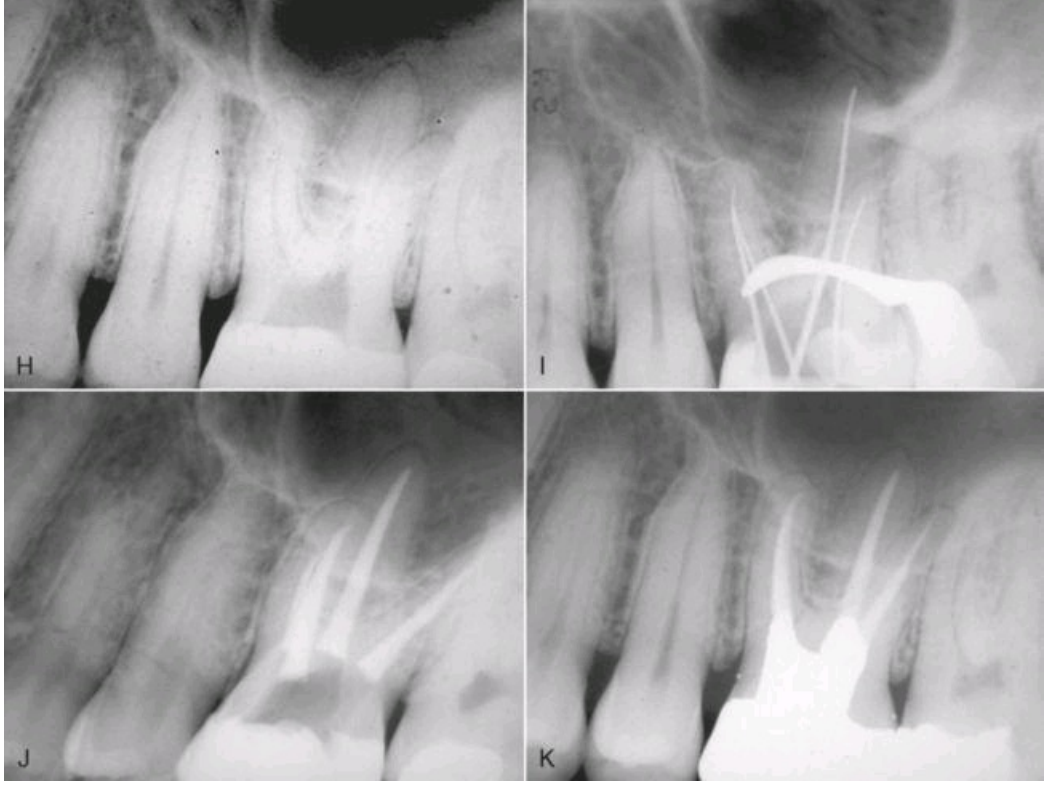


Figure 19-14 A, Tooth is symptomatic, and three canals have been obturated with silver cones. **B,** Cement base is removed, and the coronal ends of the silver cones extending into the pulp chamber are exposed *(arrows)*. **C,** A hand file is used to create space between the silver cone (*arrow*) and root canal wall. **D,** Hand files braided around one of the silver cones. **E,** An ultrasonic tip is activated while in contact with the files to deliver energy to the silver cone. This technique aids in breaking the seal between the silver cone and the root canal cement. **F,** Steiglitz forceps grasp the coronal ends of the loosened silver cones. **G,** All three silver cones were successfully removed intact. **H,** Radiograph confirming that all of the silver cones have been successfully removed. **I,** Working length radiograph demonstrates the presence of a second canal in the mesiobuccal root that had not been previously treated. **J,** Obturation of all four root canals with a gutta-percha technique. Note that the second mesiobuccal canal exits the root apex as a completely separate canal. **K,** One year follow-up. Tooth is now restored with a core and crown. The tooth is asymptomatic and there is no evidence of any periapical radiolucencies.

Once the cone is loosened, the portion extending into the pulp chamber may be grasped with one of the following to complete the removal: regular or modified Steiglitz forceps (Figure 19-14, *F* and *G*),[10,22,51] Caufield silver point retrievers,[10,22] gold foil pliers,[10] splinter forceps,[10] needle holders,[22] or various types of hemostats.[10,22] If the coronal portion of the cone is not present or was inadvertently removed during removal of the coronal restoration, one or more hand files may be used to engage and remove the cone.[10,51] Other devices that may be used in this latter situation include needle-sleeve or tube devices[22,49] where the cone is grasped with a wire,[10,22] file,[50] or cyanoacrylate glue[10,52] to facilitate removal of the cone. In these situations, the end of the cone will have to be exposed and space will have to be created around the coronal portion of the cone with a trepan bur.[10,22,49,52] Once the silver cone is successfully removed, the canal preparation can proceed in a normal fashion (Figure 19-14, *H* to *K*).

Removal of Soft and Hard Pastes

If a soft paste has been used to obturate the root canal system, the material can easily be penetrated and removed with either hand or rotary files. In the removal of the soft material, it is important that copious irrigation be used with a crown-down canal preparation technique.[22] This will prevent the extrusion of the material into the periapical tissues that potentially result in painful postoperative flare-ups (Figure 19-15).

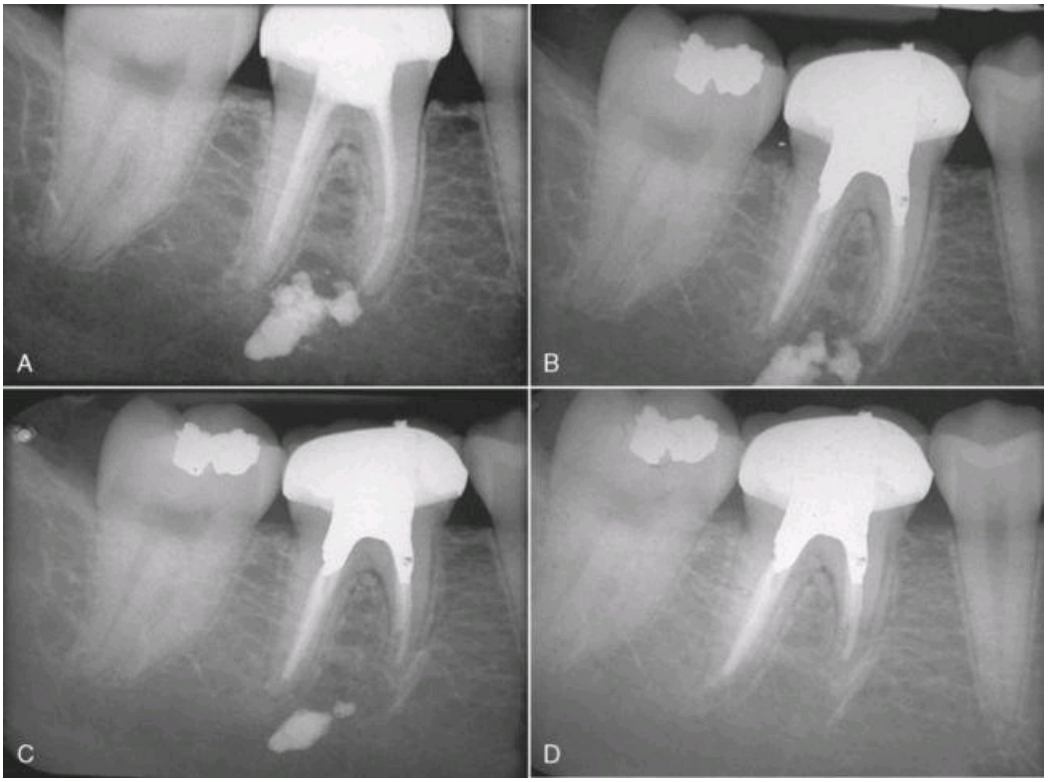


Figure 19-15 A, Symptomatic No. 30 and paresthesia of the lower lip on the right side. History of a soft paste (N2) obturation 6 months before this visit. **B,** The soft paste was removed from all the canals, and the canals were obturated with a gutta-percha technique. **C,** Radiograph made during the surgical procedure performed to remove the soft paste from the bone lesion. **D,** Four-year follow-up reveals complete bone healing. The tooth is asymptomatic, and normal sensation has returned to the lower lip.

Hard setting pastes are more difficult to remove, and, in some cases, may even be impossible to remove. In the past, it was suggested that removal of approximately 4 mm from the tip of a K-file would create a shaped edge that might prove to be useful in the removal of the hard paste material.[53] Today, several dental companies manufacture a file with a sharp point that can be used to gain initial penetration into these materials. If the sharp file system does not work, burs with small-diameter heads or ultrasonic tips are used to remove the hard paste.[8,22,54] There is a danger of root perforation and transportation (Figure 19-16) of the root canal system if the entire length of the canal must be initially negotiated with either of these methods.[22] It is obvious that the latter two methods are primarily for use in straight root canals.

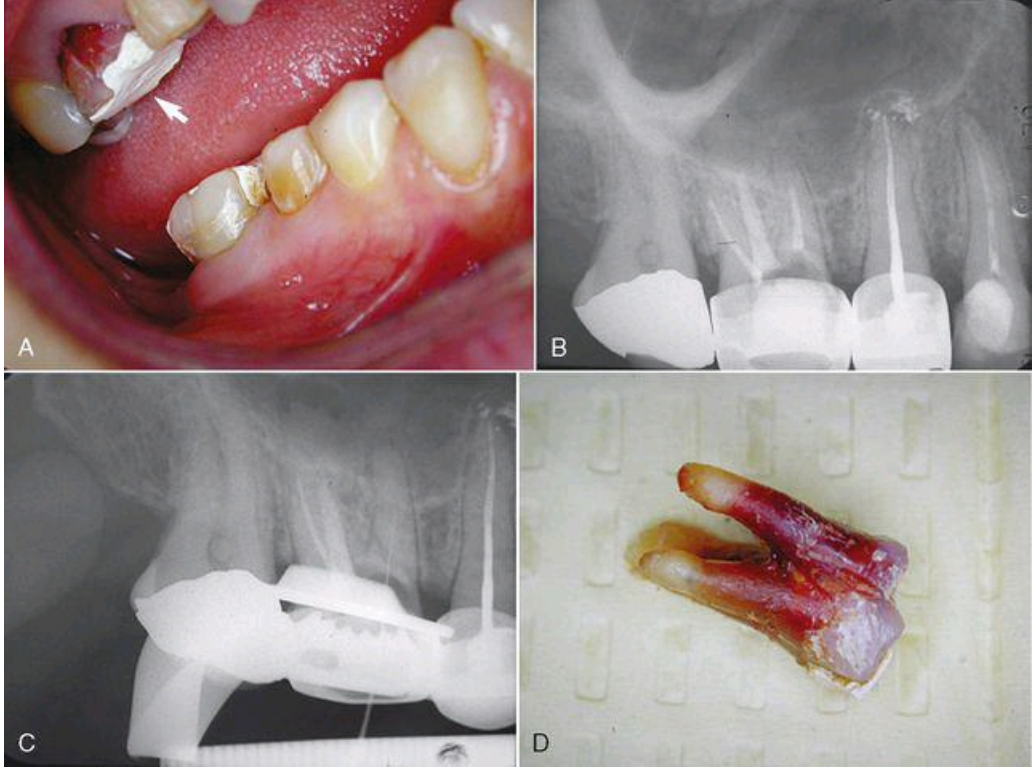


Figure 19-16 A, Tooth (*arrow*) exhibits red discoloration of the crown as a result of root canal obturation being performed with a hard resorcinol paste material. **B,** The resorcinol obturations are short of ideal length in all three canals of tooth No. 3. Resorcinol paste was also present in premolars. **C,** The resorcinol material was successfully removed from the coronal portion of the mesiobuccal canal, but the canal was calcified apical to the level of the previous obturation. A root perforation occurred during attempts to remove the material from the palatal canal. The treatment plan was altered, and the tooth was extracted and replaced as part of a bridge. **D,** Note that the red discoloration of the roots extends to the level of the previous resorcinol obturations.

Endodontic solvents have been investigated to facilitate softening and removal of a resorcinol-formalin paste. One in vitro study[55] reported that 5.25% sodium hypochlorite was capable of softening this paste material, but a follow-up study determined that the sodium hypochlorite did not produce the same result when an extracted tooth model was used.[56] This study compared six different potential solvents and found that none were any better than the water control in softening this kind of hard paste.[56]

At present, the ultrasonic method is the most predictable for removal of the hard paste material. When the paste is removed, normal canal preparation can proceed. If not, a root-end surgical endodontic procedure might have to be performed if the tooth should be retained.

POST-TREATMENT CONSIDERATIONS

Flare-Ups

Flare-ups tend to occur more frequently in teeth that have been retreated when compared to those teeth with initial root canal therapy only.[57-59] To prevent or at least limit the number of flare-ups associated with retreatments, material removal and instrumentation techniques should be selected that minimize the extrusion of debris and microorganisms beyond the apical foramen.[60] These techniques would include the frequent use of irrigants to flush debris from the canal and the use of a crown-down preparation technique that removes debris in a coronal direction rather than forcing material and microorganisms apically. Other preventive measures include thoroughly cleansing the canal at the initial visit, using an intracanal medicament between the instrumentation and obturation visits, not leaving the root canal system open for drainage between visits, and maintaining asepsis throughout the entire retreatment procedure.[60] Because of the increased chance of flare-ups in retreatment cases, these cases are generally treated in two visits rather than one.

Final Coronal Restoration

Teeth that have been successfully retreated require an appropriate coronal restoration the same as teeth that have undergone initial root canal therapy. The objective is to protect tooth structure and prevent coronal microleakage so that additional retreatment procedures or tooth extraction will not be necessary.

Follow-Up Visits

As with any other endodontically treated tooth, follow-up visits should be scheduled to ensure that the tooth is restored, clinical signs and symptoms disappear or do not recur after completion of treatment, and radiographic healing occurs. If the cause of the initial treatment is identified and corrected, the initial follow-up visit should occur at a minimum of 6 months post-treatment and then again at 1 year. If the etiology of the previous failure was not identified or there were complications that did not permit complete retreatment of the root canal system, then a 3-month initial follow-up visit is warranted.

Prognosis for Healing

Most of the reports dealing with the prognosis for retreatment cases were accomplished 18 or more years ago[3,61-64] and do not reflect the manner in which nonsurgical retreatment cases are treated today. The reported success rates in these reports varied from a low of 48% for teeth with apical radiolucencies and canals that could not be negotiated to the apical terminus to 94% for teeth in which there were no apical lesions and the canals could not be negotiated to proper length.

Today, retreatment cases are being managed in a much more predictable manner because of the technological advances that have occurred over the past 10 to 15 years. The most recent report was published in 2004,[65] but the results reported related to cases that had been treated 20 to 27 years before. In this report, 95.5% of the roots were "normal" radiographically. A prognosis study is needed that will take into account the retreatment procedures currently being used.

Chapter Review Questions available in Appendix B or on the DVD

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Endodontic Surgery

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CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Discuss the role of endodontic surgery in treatment planning for a patient.

2. Recognize situations in which surgery is the treatment of choice.

3. Recognize the medical or dental situations in which endodontic surgery is contraindicated.

4. Define the terms incision for drainage, apical curettage, root-end resection, root-end preparation and filling, root amputation, hemisection, and bicuspidization.

5. Describe in brief the step-by-step procedures involved in periapical surgery, including those for incision and reflection, access to the apex, apical curettage, root-end resection, root-end preparation and filling, flap replacement, and suturing.

6. Discuss the indications for each procedure listed in Objective 4.

7. Discuss the prognosis for each procedure listed in Objective 4.

8. State the principles of flap design.

9. Diagram the various flap designs and describe the indications, advantages, and disadvantages of each.

10. List the more common root-end filling materials.

11. Review the basic principles of suturing.

12. Describe general patterns of soft and hard tissue healing.

13. Write out instructions to be given to the patient concerning postoperative care after endodontic surgery.

14. List and describe conditions that indicate referral to a specialist for evaluation or treatment.

Nonsurgical root canal therapy is a highly successful procedure if diagnosis and technical aspects are carefully

performed.[1,2]

There is a common belief that if root canal therapy fails, surgery is indicated for correction. This is not necessarily true; most failures are best corrected by retreatment (revision). Studies have shown that more than two-thirds of retreated cases are successful after retreatment of the original root canal therapy.[3-5] There are, however, situations in which surgery is necessary to retain a tooth that would otherwise be extracted.[6]

Endodontic surgery is not “oral surgery” in the traditional sense. Rather, it is actually “endodontic treatment through a surgical flap.” Simply cutting off the apex of a root and placing a filling in the vicinity of the canal does not accomplish the goals of endodontic surgical treatment. The purposes of endodontic surgery include sealing of all portals of exits to the root canal system and the isthmuses, eliminating bacteria and their byproducts from contaminating the periradicular tissues, and providing an environment that allows for regeneration of periradicular tissues.

During the past decade, the art and science of endodontic surgery have changed dramatically. Endodontic surgery was previously limited to the anterior teeth where access was considered adequate. With the introduction of the operating microscope, ultrasonic tips, and new root-end filling materials, teeth that might otherwise be extracted now have a chance for retention.[7]

This chapter describes both the indications and the procedures involved in incision for drainage, periapical surgery, and corrective surgical procedures such as root amputation, hemisection, and bicuspidization.

INCISION FOR DRAINAGE

The objective of incision for drainage is to evacuate exudates and purulence from a soft tissue swelling. Incision for drainage reduces discomfort resulting from the buildup of pressure and speeds healing.

Indications

The best treatment for swelling originating from a symptomatic apical abscess of pulpal origin (Figure 20-1, *A*) is to establish drainage through the offending tooth (Figure 20-1, *B*). When adequate drainage cannot be accomplished through the tooth itself, drainage is obtained through soft tissue incision. Occasionally, drainage is performed through the soft tissue even if it has also been obtained through the tooth (Figure 20-1, *C*). The reason is that there may be separate noncommunicating abscesses—one at the apex and another in a submucosal location or in an anatomic space.

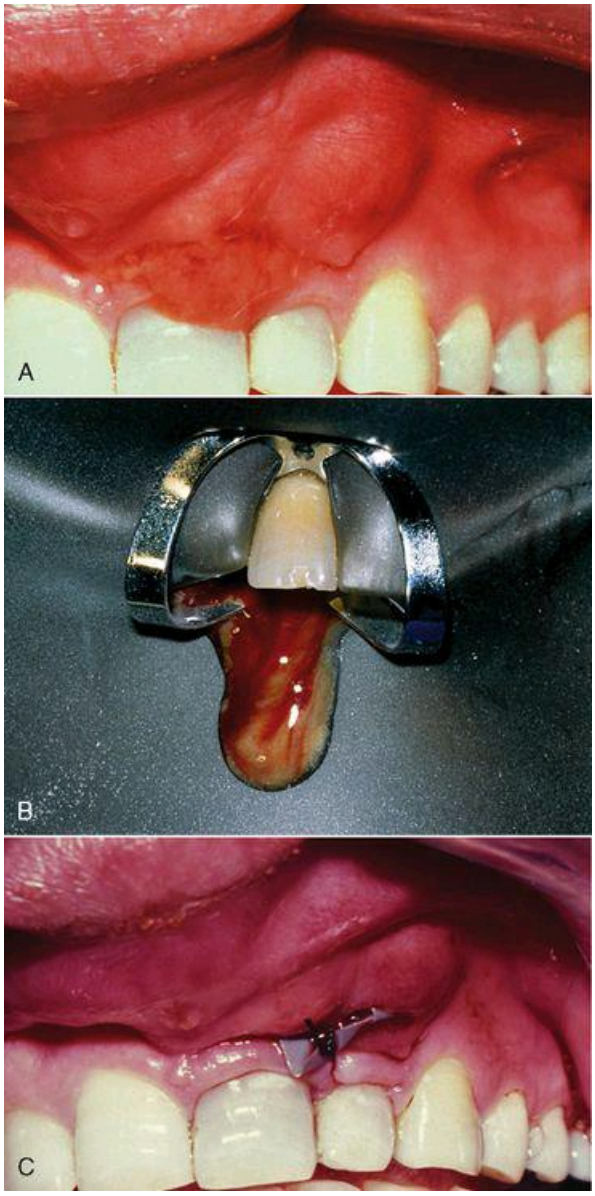

Figure 20-1 A, Fluctuant swelling is present as a result of presence of an infection in the right lateral incisor. **B,** Establishment of drainage through an offending tooth. **C,** Incision for drainage is made horizontally into the swelling, and a rubber drain is sutured in place to prevent immediate closure of the incision.

Drainage through the soft tissue is accomplished most effectively when the swelling is fluctuant. A fluctuant swelling is a fluid-containing mass in which a wavelike sensation (like pushing on a water balloon) is felt when pressure is applied (see Figure 20-1, *A*). Incising a fluctuant swelling releases purulence immediately and provides rapid relief. If the swelling is nonfluctuant or firm, incision for drainage often result in drainage of only blood and serous fluids. Incision and drainage of a nonfluctuant abscess reduces pressure and facilitates healing by reducing irritants and increasing circulation in the area.

Contraindications

There are relatively few contraindications to the use of incision for drainage. Patients with prolonged bleeding or clotting times must be approached with caution, and hematological screening is often indicated. An abscess in or near an anatomic space should be handled very carefully.

Procedures

Anesthesia

Profound anesthesia is difficult to obtain in the presence of inflammation, swelling, or exudates. Because direct subperiosteal infiltration is ineffective and may be quite painful, regional block anesthetic techniques are preferred. Mandibular blocks for posterior areas, bilateral mental blocks for the anterior mandible, posterior superior alveolar blocks for the posterior maxilla, and infraorbital blocks for the premaxilla area are the preferred choices. These injections may be supplemented by regional infiltration.

In addition to block anesthesia, one of the following methods may also be used. The first technique is infiltration that starts peripheral to the swelling. After the application of topical anesthetic, the solution is injected *slowly* with limited pressure and depth, and this is followed by additional injections in previously anesthetized tissue, moving progressively closer to the center of the swelling. This procedure results in improved anesthesia without extreme discomfort.

The second technique is the use of topical ethyl chloride.[8] A stream of this solution is directed onto the swelling from a distance, permitting the liquid to volatilize on the tissue surface. Within seconds, the tissue at the site of volatilization turns white. The incision is quickly accomplished with continued ethyl chloride spray. This topical anesthesia is a supplement to block anesthesia when a quick incision is required. If none of these procedures work, intravenous (IV) sedation can be used for incisions and drainage.

Incision

After anesthesia, the incision is made vertically with a No. 11 scalpel. Vertical incisions are parallel with the major blood vessels and nerves and leave very little scarring. The incision should be made firmly through periosteum to bone. If the swelling is fluctuant, pus usually flows immediately, followed by blood. If the swelling is nonfluctuant, the predominant drainage is blood.

Drainage

After the initial incision, a small closed hemostat may be placed in the incision and then opened to enlarge the draining tract.[9] This procedure is indicated with more extensive swellings. To establish drainage, an I-shaped or “Christmas tree” drain cut from a rubber dam or a piece of iodoform gauze can be placed (suturing is optional) in the incision. The drain should be removed after 2 to 3 days; if it is not sutured, the patient may remove the drain at home.

PERIAPICAL SURGERY

Periapical surgery (PAS) is commonly performed to remove a portion of the root with undébrided canal space or to seal the canal apically when a complete seal cannot be accomplished with nonsurgical root canal treatment through the crown approach.

Indications

The main indications for PAS are anatomic problems, procedural accidents, irretrievable materials in the root canal, symptomatic cases, and horizontal apical fracture, as well as biopsy and corrective surgery.

Anatomic Problems

A nonnegotiable, blocked canal, or severe root curvature may prevent adequate cleaning and shaping or obturation. Nonsurgical, as well as surgical, endodontic treatments are indicated in these cases (Figure 20-2). Nonsurgical root canal therapy or revision (if possible) before surgery improves the surgical success rate.[10,11] However, if neither is feasible, removal of the uninstrumented and unfilled portion of the root or performing PAS may be necessary (Figure 20-3). Anatomic perforation of the root apex through the bone (fenestration), although infrequent, may necessitate PAS after root canal treatment. Beveling the root apex and placing it within the bone correct this condition. Occasionally, adequate root canal therapy is compromised by extensive apical root resorption. It may then be necessary to expose the root, remove the resorbed area, and repair it.

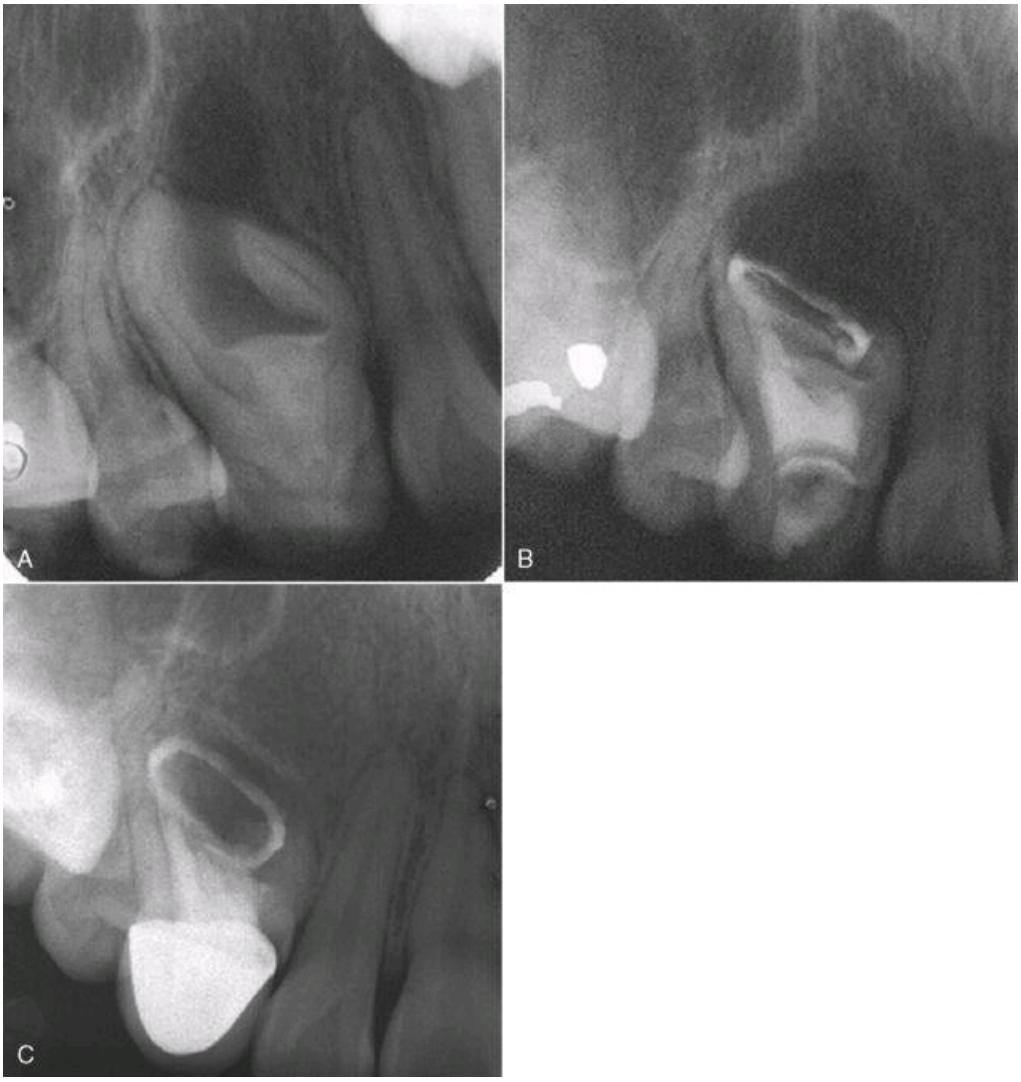

Figure 20-2 A, Preoperative radiograph of the right region shows presence of dens invaginatus (dens in dente) in the cuspid tooth. **B,** Because of the presence of this anomaly in the cuspid and a large lesion, as well as our inability to perform nonsurgical root canal treatment, a periapical surgery was performed on this tooth. **C,** Postoperative radiograph taken 20 months later shows complete resolution of the lesion in the cuspid.

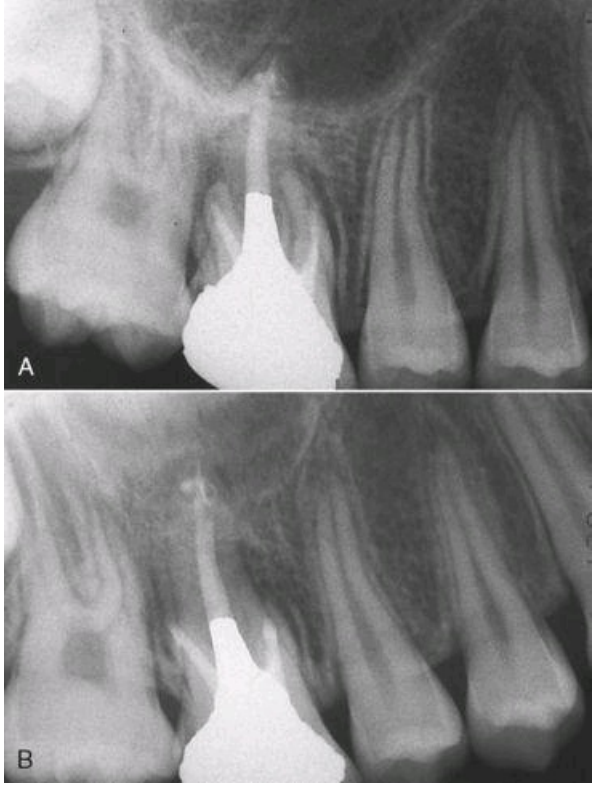

Figure 20-3 A, A nonnegotiable ledge is present in the mesiobuccal root of the first maxillary molar. **B,** An endodontic surgery was performed to correct this accidental procedure on this root and deficiencies present in the distobuccal root.

Procedural Accidents

Separated instruments, ledging, perforations, and gross overfills may cause failure of root canal treatment, which will require surgical intervention. If symptoms or lesions develop or persist after the accidents, PAS is usually necessary (Figure 20-4). (See Chapter 18 for procedural accidents.)

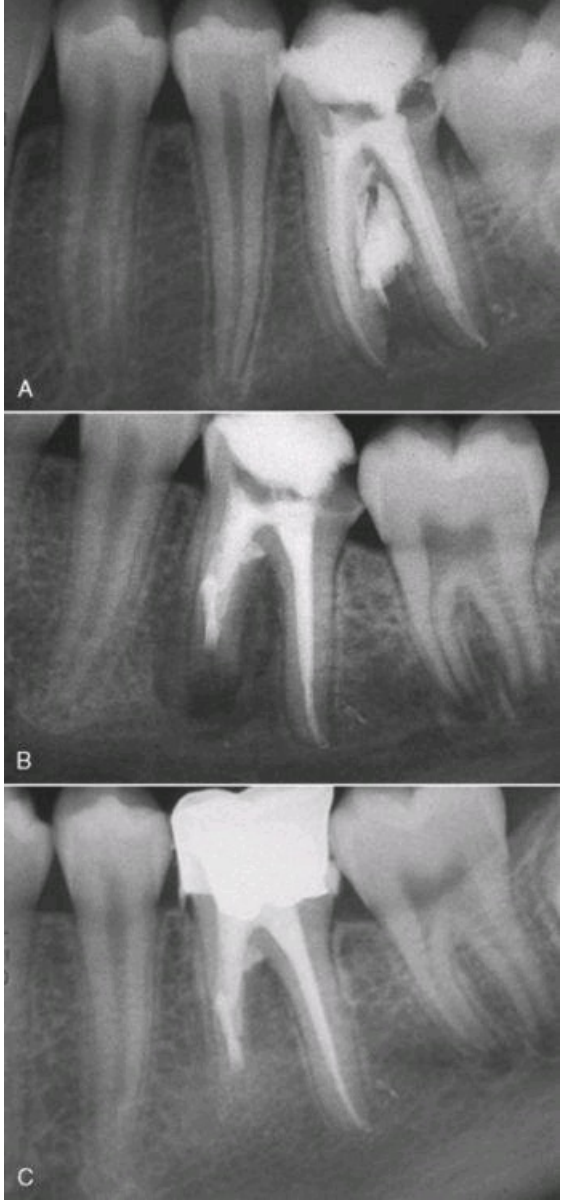

Figure 20-4 A, Preoperative radiograph of the mandibular first molar shows presence of “strip” perforation in the mesial roots and extrusion of sealer into the furcation region. **B,** Because of the presence of this accidental procedure, a periapical surgery was performed on this tooth. **C,** Postoperative radiograph taken 18 months later shows complete resolution of the lesion.

Irretrievable Materials in the Root Canal

Retreatment (revision) is recommended for treatment of failures. However, irretrievable posts or dowels or root filling materials, such as silver cones, amalgam, or nonabsorbable pastes, often prevent revision or their removal would result in further damage to the root structure. The best alternative is a surgical approach and placement of a root-end filling material (Figure 20-5).

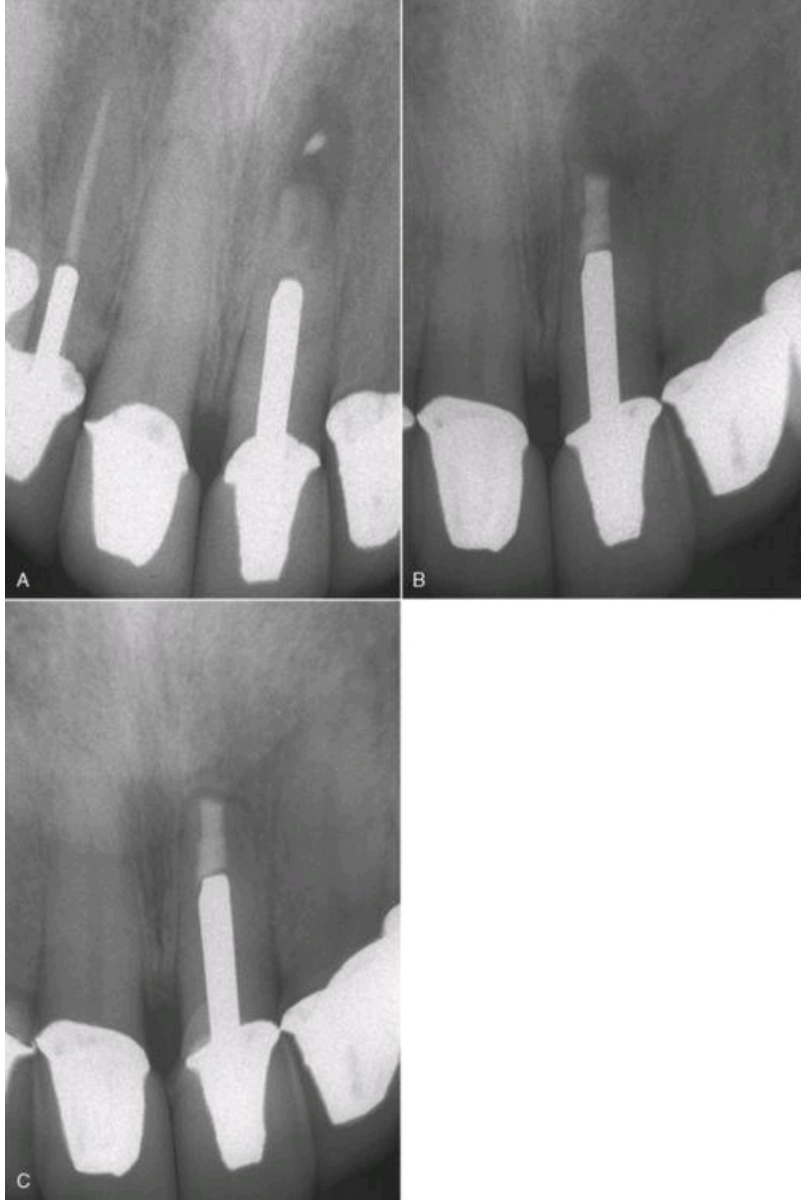

Figure 20-5 A, Failed root canal treatment in the left maxillary lateral incisor requires periapical surgery. **B,** The root end is resected; a cavity is prepared and filled with MTA. **C,** Postoperative film after 1 year showing complete healing.

(Courtesy Dr. R. Rubinstein.)

Symptomatic Cases

Most symptoms disappear after complete cleaning and obturation of root canals. However, in cases where symptoms persist after meticulous performance of these procedures, PAS should be considered to identify the cause or causes for the persistence of the symptoms. The main cause for the persistence of pain in these cases is usually presence of inflammation that results from the inability of the operator to completely clean the root canal or canals. Exploratory surgery may identify undetected vertical root fractures, additional apical and lateral foramina (possible missed canals), perforations, apical ramifications, overfills, or other causes of failure. Once the cause has been eliminated, the symptoms disappear.

Horizontal Apical Fracture

Although most traumatic horizontal apical fractures usually heal without intervention, occasionally the apical portion of a root becomes necrotic and cannot be treated nonsurgically. In these cases, the apical portion of the root should be removed and the apical seal should be evaluated.

Biopsy

Although most periapical lesions are of pulpal origin, nonpulpal lesions do exist (see Chapter 5 for diagnosis and treatment planning). Presence of a vital pulp in a tooth with a radicular radiolucency (Figure 20-6), undefined periapical lesions in teeth

with vital pulps in patients with a history of previous malignancy, or lip paresthesia or anesthesia are indications for biopsy.

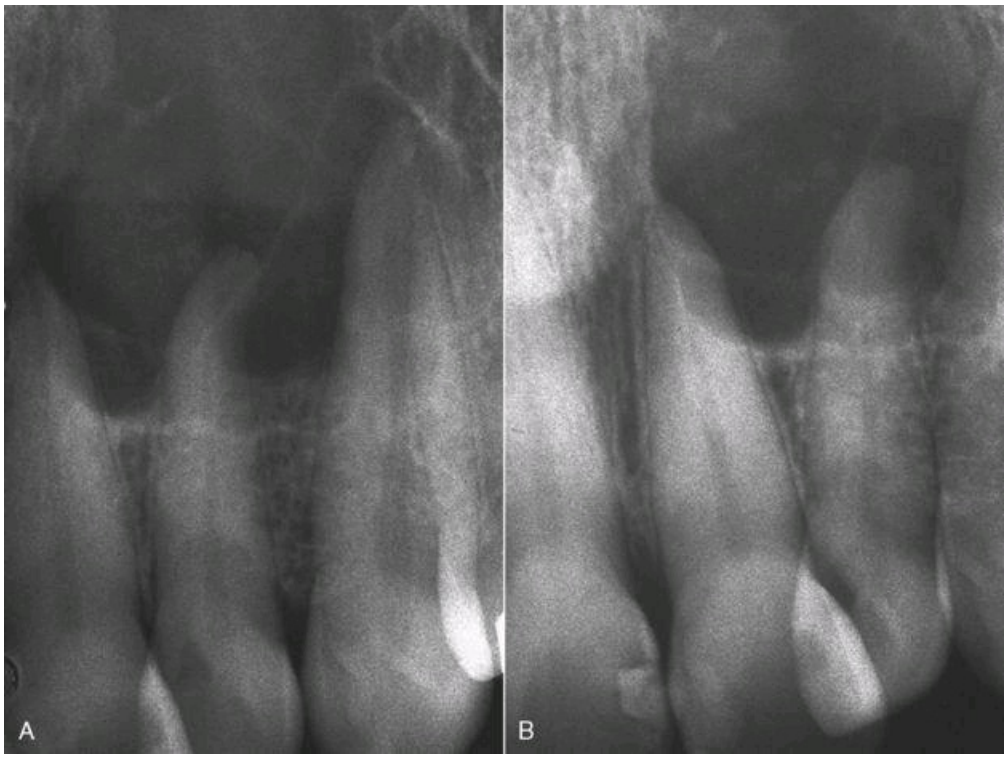

Figure 20-6 A, Presence of vital pulps in the left anterior teeth and a multilocular large radiolucency indicated presence of a possible lesion of a nonpulpal origin. **B,** Another radiograph shows the extent of this lesion. A biopsy revealed the presence of a keratocyst.

Contraindications

The four major contraindications for PAS are (1) anatomic factors, (2) medical or systemic complications, (3) indiscriminate use of surgery, and (4) an unidentified cause of treatment failure.[9]

Anatomic Factors

Inaccessibility to surgical site because of tooth location, spaces such as maxillary sinus or nasal fossa, unusual bony configuration, or proximity of neurovascular bundles may be contraindications or at least require caution or special approaches (Figure 20-7). For example, a thick external oblique ridge associated with a mandibular molar or apices contiguous with the mandibular canal may compromise surgical access. Other situations that may contraindicate PAS or modify the approaches used include very short root length (precluding root-end re-section), severe periodontal disease (prognosis hopeless, even with surgery), or unrestorable teeth.

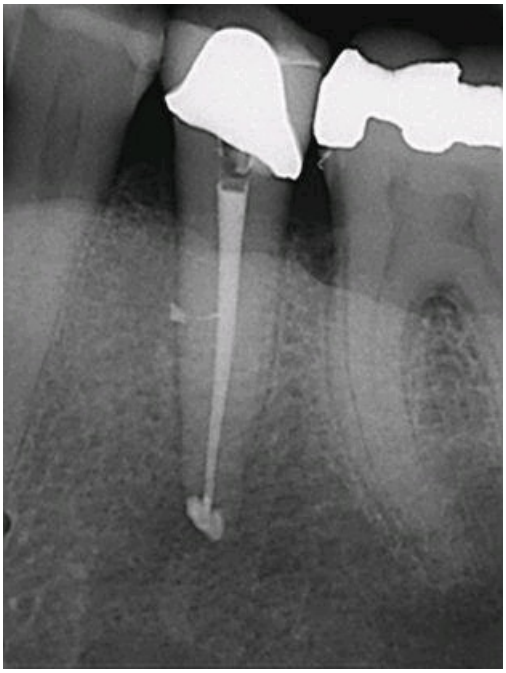

Figure 20-7 Proximity of the apex of the mandibular first premolar to the neurovascular bundle dictates caution during endodontic surgery.

Medical or Systemic Complications

Serious systemic health problems or extreme apprehension make the patient a poor candidate for PAS. Surgery may also be contraindicated in patients with blood disorders, terminal disease, uncontrolled diabetes, or severe heart disease and for those whose immune systems are compromised.

Indiscriminate Use of Surgery

As previously stated, surgery is not indicated when a nonsurgical approach would probably result in success. The practice of managing all accessible periapical lesions or large periradicular lesions surgically is unethical and contraindicated.

Unidentified Cause of Treatment Failure

Using surgery to correct a treatment failure for which the cause cannot be identified is unlikely to be successful.

Recent Advances in Endodontic Surgery

Many advances in surgical technique and instrumentation have occurred over the past decade.[7] These include enhanced magnification and illumination, ultrasonic tips, microinstruments, and newer root-end filling materials. Enhanced illumination and magnification have greatly improved the procedures that practitioners can perform. Magnification in endodontic surgery has led to miniaturization of endodontic surgical instruments. Developments in root-end filling materials have increased both quality and biocompatibility of apical seals. Together, these advances have significantly improved the state of the art and science of endodontic surgery, giving a second chance to a tooth that was considered for extraction.

Procedures Involved in Periapical Surgery

The typical sequence of procedures used in PAS are flap design, incision and reflection, apical access, periradicular curettage, root-end resection, root-end cavity preparation, root-end filling, flap replacement and suturing, postoperative care and instructions, and suture removal and evaluation.

Flap Design

The first step in PAS is designing a flap that allows adequate exposure to the surgical site of the surgery for the operator. The following general guidelines and principles should be used during flap design[12,13]:

1. The flap should be designed for maximum access to the site of surgery.

2. Adequate blood supply to the reflected tissue is maintained with a wide flap base.

3. Incisions over bony defects or over the periradicular lesion should be avoided; these might cause postsurgical soft tissue fenestrations or nonunion of the incision.

4. The actual bony defect is larger than the size observed radiographically.

5. A minimal flap, which should include at least one tooth on either side of the intended tooth, should be used.

6. Acute angles in the flap must be avoided. Sharp corners are difficult to reposition and suture and may become ischemic and slough, resulting in delayed healing and possibly scar formation.

7. Incisions and reflections include periosteum as part of the flap. Any remaining pieces or tags of cellular nonreflected periosteum will hemorrhage, compromising visibility.

8. The interdental papilla must not be split (incised through) and should be either fully included or excluded from the flap.

9. Vertical incisions must be extended to allow the retractor to rest on bone and not crush portions of the flap.

Although there are numerous flap designs, two meet most periapical surgery needs: the submarginal (curved, triangular, and rectangular) and the full mucoperiosteal (triangular and rectangular) flaps.

Submarginal Curved Flap

The submarginal curved flap is a slightly curved, half-moon–shaped, horizontal incision made in the attached gingiva with the convexity nearest the free gingival margin (Figure 20-8). It is simple and easily reflected and provides access to the apex without impinging on the tissue surrounding the crowns. Its disadvantages include restricted access with limited visibility, tearing of the incision corners if the operator tries to improve access by stretching the tissue, and leaving the incision directly over the lesion if the surgical defect is larger than anticipated. The incision margins of this flap frequently heal with scarring.[14] The submarginal curved flap is limited by the presence of the frenum, muscle attachments, or canine and other bony eminences. Because of its many disadvantages, this design is generally not indicated or used.

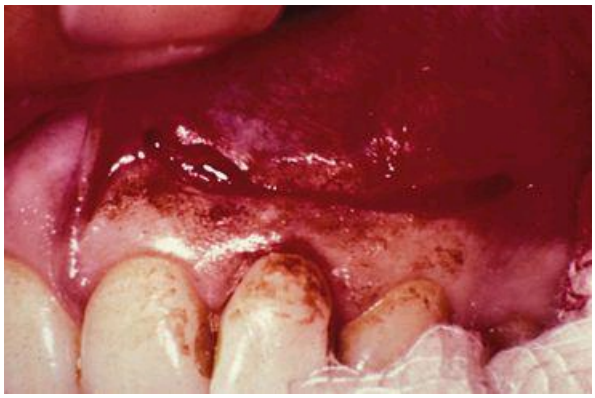

Figure 20-8 A submarginal curved (semilunar) flap is made in the attached gingiva to perform a periapical surgery on the left lateral incisor.

Submarginal Triangular and Rectangular Flaps

Triangular or rectangular flaps are known as modified submarginal curved flap. A scalloped horizontal incision (Ochsenbein-Luebke) is made in the attached gingiva with one or two accompanying vertical incisions (Figure 20-9). This flap is used most successfully in the maxillary anterior teeth with crowns. An alternative submarginal flap design to this is the papilla-based incision, in which the interdental papillae are left intact.[15] Prerequisites are 4.0 mm of attached gingiva and good periodontal health.

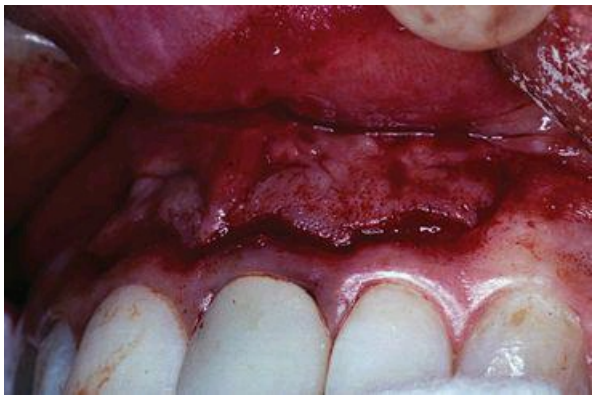

Figure 20-9 Scalloped horizontal incision (OchsenbeinLuebke) is made in the attached gingiva with two accompanying vertical incisions to perform a surgery on the left central incisor.

This flap design provides better access and visibility compared to the submarginal curved flap and has less risk of incising tissue over a bony defect. Disadvantages are possible scarring and hemorrhaging from the cut margins to the surgical site.[16] It also provides less visibility than the full mucoperiosteal flap.

Full Mucoperiosteal Flap

The full mucoperiosteal (sulcular) flap consists of an incision at the gingival crest with full elevation of the interdental papillae, free gingival margin, attached gingiva, and alveolar mucosa. It may have either a single (triangular) or double (rectangular) vertical-releasing incision (Figure 20-10). It allows maximal access and visibility, precludes incising over a bony defect, and has fewer tendencies toward hemorrhage. This design permits periodontal curettage, root planing, and bony reshaping and heals with minimal scar formation. Its disadvantages include the difficulty of replacement, suturing, and making alterations (height and shape) to the free gingival margin, as well as possible gingival recession after surgery and exposing the crown margins.[14,17]

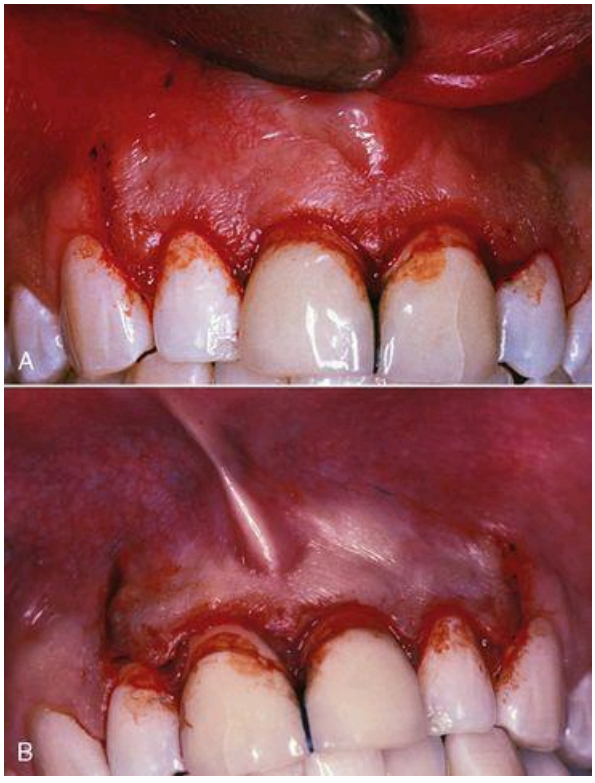

Figure 20-10 A, Triangular full mucoperiosteal (sulcular) flap, with one vertical incision made to access the right central incisor. **B,** Rectangular full mucoperiosteal flap, with two vertical incisions made to access both central incisors.

Incision and Reflection

A firm incision is made with a CK-2, CK-3, or other suitable blade into the base of the sulcus or initiation of the horizontal incision. To prevent tearing during reflection, the incision must be made through periosteum to bone. Once the horizontal incision has been made, the same blade or a No. 15 can be used to place the vertical incision or incisions. The tissue is reflected with a sharp periosteal elevator. Because periosteum is reflected as part of the flap, the elevator must firmly contact bone as the tissue is relieved, using firm controlled force. The tissue is reflected beyond the mucogingival junction to a level that will provide adequate access to the root apex, provide visibility of the surgical site, and allow a retractor to be placed on sound bone (Figure 20-11).

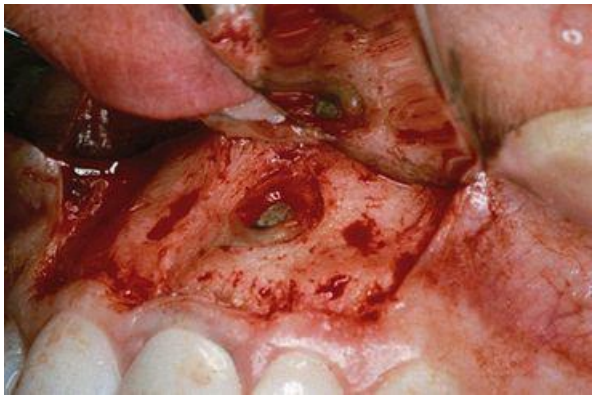

Figure 20-11 The flap is reflected with a periosteal elevator and held with a retractor to allow visibility and access to the surgical site. The retractor must be placed on sound bone.

Osteoectomy

In many cases, the presence of a periradicular lesion creates a defect in the cortical bone that is visible after flap reflection or is identified when firm probing with an explorer is applied on the bone. If the opening is small, a sharp round bur can be used to remove the bone until the apex is located (Figure 20-12). If there is limited cortical bone destruction, after placement of a radio opaque object near the apex, a radiograph should be taken to locate the apex. Removal of bone with a bur is performed by a light brushing motion in the presence of copious sterile saline irrigation.[12,13,18]

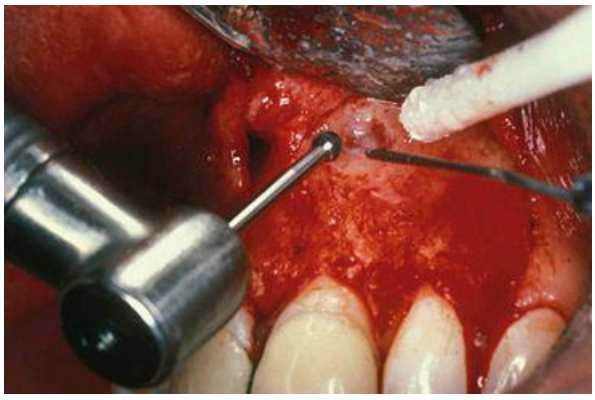

Figure 20-12 A sharp round bur in a high-speed handpiece is used to remove the bone, with continuous spray of sterile water to locate the tooth apex.

Periradicular Curettage

Removal of pathologic soft tissue surrounding the apex does the following:

- Provides access and visibility of the apex

□ Removes inflamed tissue

□ Provides a biopsy specimen for histological examination

□ Reduces hemorrhage

The tissue should be carefully peeled out, ideally in one piece, with a suitably sized sharp curette (Figure 20-13). This process should leave a clean bony cavity. When the lesion is very large, portions of tissue can be left without compromising the blood supply to an adjacent tooth. This should not affect periradicular healing.

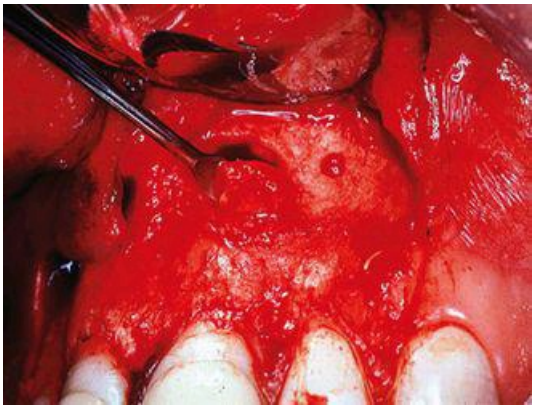

Figure 20-13 Apical curettage and removal of diseased tissue at the apex enhance visualization of the apex and surrounding bone. This tissue should be submitted for histologic evaluation.

Root-End Resection

Root-end resection involves beveling the apical portion of the root. This step is often an integral part of PAS and serves the following two purposes:

□ It removes the untreated apical portion of the root and enables the operator to determine the cause of failure.

□ It provides a flat surface to prepare a root-end cavity preparation and pack it with a root-end filling material.

Apical sectioning is done with a tapered fissure bur in a high-speed handpiece and copious sterile saline irrigation (Figure 20-14). The bevel should be made at as close to 0 degrees in a faciolingual direction as possible to still enable maximum visibility to the root apex.[12,13,16] In general, the amount of root removed depends on the reason for performing the root-end resection. However, sufficient resection must be performed to do the following:

□ Provide access to the palatal-lingual root surface

□ Place the canal in the center of the sectioned root

□ Expose additional canals, apical deltas, or fractures

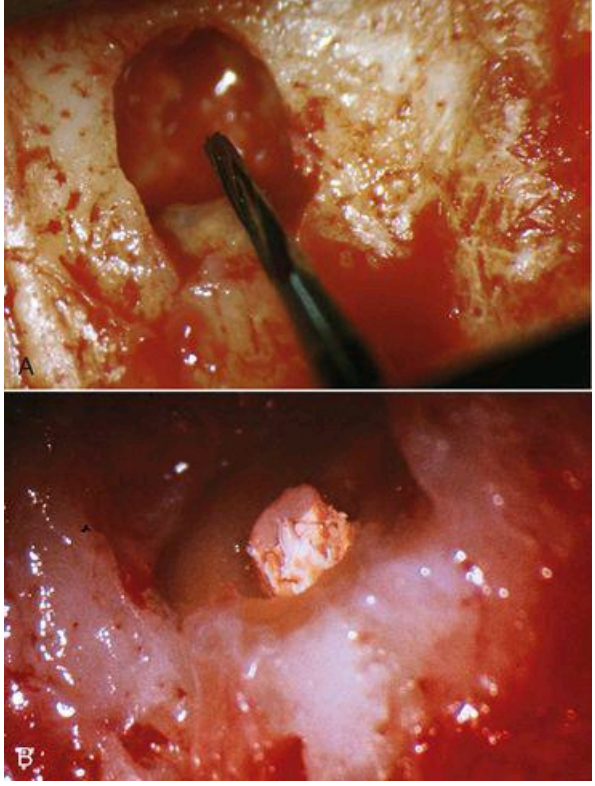

Figure 20-14 A, Root-end resection (apicoectomy) is performed using a fissure bur in a high-speed handpiece. **B,** The entire resected portion of the tooth should be visible.

Root-End Cavity Preparation and Filling

Root-end cavity preparation and filling are indicated in most endodontic surgeries. Apical preparations are now made with ultrasonic tips. A variety of tips are available to accommodate virtually all access situations (Figure 20-15). When used, they are placed in the long axis of the root so that the walls of the preparation will be parallel with long access of the root. A class I type preparation is made with ultrasonic tips to a minimum depth of 3 mm into the canal[16,19,20] (Figure 20-16). More complicated apical root anatomy may require other types of preparation.[12] The ultrasonic instrument offers advantages of control and ease of use and permits less apical root beveling and uniform depth of preparation.[19-21] In addition, the ultrasonic tips produce smaller apical preparations, allow easier preparation of isthmus, follow the direction of the canals (Figure 20-17), clean the canal surfaces better than burs, and create less fatigue for the operator.

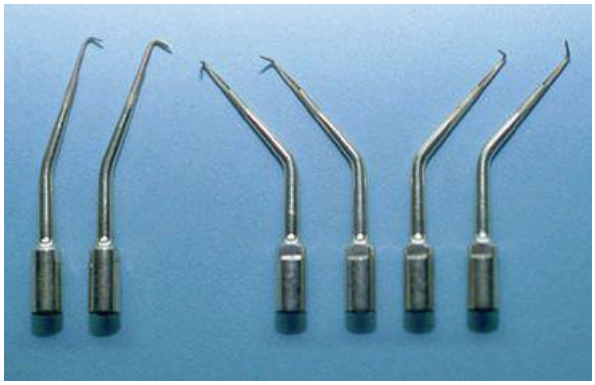

Figure 20-15 A variety of ultrasonic tips are available to prepare root-end cavities for various roots.

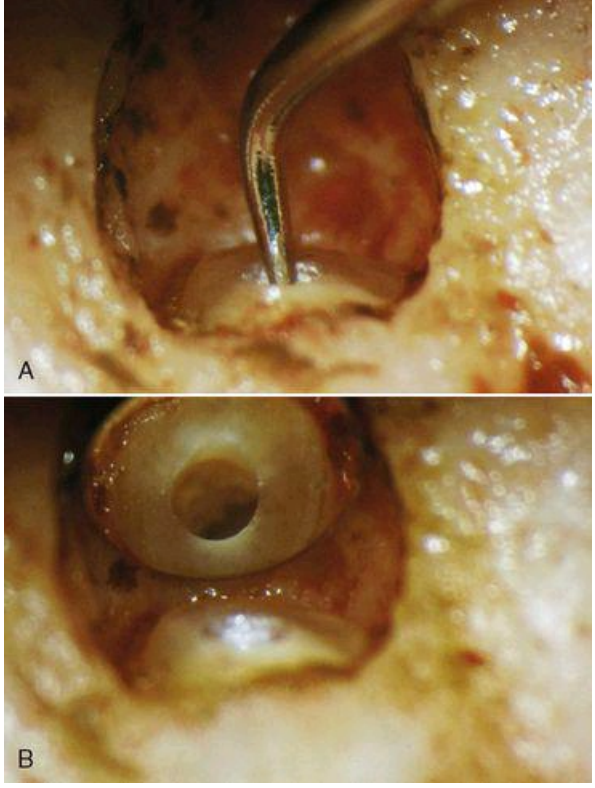

Figure 20-16 A, An ultrasonic tip is used to prepare a class I preparation in the apical portion of the root canal. **B,** A mirror view of a root-end cavity preparation.

(Courtesy Dr. R. Rubinstein.)

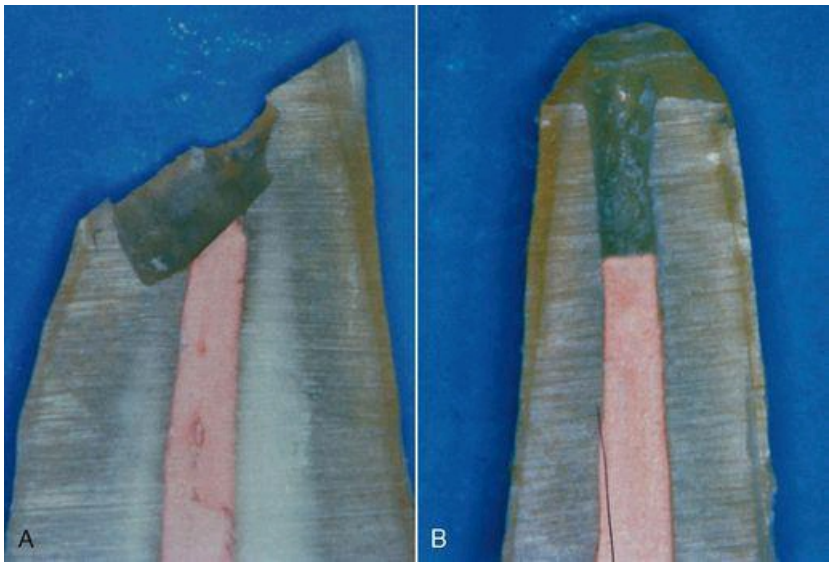

Figure 20-17 A, Root-end cavity prepared by a small inverted bur. **B,** Root-end cavity prepared by an ultrasonic bur.

After the apical preparation is made and thoroughly examined, it should be filled with a root-end filling material (Figure 20-18). Root-end filling materials should:

1. Seal well
2. Be biocompatible
3. Be unresorbable
4. Be easily inserted
5. Be unaffected by moisture
6. Be visible radiographically

7. Be capable of regenerating periradicular tissues

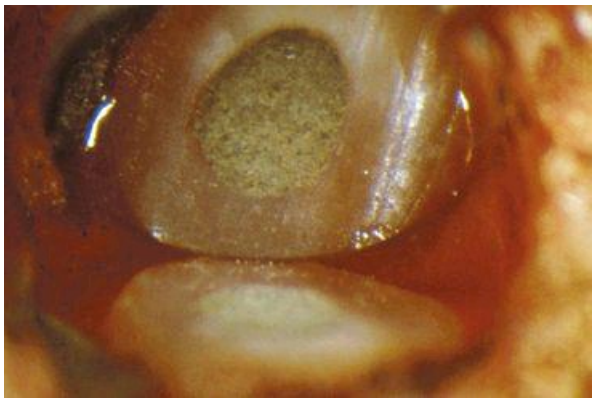

Figure 20-18 MTA (3 mm) is placed in the root-end cavity preparation to provide a fluid-tight apical seal.

(Courtesy Dr. R. Rubinstein.)

Many materials have been used as root-end filling materials.[22-30] Root-end filling materials that are the consistency of cement, such as SuperEBA (Boswoth, Skokie, Ill) and ProRoot MTA (Dentsply, Tulsa Dental, Johnson City, TN), are currently the materials of choice.[7] In several studies, histologic sections demonstrate the regeneration of new cementum over the mineral trioxide aggregate (MTA) root-end filling,[26,27,31] which is a phenomenon not seen with other commonly used root-end filling materials (Figure 20-19).

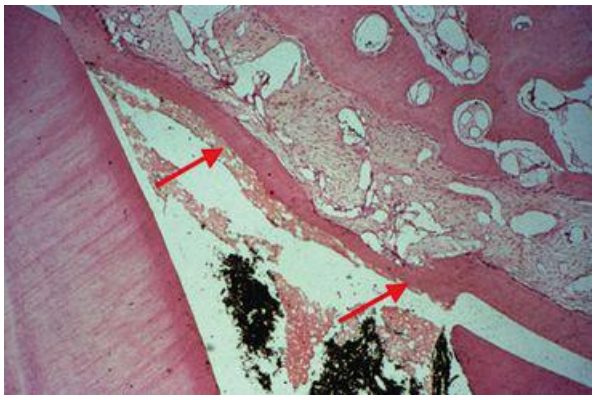

Figure 20-19 Complete periapical healing and formation of cellular cementum *(arrows)* adjacent to MTA when used as a root-end filling material in monkeys.

Flap Replacement and Suturing

After placing a root-end filling material and taking a radiograph, the flap should be placed in its original position and held in place for 5 minutes using moderate digital pressure with moistened gauze. This allows expression of hemorrhage from under the flap, initial adaptation, easier suturing, and less postoperative swelling and bleeding.

Suturing is commonly done with 5-0 Tevdek, although other materials are acceptable.[32-34] There are many suturing techniques, including interrupted, continuous mattress, and sling sutures.[13] Interrupted sutures are commonly used (Figure 20-20, *A*). When suturing, the needle passes first through reflected and then through attached tissue. The sutures are tied with a simple double surgeon's knot. The knot should not be placed over the incision line because it collects debris and bacteria that will promote inflammation, infection, and delayed healing. The sutures are usually removed 3 to 7 days after surgery (Figure 20-20, *B*).

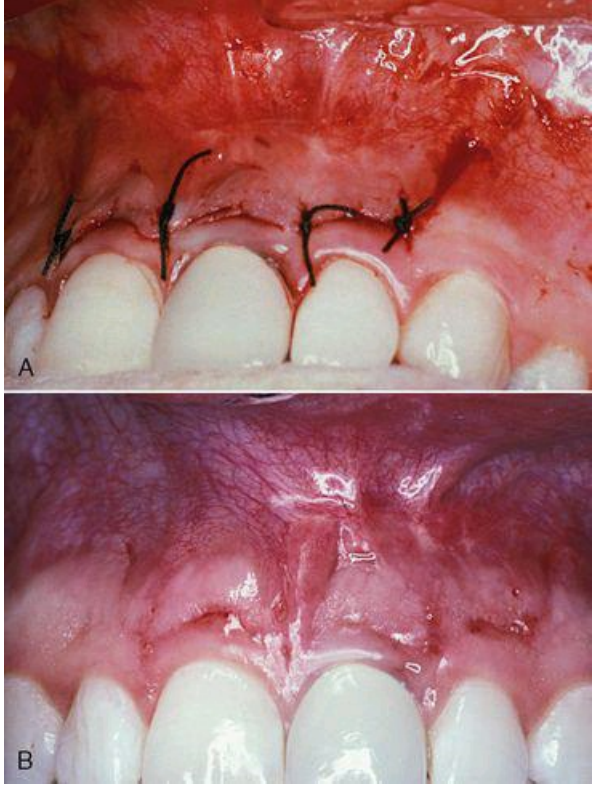

Figure 20-20 A, Interrupted sutures are commonly used to hold the soft tissue flap in its original location. **B,** Sutures are removed 3 to 7 days after surgery.

Postoperative Care and Instructions

Both oral and written postoperative instructions should be given to the patient. Instructions should be written in simple, straightforward language. They should minimize patient anxiety arising from normal postoperative symptoms by describing how to promote healing and comfort.

The following instructions are for patients.

1. Some swelling and discoloration are common. Use an ice pack with moderate pressure on the outside of your face (20 minutes on, 5 minutes off) until you go to bed tonight. Application of ice and pressure decrease bleeding and swelling and provide an analgesic effect.

2. Some oozing of blood is normal. If bleeding increases, place a moistened gauze pad or facial tissues over the area and apply finger pressure for 15 minutes. If bleeding continues, call the doctor's office.

3. Do not lift your lip or cheek to look at the area. The stitches are tied, and you may tear them out.

4. Starting tomorrow, dissolve one teaspoon of salt in a glass of warm water and *gently* rinse your mouth three or four times daily. Rising with a mouthwash, such as 0.12% chlorhexidine, can promote healing. Careful brushing is important, but vigorous brushing may damage the area. Tonight you should brush and floss all areas except the surgery site. Tomorrow night *carefully* brush the surgery site.

5. Proper diet and fluid intake are essential after surgery. Eat a soft diet and chew on the opposite side of your mouth. Drink lots of fluids and eat soft foods such as cottage cheese, yogurt, eggs, and ice cream.

6. Pain is usually minimal after PAS, and strong analgesics are normally not required. Some discomfort is normal. If pain medication was prescribed, follow the instructions. If no medication was prescribed, take your preferred nonprescription pain remedy if needed. If this is not sufficient, call the doctor's office.

7. If you are a smoker, do not smoke for the first 3 days after the procedure.

8. If you experience excessive swelling or pain or if you run a fever, call the doctor's office immediately.

9. Keep your appointment to have the stitches removed. (Note to practitioners: Sutures are removed 3 to 7 days after surgery.)

10. Call the doctor’s office if you have any concerns or questions.

Healing

Surgery involves the manipulation of soft and hard tissues. Handling of both soft tissues (periosteum, gingiva, periodontal ligament, and alveolar mucosa) and hard tissues (dentin, cementum, and bone) is accomplished by incision, dissection, and excision.

Soft Tissue

The healing involves clotting, inflammation, epithelialization, and connective tissue healing, as well as maturation and remodeling.[35] Clotting and inflammation consist of both chemical and cellular phases. The clotting mechanism is important because it is based on the conversion of fibrinogen to fibrin. Under pressure, the clot should be a thin layer. Failure of the clot to form results in leakage of blood into the wound site. The inflammatory components of healing are a complex network of both extrinsic and intrinsic elements.[35]

Initial epithelial healing consists of the formation of the epithelial barrier, which is made up of layers of epithelial cells that depend on the underlying connective tissue for nutrients. This epithelial layer migrates along the fibrin surface until it makes contact with epithelial cells from the opposite border of the wound, forming an epithelial bridge.

The connective tissue component comes from fibroblasts, which are differentiated from ectomesenchymal cells and are attracted to the wound site. Adjacent blood vessels provide nutrients for the fibroblasts and their precursors, which elaborate collagen, initially type III, followed by type I. Macrophages are an important part of these processes. As healing matures, there is a decrease in the amount of inflammation and numbers of fibroblasts, accompanied by deaggregation and reaggregation of collagen with formation of collagen fibers into a more organized pattern.[35-38]

Hard Tissue

As with healing in soft tissue, the hard tissue response is also based on the presence of cells such as fibroblasts, osteoblasts, and cementoblasts, which produce ground substance, cementum, and bone matrix formation. New cementum deposition from cementoblasts begins about 12 days after surgery; eventually, a thin layer of cementum may cover resected dentin and even certain root-end filling materials (see Figure 20-19). The exposed dentin acts as an inductive force, with new cementum forming from the periphery to the center.

Osseous healing begins by the proliferation of endosteal cells into the coagulum of the wound site. At 12 to 14 days, woven trabeculae and osteocytes appear, leading to early maturation of the collagen matrix at about 30 days. This process occurs from inside to outside, ending in the formation of mature lamellar bone,[39-41] which is visible radiographically (see Figure 20-5, *C*).

CORRECTIVE SURGERY

Corrective surgery procedures are especially designed to correct pathologic or iatrogenic entities.

Indications

Procedural Errors

Root perforations typically occur during access, canal preparation, or restorative procedures (usually post placement). They require restorative and endodontic management. Most perforations can be managed using MTA (see Chapter 18). However, there are some cases that need to be managed surgically.

The location of the perforation is often the factor limiting the success of surgical treatment. If the defect is on the proximal root surfaces in close proximity to adjacent teeth, repair is a problem because access to the site is difficult without damaging adjacent teeth (Figure 20-21). This is particularly true of the lingual surface of mandibular teeth. However, defects on the facial surface are easier to treat.

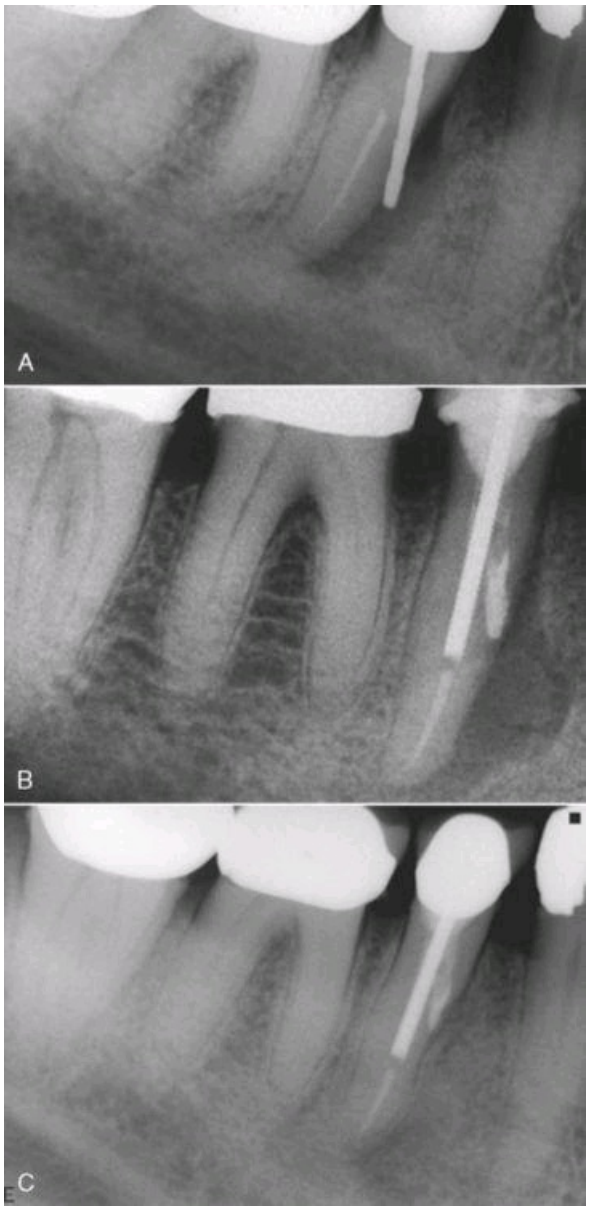

Figure 20-21 Perforation repair. **A,** An off-center post has perforated the root and caused a bony lesion. **B,** Internal and external perforation repairs with MTA. **C,** Complete repair of the bony lesion in 3 years.

(Courtesy Dr. N. Chivian.)

Resorptive Perforations

Resorptive root perforations typically occur as a result of trauma or internal bleaching procedures. The defects may be localized to the root surface or communicate with the canal.

Repair of these defects presents unique problems. Often a defect on the root surface wraps onto the palatal or lingual surface, compromising access and homeostasis. Repair can be accomplished with various materials. If the field can be kept dry, glass ionomer, dentin-bonding agent with composite resin, or White ProRoot MTA can be used. Esthetically pleasing materials are preferred for facial repairs because dark materials, such as amalgam or gray MTA, may stain the teeth.

If a post perforates the root, it must be reduced so that it is well within the root structure. Then the defect is repaired with ProRoot MTA.

Repairs in the cervical portion of the root are often difficult to manage and maintain because communication with the gingival sulcus leads to periodontal breakdown. This often means that guided bone regeneration (GBR) procedures,[42] combined with the endodontic surgical treatment, periodontal treatment (such as crown lengthening), orthodontic extrusion, or a combination thereof, may be necessary in conjunction with the repair.[43]

ROOT AMPUTATION, HEMISECTION, AND BICUSPIDIZATION

The three categories described previously, incision for drainage, PAS, and corrective surgery, involve cutting bone, soft tissue, and root. This last category involves resecting the root(s) and crown.[44] Box 20-1 lists the indications and contraindications for root amputation, hemisection, and bicuspidization.

Box 20-1 Indications and Contraindications for Root Amputation, Hemisection, and Bicuspidization

INDICATIONS FOR ROOT AMPUTATION OR HEMISECTION

□ Presence of severe bone loss in a nonsurgical treatable periodontally involved root or furcation

□ Untreatable roots with broken instruments, perforations, caries, resorption, and vertical root fracture or calcified canals

□ Preservation of strategically important root(s) and its accompanying crown

CONTRAINDICATIONS FOR ROOT AMPUTATION OR HEMISECTION

□ Insufficient bony support for the remaining root(s)

□ Root fusion or proximity so that root separation is not possible

□ Strong abutment teeth available (the involved tooth should be extracted and a prosthesis fabricated)

□ Inability to complete root canal treatment on the remaining root(s)

INDICATIONS FOR BICUSPIDIZATION

□ Furcation perforation

□ Furcation pathosis from periodontal disease

□ Buccolingual cervical caries or fracture into furcation

CONTRAINDICATIONS FOR BICUSPIDIZATION

- □ Deep furcation (thick floor of pulp chamber)
- □ Unrestorable half
- □ Periodontal disease (each half must be periodontally sound)
- □ Inability to complete root canal treatment on either half
- □ Root fusion
- □ Severe periodontal disease

Root amputation is the removal of one or more roots of a multirooted tooth. The involved root, or roots, is separated at the junction of the root and the crown (Figure 20-22). In general, this procedure is performed in maxillary molars but can be performed for mandibular molars.

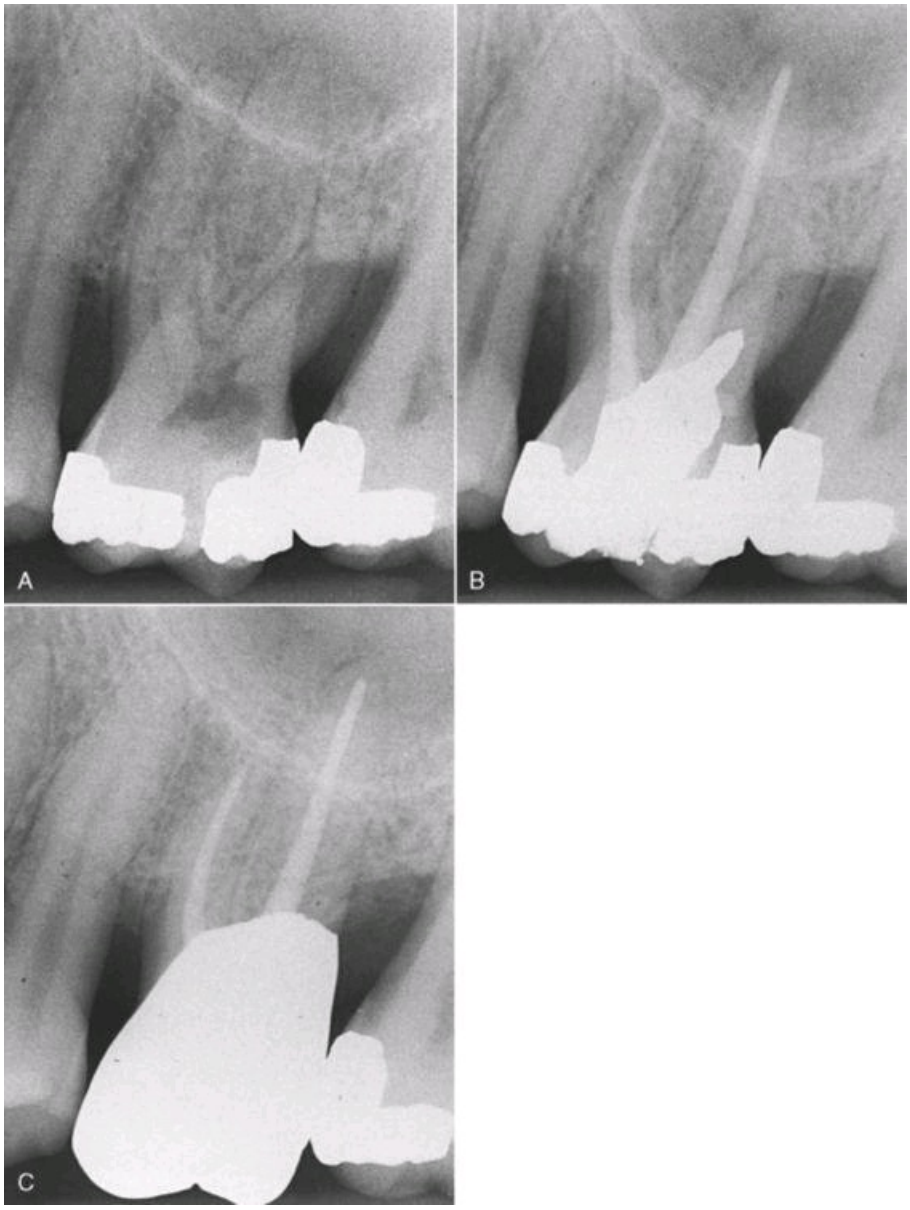

Figure 20-22 Root amputation. **A,** Internal resorption with severe bone loss around the distobuccal root was planned. **B,** Root canal treatment was followed by an amalgam core extending 4 mm into the distobuccal canal. **C,** The root was amputated and a crown subsequently placed.

Hemisection is the surgical division of a multirooted tooth. In mandibular molars the tooth is divided buccolingually through the bifurcation (Figure 20-23). In maxillary molars the cut is made mesiodistally, also through the furcation. The defective or periodontally involved root, or roots, and its coronal crown are then removed.[45]

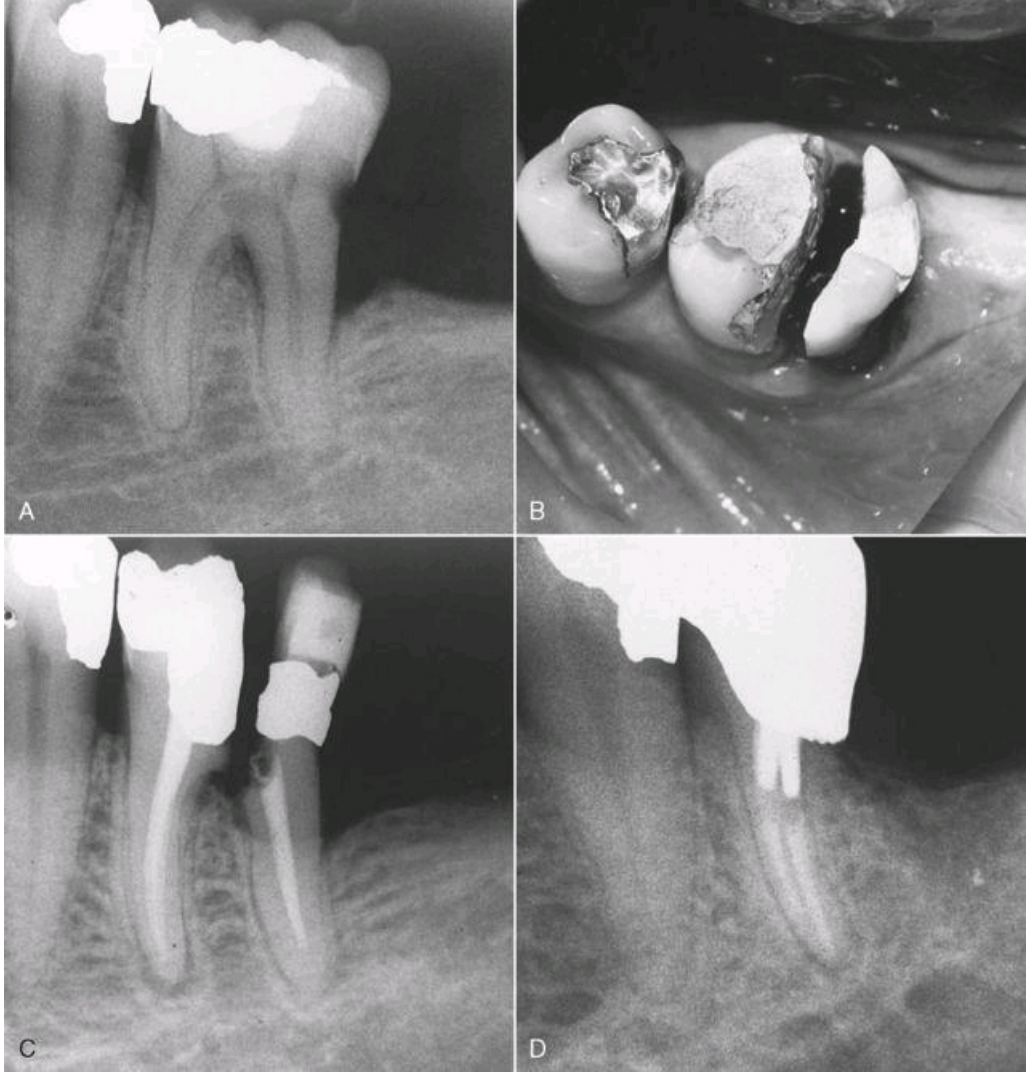

Figure 20-23 Hemisection. **A,** Furcation caries and bone loss have compromised the distal root. **B,** After root canal treatment, the crown was divided through the furcation **(C). D,** Twenty-month recall, after posts and core and a crown were placed. The extraction socket has healed.

Bicuspidization is typically a surgical division of a mandibular molar. The crown and root of both halves are retained. If severe bone loss or destruction of tooth structure is confined primarily to the furcation area, hemisection and furcal curettage may allow retention of both halves (Figure 20-24). Each half may be restored to approximate a bicuspid; thus the term *bicuspidization* is used for this procedure.

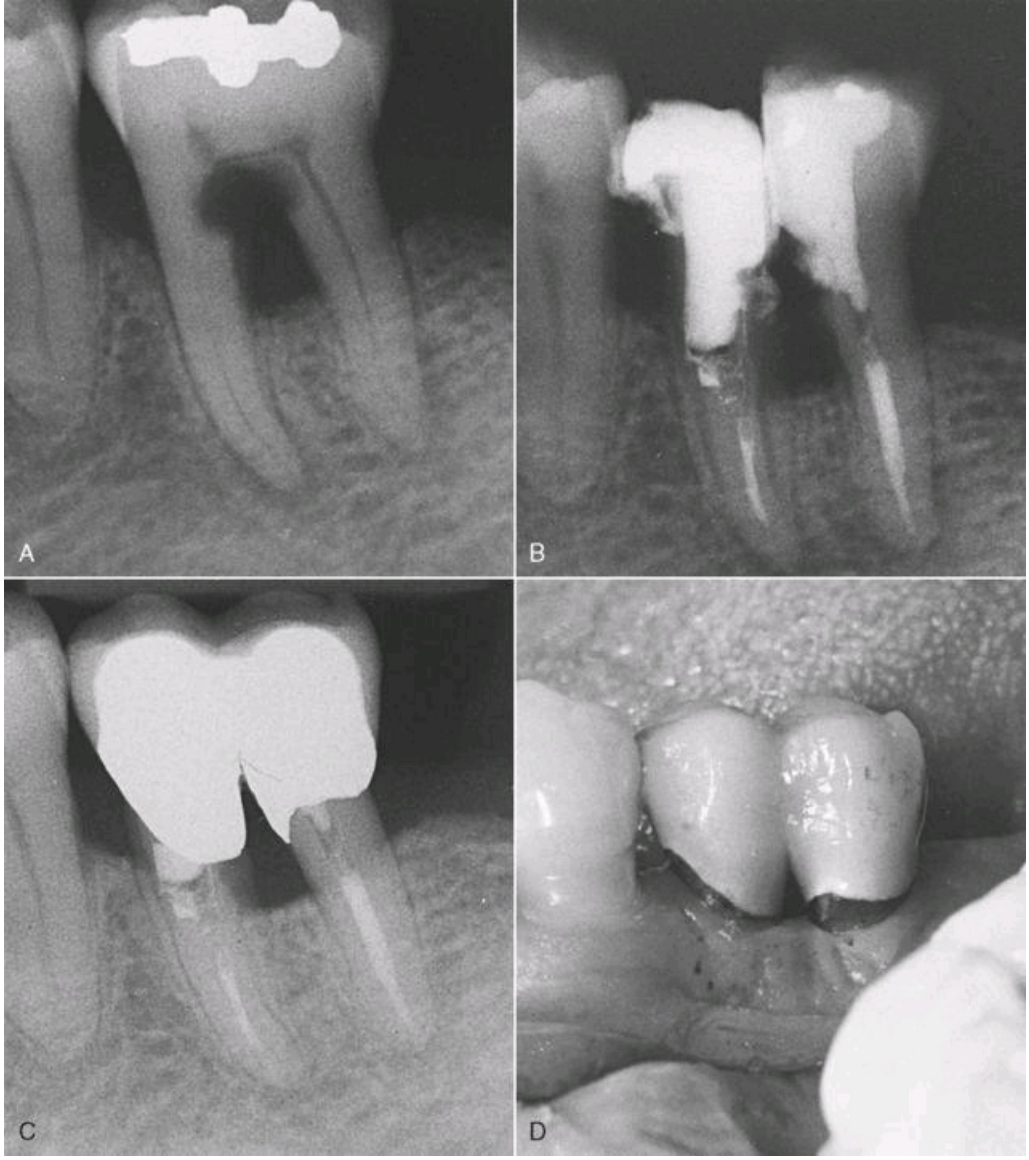

Figure 20-24 Bicuspidization. **A,** Caries in the furcation and furcal bone loss are evident, but there is adequate support for both roots. **B,** Root canal treatment and bur separation through furcation and crown. **C,** Restoration with a porcelain fused to metal crown splinting the two roots. **D,** Good gingival response at 30-month recall with no probing defects. Note that the furcation is open to facilitate special oral hygiene procedures.

Techniques

Root amputation is performed by making a horizontal cut to separate the root from the crown. The crown remains intact, and the root segment is removed. Therefore the crown is cantilevered over the extracted root segment (see Figure 20-22). A second approach is to use an angled vertical cut in which the crown above the root to be amputated is recontoured, decreasing the occlusal forces and making the procedure easier. As the crown is shaped, the bur is gradually angled into the root, resulting in good anatomic contour.

Hemisection is carried out by making a vertical cut through the crown into the furcation, which results in complete separation of the hemisected section (crown and root) from the tooth segment that is retained. These techniques may or may not require a flap. Often, if the root is periodontally involved, it is removed without a flap (see Figure 20-23). If bony recontouring is indicated, a flap is necessary before root resection is carried out. A sulcular flap design is often adequate without a vertical-releasing incision. However, when in doubt, a flap should be raised because doing so will always help.

Bicuspidization is performed after making a vertical cut through the crown into the furcation with a fissure bur. This procedure results in complete separation of the roots and creation of two separate crowns. After healing of tissues the teeth can be restored like two separate premolars (see Figure 20-24).

Prognosis

Each case is unique and has a different prognosis according to the situation. The results of root removal have been reported as good by some but only fair by others.[46] Success is defined by tooth retention with absence of pathosis. Success depends on the following factors:

- □ Case selection
- □ Cutting and preparing the tooth without creating additional damage
- □ Good restoration
- □ Good oral hygiene
- □ Development of caries (the most frequent cause of failure)
- □ Root fractures
- □ Excessive occlusal forces
- □ Untreatable endodontic problems
- □ Periodontal disease

If these procedures are performed correctly and the tooth is restored properly, the major factor affecting success is the patient's oral hygiene. The patient must be willing and able to perform extra procedures to prevent plaque accumulation, particularly in the area adjacent to what was once the furcation. Failure to do so will result in untreatable caries or periodontal disease. The dentist must work carefully with the patient to render this area plaque-free. A procedure that appears to be a success at 5 years may fail later. Thus the judgment of success or failure should be guarded and should extend over many years.

Recently, Mead and associates searched clinical articles pertaining to success and failure of PAS and assigned levels of evidence to these studies.[47] Their search located many clinical studies, most of which were case series. Rubinstein and Kim in a prospective study showed that the 1-year healing rate of endodontic surgery performed under the surgical operating microscope (SOM) in conjunction with microsurgical technique was 96.8%.[48] A long-term follow-up of these cases showed that 91.5% of them remained healed after 5 to 7 years.[49] Maddalone and Gagliani, who monitored the outcome of periradicular surgery in teeth treated with microsurgical technology and ultrasonic root-end preparation, reported a similar high success rate.[50]

Although it is impossible to tell whether the unusually high success rate resulted from the technique or the material, the clinical impression is that it is both the technique and the material, with the emphasis on technique. Recently, Sechrist et al[51] reviewed the clinical records of 294 patients who had MTA used during endodontic treatment from 1996 to 2001. From these, 75 patients whose root-end cavities had been filled with MTA were identified for recall. Twenty-five patients responded for clinical and radiographic evaluations, providing a total of 27 cases. Twenty-five (93%) of the recalled cases were functional and asymptomatic. Based on their results, it appears that the use of MTA should promote healing in a majority of surgical endodontic cases.

CONDITIONS THAT INDICATE REFERRAL

Except for incision for drainage, remaining endodontic surgical procedures should be referred to an endodontist. Although the procedures described in this chapter may appear straightforward, endodontic surgery requires advanced training, experience, and considerable surgical skill. The concern about standard of care and litigation in today's society, coupled with the availability of experienced specialists, means that general dentists must carefully examine their own expertise and accurately assess the difficulty of the case before attempting a surgical procedure. These procedures are often the last hope for retaining the tooth and require the highest level of skill and expertise to achieve success. Lack of training may result not only in the loss of the tooth but also in damage to adjacent structures, paresthesia from nerve injury, sinus perforations, soft tissue fenestrations, and postoperative complications such as hemorrhage and infection.

In many situations, access to the surgical site is limited and potentially hazardous. Long-standing large lesions may impinge on adjacent structures, requiring special techniques for resolution (Figure 20-25). The neurovascular bundle near the apexes of mandibular molars, premolars, and maxillary palatal roots predisposes the patient to postoperative surgical paresthesia or excessive hemorrhage. The treatment of endodontic problems in these areas requires careful preoperative assessment and considerable surgical skill. The presence of thick cortical bone and bony eminences throughout the mandible and in the palate, frenum, and muscle attachments; fenestrations of the cortical bone; and sinus cavities all require considerable surgical skill and experience in gaining access to most teeth.

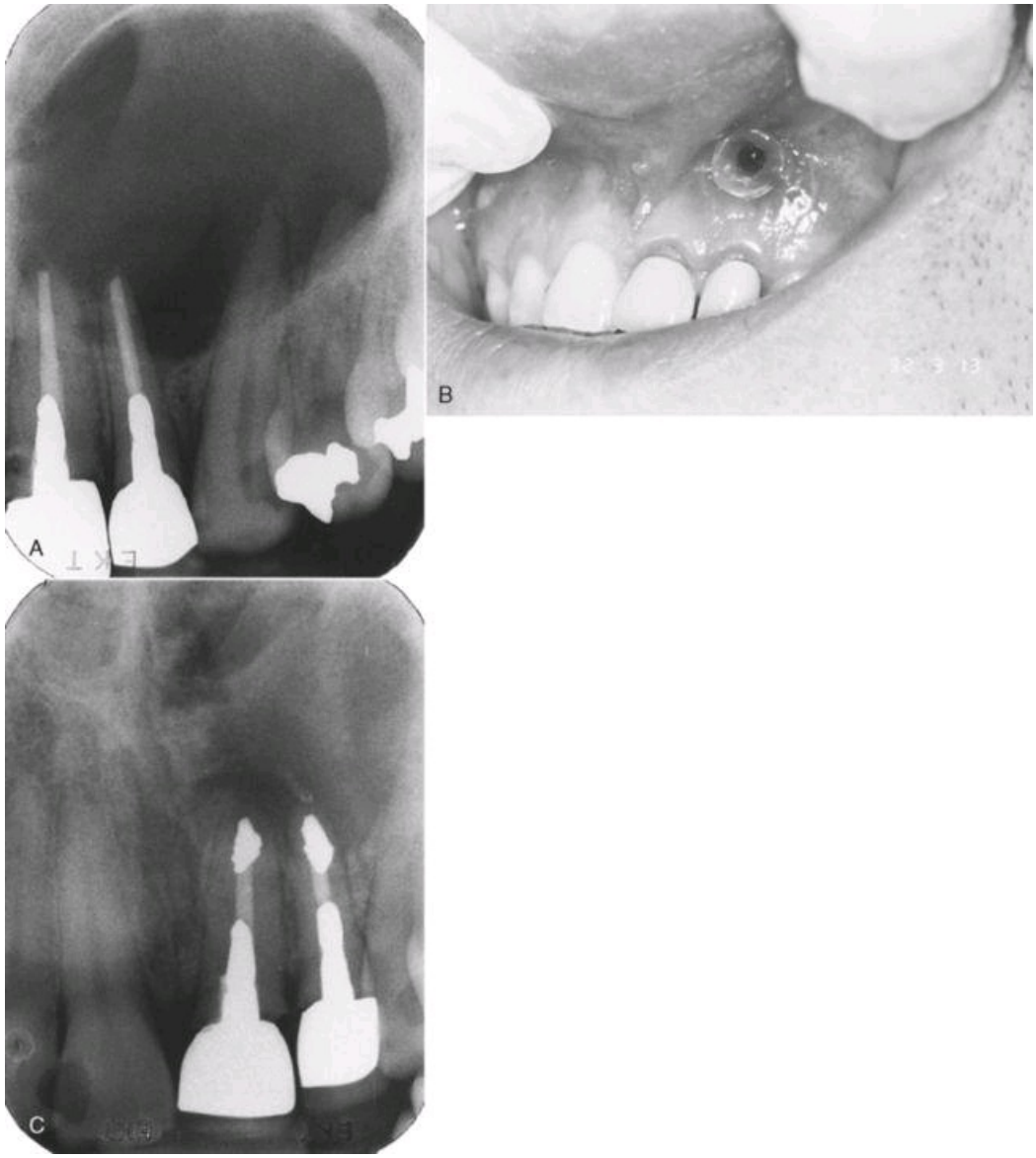

Figure 20-25 Decompression. Some cases require special procedures. **A,** Very large cyst fails to heal after root canal treatment. **B,** After surgical exposure and root-end surgery, a polyethylene tube is placed for several weeks to allow communication between the cyst cavity and the oral cavity. This allows collapse of the cyst wall. **C,** The lesion has resolved 1 year later, showing regeneration of bone.

(Courtesy Dr. S. Gish.)

Most important is the need for appropriate diagnosis, treatment planning, case assessment, prognostication, and follow-up evaluation. The general dentist should have knowledge in these areas but may prefer to refer to or request input from an

endodontist. The specialist is better able to accomplish these goals and assess the short- and long-term outcome.[52]

Chapter Review Questions available in Appendix B or on the DVD

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Evaluation of Endodontic Outcomes

Mahmoud Torabinejad, Asgeir Sigurdsson

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Describe signs of successful and unsuccessful root canal treatment.
2. Describe the most common modalities used to determine success or failure.
3. State the approximate range of expected outcomes of routine, uncomplicated root canal treatment based on pretreatment conditions.
4. State predictors of success and failure.
5. Identify causes, both endodontic and nonendodontic, that may lead to failure of treatment.
6. State the outcomes of retreatment, endodontic surgery, and intentional replantation.
7. State the outcomes of fixed partial denture and single tooth implant.
8. Approach treatment planning of root canal failure, recognizing advantages and disadvantages of different treatment modalities.

Providing the highest possible level of comfort, function, longevity, and esthetics are the main objectives of root canal treatment for patients who have been afflicted by oral diseases or traumatic injuries. These objectives are achieved by elimination or significant reduction of bacteria (subsequent inflammation) during cleaning and shaping of root canals, prevention of recontamination of root canals by 3-dimensional obturation, and placement of functional and esthetic permanent restorations during the lifetime of the afflicted teeth. Human studies have shown successful root canal treatment depends primarily on the effective elimination of microorganisms from the infected root canals.[1-3] Because of the complexity of root canal systems,[4,5] the inadequacy of current instrumentation and obturation methods, and leakage of permanent restorations, elimination of bacteria from the root canal systems cannot be achieved in some cases. Adherence to proper protocol significantly improves the prognosis of root canal–treated teeth. However, because of the complex interactions between host and parasite, unusual outcomes are occasionally seen in cases that have followed the proper or improper protocols for

successful outcomes.

The purposes of this chapter are to (1) define success and failure, (2) describe methods for evaluation of endodontic outcomes, (3) provide success rates, (4) recognize signs and symptoms of negative outcomes, (5) discuss factors influencing outcomes, and (6) compare the outcome of initial nonsurgical root canal treatment with that of retreatment, endodontic surgery, and alternative treatments such as single tooth implants.

DEFINITION OF SUCCESS AND FAILURE

The stakeholders in the dental delivery system (patients, third-party payers, and dentists) have different perspectives and expectations regarding the outcome of root canal treatment.[6] Patients are usually satisfied if their teeth are functional and esthetically pleasing. Insurance companies measure success by access to care, quality of care, and cost, as well as the survival rate of treatment. Dentists are usually most concerned with the delivery of optimal care (elimination of disease as measured clinically and radiographically) and with the fair compensation. The real art of dentistry is to coordinate and interface these perspectives and expectations amongst the stakeholders without sacrificing the quality of care to the patient.

Absence of clinical symptoms and apical lesions are the major indicators of successful root canal treatment. It is important to remember that apical periodontitis, which is often a principal indication of a failing endodontic treatment, is frequently asymptomatic and the radiograph is the only way to demonstrate the lesion.[7]

In a tooth with vital pulp, success means that the treated tooth remains asymptomatic and does not form an apical lesion during the lifetime of the afflicted tooth. A symptomatic tooth or a root canal–treated tooth with an apical lesion is a sign of a failed root canal in a tooth with vital pulp. In a tooth with a necrotic pulp, the treatment is considered successful if it remains asymptomatic, its apical lesion heals, and it does not develop a new apical lesion during the lifetime of the afflicted tooth. The presence of symptoms or a lesion in a tooth with necrotic pulp and an apical lesion is a sign of failed root canal treatment. Unfortunately, not all endodontic outcomes are straightforward. Many asymptomatic root canal–treated teeth have varying degrees of radiolucency. By strict radiographic criteria, they are unsuccessful, but by clinical criteria, they could be considered successful procedures. These cases can be classified as functional teeth with uncertain prognosis. Although these teeth are asymptomatic, a radiographic lesion indicates disease. The responsibility of the dentist is to use his or her judgment to treat these cases.

WHEN TO EVALUATE

There has been debate as to the length of follow-up period to demonstrate success.[8-13] Suggested follow-up periods in these studies range from 6 months to 5 years; 6 months is a reasonable interval for a recall evaluation for most patients. However, an important question is at what point is it unlikely that a treatment outcome will change? In other words, when can it be determined that treatment is successful or has failed and the outcome is unlikely to change so that no further recall is necessary? There is good evidence that a radiographic lesion that is unchanged or has increased in size after 1 year is unlikely to ever resolve; therefore the treatment is deemed unsuccessful. If at 6 months the lesion is still present but smaller in size, there is an indication that it might heal but additional recall is needed. It takes longer for larger periradicular lesions to heal compared to smaller lesions. Unfortunately, apparent success may revert to failure at a later time (often as a result of coronal leakage), so clinical and radiographic examination of teeth treated with root canals is indicated as a part of routine full-status evaluation of all dental patients.

It may be speculated that a persistent radiolucent lesion represents “healing by scar tissue” after root canal treatment. This is unlikely because scars occasionally form but only after endodontic surgery in maxillary anterior teeth.

METHODS FOR EVALUATION OF ENDODONTIC OUTCOMES

Clinical findings and radiographic examinations are the most common procedures used to determine outcomes of root canal therapy. Histologic examination of periapical tissues with surgical intervention is another method for evaluation of success or failure of root canal treatment. This method is not routinely used and is an impractical approach to determine clinical outcomes of root canal treatments.

Clinical Examination

Presence of persistent signs or symptoms is usually an indication of disease and of failure. However, absence of symptoms does not portray success. Periapical pathosis without significant symptoms is usually present in teeth before and after root canal treatment.[7] There is little correlation between the presence of pathosis and corresponding symptoms; yet when adverse signs or symptoms are evident, there is a strong likelihood that there is a pathosis.[14] Persistent signs (e.g., swelling or sinus tract) or symptoms (e.g., spontaneous pain, dull continuous ache, or mastication sensitivity) usually indicate failure.

Clinical criteria for success include the following[14]:

- □ Absence of pain and swelling
- □ Disappearance of sinus tract
- □ No evidence of soft tissue destruction, including probing defects

Radiographic Findings

According to the radiograph findings, the outcome of each treatment can be classified as success, failure, or questionable status. To be able to accurately compare radiographs made at different times, it is important that they are made in a reproducible fashion and with minimal distortion. The best way to ensure reproducibility is with paralleling radiographic devices (see Chapter 12).

Radiographic *success* is the absence of an apical radiolucent lesion. This means that a resorptive lesion present at the time of treatment has resolved or, if there was no lesion present at the time of treatment, none has developed. Thus radiographic success is evident by the elimination or lack of development of an area of rarefaction for a minimum of 1 year after treatment (Figure 21-1) . *Failure* is the persistence or development of pathosis radiographically. Specifically, this is a radiolucent lesion that has remained the same, has enlarged, or has developed since treatment (Figure 21-2). Nonfunctional, symptomatic teeth with or without radiographic lesions are considered failure (nonhealed).

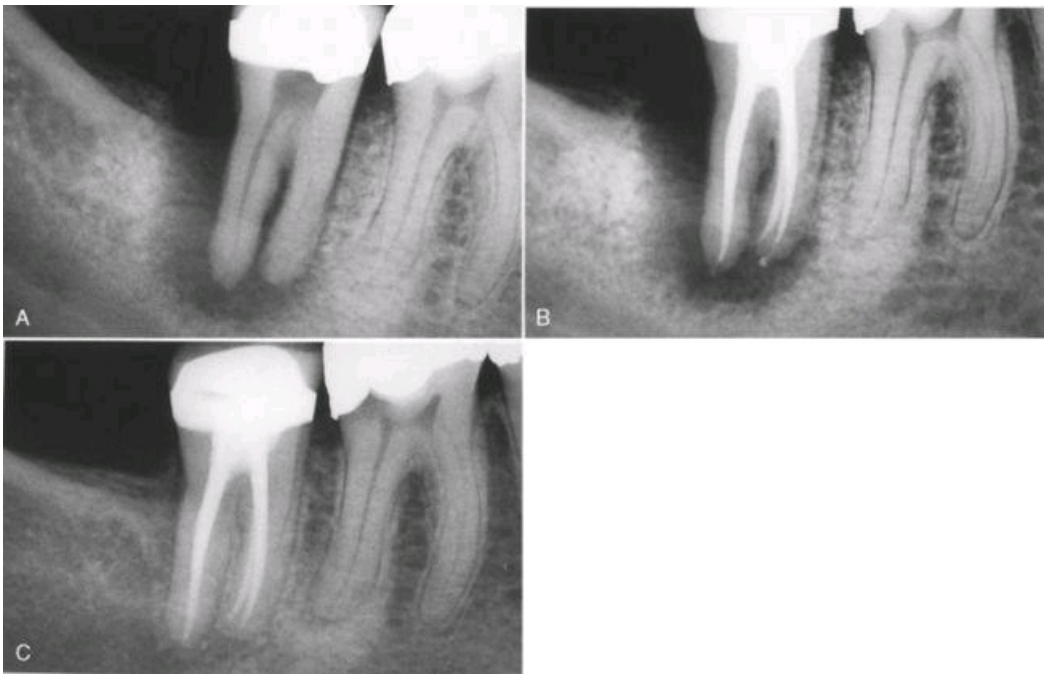

Figure 21-1 Success. **A,** This radiolucent lesion has the radiographic appearance of a periodontal lesion. The initial radiograph indicates bone loss from the crest of the ridge around the apices of the tooth. Periodontal probing demonstrates that the gingival sulcus is intact. There is no response to pulpal tests. **B,** Root canal treatment completed. **C,** Four-year recall shows resolution of the radiolucency.

*(**A** from Harrington GW: Dent Clin North Am 23:673, 1979.)*

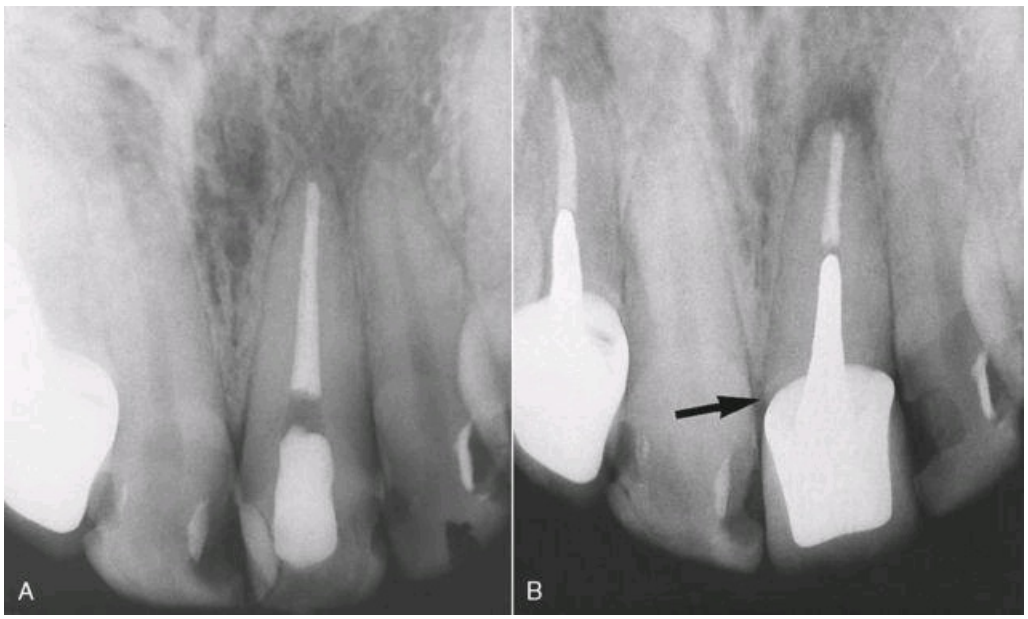

Figure 21-2 Failure. **A,** Apparently adequate root canal treatment. Tooth was restored later with a post and core and crown. **B,** The patient reports persistent discomfort after 2 years. Periradicular radiolucency indicates *failure,* probably a result of coronal leakage at a defective margin *(arrow).* Surgery was required (root-end resection and filling) because of restoration.

Questionable status indicates a state of uncertainty. This classification includes teeth with pathosis that are asymptomatic and functional. The radiolucent lesion in these teeth has neither become larger nor significantly decreased in size. A questionable status can be considered to be nonhealing or healing, depending on initial radiographic findings (Figure 21-3).

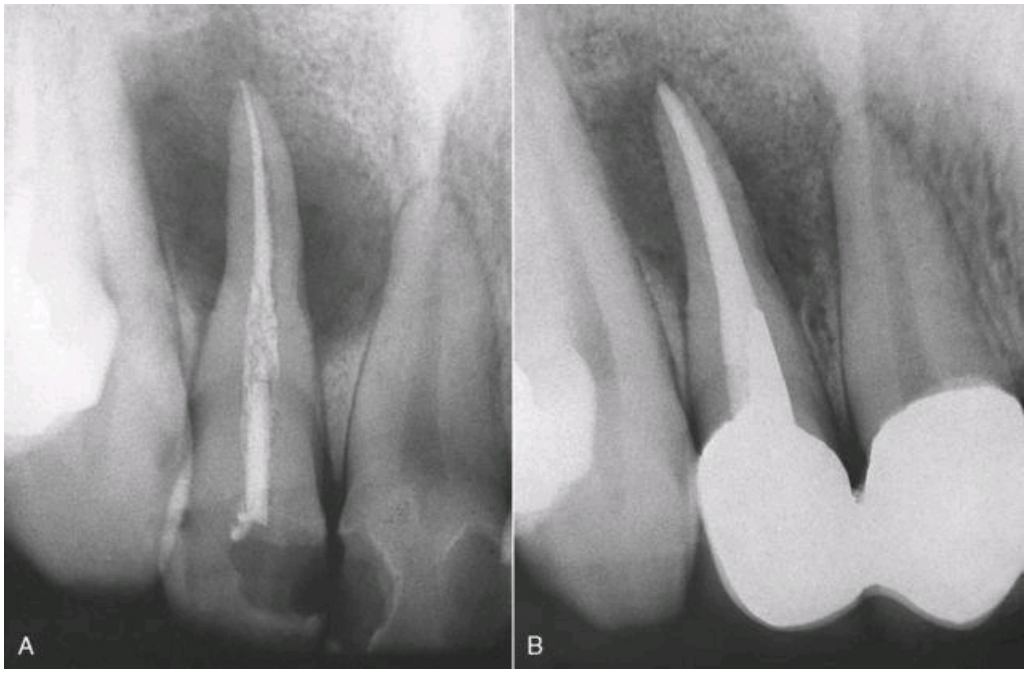

Figure 21-3 Questionable. **A,** Inadequate canal preparation, poor obturation, and coronal leakage all contributed to failure. The tooth is suitable for conventional retreatment and restoration. **B,** Twelve-month recall after retreatment. A sinus tract has disappeared, and the patient reports absence of symptoms. The radiolucent lesion is decreasing in size but has not resolved. Because outcome is still *questionable,* additional recall evaluation is necessary.

(Courtesy Dr. A. Stabholz.)

The shortcoming of this method of evaluation is that it determines "success" based strictly on radiographic findings. As early as 1966, Bender and coworkers[14] noted that radiographic interpretation is often subject to personal bias and that a change in angulations can often give a completely different appearance to the lesion, making it appear either smaller or larger. Also, different observers may not agree on what they see in a radiograph and the same observer may disagree with himself if asked to review the same radiograph at a different time period.[15]

Ørstavik and associates[16] suggest the use of the periapical index (PAI) for radiographic evaluation of the outcome of root canal treatment. The PAI relies on the comparison of the radiographs with a set of five radiographic images reported by Brynolf in 1967.[17] These images represent a radiographically healthy periapex (score 1) to a large periapical lesion (score 5). Each of the preoperative and recall radiographs is assigned a score according to its resemblance to one of the five reference images. The outcome of root canal treatment using PAI can be classified as "healing" if the lesion size is reduced, "healed" if the lesion has been completely eliminated, or "developing" if a new lesion has formed.

Histologic Examination

Routine histologic evaluation of periapical tissues after root canal treatment is impractical and impossible without surgery.[18] If a treated tooth were to be evaluated histologically, ideal treatment would be signified by reconstitution of periradicular structures and an absence of inflammation (Figure 21-4).

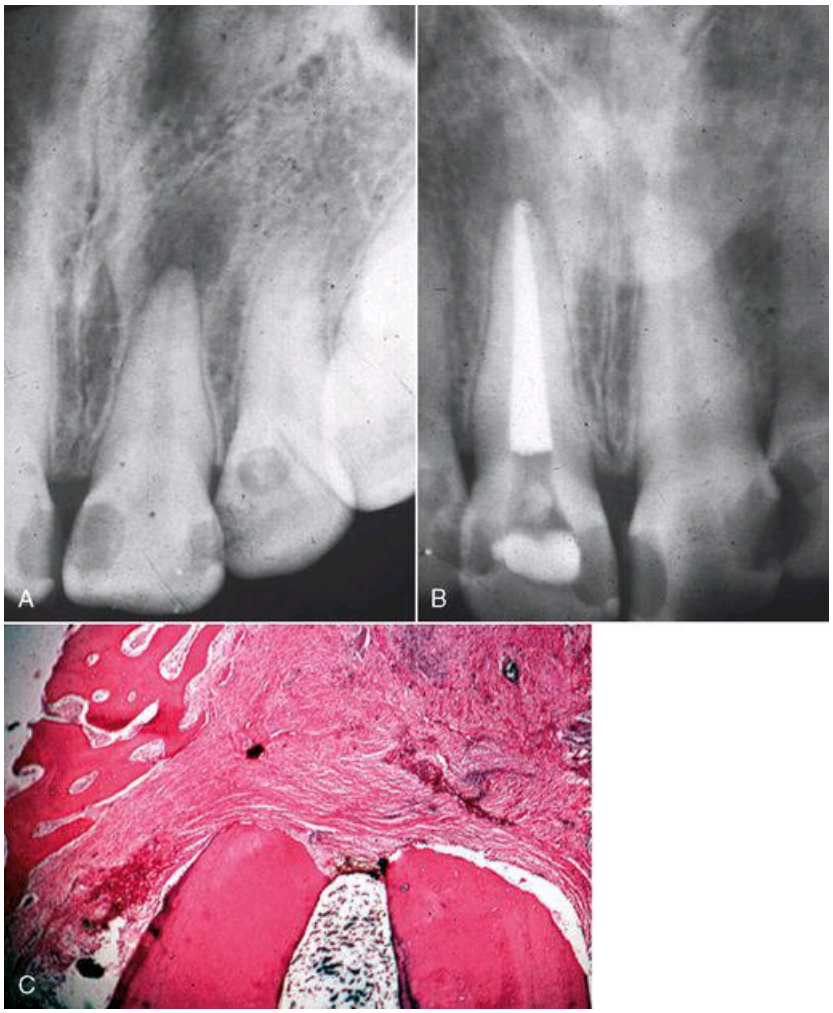

Figure 21-4 A, Periapical radiograph revealed an apical radiolucency on the central incisor. **B,** Periapical film taken 6 months later shows resolution of most of the radiolucency associated with the apex. Despite radiographic changes, the patient stayed symptomatic and a surgery had to be performed. **C,** Histologic examination of this tooth shows resolution of most of the inflammatory cells near the apex.

(Courtesy Dr. A. Khayat.)

There is uncertainty about the degree of correlation between histologic findings and negative radiographic appearance. Two histologic investigations of teeth treated with root canals in cadavers reached very different conclusions.[17,19] Brynolf[17] concluded that almost all root canal–treated teeth showed some periradicular inflammation despite the appearance of successful treatment on radiographs. In contrast, Green et al[19] observed that most root canal–treated teeth with normal periapex radiographically were indeed free of inflammation histologically. Thus, with current technology, clinical findings (signs, symptoms) and radiographic evaluation are the only practical means of assessing degree of healing after root canal treatment.

SUCCESS RATES

As is the case for other dental and medical procedures, unfortunately, not all root canal treatments are successful. Recognition, acceptance, and management of treatments that do not resolve and heal can be difficult and often involve a complex set of factors. Historically, the popular belief has been that the success and survival rates for root canal treatment are between 80% and 95%. However, general percentages should be taken with caution, and *each case should be individually assessed to determine the percentage probability of success.*

Torabinejad and associates[20] performed a systematic review of the literature pertaining to success and failure of nonsurgical root canal therapy and assigned levels of evidence to the studies. They reported that in the past 40 years, 306 articles have been published related to the outcome of nonsurgical root canal treatment. Fifty-one of these articles reported studies involving at least 100 teeth. From these, the success rate at 1, 2, and 5 years was recorded and analyzed with 95% confidence interval estimates. Their data show an overall radiographic success rate of 81.5% over a period of 5 years. Others who assessed the 4- to 6-year outcome of initial endodontic treatment have reported similar overall healing rates.[21] Based on survival rates, it appears that more than 90% of teeth that have had root canal treatment remain functional (in place, with or without disease) over time.[22,23] In a recent systematic review, Torabinejad et al[24] compared the outcomes of endodontically treated teeth with those of single dental implant–supported crown, fixed partial denture, and no treatment after extraction. Success data in this review consistently ranked implant therapy as being superior to endodontic treatment, which in turn was ranked as being superior to fixed prosthodontic treatment (Table 21-1). The same systematic review shows that root canal and implant treatments resulted in superior long-term (6 years plus) survival rates compared to extraction and replacement with a fixed partial denture. Iqbal and Kim reported similar findings when they compared the survival rates of restored endontically treated teeth with those of implant-supported restorations.[25]

Table 21-1 Percentages of Pooled and Weighted Survival and Success Rates of Dental Implants, Root Canal Treatment, and Three Unit Bridges over 2-4, 4-6, and 6+ Years

Procedure	Success (%)	Survival (%)
2 TO 4 YEARS		
Dental implant (pooled)	98 (95-99)	95 (93-97)
Dental implant (weighted)	99 (96-100)	96 (94-97)
Root canal treatment (pooled)	90 (88-92)	94
Root canal treatment (weighted)	89 (88-91)	—
Three unit bridge (pooled)	79 (69-87)	94
Three unit bridge (weighted)	78 (76-81)	—
4 TO 6 YEARS		
Dental implant (pooled)	97 (96-98)	97 (95-98)
Dental implant (weighted)	98 (97-99)	97 (95-98)
Root canal treatment (pooled)	93 (87-97)	94 (92-96)
Root canal treatment (weighted)	94 (92-96)	94 (91-96)
Three unit bridge (pooled)	82 (71-91)	93
Three unit bridge (weighted)	76 (74-79))	—
6+ YEARS		
Dental implant (pooled)	95 (93-96)	97 (95-99)
Dental implant (weighted)	95 (93-97)	97 (96-98)
Root canal treatment (pooled)	84 (82-87)	92 (84-97)
Root canal treatment (weighted)	84 (81-87)	97 (97-97)
Three unit bridge (pooled)	81 (74-86)	82
Three unit bridge (weighted)	80 (79-82)	—

PREDICTORS OF SUCCESS AND FAILURE

An early "classic" comprehensive study performed by Strindberg[26] related treatment outcomes to biologic and therapeutic factors. With time, other variables have been related to success and failure. Few of these variables have been proven to have a clear effect on the final outcome of the root canal treatment. Others are generalities, and their full effects are unknown because of the nature and complexity of the problems. Some of the factors that are considered predictors of success and failure include (1) apical pathosis, (2) bacterial status of the canal, (3) extent and quality of the obturation, and (4) quality of the coronal restoration. The role of these factors should be communicated to the patient and discussed before and after treatment.

Several investigations have shown that presence of lesions negatively impacts root canal treatment.[20] The presence of bacteria in the canal before obturation predicts a poorer prognosis.[27] In relation to extension of the obturation, healing is less predictable if the filling is too short (more than 2 mm from the radiographic apex) or too long (exiting the apex).[28-30] More voids or less density of the obturating materials are also related to lower success rates.[31] The quality of the coronal restoration plays an important role in the outcomes of root canal treatment.[32]

Other factors, such as tooth type, age and sex of the patient, technique of obturation, observation period, and type of intracanal medication, have been shown to have a slight effect (but usually none) on the outcomes of root canal treatment.[33] Medical (health) status or age of the patient has no significant bearing on success or failure.[34] No specific systemic disease or condition has been related to delayed or impaired healing or seems to precipitate failure. Obviously, a patient who is debilitated or whose condition is severely compromised medically may be a poor candidate for root canal treatment. Patients with insulin-dependent diabetes mellitus have a significantly lower healing rate after root canal therapy in teeth with apical lesions than patients who are not diabetic.[35] Further studies are needed to investigate possible relationship between systemic diseases and healing of periapical tissues after root canal treatment.

CAUSES OF NONHEALED (FAILED) ROOT CANAL TREATMENTS

Several factors affect the outcome of a root canal treatment.[36] Presence of bacteria is the main cause of most nonhealed (failed) root canal treatments. In general, the most common causes of nonhealed root canal treatments are (1) errors in diagnosis and treatment planning, (2) coronal leakage, (3) lack of knowledge of pulp anatomy, (4) inadequate débridement and/or disinfection of the root canal system, (5) inadequate restorative protection, (6) operative errors, (7) obturation deficiencies or errors, and (8) vertical root fracture.

These factors can be traced backed to preoperative, operative, and postoperative phases of root canal treatment.

Preoperative Causes

Failure of root canal treatment is often traced to misdiagnosis, errors in treatment planning, poor case selection (dentists attempting treatment beyond their skill levels), or treatment of a tooth with a poor prognosis. All of these are preoperative factors. Diagnosis should be based on all available information: history of signs and symptoms, current signs and symptoms, radiographic evaluation, and vitality tests (see Chapter 5). Without evaluating all factors and forming a firm diagnosis, there is a risk of inappropriate treatment or treating the wrong tooth.

Not using good radiographic projection, including different mesiodistal angulations to determine various root canal system aberrations, such as extra canals (e.g., the mesiolingual canal in maxillary molars and second canals in mandibular incisors), often results in failure, even with correct diagnosis (Figure 21-5).

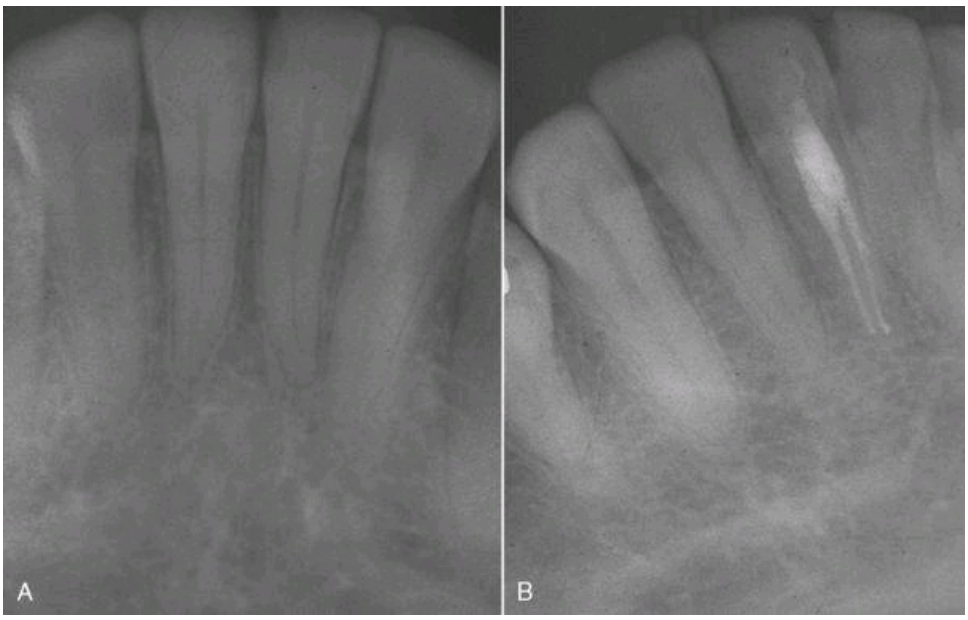

Figure 21-5 A, Sudden disappearance of the canal in the mandibular incisors indicates presence of a second canal or root. **B,** Postoperative radiograph shows presence of two separate canals in the right central incisor.

Coronal or root dentin fractures are also often misdiagnosed or escape early detection. Periodontal defects with associated bone loss often appear after the fracture has been on the crown and root long enough for the crack to become infected (see Chapter 7).[26-29] However, if there is an isolated, deep probing defect associated with the suspect tooth, vertical root fracture must be considered (Figure 21-6).

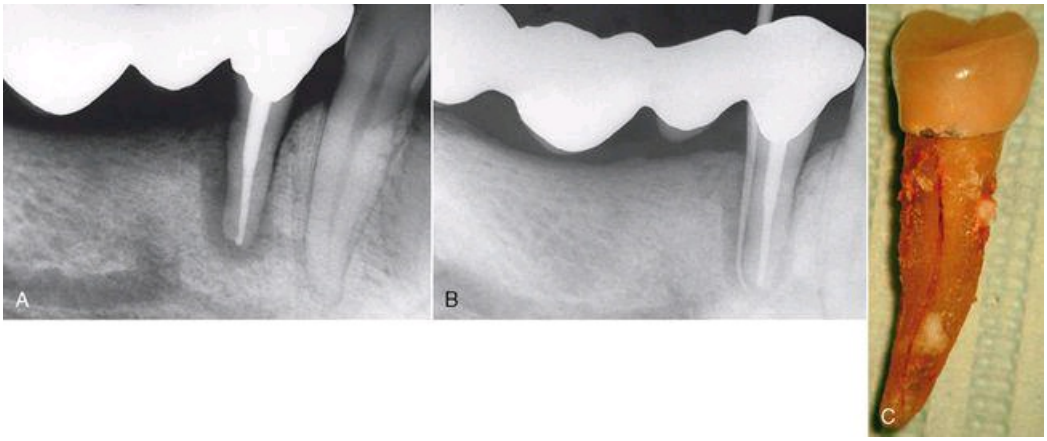

Figure 21-6 Indicators of vertical root fracture. **A,** "Tear-drop" lateral radiolucency is noted along the root. **B,** Narrow probing defect extends to the apex.**C,** Confirmation of a vertical fracture after tooth extraction.

Operative Causes

Many failures result from errors in operative procedures (see Chapter 18). For predictable success, several steps need to be followed. These include chemomechanical cleaning and shaping of the root canal space followed by a complete obturation that is confined to the root canal system.

Mechanical Objectives

An overlooked but important part of successful root canal treatment is a straight-line access preparation that will facilitate débridement and obturation. The access is even more important with the use of rotary nickel-titanium file systems. If the access is underextended, several mishaps may occur that ultimately will lead to failure. A canal may be missed, and the treatment is likely to fail although the located canals were appropriately treated. If the pulp horns are not opened in anterior teeth, debris and sealer may remain in the coronal pulp space. Such remnants often result in discoloration and therefore treatment failure. In addition, with a too-small access, instrument maneuverability is limited, resulting in insufficient cleaning and aberrant shaping or even instrument breakage.

Overextended access cavities, prepared at the expense of dentin, are also a problem. Excessive loss of dentin weakens the tooth, possibly allowing fractures, and increases the risk of perforation.[37,38]

A common error of instrumentation is failure to maintain canal curvature because files cut to the outside of the curve ("transportation" of the canal or apex). This alters canal morphology and leaves potentially infected debris in the canal system. Marked deviation or overzealous flaring or overpreparation in the "danger" zone or in the apical third may result in perforation (see Figures 15-13 and 18-8). Most of these perforations are possible to repair nonsurgically with mineral trioxide aggregate (MTA) but some require endodontic surgery (see Figure 21-4).[39] Leakage or mechanical irritation may result, and a lesion may develop.[40]

The outcome of a separated instrument (broken endodontic file) in a root canal system depends on the stage of canal preparation and the pretreatment pulp status (vital versus necrotic) (see Figures 18-13 and 18-14).[41,42] The outcome may be unaffected if the instrument can be removed or bypassed.

Confining operative procedures and materials to the canal space enhances repair.[28,43] Overinstrumentation causes some tissue damage, periradicular hemorrhage, and transitory inflammation. Continuous overinstrumentation provokes a persistent inflammatory response capable of resorbing dental and osseous tissues.[44] Overinstrumentation may also transfer microorganisms from the canal into the periapex, possibly compromising the outcome.[45]

Likewise, overextended obturation may lead to treatment failure. In many cases, the material does not cause the apical lesion because gutta-percha is relatively inert. Rather, the combination of an inadequate seal probably preceded by overinstrumentation has caused the failure. The gutta-percha cone slips through the apex because there was inadequate taper or shelf, resulting in inadequate matrix to confine, condense, and seal with the gutta-percha. In addition, sealers are irritating or toxic to the tissues.[18,46,47] Errors in obturation result from poor canal shaping or selecting an inappropriate obturating technique. A poorly condensed obturation (either underfilled or containing voids) is related to apical and/or coronal percolation.[18,48,49] Either underobturation or overfilling is likely to result in failure, particularly in the presence of pulp necrosis and an apical lesion.[50]

Biologic Objectives

Ideally, after preparation the root canal would be free of bacteria.[51] If the pulp is vital, preparation prevents contamination, and with a necrotic pulp, disinfection is achieved. However, as shown experimentally, complete débridement of the canal is virtually impossible.[52,53] Therefore bacterial counts are minimized by careful instrumentation, with copious sodium hypochlorite irrigation.[54] The intracanal medicament, calcium hydroxide, will reduce the number of bacteria,[55] enhance the speed of healing, and reduce inflammation.[56,57] However, there is uncertainty whether use of this medicament ultimately results in a better prognosis.[58]

Postoperative Causes

Lack of a coronal seal is probably the most common and best controlled problem. A coronal restoration protects and seals the tooth, preventing percolation of saliva and bacteria apically (Figure 21-7) that results in failed treatment.[32,50-58] There is a definite correlation between poorly restored crowns of endodontically treated teeth; leakage of dye, bacteria, or endotoxin through the canals; and a poorer prognosis.[32,59-62] The coronal access to a root canal–treated tooth must be sealed completely for the lifetime of this organ.

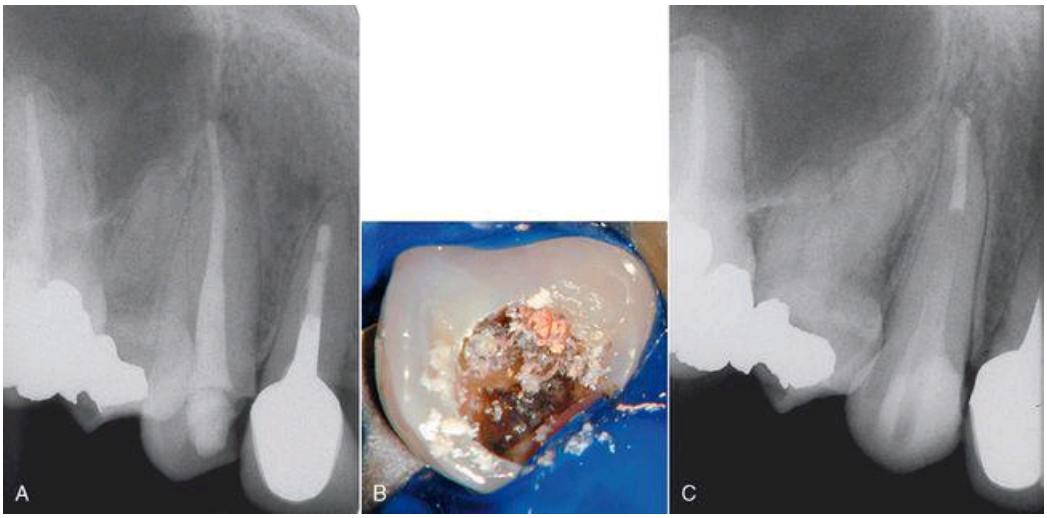

Figure 21-7 A, Lack of coronal seal has resulted in clinical symptoms and presence of a periapical lesion in the maxillary right cuspid. **B,** Clinical photograph shows lack of permanent restoration and decay in this tooth. **C,** After retreatment of previous root canal treatment and placement of a C-fiber post, the coronal access to root canal–treated tooth was sealed permanently.

(Courtesy Dr. D. Roland.)

Restoration should occur soon after obturation, using rubber dam isolation in a saliva-free environment. There should be no space between the coronal filling and the obturation in the cervical area because there is a risk for bacterial contamination through exposed cervical root dentin. Restorative errors also may compromise success. For example, excessive dentin removal for posts weakens the root and increases susceptibility to fracture (Figure 21-8).[37]

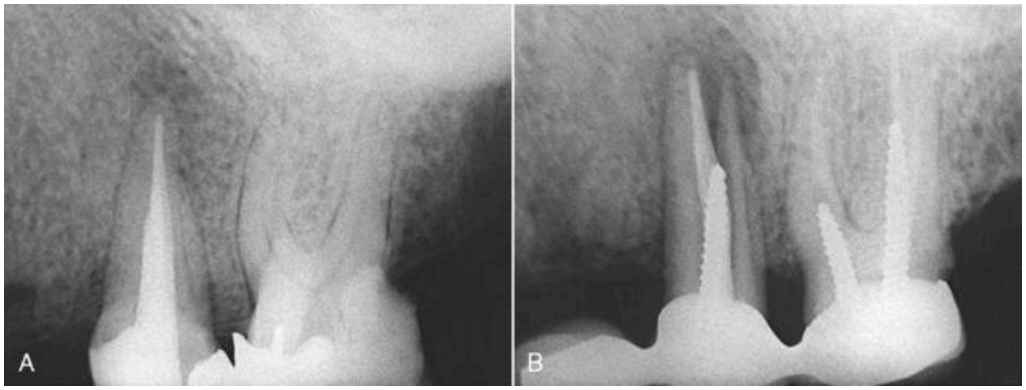

Figure 21-8 A, Overenlargement of a canal has weakened the root. **B,** Placement of an oversized post combined with earlier condensation forces resulted in a vertical fracture and apical-lateral pathosis. The tooth had to be extracted.

OUTCOMES OF TREATMENTS AFTER FAILURE OF NONSURGICAL ENDODONTICS

For decades, a primary goal of dentistry has been the preservation of natural dentition. In previous years, all efforts would have been made to save teeth with pulpal and periodontal diseases or to extract hopeless teeth and place fixed or removable prostheses. The relatively high success rate of dental implants has created a good treatment alternative and a dilemma for the dental profession, resulting in a paradigm shift in treatment planning. Clinicians are regularly confronted with difficult choices after failure of root canal treatment. Treatment options now include (1) nonsurgical retreatment; (2) endodontic surgery; (3) intentional replant, auto-transplantation; (4) extraction without replacement; (5) extraction and replacement with a fixed partial denture; or (6) single tooth implant.

Nonsurgical Retreatment

Recently, investigators from Loma Linda University completed a thorough search of published literature related to clinical studies on the success and failure of nonsurgical retreatment and assigned levels of evidence (LOE) to publications since 1970.[63] Their search resulted in the identification of 31 clinical studies and 6 review articles related to this subject. The success rate of nonsurgical retreatment ranged between 40% and 100%.[63] In the new prospective Toronto Study the "healed" rate of endodontic retreatment cases is reported to be 81%.[64] Based on these results, it appears that if a failure is retreated by conventional means (Figure 21-9), the success rate is very high in teeth without periapical lesions and when *the cause of failure is identified and corrected*[65] (see Chapter 19).

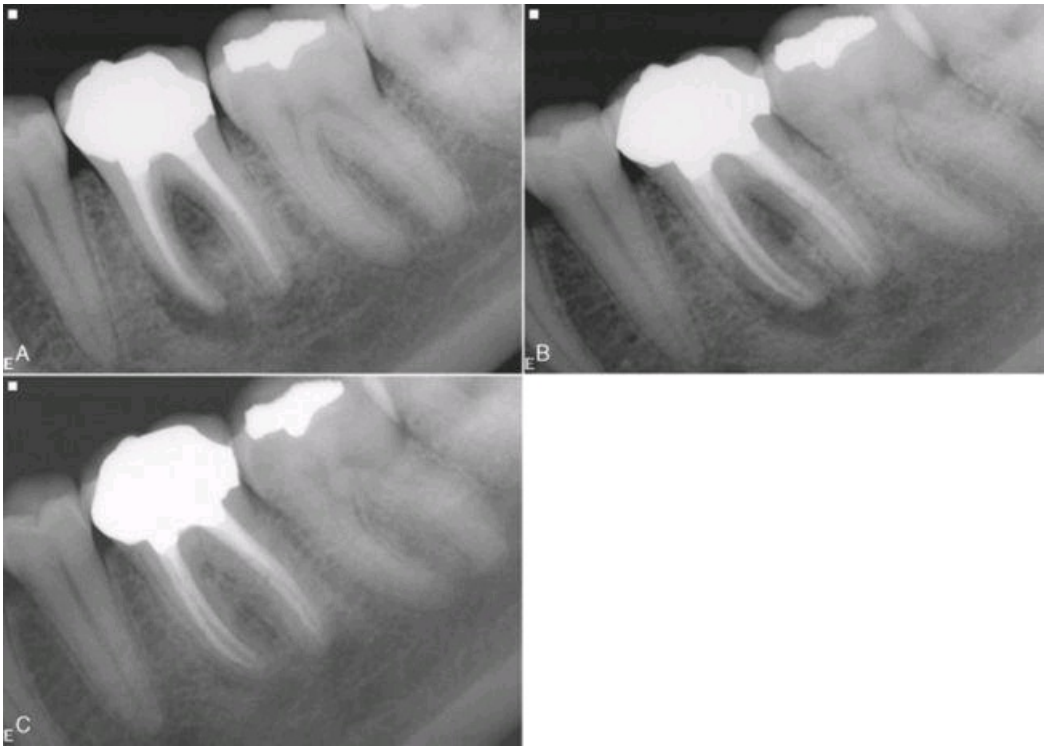

Figure 21-9 A, A periapical radiograph shows extensive radiolucency around the mesial root of first mandibular molar with previous root canal treatment. **B,** The root canal treatment was retreated nonsurgically and restored permanently. **C,** A radiograph 1 year later shows complete resolution of the periradicular lesion.

(Courtesy Dr. S. Paik.)

In another study, investigators from Loma Linda University searched the literature for clinical articles pertaining to success and failure of periapical surgery and assigned LOE to these studies.[66] Their electronic and manual searches showed that the majority of frequently quoted "success and failure" studies are case series (LOE 4). Recent long-term follow-up studies of endodontic surgery show high success rates (Figure 21-10)[67-69] (see Chapter 20).

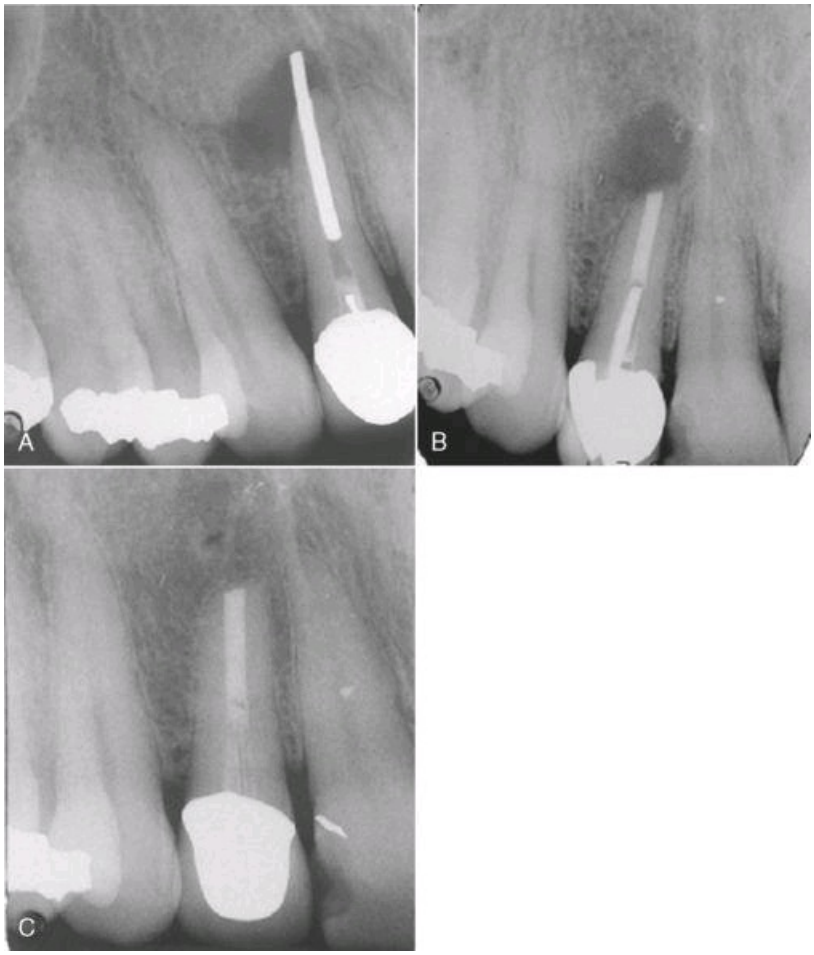

Figure 21-10 A, Periapical radiograph shows extensive radiolucency around the apex of the maxillary right incisor with an overextended silver point and an inadequate coronal seal. **B,** The root canal treatment was retreated nonsurgically and surgically using MTA as root canal filling material. **C,** Radiograph 3 years later shows complete resolution of the periradicular lesion.

(Courtesy Dr. C. Sechrist.)

Intentional Replantation and Autotransplantation

Intentional replantation is the reinsertion of a tooth into its alveolus after the tooth has been extracted for the purpose of performing root-end surgery while out of the socket (in hand).[70] Intentional replantation is indicated when there is no other treatment alternative to maintain a strategic tooth (see Figure 18-10). Intentional replants are often successful long-term[71-73] but require careful case selection (see Chapter 20).

An autotransplant is the transfer of a tooth from one alveolar socket to another in the same patient.[74,75] The clinical procedures involved include socket preparation, extraction, transplantation, and stabilization (Figure 21-11). Properly indicated and performed autotransplanted teeth have a good prognosis.[76-79]

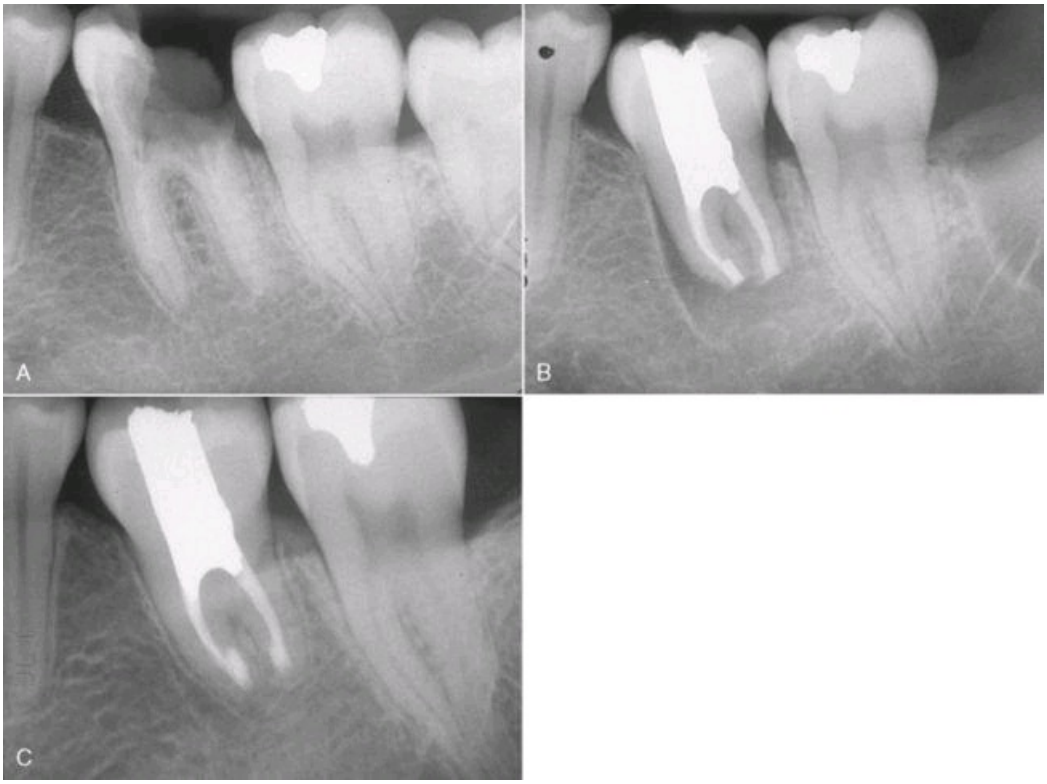

Figure 21-11 A, Periapical radiograph shows a nonrestorable mandibular first left molar. **B,** After root canal treatment and endodontic surgery using MTA as root-end filling on the third molar on the same side, the first molar was extracted and the third molar was placed in its socket. **C,** Radiograph 5 years later shows complete healing of periradicular tissues.

Extraction without Replacement

Little information is available regarding beneficial or harmful effects, as well as psychosocial or economic outcomes, of extracted teeth without replacement. Most of the available information is related to a shortened dental arch.

Fixed Partial Denture

Traditionally, after extraction of hopeless teeth, adjacent teeth were prepared for placement of fixed or removable prostheses. These teeth were prone to future complications, such as caries or pulpal or periodontal problems, as well as technical complications such as porcelain fracture with fixed prosthodontics over a 5-year period (Figure 21-12).[80] The success and survival rates for fixed partial denture have been reported between 48% and 95%. A meta-analysis of the literature reported an 87% 10-year survival rate and a 69% 15-year survival rate for fixed partial dentures.[81] Other investigators have reported similar results[24,82,83] (see Table 21-1). Treatment planning in prosthodontics has changed significantly because of the recent advances in the success rate of single tooth implants.[84]

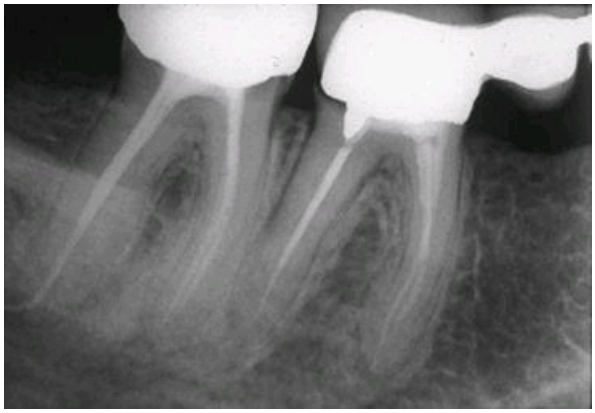

Figure 21-12 Restored mandibular right molars have developed pulpal and periapical problems, as well as caries under the margins of crowns. Coronal leakage might have contributed to the presence of periapical lesions.

Single Tooth Implant

Advances in implant dentistry have provided thousands of edentulous patients with a more functional and attractive alternative to removable prosthetics. Introduction of cylindrical-style endosseous implants to the dental profession has obviated the need for many partial- or full-coverage restorations on the adjacent teeth.[85,86] As in prosthodontics, the new innovations in implant dentistry have also affected periodontal treatment. This paradigm shift in periodontics is evident in recent surveys conducted by the American Academy of Periodontics that show 63% of periodontists are placing their primary emphasis on periodontics and 27% are placing their primary emphasis on implants.[87] These advances also affect treatment planning for teeth with pulp and periapical diseases.

The confusion over the definition of success is an issue in implant dentistry. Examination of the highly quoted studies on the outcomes of implants shows that investigators have used qualitative and quantitative criteria to determine success and failure of implants. The success and survival rates for single tooth implants have been reported between 76% and 100% (see Table 21-1). Very low (9%), as well as very high (over 97%), success rates have been reported in the literature.[88-99]

The criteria vary from very stringent to very lenient. Because of fundamental differences in indications and contraindications, procedures and techniques involved, factors affecting success and failure, criteria used to determine success and failure of implants, and treatment options after unsuccessful implants, implants cannot be directly compared to procedures that save natural dentition. Each procedure has its own indication (Figure 21-13).

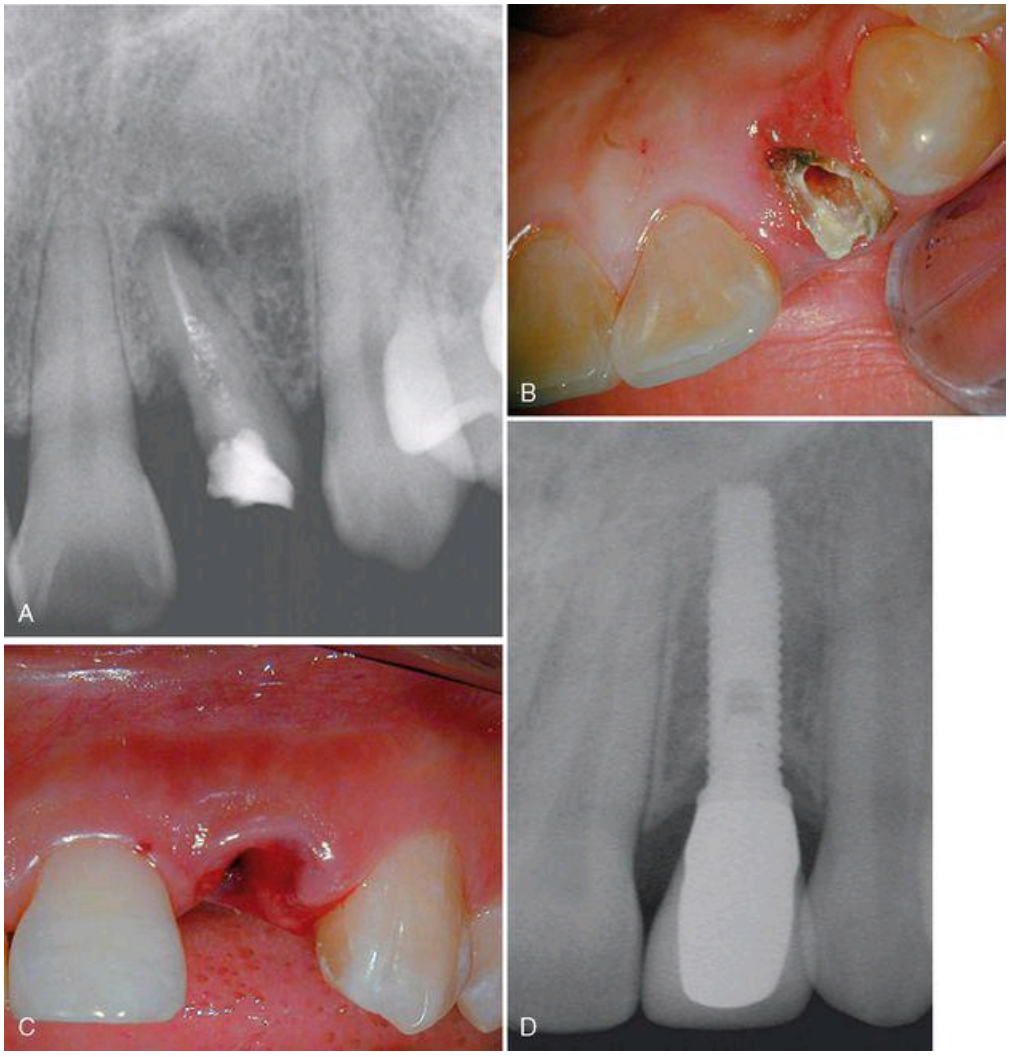

Figure 21-13 A nonrestorable tooth (**A** and **B**) was extracted **(C)** and an endosseous implant was placed **(D)** adjacent to two intact teeth.

Several factors affect the decision as to whether a tooth will receive root canal treatment or be extracted and an implant placed. These factors relate to the patient, tooth, periodontium, and type of treatment required. Considering these factors during treatment planning will provide the highest possible level of comfort, function, longevity, and esthetics for those patients who have been afflicted by oral diseases or traumatic injuries.[100]

Chapter Review Questions available in Appendix B or on the DVD

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Bleaching Discolored Teeth: Internal and External

Ilan Rotstein, Richard E. Walton

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Identify the cause and nature of tooth discoloration.
2. Describe means of preventing coronal discolorations.
3. Differentiate between dentin and enamel discolorations.
4. Evaluate both the short- and long-term prognosis of bleaching treatments.
5. Select the bleaching agent and technique according to the cause of discoloration.
6. Describe each step of the internal "walking bleach" technique.
7. Describe the indications for the microabrasion technique and the procedure.

8. Describe how bleaching agents may alter dentin.

9. Select the appropriate method to restore the access cavity after bleaching.

10. Recognize the potential adverse effects of internal bleaching and discuss means of prevention.

Discoloration of anterior teeth is a cosmetic problem that is often significant enough to induce patients to seek corrective measures. Although restorative methods, such as crowns and veneers, are available, discoloration can often be corrected totally or partially by bleaching. Bleaching procedures are more conservative than restorative methods, relatively simple to perform, and less expensive. Procedures may be internal (within the pulp chamber) or external (on the enamel surface) and involve various approaches.[1]

To better understand bleaching techniques, it is important to know the causes of discoloration, location of the discoloring agent, and the treatment modalities available. Also important is the ability to predict the outcome of treatment (i.e., how successfully can various discolorations be treated and how long will the esthetic result last). In other words, before attempting to correct discoloration, a diagnosis must be made (determine the cause and location of the discoloration), treatment planning must be done (internal or external bleaching and technique), and a prognosis assessed (anticipated short- and long-term success). Patients must be informed of these factors before undergoing the procedure; any discoloration treatment must be tempered by the explanation that substantial improvement may or may not occur. However, bleaching is worth a try because with proper and careful technique, no irreversible damage to the crown or root occurs.

This chapter reviews tooth discoloration and its correction. Discussed are the causes and management of discoloration as related to (1) location of discoloration, (2) approach used for correction, and (3) anticipated short- and long-term success of bleaching. The following aspects of discoloration and bleaching procedures are discussed:

1. Causes and location of discoloration

2. Commonly used bleaching agents

3. Internal bleaching techniques (usually in conjunction with or after root canal treatment)

4. Microabrasion, which is a technique for removing surface discolorations

5. Predictability and permanence of each procedure

6. Possible complications and safety of the various procedures

CAUSES OF DISCOLORATION

Tooth discolorations occur during or after enamel and dentin formation. Some discolorations appear after tooth eruption, and others are the result of dental procedures. Natural (acquired) discolorations may be on the surface or incorporated into tooth structure. Sometimes they result from flaws in enamel or a traumatic injury. Iatrogenic (inflicted) discolorations, which result from certain dental procedures, are usually incorporated into tooth structure and are largely preventable.

"Natural" or Acquired Discolorations

Pulp Necrosis

Bacterial, mechanical, or chemical irritation of the pulp may result in necrosis. Tissue disintegration byproducts are then released, and these colored compounds may permeate tubules to stain surrounding dentin. The degree of discoloration is directly related to how long the pulp has been necrotic. The longer the discoloration compounds are present in the pulp chamber, the greater the discoloration. This type of discoloration can be bleached internally, usually with both short- and long-term success (Figure 22-1).

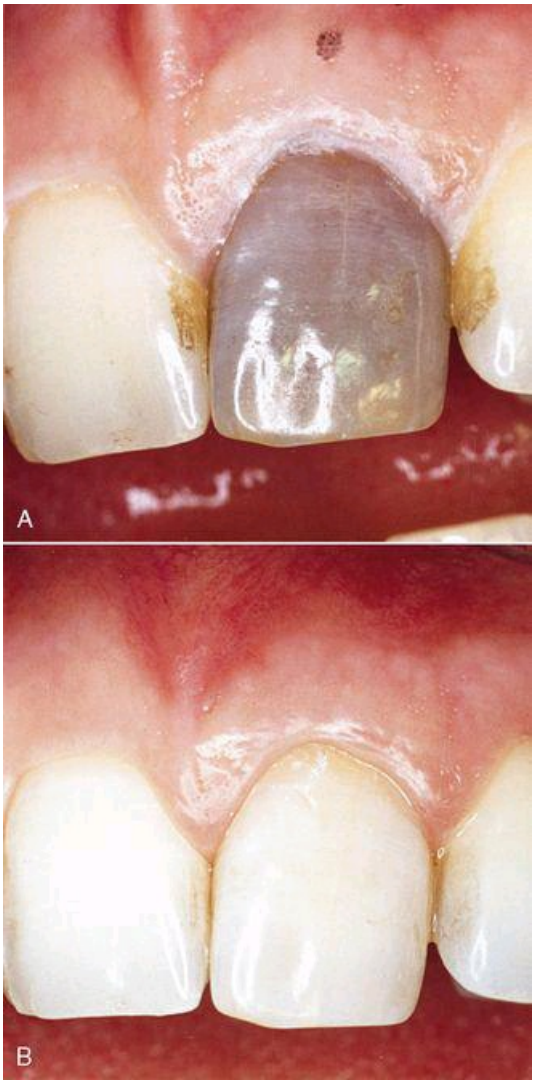

Figure 22-1 A, Discoloration as a result of a traumatic injury followed by pulp necrosis. **B**, After root canal treatment, a paste of sodium perborate and water mixed to a consistency of wet sand was sealed in the pulp chamber. After 21 days of "walking bleach," the tooth regained its original shade.

(Courtesy Dr. A. Claisse.)

Intrapulpal Hemorrhage

Generally, intrapulpal hemorrhage is associated with an impact injury to a tooth that results in disrupted coronal blood vessels, hemorrhage, and lysis of erythrocytes. It has been theorized that certain blood disintegration by-products, presumably iron sulfides, permeate tubules to stain surrounding dentin. Discoloration tends to increase with time.

If the pulp becomes necrotic, the discoloration usually remains. If the pulp survives, the discoloration may resolve and the tooth regains its original shade. Sometimes, mainly in young individuals, the tooth remains discolored even if the pulp responds to vitality tests.

Internal bleaching of discoloration after intrapulpal hemorrhage is usually successful both short and long term.[2,3]

Calcific Metamorphosis

Calcific metamorphosis is extensive formation of tertiary (irregular secondary) dentin in the pulp chamber or on canal walls. This phenomenon usually follows an impact injury that did not result in pulp necrosis. There is temporary disruption of blood supply with partial destruction of odontoblasts. These are usually replaced by cells that rapidly form irregular dentin on the walls of the pulp chamber and root canal space. As a result, the crowns take on a "flat" appearance as they gradually decrease in translucency and acquire a yellowish or yellow-brown discoloration (Figure 22-2). The pulp usually remains vital and does not require root canal treatment.

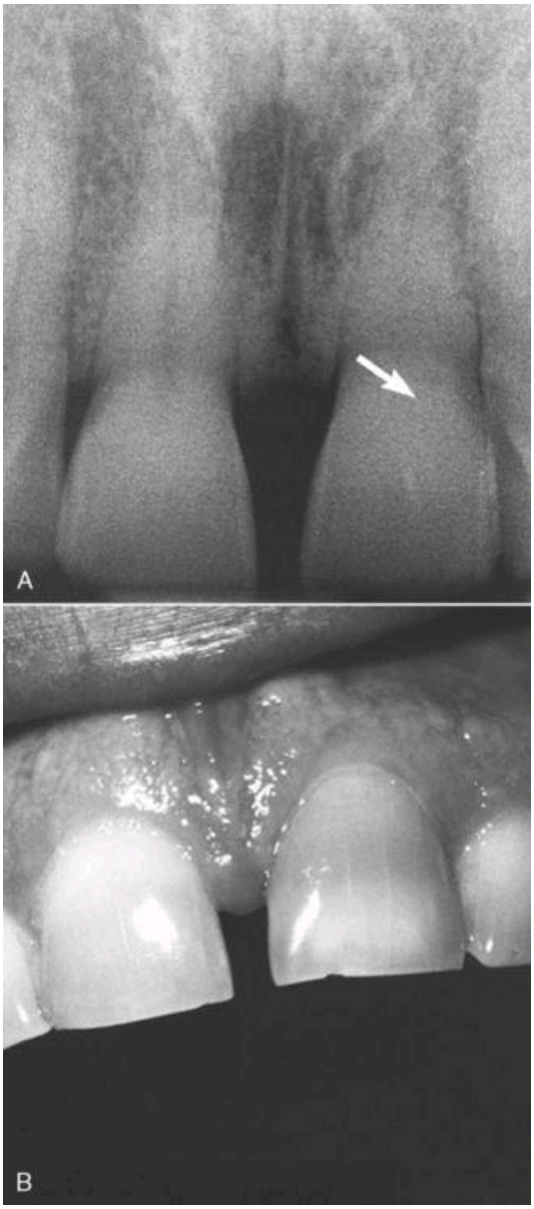

Figure 22-2 Calcific metamorphosis. Impact trauma resulted in reversible pulp damage with **(A)** extensive tertiary dentin formation *(arrow)* and **(B)** lost translucency and darkening of the crown. These teeth present difficulties with root canal treatment and internal bleaching.

If the patient desires color correction, external bleaching should be attempted first. If this is unsuccessful, root canal treatment is performed (sometimes with difficulty) and internal bleaching is done. This may be carried out whether the pulp is vital or necrotic. The esthetic prognosis of such bleaching is fair (unpredictable).

Age

In older patients, color changes in the crown occur physiologically as a result of extensive dentin apposition, as well as thinning of and optical changes in the enamel. Food and beverages also have a cumulative discoloring effect because of the inevitable cracking and other changes on the enamel surface and in the underlying dentin. In addition, previously applied restorations that degrade over time cause further discoloration. There is an increasing demand for bleaching among elderly patients. Bleaching is usually external because the discoloration is primarily on the enamel surface.

Developmental Defects

Discolorations may also result from development defects or from substances incorporated into enamel or dentin during tooth formation.

Endemic Fluorosis

Ingestion of excessive amounts of fluoride during tooth formation produces defects in mineralized structures, particularly enamel matrix, with resultant hypoplasia. The severity and degree of subsequent staining generally depend on the degree of hypoplasia, which depends in turn on the amount of fluoride ingested during odontogenesis.[4] The teeth are not discolored on eruption but may appear chalky. Their surface, however, is porous and gradually absorbs stains from chemicals in the oral cavity.

Because the discoloration is in the porous enamel, such teeth are bleached (or corrected) externally. Esthetic success depends mainly on the degree and duration of the discoloration. Some regression and reoccurrence of discoloration tend to occur but can be corrected with future rebleaching.

Systemic Drugs

Administration or ingestion of certain drugs or chemicals (many of which have not yet been identified) during tooth formation may cause discoloration, which is occasionally severe.[5]

The most common, as well as the most dramatic, discoloration of this type occurs after tetracycline ingestion, usually in children. Discoloration is bilateral, affecting multiple teeth in both arches. It may range from yellow through brownish to dark gray, depending on the amount, frequency, and type of tetracycline as well as the patient's age (stage of development) during administration. Tetracycline discoloration has been classified into three groups according to severity.[6] First-degree discoloration is light yellow, light brown, or light gray and occurs uniformly throughout the crown without banding. Second-degree discoloration is more intense and is also without banding. Third-degree discoloration is very intense, and the clinical crown exhibits horizontal color banding. This type of discoloration usually predominates in the cervical region.

Tetracycline binds to calcium, which then is incorporated into the hydroxyapatite crystal in both enamel and dentin. Most of the tetracycline, however, is found in dentin. Chronic sun exposure of teeth with the incorporated drug may cause formation of a reddish-purple tetracycline oxidation by-product, resulting in further discoloration of permanent teeth.

A phenomenon of adult-onset tetracycline discoloration is also reported.[7] This type of discoloration occurs occasionally in mature teeth in patients receiving long-term minocycline therapy, which was usually given for control of cystic acne. The discoloration is gradual because of incorporation of minocycline in continuously forming dentin.[5] Staining generally is not severe.

Two approaches have been used for bleaching tetracycline discoloration. The first, which involves bleaching the external enamel surface, is limited to lighter, yellowish discoloration and requires multiple appointments to achieve a satisfactory result.[8] The second, root canal treatment followed by internal bleaching, is a more predictable procedure, is useful for all degrees of discoloration severity, and has proved successful in both the short and long term.[9]

Defects in Tooth Formation

Defects in tooth formation are confined to the enamel and are either hypocalcific or hypoplastic. Enamel *hypocalcification* is common, appearing as a distinct brownish or whitish area, often on the facial aspect of a crown. The enamel is well formed and intact on the surface and feels hard to the explorer. Both the whitish and the brownish spots are amenable to bleaching with the pumice and acid technique (described later in this chapter) with good results.

Enamel *hypoplasia* differs from *hypocalcification* in that the enamel in the former is defective and porous. This condition may be hereditary (amelogenesis imperfecta) or may result from environmental factors. In the hereditary type, both deciduous and permanent dentitions are involved. Defects caused by environmental factors may involve only one or several teeth. Presumably during tooth formation the matrix is altered and does not mineralize properly. The porous enamel readily acquires stains from the oral cavity. Depending on the severity and extent of hypoplasia and the nature of the stain, these teeth may be bleached (or corrected by the acid pumice method) from the enamel surface with some degree of success.[10] The bleaching effect may not be permanent, and stains may recur with time. These stains, however, can be recorrected. As stated earlier, it is most important to inform the patient of the likely reoccurrence of discoloration of these teeth.

Blood Dyscrasias and Other Factors

Various systemic conditions may cause massive lysis of erythrocytes. If this occurs in the pulp at an early age, blood disintegration products are incorporated into and discolor the forming dentin. An example of this phenomenon is the severe

discoloration of primary teeth that usually follows erythroblastosis fetalis. This disease in the fetus, or newborn, results from Rh incompatibility factors, which lead to massive systemic lysis of erythrocytes. Large amounts of hemosiderin pigment then stain the forming dentin of the primary teeth. This discoloration is not correctable by bleaching. However, this type of lysis is now uncommon because of new preventive measures.

High fever during tooth formation may result in linear defined hypoplasia. This condition, known as chronologic hypoplasia, is a temporary disruption in enamel formation that results in a banding type of surface defect that acquires stain. Porphyria, a metabolic disease, may cause deciduous and permanent teeth to show a red or brownish discoloration. Thalassemia and sickle cell anemia may cause intrinsic bluish, brown, or green discolorations. Amelogenesis imperfecta may result in yellowish or brownish discolorations. Dentinogenesis imperfecta can cause brownish violet, yellowish, or gray discoloration. These conditions are also not amenable to bleaching and should be corrected by minimally invasive restorative means.

Other staining factors related to systemic conditions or ingested drugs are rare and may not be identifiable.

Iatrogenic or Inflicted Discolorations

Discolorations caused by various chemicals and materials used in dentistry are usually avoidable. Many of these discolorations are difficult to correct by bleaching alone.

ENDODONTICALLY RELATED DISCOLORATIONS

Obturating Materials

Obturating materials are the most common and severe cause of single tooth discoloration. Incomplete removal of materials from the pulp chamber on completion of treatment often results in dark discoloration (Figures 22-3 and 22-4). Removing all obturation materials to a level just cervical to the gingival margin can prevent such discoloration. Primary offenders are sealer remnants, whether of the zinc oxide–eugenol type or plastics, which themselves also darken with time.[11] Sealer remnants gradually cause progressive coronal discoloration.[12] The prognosis of bleaching in such cases depends on the constituents of the sealer. Sealers with metallic components often do not bleach well, and any bleaching effect tends to regress with time.

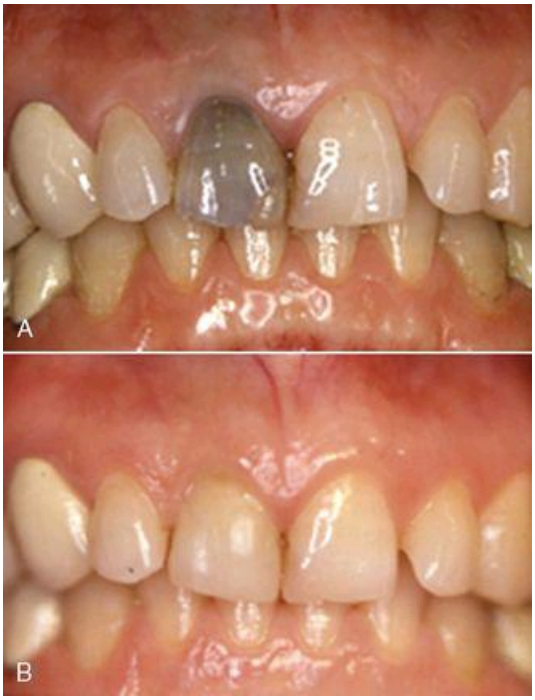

Figure 22-3 A, Discoloration as a result of trauma and subsequent treatment. The patient was involved in an accident causing coronal fracture. Root canal treatment was done, but gutta-percha and sealer were not completely removed from the pulp chamber. An additional discoloration factor was the defective leaking restoration. **B**, Two appointments of "walking bleach" and placement of a new well-sealed composite restored esthetics.

(Courtesy Dr. M. Israel.)

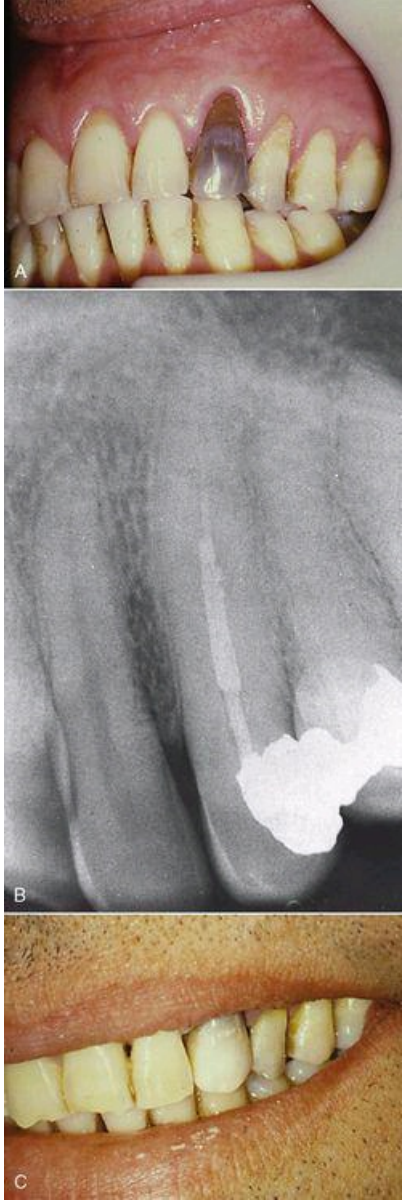

Figure 22-4 **A**, Severely discolored canine. **B**, Poor root canal treatment in which material extended into the pulp chamber caused some of the discoloration. **C**, After retreatment and three appointments of "walking bleach," esthetics has markedly improved. Although some cervical discoloration remains, this is largely hidden by the upper lip.

(Courtesy Dr. H. Libfeld.)

Remnants of Pulpal Tissue

Pulp fragments remaining in the crown, usually in pulp horns, may cause gradual discoloration. Pulp horns must be “opened up” and exposed during access to ensure removal of pulpal remnants and to prevent retention of sealer at a later stage. Internal bleaching in such cases is usually successful (Figure 22-5).

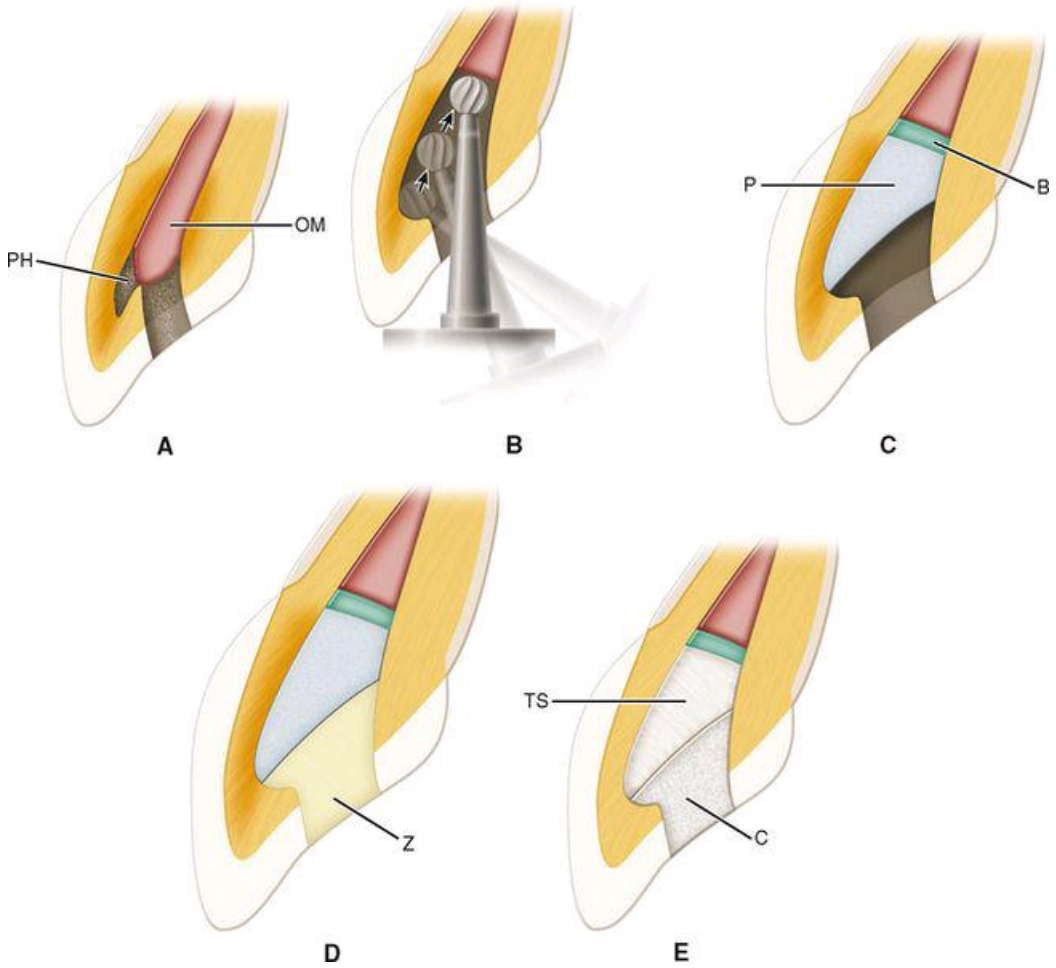

Figure 22-5 Walking bleach. **A**, Internal staining of dentin caused by remnants of obturating materials *(OM)* in the pulp chamber, as well as by materials and tissue debris in pulp horns *(PH)*. **B**, Coronal restoration is removed completely, access preparation is improved, and gutta-percha is removed apically to just below the cervical margin. Next, the pulp horns are cleaned with a round bur. (Shaving a thin layer of dentin from the facial wall is optional and may be attempted at later appointments if discoloration persists.) **C**, An optional protective cement base *(B)* is placed over the gutta-percha, not extending above the cervical margin. After removal of sealer remnants and materials from the chamber with solvents, a paste (*P*) composed of sodium perborate and water (mixed to the consistency of wet sand) is placed. The incisal area is undercut to retain the temporary restoration. **D**, A thick mix of zinc oxide–eugenol type temporary filling *(Z)* seals access. **E**, At a subsequent appointment, when the desired shade has been reached, a permanent restoration is placed. A suggested method is to fill the chamber with white temporary stopping *(TS)* or with light polycarboxylate or zinc phosphate base. Acid-etched composite *(C)* restores lingual access and extends into the pulp horns for retention and to support the incisal edge.

(From Walton RE: Bleaching procedures for teeth with vital and nonvital pulps. In Levine N, ed: Current treatment in dental practice, Philadelphia, 1986, Saunders.)

Intracanal Medicaments

Several medicaments have the potential to cause internal discoloration of the dentin.[13] Phenolic or iodoform-based intracanal medications, sealed in the root canal space, are in direct contact with dentin, sometimes for long periods, allowing for their penetration and oxidization. These compounds have a tendency to discolor the dentin gradually. Fortunately, most discolorations are not marked and are readily and permanently corrected by bleaching. Iodoform-induced discolorations tend to be more severe.

Coronal Restorations

Restorations are generally metallic or composite. The reasons for discoloration (and therefore the appropriate correction) are quite different.

Metallic Restorations

Amalgam is the worst offender because its dark-colored elements may turn dentin dark gray. If used to restore an access preparation, amalgam often discolors the crown (Figure 22-6). Such discolorations are difficult to bleach and tend to reoccur with time. However, bleaching them is worth a try. The result may be an improvement that satisfies the patient.

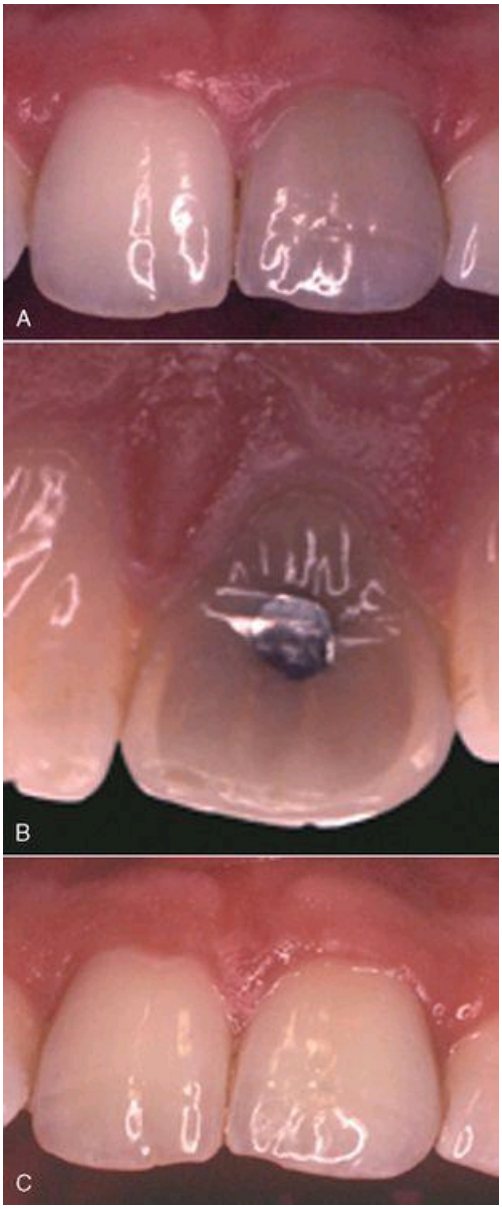

Figure 22-6 **A**, Discoloration of endodontically treated incisor. **B**, Failure to remove all remnants of pulpal tissue from the chamber and amalgam placed in the access cavity appear to be the causes of discoloration, **C**, Removal of amalgam, intracoronal bleaching, and placement of a new composite restored esthetics.

(Courtesy Dr. A. Claisse.)

Discoloration from inappropriately placed metal pins and prefabricated posts in anterior teeth may sometimes occur. This is caused by metal that is visible through the composite or tooth structure. Occasionally, discoloration from amalgam is also caused by visibility of the restoration through translucent tooth structure. In such cases, replacement of old metallic restorations with an esthetically pleasing composite may suffice.

Composite Restorations

Microleakage of composites causes discoloration. Open margins may permit chemicals to permeate between the restoration and tooth structure to stain the underlying dentin. In addition, composites may become discolored with time and alter the shade of the crown. These conditions can sometimes be corrected by replacing the old composite with a new well-sealed esthetic restoration. In many cases, internal bleaching is carried out first with good results.

BLEACHING MATERIALS

Bleaching chemicals may act as either oxidizing or reducing agents. Most bleaching agents are oxidizers, and many preparations are available. Commonly used agents are solutions of hydrogen peroxide of different strengths, sodium perborate, and carbamide peroxide. Sodium perborate and carbamide peroxide are chemical compounds that are gradually degraded to release low levels of hydrogen peroxide. Hydrogen peroxide and carbamide peroxide are mainly indicated for external bleaching, whereas sodium perborate is mostly used for internal bleaching. All have proved effective.

Hydrogen Peroxide

Hydrogen peroxide is a powerful oxidizer that is available in various strengths, but 30% to 35% stabilized solutions (Superoxyl, Perhydrol) are the most common. These high-concentration solutions must be handled with care because they are unstable, lose oxygen quickly, and may explode unless they are refrigerated and stored in a dark container. Also, these are caustic chemicals and will burn tissue on contact.

Although 30% to 35% hydrogen peroxide will bleach quickly, other chemicals that release much lower levels of peroxide are available; usually they will bleach effectively with longer application periods.[14]

Sodium Perborate

Sodium perborate is available in powder form or in various commercial proprietary combinations. When fresh, it contains about 95% perborate, corresponding to 9.9% available oxygen. Sodium perborate is stable when dry, but in the presence of acid, warm air, or water, it decomposes to form sodium metaborate, hydrogen peroxide, and nascent oxygen.[15] Various types of sodium perborate preparations are available: monohydrate, trihydrate, and tetrahydrate. They differ in oxygen content, which determines their bleaching efficacy.[16] Commonly used sodium perborate preparations are alkaline; their pH depends on the amount of hydrogen peroxide released and the residual sodium metaborate.[17]

Sodium perborate is more easily controlled and safer than concentrated hydrogen peroxide solutions.[2,15,18] Therefore it should be the material of choice for internal bleaching.

Carbamide Peroxide

Carbamide peroxide, also known as urea hydrogen peroxide, is usually available in concentrations varying between 3% and 15%. Popular commercial preparations contain about 10% carbamide peroxide and have an average pH of 5 to 6.5. They usually also include glycerin or propylene glycol, sodium stannate, phosphoric or citric acid, and flavor. In some preparations, Carbopol, a water-soluble resin, is added to prolong the release of active peroxide and to improve shelf-life. Ten percent carbamide peroxide breaks down into urea, ammonia, carbon dioxide, and approximately 3.5% hydrogen peroxide.

Carbamide peroxide systems are mostly used for external bleaching and have been associated with varying degrees (usually slight) of damage to teeth and surrounding mucosa.[19,20] They may adversely affect the bond strength of composite resins and their marginal seal.[19,21,22] Therefore these materials must be used with caution and usually under strict supervision of the dentist.

Other Agents

In the past, a preparation of sodium peroxyborate monohydrate (Amosan), which releases more oxygen than does sodium perborate, was recommended for internal bleaching.[23] Today, its clinical use is less common.

Sodium hypochlorite is a common root canal irrigant that is available commercially as a 3% to 6% household bleach. Although used as a household bleaching agent, it does not release enough oxidizer to be effective and is not recommended for routine bleaching.

Other nonperoxide bleaching agents were also suggested for clinical use; however, these have been no more effective than traditional agents.[24,25]

INTERNAL (NONVITAL) BLEACHING TECHNIQUES

The methods most commonly used to bleach teeth in conjunction with root canal treatment are the *thermocatalytic* technique and the so-called *walking bleach* technique.[15,23] These techniques are somewhat different, but both produce similar results.[2,3] The walking bleach technique is preferred because it requires the least chair time and is more comfortable and safer for the patient. The walking bleach technique is described in a later section. Whatever technique is used, the active ingredient is the oxidizer, which is available in different chemical forms. The least potent form is preferred.

Indications for internal bleaching technique are (1) discolorations of pulp chamber origin, (2) dentin discolorations, and (3) discolorations that are not amenable to external bleaching. Contraindications are (1) superficial enamel discolorations, (2) defective enamel formation, (3) severe dentin loss, (4) presence of caries, and (5) discolored proximal composites (unless they are replaced after bleaching).

Thermocatalytic Technique

The thermocatalytic technique involves placing the oxidizing agent in the pulp chamber and then applying heat. Heat may be supplied by heat lamps, flamed instruments, or electrical heating devices, which are manufactured specifically to bleach teeth.

Potential damage from the thermocatalytic approach includes the possibility of external cervical root resorption because of irritation to cementum and the periodontal ligament, possibly from the oxidizing agent in combination with heat.[25,26] Therefore the application of heat during bleaching should be limited. The thermocatalytic technique has not proved more effective than other methods and is not recommended for routine internal bleaching.

A thermocatalytic variation is ultraviolet photooxidation. A 30% to 35% hydrogen peroxide solution is placed in the chamber on a cotton pellet, followed by a 2-minute exposure to ultraviolet light applied to the labial surface. Supposedly this causes the release of oxygen similar to that seen in other thermocatalytic bleaching techniques.[27,28]

There has been little clinical experience with ultraviolet photo-oxidation. It is probably no more effective than the walking bleach technique and requires more chair time. Because of toxicity considerations of concentrated hydrogen peroxide, this technique is not recommended.

Walking Bleach

The walking bleach technique should be used in all situations requiring internal bleaching. Not only is it as effective as the techniques previously described, but it also is the safest and requires the least chair time (Box 22-1).[29-32]

Box 22-1 Walking Bleach Technique

The steps involved in walking bleach are as follows (see Figure 22-5):

1. As previously stated, the patient is familiarized with the probable causes of staining, the procedure to be followed, the expected outcome, and the possibility of future reoccurrence of discoloration (regression). To avoid disappointment or misunderstanding, effective communication before, during, and after treatment is absolutely necessary.

2. Radiographs are made to assess the status of the periapical tissues and the quality of root canal treatment. Treatment failure or questionable obturation requires retreatment before bleaching.

3. The quality and shade of any restoration present are assessed. If defective, the restoration is replaced. Often, tooth discoloration results from leaking or discolored restorations. Also, the patient is informed that the bleaching procedure may temporarily (or permanently) affect the color of the restoration, requiring its replacement.

4. Tooth color is evaluated with a shade guide, and clinical photographs are taken at the beginning of and throughout the procedure. These provide a point of reference for future comparison by both dentist and patient.

5. The tooth is isolated with a rubber dam. Interproximal wedges may also be used for better isolation. If Superoxyl is used, protective cream (such as petroleum jelly, Orabase, or cocoa butter) must be applied to the gingival tissues before dam placement. This protection is not required with sodium perborate use.

6. The restorative material is removed from the access cavity (see Figure 22-5, *B*). Refinement of access and removal of all old obturating materials from the pulp chamber comprise the most important stage in the bleaching process. There must be a check that pulp horns or other "hidden" areas are opened.

A chamber totally filled with composite resin presents a clinical problem. First, this material is resistant to cutting with burs. Second, its shade is often indistinct from that of dentin. However, all composite must be removed to allow the bleaching agent to contact and penetrate the dentin. Care must be taken during restoration removal to avoid inadvertent cutting of sound dentin. The operating microscope or magnifying loupes are beneficial.

7. (Optional) This step may be necessary if the discoloration seems to be of metallic origin or if on the second or third appointment bleaching alone does not seem to be sufficient. A thin layer of stained dentin is carefully removed toward the facial aspect of the chamber with a round bur in a slow-speed handpiece (see Figure 22-5, *B*). This will remove much of the discoloration (which is concentrated in the pulpal surface area). It may also open the dentinal tubules for better penetration by the bleaching agents.

8. All materials should be removed to a level just apical to the gingival margin. Appropriate solvents (such as orange solvent, chloroform, or xylol on a cotton pellet) are used to dissolve remnants of the common sealers.

9. If Superoxyl is used, a sufficient layer of protective cement barrier (such as polycarboxylate, zinc phosphate, glass ionomer, intermediate restorative material [IRM], or Cavit at least 2 mm thick) is applied on the obturating material. This is essential to minimize leakage of bleaching agents.[29] The barrier should protect the dentin tubules and conform to the external epithelial attachment.[30] It should not extend incisal to the gingival margin (see Figure 22-5, *C*).

Acid etching of dentin internally with phosphoric (or other) acid to remove the smear layer and open the tubules is not effective.[31] The use of any caustic chemical in the chamber is unwarranted because periodontal ligament irritation or external root resorption may result. The same reservation applies to solvents such as ether or acetone before application of the bleaching agent. The application of concentrated hydrogen peroxide with heat (thermocatalytic) has been suggested as the next step. As mentioned earlier, this is questionable from a safety standpoint.

10. The walking bleach paste is prepared by mixing sodium perborate and an inert liquid, such as water, saline, or anesthetic solution, to a consistency of wet sand (approximately 2 g/ml). Although sodium perborate mixed with 30% hydrogen peroxide will bleach faster, in most cases, the long-term results are similar to those of sodium perborate mixed with water and therefore the former mixture should not be used routinely.[2,3,15,32] Another advantage of sodium perborate and inert liquid is that the protective cement barrier and gingival protection are unnecessary. With a plastic instrument, the pulp chamber is packed with the paste. Excess liquid is removed by tamping with a cotton pellet. This also compresses and pushes the paste into the recesses (see Figure 22-5, *C*).

11. Excess oxidizing paste is removed from undercuts in the pulp horns and gingival area with an explorer. A cotton pellet is not used but a thick mix of Cavit or zinc oxide–eugenol (preferably IRM) is packed carefully to a thickness of at least 3 mm to ensure a good seal (see Figure 22-5, *D*).

12. The rubber dam is removed. The patient is informed that the bleaching agent works slowly and that significant lightening may not be evident for 2 or more weeks. It is common to see no change initially, but dramatic results occur in successive days or weeks or after a future reapplication.

13. The patient is scheduled to return approximately 2 to 6 weeks later, and the procedure is repeated. If at any future appointment (third or fourth), progressive lightening is not evident, further walking bleach treatments with sodium perborate and water solution may not prove

beneficial.[32]

It is commonly believed that “overbleaching” is desirable because of future reoccurrence of discoloration. However, bleaching a tooth to a lighter shade than its neighbors should be performed with caution because the overbleached tooth may not discolor again.[33] A tooth that is too light may be as unesthetic as one too dark.

Repeat treatments are similar. If early bleaching does not provide satisfactory results, the following additional procedures may be attempted: (1) a thin layer of stained facial dentin is removed with a small round bur (see step 7) and (2) the walking bleach paste is strengthened by mixing the sodium perborate with increasing concentrations of hydrogen peroxide (3% to 30%) instead of water. The more potent oxidizer may enhance the bleaching effect but may increase the risk of subsequent root resorption.[26,34]

Carbamide peroxide was also suggested for internal bleaching.[35] This agent, however, is probably not superior to sodium perborate.

Although usually the final results are excellent, occasionally only partial lightening is achieved. Surprisingly, the patient often is very pleased and satisfied with a modest improvement and does not expect perfection.[35] Therefore internal bleaching is worth the attempt.

Final Restoration

Proper tooth restoration is essential for long-term successful internal bleaching results.[36] The pulp chamber and access cavity are restored at the final visit (see Figure 22-5, *E*). Although it has been proposed that substances such as acrylic monomer or silicones be placed in the chamber to fill the dentinal tubules, this is not beneficial. Furthermore, these substances may themselves lead to discoloration with time. However, it is important to restore the chamber carefully and to seal the lingual access to enhance the new shade and prevent leakage. The ideal method for filling the chamber after tooth bleaching has not been determined. However, the chamber must not be filled totally with composite; this may cause a loss of translucency of the tooth.[37]

It is easy and effective to fill the chamber with a light-colored gutta-percha temporary stopping, glass ionomer, or a light shade of zinc phosphate cement and then to restore the lingual access with a light-cured acid-etched composite.[38] An adequate depth of composite should be ensured to seal the cavity and provide some incisal support. Light curing from the labial, rather than the lingual, surface is recommended because this results in shrinkage of the composite resin toward the axial walls, reducing the rate of microleakage.[39] Coronal microleakage of lingual access restorations is a problem[40]; a leaky restoration may lead to reoccurrence of discoloration.

Residual peroxides of bleaching agents, mainly hydrogen peroxide and carbamide peroxide, may affect the bonding strength of composites to the tooth.[22,41,42] Sodium perborate mixed with water results in much less loss of bond strength than does concentrated hydrogen peroxide.[43] Therefore it is not recommended that the tooth be restored with composite immediately after bleaching but only after an interval of a few days. The use of catalase has also been proposed for fast elimination of residual peroxides from the access cavity[44]; this merits further investigation.

It has been suggested that packing calcium hydroxide paste in the chamber for a few weeks before the final restoration is placed would reverse the acidity caused by bleaching agents and prevent resorption; however, this procedure is ineffective and unnecessary.[17,34]

Future Rediscoloration

Although initial bleaching is successful, many of these teeth will rediscolor after several years.[45] Patients must be informed of this possible occurrence and that rebleaching usually will be successful.

When to Bleach

Internal bleaching may be performed at various intervals after root canal treatment (see Figure 22-1; Figure 22-6). The appearance of the discolored tooth may be improved soon after treatment. However, the walking bleach technique may be initiated at the same appointment as the obturation. In fact, this may motivate the patient to accept bleaching because the appearance of the discolored tooth may be improved soon after treatment. Bleaching may also be attempted successfully many years after discoloration has occurred (see Figures 22-3 and 22-4), even in the presence of porcelain veneer restorations (Figure 22-7). Such teeth show no markedly greater tendency toward reoccurrence of discoloration than teeth stained for shorter discoloration periods.[33] However, it is probable that a shorter discoloration period tends to improve the chances for successful bleaching, as well as to reduce the likelihood of reoccurrence of discoloration.[46]

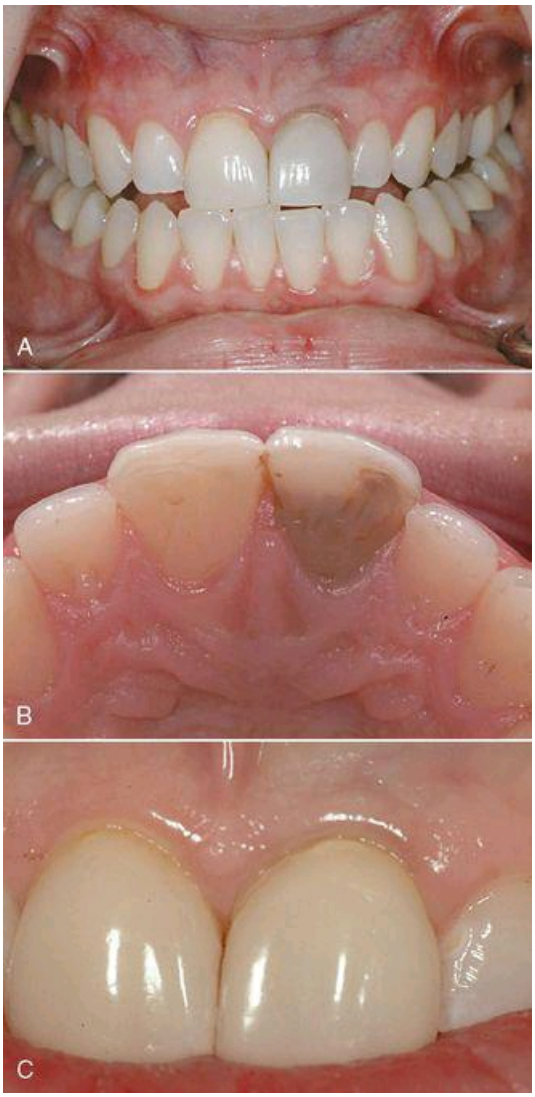

Figure 22-7 A, Discoloration of endodontically treated incisor restored with a porcelain veneer. Discoloration is reflected through the veneer and is most evident at the cervical area. **B**, Lingual view reveals extensive discoloration of the dentin and composite that was used to restore the access cavity. **C**, Removal of discolored composite, internal bleaching, and placement of a new well-sealed composite restored tooth esthetics.

(Courtesy Dr. A. Sameni.)

Other factors that may influence long-term success have also been evaluated clinically. The patient's age and the rate of discoloration have no major effect on the long-term stability of bleaching.[33]

COMPLICATIONS AND SAFETY

Patient safety is always the major concern in any procedure. Some possible adverse effects produced by chemicals and bleaching procedures are listed in the following section.

External Root Resorption

Clinical reports[47-49] and histologic studies[26,34] have shown that internal bleaching may induce external root resorption. The oxidizing agent, particularly 30% hydrogen peroxide, may be the culprit. However, the exact mechanism by which periodontium or cementum is damaged has not been elucidated. Presumably, the irritating chemical diffuses through the dentinal tubules[50] and reaches the periodontium through defects in the cementoenamel junction.[51] Chemicals combined with heat are likely to cause necrosis of the cementum, inflammation of the periodontal ligament, and subsequent root resorption.[26,34] The process is liable to be enhanced in the presence of bacteria.[52] Previous traumatic injury and young age may also act as predisposing factors.[47]

Therefore injurious chemicals and procedures should be avoided if they are not essential for bleaching. Also, apical to the cervical margin, oxidizing agents should not be exposed to more of the pulp space and dentin than is absolutely necessary to obtain a satisfactory esthetic clinical result.

Coronal Fracture

Increased brittleness of the coronal tooth structure, particularly when heat is applied, is also thought to result from bleaching. This supposedly is a result of either desiccation or alterations to the physicochemical characteristics of the dentin and enamel.[53-55] Clinical experience suggests that bleached teeth are no more susceptible to fracture, although this has not been proved conclusively.

Chemical Burns

As mentioned earlier, sodium perborate is safe, but 30% hydrogen peroxide is caustic and will cause chemical burns and sloughing of the gingiva. When this strong chemical is used, the soft tissues should be coated with an isolation cream such as petroleum jelly, Orabase, or cocoa butter. Animal studies suggest that catalase applied to oral tissues before hydrogen peroxide treatment fully prevents the associated tissue damage.[56]

INTRINSIC DISCOLORATIONS

Intrinsic discolorations are those incorporated into tooth structure during tooth formation.[57] Significantly, most of these discolorations are in dentin and are relatively difficult to treat externally.[58] A good example is staining from tetracycline, which is incorporated into the mineral structure of the developing tooth. The incorporated tetracycline imparts its color to the dentin.

Tetracycline

Both external and internal bleaching techniques have been advocated as a means of improving the appearance of tetracycline-discolored teeth. As noted earlier, the internal technique is more effective, with very good long-term prognosis.[9,57,58] However, the best resolution for tetracycline discolorations is prevention.

The technique involves root canal treatment followed by an internal walking bleach technique, as outlined earlier in this chapter. If the procedure is explained to patients, they usually accept this approach with gratifying results (Figure 22-8).

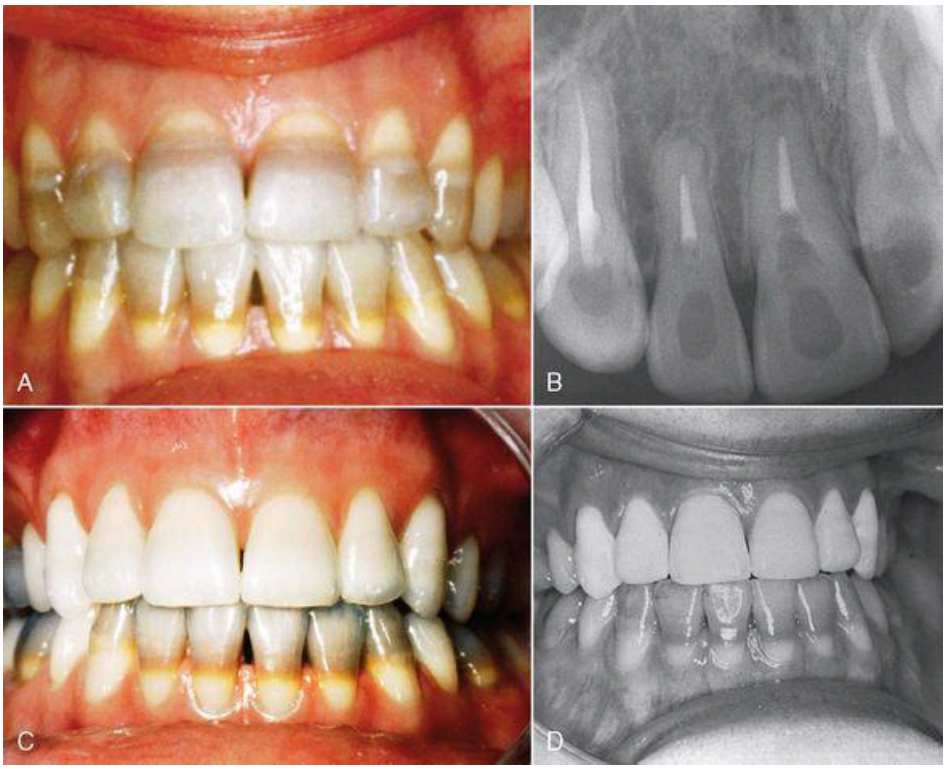

Figure 22-8 A, Characteristic grayish discoloration and banding of tetracycline discolorations. Cervical regions on maxillary and mandibular teeth show no discoloration; tetracycline was not administered during those periods of tooth development. **B**, Root canal treatment has been completed on the maxillary anterior teeth, with subsequent "walking bleach" procedures. **C**, After the necessary number of bleaching appointments, the teeth are restored permanently. Note the marked contrast with the mandibular incisors, which remain untreated. **D**, A 4-year follow-up shows no regression and no recurrence of discoloration.

(Courtesy Dr. H. Wayne Mohom.)

Other Intrinsic Discolorations

Other drugs or ingested chemicals are incorporated into teeth that are forming and cause discoloration. There are no reports of attempts to bleach these teeth. Presumably, attempts to lighten teeth with dentinal discolorations by the external application of bleaching agents would be only marginally effective.

EXTRINSIC DISCOLORATIONS

Extrinsic discolorations are more superficial and are obviously more amenable to external bleaching. The success of bleaching, however, depends more on the depth of the stain in the enamel rather than on the color of the stain itself.

Superficial Defects

Although a number of conditions may result in hypoplasia of enamel accompanied by porosity, the most common and often the most disfiguring is endemic fluorosis. This hypoplastic defect may result in various degrees and colors of superficial stains.

Mechanism of Discoloration

Ingestion of high levels of fluoride during tooth formation disrupts the ameloblasts and leads to hypoplasia of the forming enamel.[59] After tooth eruption, the porous enamel is gradually discolored. The discoloration may vary from chalky white through light yellow and brown to almost black, depending on the degree of fluorosis (enamel porosity) and the type of chemical stain.

The appearance of all such discolorations may be improved with bleaching or other measures, some dramatically. Although a variety of techniques involving different chemicals and procedures have been suggested, probably the most effective is the microabrasion "controlled hydrochloric acid–pumice abrasion" technique.[10] This is not a true bleaching (oxidizing) technique but decalcification and removal of a thin layer of stained enamel. This technique has been modified somewhat since its development in the mid 1980s.[10,60] This technique is useful mainly for fluorosis and other extrinsic discolorations and has proved to be very effective.[61]

Microabrasion Technique

The procedure is as follows (Figure 22-9):

1. The teeth to be treated are photographed to serve as a permanent record and as a basis for future comparison.

2. The gingiva is protected, and the teeth are carefully isolated with an inverted rubber dam and ligatures. The rubber dam is extended over the patient's nostrils.

3. Exposed areas of the patient's face and eyes are covered with a suitable drape or towel for added safety from acid spatter.

4. A 36% hydrochloric acid solution is mixed with an equal volume of distilled water to make an 18% hydrochloric acid solution. A substantial amount of fine flour of pumice is added to form a thick paste. In another dappen dish, sodium bicarbonate and water are mixed to a thick paste, which will be used later for acid neutralization. Ready-made commercial products are also available.

5. The hydrochloric acid and pumice paste is applied to the enamel surface with a piece of wooden tongue blade or crushed orangewood stick. Exerting firm pressure, the paste is worked into the enamel surface with a scrubbing motion for 5 seconds. The enamel surface is then rinsed for 10 seconds with water.

6. The paste is reapplied until the desired color is achieved.

7. The surface is neutralized with sodium bicarbonate and water. The rubber dam is removed, and the teeth are pumiced with a fine prophylactic paste to smooth the abraded surface. Usually the desired shade is obtained in a single appointment. If not, the stains may be too deep and not amenable to lightening.

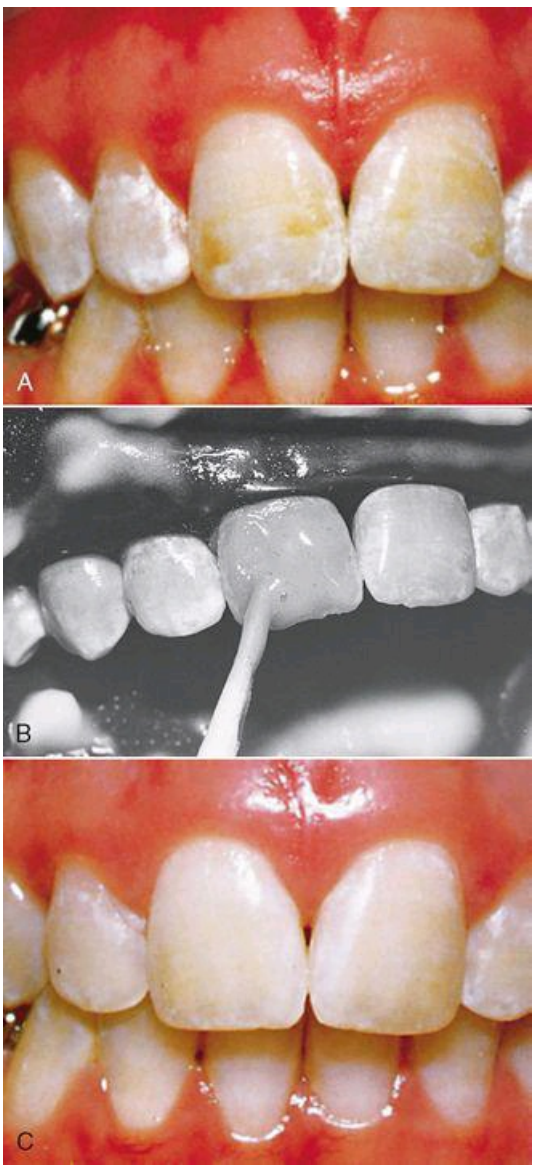

Figure 22-9 Controlled hydrochloric acid–pumice microabrasion technique. **A**, Marked discoloration results from surface hypoplastic defects. **B**,

Hydrochloric acid 18% is mixed with fine flour of pumice to form a thick paste, which is applied to the surface with a crushed orangewood stick. The paste is worked into the enamel with rubbing motions for 5 seconds, then followed with a water rinse. Paste is reapplied as necessary. Finally, acid is neutralized with sodium bicarbonate. **C**, The desired improvement was obtained in a single appointment. There was no regression.

(Courtesy Dr. S. Goepferd.)

Prognosis

The acid-pumice abrasion technique is relatively permanent if initial lightening is achieved. Many patients have been followed for long periods of time with no reoccurrence of discoloration.[10]

Safety

There are two areas in which safety is a concern: the effects on enamel (excessive decalcification) and chemical burns of soft tissue.

With care and judicious application of acid in either of the hydrochloric acid techniques, an insignificant amount of enamel is removed. Chemical burns of the gingiva by either concentrated acid or hydrogen peroxide may be easily prevented by coating the gingiva with isolating materials, inverting the rubber dam, and ligating the teeth. There are minimal to no pulpal effects.

WHEN AND WHAT TO REFER

Most bleaching procedures can be performed by general dentists, particularly if the cause of the discoloration is diagnosed. If the general practitioner cannot make this identification, referral to a specialist should be considered.

The practitioner may also wish to refer patients whose tooth discoloration does not respond to conventional methods of bleaching, either external or internal. Unidentified factors may be preventing the bleaching chemicals from effectively reaching the stain. The specialist may be able to identify and correct these factors.

Chapter Review Questions available in Appendix B or on the DVD

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Geriatric Endodontics

Richard E. Walton

CHAPTER OUTLINE

LEARNING OBJECTIVES

After reading this chapter, the student should be able to:

1. Identify those biologic aspects in the elderly patient that are similar to and different from those in the younger patient.

2. Discuss age changes in the older dental pulp, both physiologic and anatomic.

3. Discuss differences in healing patterns in the older patient.

4. Describe complications presented by the medically compromised older patient.

5. Describe each step of the process of diagnosis and treatment planning in the elderly patient.

6. Identify factors that complicate case selection.

7. Discuss why there are differences and what those differences are when root canal treatment is performed in the older patient.

8. Recognize the complications of endodontic surgery.

9. Select the appropriate restoration after root canal treatment.

10. Identify those elderly patients who should be considered for referral.

Endodontic considerations in the elderly patient are similar in many ways to those in the younger patient but with differences. This chapter discusses the similarities and concentrates on the differences. The topics include the biologic aspects of pulpal and periradicular tissues, healing patterns, diagnosis, and treatment aspects in the geriatric patient.

The number of persons aged 65 and over in the United States exceeds 35 million and is expected to comprise 20% of the population by 2020. Their dental needs will also continue to increase.[1-3] More elderly patients will not accept tooth extraction unless there are no alternatives.[4,5] They have a high utilization rate of dental services.[6] Their expectations for dental health parallel their demands for quality medical care. An even more important consideration is that their dentitions will have experienced decades of dental disease, as well as restorative[4] and periodontal procedures (Figure 23-1). These all have compound adverse effects on the pulp, periradicular, and surrounding tissues (Figure 23-2). In other words, the more injuries that are inflicted, the greater the likelihood of irreversible disease and thus the greater the need for treatment. A recent survey reported that the number of elderly endodontic patients is increasing; this will continue.[7]

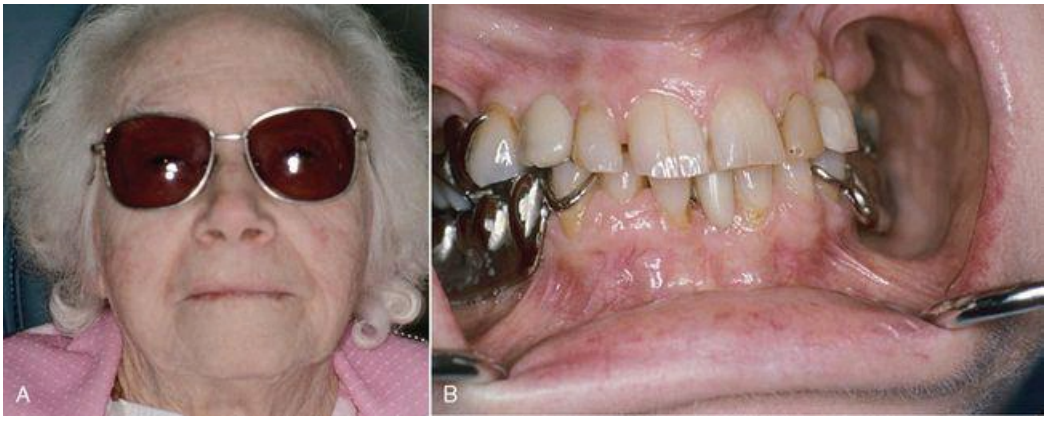

Figure 23-1 A, This 87-year-old woman has Alzheimer's disease. **B,** Her dentition shows diverse problems caused by many years of disease, restorations, and oral and systemic changes. Diagnosis is challenging, and the dentition will be difficult to restore to acceptable function and esthetics, particularly in a patient with mental impairment.

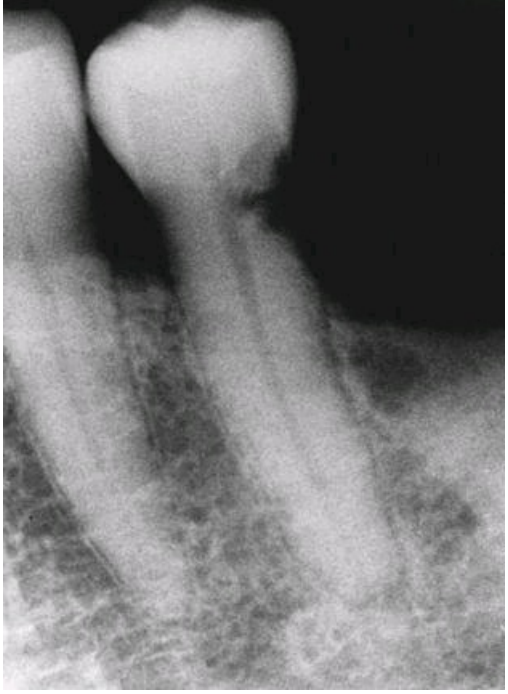

Figure 23-2 Cervical external resorption exposing the pulp. A free-end removable partial denture has settled posteriorly, exerting pressure on the gingiva and inducing inflammation and root resorption.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

The combination of an increase in pathosis and dental needs coupled with greater expectations has resulted in more endodontic procedures among these aging patients (Figure 23-3). Furthermore, expanded dental insurance benefits for retirees and more disposable income have made complex treatment more affordable.[1] Other means will likely be available to finance the costs of oral health care in the future.[8]

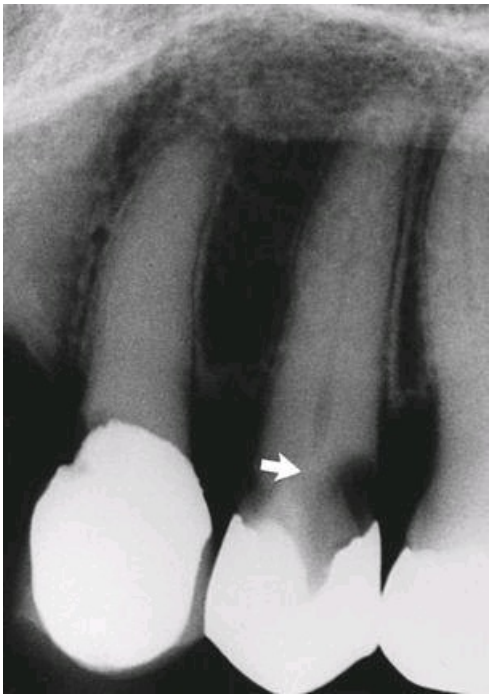

Figure 23-3 Restorations, caries, and time have all combined to result in dentin formation. The first premolar shows calcific metamorphosis (a very small pulp space is present). The second premolar has dentin formation *(arrow)* in response to recurrent caries. Both will be difficult to treat and restore.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

Endodontic considerations in elderly patients include biologic, medical, and some psychologic differences from younger patients, as well as treatment complications. These considerations are further discussed in this chapter.

BIOLOGIC CONSIDERATIONS

Biologic considerations are both systemic and local. The wide variety of systemic changes related to the patient's medical status are covered in other textbooks. In the older patient, systemic or local changes unique to endodontics are no different from those for other dental procedures. Similarly, pulp and periradicular tissues do not respond markedly differently.

PULP RESPONSE

Changes with Age

There are two considerations: (1) structural (histologic) changes that take place as a function of time and (2) tissue changes that occur in response to irritation from injury. These tend to have similar appearances in the pulp. In other words, injury may prematurely "age" a pulp. Therefore an "old" pulp may be found in a tooth of a younger person (i.e., a tooth that has experienced caries, restorations, and so on). Whatever the etiology, these older (or injured) pulps react somewhat differently than do younger (or noninjured) pulps.

Chronologic Versus Physiologic

Does a pulp in an older individual react differently than an injured pulp in a younger individual? This question has not been answered totally. Probably a previously injured pulp (from caries, restoration, and so on) in a younger person has *less* resistance to injury than an undamaged pulp in an older individual. At a histologic level, there are some consistent changes in these older pulps, as well as in irritated pulps.

Structural

The pulp is a dynamic connective tissue. It has been well documented that with age there are changes in cellular, extracellular, and supportive elements (see Chapter 1). There is a decrease in cells, including both odontoblasts and fibroblasts. There are also fewer supportive elements (i.e., blood vessels and nerves).[9,10] Fewer and smaller vessels result in a decrease in blood flow in the pulp[11]; the significance of this decrease is unknown. Capillaries show somewhat degenerative changes in the endothelium with age.[12] There is presumably an increase in the percentage of space occupied by collagen but less ground substance; these changes in proportions have not been measured but only have been observed histologically.[13]

Calcifications

Calcifications include denticles (pulp stones) and those that are diffuse (linear). These increase in the aged pulp,[14] as well as in the irritated pulp.[15] Pulp stones tend to be found in the coronal pulp, and diffuse calcifications are found in the radicular pulp. It has been speculated that the niduses of calcification arise from degenerated nerves or blood vessels, but this has not been proved. Another common speculation is that pulp stones may cause odontogenic pain; however, this is not true.

Dimensional

Generally, pulp spaces progressively decrease in size and often become very small.[16,17] Dentin formation is not necessarily continuous throughout life, but it often does occur and may be accelerated by irritation from caries, restorations, and periodontal disease. Dentin formation with time or irritation is not uniform. For example, in molar pulp chambers there is more dentin formation on the roof and floor than on the walls.[9] The result is a flattened (disclike) chamber (Figure 23-4).

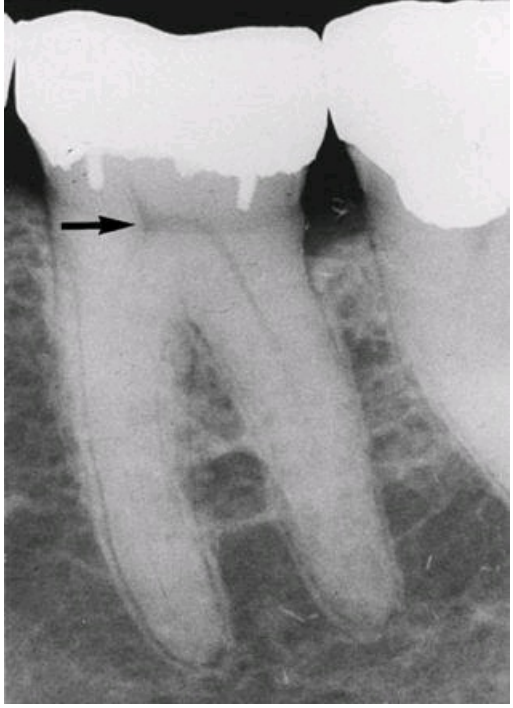

Figure 23-4 Disclike chamber *(arrow)*. The chamber is flattened because of dentin formation on the roof and floor. These chambers and canals are a challenge to locate.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

Nature of Response to Injury

The older patient does tend to have more adverse pulpal reactions to irritation than the reactions that occur in the younger patient. The reason for these differences is debatable and not fully understood, but they are probably the result of a lifetime of cumulative injuries.

From Irritation

There are reasons for pulp pathosis after restorative procedures. First, the tooth may have experienced several injuries in the past. Second, there are likely to be more extensive procedures that involve considerable tooth structure such as crown preparation. There are multiple potential injuries associated with a full crown such as foundation placement, bur preparation, impressions, temporary crown placement (often these leak), cementation, and unsealed crown margins. The coup de grâce of a pulp that is already stumbling along may be that final restoration.

Age

Although it would seem that a pulp with fewer cells, blood vessels, and nerves would be less resistant to injury, this has not been proven. Pulp responses to various procedures in different age groups have not shown differences, although the large number of variables in these types of clinical studies make it difficult to isolate age as a factor. This is not necessarily the case with the immature tooth (open apex) in which pulps have indeed been shown to be more resistant to injury. There is a theory that pulps in older teeth may in fact be *more* resistant because of decreased permeability of dentin.[18] Again, this resistance to injury in old teeth has not been proven.[19] The bottom line is that older pulps in older patients do require more care in preparation and restoration; this is probably the result of a history of previous insults rather than age per se.

Systemic Conditions

There is no conclusive evidence that systemic or medical conditions directly affect (decrease) pulp resistance to injury. One proposed condition is atherosclerosis, which has been presumed to directly affect pulp vessels[20]; however, the phenomenon of pulpal atherosclerosis could not be demonstrated.[21]

PERIRADICULAR RESPONSE

Little information is available on changes of bone and soft tissues with age and how these might affect the response to irritants or to subsequent healing after removal of those irritants. The indicators are that there is relatively little change in periradicular cellularity, vascularity, or nerve supply with aging.[22] Therefore it is unlikely that there are significantly different periapical responses in older compared with younger individuals.

HEALING

A popular concept is that healing in older individuals is impaired, compromised, or delayed when compared with healing in younger patients. This is not necessarily true. Studies in animals have shown remarkably similar patterns of repair of oral tissues in young versus old but with a slight delay in healing response.[23] Radiographic evidence of healing of younger versus older patients after root canal treatment demonstrated no apparent difference in success and failure.[24] No evidence exists that vascular or connective tissue changes in older individuals result in significantly slower or impaired healing. Overall, there is little difference in the nature of healing between the age groups, including healing of both bone and soft tissue. Vascularity is critical to healing, and in healthy individuals, periradicular blood flow is not impaired with age.[25]

MEDICALLY COMPROMISED PATIENTS

Certainly, systemic problems in the older patient tend to occur more often and with greater severity. In general, medical conditions are no more significant for endodontic procedures in the older patient than for other types of dental treatment. In fact, there is little information on the relationship of medical conditions or medically compromised patients as to adverse reactions during or after endodontic procedures. It has been presumed that systemic conditions, such as human immunodeficiency virus (HIV), diabetes, or immunosuppressant therapy, would predispose an endodontic patient to infection or to delayed healing. A recent retrospective study compared periradicular healing between HIV positive and negative patients 1 year after endodontic treatment of teeth with necrotic pulps and chronic apical periodontitis. It found no statistically significant differences between the two groups with respect to the degree of periradicular healing.[26] There is no definitive evidence that immunosuppressants compromise healing or predispose a patient to infection. However, both type I and II diabetes will make healing less predictable,[27] although this is true at any age. Garber and associates found less dentin bridge formation in exposed pulps of rats with induced diabetes compared with those rats without diabetes.[28] There is particular concern about the person with severe, uncontrolled diabetes who may require additional precautions and careful monitoring.[29]

Another common condition is hypertension. Contrary to popular belief, using epinephrine in local anesthetics in hypertensive patients carries a very low risk of adverse effects.[30]

Evidence exists that osteoporosis, a rather common condition of postmenopausal women, is associated with a decrease in trabecular bone density in the jaws, particularly in the anterior maxilla and the posterior mandible.[31,32] However, it is not known whether patients with osteoporosis have impaired bony healing after root canal treatment or surgery. As related to diagnosis of periapical pathosis, osteoporotic changes are probably not of sufficient magnitude[33] to confuse pretreatment or posttreatment evaluation. Interestingly, analysis of optical density from periapical radiographs from the posterior mandible is an indicator of osteoporotic changes in lumbar and femoral regions in the elderly.[34]

Bisphosphonates are one therapeutic agent that is of concern. This drug counteracts conditions or diseases that are associated with bone resorption. Oral bisphosphonates are extensively prescribed to postmenopausal females (and also to males) who are osteoporotic. Potent intravenous (IV) bisphosphonates are used to treat the effects of metastatic breast and prostate cancers on bone, as well as bone resorption defects from multiple myeloma and acute hypercalcemia. A rare side effect of this family of drugs is osteonecrosis of the jaw, particularly with IV administration. Patients on bisphosphonates should be carefully monitored to attempt to minimize the occurrence of pathosis.[35] Dental treatment should be noninvasive. Rubber dam placement should be used with care to avoid hard and soft tissue trauma. Root canal treatment should be performed without injuring apical tissues with instruments, irrigants, and materials. In addition, surgical procedures, including extractions, root-end surgery, and periodontal surgery, should be avoided if possible. Evidence does *not* suggest that bisphosphonate therapy should be altered or interrupted during dental procedures.[36]

In summary, elderly medically compromised patients are generally at no more risk for complications than are other age groups. In fact, for a medically compromised patient, root canal treatment or other endodontic procedures are far less traumatic and damaging than extraction. A good example is the patient taking (or having taken) bisphosphonates. Root canal treatment is preferred in order to avoid the trauma of extraction.

However, one important consideration is that older patients are more likely to be taking more and stronger medications.[37] Caution is required to avoid interactions, particularly when prescribing additional medications.

DIAGNOSIS

Again, the same basic principles apply with older as with younger patients.

Diagnostic Procedure

It is important that a routine sequence be applied to diagnosis, particularly with elderly patients. The most important findings are from the subjective examination to determine symptoms and history. Careful questioning and allowing sufficient time for the older patient to recall and answer often yields valuable information.

Chief Complaint

The patient must be allowed to express the problem(s) in his or her own words. Not only will this divulge symptoms, but it also provides an opportunity to determine the patient's dental knowledge and ability to communicate. This ability may be impaired because of problems with sight, hearing, or mental status.

Medical History

The prudent diagnostician not only discusses positive responses marked on the medical history form but repeats important items that may not have been marked or were overlooked by the patient. Systemic conditions, medications, and related considerations should be discussed in-depth. It is appropriate at this time to explain to the patient how medical conditions might affect diagnosis, treatment planning, treatment, and outcomes.

Dental History

In general, elderly patients have a lot of history to review and recall. Important dental occurrences may be only a dim memory and these will require prompting by the examiner. Examples include a history of traumatic injury, fractures, caries, or pain and swelling.

Subjective Findings

Subjective findings include information obtained by questioning the patient's description of current signs and symptoms. Many older patients are stoic, do not readily express adverse symptoms, and may consider them to be minor relative to other systemic problems or pains. A careful, concerned discussion about these seemingly minor problems also helps establish rapport and confidence.

Overall, symptoms of pulpitis do not seem to be as acute in the older patient. One reason may be that there is a reduced pulp volume and a decrease in sensory nerves,[38] particularly in dentin.

The *absence* of significant signs and symptoms is also very common, more so than the *presence*. Of course, the absence does not indicate the lack of significant pathosis; most irreversible pulpal and apical pathoses are asymptomatic at any age. Thus when pathosis is suspected, objective tests are required regardless of whether significant signs and symptoms are present.

Objective Tests

Objective findings are primarily related to pulpal and periapical tests. Oral examination and transillumination are also commonly required.

1. *Pulp testing:* although similar in older and younger patients, there are some differences. The pulp becomes less responsive to stimuli with age (Figure 23-5). Thus testing in older patients should be done slowly and carefully, with the use of different stimuli. It is common for a tooth with a vital pulp to be nonresponsive to one form of testing (e.g., cold) but respond to another stimulus (e.g., electric). These results must be correlated with other tests and findings, as well as with radiographs.

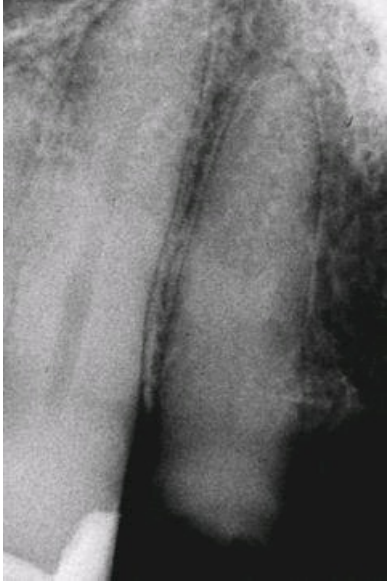

Figure 23-5 Calcific metamorphosis. Although there usually is vital pulp tissue, the teeth in older adults often do not respond to pulp testing because of decreased nerve supply and an increase in insulating dentin.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

There is a question of whether electric pulp tests should be used in patients with pacemakers.[39] Although it is unlikely that these tests could cause a pacemaker to malfunction, other tests can be used safely to give information on pulp status. It is recommended that electrical tests *not* be used when there is a pacemaker.

A test cavity is often indicated but may not be as useful in the older patient because of reduced dentin innervation. A false-negative (no response/vital pulp) response is not unusual, even with a test cavity.

2. *Periapical testing:* percussion (biting and tapping) and palpation tests indicate periapical inflammation but are not particularly useful unless the patient reports significant pain. These are most useful to confirm that such symptoms are indeed from a particular tooth and to determine the severity of response.

Radiographic Findings

Current, good quality periapical films are always necessary, and the same principles apply as in the younger patient. The techniques of making radiographs are similar but with some differences. Bony growths, such as tori and muscle attachments (frena), may affect film positioning. Also, the older patient may have difficulty in placing the film, thus holders should be used. Generally, a parallel film is preferred for diagnosis with occasional supplementation of mesially or distally angled cone positioning or a Panelipse or occlusal view. Often, bitewing projections are helpful in showing chamber size and location and relative depths of caries and restorations.

Apically, there may be some differences in the older patient. The incidence of nonendodontic pathosis of the jaws tends to increase with age; careful determination of pulp status is even more important in these situations when the nature of the pathosis is uncertain. If the pulp is vital, a lesion in the apical region is not endodontic.

Radiographs are studied for pulp size and for root and pulp anatomy. Again, pulps tend to be smaller and may disappear radiographically (Figure 23-6). It is important to note that nonvisualization of a pulp space does not mean that a pulp is not present. In fact, it has been demonstrated that there is always a pulp space,[40] even when it is not visible radiographically. Apical root and canal anatomy tends to be somewhat different in elderly patients because of continued cementum formation.[41,42] This may be further complicated by apical root resorption from pathosis.[43]

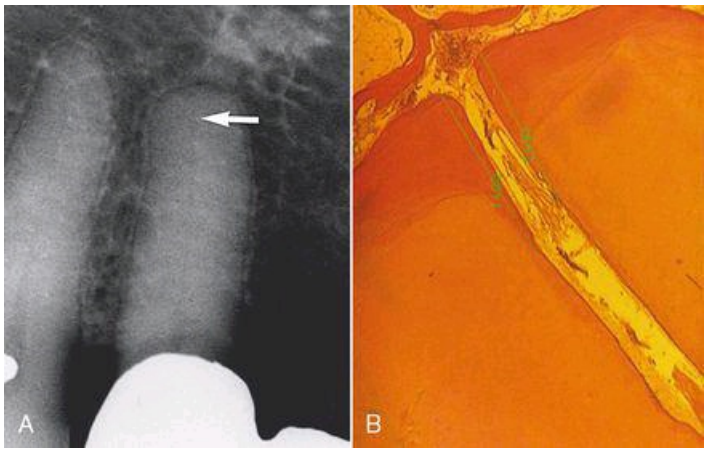

Figure 23-6 A, Although the pulp is barely visible apically *(arrow)*, a corresponding histologic section of this region **(B)** shows a sizable pulp space containing vital tissue.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

DIFFERENTIAL DIAGNOSIS

Differential diagnosis is the ultimate determination of whether there is an endodontic or another type of pathosis and if endodontic, the specific details of the pulp or periapical lesion.

Endodontic Pathosis

Signs and symptoms, test results, and other observations in the older patient should follow a fairly consistent pattern. Other complications may be mind-altering medications, as well as occasional perceptive problems in elderly patients. Vague symptoms that cannot be localized or do not follow an identifiable pattern probably are not endodontic in origin. Other pathosis or nonpathologic entities must then be considered, including psychosomatic conditions.

Other Pathoses

Other pathoses include numerous entities, and many are more common in elderly patients. The lesion that commonly mimics endodontic pathosis is the periodontal lesion. Nonendodontic symptomatic disorders that may mimic endodontic pathosis include sinus infection, muscle spasm, headache, temporomandibular joint dysfunction, and neuritis and neuralgia. The incidence of these tends to increase somewhat with age, particularly in patients who have specific disorders, such as arthritis, that may affect the joints.

Differentiating periodontal from endodontic pathosis is a common problem because of the increasing incidence of both endodontic and periodontal disease. Usually the underlying problem is either periodontic or endodontic, with few true combined lesions (see Chapter 6). Radiographic changes, swellings, sinus tracts, and deep probing defects may be either endodontic or periodontic in origin. Although all findings should be considered, the ultimate indicator is pulp testing. If the pulp is indeed vital, the problem is periodontal. If the pulp is necrotic, the likelihood is that the problem is endodontic. Pulp tests are critical, thus a test cavity may be helpful.

TREATMENT PLANNING AND CASE SELECTION

After differential diagnosis, a definitive treatment plan is determined’usually root canal treatment but additional procedures may be included. Everything should be considered (restorability, periodontal status, and overall treatment plan), and this would be the time to consider referral of the patient to an endodontist if the situation is deemed too complex.

Procedure

Whatever the treatment, procedures are generally more technically complex in older patients. Extensive restorations, a history of multiple carious insults, periodontal involvement, decreasing pulp size, tipping (Figure 23-7), and rotation are all factors. An original treatment plan often has to be modified during the procedure because of unexpected findings. For example, root canal treatment may be initiated only to find that a canal cannot be located or negotiated. Periradicular surgery then becomes a necessity (Figure 23-8). These possibilities should be explained to the patient, preferably before treatment is begun.

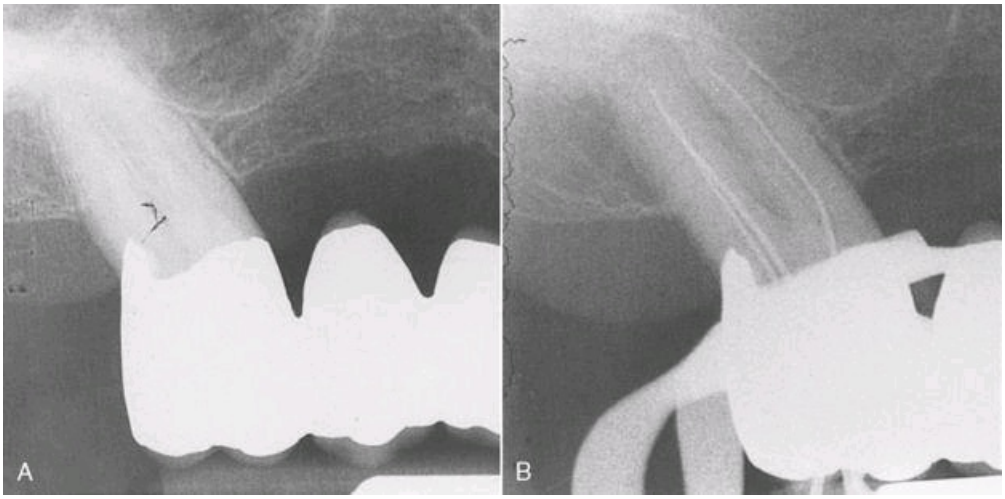

Figure 23-7 A, Castings are frequently misoriented because of tipping and rotation. **B,** Access is more challenging. Observation before and caution during access are critical to avoid perforation.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

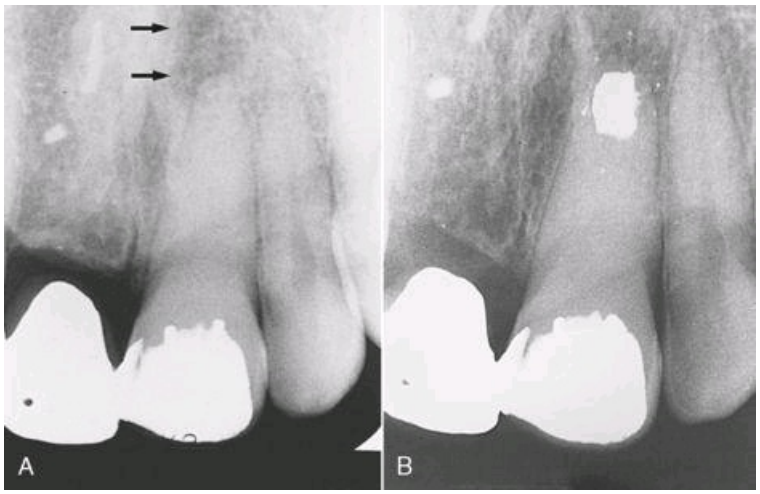

Figure 23-8 A, Calcific metamorphosis and apical pathosis *(arrows)* after trauma. In this tooth, conventional access would be difficult and would jeopardize retention of the bridge. **B,** A surgical approach was used with the hope of sealing in irritants apically.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

Prognosis

Although periradicular tissues will heal as readily in elderly as in young patients,[44,45] there are many factors that reduce the rate of success. The same factors that complicate treatment also may compromise ultimate success. An extensively restored tooth is more prone to coronal leakage. Canals that cannot be negotiated to length may contain persistent irritants. Tipped or rotated teeth restored with castings that are misaligned are more difficult to access and therefore more difficult to clean, shape, and obturate.

Each patient should have a pretreatment and posttreatment assessment of prognosis. The pretreatment assessment is the anticipated outcome, and the posttreatment assessment reviews what should happen according to modifiers determined during treatment. Many teeth are severely compromised and would be a problem to retain (Figure 23-9). Extraction is often the preferred approach. A study[46] on the outcome of not replacing a missing tooth showed that the consequences generally were not significant. Thus when extraction is discussed as an option, the patient is informed that “filling the space” may be unnecessary.

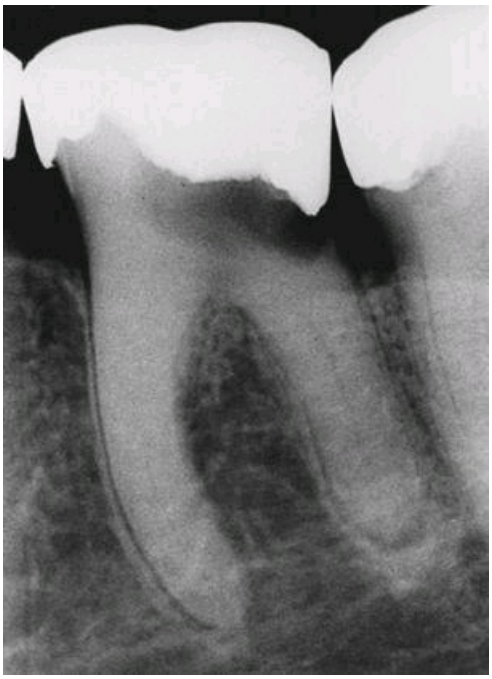

Figure 23-9 Recurrent caries have created challenges in retaining this molar. Crown lengthening would be necessary for both restoration and isolation during root canal treatment. Crown lengthening may infringe on the furcation. Canals would be difficult to locate and negotiate. The tooth probably should be extracted.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

Number of Appointments

Whether to treat in a single visit or in multiple visits has always been a subject of debate and conjecture. Studies have shown that there are no advantages overall to multiple appointments relating to posttreatment pain or prognosis. However, with pulp necrosis, treatment in multiple appointments and the use of calcium hydroxide as an intracanal medicament may speed healing[47] and possibly promote better long-term outcomes.[48,49]

Single appointment procedures are beneficial in elderly patients. Longer appointments may be less of a problem than several shorter appointments if the patient must rely on others for transportation or requires assistance to reach the office or to get in and out of the chair. At times, the elderly patient may require special positioning of the chair, support of the back or neck or limbs, or other such considerations (Figure 23-10). Conversely, these problems may require shorter, multiple appointments.

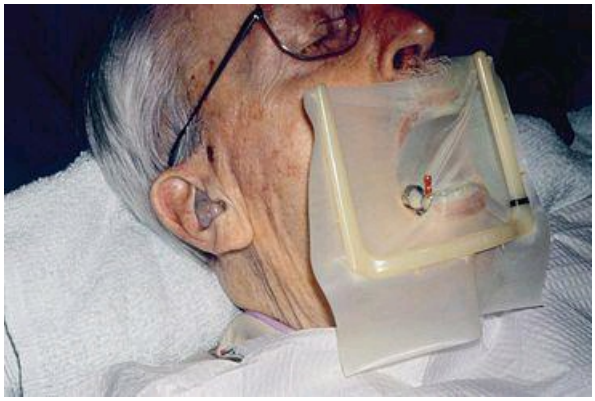

Figure 23-10 Elderly patients often have postural problems. This patient is made comfortable with a rolled-up towel, forming a brace under his neck.

Additional Considerations

In treatment planning for elderly patients, the tendency is to plan according to anticipated longevity.[50] It is natural to assume that procedures need not be as permanent because the patient may not live for very long. The concept that treatment should not outlast the patient is not accepted by most elderly patients, who desire health care equivalent to that rendered to younger patients. Esthetic and functional concerns may be no different.

ROOT CANAL TREATMENT

Treatment Considerations

Time Required

On average, longer appointments are necessary to accomplish the same procedures in elderly patients for the reasons discussed earlier.

Anesthesia

Primary Injections

The need for anesthesia is somewhat less in the older patient. It is necessary for vital pulps but is often unnecessary for pulp necrosis, obturation appointments, and retreatments. Older patients tend to be less sensitive and are more likely to prefer procedures without anesthetic. Also, they tend to be less anxious and therefore have a higher threshold of pain. Although there are no differences in effectiveness of anesthetic solutions, various systemic problems or medications may preclude the use of vasoconstrictors.

Supplemental Injections

Intraosseous, periodontal ligament (PDL), and intrapulpal anesthesia are effective adjuncts if the primary anesthesia is not adequate. Again, certain cardiac conditions may preclude the use of epinephrine, particularly with the intraosseous and PDL techniques. Duration of anesthesia is considerably decreased without a vasoconstrictor, and reinjection during the procedure may be required.

Procedures

Isolation

Isolation is often difficult because of subgingival caries or defective restorations. However, placement of a rubber dam is imperative and often requires ingenuity (see Chapter 14). A fluid-tight isolation reduces salivary contamination of the pulp space and prevents introduction of irrigants into the mouth. If there are questions about the integrity of a restoration, it should be removed before rubber dam placement. Also, temporary crowns, orthodontic bands, or temporary restorations should be removed in their entirety. Improved visibility and good isolation are more predictable.

Access Preparation

Achieving good access to enable locating and then negotiating canal orifices is challenging in older teeth because of internal anatomy (Figure 23-11). Radiographs are helpful. A slightly larger rather than a too small access opening is preferred, particularly through large restorations such as crowns. Magnification is also helpful, either from a microscope or from other visual aids.

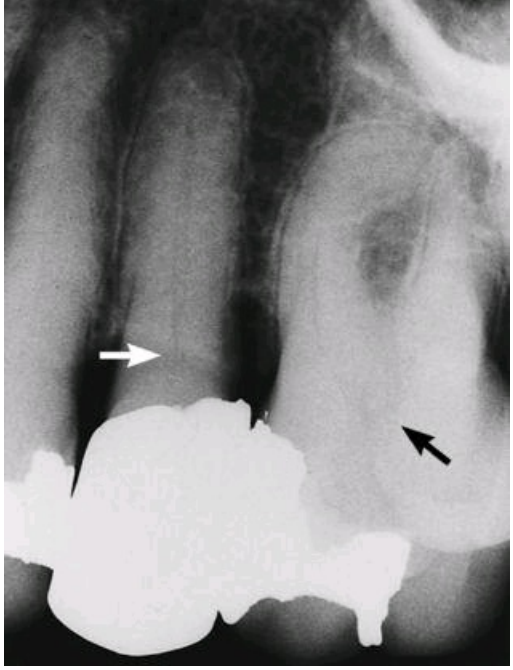

Figure 23-11 Age, caries, and restorations have resulted in small chambers *(arrows)*. Either would be a challenge to access, and referral should be considered.

A supraerupted tooth, as a result of caries or restoration, has a short clinical crown, requiring a less deep access preparation. The distance from the reference cusp to the chamber roof should be measured on the bur radiographically. A very small or nonvisible chamber may be an indication for beginning the access without the rubber dam; this aids in staying in the long axis of the tooth (Figure 23-12). Once the canal is located, the rubber dam is immediately placed before working length radiographs are made.

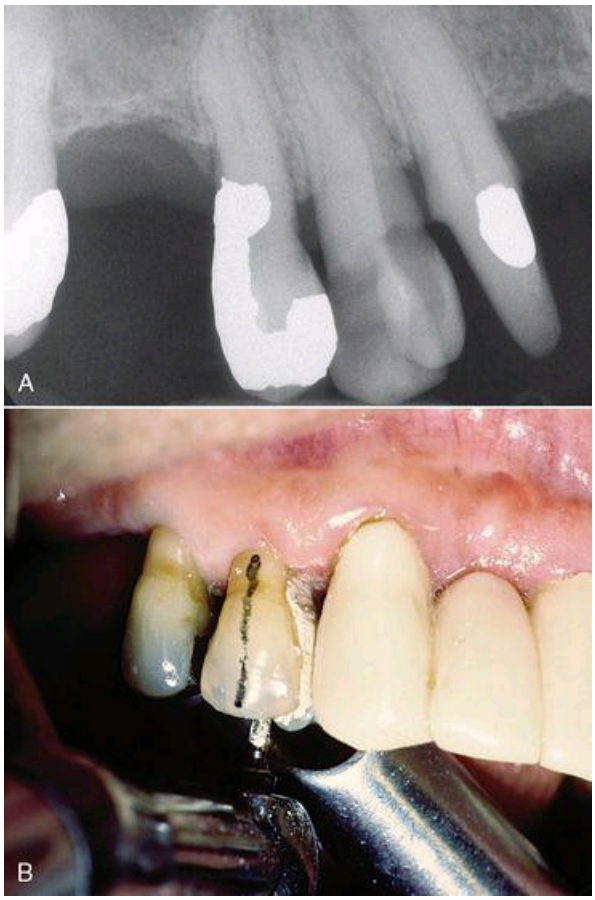

Figure 23-12 A, The first premolar is tilted and has a “receded” pulp chamber. **B,** Aids in orientation during access. The preparation is initiated without the rubber dam in place. A pencil mark is placed on the crown to guide the bur in the long axis of the root.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

Locating canal orifices is often fatiguing and frustrating to both clinician and patient. Although a reasonable period of time

should be allocated, there is a limit. It may be best to stop and have the patient return for another appointment. Often, the canals are readily located at a subsequent visit. This also is a time to consider a referral because another procedure, such as surgery, may be indicated.

Working Length

There are some differences in working length in the older patient.[51] Because the apical foramen varies more widely (Figure 23-13) than in the younger tooth and because of decreased diameter of the canal apically, it is more difficult to determine the preferred length.[42] In teeth of any age, materials and instruments are best confined to the canal space. One to 2 mm short of the radiographic apex is the preferred working and obturation length[52]; this should be decreased if an apical stop is not detected. Electronic apex locators are also useful, particularly when there is difficulty obtaining adequate working length radiographs.[53]

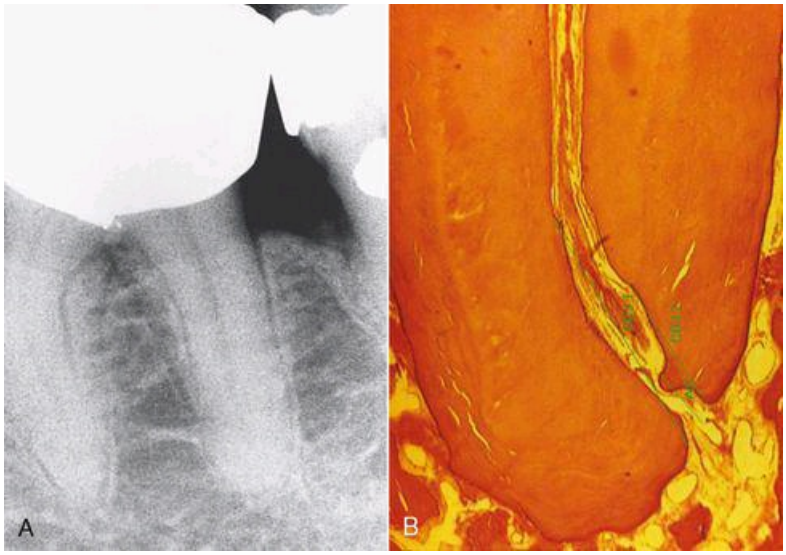

Figure 23-13 Variability in apical foramen location. **A,** The foramen is not visible radiographically. **B,** Histologically, the distal root shows the foramen to be well short of the apex.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

Cleaning and Shaping

A common challenge is a much smaller canal that requires more time and effort to enlarge. A very small canal may be more easily negotiated and initially prepared with a lubricant such as glycerin. This may be used through two or three smaller sizes of files to facilitate enlarging, as well as to reduce the risk of binding and separation. The same principles of débridement and adequate shaping are followed.

Intracanal Medicaments

Intracanal medicaments are contraindicated, with the exception of calcium hydroxide. This chemical is antimicrobial, inhibits bacterial growth between appointments, and may reduce periradicular inflammation.[54] It is indicated if the pulp is necrotic, and the canal preparation is essentially complete.

Obturation

There is no demonstrated preferred approach, although cold-lateral and warm-vertical gutta-percha obturations are the most commonly used and the best documented.

IMPACT OF RESTORATION

Generally the larger and deeper the restoration, the more complicated the root canal treatment. The old tooth is more likely to have a full crown. There are two concerns when there is a crown: (1) potential damage to retention or components of the crown and (2) blockage of access and poor internal visibility.

The porcelain-fused-to-metal (PFM) crown is more common than a full metal crown and creates additional problems. Porcelain may fracture or craze. This problem is minimized by using burs specifically designed to prepare through porcelain,[55] combined with slow cutting and copious use of water spray. Occlusal access is wide (Figure 23-14). Metal should not be removed after the chamber is opened to prevent metal shavings from entering and blocking canals. Access through a PFM or a gold crown (either anterior or posterior) that is to be retained is best permanently repaired with amalgam. Anterior nonmetallic crowns may be repaired with composite.

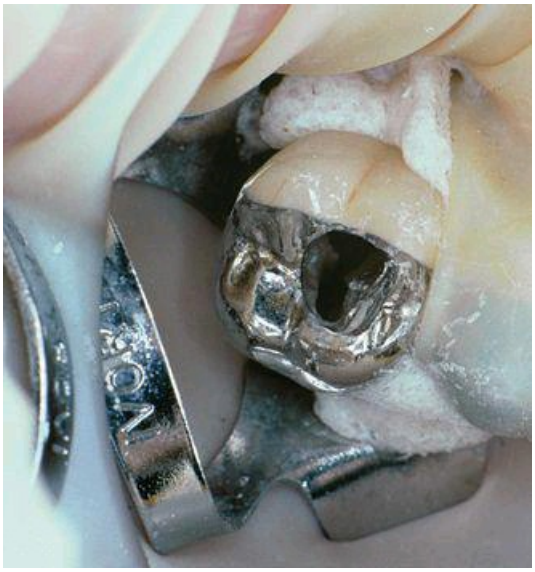

Figure 23-14 Access through a porcelain-fused-to-metal crown. The outline is large for visibility. Also, the preparation does not extend to the porcelain to avoid fracture to the porcelain.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

RETREATMENT

Factors that lead to failure tend to increase with age; thus retreatment is more common in older patients. Retreatment at any age is often complicated and should be approached with caution; these patients should be considered for referral. Retreatment procedures and outcomes are similar in both older and younger teeth (see Chapter 19).

ENDODONTIC SURGERY

Considerations and indications for surgery are similar in elderly and younger patients. These include incision for drainage, periradicular procedures, corrective surgery, root removal, and intentional replantation. Overall, the incidence of most of these will increase with age. Small nonnegotiable canals, resorptions, and canal blockages occur more often with age. Perforation during access or preparation, ledging, and instrument separation are related to restorative and anatomic problems.

Medical Considerations

Medical considerations may require consultation and are of concern but generally do not contraindicate a surgical approach.[56] This is particularly true when extraction is the alternative; surgery is often less traumatic.[57]

Excessive hemorrhage during or after surgery is of concern; many elderly patients are receiving anticoagulant therapy. Interestingly, recent studies examined bleeding patterns in oral surgery patients taking low-dose aspirin[58] and prescribed anticoagulants.[59,60] The findings were that anticoagulant therapy should preferably not be altered and that hemorrhage was controllable by local hemostatic agents.

Biologic and Anatomic Factors

Bony and soft tissues are similar and respond the same in older and younger patients. There may be somewhat less thickness of overlying soft tissue; however, alveolar mucosa and gingiva seem to be structurally similar. Anatomic structures, such as the sinuses, floor of the nose, and location of neurovascular bundles, are essentially unchanged. Often, periodontal and endodontic surgery must be combined. Also, crown-to-root ratios may be compromised because of periodontal disease or root resorption.

Healing After Surgery

Hard and soft tissues will heal as predictably, although somewhat more slowly.[61-63] Postsurgical instructions should be given both verbally and in writing to minimize complications. If the patient has cognitive problems, instructions are repeated to the person accompanying the patient. Even very elderly patients will have good healing, provided they follow posttreatment protocols. Ice and pressure (in particular) applied over the surgical area reduces bleeding and edema and minimizes swelling. Overall, older patients experience no more significant adverse affects from surgery than do younger patients. Outcomes depend more on oral hygiene than on age, as has been shown in periodontal surgery patients.[64]

One problem that seems to be more prevalent in older patients is ecchymosis after surgery. This is hemorrhage that often spreads widely through underlying tissue and commonly presents as discoloration (Figure 23-15). Patients are informed that this may occur and should not be a concern. Normal color may take 1 to 2 weeks or longer to return. In addition, the discoloration may go through different color phases (purple, red, yellow, green) before disappearing.

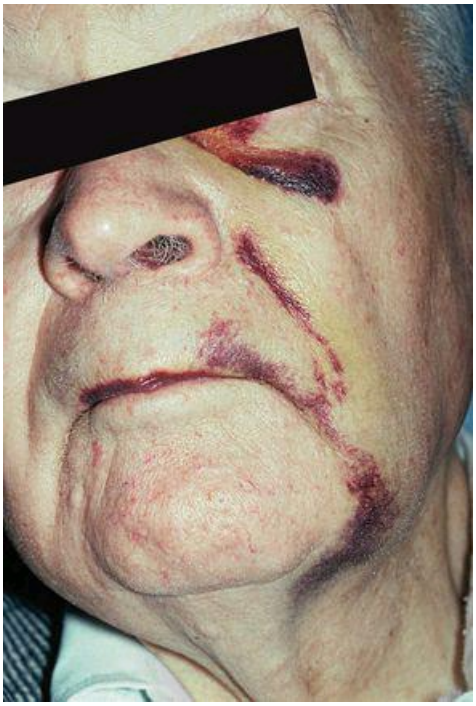

Figure 23-15 Postsurgical ecchymosis. Root-end surgery of a maxillary lateral incisor resulted in widespread migration of hemorrhage into the tissues, with resultant discoloration. This is not an uncommon occurrence in elderly patients. No treatment is indicated, and the problem resolves in 1 to 2 weeks.

BLEACHING

Both internal and external tooth discoloration occurs in older patients.[18] Internal discoloration is related to dental (restorative or endodontic) procedures or to an increase in dentin formation with a loss of translucency. External discoloration occurs from stains and from restorative procedures as well (see Figure 23-1). Overall, teeth tend to discolor with time and with age. Both external and internal bleaching procedures can be successful in these patients.

External Stains

External stains are on or close to the enamel surface and are best managed by conventional night guard/oxidizing gel techniques.

Internal Stains

Stains that are most amenable to internal bleaching are related to discoloration after root canal treatment or pulp necrosis. The considerations related to diagnosis, etiology, treatment planning, and prognosis for successful short- and long-term internal bleaching are detailed in Chapter 22. Often, discolorations in these teeth can be significantly resolved, much to the satisfaction of the older patient.

Teeth that are discolored because of increased amounts of dentin formation and loss of translucency generally should not be considered for internal bleaching because these would require root canal treatment first. External bleaching may lighten the teeth somewhat.

RESTORATIVE CONSIDERATIONS

Overdenture Abutments

Overdenture abutments involve the reduction of a root to permit the resting of a removable partial or complete denture on a restored or natural root face.

An important consideration is that although a pulp space may not be evident on a radiograph (Figure 23-16), small components of the pulp chamber usually extend into the crown.[40] Reduction would create a clinically undetectable exposure. If untreated, this will result in pulp necrosis. Teeth to be reduced for overdenture abutments should have root canal treatment followed by an appropriate restoration to seal the access. Amalgam, composite resin, and glass ionomer are adequate materials.[65] Reduction of the crown and placement of a sealing material, without root canal treatment, is another approach (Figure 23-17).

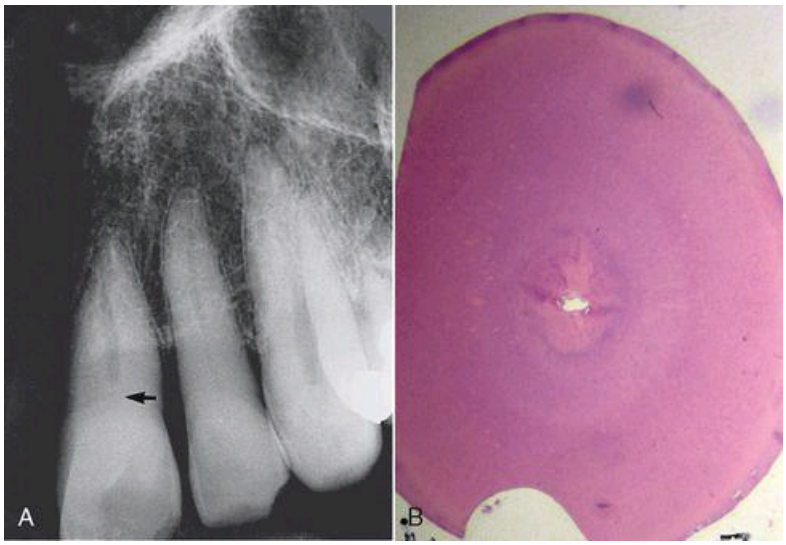

Figure 23-16 A, This central incisor was cross-sectioned at the level of the arrow, where no pulp space is visible. **B,** Histologically, a small pulp space is apparent. Reducing such a tooth for overdentures would result in pulp exposure.

(From Walton RE: Dent Clin North Am 41:795, 1997.)

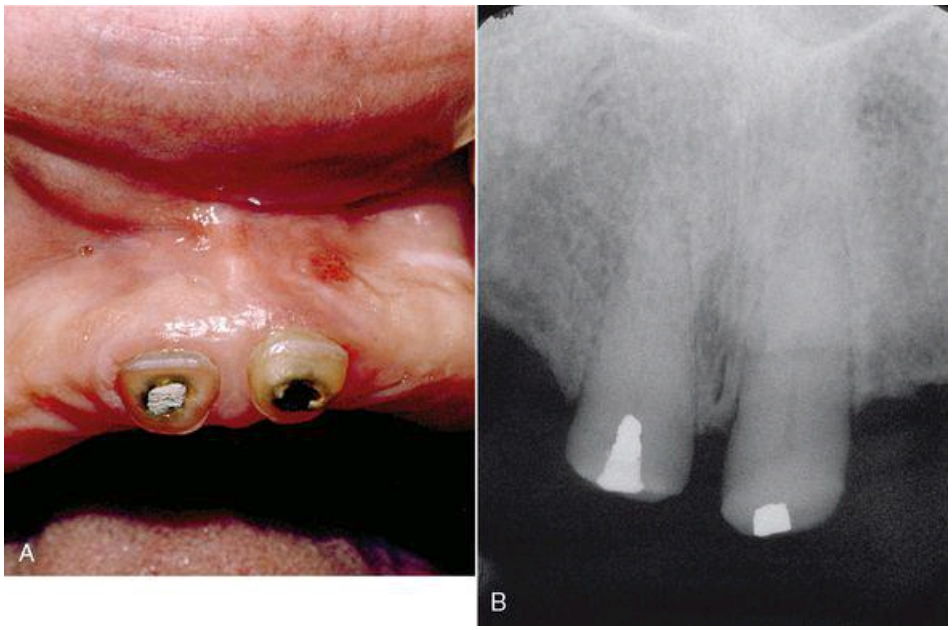

Figure 23-17 A, Overdenture abutments must be restored to **(B)** seal a pulp space, which is always present. Root canal treatment is not necessary in some situations.

Coronal Seal

In the elderly (as in the younger) patient, the coronal surface must be sealed from oral fluids forever to prevent failure. A concern for elderly patients is that the dentist will be less careful with the design and placement of a restoration or select less durable materials. In addition, the older patient is likely to be more susceptible to recurrent caries or abrasion, particularly on cervical root surfaces. These lesions do not have to penetrate as deeply to expose the obturating material. Subsequent to this exposure will be contamination of the obturating material by saliva and bacteria with resultant periapical pathosis. Again, this is probably the number one cause of treatment failure and reason for retreatment.

TRAUMA

Traumatic injuries occur in elderly patients more commonly with recreational activity and because of postural instability and loss of coordination. Generally, the older patient who experiences facial trauma will have different concerns and require some differing approaches than the younger patient.[66]

A major issue is that there may be cranial injuries that are masked by the obvious superficial facial trauma. Evidence of such injuries, as shown by in-office tests (see Chapter 10), would require an immediate hospital emergency room visit. Other concerns would be similar to those discussed earlier in this chapter: medical status, cognitive factors, and patient expectations. In conjunction with these considerations, the actual management of the hard and soft tissues would be similar to and have an expected outcome much like that of a younger patient.

Many of these elderly trauma patients may have initial injury management by a generalist and then be referred to an oral surgeon for facial injury assessment. Follow-up and long-term dentition care may then be best managed by the endodontist.

Chapter Review Questions available in Appendix B or on the DVD

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Pulpal Anatomy and Access Preparations

Lisa R. Wilcox

The illustrations in this appendix depict the size, shape, and location of the pulp space within each tooth, as well as the more common morphologic variations. Based on this knowledge of the shape of the pulp and its spatial relationship to the crown and root, the correct outline form for access preparation is presented from the occlusal, lingual, and proximal views. From these illustrations, the following features can be observed:

1. The *location* of access on posterior teeth relative to occlusal landmarks such as marginal ridges and cusp tips

2. The *size* and *appearance* of the access on anterior teeth as viewed from the incisal surface

3. The *approximate size* of the access opening

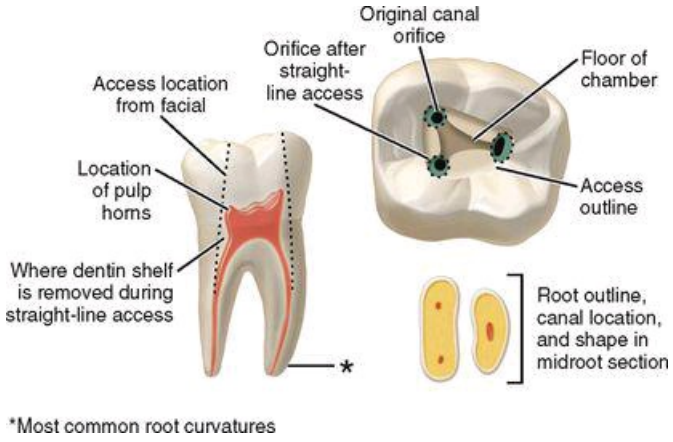
4. The *location of canal orifices* and their positions relative to occlusal landmarks and to each other

5. The *canal curvatures* and the location of the apical foramina

6. The *configuration of the chamber* and cervical portion of the canals after straight-line access preparation

7. The *root curvatures* that are most common

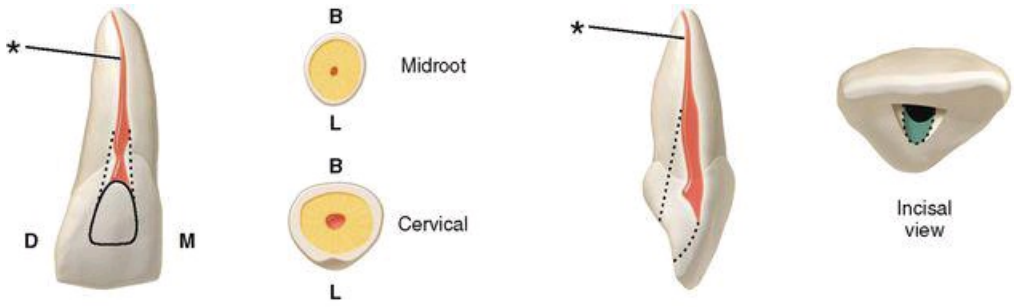
Each illustration gives the following information:



*Most common root curvatures

In addition, the percentages of the more common morphologic variations of the roots and canals are given. With many of the tooth groups, the percentages do not total 100%. The remaining percentage represents the less common variations not illustrated. Percentages are approximate to give general information, primarily to demonstrate relative occurrences. The more common root and canal curvatures are included. These are curvatures not readily identified on radiographs (i.e., toward the facial and lingual aspects).

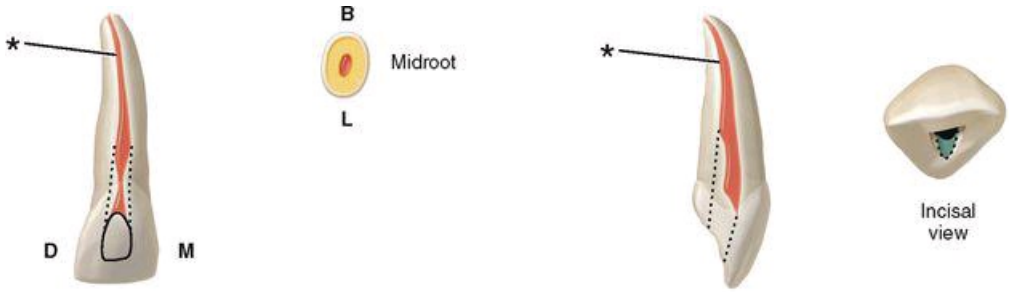
Maxillary Right Central Incisor



1 canal: 100%

*Most common root curvatures

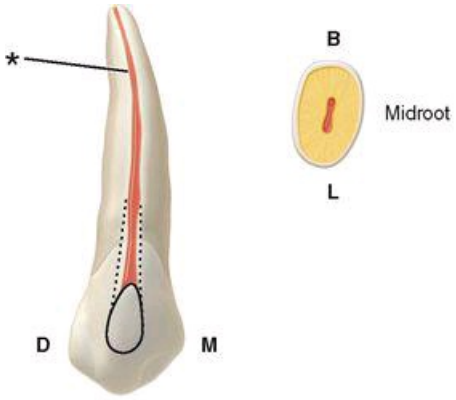
Maxillary Right Lateral Incisor



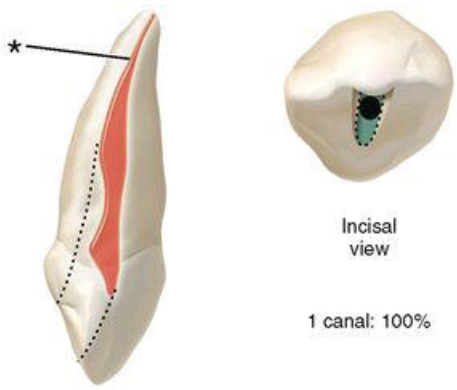
1 canal: 100%

*Most common root curvatures

Maxillary Right Canine

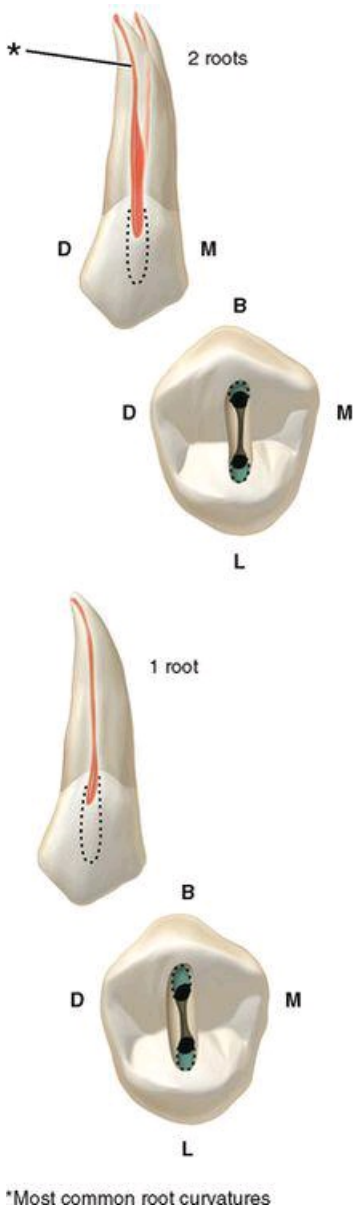


*Most common root curvatures

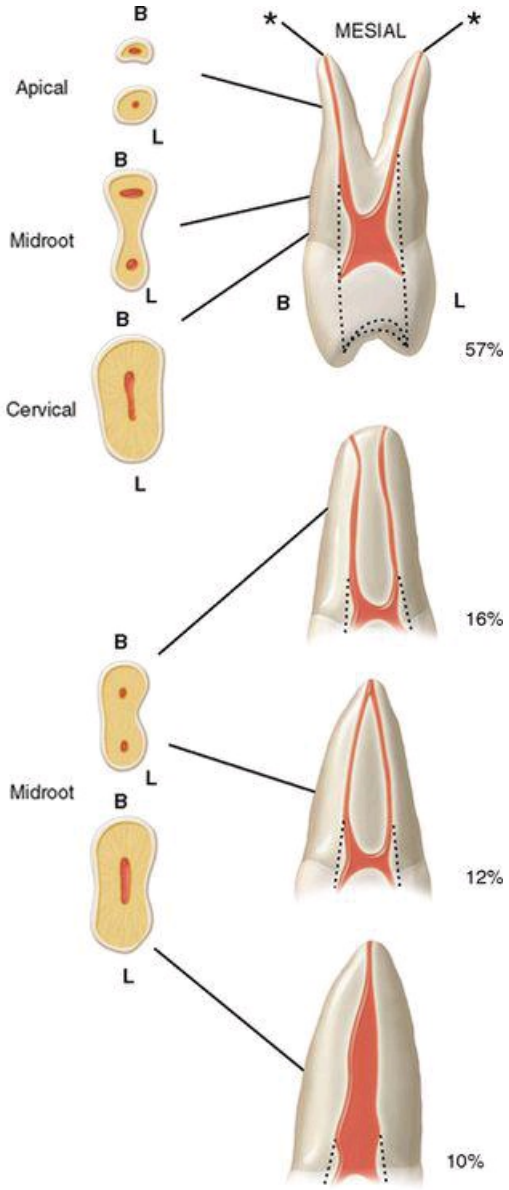


1 canal: 100%

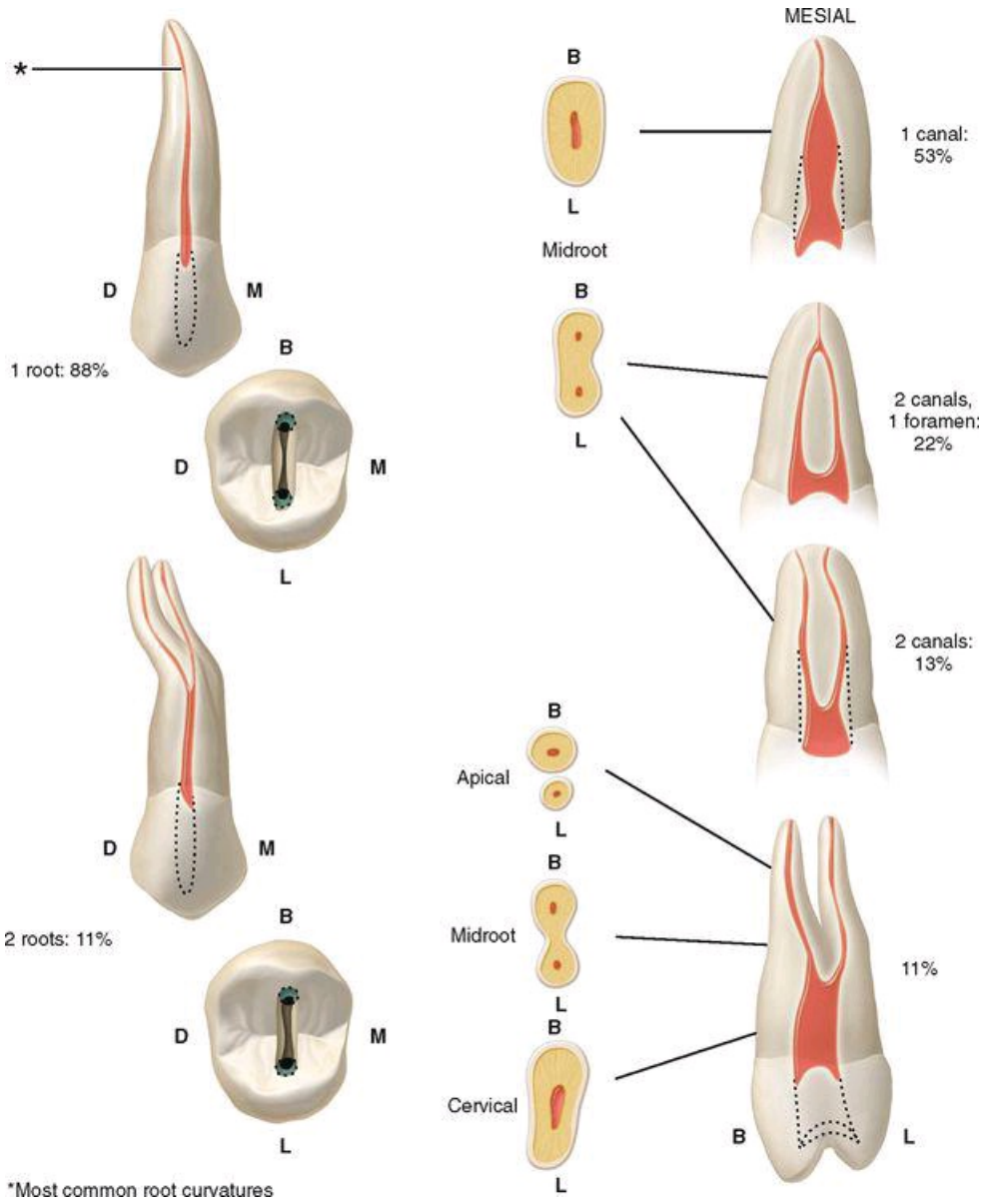
Maxillary Right First Premolar



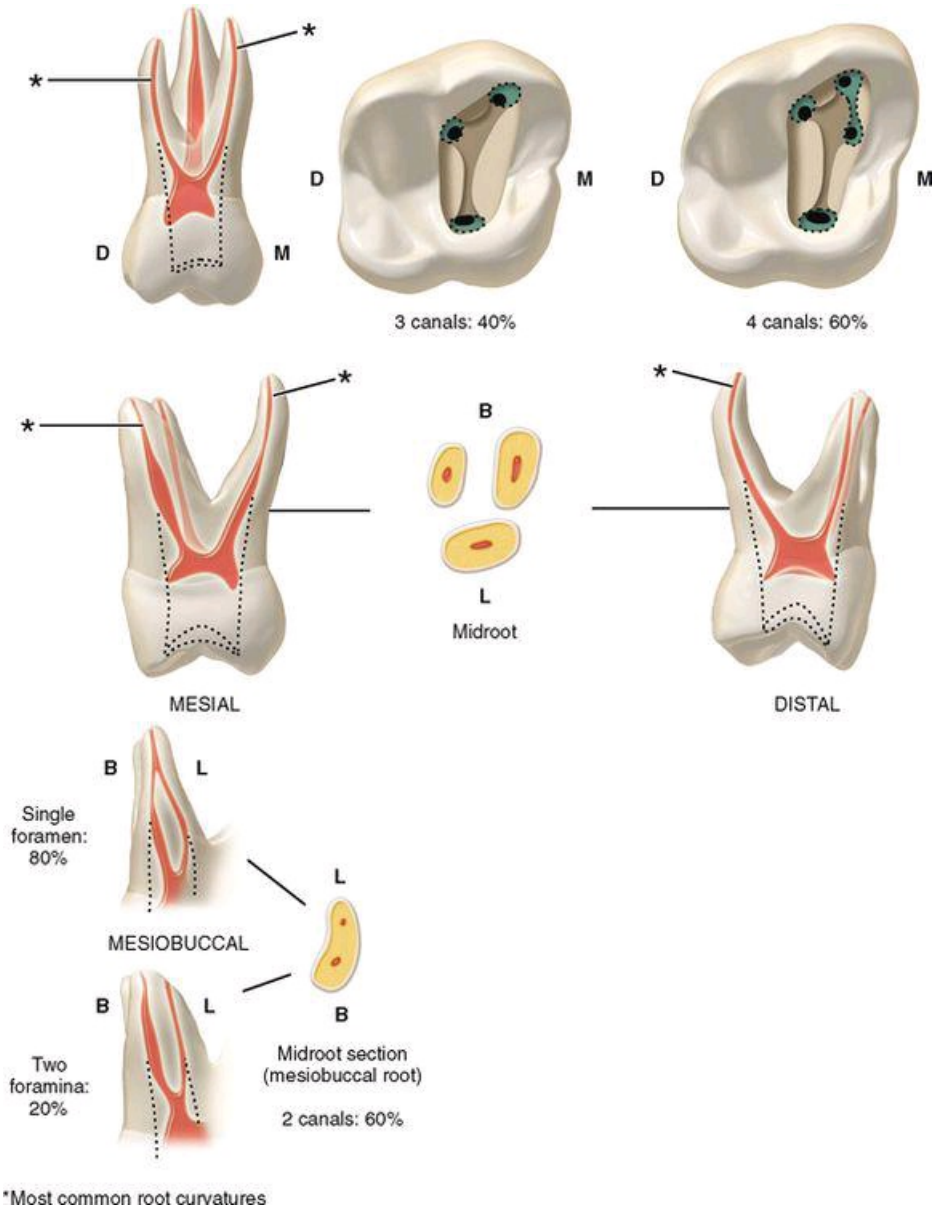
*Most common root curvatures



Maxillary Right Second Premolar

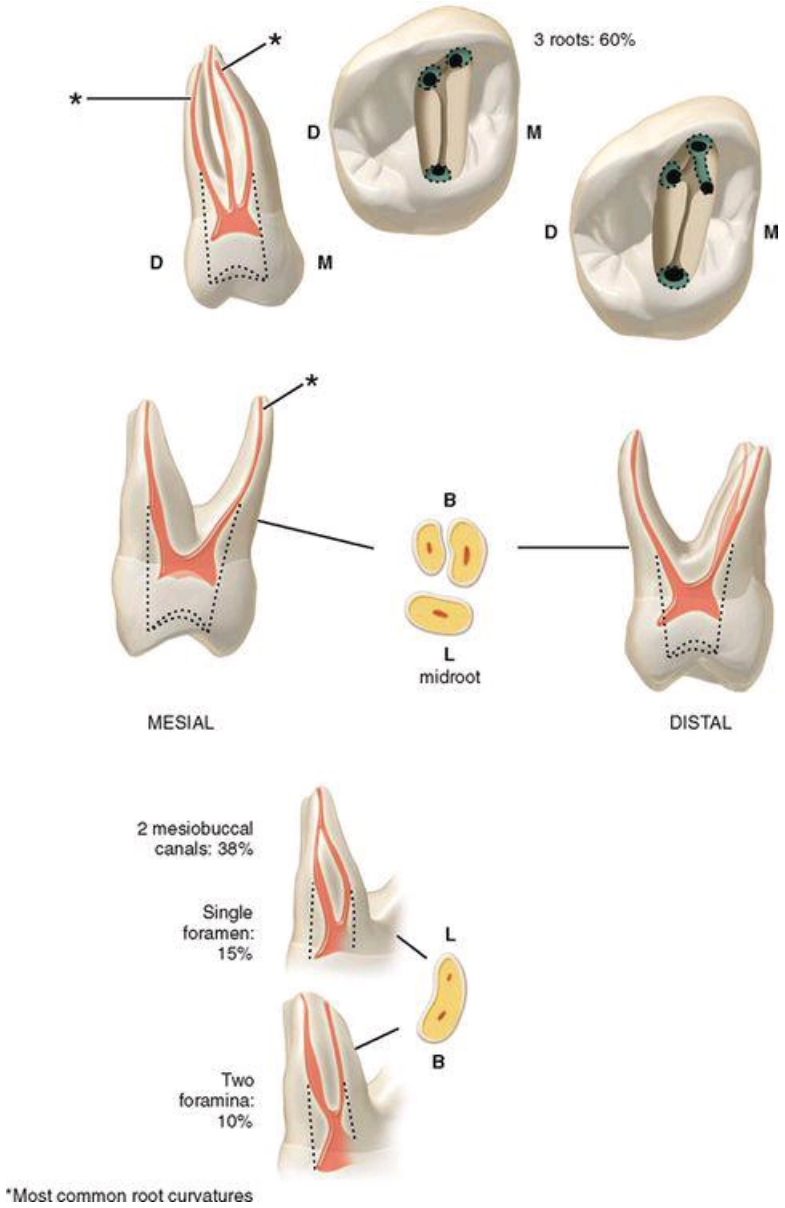


Maxillary Right First Molar

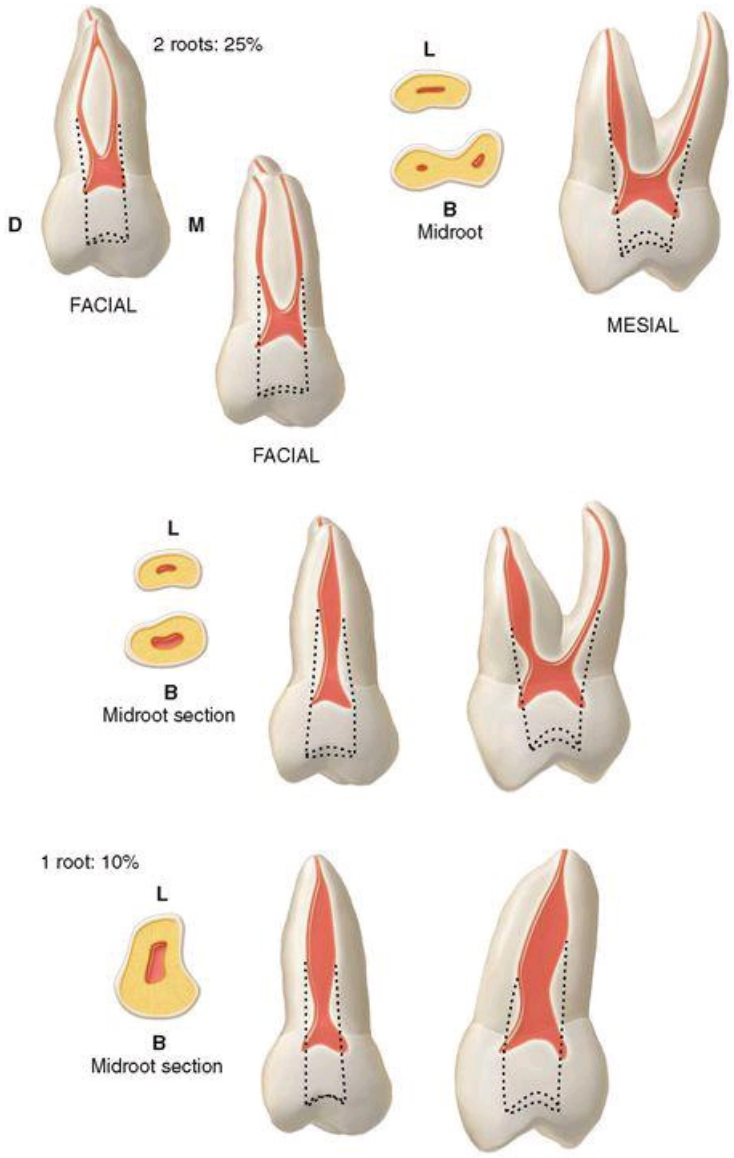


*Most common root curvatures

Maxillary Right Second Molar

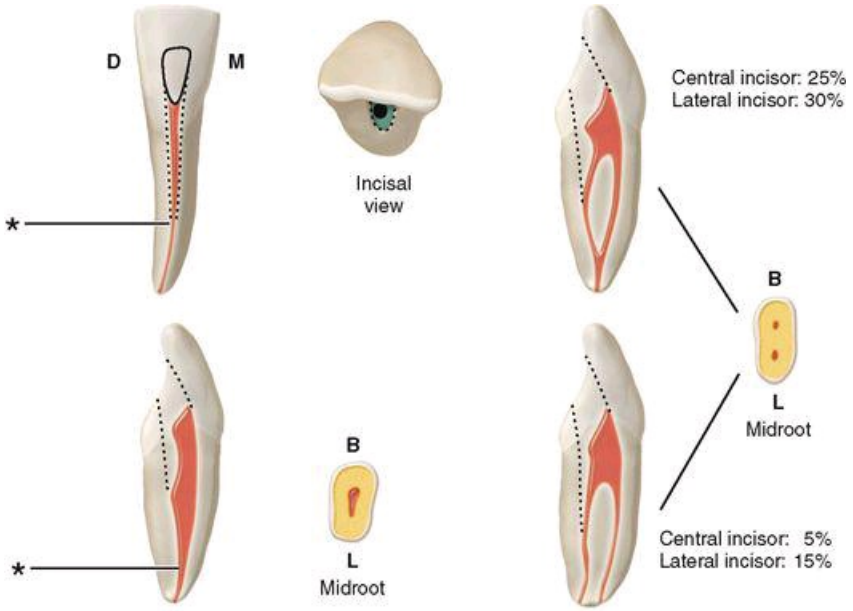


Maxillary Right Second Molar



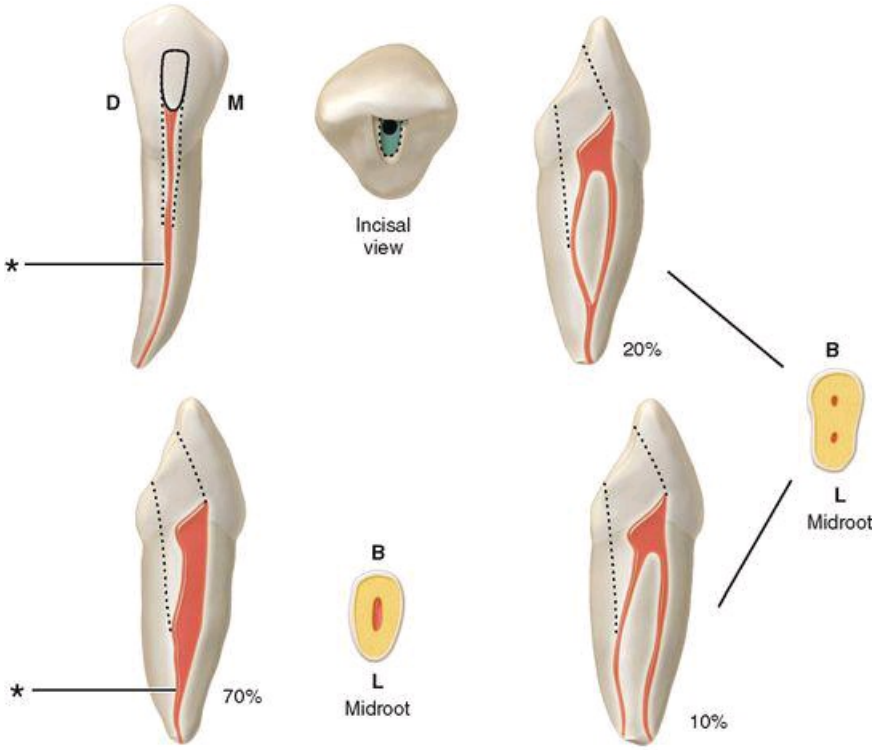
*Most common root curvatures

Mandibular Right Central and Lateral Incisor



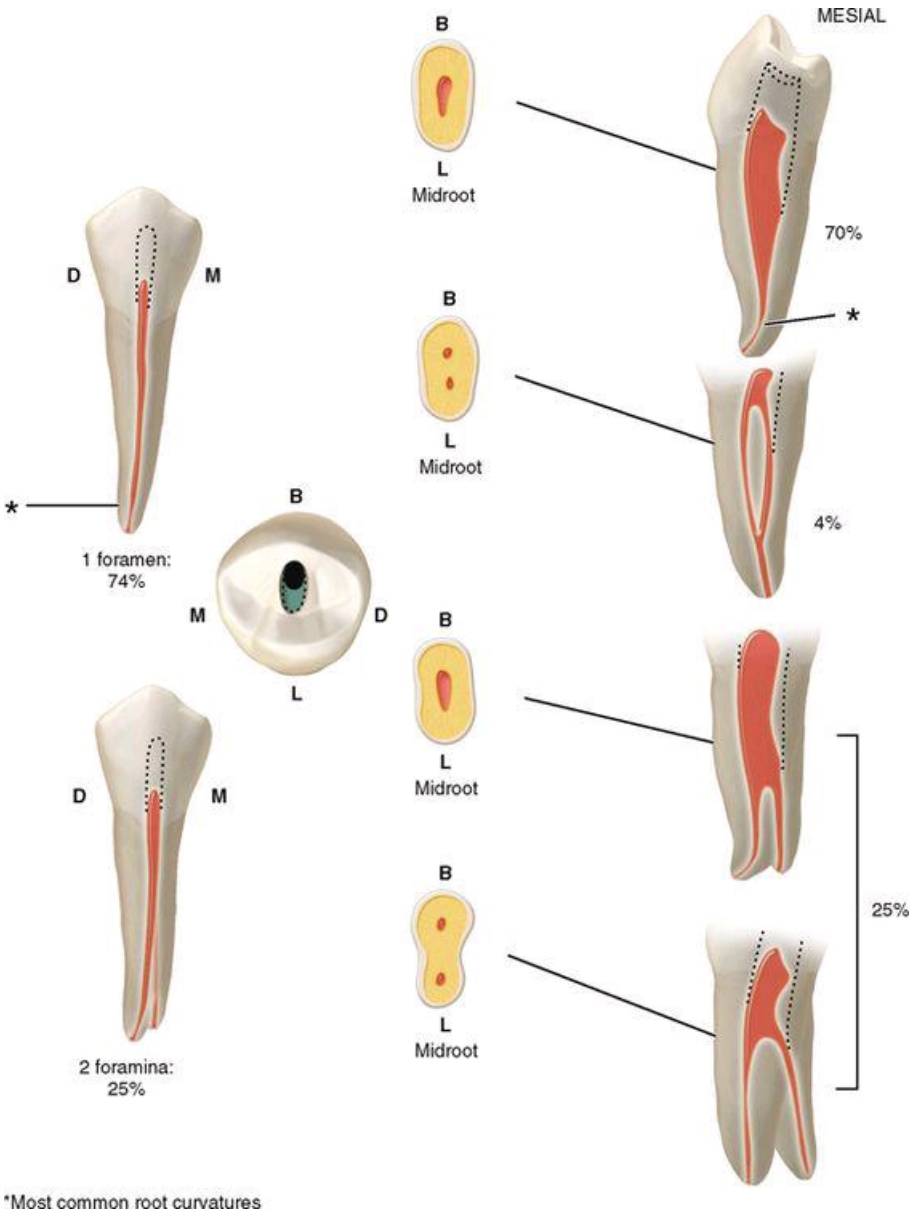
*Most common root curvatures

Mandibular Right Canine



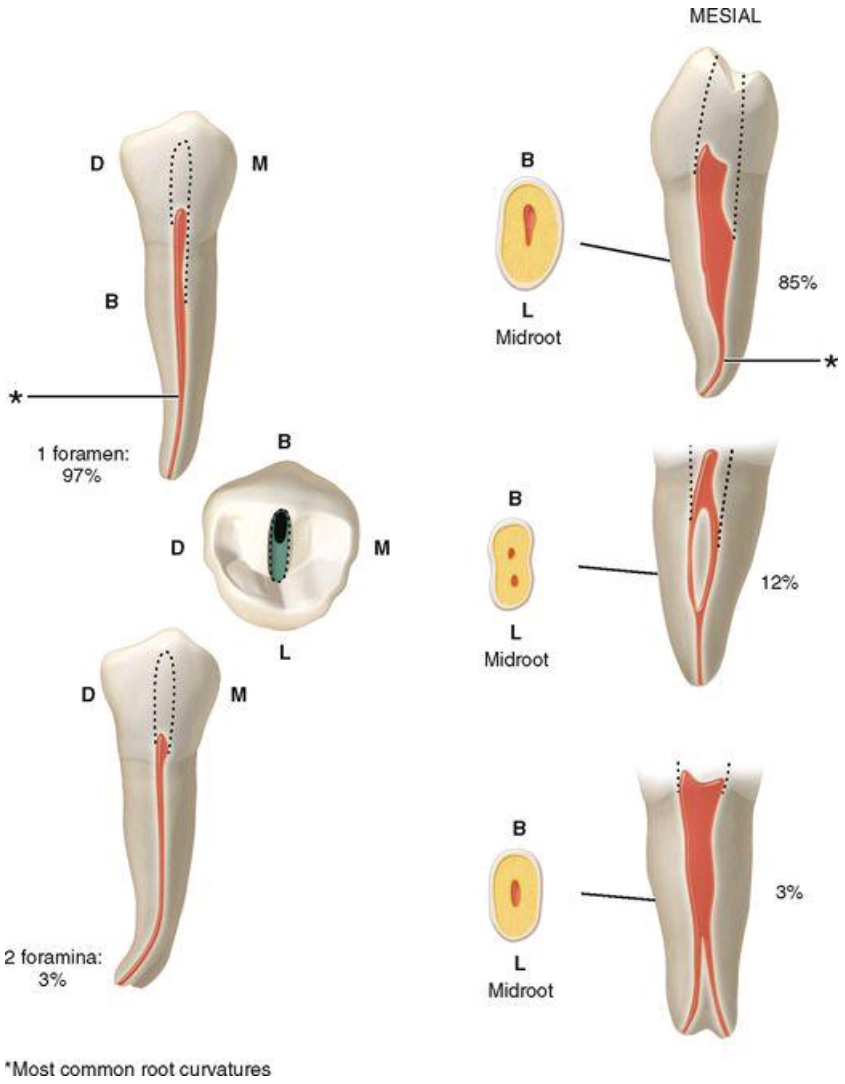
*Most common root curvatures

Mandibular Right First Premolar



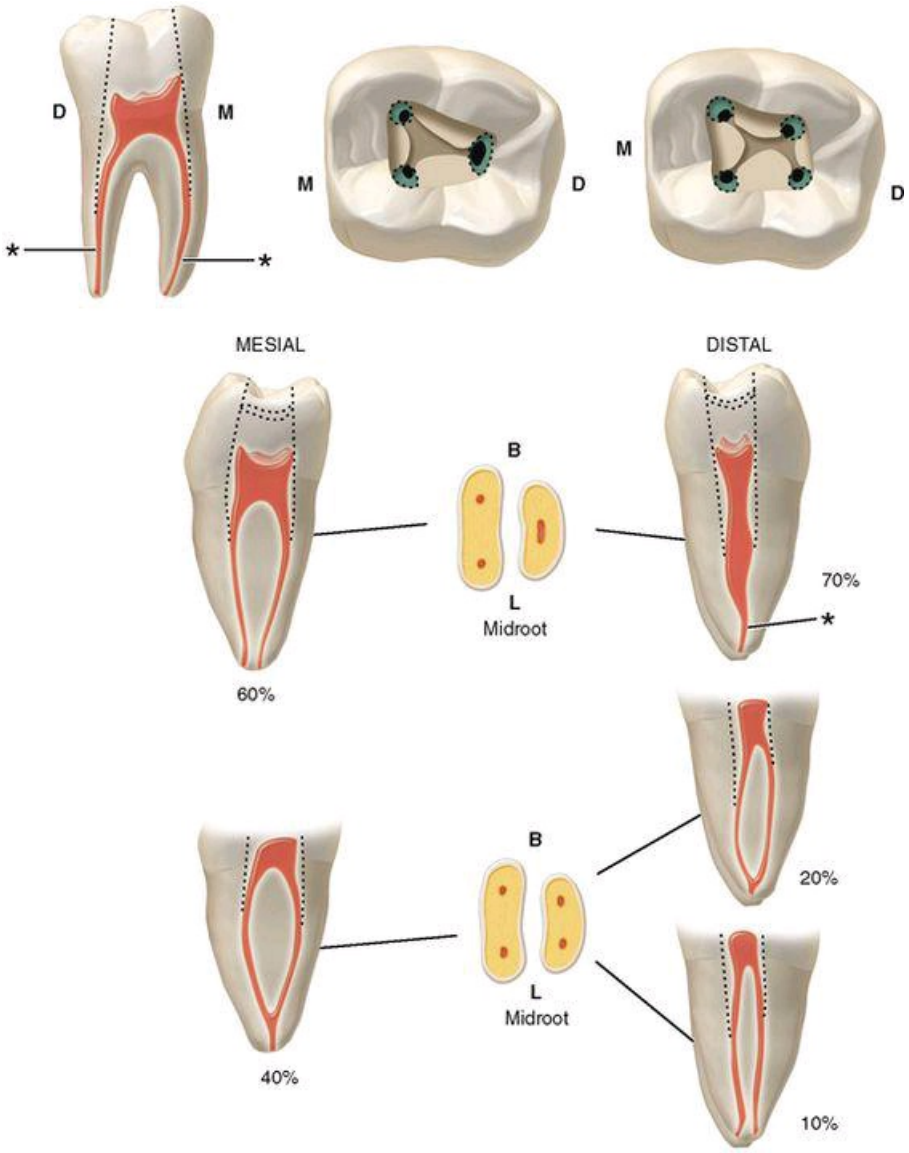
*Most common root curvatures

Mandibular Right Second Premolar



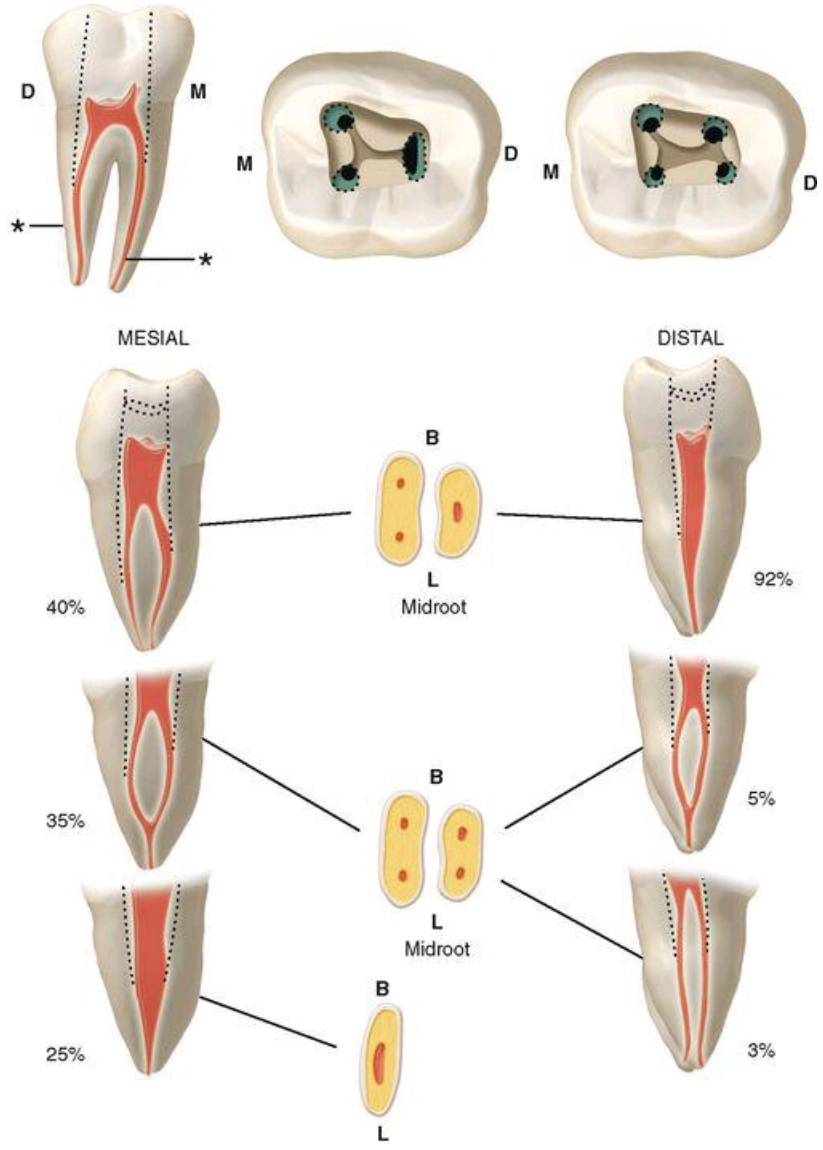
*Most common root curvatures

Mandibular Right First Molar



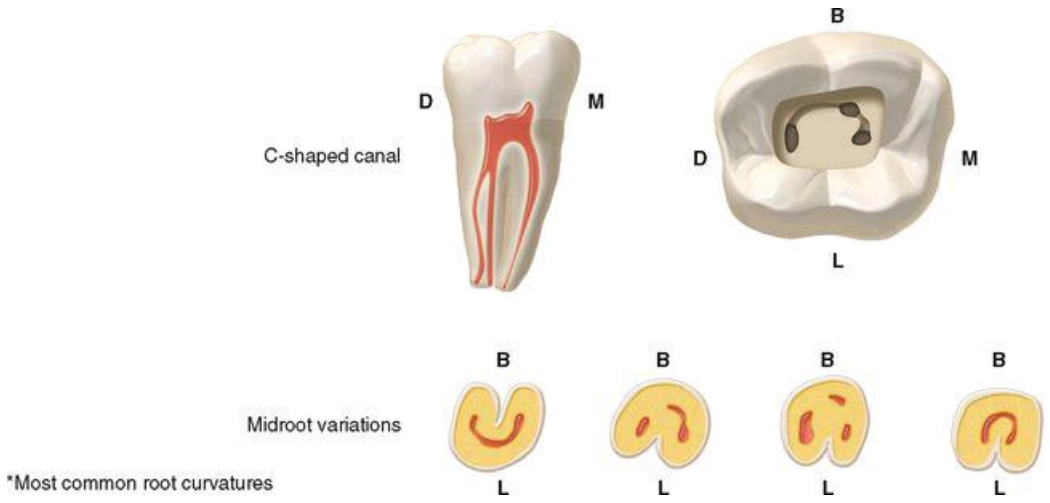
*Most common root curvatures

Mandibular Right Second Molar

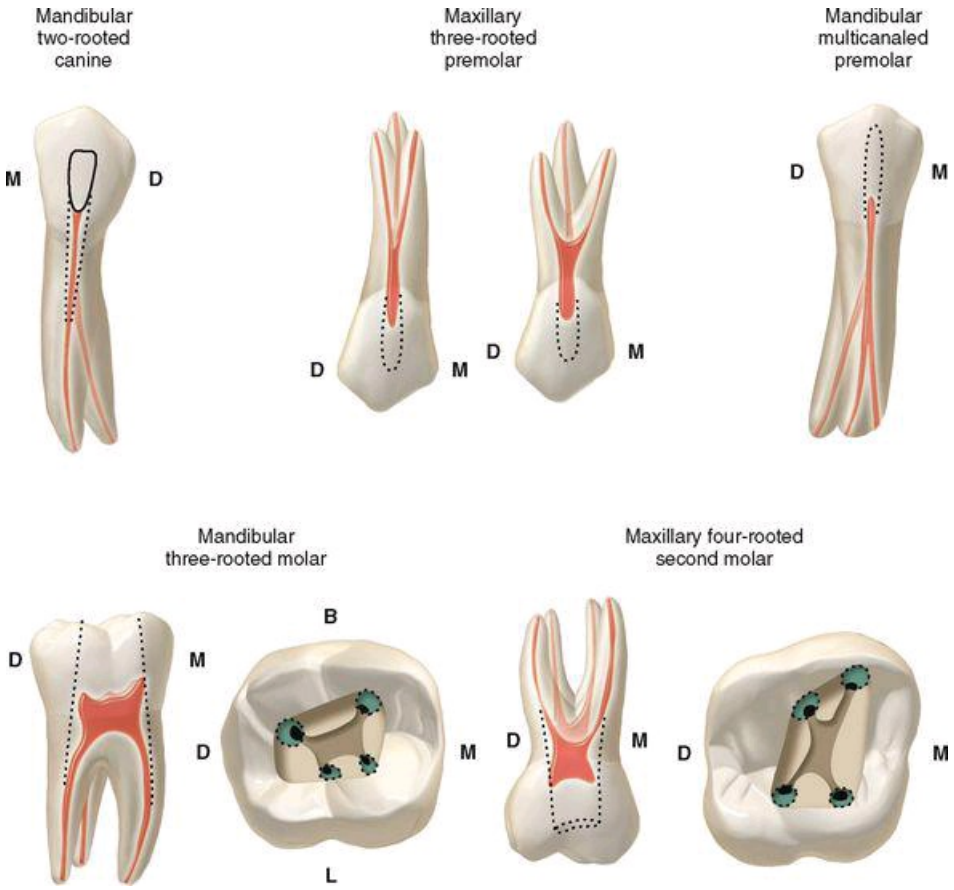


*Most common root curvatures

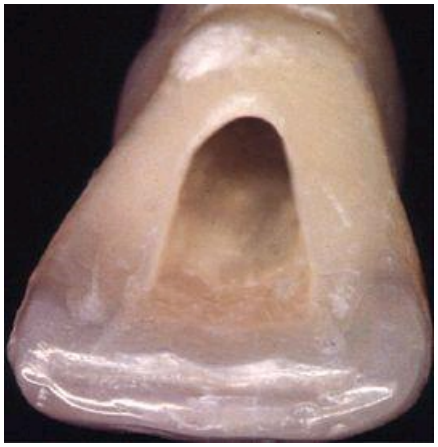
Mandibular Right Second Molar



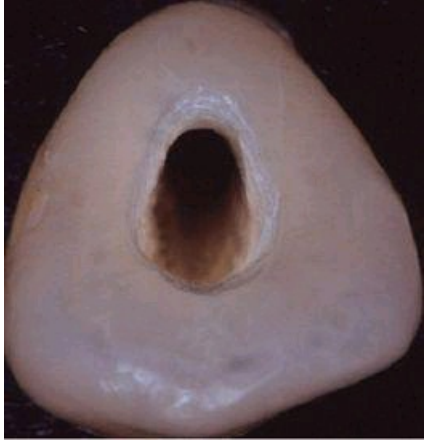
Some Uncommon Variations



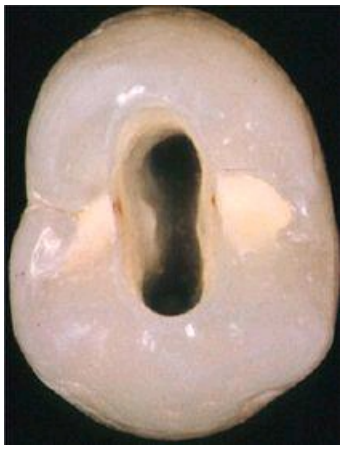
Examples of access openings prepared in extracted teeth are given here. It is important to recognize: (1) the location of the access relative to occlusal or lingual landmarks (marginal ridge and cusp tips) and (2) the size and shape of the access relative to the size and shape of the occlusal or lingual surface.



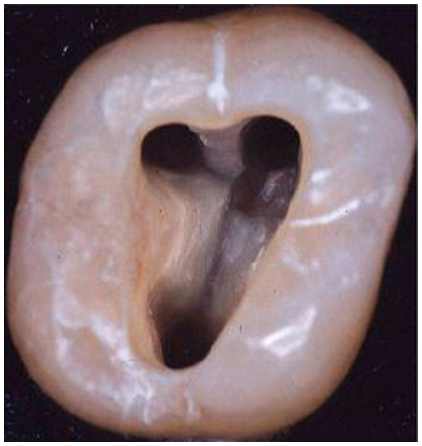
1. Maxillary central incisor



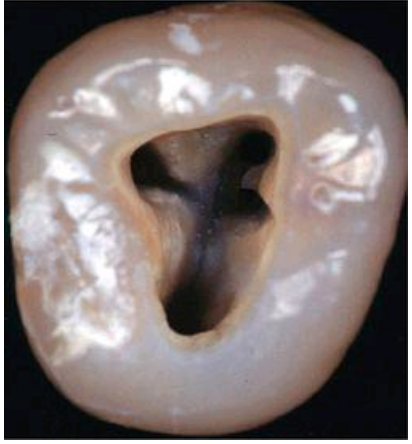
2. Maxillary canine



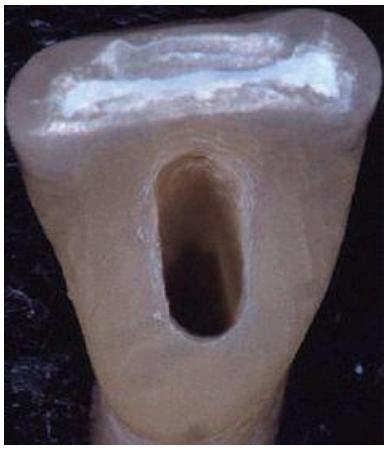
3. Maxillary first premolar



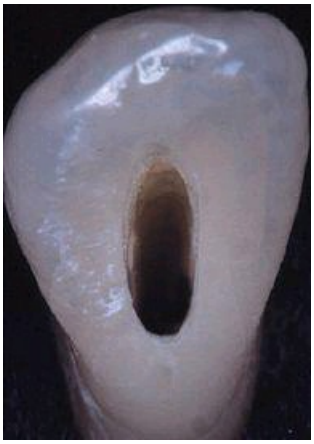
4A. Three-canal maxillary molar



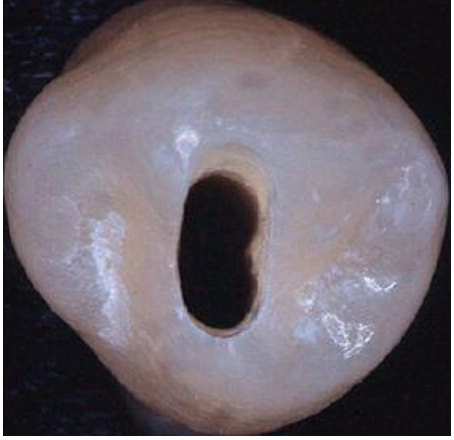
4B. Four-canal maxillary molar



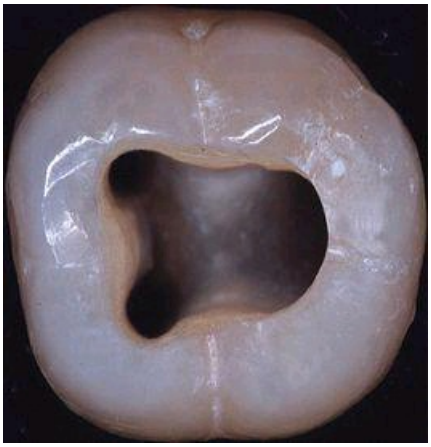
5. Mandibular incisor



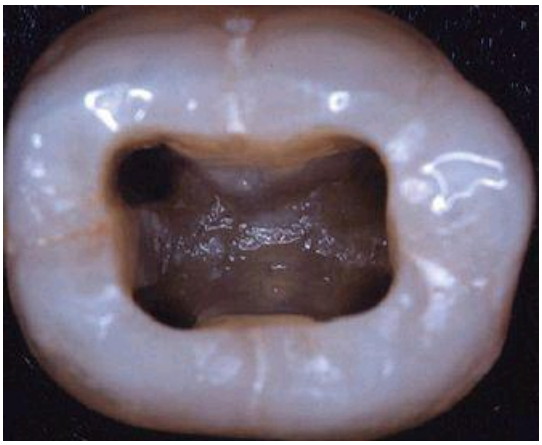
6. Mandibular canine



7. Mandibular premolar



8A. Three-canal mandibular molar



8B. Four-canal mandibular molar

Chapter Review Questions

Bruce C. Justman, W. Craig Noblett

Appendix B contains a series of questions for each of the chapters. These questions have been developed not only to test the reader's comprehension of the material, but also to ensure that the important concepts from each chapter are highlighted. In answering these questions, the reader will become acquainted with many of the critical points of information contained within each chapter.

The questions are set in a multiple choice format and are drawn from the chapters; however, a specific answer may not be immediately evident because a question may draw on several statements contained within the text. The review questions are available in two formats, print or electronic on the accompanying DVD. This appendix includes a correct answer key. The DVD includes a rationale for the correct answer, as well as the page cross reference in which those concepts and answers can be found in the book.

It is the authors' intention that by completing this exercise and understanding the answers, the reader will be able to organize the information into a basic understanding of endodontics.

CHAPTER 1

The Dental Pulp and Periradicular Tissues

1. What is the primary function of the dental pulp?
   a. induction
   b. formation
   c. nutrition
   d. defense

2. Which of the following is *not* a stage of tooth formation?
   a. bud
   b. cap
   c. bell
   d. basal

3. From what cells are odontoblasts derived?
   a. undifferentiated basal
   b. undifferentiated ectomesenchymal
   c. undifferentiated cartilage
   d. dental papilla

4. Where does deposition of unmineralized dentin matrix begin and in what direction does it proceed?
   a. It begins at the cusp tip and progresses laterally.
   b. It begins at the cusp tip and progresses cervically.
   c. It begins at the cementoenamel junction and progresses laterally.
   d. It begins at the cementoenamel junction and progresses coronally.

5. The cervical loop is the location of which of the following?
   a. the apical constriction
   b. formation of the pulp horn
   c. formation of the furcation
   d. where inner and outer dental epithelium meet

6. What is the first thin layer of dentin that is formed?
   a. predentin
   b. primary dentin
   c. mantle dentin
   d. root sheath

7. Epithelial cell rests of Malassez are remnants of what?
   a. odontoblasts
   b. cementoblasts
   c. epithelial root sheath
   d. teeth of the deciduous dentition

8. Why are lateral canals clinically significant?
   a. Symptoms may persist if not treated.
   b. They allow pulp disease to extend to periodontal tissues.
   c. They effectively block periodontal disease from extending to the pulp.
   d. They contain the greatest amount of pulp tissue.

9. What morphological changes occur to the dental pulp over time?
   a. Root canal diameter increases in size.
   b. Pulp horns grow higher into the cup tips.
   c. Overall size of the pulp chamber is reduced.
   d. Mesiodistal dimension of pulp space is reduced more than apical-occlusal dimension in molars.

10. The cementodentinal junction (CDJ) is which of the following?
    a. the area where cementum contacts dentin inside the canal
    b. located at the same level for each tooth type
    c. usually located 1.0 to 2.0 mm coronal to the apical constriction
    d. the widest portion of the canal

11. What is the defensive function of the dental pulp?
    a. odontoclast activation for resorption of dentin from areas of inflammation
    b. odontoblast formation of enamel to increase enamel thickness

c. odontoclast differentiation into macrophages
d. odontoblast formation of dentin in response to injury

12. Which of the following is *not* a major component of the odontoblast?
a. cell body
b. basally located nucleus
c. cell process
d. synaptic junction

13. What cell type is primarily related to the immune system in the dental pulp?
a. dendritic
b. macrophages
c. neutrophils
d. all of the above

14. What type of collagen is the most prominent found in the dental pulp?
a. type I
b. type II
c. type III
d. type IV

15. Which of the following is *not* a type of pulp stones?
a. free
b. attached
c. embedded
d. floating

CHAPTER 2

Protecting the Pulp, Preserving the Apex

1. What is a “pulp cap”?
   a. an early stage of tooth development
   b. capping the exposed pulp with a thin layer of lining or base material
   c. capping exposed necrotic tissue by placing a layer of mineral trioxide aggregate (MTA)
   d. a method of isolation used during root canal treatment

2. What is the effect on blood flow to the pulp when anesthetics with vasoconstrictors are used during restorative procedures?
   a. reduced by 10% its normal rate
   b. reduced to less than half its normal rate
   c. unchanged
   d. increased by 25% because of stress on the pulp tissue

3. What is dentin “blushing”?
   a. the color of newly erupted teeth due to large pulp chambers
   b. use of a masking color during restorative procedures
   c. vascular hemorrhage of pulp tissue, often during crown preparation
   d. an esthetic concern requiring laminate restorations

4. Why are deeper carious lesions more injurious to the dental pulp?
   a. increased dentin permeability in deeper areas
   b. increased length of the dentinal tubule in deeper areas
   c. decreased density of dentinal tubules in deeper areas
   d. decreased diameter of dentin tubules in deeper areas

5. Why does a blast of compressed air directed at freshly exposed dentin create a sensation of pain?
   a. frightens the patient
   b. air is cold
   c. rapid outward movement of fluid in patent dentinal tubules
   d. rapid inward movement of fluid in patent dentinal tubules

6. What is the most important characteristic of any restorative material in determining its effect on the pulp tissue?
   a. heat generated by the material
   b. speed the material sets
   c. ability to form a marginal seal
   d. life expectancy of the restorative material

7. What effect does orthodontic tooth movement have to the dental pulp?
   a. no clinically significant changes
   b. continued reliability to electric pulp testing
   c. extrusion reduces pulpal blood flow for a few minutes
   d. intrusive forces have no effect on pulpal blood flow

8. Should bases be used to protect the pulp beneath metallic restorations?
   a. Yes, a thin cement base should be used.
   b. Yes, a thick layer of varnish should be used.
   c. No, a base is only necessary if the tooth is to be crowned.
   d. No, additional thermal insulation is rarely needed.

9. Vital pulp therapies have variable rates of success. What is the most significant determinant of the success of vital pulp therapy?
   a. periapical status before the procedure
   b. periodontal status before the procedure
   c. pulp status before the procedure
   d. type of restorative material to be used

10. Which of the following steps are used in step-wise evacuation of caries?
   a. removing all the caries in a single visit
   b. placing a calcium hydroxide base at the initial visit
   c. placing a glass ionomer base at each visit
   d. removing only a superficial layer of caries at the first visit

11. How does a direct pulp cap for an accidental mechanical pulp exposure differ from a direct pulp cap for an exposure caused by caries?
   a. Pulp is likely to be severely inflamed beneath a deep carious lesion.
   b. Long-term success for a mechanical exposure is low.

c. Long-term success for a carious exposure is high.
d. A mechanical exposure should be immediately root canal treated.

12. When does apical closure occur in the developing root?
a. at the time of eruption
b. approximately 1 year after eruption
c. approximately 3 years after eruption
d. approximately 5 years after eruption

13. If the pulp becomes necrotic before root growth is complete, the resultant root is
a. short with thick dentin walls
b. short with thin dentin walls
c. normal length with thick dentin walls
d. normal length with thin dentin walls

14. What is apexogenesis?
a. induction of a calcific barrier across an open apex
b. removal of the necrotic pulp
c. determination of corrected working length
d. continued physiological root formation

15. Which of the following has *not* been demonstrated with MTA when used in apexification?
a. good biocompatibility
b. good sealability
c. high pH value
d. adjacent zone of necrosis

CHAPTER 3

Endodontic Microbiology

1. Which of the following is *not* a main portal of entry for microorganisms to enter the dental pulp?
   a. dentinal tubules
   b. direct pulp exposure
   c. periodontal disease
   d. occlusal grooves

2. Why is there greater dentin permeability near the pulp?
   a. increased thickness of dentin
   b. smaller diameter dentinal tubules
   c. higher density of dentinal tubules
   d. longer length of dentinal tubules

3. Exposed dentin provides an unimpeded access for bacteria to enter the pulp.
   a. True
   b. False

4. What is anachoresis?
   a. artificial formation of an apical barrier
   b. induction of a biologic calcific apical barrier
   c. microorganism transport from blood vessels into damaged tissue
   d. systemic infection resulting from infected pulp tissue

5. Root canals can become infected through anachoresis?
   a. true
   b. false

6. Which of the following is *not* a category of intraradicular infections?
   a. primary
   b. secondary
   c. tertiary
   d. persistent

7. The most common microorganisms in primary endodontic infections are
   a. gram-negative bacteria.
   b. gram-positive bacteria.
   c. facultative anaerobes.
   d. facultative aerobes.

8. Which of the following is *not* a source of nutrients for bacteria within the root canal system?
   a. necrotic pulp tissue
   b. inflamed vital pulp tissue
   c. proteins and glycoproteins seeping into the root canal system
   d. components of saliva penetrating into the pulp tissue

9. Which of the following microorganisms are commonly present in large percentages of root canal–treated teeth that present with persistent apical periodontitis, indicative of failed treatment?
   a. *Enterococcus faecalis*
   b. *Pseudoramibacter alactolyticus*
   c. *Tannerella forsythia*
   d. *Dialister invisus*

10. Gram-positive bacteria have been demonstrated to
   a. have a higher occurrence in post-instrumentation samples
   b. are more resistant to antimicrobial treatments
   c. are able to adapt to harsh environmental conditions
   d. all of the above

CHAPTER 4

Pulp and Periapical Pathosis

1. A direct pulp exposure of a carious lesion is necessary to have a pulpal response and inflammation.
   a. True
   b. False

2. What factor is *the* most important in determining if pulp tissue becomes necrotic slowly or rapidly after carious pulp exposure and pulpal inflammation?
   a. virulence of bacteria
   b. host resistance
   c. amount of circulation
   d. lymph drainage

3. What is necessary for pulp and periradicular pathosis to develop?
   a. exposure of pulp tissue
   b. exposure of dentin
   c. presence of bacteria
   d. trauma

4. Which of the following statements is true regarding mechanical irritants?
   a. Changes to the underlying pulp, such as odontoblast aspiration, are irreversible.
   b. Potential for pulp injury decreases as more dentin is removed.
   c. Operative procedures without water coolant cause more irritation than those performed under water spray.
   d. There is decreased permeability and constriction of blood vessels in the early stages of pulpitis.

5. What nonspecific inflammatory mediators are *not* present when the dental pulp is irritated?
   a. histamine
   b. epinephrine
   c. bradykinin
   d. arachidonic acid metabolites

6. What cell type associated with immune response is *not* present in severely inflamed dental pulp?
   a. T lymphocytes
   b. B lymphocytes
   c. macrophages
   d. odontoclasts

7. What is the cause of pain during the progression of pulpal injury?
   a. elevation of the sensory nerve threshold
   b. decrease of arteriole vasodilatation
   c. increase of venule vascular permeability
   d. decrease of pulp tissue pressure

8. What is reversible pulpitis?
   a. severe inflammation of pulp tissue
   b. yields a negative response to electric pulp testing
   c. yields a positive response to thermal pulp testing
   d. requires root canal treatment

9. What is irreversible pulpitis?
   a. a severe inflammatory process
   b. precedes reversible pulpitis
   c. resolves when the causative agent is removed
   d. yields a negative response to thermal pulp testing

10. Which of the following is *not* a hard tissue change that may result from pulpal irritation or inflammation?
    a. calcification of pulp tissue spaces
    b. resorption of pulp tissue spaces
    c. formation of pulp stones
    d. thickening of periodontal ligament

11. What are the signs and symptoms associated with symptomatic apical periodontitis (acute apical periodontitis)?
    a. normal sensation on mastication
    b. normal sensation on finger pressure
    c. marked or excruciating pain on tapping with a mirror handle

d. presence of a large periapical lesion

12. What histologic feature differentiates a periapical granuloma from a periapical cyst?
    a. presence of mast cells
    b. presence of lymphocytes
    c. presence of plasma cells
    d. presence of an epithelial lined cavity

13. Which of the following is *not* associated with acute apical abscess?
    a. moderate to severe discomfort
    b. intense and prolonged response to thermal stimulus
    c. negative response to electric pulp testing
    d. tenderness to percussion and palpation

14. What factors may impact and influence whether periradicular lesions heal completely or incompletely?
    a. size of the lesion
    b. blood supply
    c. systemic disease
    d. all of the above

15. What is the most important aid in distinguishing between endodontic and nonendodontic periradicular lesions?
    a. radiographic location
    b. radiographic appearance
    c. pulp vitality testing
    d. patient's history

CHAPTER 5

Diagnosis and Treatment Planning

1. Which of the following is *not* one of the five basic steps in the diagnostic process?
   a. chief complaint
   b. medical and dental history
   c. oral examination
   d. review of insurance coverage

2. Are patients that seek endodontic treatment usually younger or older than the general population?
   a. Age has not been shown to be a factor.
   b. Patients seeking endodontic treatment are usually younger.
   c. Patients seeking endodontic treatment are usually older.

3. During a review of the patient's healthy history, it is noted that the patient is on a regimen of intravenous bisphosphonate medication. What significance does this hold for the patient and their treatment plan?
   a. possible side effect of bleeding disorders
   b. possible side effect of osteonecrosis of the jaw
   c. lowered pain threshold
   d. inability to obtain adequate anesthesia

4. When pain is one of the patient's complaints, what question is less relevant regarding their pain and does not need to be asked of the patient?
   a. When did the pain begin?
   b. Is the pain always in the same place?
   c. Why did you not seek treatment when the pain began?
   d. Once initiated, how long does the pain last?

5. Why is it important to use control teeth during the clinical tests?
   a. to calibrate the patient's response
   b. so the patient can predict which tooth is being tested
   c. so teeth can be tested repeatedly
   d. to test whether isolation is adequate

6. A painful response obtained by pressing or by tapping on the crown indicates the presence of which of the following?
   a. periapical inflammation.
   b. pulpal inflammation.

7. What is palpation testing used to determine?
   a. pulpal inflammation
   b. periapical inflammation
   c. periodontal inflammation
   d. periapical histology

8. Which of the methods of cold testing are preferred for pulp testing?
   a. regular ice (frozen water)
   b. refrigerant spray or CO_2
   c. flooding the arch with chilled water
   d. blast of air from the air/water syringe

9. How does electrical pulp testing determine the degree of pulpal inflammation?
   a. A shorter response indicates a healthier pulp.
   b. A midrange response indicates pulp inflammation.
   c. A midrange response indicates partial necrosis.
   d. It can only be used to determine the presence or absence of vital tissue.

10. What are the four characteristics of a periapical lesion of endodontic origin?
   a. The lamina dura of the tooth socket is intact.
   b. The lucency remains at the apex in radiographs made at different cone angles.
   c. The lucency tends to resemble a round circle.
   d. It is usually associated with an irreversible pulpitis.

11. In which situation is caries removal necessary to obtain a definitive pulpal diagnosis?
   a. deep caries with no symptoms and negative pulp testing
   b. deep caries with no symptoms and positive pulp testing
   c. shallow caries with mild symptoms and positive pulp testing
   d. shallow caries with mild symptoms and negative pulp testing

12. How may selective anesthesia be an aid in diagnosis?
    a. It can localize a painful tooth to a specific arch.
    b. It can localize an individual painful tooth in the mandibular arch.
    c. It can confirm the tooth the patient identifies as the offending tooth.
    d. It can be used to start posterior and work toward the anterior teeth.

13. Using the Case Difficulty Assessment system developed by the American Association of Endodontists, cases in which any factors score 3 should be
    a. treated by a general dentist.
    b. treated by an endodontist.

14. Which of the following is *not* a category of external resorption?
    a. inflammatory
    b. replacement
    c. regenerative
    d. surface

15. If a patient is to be referred to an endodontist for treatment, when is the most appropriate time for the referral?
    a. before beginning treatment
    b. during treatment when expected difficulties arise
    c. before weekends or holidays
    d. after obturation when separated instruments are present

CHAPTER 6

Endodontic and Periodontal Interrelationship

1. Which of the following are potential avenues for communication between the dental pulp and periodontium?
   a. dentinal tubules
   b. apical foramen
   c. lateral/accessory canals
   d. all of the above

2. What characterizes the diameter of dentinal tubules in radicular dentin?
   a. The diameter is 1 µm on the root surface, 3 µm at the pulp.
   b. The diameter is constant.
   c. The diameter is 3 µm on the root surface, 1 µm at the pulp.
   d. The diameter increases with age.

3. Patent accessory canals are characterized by which of the following?
   a. They serve as a pathway for microorganisms from pulp to periodontium.
   b. They can be the result of scaling and root planing.
   c. In the apical third, they may lead to pulp necrosis if exposed to oral environment.
   d. All of the above.

4. A narrow, vertical probing depth associated with pulp necrosis, but only mild periodontal disease is probably
   a. a vertical root fracture
   b. a fistula
   c. a sinus tract
   d. a periodontal abscess

5. The effect of periodontal disease on the pulp is thought to occur by migration of _____ through dentinal tubules, accessory canals, or the apical foramen.
   a. dental plaque
   b. saliva
   c. microorganisms
   d. exudates

6. Poor endodontic treatment can allow reinfection of the canal, leading to treatment failure and subsequent inflammatory response of the periodontal tissues.
   a. True
   b. False

7. Periodontal inflammation resulting from primary endodontic disease may mimic periodontal disease by which of the following?
   a. generalized increase in probing depths in the affected quadrant
   b. an apical radiolucency
   c. a narrow deep solitary probing defect associated with an affected tooth
   d. all of the above

8. A patient presents with a chief complaint of pain to cold temperatures. Examination and testing reveal a maxillary left premolar exhibiting severe lingering pain in response to cold. No caries or fractures are noted. Periodontal probings are 6 to 9 mm around that specific tooth and 6 to 7 mm around the other posterior teeth in the quadrant. The patient reports having a “deep cleaning” (root planing) three times per year. What would the diagnosis in this case be?
   a. primary endodontic disease with secondary periodontal involvement
   b. primary periodontal disease with secondary endodontic involvement
   c. separate and unrelated endodontic and periodontal disease
   d. a true combined endodontic-periodontic (endo-perio) disease process

9. What will the long-term prognosis for the patient described in question 8 *primarily* depend on?
   a. successful periodontal treatment
   b. successful endodontic treatment
   c. treatment sequencing
   d. timing of treatment

10. Which of the following best represents similarities between endodontic apical and periodontal pathosis?
   a. Both are often the result of traumatic occlusion.
   b. Both are usually symptomatic.
   c. Both are mediated by microorganisms.
   d. Both are associated with loss of attachment.

11. The best description of the effect of moderate periodontal disease (loss of attachment in the apical/middle third) on the underlying pulp is usually characterized by
    a. no or slight regional inflammation
    b. generalized acute inflammation
    c. generalized chronic inflammation
    d. necrosis
    e. bacterial invasion through tubules into the pulp

12. Which of the following is characteristic of the true combined endo-perio lesion?
    a. It occurs much less frequent than the primary endodontic lesion.
    b. It is usually the end result of a severe endodontic lesion that causes loss of attachment.
    c. It is usually the end result of a severe periodontal lesion.
    d. It is usually successfully managed with both endodontic and periodontal treatment.

13. What is the best means of differentiating endodontic from periodontal pathosis?
    a. pulp vitality testing
    b. percussion
    c. radiographs
    d. probing patterns
    e. location of swelling

14. Treatment sequencing for primary endodontic disease with secondary periodontal involvement includes
    a. scaling and root planing followed by endodontic treatment
    b. endodontic treatment followed by scaling and root planing
    c. endodontic treatment followed by periodontal surgery
    d. endodontic treatment followed by reevaluation of periodontal status in 2 to 3 months

CHAPTER 7

Longitudinal Tooth Fractures

1. What category of longitudinal tooth fractures is *most* severe?
   a. craze lines
   b. fractured cusp
   c. cracked tooth
   d. vertical root fracture

2. What clinical conditions or situations are often associated with cusp fractures?
   a. teeth with minimal caries
   b. strong support of the remaining cusps
   c. missing marginal ridge
   d. occlusal composite restorations

3. What is the common direction that fractures extend in cracked teeth?
   a. mesiodistal
   b. faciolingual
   c. apical to coronal
   d. horizontal

4. Which of the following is true as related to pulp tissue involvement in a cracked tooth?
   a. The fracture always includes the pulp tissue.
   b. The fracture never includes the pulp tissue.
   c. The more centered the fracture, the greater chance for pulp exposure.
   d. The more facial or lingual oriented the crack, the greater the chance for pulp exposure.

5. Are pulp and periapical tests for cracked teeth consistent and reliable?
   a. Both pulp and periapical testing are consistent.
   b. Pulp testing is consistent, but periapical testing is variable.
   c. Pulp testing is variable, but periapical testing is consistent.
   d. Both pulp and periapical testing are variable.

6. How can transillumination be used to distinguish between a craze line and a crack line?
   a. It cannot be used to distinguish between the two entities.
   b. Transmitted light readily passes through the air space of a fracture.
   c. Transilluminated light is blocked by craze lines.
   d. Transilluminated light is blocked by a cracked tooth.

7. After access preparation of a suspected cracked tooth, the crack line is seen to extend through the chamber floor. In this case, what is the prognosis and recommended treatment?
   a. Prognosis is favorable and continue with root canal treatment.
   b. Prognosis is questionable, inform patient and continue with treatment.
   c. Prognosis is hopeless, and extraction is recommended.
   d. It does not change the original prognosis, and the treatment plan is not altered.

8. What is the preferable restoration of a cracked tooth?
   a. post and core and crown
   b. amalgam core
   c. cast inlay
   d. full coverage crown

9. How does a split tooth differ from a cracked tooth?
   a. A split tooth precedes a cracked tooth.
   b. A split tooth has an incomplete fracture.
   c. A split tooth has separable tooth segments.
   d. A cracked tooth has the fracture extending faciolingually.

10. What direction does a vertical root fracture (VRF) primarily occur?
   a. mesiodistal
   b. faciolingual
   c. coronal and extending apically
   d. no primary direction

11. What is a demonstrated major cause of VRFs?
   a. traumatic occlusion

b. occlusal biting habits
c. loss of one or both marginal ridges
d. condensation forces during obturation

12. Which is *not* a possible treatment of a VRF in a multirooted tooth?
a. tooth extraction
b. nonsurgical retreatment of the affected root
c. root amputation of the affected root
d. hemisection and extraction of the affected root

CHAPTER 8

Local Anesthesia

1. What is the allodynia phenomenon?
   a. Inflamed tissue has an increased threshold of pain.
   b. Inflamed tissue has a decreased threshold of pain.
   c. Inflamed tissue is much less sensitive to a mild stimulus.
   d. Inflamed tissue responds mildly to a stimulus that would otherwise be very painful.

2. Which of the following is *not* a component of the psychologic approach to pain management?
   a. control
   b. communication
   c. conservation
   d. confidence

3. What is the effect of warming the anesthetic solution on the amount of pain during the injection?
   a. Warmed anesthetic results in less pain during injection.
   b. Warmed anesthetic solution results in greater pain during injection.
   c. There is no difference in pain perception regardless of warming.

4. What is a two-stage injection?
   a. Injection of one cartridge of anesthetic, waiting 5 minutes followed by injection of a second cartridge of the same anesthetic solution.
   b. Injection of one cartridge of anesthetic, followed by injection of a second cartridge of a different anesthetic solution.
   c. Injection of a quarter cartridge of anesthetic under the mucosal surface, waiting until regional anesthesia, then injection of the remainder of the cartridge to full depth.
   d. Injection of a quarter cartridge of anesthetic under the mucosal surface, waiting until regional anesthesia, then injection of a cartridge of different anesthetic solution to full depth.

5. When does the onset of pulpal anesthesia occur after the inferior alveolar injection?
   a. immediately
   b. 0 to 5 minutes
   c. 10 to 15 minutes
   d. 30 minutes

6. Does the direction of the needle bevel affect the effectiveness of the inferior alveolar nerve block?
   a. Needle bevel toward the mandibular ramus improves success.
   b. Needle bevel away from the mandibular ramus improves success.
   c. Half the cartridge should be injected with the bevel toward the ramus, the needle rotated, and the second half of the cartridge injected with the bevel away from the ramus.
   d. Direction of the needle bevel does not affect success.

7. Is anesthesia of the maxilla commonly more or less successful than anesthesia in the mandible?
   a. more successful
   b. less successful
   c. comparable success rates

8. Does increasing the volume of anesthetic solution affect the duration of pulpal anesthesia?
   a. For mandibular anesthesia, increasing the volume improves the success rate with the inferior alveolar nerve block.
   b. For maxillary infiltrations, increasing the volume increases the depth of pulpal anesthesia.
   c. For maxillary infiltrations, increasing the volume increases the duration of pulpal anesthesia.
   d. For maxillary infiltrations, increasing the volume has no effect on the success rate of pulpal anesthesia.

9. What additional anesthesia procedure should be administered if the classic signs of anesthesia are present after a standard injection, but the patient still has sharp pain when the bur enters the dentin?
   a. repeat the initial injection
   b. wait an additional 15 minutes and attempt access again
   c. repeat the injection using a different type of anesthetic solution
   d. use a supplemental injection technique for a second injection

10. What is a consideration of the intraosseous (IO) injection?
   a. It has not been proved effective.
   b. It has been recommended as the primary injection technique.
   c. It allows the anesthetic solution to be deposited directly into the pulp tissue of the tooth.
   d. It allows the anesthetic solution to be deposited directly into the cancellous bone adjacent to the tooth.

11. What is the best site for an IO injection of a premolar?

a. mesial perforation and injection
b. apical perforation and injection
c. distal perforation and injection
d. site of injection not important

12. What is an important requirement for effectiveness when using a periodontal ligament (PDL) injection?
a. back-pressure during injection
b. direction of the needle bevel toward the root surface
c. direction of the needle bevel away from the root surface
d. all four line angles receive the injection

13. Can a PDL injection be used for individual tooth selective anesthesia as an aid in diagnosis?
a. PDL injection is useful for single tooth anesthesia.
b. PDL injection is not useful for single tooth anesthesia.

14. Which of the following is an important consideration of the intrapulpal injection (IP)?
a. The injection should be given with back-pressure.
b. It will take several minutes for the injection to take effect.
c. A long-acting anesthetic should be used.
d. Another supplemental injection should not be attempted first.

15. What are the most difficult teeth to anesthetize with irreversible pulpitis?
a. maxillary molars
b. mandibular molars
c. maxillary anterior teeth
d. maxillary premolars

16. Why should an anesthetic agent not be injected directly into a swelling before an incision for drainage?
a. The anesthetic will cause a decreased flow of exudate following incision.
b. A direct injection will spread the infection.
c. There is an increased chance of aspirating blood.
d. The swelling has increased blood supply so the anesthetic is transported quickly into systemic circulation, diminishing the effect.

CHAPTER 9

Endodontic Emergencies and Therapeutics

1. What is the difference between a true endodontic emergency and urgency?
   a. A true emergency is a condition requiring an unscheduled office visit.
   b. A true emergency may be rescheduled for convenience of the patient.
   c. An urgency indicates a more severe problem.
   d. An urgency may need to be seen after normal office hours.

2. How many teeth are usually involved in a true emergency?
   a. one tooth
   b. two teeth
   c. often several teeth throughout the mouth
   d. often teeth in only one quadrant

3. Which of the following is *not* an important factor in assessing the quality and quantity of pain?
   a. spontaneity
   b. intensity
   c. time of day of occurrence
   d. duration

4. Which of the following is *not* an immediate goal of the emergency treatment plan?
   a. pharmacotherapeutic management of swelling
   b. reducing the irritant
   c. reduction of pressure
   d. removal of the inflamed pulp or periradicular tissue

5. What is the most critical factor in a pretreatment emergency?
   a. adequate health history
   b. pain management
   c. patient management
   d. adequate provisional restoration

6. What is the preferred treatment for an emergency appointment with a diagnosis of irreversible pulpitis with symptomatic apical periodontitis?
   a. caries excavation with provisional restoration
   b. trephination through the mucosa and bone
   c. partial or total pulpectomy
   d. pharmaceutical management of swelling

7. What is the emergency treatment of choice for pulp necrosis without swelling?
   a. trephination for drainage
   b. pulpotomy
   c. canal débridement to corrected working length
   d. single visit root canal treatment

8. A patient may present with localized swelling; incision for drainage may be indicated. What does drainage accomplish?
   a. patient reassurance and management
   b. decrease in blood flow to the area
   c. allows administration of anesthetic solution to the apex
   d. removal of a very potent irritant'purulence

9. At an emergency appointment, should teeth be left open to drain?
   a. Yes, only if there is swelling.
   b. Yes, only if there is no swelling.
   c. No, teeth should have an interappointment temporary restoration placed.

10. What is the most important consideration of administering antibiotics with a localized apical abscess?
   a. The dosage should be for 10 days.
   b. It should be a broad-spectrum antibiotic.
   c. The dosage levels should be higher than usual.
   d. Antibiotics are ineffective and should not be prescribed.

11. What is the incidence of interappointment flare-ups?
   a. primarily after obturation at a range of 4.4 to 6.0%
   b. overall at a range of 1.8 to 3.2%
   c. overall at a range of 10.7 to 21.3%

d. primarily after vital pulp removal at a range of 7.5 to 10.7%

12. Which of the following has been identified as significant factor related flare-ups?
    a. teeth with vital pulps
    b. teeth without periradicular radiolucency
    c. patient presenting with preoperative pain or swelling
    d. completing endodontic treatment in a single visit

13. What is a consideration of postobturation emergencies?
    a. more common than flare-ups following cleaning and shaping
    b. infrequent and usually resolve spontaneously
    c. approximately 10% to 15% occurrence
    d. often require nonsurgical root canal retreatment

CHAPTER 10

Management of Traumatic Dental Injuries

1. Why is age a “good news/bad news” situation with dental trauma?
   a. Pulps have an incomplete and decreased blood supply.
   b. Pulps are better able to recover and have a better repair potential.
   c. Poor development will continue in teeth with damaged necrotic pulps.
   d. Dentin has more strength in younger teeth.

2. Which of the following factors does *not* need to be considered when evaluating a crown fracture with pulp exposure?
   a. extent of fracture
   b. stage of root development
   c. position in the arch
   d. length of time since the injury

3. Which of the following is *not* a step as part of the technique for a shallow (partial) pulpotomy?
   a. rubber dam isolation
   b. pulp tissue removed to about 2 mm below the exposure
   c. use of a large round carbide bur in the slow-speed handpiece to remove tissue
   d. restoration of the cavity with a hard-setting cement

4. How long should horizontal root fractures be splinted if the coronal section was displaced and repositioned?
   a. not indicated
   b. 7 to 10 days
   c. 4 to 6 weeks
   d. 3 months

5. Which of the following are types of luxation injuries?
   a. concussion
   b. intrusion
   c. extrusion
   d. all of the above

6. What is recommended with pulp testing for teeth with traumatic injuries?
   a. use of electric pulp testing or carbon dioxide ice to test the injured and adjacent teeth
   b. opposing teeth also be tested
   c. retesting is done in 4 to 6 weeks
   d. all of the above

7. What information does the color change of the clinical crown provide?
   a. There has been a pulp exposure.
   b. The initial change is gray in color, which always indicates pulp necrosis.
   c. Discoloration may be reversed without treatment.
   d. Calcific metamorphosis discoloration tends to be yellow to brown and always indicates pulp necrosis.

8. What factor should be considered that determines the treatment of an intrusive luxation injury?
   a. depth of intrusion
   b. stage of root development
   c. availability of adjacent teeth for stabilization
   d. amount of soft tissue injury

9. Of the following, what is the best transport medium to use for transporting an avulsed tooth?
   a. saliva
   b. distilled water
   c. wrapped in a tissue
   d. milk

10. Which type of medication is indicated for patients with avulsed teeth?
    a. narcotic analgesic
    b. steroid
    c. tetanus booster if more than 5 years since last administered
    d. all of the above

11. What additional treatment should be used on the root surface if an avulsed tooth is replanted after more than 1 hour after avulsion?
    a. thorough scrubbing with antimicrobial soap for disinfection
    b. scaling of the root surface

c. soaking the tooth in 2.4% doxycycline for 5 to 20 minutes
d. soaking the tooth in a 2.4% solution of sodium fluoride for 5 to 20 minutes

12. Which of the following types of external resorption has *not* been identified with replanted avulsed teeth?
a. surface
b. inflammatory
c. refractory
d. replacement

13. When is root canal treatment indicated in a mature avulsed, replanted tooth?
a. at the time of replantation
b. within 7 to 10 days after replantation
c. after 3 months if there is no response to pulp testing
d. when periapical pathosis is noted

14. A deciduous tooth that has suffered an intrusive luxation should be extracted if what occurs?
a. The child cries but is compliant.
b. The permanent successor is partially erupted.
c. The intruded tooth appears foreshortened on the radiograph.
d. The intruded tooth appears elongated on the radiograph.

CHAPTER 11

Endodontic Radiography

1. Diagnostic radiology is helpful in all of the following except:
   a. identifying pathosis
   b. determining root anatomy
   c. determining pulp anatomy
   d. determining pulp responsiveness

2. What are working length radiographs?
   a. Radiographs made by removing the rubber dam.
   b. Radiographs placed using an XCP positioning device.
   c. Radiographs that help establish an estimated working length.
   d. Radiographs determine the distance from the radiographic apex to a reference point.

3. Radiographs are useful to evaluate the following qualities of an obturation *except* which of the following?
   a. length
   b. density
   c. sealer thickness
   d. canal configuration

4. Radiographs are useful in evaluating success and failure at recalls because they do which of the following?
   a. record subjective symptoms
   b. show pulp vitality
   c. may show failures that often occur without adverse signs or symptoms
   d. accurately diagnose apical pathosis

5. The most accurate radiographs are made by doing which of the following?
   a. having the patient hold the film in place with their index finger
   b. using a paralleling device
   c. increasing/decreasing the vertical angulation to move superimposed objects out of the field of vision
   d. having the rubber dam in place for isolation

6. Use of a paralleling technique may not be feasible when which of the following occurs?
   a. There is a high palatal vault.
   b. There are maxillary tori.
   c. A fixed prosthesis is present.
   d. There are exceptionally short roots.

7. F Speed film requires how much less exposure compared to E Speed film?
   a. 10% to 15%
   b. 20% to 25%
   c. 30% to 35%
   d. 50%

8. What does the cone-image shift do?
   a. It gives a clear 2-dimensional image.
   b. It superimposes facial and lingual structures.
   c. It assists in identifying superimposed canals.
   d. It moves apical endodontic lesions away from the root apex.

9. What occurs as the cone position moves away from parallel?
   a. Objects on the film shift toward the direction of the cone.
   b. The facial or buccal object shifts less than the lingual object.
   c. The lingual object moves relatively in the same direction as the cone.
   d. The buccal object moves relatively in the same direction as the cone.

10. What is a disadvantage to the cone-image shift?
   a. Lingual objects become more distorted than buccal objects.
   b. There is excessive contrast between radiolucent and radiopaque objects.
   c. It may superimpose normal anatomic structures over the root apices.
   d. It does not reveal additional canals within a root.

11. Which of the following is a distinguishing characteristic of a radiolucent lesion of endodontic pathosis?
   a. Apical/radicular lamina dura is present and intact.
   b. A round ball shape is characteristic.

c. The radiolucency stays at the apex regardless of cone angulation.
d. There is no apparent cause of pulpal necrosis.

12. A radiolucency of endodontic origin is usually present with what type of pulpal diagnosis?
a. normal pulp
b. reversible pulpitis
c. irreversible pulpitis
d. necrotic pulp

13. What is the usual radiographic appearance of condensing osteitis?
a. diffuse radiopaque appearance
b. uniform smooth borders
c. irregular moth-eaten appearance around the apex
d. presence of a radiolucent inflammatory lesion

14. A mesial projection cone adjustment during working length radiographs is indicated for what?
a. maxillary anterior teeth
b. maxillary molars with a mesiolingual canal
c. mandibular incisors
d. mandibular molars with a second distal canal

15. Digital radiography has *not* been proven to do which of the following?
a. provide superior image quality
b. reduce radiation to the patient
c. increase speed of obtaining an image
d. accurately and reliably be transmitted between computers

CHAPTER 12

Endodontic Instruments

1. What must an instrument do to completely clean the canal space?
   a. be deflected at the canal orifice
   b. be 2 to 3 mm short of the radiographic apex
   c. fit loosely into the canal
   d. contact all walls and surfaces

2. What motion is employed with a hand instrument to clean and shape canal walls?
   a. pushing
   b. broaching
   c. reaming
   d. vibration

3. Nickel-titanium alloy has increased flexibility over stainless steel. How does the modulus of elasticity for nickel-titanium alloy compare to that of stainless steel?
   a. similar to stainless steel
   b. one-fourth to one-fifth that of stainless steel
   c. half that of stainless steel
   d. 2 to 3 times that of stainless steel

4. What is a disadvantage as a result of the increased flexibility of nickel-titanium instruments?
   a. difficulty in negotiating curvatures
   b. inability to rotate in the canals
   c. cannot precurve the files to bypass ledges
   d. tendency to bind in small canals

5. According to ADA Specification No. 28, what is the rate of increase in file diameter per running millimeter of length for a K-type file from point D_0 to point D_{16}?
   a. 0.02 mm per running millimeter of length
   b. 0.04 mm per running millimeter of length
   c. 0.06 mm per running millimeter of length
   d. parallel sided so no increase in diameter

6. What is torsional limit?
   a. amount of apical pressure that can be applied to a file to the point of breakage
   b. the beginning of plastic deformation of the instrument
   c. amount of rotational torque that can be applied to a “locked” instrument to the point of breakage
   d. amount of force necessary so that a file does not return to its original shape upon unloading of the force

7. Which is a stronger metal alloy: carbon steel or stainless steel?
   a. Carbon steel is stronger.
   b. Stainless steel is stronger.

8. How do Gates-Glidden drills differ from Peeso reamers?
   a. Gates-Glidden drills are a greater length of cutting surface.
   b. Gates-Glidden drills are more aggressive cutters.
   c. Gates-Glidden drills have an elliptical-shaped cutting area.
   d. Gates-Glidden drills are less flexible.

9. How are broaches intended to be used in the canal?
   a. planing of canal walls by a push-pull motion
   b. planing of canal walls by a reaming motion
   c. placed to the corrected working length around canal curvatures
   d. entangling and removing canal contents by rotation

10. Which of the following describes the filing motion?
   a. a single-step motion
   b. used only on the furcation side of a molar root canal
   c. with a 360-degree rotation motion
   d. circumferential movement around the canal walls

11. Regular inspection of hand files may aid in avoidance of instrument separation. What file defects should be looked for on inspection?
   a. unwinding of the flutes
   b. rolling up or tightening of the flutes

c. distortion of the tip
d. all of the above

12. What are the characteristics of finger spreaders and pluggers compared to handled instruments when used for lateral condensation?
a. They are annealed to give them greater strength.
b. They are best suited for straight canals.
c. They are more rigid to access the canal orifice.
d. They have greater flexibility.

13. Is pressure sterilization superior to dry heat sterilization for sterilization of sharp-edged instruments?
a. Both are equal and comparable and effective.
b. Neither should be used for sterilization.
c. Yes, pressure sterilization is superior.
d. No, dry heat sterilization is superior.

14. What are the time, temperature, and pressure necessary for sterilization of gauze-wrapped instruments using pressure sterilization?
a. 10 minutes at 121°C and 15 psi
b. 10 minutes at 100°C and 15 psi
c. 20 minutes at 121°C and 15 psi
d. 20 minutes at 100°C and 15 psi

CHAPTER 13

Internal Anatomy

1. Lack of knowledge of pulp anatomy is the _____ most common cause of treatment failure.
   a. least
   b. second
   c. third
   d. fourth

2. Of the following, which is the best technique to determine if a root contains two canals?
   a. apex locator
   b. viewing access with a microscope
   c. searching with an explorer
   d. interpreting angled radiographs

3. The shape of the canal in cross-section is variable but is almost always round in the apical third.
   a. Both parts of the sentence are true.
   b. The first part of the sentence is false, the second part is true.
   c. The first part of the sentence is true, the second part is false.
   d. The entire sentence is false.

4. Multiple canals in mandibular premolars occur most often in which population?
   a. Asians
   b. African-Americans
   c. Caucasians
   d. No difference by ethnicity

5. Alterations in the anatomy of the pulp space occur because of which of the following?
   a. resorption
   b. age
   c. calcifications
   d. all of the above

6. Calcifications encountered in the pulp space do which of the following?
   a. represent additional dentin formation
   b. can always be detected by radiograph
   c. are always attached to the chamber or canal walls
   d. often prevent instruments from negotiating canals

7. Which of the following is *not* associated with the radicular pulp?
   a. lateral canals
   b. apical foramen
   c. pulp horns
   d. canal orifices

8. Accessory canals are more common in the apical third, and more common in posterior teeth.
   a. The entire sentence is true.
   b. The first statement is true, the second is false.
   c. The first statement is false, the second is true.
   d. The entire sentence is false.

9. Which of the following are true regarding the apical foramen?
   a. The diameter remains constant throughout life.
   b. The position of the apical foramen is often visible on radiograph.
   c. The foramen is most commonly located 0.5 mm to 1.0 mm away from the anatomic root apex.
   d. None of the above.

10. Dens invaginatus (dens in dente) occurs most commonly in which teeth?
    a. maxillary canines
    b. maxillary lateral incisors
    c. maxillary and mandibular lateral incisors
    d. mandibular first premolars

11. The lingual groove defect is (1) found most frequently in maxillary central incisors and (2) has a poor prognosis for treatment.
    a. Statements 1 and 2 are true.
    b. Statement 1 is true, statement 2 is false.

c. Statement 1 is false, statement 2 is true.
d. Statements 1 and 2 are false.

12. A C-shaped canal is characterized by which of the following?
    a. has complex internal anatomy
    b. is most commonly found in Asian populations
    c. usually occurs in mandibular second molars
    d. should be referred to an endodontist for treatment
    e. all of the above

13. Of the following, which tooth or root is the most likely to have two canals?
    a. Maxillary second premolar
    b. Mandibular first molar mesial root
    c. Mandibular lateral incisor
    d. Maxillary first molar mesiobuccal root

14. The lingual root of the maxillary first molar often has a curvature in the apical third to which of the following?
    a. buccal
    b. lingual
    c. mesial
    d. distal
    e. none of the above, the root is usually straight

CHAPTER 14

Isolation, Endodontic Access, and Length Determination

1. What is true of the rubber dam?
   a. The rubber dam is elective for endodontic treatment.
   b. The rubber dam allows irrigating solution to contact and disinfect surrounding soft tissues.
   c. The rubber dam protects the patient from swallowing or aspirating instruments and materials.
   d. The rubber dam injures soft tissue from the pressure of the clamp.

2. What is the recommended rubber dam weight for endodontic procedures?
   a. light
   b. medium
   c. heavy
   d. extra heavy

3. Why are plastic rubber dam frames recommended over metal frames?
   a. They are radiolucent.
   b. They are easier to remove during exposure of interim radiographs.
   c. They are more comfortable for the patient.
   d. They are easier to place.

4. Which of the following clamps is designed for an anterior tooth?
   a. No. 8
   b. No. 212
   c. No. 0
   d. No. 24/25

5. What is an advantage of a provisional crown used to replace missing tooth structure before root canal treatment?
   a. It accurately reproduces tooth anatomic landmarks.
   b. It maintains tooth orientation for access and canal location.
   c. It is easily removed and replaced during root canal treatment appointments.
   d. It increases visibility of the root canal chamber.

6. What is the preferred method for rubber dam placement on molars?
   a. placement as a unit
   b. placement of a clamp and rubber dam, followed by attachment of the frame
   c. placement of a clamp, followed by the dam and then the frame
   d. placement of the rubber dam and frame, followed by placement of the clamp

7. What is a *major* objective of the access opening?
   a. Locate the primary or largest canal.
   b. Achieve unimpeded straight-line access of the instruments to the first canal curvature or apical one third.
   c. Expose the pulp horns.
   d. Remove all restorative materials.

8. Outline form for access is described best by which of the following?
   a. It mimics the shape of the canal or canals.
   b. It is toward the distal in the occlusal surface in molars.
   c. It is a projection of the internal tooth anatomy onto the external surface.
   d. It is a constant and unchanging shape regardless of age.

9. What is an advantage of caries removal during access?
   a. It enhances the effectiveness of NaOCl.
   b. It reduces interappointment pain.
   c. It strengthens tooth structure.
   d. It allows assessment of the restorability prior to the endodontic treatment.

10. Estimated depth of access is a measurement from which of the following?
    a. incisal edge of anterior teeth to the coronal portion of the pulp chamber
    b. occlusal reference of posterior teeth to the coronal portion of the pulp chamber
    c. incisal edge of anterior teeth to the radiographic apex of the tooth
    d. occlusal reference of posterior teeth to the radiographic floor of the chamber

11. What is the shape of the access opening of a maxillary central incisor in a young patient?
    a. round
    b. triangular

c. trapezoidal
d. square

12. What is the outline shape of the access for a maxillary first molar?
a. round
b. triangular
c. trapezoidal
d. square

13. What is the outline shape of the access for a mandibular first molar with four canals?
a. round
b. triangular
c. trapezoidal
d. square

14. To obtain an accurate measurement, how should the working length radiographs be made?
a. They should be made with a loosely fitting file in place.
b. They should be made with a minimum of a No. 20 file.
c. They should be made with a positioning device and a parallel technique.
d. They should be made with the rubber dam removed for visibility and access.

15. An apex locator is helpful in patients with which of the following?
a. with high palatal vaults
b. with implanted cardiac pacemakers
c. with missed canals
d. with a strong gag reflex

CHAPTER 15

Cleaning and Shaping

1. What is the preferred method to evaluate if a canal has been adequately cleaned?
   a. The canal is three file sizes larger than the initial master apical file.
   b. The canal walls are “glassy smooth” when explored with a file.
   c. Dentin shavings obtained are clean and white.
   d. Irrigant runs clear with no visible debris.

2. The degree of canal enlargement during shaping is dictated by which of the following?
   a. method of obturation
   b. anatomy of the root
   c. plan for post placement
   d. all of the above

3. The apical termination point for cleaning and shaping the root canal should be which of the following?
   a. the radiographic apex
   b. at the major diameter of the apical foramen
   c. within 0 to 2 mm of the radiographic apex
   d. 0.5 mm beyond the radiographic apex

4. To prevent extrusion of obturating material, cleaning and shaping procedures must be confined to the radicular space. Canals filled to the radiographic apex would be considered to be the perfect result.
   a. Both statements are true.
   b. The first statement is true; the second statement is false.
   c. The first statement is false; the second statement is true.
   d. Both statements are false.

5. For the irrigating solution to effectively reach the apical third of the canal, the apical canal should be enlarged to at least a No. _____ file.
   a. 25 or 30
   b. 20 or 25
   c. 35 or 40
   d. 45 or 50

6. Which of the following is the most widely used irrigant solution?
   a. sodium hypochlorite
   b. ethylenediaminetetraacetic acid (EDTA)
   c. MTAD
   d. saline

7. The best description of a difference between nickel-titanium and stainless steel instruments is which of the following?
   a. Nickel-titanium tends to result in better shaping (less transportation) in curved canals.
   b. Nickel-titanium usually results in better debridement.
   c. Nickel-titanium can usually be reused many more times than stainless steel.
   d. Nickel-titanium has sharper cutting edges.

8. What is the primary purpose of an irrigant such as sodium hypochlorite (NaOCl)?
   a. kill bacteria
   b. dissolve tissue remnants
   c. flush out debris
   d. lubricate instruments

9. A major advantage to using a lubricant during cleaning and shaping is:
   a. It ensures that canal transportation will not occur.
   b. It reduces torsional force on the instrument, decreasing the possibility of fracture.
   c. It minimizes debris production.
   d. It reduces operator fatigue.

10. Removal of the smear layer after cleaning and shaping does which of the following?
   a. promotes coronal leakage
   b. decreases dentin permeability
   c. allows better adaptation of obturating materials to canal walls
   d. forces bacteria into dentinal tubules

11. EDTA is most effective for which of the following uses?
   a. decalcifying small canals to allow instruments to negotiate to length

b. lubricating canals to facilitate instrumentation
c. bacterial elimination in canals
d. removing smear layer after cleaning and shaping

12. How does the “crown-down” technique differ from the “step-back” technique?
a. It creates a funnel-shaped preparation.
b. It facilitates tissue removal.
c. It requires fewer instruments.
d. It creates coronal flare early, reducing torsional stress on the instruments.

13. Recapitulation is defined as:
a. The removal of accumulated debris using a small file at the corrected working length.
b. Confirmation of working length after completing cleaning and shaping.
c. The last irrigation before drying the canal.
d. Verifying the master apical file after cleaning and shaping.

14. Of the following, which is the most important consideration of a temporary restoration?
a. antimicrobial
b. placed over a cotton pellet
c. resistant to acids
d. at least 4 mm thick

CHAPTER 16

Preparation for Restoration

1. What is the leading cause for loss of endodontically treated teeth?
   a. inadequate cleaning and shaping of the canals
   b. inadequate obturation of the root canal system
   c. restorative factors
   d. vertical root fracture

2. How does the survival rate for a tooth restored with cusp protection compare to a tooth without cusp protection?
   a. about the same
   b. a tooth with protected cusps has an enhanced survival rate
   c. a tooth without protected cusps has an enhanced survival rate
   d. restorations have no effect on tooth survival rates

3. Dentin becomes more brittle following the endodontic treatment due to loss of moisture content.
   a. True
   b. False

4. The greatest contributing factor to reduced cuspal stiffness (strength) that predisposes to fracture is which of the following?
   a. occlusal access opening
   b. loss of one or both marginal ridges
   c. an amalgam restoration placed after root canal treatment
   d. a bonded composite restoration placed after root canal treatment

5. Which of the following describes a definitive restoration after root canal treatment?
   a. It should be placed at the time of obturation.
   b. It should allow cuspal flexure to absorb occlusal forces.
   c. It should provide a coronal seal.
   d. It should always be a full-coverage crown on posterior teeth.

6. Exposure of obturating materials to oral fluids can be described by which of the following?
   a. It is not a factor if a sealer is properly used during obturation.
   b. It is a major cause of failure.
   c. It leads to rapid failure.
   d. It may cause pain to thermal changes.

7. Which of the following describes a definitive restoration?
   a. It should be placed as soon as practical.
   b. It should be placed at the 6-month recall visit to assure symptoms do not recur.
   c. It should be placed when radiographic evidence of healing becomes evident.
   d. It should be delayed if there is a questionable prognosis.

8. What is the only reason to delay the definitive restoration?
   a. to maximize the patient's insurance benefits
   b. if the patient is unable to pay for the restoration
   c. to allow radiographic healing to become evident
   d. if there is a questionable prognosis and failure would lead to extraction

9. The practical principles for function and durability when designing a definitive restoration include the following *except* which of the following?
   a. conservation of tooth structure
   b. retention
   c. placement of a post
   d. protection of the remaining tooth structure

10. What is an indication for use of a direct (amalgam or composite) restoration?
   a. Excessive tooth structure has not been lost.
   b. The opposing arch has been restored with full coverage crowns.
   c. Esthetics is not a concern.
   d. Only one of the marginal ridges has been lost.

11. Fewer root fractures have been recorded in laboratory studies when what type of post has been used?
   a. cobalt chromium alloy post
   b. stainless steel post
   c. titanium post
   d. carbon fiber post

12. Which of the following describes removal of gutta-percha for post space preparation?
    a. immediately after obturation
    b. after the sealer has completely set
    c. to a depth that allows 2 to 3 mm of remaining gutta-percha
    d. completed using solvents

13. When using a prefabricated post system to restore a posterior tooth, the most desirable post design is
    a. tapered, passively cemented.
    b. tapered, threaded screw type.
    c. parallel sided, passively cemented.
    d. parallel sided, threaded screw type.

14. Which of the following describes retentive pins?
    a. Retentive pins help strengthen the restoration.
    b. Retentive pins minimize stresses to dentin.
    c. Retentive pins are the most effective antirotation method for post and cores.
    d. Retentive pins should not be used because the risks outweigh any potential gain.

15. Placement of a dowel or post through a crown or an existing restoration is described by which of the following?
    a. It adds support for the existing restoration.
    b. It helps maintain integrity of the existing restoration.
    c. It improves the seal of the root canal.
    d. It is rarely indicated.

CHAPTER 17

Obturation

1. What is a possible outcome when there is an overfill of the obturation materials?
   a. decreased periapical inflammation
   b. improved and rapid healing of periapical lesions
   c. inadequate apical seal
   d. decreased postobturation discomfort

2. What is the optimal preparation/obturation length relative to the radiographic apex with pulp necrosis?
   a. flush
   b. 0.5 to 1.0 mm short
   c. slight extrusion of sealer but not gutta-percha
   d. 1.0 to 3.0 mm short

3. Prognosis and histologic studies show that if there is a length error, there are less problems resulting with which of the following?
   a. overfills
   b. underfills

4. Which of the following describes lateral canals?
   a. They connect adjacent canals within the same root.
   b. They may allow bacterial and necrotic debris to leak into the periodontium.
   c. They are débrided with copious irrigation.
   d. They are significant in the outcome of most root canal treatments.

5. What factors should be considered when deciding the timing for obturation?
   a. signs and symptoms present
   b. pulp and periapical status
   c. difficulty of the procedure
   d. all of the above

6. What pulp and periapical diagnosis may have completed treatment in a single visit?
   a. symptomatic (acute) apical periodontitis
   b. asymptomatic apical periodontitis (chronic apical periodontitis)
   c. acute apical abscess
   d. painful irreversible pulpitis

7. What material is currently the only universally accepted solid core obturation material?
   a. gutta-percha
   b. synthetic polyester resin-based polymers
   c. silver points
   d. solid core (carrier) gutta-percha

8. Which of the following is a disadvantage of guttapercha?
   a. poor adaptation to canal irregularities with compaction
   b. shrinkage if altered by heat or solvents
   c. not easily managed and manipulated
   d. difficult to partially remove from a canal

9. Which of the following is an advantage of guttapercha?
   a. adhesiveness to dentin
   b. slight elasticity and rebound effect
   c. expansion on cooling of warmed gutta-percha
   d. adaptation to canal irregularities with compaction

10. What have recent studies shown synthetic polyester resin-based polymers to be?
    a. adhesive to canal walls throughout their length
    b. inflammatory to tissues
    c. mutagenic
    d. no difference in leakage when compared to guttapercha

11. Which of the following have semisolid obturation materials (pastes or cements) been shown to do?
    a. They provide easy control of obturation length.
    b. They exhibit no shrinkage upon setting.
    c. They provide an unpredictable and inconsistent apical seal.
    d. They are biocompatible and nonirritating to periapical tissues.

12. Which of the following describes the complete setting of zinc oxide–eugenol (ZnOE)-based sealers?
    a. The setting occurs at approximately 1 hour if left exposed to air.
    b. The setting depends on contact with dentin.
    c. The setting usually requires weeks or months.
    d. The setting usually is approximately 24 hours.

13. What type of sealer may have a problem with long-term solubility?
    a. ZnOE-based sealers
    b. plastic sealers
    c. glass ionomer sealers
    d. calcium hydroxide sealers

14. Which of the following describes lateral compaction of gutta-percha?
    a. It is indicated for cases with internal resorption.
    b. It involves multiple steps and special armamentarium.
    c. It manages length control well.
    d. It is difficult to retreat.

15. What is a disadvantage of finger spreaders as compared to standard long-handled spreaders?
    a. tactile sensation
    b. instrument length control
    c. fracture potential in curved canals
    d. dentin stress during obturation

CHAPTER 18

Procedural Accidents

1. What should a patient *not* be told when there has been a procedural accident?
   a. the incident did occur
   b. the possible procedures that may be necessary for correction
   c. how this might affect the outcome
   d. the root canal treatment will be completed free of charge

2. Which of the following is *not* a common cause of a perforation during access preparation?
   a. mandibular molar with a lingual axial inclination of the tooth
   b. searching for canals through an under-prepared access opening
   c. directing the bur parallel to the long axis of the tooth
   d. presence of a misaligned cast restoration

3. What measures are used to prevent perforation during access preparation?
   a. relating tooth angulations independent of the adjacent tooth
   b. using only straight-on radiographs
   c. always having a rubber dam in place before beginning access preparation
   d. having a thorough knowledge of both surface and internal tooth anatomy

4. Which of the following is *not* an early sign or indication of a perforation?
   a. pain during access preparation
   b. sudden appearance of hemorrhage
   c. burning pain and bad taste during irrigation with NaOCl
   d. radiographically malpositioned file

5. If a lateral root perforation does occur, what is the most favorable location for perforation repair?
   a. at or above the height of crestal bone
   b. below the crestal bone in the coronal third of the root
   c. on the furcal side of the coronal root surface
   d. a zipping perforation at the apex of the root

6. What is the ideal time and material for a nonsurgical repair of a furcation perforation?
   a. immediate repair with amalgam
   b. immediate repair with mineral trioxide aggregate (MTA)
   c. delayed repair with amalgam
   d. delayed repair with MTA

7. What is a common cause of ledge formation?
   a. straight-line access into the canal
   b. excess irrigating solution
   c. overenlargement of a curved canal with files
   d. constant recapitulation and irrigation into the apical portion of the canal

8. What type of canal is most prone to ledge formation?
   a. long, small, and curved
   b. incomplete apex formation, curved
   c. large, long
   d. short, straight

9. What is a possible etiology for an apical (zipping) root perforation?
   a. inability to negotiate canals with ledges
   b. working length determination with radiographs only
   c. trying to locate canals in a small chamber
   d. failure to adjust working length after curved canals are straightened during cleaning and shaping

10. What type of perforation has the poorest long-term prognosis?
    a. zipping (apical third) root perforation
    b. stripping perforation in the apical third of the root
    c. stripping perforation in the coronal third of the root below the crest of bone
    d. direct floor to furcation perforation in a multi-rooted tooth

11. Which of the following is *not* a common cause of file separation?
    a. limited flexibility
    b. manufacturing defects

c. amount of use
d. amount of force applied

12. What approaches may be used to treat a case with a separated instrument?
a. attempt to remove
b. attempt to bypass
c. prepare and obturate to the level of the segment
d. all of the above

13. Which of the following scenarios yields the most favorable prognosis in cases with a separated instrument?
a. a small instrument short of the working length
b. a small instrument beyond the apical foramen
c. a large instrument at an early stage of instrumentation
d. a large instrument close to the working length

14. Which of the following causes extrusion of sodium hypochlorite (NaOCl) irrigating solution into periapical tissues?
a. Fitting irrigation needle loosely in the canal space.
b. Wedging irrigation needle in the canal space.
c. Using perforated needles during irrigation.
d. Using regular needles during irrigation.

15. Which of the following occurs after minor extrusion of obturation materials into the periapical tissue?
a. It results in significant swelling
b. It results in significant symptoms.
c. It causes some tissue inflammation.
d. It causes more apical leakage.

CHAPTER 19

Nonsurgical Retreatment

1. Nonsurgical retreatment should be the first treatment option for correction when
   a. A large well-fitting post and core is present.
   b. There is a separated instrument present that cannot be retrieved.
   c. External resorptive root defects are present.
   d. A negotiable canal was not initially treated.

2. Which of the following is *not* a potential contraindication for nonsurgical root canal retreatment?
   a. post and core restorations
   b. ledges in the root canal walls
   c. amalgam core restorations into the chamber
   d. separated root canal instruments

3. Which of the following is *not* a potential risk associated with nonsurgical root canal retreatment?
   a. thinning and weakening of the root canal walls
   b. inability to remove the initial root canal obturation material
   c. creating a compromised crown-root ratio
   d. loosening of a well-fitting fabricated crown

4. Which of the following describes removal of coronal restorations before nonsurgical retreatment?
   a. Removal may prolong retreatment procedures.
   b. Removal complicates removal of post and core restorations.
   c. Removal may be necessary to assess restorability.
   d. Removal should never be done if the previous restoration is a full-coverage crown.

5. What steps are involved in retrieval of a prefabricated post during retreatment?
   a. Section and remove the core material and the post at the level of the chamber floor.
   b. An ultrasonic activated tip is used for 3 to 5 minutes each at several locations circumferentially around the post, without water for visibility.
   c. Grasp the post with a hemostat or Steiglitz pliers to rock it back and forth to break the cement seal.
   d. Use ultrasonics, then grasp a threaded screw type post with a hemostat or small-tipped forceps to unscrew it from the canal.

6. The following steps are used to attempt removal of a canal ledge during nonsurgical retreatment *except* which one?
   a. remove all obstructions coronal to the ledge
   b. bypass the ledge with a flexible nickel-titanium hand file
   c. file in a circumferential motion after bypassing the ledge
   d. proceed from small-size files to larger size files

7. Which of the following is *not* a factor that may affect the successful removal of a separated instrument fragment?
   a. size of the instrument separated
   b. length of the separated fragment
   c. location of the fragment within the canal
   d. length of time the fragment has been in place

8. What method or methods have been used to successfully remove gutta-percha from root canals?
   a. heat
   b. ultrasonics
   c. rotary instruments
   d. all of the above

9. When should Hedstrom hand files or regular hand reamers be the instruments of choice for gutta-percha removal, without the addition of solvents?
   a. The root canal is well sealed with gutta-percha.
   b. The gutta-percha is well adapted to canal walls.
   c. A space can be created between the gutta-percha and the root canal.
   d. Gutta-percha fills the root canal chamber.

10. What solvent has been shown to be the most efficient (fastest) in softening gutta-percha?
   a. chloroform
   b. halothane
   c. methylchloroform
   d. xylene

11. During the removal of a carrier-based gutta-percha obturator which of the following should occur?
   a. The first step is to remove the solid core material.

b. A combination of techniques for removal of gutta-percha, silver cones, and posts is employed.
c. A small rotary file may be used to engage and remove the plastic carrier.
d. Different solvents are used that would routinely be used to remove gutta-percha alone.

12. What is the key to success in the retrieval of silver points?
a. The key is to engage the silver point with the ultrasonic tip.
b. The key is to remove the silver point and core material simultaneously.
c. The key is to retain as much of the coronal extent of the point as possible.
d. The key is to remove the core material and silver point to the level of canal orifices first.

13. Which of the following is *not* true regarding the removal and retreatment of hard-setting pastes?
a. They are more difficult to remove than a soft paste.
b. They may be impossible to remove.
c. Solvents have been shown to soften hard-setting pastes.
d. Use of ultrasonics is the most predictable method.

14. Which of the following is true regarding interappointment flare-ups with nonsurgical root canal retreatments?
a. Flare-ups occur less frequently when compared to initial root canal treatments.
b. Flare-ups occur frequently, even when debris and microorganisms are confined to the canals.
c. Flare-ups occur less frequently if irrigation is kept to a minimum.
d. High incidence dictates that retreatment should generally be treated in two visits rather than one visit.

15. The prognosis for nonsurgical root canal retreatment is which of the following?
a. It is increased with periapical lesions.
b. It has the lowest rate of success without a periapical lesion.
c. It is similar to initial root canal treatment success rates.
d. It is best if the etiology of failure can be identified.

CHAPTER 20

Endodontic Surgery

1. What is the purpose of incision for drainage?
   a. to evacuate exudates from a soft tissue swelling
   b. to obtain a biopsy specimen
   c. to prevent a postoperative swelling
   d. to avoid emergency cleaning and shaping

2. Profound anesthesia is difficult to attain before incision for drainage. What is a preferred approach for a maxillary cuspid with extensive swelling?
   a. Start with an infraorbital block and then infiltrate at the margins of the swelling.
   b. Start with posterior superior alveolar block and then use refrigerant spray.
   c. Inject buffer and anesthetic directly into the swelling.
   d. Use topical anesthetic and then refrigerant spray. No anesthetic is needed.

3. Which of the following is *not* an indication for periapical surgery?
   a. a nonnegotiable or blocked canal associated with symptomatic periradicular pathosis
   b. gross overextension of obturating material
   c. obtain a biopsy
   d. to resolve any endodontic treatment failure

4. Which of the following would contraindicate periapical surgery?
   1. anatomical structures in the area
   2. medical complications
   3. lip paresthesia
   4. previous malignancies
   5. unidentified cause of treatment failure
      a. 1, 2, and 3
      b. 1, 3, and 5
      c. 1, 2, and 5
      d. 2, 3, and 4
      e. all of the above

5. Which of the following is true regarding an incision over a bony defect?
   a. It should be avoided.
   b. It may cause a postsurgical fenestration.
   c. It may prevent healing of the incision.
   d. All of the above.

6. Which of the following describes a submarginal flap design?
   a. It is ideal for mandibular posterior teeth.
   b. It causes less scarring.
   c. It is associated with less gingival recession.
   d. It causes less intraoperative hemorrhage.

7. What is the purpose of root end resection?
   a. removes irritants encased in the apical portion of the root
   b. to examine the root
   c. exposes additional canals or fractures
   d. all of the above

8. Which of the following is true regarding a root-end cavity preparation?
   a. It should be as shallow as possible to preserve tooth structure.
   b. It should be made to a minimum depth of 3 mm.
   c. It should only encompass the main portion of the canal.
   d. It should be made with a very small bur.

9. An ideal root-end filling material should satisfy all of the following *except* which one?
   a. The material should be well tolerated by periradicular tissues.
   b. The material should be easily placed.
   c. The material should be absorbable.
   d. The material should be visible radiographically.

10. Which of the following cell types are important in the healing process after periapical surgery?
   1. epithelial cells

2. macrophages
3. dendritic cells
4. fibroblasts
5. osteocytes
    a. 1, 2, 4, and 5 only
    b. 1 and 2 only
    c. 1, 2, and 5 only
    d. 1, 2, 3, 4, and 5
    e. 1, 3, and 4 only

11. With root amputation, the factor that most affects success is which of the following?
    a. occlusal force patterns
    b. the type of restoration
    c. the length of the root
    d. the patient's oral hygiene

12. All of the following procedures should be referred to a specialist with specific training in endodontic surgery *except* which one?
    a. root-end resection/root-end filling
    b. incision for drainage
    c. root amputation
    d. perforation repair

CHAPTER 21

Evaluation of Endodontic Outcomes

1. What is the primary determinant for successful endodontic treatment?
   a. selecting the proper obturation technique
   b. effective elimination of microorganisms from the pulp space
   c. using rotary instruments to shape the canals
   d. using an effective irrigation regimen

2. What are the major indicators of successful endodontic treatment?
   a. lack of discoloration, no tenderness to biting
   b. no swelling or redness of the gingival area
   c. absence of symptoms and apical radiolucency
   d. a happy patient who has paid the bill

3. A patient presents for a posttreatment examination with no complaint of symptoms except the apical radiolucency that was present before treatment, although it appears smaller. Treatment for this patient would be classified as which of the following?
   a. a failure
   b. a success
   c. a clinical success but radiographic failure
   d. a functional tooth with uncertain prognosis

4. Which of the following is not a clinical criterion to evaluate treatment outcome?
   a. absence of a radiolucency
   b. no evidence of a sinus tract
   c. no swelling present
   d. no response to percussion or palpation

5. To make valid comparisons between radiographs to assess healing, which of the following describes how films should be made?
   a. in a reproducible manner
   b. 6 months apart
   c. at different angles
   d. by the same person to ensure consistency

6. Which of the following criteria is *not* considered to be a predictor of success or failure?
   a. the patient's medical history
   b. apical pathosis
   c. quality of the coronal restoration
   d. extent and quality of obturation

7. The most common preoperative cause of endodontic treatment failure includes all of the following *except* which one?
   a. misdiagnosis
   b. leaking coronal restoration
   c. poor case selection
   d. error in treatment planning

8. The most common postoperative cause of endodontic treatment failure is which of the following?
   a. overextension of obturating material
   b. a separated instrument
   c. coronal leakage
   d. placement of a post unnecessarily

9. The prognosis for nonsurgical retreatment depends primarily on which of the following?
   a. identification and correction of the cause of failure
   b. using a different obturation technique
   c. placing the definitive restoration at the obturation appointment
   d. all of the above

CHAPTER 22

Bleaching Discolored Teeth: Internal and External

1. Which of the following is *not* considered a natural or acquired discoloration source?
   a. tetracycline stain
   b. intrapulpal hemorrhage
   c. stain from amalgam
   d. calcific metamorphosis

2. Which of the following is *not* part of the mechanism of staining caused by fluorosis?
   a. Hypoplastic defects are produced in enamel by excess fluoride.
   b. Stain is acquired from chemicals in the oral cavity.
   c. Stain is present in the enamel.
   d. Stain is solely caused by fluoride deposits in the enamel.

3. Which of the following describes tetracycline stain?
   a. It is classified into three groups based on severity.
   b. It is often associated with a “banding” pattern.
   c. It is located in the dentin.
   d. All of the above.

4. What is the most common iatrogenic etiology related to tooth discoloration?
   a. incomplete removal of pulp tissue
   b. incomplete removal of obturating material from the chamber
   c. the use of intracanal medicaments
   d. intracanal irrigants

5. Which of the following restorative materials can contribute to staining?
   a. amalgam
   b. pins and posts
   c. composite
   d. all of the above

6. What is the most common agent used for internal bleaching?
   a. carbamide peroxide
   b. sodium perborate
   c. hydrogen peroxide
   d. sodium peroxyborate monohydrate

7. Which of the following is *not* an indication for internal bleaching?
   a. defective enamel formation
   b. intrapulpal hemorrhage-induced stain
   c. tetracycline-induced stain
   d. sealer stain

8. One potential complication of internal bleaching is external root resorption, which has been associated with which of the following?
   a. high concentration of hydrogen peroxide
   b. heat
   c. damage to cementum and periodontal tissues
   d. all of the above

9. What is the most common agent employed in external bleaching?
   a. sodium perborate
   b. hydrochloric acid
   c. carbamide peroxide
   d. sodium hypochlorite

10. Which of the following describes the microabrasion technique?
   a. It is not a true bleaching technique.
   b. It uses hydrochloric acid.
   c. It requires meticulous soft tissue isolation.
   d. All of the above.

CHAPTER 23

Geriatric Endodontics

1. Which of the following are changes that occur in the pulp with age?
    1. decreased vascular elements
    2. decreased amount of collagen
    3. increase in numbers of fibroblasts
    4. decrease in numbers of odontoblasts
    5. increase in occurrence of calcifications
        a. 1, 2, and 3 only
        b. 1, 3, and 5 only
        c. 1, 4, and 5 only
        d. 2, 3, and 5 only
        e. 1, 2, 3, 4, and 5

2. Which of the following statements are true regarding calcifications in the pulp space?
    a. Pulp stones are usually found in the radicular pulp.
    b. Pulp stones can increase the incidence of odontogenic pain.
    c. Calcifications increase with both age and irritation.
    d. Diffuse calcifications are most commonly found in the pulp chamber.
    e. All of the above.

3. With age, which of the following describes the pulp chamber in molars?
    a. decreases primarily in a mesiodistal dimension
    b. decreases primarily in an occlusal-apical dimension
    c. remains the same in volume
    d. increases in size in response to irritation

4. The healing capacity of older patients is significantly less than younger patients because of a decrease in periradicular vascularity. The vascularity of the periradicular tissues is a critical determinant in healing.
    a. The first statement is false, and the second statement is true.
    b. The first statement is true, and the second statement is false.
    c. Both statements are true.
    d. Both statements are false.

5. Which of the following medical conditions may influence healing in geriatric patients?
    a. osteoporosis
    b. hypertension
    c. immunosuppression
    d. diabetes

6. Which of the following describes bisphosphonates?
    a. They are used in treatment of osteoporosis.
    b. They are used in treatment of malignancies.
    c. They are linked to idiopathic osteonecrosis of the jaws.
    d. They interfere with osteoclast function.
    e. All of the above.

7. Which of the following is a common finding on examination in geriatric patients?
    a. extensive restorative experience with multiple large restorations and crowns
    b. lower incidence of periodontal disease
    c. exaggerated symptoms associated with pulp pathosis
    d. excessive salivation

8. Which of the following is a consideration between geriatric and younger patients that may affect the ability to make a diagnosis?
    a. Older patients are more stoic.
    b. Decreased response to pulp testing is common.
    c. Symptoms of pulpitis are not as acute in older patients.
    d. All of the above.

9. A common modification in performing root canal treatment for older patients is which of the following?
    a. treatment planning for a shorter lifespan
    b. access cavity without a rubber dam to locate a smaller chamber
    c. greater need for anesthetic
    d. larger restorations make isolation easier

10. Working length determination in elderly patients may be more difficult because of which of the following?
    a. increased bone density, making radiographs harder to interpret
    b. increased cementum deposition, modifying the apical anatomy
    c. differences in tissue electrical resistance, making apex locators less accurate
    d. patients unable to sit still for radiographs

Chapter Review Questions Answer Key

CHAPTER 1

1. b
2. d
3. b
4. b
5. d
6. c
7. c
8. b
9. c
10. a
11. d
12. d
13. a
14. a
15. d

CHAPTER 2

1. c
2. b
3. c
4. a
5. c
6. c
7. a
8. d
9. c
10. c
11. a
12. c
13. b
14. d
15. d

CHAPTER 3

1. d
2. c
3. b
4. c
5. b
6. c
7. a
8. b
9. a
10. d

CHAPTER 4

1. b
2. d
3. c
4. c
5. b
6. d
7. c
8. c
9. a
10. d
11. c
12. d
13. b
14. d
15. c

CHAPTER 5

1. d
2. c
3. b
4. c
5. a
6. a
7. b
8. b
9. d
10. b
11. b
12. a
13. b
14. c
15. a

CHAPTER 6

1. d
2. a
3. d
4. c
5. c
6. a
7. c
8. b
9. a
10. c
11. a
12. a
13. a
14. d

CHAPTER 7

1. d
2. c
3. a
4. c
5. d
6. d
7. c
8. d
9. c
10. b
11. d
12. b

CHAPTER 8

1. b
2. c
3. c
4. c
5. c
6. d
7. a
8. c
9. d
10. d
11. c
12. a
13. b
14. a
15. b
16. d

CHAPTER 9

1. a
2. a
3. c
4. a
5. c
6. c
7. c
8. d
9. c
10. d
11. b
12. c
13. b

CHAPTER 10

1. b
2. c
3. c
4. c
5. d
6. d
7. c
8. b
9. d
10. c
11. d
12. c
13. b
14. d

CHAPTER 11

1. d
2. d
3. c
4. c
5. b
6. b
7. b
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