ENDODONTICS REVIEW

A STUDY GUIDE

PERFECT FOR BOARD REVIEW

Brooke Blicher, DMD
Rebekah Lucier Pryles, DMD
Jarshen Lin, DDS

quintessence books

HIGH DENT

PRODUCTS SERVICES TRAINING

همیار دندانسازان و دندانپزشکان در پروتز

مرکز تخصصی پروتزهای دندانی

های دنت

طراحی و ساخت انواع پروتزهای دندانی بویژه ایمپلنت

برگزار کننده دوره های آموزشی تخصصی و جامع دندانسازی و...

با ما همراه باشید...

WWW.HIGHDENT.IR

@highdent

@highdent

Endodontics Review: *A Study Guide*

ENDODONTICS REVIEW A STUDY GUIDE

Brooke Blicher, DMD
Private Practice Limited to Endodontics
White River Junction, Vermont

Clinical Instructor, Department of Restorative
Dentistry and Biomaterials Sciences
Harvard School of Dental Medicine
Boston, Massachusetts

Assistant Clinical Professor, Department of Endodontics
Tufts University School of Dental Medicine
Boston, Massachusetts

Instructor in Surgery
Dartmouth Medical School
Hanover, New Hampshire

Rebekah Lucier Pryles, DMD
Private Practice Limited to Endodontics
White River Junction, Vermont

Assistant Clinical Professor, Department of Endodontics
Tufts University School of Dental Medicine
Boston, Massachusetts

Jarshen Lin, DDS
Director of Predoctoral Endodontics
Department of Restorative Dentistry and Biomaterials Science
Harvard School of Dental Medicine

Clinical Associate, Department of Oral and Maxillofacial Surgery
Massachusetts General Hospital
Boston, Massachusetts

quintessence books

Quintessence Publishing Co, Inc
Chicago, Berlin, Tokyo, London, Paris, Milan, Barcelona, Istanbul,
Moscow, New Delhi, Prague, São Paulo, Seoul, and Warsaw

Library of Congress Cataloging-in-Publication Data

Names: Blicher, Brooke, author. | Lucier Pryles, Rebekah, author. | Lin, Jarshen, author.
Title: Endodontics review : a study guide / Brooke Blicher, Rebekah Lucier Pryles, Jarshen Lin.
Description: Hanover Park, IL : Quintessence Publishing Co. Inc., [2016] | Includes bibliographical references.
Identifiers: LCCN 2016003847 (print) | LCCN 2016004668 (ebook) | ISBN 9780867156966 (soft cover) | ISBN 9780867157338 ()
Subjects: | MESH: Endodontics--methods | Periodontal Diseases | Dental Pulp Diseases | Review
Classification: LCC RK351 (print) | LCC RK351 (ebook) | NLM WU 230 | DDC 617.6/342--dc23
LC record available at http://lccn.loc.gov/2016003847

Quintessence Publishing Co Inc
4350 Chandler Drive
Hanover Park, IL 60133
www.quintpub.com

5 4 3 2 1

Editor: Bryn Grisham
Design: Ted Pereda
Production: Angelina Sanchez

Printed in the United States of America

Contents

Preface

Congratulations on taking the important leap toward board certification through the American Board of Endodontics (ABE). Although challenging, the path ahead is fair and rewarding, and achieving diplomate status inevitably results in improved clinical skills. For information regarding specific portions of the ABE examination, please refer to www.aae.org/board.

This text provides a comprehensive guide for both the written and oral portions of the ABE examination. Chapters are organized according to the oral examination structure, including the basic sciences, medicine, diagnosis, treatment protocols, prognosis, and complications. Given their relative complexity, the subjects of trauma and resorption are presented individually and include their own unique diagnosis and treatment protocols.

Consultation of textbooks like this one provides an important framework in preparing for the ABE examination; however, other resources are necessary for preparation. This text therefore contains several references to other textbooks considered useful in examination preparation.

Citation of specific references is essential during the written and oral portions of the ABE examination, including a vast body of endodontic literature dating back nearly a century. Throughout the text, frequent references are made to primary resources, as depth of knowledge and overall comprehension of endodontics obtained by reading such references is irreplaceable and cannot be acquired through short cuts.

Knowledge of the most up-to-date endodontic literature, American Association of Endodontics position statements, and presentations at international conferences all contribute to the examination. Readers are encouraged to pay close attention to all literature preceding their examination, including information disseminated after publication of this textbook.

1

Evidence-Based Dentistry

In its quest to certify endodontists with the highest levels of knowledge, the American Board of Endodontics (ABE) examination process focuses on the practice of evidence-based dentistry, wherein the provider makes treatment decisions based on a comprehensive and constantly evolving evaluation of the bodies of research and literature in their field. Practitioners must sift through the available resources with a discerning eye.

In each section of the ABE examination, candidates must demonstrate their ability to justify their decisions and recommendations based on the highest-quality evidence available. Research published in peer-reviewed journals is considered to be unbiased and therefore most useful. Although textbooks and lectures are effective means of disseminating information, quality versions of these resources will refer back to primary resources in peer-reviewed journals. Consequently, it is imperative that providers familiarize themselves with the primary references cited in all examples. This chapter will cover study design, measures of statistical significance and validity, and epidemiology. For a more in-depth review of research design and biostatistics, please refer to Hulley et al's *Designing Clinical Research* or Glaser's *High-Yield Biostatistics*.

Study Design

Beyond citing peer-reviewed journals as the ideal reference source, certain study designs are generally considered more scientifically sound. The Oxford Centre for Evidence-Based Medicine (OCEBM) outlines a hierarchy of levels of evidence by study design, illustrated in Fig 1-1.

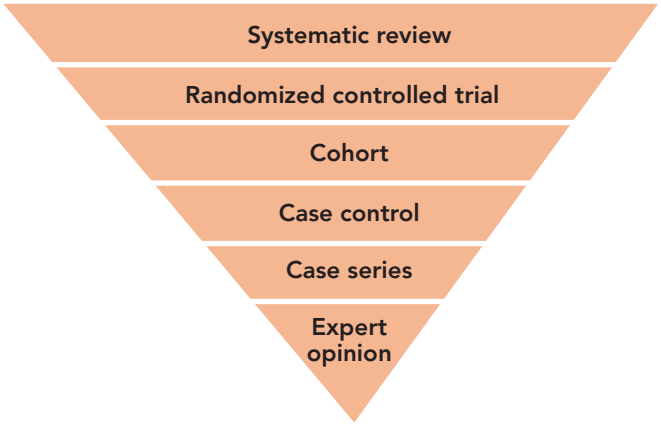

Fig 1-1 OCEBM hierarchy of levels of evidence by study design.

Systematic reviews, including meta-analyses, are considered the highest level of evidence, and their quality improves based on the compiled levels of evidence of the studies reviewed. Systematic reviews involve a comprehensive search and review of all of the literature on a topic, and a meta-analysis delves deeper by doing statistical analyses to make direct comparisons between studies. Depending on the variability of the statistics reported in the literature available on a topic, a meta-analysis may not be achievable. Systematic reviews and meta-analyses are limited by the quality of the studies included, and the following discussion of levels of evidence should be taken into account in evaluation of the quality of literature reviews.

Looking next to clinical research studies, randomized controlled trials are considered the highest level of evidence (OCEBM). Randomized controlled trials involve a planned intervention on a diseased population with matched controls. These studies are both resource- and time-intensive and are consequently difficult to perform. Futhermore, ethical concerns often arise in the discussion of this study type. Prior knowledge of superior intervention outcomes cannot be denied from a diseased population, and it is considered unethical to study certain populations, such as children or the disabled.

Cohort studies are considered next best among the levels of evidence hierarchy (OCEBM). Cohort studies are prospective and longitudinal and measure for the incidence of new cases of a disease while assessing risk or protective factors. These types of studies can be resource intensive and are not practical for rare outcomes.

Case-control studies follow cohort studies in the OCEBM hierarchy. This type of study compares past risk factors and exposures of cases with disease and controls without disease in a retrospective fashion. These studies are often less expensive to perform and less time intensive and can be useful to study rare outcomes. They are considered lower quality due to recall bias, difficulties with misdiagnosis, and assignment of controls.

Publications of case series or case reports represent the lowest level of evidence for observational studies (OCEBM). They involve a simple presentation of an outcome without provision of a control.

Lastly, expert opinions offer the lowest level of evidence. Their utility is limited in the justification of evidence-based diagnosis and treatment. Rather, they serve to introduce innovation and new techniques as clinical empiricism is oftentimes the starting point for further higher-level research.

Statistics

Although a comprehensive review of biostatistics will not be addressed in this textbook, a review of the more commonly encountered concepts in biostatistics, particularly those encountered in later parts of this text, will be presented. Readers are encouraged to seek out further resources, particularly if questions arise during the reading of primary references.

Measures of statistical significance

The ultimate goal of research is to test a hypothesis. Although absolute statements regarding proof or disproof of a hypothesis cannot be made based on limited populations and study parameters, researchers look to determine the likelihood that results support the hypothesis. Similarly, determination of cause and effect is extremely difficult to prove, requiring large-scale randomized controlled trials with longitudinal follow-ups. Most studies fall short of determining causation but can identify associations or relationships between two factors. It is important in quoting literature to never overstate results.

One way researchers can increase the odds of obtaining statistically significant results is to ensure that the sample population under study is both large and diverse enough to demonstrate outcomes. Although successful completion of the ABE examination does not require an intimate understanding of the methods researchers use to determine the adequacy of sample sizes, familiarity with the concept of power to rule out errors in hypothesis testing is imperative. Well-designed research studies involve power calculations to assure adequate sample sizes, and in critical review of literature articles, one should note if appropriate power calculations were made to justify the use of a particular sample size.

With samples selected and the experiment performed, results must be analyzed to determine their statistical relevance. The most common measure of statistical significance encountered in the endodontic literature is the *P* value. The *P* value refers to the likelihood of the outcome having occurred by chance. A *P* value less than or equal to .05 generally in-

dicates statistical significance (Fig 1-2). In other words, with a *P* value of less than .05, the probability that the study results were obtained by chance is less than 5%. For example, in a retrospective case-control study performed by Spili et al investigating the outcomes of teeth with and without fractured nickel-titanium instruments, 91.8% success was found in cases with retained fractured instruments compared with 94.5% success in controls. Statistical analysis using the Fisher exact test, a tool used to determine deviation from a null hypothesis, resulted in a *P* value of .49. This corresponds to a 49% chance that the difference in healing rates was due to chance. As the authors set the significance value at $P = .05$, the difference in healing rates obtained from the study was deemed statistically insignificant.

Fig 1-2 The relationship between *P* value and statistical significance. The *P* value describes the probability that results occurred by chance.

Measures of validity

When new testing modalities are compared to the current standard, the validity or accuracy of the new approach must be verified. Sensitivity, specificity, and predictive values provide the means by which validity can be confirmed (Fig 1-3). These values are often encountered in descriptions of pulp sensitivity tests. Jespersen et al's study on the validity of cold sensitivity testing using Endo Ice [Hygenic] provides an excellent example in the discussion of validity measures.

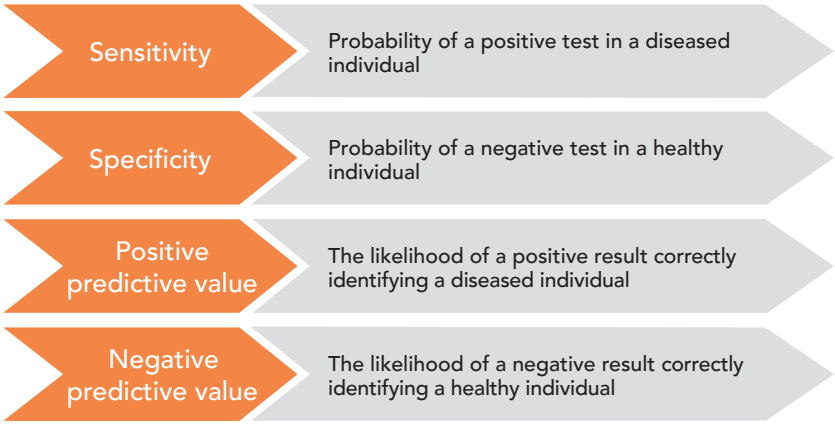

Fig 1-3 The validity measures often encountered in the endodontic literature.

Understanding validity measures requires familiarity with the concepts of both true positive and negative results and false positive and negative results (Table 1-1). True positive

Table 1-1 The possible outcomes of a test

Test result	Disease present	Disease absent
Positive	True positive	False positive
Negative	False negative	True negative

or negative results correctly identify individuals as healthy or diseased. False positive or negative results incorrectly identify the individual's disease status.

Sensitivity is defined as the ability of a test to detect diseased individuals. It is calculated by comparing the number of true positives detected by the test with the total number of diseased subjects, including the true positives plus false negatives. In Jespersen's study, the sensitivity was 0.92 for cold testing. In other words, 92% of teeth with pulpal necrosis were correctly identified.

Specificity is defined as the ability of a test to correctly identify a healthy individual. It is calculated by comparing the number of true negatives detected by the test with the total number of nondiseased subjects, including the true negatives and false positives. In Jespersen's study, the specificity was 0.90 for cold testing. In other words, the cold test correctly identified vital teeth 90% of the time.

Predictive values describe the likelihood of the test to correctly identify health or disease. The *positive predictive value* is calculated as the proportion of true positives compared with positive results. The *negative predictive* value is calculated as the proportion of true negatives compared with negative results. Jespersen reported a positive predictive value of 0.86 and a negative predictive value of 0.94 for cold testing. In other words, 86% of positive results indicated pulpal necrosis, and 94% of negative results indicated the presence of vital pulp tissue.

Epidemiology

Epidemiology involves the study of health and disease in populations. Descriptive statistics are used in epidemiology to determine the impact of health or disease measures on the population under study. Commonly reported descriptive statistics include both prevalence and incidence (Fig 1-4). *Prevalence* refers to the total number of people affected by a disease at a particular time point. *Incidence* refers to the number of new disease cases arising during a defined period of time.

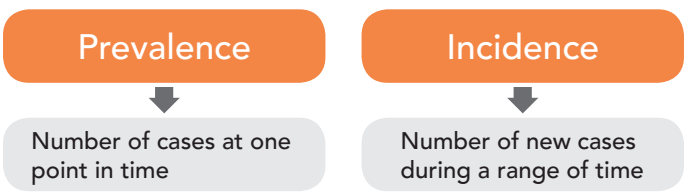

Fig 1-4 Descriptive statistics often encountered in the endodontic literature.

For example, Eriksen et al reviewed several European studies that reported the prevalence of apical periodontitis with a range from 26% to 70%. These results indicate that screening via periapical radiographs found that between 26% and 70% of patients sampled had apical periodontitis at a particular time point. An additional example is found in a study by Lipton et al, which reported a 12% incidence of toothache in the United States population in the preceding 6 months. Prevalence is a good measure for apical periodontitis since it develops slowly over a long time period, wherein it might be difficult to truly detect new cases. Incidence is a better measure for toothache since they generally have a rapid onset and decline, so a point in time assessment might miss many cases.

Prognosis

Success rates of therapy are frequently utilized to justify treatment choices. Chapter 11 presents an in-depth discussion of endodontic success rates. Success can have multiple definitions depending on the text, and it is important to understand how each study defines success. Oftentimes, a distinction can be made between *success*, defined as the absence of symptoms and radiographic periapical pathology, and *survival*, referring to the absolute presence or absence of the tooth in the mouth without consideration of symptoms or pathology. When examining primary sources, it is important to understand the authors' definition of success as results will vary accordingly. Furthermore, the advent of newer imaging modalities like cone beam computed tomography (CBCT) may alter our future definitions. Wu et al suggested that the lines between success and survival may be blurred once prognosis studies utilizing CBCT imaging become available because CBCT images will inevitably detect more lesions than traditional radiography.

Bibliography

Introduction

Glaser AN. High-Yield Biostatistics, Epidemiology, and Public Health, ed 4. Philadelphia: Lippincott Williams & Wilkins, 2014.

Hulley SB, Cummings SR, Browner WS, Grady DG, Newman TB. Designing Clinical Research, ed 4. Philadelphia: Lippincott Williams & Wilkins, 2013.

Study Design

Oxford Centre for Evidence-Based Medicine. OCEBM Levels of Evidence. http://www.cebm.net/ocebm-levels-of-evidence/. Accessed 6 January 2016

Statistics

Jespersen JJ, Hellstein J, Williamson A, Johnson WT, Qian F. Evaluation of dental pulp sensibility tests in a clinical setting. J Endod 2014;40:351–354.

Spili P, Parashos P, Messer HH. The impact of instrument fracture on outcome of endodontic treatment. J Endod 2005;31:845–850.

Epidemiology

Eriksen H, Kirkevang L, Petersson K. Endodontics epidemiology and treatment outcome: General considerations. Endod Topics 2002;2:1–9.

Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc 1993;124:115–121.

Prognosis

Wu MK, Shemesh H, Wesselink PR. Limitations of previously published systematic reviews evaluating the outcome of endodontic treatment. Int Endod J 2009;42:656–666.

2

Microbiology

Endodontic pathology results from interactions between microbes and host immune responses. The seminal work of Kakehashi et al on germ-free rats illustrated the role of bacteria as a major etiologic force in the progression of pulpal inflammation to apical periodontitis (Fig 2-1). In their study, gnotobiotic, or germ-free, rats did not develop apical periodontitis following pulpal exposures, whereas conventional rats with normal oral flora rapidly developed apical pathology. Moller et al and Sundqvist noted similar results in their work with monkeys and humans, respectively. Both found bacteria in necrotic pulps with apical periodontitis but not in necrotic pulps without apical disease.

This chapter covers historically significant events in endodontic microbiology, research methods for microbial analysis, and commonly encountered microbes in endodontic infections. A review of biofilm biology is also presented, and the chapter concludes with a discussion of pathways of microbial spread.

Fig 2-1 The relationship between pulp necrosis, bacteria, and the development of apical periodontitis. Bacteria are essential for progression of pulpal necrosis to apical periodontitis.

History of Endodontic Microbiology

One cannot study endodontic microbiology without understanding the complicated history of the focal infection theory. This theory dates back to medical literature of the 19th century and asserts that localized or generalized infection can result from dissemination of bacteria and toxic byproducts from a focus of infection. Weston Price brought the theory to endodontics in 1925 when he inferred that bacteria trapped in dentinal tubules after root canal therapy could "leak" from the root canal space and cause systemic disease. He strongly advocated extraction of all diseased teeth. In 1952, Easlick pointed out the fallacies in Price's research methods, including the inadequate use of controls, large amounts of bacteria in the cases presented, and contamination of root canal–treated teeth studied during extraction. Doing so, he effectively refuted the associations between endodontically treated teeth and systemic disease. The work of Fish also refuted Price's claims. Fish described the encapsulation of infections into the so-called "Zones of Fish": the zones of infection, contamination, irritation, and stimulation extending outward concentrically (Fig 2-2). If the nidus of infection is removed, the body can recover, providing a basis for the success of root canal therapy.

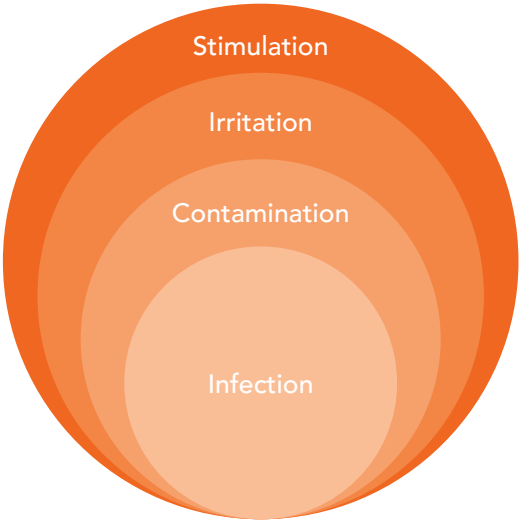

Fig 2-2 The Zones of Fish describe a means of infection containment.

Research Methods

With the advent of new research methods, the understanding of endodontic microbiology has changed. Culture methods have been available for many years but have several limitations. Certain species are unable to grow outside of physiologic conditions, and it is difficult to take a truly anaerobic sample for growth in culture. The advent of molecular techniques facilitated the detection of uncultivable species. These techniques include polymerase chain reactions (PCR), fluorescent in situ hybridization (FISH), and DNA checkerboard analysis. PCR amplifies DNA, which can subsequently be sequenced to identify the presence of known and novel species. Variants of DNA techniques, such as FISH and DNA checkerboard analysis, allow detection of vast libraries of known species. Molecular techniques are also useful in the detection of nonbacterial infection sources. They can be used to identify the DNA from fungal infections, including candida, and viruses, including viruses in the herpes family.

Though molecular techniques offer superior species detection, some utility remains in classical microbiology laboratory techniques, including gram staining. Gram-positive bacteria are labeled as such due to the affinity of the crystal violet dye for their thick peptidoglycan cell walls. Gram-positive bacteria include those in the *Streptococcus*, *Peptostreptococcus*, *Enterococcus*, *Lactobacillus*, *Eubacterium*, and *Actinomyces* genera. Gram-negative bacteria have a lesser affinity for the crystal violet stain due to the presence of a cell wall containing lipopolysaccharide (LPS), often referred to as *endotoxin*. LPS is important in the progression of pulpal and periapical inflammation. Dwyer and Torabinejad found that it stimulates cytokine production by macrophages (Fig 2-3). Gram-negative bacteria include those in the *Fusobacterium*, *Treponema*, *Prevotella*, *Porphyromonas*, *Tannerella*, *Dialister*, *Campylobacter*, and *Veillonella* genera.

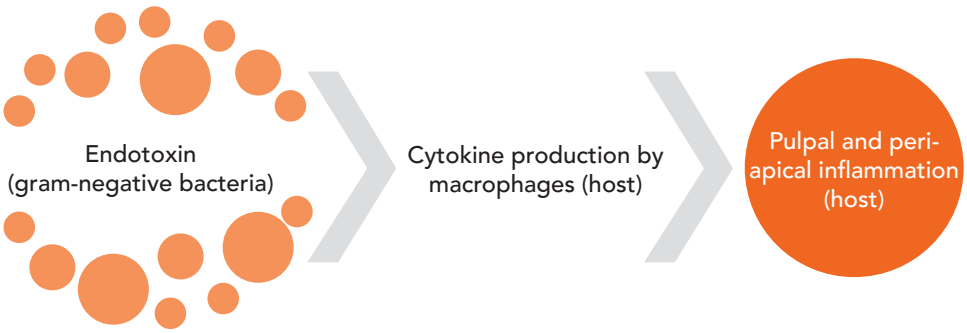

Fig 2-3 Endotoxin (ie, LPS) is the key component inducing an inflammatory response in pulpal and periapical disease (Dwyer and Torabinejad).

Endodontic Infections

Not all oral microbes are pathogenic. Our bodies host a vast, complex, and symbiotic microbiome. Most simply, this microbiome maintains an important equilibrium that serves to exclude pathogenic or opportunistic bacteria from invasion. While a large amount of the human body is colonized by bacteria, the dental pulp and associated periapical tissues are normally sterile spaces. When the body's physiologic microbiome is interrupted, or pathogenic microbes enter normally sterile tissues such as the dental pulp, the balance shifts, and pathogenic infection can occur.

Some degree of protective barrier interruption must occur for bacterial contamination of the pulp and periapex, and theories abound. Caries and direct exposure via fracture are the most obvious means for microbial contamination of the dental pulp. However, endodontic pathology may have alternative origins, such as traumatic injuries without direct pulpal exposures. Bergenholtz proposed that microcracks caused by traumatic injuries allow ingress of bacteria to infect an already compromised, inflamed pulp. Gier and Mitchell proposed *anachoresis*—the homing of bacteria to traumatized, unexposed pulps—as another means of infection. However, work by Delivanis et al effectively disproved this. Figure 2-4 illustrates the theorized means of bacterial introduction to the dental pulp.

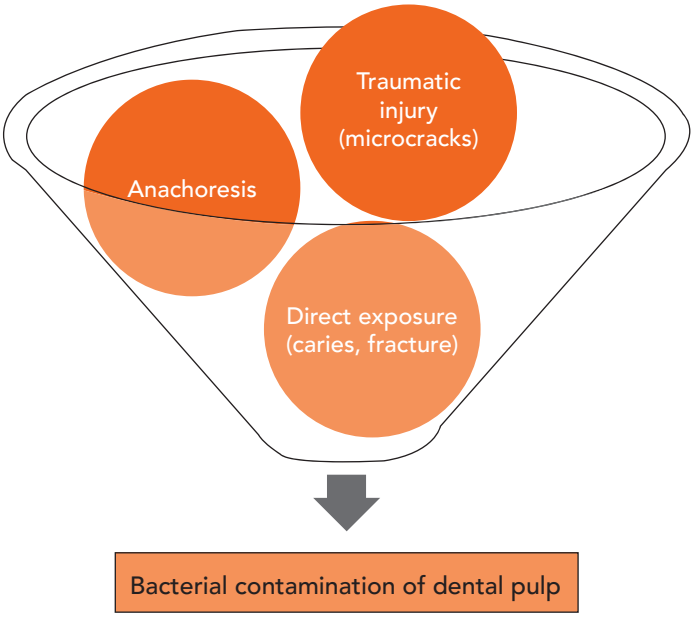

Fig 2-4 Theorized means of bacterial introduction to dental pulp.

Regardless of the means of pulp inoculation, endodontic infections are polymicrobial. Both culture-based and molecular methods confirm this finding. Molecular research has provided greater understanding of the complex microbial communities present in end-

odontic infections. These communities often exist in the form of biofilms. Donlan and Costerton defined *biofilms* as microbial-derived, sessile communities characterized by cells irreversibly attached to a substratum or interface or to one other, embedded in a self-produced matrix of extracellular polymeric substances, and exhibiting an altered phenotype with respect to growth rate and gene transcription compared with their planktonic counterparts.

Svensater and Bergenholtz described several qualities unique to biofilms including metabolic diversity, concentration gradients, genetic exchange, and quorum sensing (Fig 2-5). Bacterial biofilms are metabolically diverse, allowing a sharing of nutritional sources and waste products and resulting in greater overall survival. The concentration gradient created by the mere density of the biofilm community allows for greater physical and chemical resistance to antimicrobials and immune responses. Genetic exchange by the microbiota in close proximity allows for sharing of favorable virulence factors. Quorum sensing serves as a communication method among the microbial community and permits the members to act as a group and increase the effectiveness of their actions. For example, quorum sensing allows the release of virulence factors as a group.

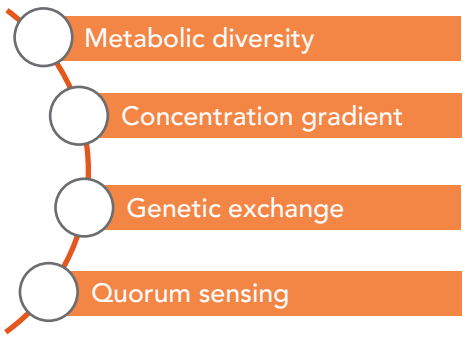

Fig 2-5 The qualities often attributed to biofilms (Svensater and Bergenholtz).

While historically abscesses were thought to be sterile (Shindell), current research supports the validity of extraradicular infections. Tronstad et al performed one of the first culture studies demonstrating the presence of bacteria, particularly anaerobes, in extraradicular infections. Sunde et al (2000) confirmed these findings using molecular techniques and noted the presence of certain species in periapical infections, in particular *Aggregatibacter actinomycetemcomitans* and *Tannerella forsythia*. Haapasalo et al cultured anaerobic bacteria in sinus tracts, and Sassone et al reported a higher prevalence of *Porphyromonas gingivalis* and *Fusobacterium nucleatum* when a sinus tract was present. Sabeti and Slots reported the presence of human cytomegalovirus and Epstein-Barr virus in apical periodontitis.

Most, though not all, teeth exhibiting pulpal necrosis are infected. In the absence of infection, Andreasen demonstrated that periapical healing could occur despite pulpal necrosis in traumatically luxated teeth without bacterial contamination. Wittgow and Sabiston found that 64% of teeth with pulpal necrosis were infected, and Bergenholtz found that teeth with pulpal necrosis and periapical lesions were more often infected.

Typically isolated species

Endodontic infections are comprised of frequently isolated species, and these are repeatedly noted in the literature. These include both facultative and obligate anaerobes, including members of the *Strepotococcus*, *Enterococcus*, *Prevotella*, and *Porphyromonas* species (Fig 2-6). With further development of microbial techniques, the diverse nature of these so-called typical species has become more apparent. Furthermore, these species may exhibit some geographic variation, as Baumgartner et al (2004) found different profiles of infections in Brazilian populations versus those from the United States.

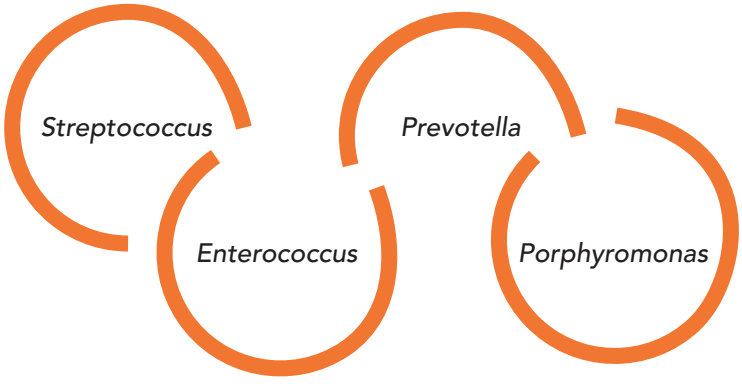

Fig 2-6 Common isolates in endodontic infections.

Streptococci are gram-positive, generally facultative anaerobic bacteria. They are classified as either alpha or beta based on their reaction with hemoglobin molecules on blood agar in a laboratory. Winkler and Van Amerongen reported that beta-hemolytic streptococci, particularly those further classified into groups F, G, C, and minorly D, were common isolates in endodontic infections. He further reported a lesser presence of *Streptococcus mitis*, an alpha-hemolytic *Streptococcus* in the viridans group.

Enterococcus faecalis is a gram-positive facultative anaerobe formerly classified as a member of group D beta-hemolytic streptococci. It is of particular interest due to its antimicrobial resistance. *E faecalis* possesses a proton pump that allows it to adapt to harsh environments (Evans et al). This proton pump is theorized to contribute to *E faecalis*' unique resistance to calcium hydroxide (Bystrom et al), an intracanal medicament known for its effectiveness against most known endodontic pathogens. Presumably, the proton pump prevents the ionization calcium hydroxide requires for effectiveness. *E faecalis* also possesses the ability to survive for long periods of time in dentinal tubules without nutrients (Love). Lastly, Distel et al found that this microbe could form biofilms. Interestingly, Penas et al reported lesser antimicrobial resistance in oral as compared to nosocomial *E faecalis* infections. The properties of *E faecalis* proposed to increase its resistance to eradication are summarized in Fig 2-7.

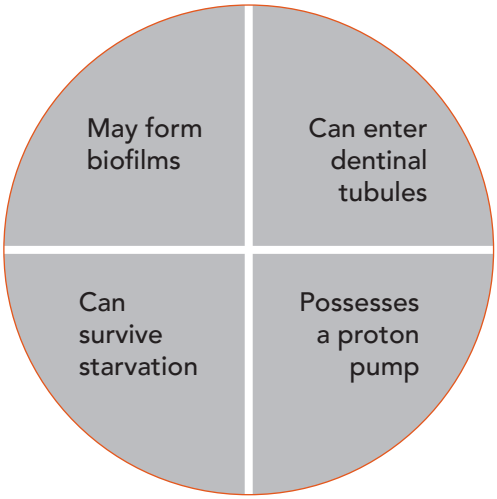

Fig 2-7 Properties attributed to *E faecalis* that increase its resistance to endodontic procedures and make it a common isolate in persistent endodontic infections.

Classic endodontic literature frequently described "black pigmented bacteroides" as common isolates in endodontic infections. In the 1980s, microbiologists recognized that this group comprised a relatively heterogenous group of bacteria and further split the genus of *Bacteroides* into *Prevotella* and *Porphyromonas* (Fig 2-8). Though both groups are gram-negative and obligate anaerobes, they are differentiated by their abilities to ferment carbohydrates. Shah and Collins described *Prevotella* as saccharolytic, or able to ferment carbohydrates, whereas Love et al labeled *Porphyromonas* as asaccharolytic. An easy way to remember these is to pair the "as" ending of *Porphyromonas* with the first two letters of asaccharolytic. Bae et al reported that *Prevotella nigrescens* was the most common isolate from endodontic infections of those previously categorized as *Bacteroides*. Gomes et al reported that *Prevotella melaninogenica* was commonly associated with painful infections.

Fig 2-8 Reclassification of prior black-pigmented *Bacteroides* by carbohydrate fermentation properties.

Atypical species

Although modern research techniques challenge the knowledge of the typical makeup of endodontic infections, certain microbes are less frequently reported in the literature than those discussed in the previous section. These include *Actinomyces*, spirochetes, fungi, and archaea (Fig 2-9).

Fig 2-9 Less frequently encountered species in endodontic infections.

Actinomyces are gram-positive bacteria that form cohesive colonies often described clinically as "sulfur granules" because of their yellow granular presentation. Sunde et al's (2002) histologic analysis of these "sulfur granules" noted that they indeed contained large quantities of clumped bacteria. Though isolation of *Actinomyces* is only rarely reported in the endodontic literature, Nair described difficulties in culturing the organism. Modern research methods, on the other hand, more frequently isolate this genus. Xia and Baumgartner noted *Actinomyces israelii*, *Actinomyces naeslundii,* and *Actinomyces viscosus* in infected root canals and aspirates from associated abscesses and cellulitis. Nair reviewed *Actinomyces'* ability to survive and thrive in the periapical area, often called *periapical actinomycosis*, and cites this entity as a common cause of persistent endodontic infections. Due to its persistence and frequent recurrence with traditional treatments, Jeansonne recommended treating periapical actinomycosis via a surgical approach along with a relatively long, 6-week course of systemic penicillin.

Spirochetes, typically gram-negative, anaerobic bacteria with flagella for motility, are reported isolates in endodontic infections. Because spirochetes are difficult to culture, molecular techniques must often be employed to detect them. Siqueira et al noted *Treponema* subspecies in endodontic infections. Sakamoto et al identified a variety of *Treponema* species present in endodontic infections, particulary *Treponema denticola*, *Treponema socranskii*, and *Treponema maltophilum*.

Though less frequently encountered, nonbacterial organisms including archaea, eukaryotes including fungi, and viruses have been reported in endodontic infections. Archaea, also known as *extremophiles*, are known to be present in hot springs and can be localized to the gastrointestinal and vaginal tracts as well as in periodontal plaque. Vianna et al first reported their presence in endodontic infections. Baumgartner et al (2000) found *Candida albicans* in primary endodontic infections. Giardino et al reported a case of an *Aspergillus* fungal infection associated with extruded zinc oxide–based endodontic sealer in the maxillary sinus potentially related to the zinc, an *Aspergillus* metabolite, present in the sealer.

Prions, infectious agents composed of misfolded proteins that target neurologic tissue, are theorized as potential pathogens in pulp tissue. Smith et al suggested that, should prions be found in pulp tissue, prion infections could be transmitted by sterilized endodontic instruments because traditional autoclave techniques do not eliminate proteinaceous contaminants. However, Azarpazhooh and Fillery performed a systematic review of the literature and found no reports of prions in the dental pulp. This theoretical, but as yet unproven, risk to reusing sterilized instruments that have been in contact with the dental pulp has spurred recommendations by dental manufacturers for the single use of such instruments.

Viruses

Viruses, particularly those in the herpesvirus family, are commonly reported in endodontic infections. Ferreira et al noted herpes simplex virus (HSV) types 1 and 2; human herpesvirus (HHV) types 6, 7, and 8; and varicella zoster virus (VZV) in aspirated samples of acute apical abscesses. Sabeti et al reported the presence of Epstein-Barr virus (EBV) and cytomegalovirus (CMV) in periapical lesions, especially larger and symptomatic lesions. Li et al also reported a possible association of EBV with irreversible pulpitis. Recent data indicates that viruses may play an active role in pulpal death. A case report by Goon and Jacobsen described devitalization of the dental pulp associated with a trigeminal VZV infection. Lastly, viruses may play a role in resorptive processes. Von Arx et al reported a potential association between feline herpesvirus and cases of invasive cervical root resorption in both humans and cats.

Nonherpetic viruses have also been described in the endodontic literature. Ferreira et al found human papillomavirus (HPV) in endodontic abscesses. Although human immunodeficiency virus (HIV) has not been correlated with the pathogenesis of endodontic disease, Glick et al located it in the dental pulp of individuals with clinical AIDS. Elkins et al found HIV in periradicular lesions of patients known to be carriers of the virus. Figure 2-10 lists a summary of the viruses reported as isolated from endodontic infections.

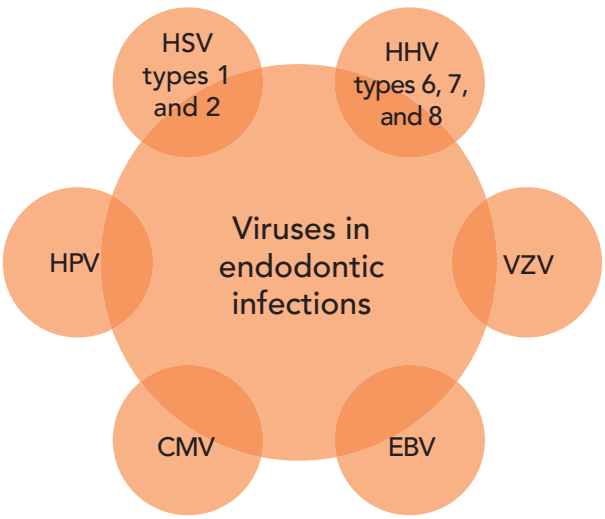

Fig 2-10 Commonly isolated viruses in endodontic infections.

Bacterial Communities

Endodontic bacterial communities continually adapt to their environment, and their characteristics may alter the clinical characteristics of that particular infection. The periodontal literature recognizes that groups of bacterial species may be more pathogenic than individuals alone. Socransky et al described the red complex including *P gingivalis*, *T denticola*, and *T forsythia* and their association with the severity of periodontitis. Similarly, certain microbial relationships are important in the progression of endodontic disease, namely primary versus secondary infections or acute versus chronic infections.

In general, *primary infections*, those that occur in untreated necrotic teeth, are believed to involve a greater number of species than *secondary infections*, ie, reinfections of previously treated teeth. Rôças and Siqueira (2008) reported roughly 20 species in primary infections versus approximately 3 species in secondary infections (Siqueira and Rôças 2004). The techniques used to detect species appear to matter significantly. A recent study by Hong et al using pyrosequencing noted hundreds of bacterial species in primary and secondary infections with no statistically significant differences in diversity among the two.

Figdor and Sundqvist reported differences in the composition of primary versus secondary infections (Fig 2-11). Primary infections consisted of an equal mix of gram-positive and gram-negative bacteria and contained mostly obligate anaerobes. Fabricius et al described the progression of primary endodontic infections from largely aerobic species to anaerobic species, a process he termed *microbial succession*. This results from a reduction in oxygen tension in the necrotic pulp tissue due to aerobic metabolism by early colonizers. Secondary infections may differ significantly from their primary counterparts. Figdor and Sundqvist reported that secondary infections contained mostly gram-positive bacteria with a more equal distribution of facultative and obligate anaerobes. Conversely, a recent study by Murad et al reported a higher prevalence of gram-negative than gram-positive species in secondary infections, particularly in the presence of a large periapical lesion.

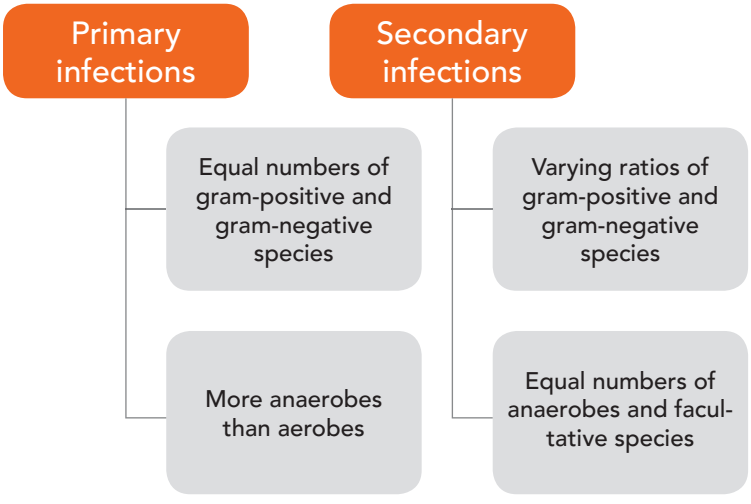

Fig 2-11 Characteristics of primary versus secondary endodontic infections.

Particular species have been associated with either primary or secondary infections. *E faecalis* is frequently associated with secondary infections. Rôças et al reported that it was nine times more likely to be present in secondary than primary infection. However, Rôças and Siqueira (2012) call these findings into question in future work, supposing that *E faecalis* is perhaps more often reported in secondary infections due to its ease of detection with laboratory methods. In a recent study using checkerboard DNA hybridization, Murad et al reported that *Enterococcus faecium* and *Streptococcus epidermidis* were the most prevalent species in secondary infections.

This species specificity may apply to other types of infections. For example, Siqueira et al (2004) reported an increased prevalence of *Fusobacterium* in symptomatic infections. Sassone et al reported an association between *T forsythia* and painful infections. Gomes et al noted an increased prevalence of peptostreptococci and *P melaninogenica* with pain. Lastly, Sabeti et al found that EBV and CMV were also associated with painful infections.

Anatomical Distribution of Infections

Endodontic infections originating from the dental pulp can spread via apical tissue into the alveolar bone. Eventually, infections may spread through *fascial spaces*, the potential spaces between the fascia and the underlying tissues and organs. Depending on the particular location of the infection, endodontic infections tend to take a particular path in their spread into peri-orofacial tissues. These typical pathways may differ based on patient anatomy.

The fascial spaces germane to endodontic infections are described below. For a more complete anatomical reference, please refer to Fehrenbach and Herring's *Illustrated Anatomy of the Head and Neck*. Following are descriptions of the defining features of the fascial spaces commonly involved with endodontic infections with credit to Siqueira and Rôças in *Cohen's Pathways of the Pulp*.

- **Buccal vestibule.** Defined by the buccinators and alveolar mucosa. Infections from posterior maxillary teeth with root apices inferior to the buccinator insertion or posterior mandibular teeth with root apices superior to the buccinator insertion may spread to the buccal vestibule.
- **Buccal space.** Defined by the buccinators and cheek mucosa. Infections from posterior maxillary teeth with root apices superior to the buccinator insertion or posterior mandibular teeth with root apices inferior to the buccinator insertion may spread to the buccal space. Infections in the buccal space can spread to the periorbital space due to its close proximity.
- **Pterygomandibular space.** Defined by the medial pterygoid and the mandibular ramus inferior to the lateral pterygoid. Infections from mandibular second or third molars often spread to this space.
- **Canine space.** Located superior to the levator anguli oris muscle and inferior to the levator labii superioris. Infections from maxillary canines and maxillary first premolars with infection breaking through the buccal cortex may spread to this space.
- **Periorbital space.** Located deep to the orbicularis occuli. Infections from maxillary canines or enlarging buccal space infections may spread to this space.

- **Submandibular space.** Found superior to the platysma and inferior to the mylohyoid muscle. Infections from mandibular posterior teeth breaking through the lingual cortex may spread to this space. This space is contiguous with the submental space across the digastric muscle.
- **Submental space.** Found superior to the platysma muscle and inferior to the mylohyoid muscle. Infections from mandibular anterior teeth may spread to this space.
- **Mental space.** Located below the mentalis muscle. Infections from mandibular anterior teeth may spread to this space.
- **Sublingual space.** Found superior to the mylohyoid muscle and inferior to the floor of the mouth. Infections from mandibular teeth that break through the lingual cortex may spread to this space. This is a bilateral space without a midline separation.

Infections in individual teeth often follow particular patterns of spread when transitioning from a localized abscess to more generalized swelling (Fig 2-12 and Table 2-1). Infections of maxillary teeth tend to spread to the buccal space or buccal vestibule, although infections of maxillary lateral incisors and palatal roots of first premolars and molars may extend

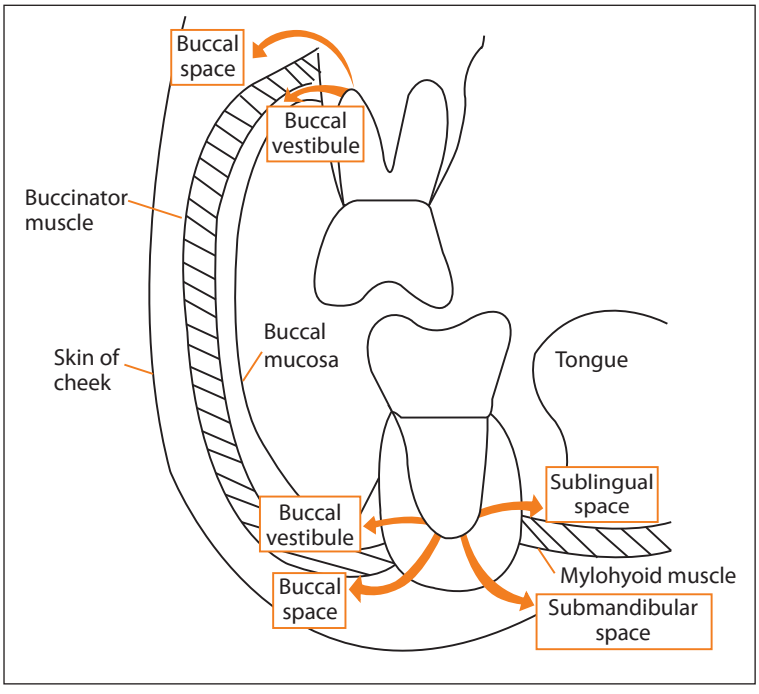

Fig 2-12 Typical pathways of infectious spread from maxillary and mandibular molars.

palatally. Maxillary canine and first premolar infections often spread to the canine space or to the periorbital space. Infections of mandibular incisors usually spread to the buccal vestibule, submental, mental, or sublingual spaces. Infections of mandibular premolars or first molars often spread to the buccal space or buccal vestibule but may also spread into the sublingual or submandibular spaces (Siqueira and Rôças). Table 2-1 summarizes common infection pathways in endodontic infections.

Table 2-1 Typical fascial space spread of periradicular infections[a]

	Location of infected tooth	Typical fascial space spread
Maxilla	Central incisors	Buccal vestibule
	Lateral incisors	Buccal vestibule Palatal
	Canines	Buccal vestibule Canine space Periorbital space
	First premolars	Buccal vestibule Buccal space Canine space Palatal roots may spread palatal
	Second premolars	Buccal vestibule Buccal space
	First, second, and third molars	Buccal vestibule Buccal space Palatal roots may spread palatal
Mandible	Central and lateral incisors and canines	Buccal vestibule Submental space Mental space Sublingual space
	First and second premolars and first molars	Buccal space Buccal vestibule Sublingual space Submandibular space
	Second and third molars	Buccal space Buccal vestibule Sublingual space Submandibular space Pterygomandibular space

[a]Data from Siqueira and Rôças.

Beyond a more localized abscess characterized by pain and swelling or cellulitis, fascial space infections may have significant medical consequences. Infections of the lateral pharyngeal space, which can develop when an infection from a mandibular second or third molar spreads beyond the pterygomandibular space, can lead to inner jugular thrombosis. Infections involving the submental, sublingual, and submandibular spaces combine to create *Ludwig angina*, a life-threatening infection characterized by difficulty swallowing, difficulty opening the mouth, and difficulty breathing. Infections of the periorbital space may spread via valveless facial veins with resultant cavernous sinus thrombosis characterized by a lateral gaze palsy. The *danger space*, defined by the alar and prevertebral fascia, may become involved in severe periradicular infections that spread beyond the lateral pharyngeal space and is so named based on its continuity with the mediastinal cavity (Siqueira and Rôças). All of these are considered medical emergencies requiring prompt intervention at an emergency medical facility.

Bibliography

Introduction

Kakehashi S, Stanley HR, Fitzgerald RJ. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. Oral Surg Oral Med Oral Pathol 1965;20:340–349.

Moller AJ, Fabricius L, Dahlen G, Ohman AE, Heyden G. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. Scand J Dent Res 1981;89:475–484.

Sundqvist G. Bacteriological Studies of Necrotic Dental Pulps [thesis]. Umea, Sweden: University of Umea, 1976.

History of Endodontic Microbiology

Easlick KA. Evaluation of the action of focal dental infections on health. Med Hyg (Geneve) 1952;10:35.

Fish EW. Bone Infection. J Am Dent Assoc 1939;26:691–712.

Price WA. Dental infections and related degenerative diseases: Some structural and biochemical factors. JAMA 1925;84:254–261.

Research Methods

Dwyer TG, Torabinejad M. Radiographic and histologic evaluation of the effect of endotoxin on the periapical tissues of the cat. J Endod 1981;7:31–35.

Endodontic Infections

Andreasen FM. Pulpal healing after luxation injuries and root fracture in the permanent dentition. Endod Dent Traumatol 1989;5:111–131.

Azarpazhooh A, Fillery ED. Prion disease: The implications for dentistry. J Endod 2008;34:1158–1166.

Bae KS, Baumgartner JC, Shearer TR, David LL. Occurrence of *Prevotella nigrescens* and *5* in infections of endodontic origin. J Endod 1997;23:620–623.

Baumgartner JC, Siqueira JF Jr, Xia T, Rôças IN. Geographical differences in bacteria detected in endodontic infections using polymerase chain reaction. J Endod 2004;30:141–144.

Baumgartner JC, Watts CM, Xia T. Occurrence of *Candida albicans* in infections of endodontic origin. J Endod 2000;26:695–698.

Bergenholtz G. Micro-organisms from necrotic pulp of traumatized teeth. Odontol Revy 1974;25:347–358.

Bystrom A, Claesson R, Sundqvist G. The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals. Endod Dent Traumatol 1985;1:170–175.

Delivanis PD, Snowden RB, Doyle RJ. Localization of blood-borne bacteria in instrumented unfilled root canals. Oral Surg Oral Med Oral Pathol 1981;52:430–432.

Distel JW, Hatton JF, Gillespie MJ. Biofilm formation in medicated root canals. J Endod 2002;28:689–693.

Donlan RM, Costerton JW. Biofilms: Survival mechanisms of clinically relevant microorganisms. Clin Microbiol Rev 2002;15:167–193.

Elkins DA, Torabinejad M, Schmidt RE, Rossi JJ, Kettering JD. Polymerase chain reaction detection of human immunodeficiency virus DNA in human periradicular lesions. J Endod 1994;20:386–388.

Evans M, Davies JK, Sundqvist G, Figdor D. Mechanisms involved in the resistance of *Enterococcus faecalis* to calcium hydroxide. Int Endod J 2002;35:221–228.

Ferreira DC, Paiva SS, Carmo FL, et al. Identification of herpesviruses types 1 to 8 and human papillomavirus in acute apical abscesses. J Endod 2011;37:10–16.

Giardino L, Pontieri F, Savoldi E, Tallarigo F. *Aspergillus mycetoma* of the maxillary sinus secondary to overfilling of a root canal. J Endod 2006;32:692–694.

Gier RE, Mitchell DF. Anachoretic effect of pulpitis. J Dent Res 1968;47:564–570.

Glick M, Trope M, Pliskin ME. Detection of HIV in the dental pulp of a patient with AIDS. J Am Dent Assoc 1989;119:649–650.

Gomes BP, Lilley JD, Drucker DB. Associations of endodontic symptoms and signs with particular combinations of specific bacteria. Int Endod J 1996;29:69–75.

Goon WW, Jacobsen PL. Prodromal odontalgia and multiple devitalized teeth caused by a herpes zoster infection of the trigeminal nerve: Report of case. J Am Dent Assoc 1988;116: 500–504.

Haapasalo M, Ranta K, Ranta H. Mixed anaerobic periapical infection with sinus tract. Endod Dent Traumatol 1987;3:83–85.

Jeansonne BG. Periapical actinomycosis: A review. Quintessence Int 2005;36:149–153.

Li H, Chen V, Chen Y, Baumgartner JC, Machida CA. Herpesviruses in endodontic pathoses: Association of Epstein-Barr virus with irreversible pulpitis and apical periodontitis. J Endod 2009;35:23–29.

Love RM. *Enterococcus faecalis*—A mechanism for its role in endodontic failure. Int Endod J 2001;34:399–405.

Love DN, Bailey GD, Collings S, Briscoe DA. Description of *Porphyromonas circumdentaria* sp. nov. and reassignment of *Bacteroides salivosus* (Love, Johnson, Jones, and Calverley 1987) as *Porphyromonas* (Shah and Collins 1988) *salivosa* comb. nov. Int J Syst Bacteriol 1992;42: 434–438.

Nair PN. On the causes of persistent apical periodontitis: A review. Int Endod J 2006;39: 249–281.

Penas PP, Mayer MP, Gomes BP, et al. Analysis of genetic lineages and their correlation with virulence genes in *Enterococcus faecalis* clinical isolates from root canal and systemic infections. J Endod 2013;39:858–864.

Sabeti M, Simon JH, Slots J. Cytomegalovirus and Epstein-Barr virus are associated with symptomatic periapical pathosis. Oral Microbiol Immunol 2003;18:327–328.

Sabeti M, Slots J. Herpesviral-bacterial coinfection in periapical pathosis. J Endod 2004; 30:69–72.

Sakamoto M, Siqueira JF Jr, Rôças IN, Benno Y. Diversity of spirochetes in endodontic infections. J Clin Microbiol 2009;47:1352–1357.

Sassone LM, Fidel R, Faveri M, Fidel S, Figueiredo L, Feres M. Microbiological evaluation of primary endodontic infections in teeth with and without sinus tract. Int Endod J 2008; 41:508–515.

Shah HN, Collins DM. Prevotella, a new genus to include *Bacteroides melaninogenicus* and related species formerly classified in the genus *Bacteroides*. Int J Syst Bacteriol 1990; 40:205–208.

Shindell E. Studies on the possible presence of a virus in subacute and chronic periapical granulomas. Oral Surg Oral Med Oral Pathol 1962;15:1382–1384.

Siqueira JF Jr, Rôças IN, Favieri A, Santos KR. Detection of *Treponema denticola* in endodontic infections by 16S rRNA gene-directed polymerase chain reaction. Oral Microbiol Immunol 2000;15:335–337.

Smith A, Dickson M, Aitken J, Bagg J. Contaminated dental instruments. J Hosp Infect 2002; 51:233–235.

Sunde PT, Olsen I, Debelian GJ, Tronstad L. Microbiota of periapical lesions refractory to endodontic therapy. J Endod 2002;28:304–310.

Sunde PT, Tronstad L, Eribe ER, Lind PO, Olsen I. Assessment of periradicular microbiota by DNA-DNA hybridization. Endod Dent Traumatol 2000;16:191–196.

Svensater G, Bergenholtz G. Biofilms in endodontic infections. Endod Topics 2004;9:27–36.

Tronstad L, Barnett F, Riso K, Slots J. Extraradicular endodontic infections. Endod Dent Traumatol 1987;3:86–90.

Vianna ME, Conrads G, Gomes BP, Horz HP. Identification and quantification of archaea involved in primary endodontic infections. J Clin Microbiol 2006;44:1274–1282.

Von Arx T, Schawalder P, Ackermann M, Bosshardt DD. Human and feline invasive cervical resorptions: The missing link?—Presentation of four cases. J Endod 2009;35:904–913.

Winkler KC, Van Amerongen J. Bacteriologic results from 4,000 root canal cultures. Oral Surg Oral Med Oral Pathol 1959;12:857–875.

Wittgow WC Jr, Sabiston CB Jr. Microorganisms from pulpal chambers of intact teeth with necrotic pulps. J Endod 1975;1:168–171.

Xia T, Baumgartner JC. Occurrence of *Actinomyces* in infections of endodontic origin. J Endod 2003;29:549–552.

Bacterial Communities

Fabricius L, Dahlen G, Ohman AE, Moller AJ. Predominant indigenous oral bacteria isolated from infected root canals after varied times of closure. Scand J Dent Res 1982;90:134–144.

Figdor D, Sundqvist G. A big role for the very small—Understanding the endodontic microbial flora. Aust Dent J 2007;52:S38–51.

Gomes BP, Lilley JD, Drucker DB. Associations of endodontic symptoms and signs with particular combinations of specific bacteria. Int Endod J 1996;29:69–75.

Hong BY, Lee TK, Lim SM, et al. Microbial analysis in primary and persistent endodontic infections by using pyrosequencing. J Endod 2013;39:1136–1140.

Murad CF, Sassone LM, Faveri M, Hirata R Jr, Figueiredo L, Feres M. Microbial diversity in persistent root canal infections investigated by checkerboard DNA-DNA hybridization. J Endod 2014;40:899–906.

Rôças IN, Siqueira JF Jr. Characterization of microbiota of root canal-treated teeth with post-treatment disease. J Clin Microbiol 2012;50:1721–1724.

Rôças IN, Siqueira JF Jr. Root canal microbiota of teeth with chronic apical periodontitis. J Clin Microbiol 2008;46:3599–3606.

Rôças IN, Siqueira JF Jr, Santos KR. Association of *Enterococcus faecalis* with different forms of periradicular diseases. J Endod 2004;30:315–320.

Sabeti M, Simon JH, Slots J. Cytomegalovirus and Epstein-Barr virus are associated with symptomatic periapical pathosis. Oral Microbiol Immunol 2003;18:327–328.

Sassone LM, Fidel RA, Faveri M, et al. A microbiological profile of symptomatic teeth with primary endodontic infections. J Endod 2008;34:541–545.

Siqueira JF Jr, Rôças IN. Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97:85–94.

Siqueira JF Jr, Rôças IN, Rosado AS. Investigation of bacterial communities associated with asymptomatic and symptomatic endodontic infections by denaturing gradient gel electrophoresis fingerprinting approach. Oral Microbiol Immunol 2004;19:363–370.

Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. J Clin Periodontol 1998;25:134–144.

Anatomical Distribution of Infections

Fehrenbach MJ, Herring SW. Illustrated Anatomy of the Head and Neck. St Louis: Elsevier/Saunders, 2012.

Siqueira JF Jr, Rôças IN. Microbiology and Treatment of Endodontic Infections. In: Hargreaves KM, Cohen S, Berman LH, (eds). Cohen's Pathways of the Pulp, ed 10. St Louis: Mosby Elsevier, 2011:559–600.

3

Pulpal and Periapical Anatomy and Physiology

A thorough understanding of endodontic pathology requires a strong foundation in normal development, histology, and physiology of both the teeth and surrounding tissues. This chapter serves to provide that foundation and enable the reader to better understand pulpal and periapical pathology.

Embryology

The development of teeth, or *odontogenesis*, results from a series of interactions between ectodermal cells of the first branchial arch and neural crest–derived mesenchymal cells (Thesleff). Signals from the oral ectoderm induce mesenchymal cells to fulfill their odontogenic role: to become odontoblasts and secrete dentin (Jernvall and Thesleff). Following the initiation of dentinogenesis, ectodermal cells form the inner and outer enamel epithelium and commence enamel formation (Thesleff). The role of several signaling molecules and growth factors important to this process have been identified, including bone morphogenetic protein 4 (BMP4), *msh homeobox 1* (*MSX1*), *paired box gene 9* (*PAX9*) (Kapadia et al), transforming growth factor (TGF), fibroblast growth factor (FGF), and insulinlike growth factor (IGF) (Martin et al).

Dentinogenesis continues until the crown is fully developed, after which root development commences. This process, like that responsible for crown development, is the result of serial epithelial-mesenchymal interactions. Cells of the inner and outer enamel epithelium contact one another to form Hertwig's epithelial root sheath (HERS). HERS activates mesenchymal cells to become odontoblasts and produce root dentin. Following dentin deposition, HERS resorbs (Diamond and Applebaum). The exposure of root dentin signals mesenchymal cells to populate the hard tissue and form cementum (Alatli et al). Following cementum formation, the periodontal ligament develops from mesenchymal precursor cells (Freeman and Ten Cate).

The dental pulp arises from the dental papilla, a mesenchymal tissue entrapped by the inner and outer enamel epithelium (Thesleff). Pulp vascularization begins prior to innervation. The onset of pulpal innervation is delayed until dentin and enamel formation begins. Sensory nerves containing calcitonin gene-related peptide (CGRP) and substance P (SP) enter the tissue earlier than sympathetic structures expressing neuropeptide Y (NPY) (Fristad et al). Figure 3-1 summarizes the embryologic origin of all dental tissues.

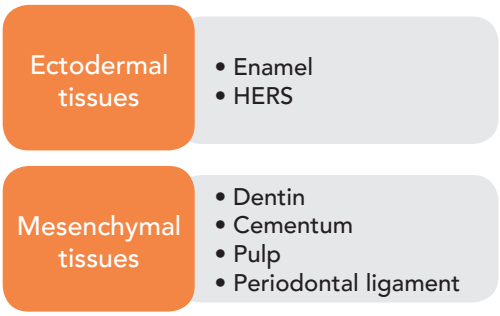

Fig 3-1 The embryologic origin of dental tissues.

Anatomy and Physiology of Dental Structures

Dentin

Dentin, a tissue of mesenchymal origin produced by odontoblasts, is intimately associated with the dental pulp (Fig 3-2). Odontoblasts secrete dentin in its unmineralized form, which then mineralizes throughout development (Butler and Ritchie). The odontoblastic process secretes both collagen and noncollagenous proteins, including dentin sialophosphoprotein and osteocalcin. These noncollagenous proteins likely serve as the impetus for dentin mineralization.

In its fully mature state, dentin is composed of 50% mineral by volume, 30% type I collagen, and 20% water (Kinney et al). It is a porous tissue containing tubules that extend from the dentinoenamel junction (DEJ) to the pulp. The number and diameter of dentinal tubules increases in proximity to the pulp. At the DEJ, Garberoglio and Brännström counted 20,000 tubules per square millimeter, whereas at the pulp surface, 45,000 tubules per square millimeter were noted. Just as tubule numbers increase in proximity to the pulp, so do the number of tubule branches (Holland).

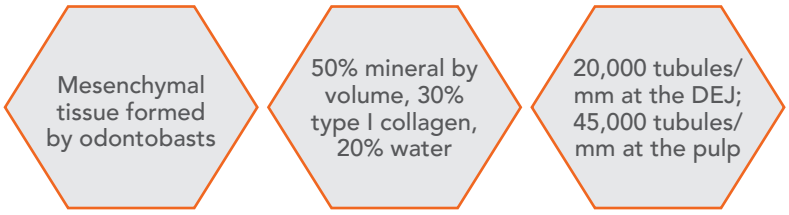

Fig 3-2 A summary of facts about dentin. DEJ, dentinoenamel junction.

The contents of the dentinal tubules and their branches have been described by several authors. Dai et al demonstrated intertubular collagen fibrils. In addition to these fibers, several authors have noted odontoblastic processes within dentinal tubules. However, the extent of penetration of these processes toward the DEJ is somewhat controversial. Garberoglio and Brännström demonstrated the extension of odontoblastic processes 0.5 mm into dentinal tubules. Byers and Sugaya found that they extended to the DEJ early in development only, while Yamada et al found that processes extended to the DEJ following completion of development. Despite the controversy, however, it is clear that the odontoblastic process extends to some degree into dentinal tubules at some point during development.

Dentin is often classified into three basic types, which Kuttler (1959) described as *primary*, *secondary*, and *tertiary* dentin (Fig 3-3). Primary dentin is that which is formed prior to eruption. Secondary dentin forms in response to the slightly aggressive effects of normal biologic function. Tertiary dentin develops in response to more intense pulpal irritants. Scott and Weber found an abrupt interface between primary and secondary dentin with a change in tubule direction in addition to a decrease in tubule number. Mjor et al categorized secondary and tertiary dentin as reactionary or reparative. Reactionary dentin is formed by an existing odontoblast, whereas reparative dentin is formed by the generation of a new odontoblast from precursor cells. Reeves and Stanley found that, following an insult, reparative dentin initially forms rapidly and then slows over time. An average of 1.49 μm of reparative dentin is formed per day.

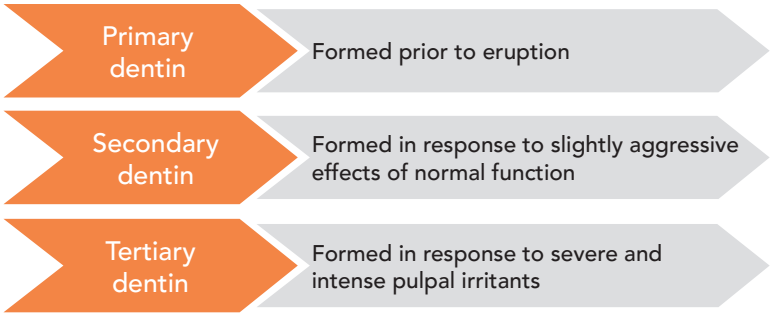

Fig 3-3 Kuttler's (1959) classification of dentin.

The dental pulp

Encased in its dentinal shell lies the dental pulp, a highly vascular and cellular organ. Pulp tissue arises from the dental papilla and thus is mesenchymal in origin (Thesleff). This tissue is composed of odontoblasts (Ruch), fibroblasts (Harris and Griffin), vascular structures (Kuttler 1955), immune cells, lymphatics (Bernick), nerves (Byers et al), and extracellular connective tissue (Linde).

Extracellular connective tissue

The extracellular connective tissue supports the vasculature, neural structures, lymphatics, and other cells that comprise the dental pulp (Fig 3-4). While dentin is comprised largely of type I collagen, the dental pulp contains types I, III, and V collagen (Pashley). According to Shuttleworth et al, 43% of the collagen found in the dental pulp tissue is type III. In addition to collagen, several noncollagenous substances comprise the extracellular connective tissue, including glycosaminoglycans (GAGs). According to Linde, the predominant GAG in pulp tissue is dermatan sulfate. However other GAGs can be found in the pulp, including chondroitin sulfate and hyaluronate.

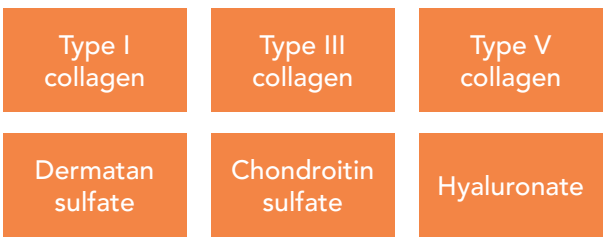

Fig 3-4 Contents of pulpal connective tissues.

Intrapulpal nerves

Coursing through the connective tissue and responding to all manner of stimuli are the pulpal nerves. These structures include both sensory and autonomic nerve fibers, and the composition is relatively unchanged between the primary and permanent dentition (Rapp et al). Sensory fibers include both the myelinated Aδ fibers and the unmyelinated C fibers (Fig 3-5). Of all axons entering the pulp, 87% are the unmyelinated C fibers (Nair). Aδ

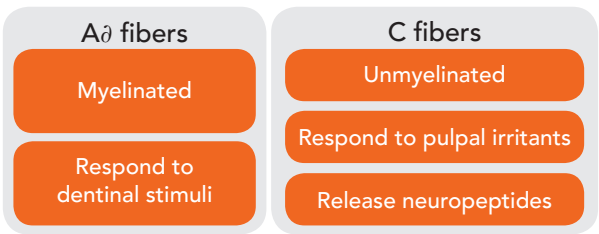

Fig 3-5 Pulpal sensory nerves.

fibers are responsible for dentin hypersensitivity and are relatively insensitive to inflammatory mediators. These fibers create short bursts of sensation. This lies in stark contrast to C fibers, responsible for lingering pulpal pain, which respond to inflammatory mediators (Olgart and Kerezoudis). In addition, these fibers may also respond directly to bacterial byproducts. Ferraz et al located toll-like receptor 4 in pulpal nerves, a receptor for lipopolysaccharide contained in the cell walls of gram-negative bacteria.

Autonomic nerves course through pulp tissue with their sensory brethren. Clear evidence of a sympathetic adrenergic plexus has been demonstrated by Pohto and Antila (1972). Avery et al found that the highest concentration of adrenergic nerve endings were located in the pulp horns. Though the presence of adrenergic nerves in the pulp is undeniable, controversy exists regarding the presence of cholinergic parasympathetic nerves. Though Pohto and Antila (1968) found nerves containing acetylcholinesterase, a marker for parasympathetic nerves, they were unable to determine the cholinergic nature of the fibers. On the other hand, Inoue et al found evidence of both adrenergic and cholinergic nerves, asserting the existence of parasympathetic fibers in the pulp.

Sensory nerves within the pulp send messages regarding dental stimuli to the brain. Several theories of pulpal sensitivity have been suggested through the years (Nair) (Fig 3-6). The theory of direct conduction is attributed to Frank. He found nerves in the dentinal tubules and theorized that free nerve endings were responsible for sensitivity. The theory of transduction is attributed to Avery et al. They located gap junctions between nerves and odontoblasts and theorized that sensation is transduced by these structures. Berger and Byers refuted this finding when they found no evidence of gap junctions in their histologic analysis. Lastly, Brännström developed the hydrodynamic theory of dental pain. Through an elegant experiment where paper points were applied to dentin, stimulating outward fluid flow, he demonstrated that fluid flow caused painful sensations in the dental pulp.

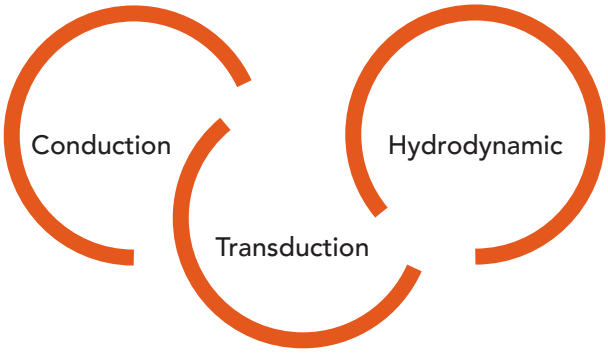

Fig 3-6 Theories of dentin sensitivity.

Dental nerves not only sense the pulpal environment but also respond to extrapulpal environmental changes. The neural influence on pulpal blood flow has been demonstrated by both histologic and physiologic experiments. The proximity of nerves to vascular structures is often offered as histologic support. Okamura et al visualized nerve endings on a large

number of arterioles, capillaries, and venules. Physiologic experiments confirmed their hypothesis. Tonder and Naess demonstrated an increase in pulpal blood flow after stimulation of the inferior alveolar nerve. Kim found the opposite in response to stimulation of sympathetic nerves. He found that administration of a β agonist, which causes vasodilation, caused a paradoxical reduction in pulpal blood flow. He theorized that the dilation of the arterioles in a low-compliance system causes compression of the venules and, hence, a reduction in blood flow. On the contrary, Tonder and Naess theorized that reduction in blood flow following the administration of a β agonist results from stealing of blood flow by adjacent tissues undergoing severe vasodilation. Figure 3-7 describes the outcomes of sensory nerve stimulation.

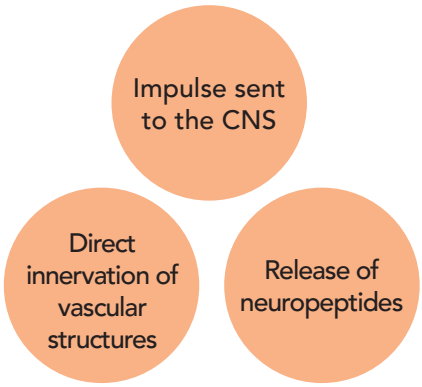

Fig 3-7 Pulpal stimulation results in impulse transmission to the central nervous system (CNS) and retrograde actions, including direct action on vascular structures and release of neuropeptides.

In addition to direct neurovascular connections, neuropeptides released from C fibers and sympathetic nerves also influence pulp vasculature. NPY, released from sympathetic nerves, causes vasoconstriction not prevented by pretreatment of the pulp with an α blocker (Edwall et al). CGRP, SP, and neurokinin A (NKA)— all released from C fibers—produce pulpal vasodilation (Caviedes-Bucheli et al 2006). The initial component of the vasodilatory response is mediated by SP, whereas the long-lasting rise in pulpal blood pressure is mediated by CGRP (Caviedes-Bucheli et al 2008).

Intrapulpal vasculature

Contained within the extracellular connective tissue along with neural structures is the vascular network that supplies oxygen and nutrients to the dental pulp. Vascular structures contained within the pulp are similar to those seen in other parts of the body and include arteries, arterioles, capillaries, venules, and veins. According to Kramer, the majority of the arterial structures contained within the pulp tissue enter via the apical foramen. Arteries and arterioles move coronally and branch to give rise to the subodontoblastic capillary plexus. Capillary structures drain into venules that course toward larger veins contained within the central part of the pulp. Venous drainage then exits the tooth through the apical foramen. In addition to those vascular connections observed through the apical foramen, several other connection points were noted, including those between the pulp spaces and periodontal tissues via lateral canals and weblike networks between adjacent roots (Kramer).

Though pulpal vasculature is, in many ways, similar to that of systemic circulation, several unique pulpal structures have been described in the literature (Fig 3-8). Kramer observed arteriovenous shunts, and Takahashi et al located U-turn loops, cross-fence capillaries, and venous-venous anastomoses. The shunting provided by these structures is significantly greater in the apex than in the coronal pulp (Kim).

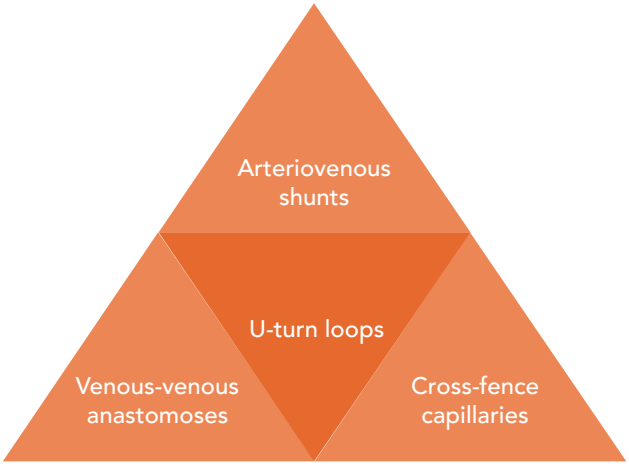

Fig 3-8 Vascular structures unique to the dental pulp.

The pulp has the highest blood flow rate per weight of tissue among all the oral tissues, and the capillary flow in the coronal portion is two times that in the root. Vascular flow is under neuropeptide controls (Kim).

Intrapulpal lymphatics

Lymphatic structures have been observed adjacent to the pulp's vascular networks and neural structures. Both histologic and physiologic studies demonstrate the existence of pulpal lymphatics. Bernick found evidence of large, valved lymphatic vessels in pulp tissue that begin as blind sacs. Matsumoto et al found the majority of these vessels in the cell-free zone beneath the odontoblast layer with a few vessels penetrating the odontoblastic layer.

Physiologic studies of fluid movement throughout pulp tissue confirm the histologic findings of pulpal lymphatics. Heyeraas found extravascular fluid drainage pathways by examining the disappearance of radioactive tracers. In a more recent paper, Oehmke et al confirmed these findings.

Changes with aging

The early dental pulp is a highly cellular and richly vascularized tissue, particularly during dentinogenesis (Avery). However, the aged pulp is more fibrotic in nature. As neural and blood vessel numbers decrease, their connective tissue sheaths persist, leading to a fibrotic appearance (Bernick and Nedelman). Furthermore, aging causes an increase in the width of predentin and cementum (Nitzan et al). As cementum thickness increases, the diameter of the major apical foramen increases, and the position of the major foramen changes with respect to the anatomical apex. However, the average width of the minor apical foramen does not change with age and remains constant at 0.189 mm (Stein and Corcoran). Figure 3-9 outlines common histologic findings associated with pulpal aging.

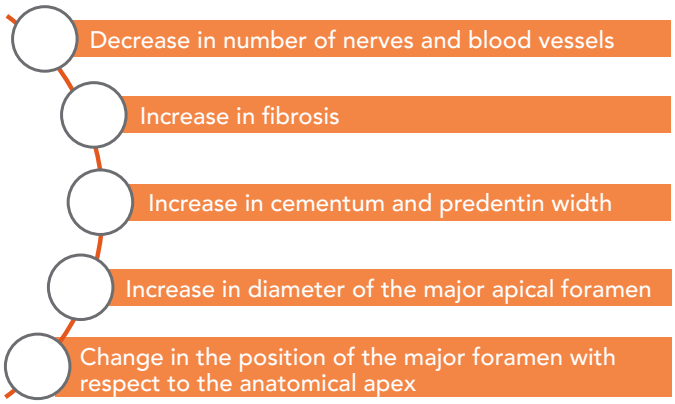

Fig 3-9 Changes seen in pulp tissues in response to aging.

Root Canal Anatomy

Canal configuration

The root canal space is a fascinating labyrinth, often reflective of the external appearance of the tooth. The cementoenamel junction (CEJ) is a consistent landmark for the location and shape of the pulp chamber (Krasner and Rankow). Furthermore, root shape predicts the number, location, and morphology of canals within the root (Bjorndal et al).

Examination of canal location and morphology uses various techniques including hematoxylin injections (Vertucci), polyester castings (Carns and Skidmore), and microscopic evaluation (Kulild and Peters). Depending on the method of study, the reported number and configuration of canal spaces varies. One of the more famous canal classification systems is that devised by Vertucci (Fig 3-10). Tables 3-1 and 3-2 display the commonly reported canal variants in maxillary teeth and mandibular teeth, respectively.

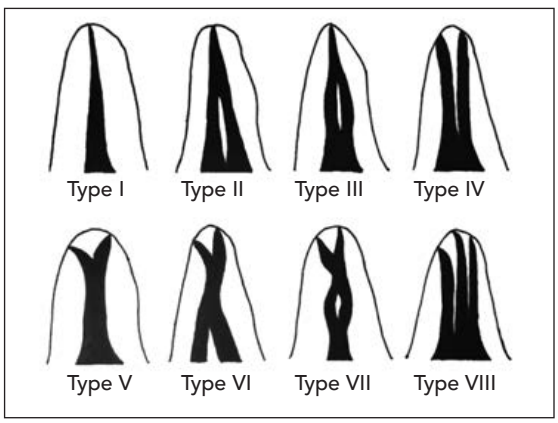

Fig 3-10 Vertucci's canal classifications in pictorial form. Type I represents a single canal. Type II indicates two canals that join at the apex. Type III begins as a single canal that splits into two canals and joins at the apex. Type IV is a root containing two canals. Type V is a single canal that bifurcates at the apex. Type VI begins as two canals, joins in the midroot area, and bifurcates at the apex. Type VII begins as a single canal, bifurcates in the midroot area, joins in the apical third, and again bifurcates. Type VIII indicates three separate canals.

Table 3-1 Maxillary root canal anatomy

Tooth	Morphology	Reference
Central incisor	1 canal 100%	Vertucci
Lateral incisor	1 canal 100%	Vertucci
Canine	1 canal 100%	Vertucci
First premolar	Root body: 1 canal 8%, 2 canals 87%, 3 canals 5% At the apex: 1 canal 26%, 2 canals 69%, 3 canals 5%	Vertucci
	Root body: 1 canal 9%, 2 canals 85%, 3 canals 6% At the apex: 1 canal 22%, 2 canals 72%, 3 canals 6%	Carns and Skidmore
	1 canal 6.5%, 2 canals 90.5%, 3 canals 3.3%	Bellizzi and Hartwell
Second premolar	Root body: 1 canal 48%, 2 canals 51%, 3 canals 1% At the apex: 1 canal 75%, 2 canals 24%, 3 canals 1%	Vertucci
	1 canal 40.3%, 2 canals 58.6%, 3 canals 1.1%	Carns and Skidmore
First molar	Root body: 3 canals 45%, 4 canals 55% At the apex: 3 canals 82%, 4 canals 18%	Vertucci
	Root body: 3 canals 48.5%, 4 canals 51.5% At the apex: 3 canals 86%, 4 canals 14%	Weine et al
	4 canals 18%	Bellizzi and Hartwell
	Root body: 3 canals 4.8%, 4 canals 95.2% At the apex: 3 canals 49.4%, 4 canals 45.8%	Kulild and Peters
Second molar	Root body: 1 canal 71%, 2 canals 29% At the apex: 1 canal 88%, 2 canals 12%	Vertucci
	4 canals 9.6%	Cecic et al
	4 canals 93.7%	Kulild and Peters

Table 3-2 Mandibular root canal anatomy

Tooth	Morphology	Reference
Central incisor	Root body: 1 canal 70%, 2 canals 30% At the apex: 1 canal 97%, 2 canals 3%	Vertucci
Lateral incisor	Root body: 1 canal 75%, 2 canals 25% At the apex: 1 canal 98%, 2 canals 2%	Vertucci
Incisors	Root body: 1 canal 89%, 2 canals 11% At the apex: 1 canal 99.5%, 2 canals 0.5%	Madeira and Hetem
	Root body: 1 canal 59%, 2 canals 41% At the apex: 1 canal 98.7%, 2 canals 1.3%	Benjamin and Dowson
Canine	Root body: 1 canal 78%, 2 canals 22% At the apex: 1 canal 94%, 2 canals 6%	Vertucci
First premolar	Root body: 1 canal 70%, 2 canals 29.5%, 3 canals 0.5% At the apex: 1 canal 74%, 2 canals 25.5%, 3 canals 0.5%	Vertucci
Second premolar	Root body: 1 canal 97.5%, 2 canals 2.5% At the apex: 1 canal 97.5%, 2 canals 2.5%	Vertucci
First molar	Mesial root body: 1 canal 12%, 2 canals 87%, 3 canals 1% Mesial root apex: 1 canal 40%, 2 canals 59%, 3 canals 1% Distal root body: 1 canal 70%, 2 canals 30% Distal root apex: 1 canal 85%, 2 canals 15%	Vertucci
	4 canals 35.1%	Hartwell and Bellizzi
Second molar	Mesial root body: 1 canal 27%, 2 canals 73% Mesial root apex: 1 canal 65%, 2 canals 35% Distal root body: 1 canal 92%, 2 canals 8% Distal root apex: 1 canal 95%, 2 canals 5%	Vertucci
	4 canals 5.5%	Hartwell and Bellizzi

Apical anatomy

The anatomy of the root apex includes the minor apical foramen—otherwise known as the *cementodentinal junction* (CDJ)—and the major apical foramen. According to Burch and Hulen, 92.4% of the major foramina of all classes of teeth open short of the anatomical apex. Furthermore, the average distance between the foramen and the anatomical root apex is 0.59 mm. Kuttler's work also demonstrated that the apical foramen is often not coincident with the root apex. He found that the average distance between the center of

the foramen and the apical center was 495 μm. A recent study by El Ayouti et al found that the distance between the apical foramen and the apex was, on average, 0.9 mm when evaluated by cone beam computed tomography (CBCT).

Just as the apical foramen is often not coincident with the root apex, the CDJ is often not flush with the apical foramen. Stein and Corcoran found that the average distance between the major and minor foramina was 0.724 mm, and the width of the CDJ remained constant with age at 0.189 mm. El Ayouti et al measured the mean distance between the apical constriction and apical foramen on CBCT scans at 0.2 mm. Interestingly, not all roots necessarily exhibit the traditionally defined apical constriction. Meder-Cowherd et al were unable to locate a defined apical constriction in 65% of the palatal roots of maxillary first molars evaluated by CBCT scans.

Accessory canals

Arborizing from the main canal system are lateral, secondary, and accessory canals (Fig 3-11). Gutmann theorized that these structures form via localized breaks in HERS around blood vessel communications. Accessory canals are defined by their location. De Deus defined a *lateral canal* as one extending from the main canal to the periodontal ligament (PDL) in the body of the root, a *secondary canal* as one extending from the main canal to the PDL in the apical region, and an *accessory canal* as one derived from the secondary canal branching toward the PDL in the apical region.

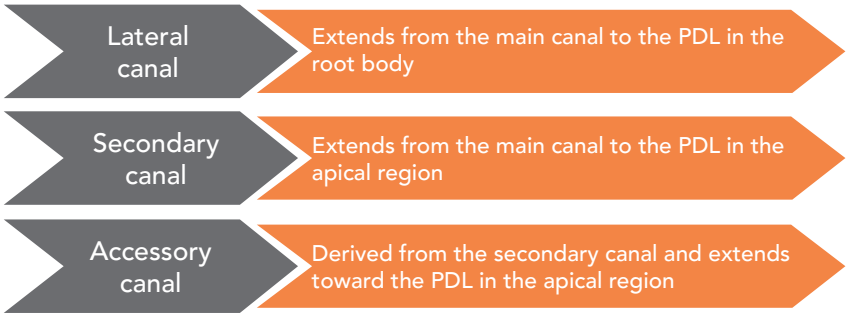

Fig 3-11 De Deus' accessory canal definitions. PDL, periodontal ligament.

The prevalence of accessory anatomy has been widely reported in the literature, and research confirms the most common location for lateral anatomy is in the apical third (De Deus, Vertucci). Rubach and Mitchell found lateral anatomy in 45% of teeth. De Deus found additional anatomy in 27.4% of teeth and 17% found in the apical third. Several authors have noted accessory canals in the furcation area of molars in addition to the more commonly found apical variants. Koenigs et al described the appearance of diametrically varying accessory foramina in the furcation region under electron microscopy. Their reported prevalence varies between Gutmann's 28% and Vertucci and Williams' 46%, depending on the method of study.

Unique anatomical variants

Though dental anatomy is often predictable, the endodontic literature is replete with examples of unique anatomical variants, including the dens invaginatus (DI), dens evaginatus (DE), and C-shaped canal systems. Furthermore, case reports are often published containing other unique anatomical presentations. These variants underscore the need for careful preoperative and intraoperative inspection of pulpal anatomy.

The DI forms via infolding of enamel and dentin into the root canal space. Narayana et al proposed several potential etiologic factors including trauma, infection, growth retardation of specific cells, disruption in the regulation of the enamel organ, and genetic predisposition. Hovland and Block reported an incidence of between 0.4% and 10% in the general population. Rotstein et al found that 42% of DI cases occurred in lateral incisors, and Hulsmann found bilateral presentation 43% of the time.

Oehlers (1957a, 1957b, 1958) classified DI severity by the extent of penetration into the root canal space. Type I represents a minor extension into the root canal space, type II extends into the root and may perforate the pulp tissue, and type III penetrates to the apex and may have its own apical foramen (Fig 3-12). Due to its proximity to pulpal structures, early pulpal involvement is often seen with DI. Consequently, early preventive treatment is often recommended (Rotstein et al). For those teeth with DI that become pulpally involved, creative treatment modalities are often employed, such as that described by Narayana et al utilizing CBCT evaluations and regenerative endodontic therapy.

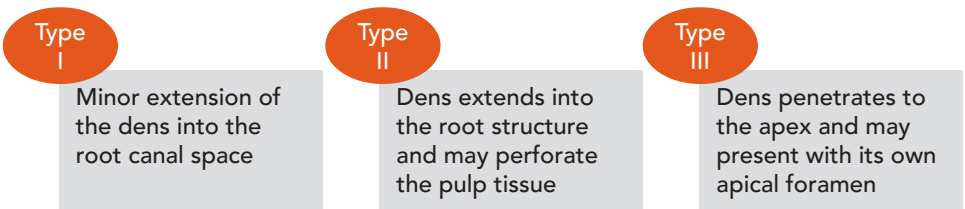

Fig 3-12 Oehler's classification of DI.

DE forms from an out-pouching of enamel and dentin onto the occlusal or lingual surfaces of the dentition (Levitan and Himel). The remaining portion of the clinical crown of teeth affected by DE is often normal in appearance. The variant can be found on any tooth but is most commonly observed on premolars and often presents bilaterally. If found on anterior teeth, it is frequently seen on the lingual surface and has been described as a *talon cusp* (Levitan and Himel). According to Oehlers (1957a, 1957b, 1958), 70% of these dens structures contain pulp horns, which can fracture during function, thus leading to early pulpal involvement. Treatment for teeth affected by DE depends on the pulpal diagnosis and stage of root development (Levitan and Himel).

C-shaped canals have been described in mandibular and maxillary molars and mandibular premolars. They present with large, interconnected tissue spaces and are often difficult to diagnose by two-dimensional radiographs alone. Although the majority of these teeth exhibit fused roots, C-shaped canals have also been noted in nonfused roots (Lu et al).

They have been observed in as many as 8% of mandibular second molars when examined clinically (Cooke and Cox). More recently, Sinanoglu and Helvacioglu-Yigit showed that C-shaped canals can be recognized by CBCT. Commonly noted pulp chamber anatomy in C-shaped molars is described in Fig 3-13.

In addition to those canal systems reported in mandibular molars, Lu et al located the anomaly in 18% of mandibular premolars in a Chinese population, and Newton and McDonald reported a case in a maxillary molar. In mandibular molars, Min et al reported several clinical variants of the C-shaped canal, including the continuous C-shaped orifice, a C-shaped mesial orifice with a separate distal canal, a continuous mesiobuccal-distal orifice with a separate mesiolingual orifice, and a non-C-shaped pulpal floor. Caution must be exercised in the endodontic treatment of teeth with a C-shaped canal because of the presence of thin canal walls (Gu et al).

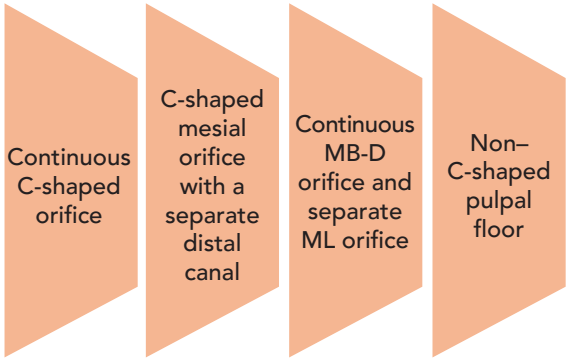

Fig 3-13 A description of the possible pulp chamber appearance in C-shaped mandibular molars (Min et al). MB-D, mesiobuccal-distal; ML, mesiolingual.

Maxillofacial Anatomy

The maxillofacial region is a richly innervated and vascularized area. This section provides a brief discussion of several anatomical features germane to endodontic treatment. For a more complete anatomical text, please refer to Moore et al's *Clinically Oriented Anatomy* or Fehrenbach and Herring's *Illustrated Anatomy of the Head and Neck*.

Arterial supply to the dentition

Oxygenated blood leaves the lungs via the pulmonary vein and enters the left atrium of the heart. It then travels into the left ventricle and into the aorta (Moore et al). On the right side of the body, the aorta branches to the brachiocephalic artery that then becomes the common carotid. However, on the left side of the body, the common carotid is a branch of the aorta. The common carotid branches into the internal and external carotid arteries at the carotid sinus. The internal carotid artery enters the base of the skull and supplies blood to the cranium. The external carotid artery supplies the remaining facial structures. Several branches of the external carotid artery supply oral structures, including the lingual artery, which provides vascularity to the floor of the mouth, sublingual salivary glands, and tongue, and the facial artery, which supplies the lips (Fehrenbach and Herring).

The external carotid artery possesses two terminal branches: the superficial temporal artery and the maxillary artery. The maxillary artery provides vascularity to the dentition. To supply the mandibular dentition, the maxillary artery branches to the inferior alveolar ar-

tery, which then branches to the mental and incisive arteries to supply the mandibular teeth and gingiva. In the maxilla, the maxillary artery gives rise to the posterior superior alveolar artery, which supplies the posterior teeth, and the infraorbital artery, which proceeds anteriorly. The anterior superior alveolar artery is a branch of the infraorbital artery and supplies the premolar and incisor teeth. The maxillary artery also provides vascularity to the hard palate via the descending palatine and greater and lesser palatine arteries (Fehrenbach and Herring). Blood flow pathways to the dentition are outlined in Fig 3-14.

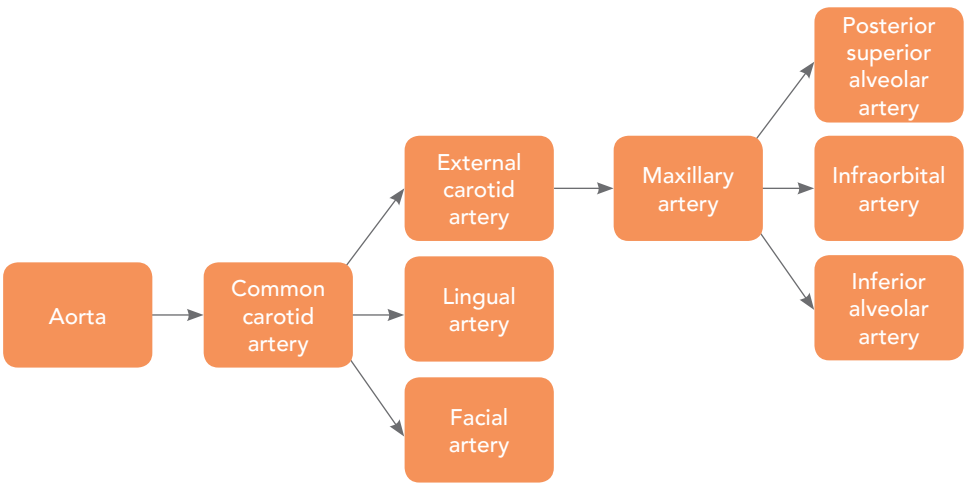

Fig 3-14 The path of blood flow from its entrance to the aorta from the left ventricle to the terminal dental branches (Fehrenbach and Herring).

Venous drainage from the alveolus

Venous drainage from the maxillary teeth and gingiva starts in the posterior superior alveolar vein. This vein enters the pterygoid venous plexus, which also communicates with the facial vein. The pterygoid plexus drains into the maxillary vein. Venous drainage from the mandibular teeth comes from the inferior alveolar vein, which, like the pterygoid plexus, drains into the maxillary vein. The maxillary vein joins with the superficial temporal vein to create the retromandibular vein. The anterior portion of the retromandibular vein enters into the internal jugular vein. The posterior portion of the retromandibular vein becomes the external jugular vein. The external jugular vein enters the subclavian vein, and then the internal jugular vein merges with the subclavian vein to form the brachiocephalic vein. The right and left brachiocephalic veins join to form the superior vena cava, which enters the right atrium. Blood then travels from the right atrium to the right ventricle into the pulmonary artery and the lungs (Fehrenbach and Herring).

The veins of the face are valveless and can occasionally provide retrograde blood flow into the calvaria. The ophthalmic vein, which drains tissues of the orbit, provides a pathway for infections in the infraorbital space to seed intracranial infections in the cavernous sinus (Moore et al).

Lymphatics

Lymphatic drainage from the head and neck progresses through several lymphatic vessels and nodes. Lymph nodes that may become enlarged due to dental infections include the submandibular, submental, and superior deep cervical lymph nodes primarily, and both the superior and inferior deep cervical lymph nodes secondarily (Fehrenbach and Herring). Lymph nodes often involved in endodontic infections are listed in Fig 3-15.

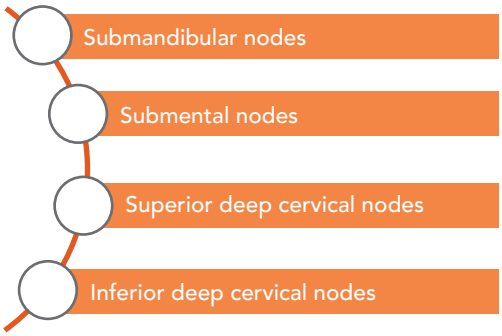

Fig 3-15 Lymph nodes that may become involved in dental infections (Fehrenbach and Herring).

Lymphatic drainage from the right side of the head proceeds through the jugular trunk, joining lymphatics from the right arm to form the right lymphatic duct, which enters the venous system at the junction of the right subclavian vein and internal jugular vein. Lymphatic drainage from the left side of the head proceeds through the right jugular trunk and into the thoracic duct. The thoracic duct then enters the venous system at the junction of the left subclavian and internal jugular veins (Fehrenbach and Herring).

Neuroanatomy

Sensation to the dentition and surrounding structures and motor function to the muscles of mastication arise from the trigeminal nerve (Fig 3-16). This structure originates in the middle cranial fossa and shortly thereafter forms the trigeminal ganglion. Sensation to the maxillary teeth comes from the second branch of the trigeminal nerve, namely the maxillary nerve. This nerve exits through foramen rotundum and then provides sensory branches to the teeth, including the posterior superior alveolar nerve, which supplies the molars; the middle superior alveolar nerve, which supplies the mesiobuccal root of the first molar and the premolars; and the anterior superior alveolar nerve, which supplies the anterior teeth. The maxillary nerve also provides sensation to the palate via the greater and lesser palatine nerves (Fehrenbach and Herring).

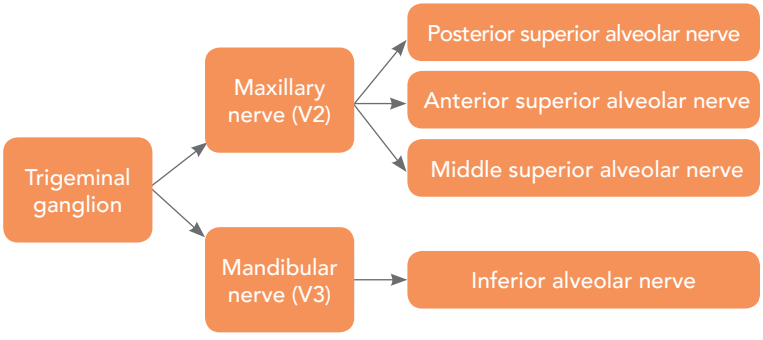

Fig 3-16 Neural pathways to the dentition (Fehrenbach and Herring).

Sensation to the mandibular teeth comes from the third branch of the trigeminal nerve, namely the mandibular nerve. This nerve exits through foramen ovale and progresses anteriorly as the inferior alveolar nerve, which supplies sensation to the mandibular teeth. Its terminal branches are the incisive nerve, which supplies sensation to the mandibular anterior teeth, and the mental nerve, which supplies sensation to the chin, lower lip, and facial gingiva. The long buccal nerve, another branch of the mandibular nerve, supplies sensation to the buccal gingiva of the posterior teeth. The lingual nerve, a branch of the mandibular nerve, supplies sensation to the lingual gingiva and tongue (Fehrenbach and Herring). According to Pogrel et al, the mandibular anterior teeth may experience cross innervation. Frommer et al asserted that additional sensation to the mandibular molars may be provided by the mylohyoid nerve, a branch of the mandibular nerve.

Noxious stimuli are sensed by Aδ and C fibers, primary afferent nerves with their cell bodies contained in the trigeminal ganglion. These fibers synapse on the subnucleus caudalis in the medullary dorsal horn. Second order projection neurons cross the midline to the thalamus via the trigeminothalamic tract. Third order neurons then travel to the cerebral cortex via the thalamocortical tract (Moore et al).

Fascial spaces

For a discussion of fascial spaces germane to endodontic infections, please refer to chapter 2.

Surgical anatomy

The endodontic literature contains numerous references to important anatomical landmarks encountered during surgical interventions, including the maxillary sinus, the inferior alveolar nerve, and the mental foramen. Recent research underscores the importance of obtaining accurate three-dimensional imaging by CBCT prior to surgery to precisely visualize the proximity of vital structures to the dentition. The maxillary sinus must be considered when root-end surgery is planned for maxillary teeth. Eberhardt et al found that the proximity of root tips to the sinus increased when moving from the maxillary premolars posteriorly to the molars. Second molars were found closest to the sinus. While Von Arx et al found that root protrusion into the maxillary sinus was uncommon in maxillary premolars, Pagin et al found that 14% of the roots of posterior maxillary teeth penetrate the sinus floor, the largest percentage of which were second molars.

If surgical intervention is planned for mandibular teeth, both the inferior alveolar nerve canal and the mental foramen must be located to avoid injury. Denio et al found that the inferior alveolar canal was S-shaped in 31% of cases; lingual, buccal, or inferior to the molar roots in 41% of cases; and could not be located precisely in 28% of cases. When evaluated by CBCT, Kovisto et al found that the inferior alveolar nerve canal was closest to the roots of the second molar and was closer to the root apices in younger patients than in older patients.

The mental foramen, which provides exit for the terminal branch of the inferior alveolar nerve, is most often located between the roots of the first and second premolars (Moiseiwitsch). Phillips et al found that it is often located two-thirds of the distance from the buccal cusp tips of the premolars to the inferior border of the mandible. Furthermore, the nerve itself exited in a posterosuperior direction in two-thirds of cases. Aminoshariae et al found that the best technique to accurately locate the mental foramen prior to surgical intervention was a CBCT scan.

Bibliography

Embryology

Alatli I, Lundmark C, Hammarstrom L. The localization of epithelial root sheath cells during cementum formation in rat molars. J Periodontal Res 1996;31:433–440.

Diamond M, Applebaum E. The epithelial sheath: Histogenesis and function. J Dent Res 1942;21:403–411.

Freeman E, Ten Cate AR. Development of the periodontium: An electron microscopic study. J Periodontol 1971;42:387–395.

Fristad I, Heyeraas K, Kvinnsland I. Nerve fibres and cells immunoreactive to neurochemical markers in developing rat molars and supporting tissues. Arch Oral Biol 1994;39:633–646.

Jernvall J, Thesleff I. Reiterative signaling and patterning during mammalian tooth morphogenesis. Mech Dev 2000;92:19–29.

Kapadia H, Mues G, D'Souza R. Genes affecting tooth morphogenesis. Orthod Craniofac Res 2007;10:237–244.

Martin A, Unda F, Begue-Kirn C, Ruch J, Arechaga J. Effects of aFGF, bFGF, TGFbeta1 and IGF-I on odontoblast differentiation in vitro. Eur J Oral Sci 1998;106(suppl 1):117–121.

Thesleff I. The genetic basis of tooth development and dental defects. Am J Med Genet A 2006;140:2530–2535.

Anatomy and Physiology of Dental Structures

Avery JK, Cox CF, Chiego DJ Jr. Presence and location of adrenergic nerve endings in the dental pulps of mouse molars. Anat Rec 1980;198:59–71.

Avery JK. Structural elements of the young normal human pulp. Oral Surg Oral Med Oral Pathol 1971;32:113–125.

Berger RL, Byers MR. Dental nerve regeneration in rats. II. Autoradiographic studies of nerve regeneration to molar pulp and dentin. Pain 1983;15:359–375.

Bernick S. Lymphatic vessels of the human dental pulp. J Dent Res 1977;56:70–77.

Bernick S, Nedelman C. Effect of aging on the human pulp. J Endod 1975;1:88–94.

Brännström M. The hydrodynamic theory of dentinal pain: Sensation in preparations, caries, and the dentinal crack syndrome. J Endod 1986;12:453–457.

Butler WT, Ritchie H. The nature and functional significance of dentin extracellular matrix proteins. Int J Dev Biol 1995;39:169–179.

Byers MR, Sugaya A. Odontoblast processes in dentin revealed by fluorescent Di-I. J Histochem Cytochem 1995;43:159–168.

Byers MR, Taylor PE, Khayat BG, Kimberly CL. Effects of injury and inflammation on pulpal and periapical nerves. J Endod 1990;16:78–84.

Caviedes-Bucheli J, Lombana N, Azuero-Holguin MM, Munoz HR. Quantification of neuropeptides (calcitonin gene-related peptide, substance P, neurokinin A, neuropeptide Y and vasoactive intestinal polypeptide) expressed in healthy and inflamed human dental pulp. Int Endod J 2006;39:394–400.

Caviedes-Bucheli J, Munoz HR, Azuero-Holguin MM, Ulate E. Neuropeptides in dental pulp: The silent protagonists. J Endod 2008;34:773–788.

Dai XF, Ten Cate A, Limeback H. The extent and distribution of intratubular collagen fibrils in human dentine. Arch Oral Biol 1991;36:775–778.

Edwall B, Gazelius B, Fazekas A, Theodorsson-Norheim E, Lundberg JM. Neuropeptide Y (NPY) and sympathetic control of blood flow in oral mucosa and dental pulp in the cat. Acta Physiol Scand 1985;125:253–264.

Ferraz CCR, Henry MA, Hargreaves KM, Diogenes A. Lipopolysaccharide from *Porphyromonas gingivalis* sensitizes capsaicin-sensitive nociceptors. J Endod 2011;37:45–48.

Frank RM. Electron microscopic study on odontoblasts and canaliculi in human dentin [in French]. Arch Oral Biol 1966;11:179–199.

Garberoglio RB, Brännström M. Scanning electron microscopic investigation of human dentinal tubules. Arch Oral Biol 1976;21:355–362.

Harris R, Griffin CJ. Histogenesis of fibroblasts in the human dental pulp. Arch Oral Biol 1967;12:459–468.

Heyeraas KJ. Pulpal hemodynamics and interstitial fluid pressure: Balance of transmicrovascular fluid transport. J Endod 1989;15:468–472.

Holland GR. The odontoblast process: Form and function. J Dent Res 1985;64:499–514.

Inoue H, Kurosaka Y, Abe K. Autonomic nerve endings in the odontoblast/predentin border and predentin of the canine teeth of dogs. J Endod 1992;18:149–151.

Kim S. Microcirculation of the dental pulp in health and disease. J Endod 1985;11:465–471.

Kinney JH, Marshall S, Marshall GW. The mechanical properties of human dentin: A critical review and re-evaluation of the dental literature. Crit Rev Oral Biol Med 2003;14:13–29.

Kramer IR. The vascular architecture of the human dental pulp. Arch Oral Biol 1960;2:177–189.

Kuttler Y. Classification of dentine into primary, secondary, and tertiary. Oral Surg Oral Med Oral Pathol 1959;12:996–999.

Kuttler Y. Microscopic investigation of root apexes. J Am Dent Assoc 1955;50:544–552.

Linde A. The extracellular matrix of the dental pulp and dentin. J Dent Res 1985;64:523–529.

Matsumoto Y, Kato S, Miura M, Yanagisawa S, Shimizu M. Fine structure and distribution of lymphatic vessels in the human dental pulp: A study using an enzyme-histochemical method. Cell Tissue Res 1997;288:79–85.

Mjor IA, Sveen OB, Heyeraas KJ. Pulp-dentin biology in restorative dentistry. Part 1: Normal structure and physiology. Quintessence Int 2001;32:427–446.

Nair PN. Neural elements in dental pulp and dentin. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;80:710–719.

Nitzan DW, Michaeli Y, Weinreb M, Azaz B. The effect of aging on tooth morphology: A study on impacted teeth. Oral Surg Oral Med Oral Pathol 1986;61:54–60.

Oehmke MJ, Knolle E, Oehmke HJ. Lymph drainage in the human dental pulp. Microsc Res Tech 2003;62:187–191.

Okamura K, Kobayashi I, Matsuo K, et al. An immunohistochemical and ultrastructural study of vasomotor nerves in the microvasculature of human dental pulp. Arch Oral Biol 1995;40: 47–53.

Olgart L, Kerezoudis NP. Nerve-pulp interactions. Arch Oral Biol 1994;39(suppl):47S–54S.

Pashley DH. Dynamics of the pulpo-dentin complex. Crit Rev Oral Biol Med 1996;7:104–133.

Pohto P, Antila R. Acetylcholinesterase and noradrenaline in the nerves of mammalian dental pulps. Acta Odontol Scand 1968;26:641–656.

Pohto P, Antila R. Innervation of blood vessels in the dental pulp. Int Dent J 1972;22:228–239.

Rapp R, Avery JK, Strachan DS. The distribution of nerves in human primary teeth. Anat Rec 1967;159:89–103.

Reeves R, Stanley HR. The relationship of bacterial penetration and pulpal pathosis in carious teeth. Oral Surg Oral Med Oral Pathol 1966;22:59–65.

Ruch JV. Odontoblast commitment and differentiation. Biochem Cell Biol 1998;76:923–938.

Scott JN, Weber DF. Microscopy of the junctional region between human coronal primary and secondary dentine. J Morphol 1977;154:133–145.

Shuttleworth CA, Ward JL, Hirschmann PN. The presence of type III collagen in the developing tooth. Biochim Biophys Acta 1978;535:348–355.

Stein TJ, Corcoran JF. Anatomy of the root apex and its histologic changes with age. Oral Surg Oral Med Oral Pathol 1990;69:238–242.

Takahashi K, Kishi Y, Kim S. A scanning electron microscope study of the blood vessels of dog pulp using corrosion resin casts. J Endod 1982;8:131–135.

Thesleff I. The genetic basis of tooth development and dental defects. Am J Med Genet A 2006;140:2530–2535.

Tonder KH, Naess G. Nervous control of blood flow in the dental pulp in dogs. Acta Physiol Scand 1978;104:13–23.

Yamada T, Nakamura K, Iwaku M, Fusayama T. The extent of the odontoblast process in normal and carious human dentin. J Dent Res 1983;62:798–802.

Root Canal Anatomy

Bellizzi R, Hartwell G. Radiographic evaluation of root canal anatomy of in vivo endodontically treated maxillary premolars. J Endod 1985;11:37–39.

Benjamin KA, Dowson J. Incidence of two root canals in human mandibular incisor teeth. Oral Surg Oral Med Oral Pathol 1974;38:122–126.

Bjorndal L, Carlsen O, Thuesen G, Darvann T, Kreiborg S. External and internal macromorphology in 3D-reconstructed maxillary molars using computerized X-ray microtomography. Int Endod J 1999;32:3–9.

Burch JG, Hulen S. The relationship of the apical foramen to the anatomic apex of the tooth root. Oral Surg Oral Med Oral Pathol 1972;34:262–268.

Carns EJ, Skidmore AE. Configurations and deviations of root canals of maxillary first premolars. Oral Surg Oral Med Oral Pathol 1973;36:880–886.

Cecic P, Hartwell G, Bellizzi R. The multiple root canal system in the maxillary first molar: A case report. J Endod 1982;8:113–115.

Cooke HG 3rd, Cox FL. C-shaped canal configurations in mandibular molars. J Am Dent Assoc 1979;99:836–839.

De Deus QD. Frequency, location, and direction of the lateral, secondary, and accessory canals. J Endod 1975;1:361–366.

El Ayouti A, Hulber JM, Judenhofer MS, et al. Apical constriction: Location and dimensions in molars—A micro-computed tomography study. J Endod 2014;40:1095–1099.

Gu YC, Zhang YP, Liao ZG, Fei XD. A micro-computed tomographic analysis of wall thickness of C-shaped canals in mandibular first premolars. J Endod 2013;39:973–976.

Gutmann JL. Prevalence, location, and patency of accessory canals in the furcation region of permanent molars. J Periodontol 1978;49:21–26.

Hartwell G, Bellizzi R. Clinical investigation of in vivo endodontically treated mandibular and maxillary molars. J Endod 1982;8:555–557.

Hovland EJ, Block RM. Nonrecognition and subsequent endodontic treatment of dens invaginatus. J Endod 1977;3:360–362.

Hulsmann M. Dens invaginatus: Aetiology, classification, prevalence, diagnosis, and treatment considerations. Int Endod J 1997;30:79–90.

Koenigs JF, Brilliant JD, Foreman DW. Preliminary scanning electron microscope investigations of accessory foramina in the furcation areas of human molar teeth. Oral Surg Oral Med Oral Pathol 1974;38:773–782.

Krasner P, Rankow HJ. Anatomy of the pulp-chamber floor. J Endod 2004;30:5–16.

Kulild JC, Peters DD. Incidence and configuration of canal systems in the mesiobuccal root of maxillary first and second molars. J Endod 1990;16:311–317.

Kuttler Y. Microscopic investigation of root apexes. J Am Dent Assoc 1955;50:544–552.

Levitan ME, Himel VT. Dens evaginatus: Literature review, pathophysiology, and comprehensive treatment regimen. J Endod 2006;32:1–9.

Lu TY, Yang SF, Pai SF. Complicated root canal morphology of mandibular first premolar in a Chinese population using the cross section method. J Endod 2006;32:932–936.

Madeira MC, Hetem S. Incidence of bifurcations in mandibular incisors. Oral Surg Oral Med Oral Pathol 1973;36:589–591.

Meder-Cowherd L, Williamson AE, Johnson WT, Vasilescu D, Walton R, Qian F. Apical morphology of the palatal roots of maxillary molars by using micro-computed tomography. J Endod 2011;37:1162–1165.

Min Y, Fan B, Cheung GS, Gutmann JL, Fan M. C-shaped canal system in mandibular second molars. Part III: The morphology of the pulp chamber floor. J Endod 2006;32:1155–1159.

Narayana P, Hartwell GR, Wallace R, Nair UP. Endodontic clinical management of a dens invaginatus case by using a unique treatment approach: A case report. J Endod 2012;38:1145–1148.

Newton CW, McDonald S. A C-shaped canal configuration in a maxillary first molar. J Endod 1984;10:397–399.

Oehlers FA. Dens invaginatus (dilated composite odontome). I. Variations of the invagination process and associated anterior crown forms. Oral Surg Oral Med Oral Pathol 1957a;10: 1204–1218 contd.

Oehlers FA. Dens invaginatus (dilated composite odontome). II. Associated posterior crown forms and pathogenesis. Oral Surg Oral Med Oral Pathol 1957b;10:1302–1316.

Oehlers FA. The radicular variety of dens invaginatus. Oral Surg Oral Med Oral Pathol 1958;11:1251–1260.

Rotstein I, Stabholz A, Heling I, Friedman S. Clinical considerations in the treatment of dens invaginatus. Endod Dent Traumatol 1987;3:249–254.

Rubach WC, Mitchell DF. Periodontal disease, accessory canals and pulp pathosis. J Periodontol 1965;36:34–38.

Sinanoglu A, Helvacioglu-Yigit D. Analysis of C-shaped canals by panoramic radiography and cone-beam computed tomography: Root-type specificity by longitudinal distribution. J Endod 2014;40:917–921.

Stein TJ, Corcoran JF. Anatomy of the root apex and its histologic changes with age. Oral Surg Oral Med Oral Pathol 1990;69:238–242.

Vertucci FJ. Root canal anatomy of the human permanent teeth. Oral Surg Oral Med Oral Pathol 1984;58:589–599.

Vertucci FJ, Gegauff A. Root canal morphology of the maxillary first premolar. J Am Dent Assoc 1979;99:194–198.

Vertucci FJ, Williams RG. Furcation canals in the human mandibular first molar. Oral Surg Oral Med Oral Pathol 1974;38:308–314.

Weine FS, Healey HJ, Gerstein H, Evanson L. Canal configuration in the mesiobuccal root of the maxillary first molar and its endodontic significance. Oral Surg Oral Med Oral Pathol 1969;28:419–425.

Maxillofacial Anatomy

Aminoshariae A, Su A, Kulild JC. Determination of the location of the mental foramen: A critical review. J Endod 2014;40:471-475.

Cooke HG 3rd, Cox FL. C-shaped canal configurations in mandibular molars. J Am Dent Assoc 1979;99:836–839.

Denio D, Torabinejad M, Bakland LK. Anatomical relationship of the mandibular canal to its surrounding structures in mature mandibles. J Endod 1992;18:161–165.

Eberhardt JA, Torabinejad M, Christiansen EL. A computed tomographic study of the distances between the maxillary sinus floor and the apices of the maxillary posterior teeth. Oral Surg Oral Med Oral Pathol 1992;73:345–346.

Fehrenbach MJ, Herring SW. Illustrated Anatomy of the Head and Neck, ed 4. St Louis: Saunders, 2012.

Frommer J, Mele FA, Monroe CW. The possible role of the mylohyoid nerve in mandibular posterior tooth sensation. J Am Dent Assoc 1972;85:113–117.

Kovisto T, Ahmad M, Bowles WR. Proximity of the mandibular canal to the tooth apex. J Endod 2011;37:311–315.

Moiseiwitsch JR. Avoiding the mental foramen during periapical surgery. J Endod 1995;21: 340–342.

Moore KL, Dalley AF, Agur AMR. Clinically Oriented Anatomy. Philadelphia: Lippincott Williams & Wilkins, 2006.

Pagin O, Centurion BS, Rubira-Bullen IR, Alvares Capelozza AL. Maxillary sinus and posterior teeth: Accessing close relationship by cone-beam computed tomographic scanning in a Brazilian population. J Endod 2013;39:748–751.

Phillips JL, Weller RN, Kulild JC. The mental foramen: 1. Size, orientation, and positional relationship to the mandibular second premolar. J Endod 1990;16:221–223.

Pogrel MA, Smith R, Ahani R. Innervation of the mandibular incisors by the mental nerve. J Oral Maxillofac Surg 1997;55:961–963.

Von Arx T, Fodich I, Bornstein MM. Proximity of premolar roots to maxillary sinus: A radiographic survey using cone-beam computed tomography. J Endod 2014;40:1541–1548.

4

Pulpal and Periapical Pathology

The dental pulp, if left undisturbed, maintains normal anatomical structure and physiology. However, various factors irritate pulpal tissues and lead to the development of pulpal and periapical disease. This chapter discusses both the causes of endodontic pathology and characteristic pulpal and periapical responses.

Pulpal Irritants

Caries

Bacteria and their byproducts have the single greatest influence on pulpal and periapical disease. Dental caries results from the bacterial consumption of fermentable sugars, namely by *Streptococcus mutans* and *Lactobacillus* (Drucker). The byproduct of fermentation is acid, which dissolves dental hard tissues, resulting in a caries lesion (Fig 4-1). Caries is a well-documented causative factor for pulpal inflammation. Mjor and Tronstad reproducibly induced severe pulpal inflammation in monkeys by inserting human carious dentin into class V cavity preparations.

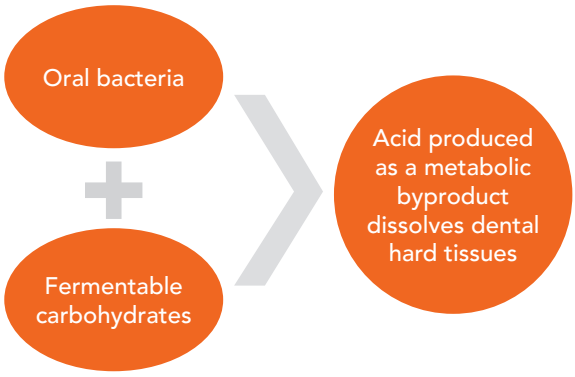

Fig 4-1 The interactions responsible for the development of a carious lesion.

The depth of caries penetration into dentin is directly associated with the severity of pulpal pathology observed both histologically and clinically. Reeves and Stanley correlated pulpal responses with depth of bacterial penetration. Mild chronic inflammation was evident once bacteria reached 1.1 mm from the pulp, whereas severe acute inflammation was observed when bacteria reached within 0.5 mm of the pulp tissue. Mitchell and Tarplee correlated these histologic findings with clinical responses when they noted histologic pulp exposures in patients with a history of spontaneous pain and radiographically detectable caries.

Fractures

Fractures are associated with pathology in vital teeth and also present a means for bacterial ingress into the pulp space. In his paper on cracked tooth syndrome, Cameron reported that coronal fractures are associated with both chewing discomfort and unexplained thermal sensitivity. This sensitivity, according to Turp and Gobetti, likely results from fluid movement within the crack causing pulpal stimulation. Fractures also provide bacteria with a direct route into pulp tissues. In a histologic study, Walton et al visualized bacteria in fractured specimens extending from the external tooth surface to the pulp spaces. This ingress may explain the development of pulp necrosis and apical periodontitis in otherwise intact traumatized teeth (Robertson et al).

Restorative treatment

Treatments aimed at eliminating caries and splinting coronal fractures have a marked effect on pulp tissues, and these effects appear to be cumulative over a lifetime. Abou-Rass described these cumulative effects as *stressed pulp syndrome*. This explains the finding that restorative treatment can cause pulpal irritation not only when treatment is initially rendered but also many years later.

Histologically, tooth preparation has a profound but reversible effect on the dental pulp. Hamilton and Kramer found that dry cavity preparation injured pulp tissues more than wet cavity preparation. Furthermore, prolonged dentin dehydration increased pulpal inflamma-

tion and caused aspiration of odontoblastic nuclei into the dentinal tubules. Despite the presence of these immediate changes, Cotton and Siegel found that both displacement and inflammation are reversible after 180 days.

The depth of restorative preparations is as influential, if not more so, than the use of coolants. Murray et al found that a remaining dentin thickness of 0.5 mm or greater is necessary to avoid evidence of pulpal injury. Deeper restorative preparations decreased odontoblast numbers. He observed reparative dentin formation following pulp exposures and reactionary dentin formation with 0.77 mm of remaining dentin thickness.

Like tooth preparation, restorative materials influence pulp tissues. These effects are depth dependent. Without pulp exposures in deep caries lesions, Tronstad and Mjor found that zinc oxide and eugenol or calcium hydroxide liners promoted pulp tissue healing. With deeper preparations, however, more serious pulpal reactions may occur. Stanley found that any material placed within 0.5 mm of the pulp caused significant and irreversible lesions. Materials placed directly on exposed pulp tissues may have additional effects. Both resin-modified glass ionomers (Tarim et al) and mineral trioxide aggregate (MTA) (Menezes et al) may promote pulpal healing when placed in direct contact with tissues. Less favorable responses are noted when pulp tissues are capped with composite (Schuurs et al).

The macro-effects of restorative treatment on pulp tissues can be measured by the frequency of endodontic pathology in restored teeth. The reported percentage of teeth requiring endodontic treatment following restorative care varies based on the type of restoration placed. In a recent study, Kwang et al reported that teeth restored with resin restorations were 1.9 times more likely to require endodontic intervention than those restored with amalgam, and 5.6 times more likely than crowned teeth. However, Dawson et al found a significantly higher prevalence of apical periodontitis in crowned teeth and no difference between teeth restored with composite or amalgam.

The percentage of crowned teeth requiring endodontic therapy is low based on literature reports (Fig 4-2). Valderhaug et al reported that only 8% of crowned vital teeth required root canal therapy after 10 years. After 25 years, 17% of crowned vital teeth required root canal therapy. However, Saunders and Saunders reported that 19% of crowned teeth required endodontic therapy after cementation. If the crown is an abutment for a partial denture, a larger percentage of teeth may require endodontic intervention. Cheung et al found that 32% of partial denture abutments required endodontic therapy after cementation of the prosthesis.

Fig 4-2 The percentages of teeth with full-coverage restorations requiring endodontic intervention after prosthetic care.

Thermal insult

The majority of published data indicate that pulpal changes caused by thermal insults are minor and pose few long-term consequences. Dowden et al found that low-temperature stimulation caused only minor injuries to the odontoblastic layer and microvascular system. Rickoff et al described similar findings with high-temperature stimuli. These studies suggest that pulp vitality assessment with either warm or cold stimuli pose no threat to pulpal health. Increases in temperature as a result of tooth preparation without coolant, however, may prove problematic. Zach and Cohen found that a temperature increase of 5.6°C, which is expected when preparation is performed without coolant, may cause permanent damage.

Orthodontic treatment

The majority of published evidence suggests that orthodontic therapy may have a transient effect on pulp tissues (Fig 4-3); however, significant and permanent changes are unlikely. Data to support these findings come from clinical, histologic, and histochemical investigations. Clinically, these effects can be measured by the altered responses to electric pulp testing (EPT) in patients undergoing treatment (Cave et al) as well as by a measurable and significant decrease in pulp size discernable by cone beam computed tomography (CBCT) evaluation (Venkatesh et al).

Histologic evidence includes several examinations of pulpal blood flow. Nixon et al found an association between increased pulp vascularity and predentin formation and the application of orthodontic force. In a 2012 review, however, Von Bohl et al suggested that contradictory evidence for the effects of orthodontic therapy on blood flow exists in the literature. A recent study by Lazzaretti et al found that orthodontic intrusion caused an increase in fibrous tissue in the pulp as well as blood vessel congestion but no alteration in the number of blood vessels.

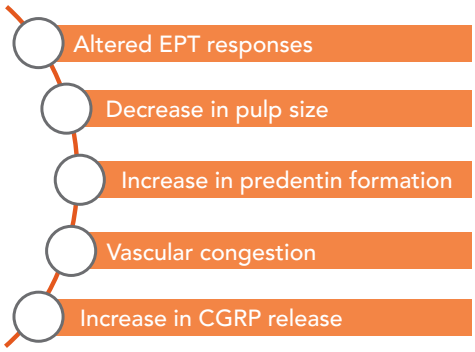

Fig 4-3 The effects of orthodontic therapy on pulp tissues. EPT, electric pulp testing; CGRP, calcitonin gene-related peptide.

Histochemical evidence for the effect of orthodontic forces on pulp tissue includes an increase in calcitonin gene-related peptide (CGRP) in the pulp tissue of teeth subjected to severe orthodontic forces (Caviedes-Bucheli et al).

Though the effects of orthodontic treatment on pulpal tissues are typically insignificant, the association between orthodontics and resorptive processes is well documented. Heithersay found an association between orthodontic therapy and invasive cervical root resorption. Reitan associated orthodontic therapy with external root resorption and found that both the precementum and predentin exerted protective effects. Iglesias-Linares et al associated the occurrence of external root resorption in orthodontic patients with interleukin-1β (IL-1β) polymorphism. Both endodontically treated and vital teeth exhibit similar ex-

ternal resorptive changes in response to orthodontic therapy (Mattison et al), though endodontically treated teeth may be less susceptible to replacement resorption (Bender et al).

Periodontal disease

The effect of periodontal disease on pulp tissue is a controversial topic. Several authors, including Mazur and Massler, Czarnecki and Schilder, and Bender and Seltzer assert that periodontitis cannot cause pulpal disease. As support for this idea, Seltzer et al pointed out that, while lateral canals are often exposed by periodontal disease, pulpal reactions are rarely observed. However, several other authors claim the converse. Both Rubach and Mitchell as well as Sinai and Soltanoff found that periodontal disease could affect pulp tissues. Sinai and Soltanoff found that pulpal lesions formed in response to periodontitis occur gradually and are more often resorptive or proliferative than inflammatory in nature. In addition to these findings, a recent clinical study appears to support the hypothesis that periodontal disease can affect pulp tissues. Giovanella et al found lower oxygen saturation rates in the pulpal tissues of teeth affected by periodontal disease than controls, though no differences in electric sensitivity testing were found. The authors supporting these positions are summarized in Fig 4-4.

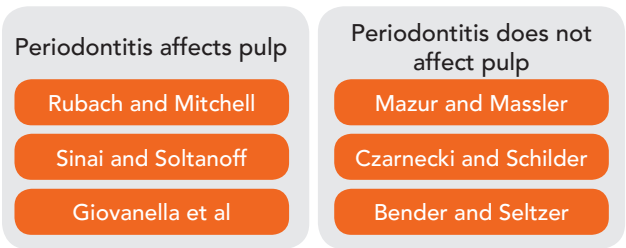

Fig 4-4 The authors who assert or deny the effects of periodontitis on pulp tissues.

Occlusion

Occlusal forces have a negligible effect on observed pulpal histology; however, histochemical responses have been documented in the literature. Landay et al found that light excessive occlusal forces over short periods did not cause significant pulpal changes in rats. Additionally, Glickman et al found that high occlusion in dogs led to the influx of inflammatory cells in the pulp furcation in only 1 in 20 animals. Despite the apparent lack of histologic data, Caviedes-Bucheli et al found an increase in substance P (SP) expression in both the pulp and periodontal ligament (PDL) after an acute episode of occlusal trauma.

Just as occlusal forces do not appear to affect pulp tissues in a meaningful manner, occlusal adjustment does not appear to relieve postoperative discomfort. Creech et al and Parirokh et al found no differences in postoperative pain between teeth that had re-

ceived occlusal reduction and controls. Rosenberg et al, on the other hand, suggested that occlusal reduction is effective in reducing postoperative pain in patients with vital pulps, percussion sensitivity, and preoperative pain, and in the absence of an apical radiolucency (Fig 4-5).

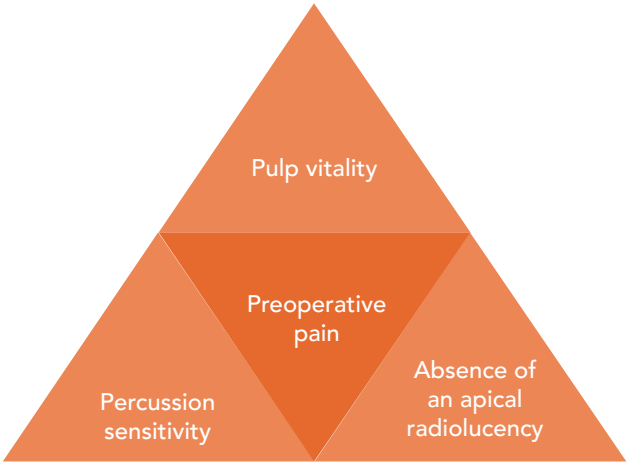

Fig 4-5 Rosenberg et al's criteria for occlusal adjustment.

Historical Perspectives on Infection and Immunology

The science of endodontics has journeyed far since its inception. With the advent of new discoveries, endodontics has moved past several previously held beliefs. A thorough understanding of the specialty of endodontics should include these ideas, as many formed the foundation of the specialty. These concepts include anachoresis, the focal infection theory, and the hollow tube theory.

The theory of anachoresis, based on the work of Gier and Mitchell, suggests that bloodborne bacteria are attracted to areas of chronic inflammation. However, Delivanis et al found that a hematogenous pathway for reinfection of unfilled root canal spaces was doubtful. Intravenously injected bacteria did not localize to unfilled root canals in a feline model, despite notable bacteremia. Their findings are often used to discredit the theory of anachoresis.

The focal infection theory represents the converse of anachoresis and suggests that a "focus" of microorganisms, like that found in a root canal space, leads to systemic dissemination of infection. Proponents of this theory included Weston Price, who brought the theory to endodontics in 1925. He inferred that bacteria trapped in dentinal tubules during root canal therapy could "leak" into the systemic circulation and cause systemic disease. He strongly advocated extraction of diseased teeth. In 1952, Easlick pointed out the fallacies in Price's research methods—including the inadequate use of controls, massive amounts of bacteria in the cases presented, as well as contamination of the endodontically treated teeth during extraction—and refuted the associations between endodontically treated teeth and systemic disease.

The hollow tube theory was developed by Rickert and Dixon. It suggests that hollow tubes contained within the body, including root canal spaces, collect circulatory elements and permit inflammation to develop. This theory was debunked by the work of both Torneck and Wenger et al, who found that open tubes, when implanted within laboratory animals, did not cause inflammatory reactions in adjacent tissues. Similarly, Dubrow reported instances of periapical healing in patients with cleaned but unfilled root canal spaces.

Pulpal Pathology

Irritants incite neurovascular, cellular, and humoral responses in pulpal tissues. These responses produce the clinical pathologic entities we know as *irreversible pulpitis* and *pulpal necrosis* through a number of orchestrated microscopic responses. Pulpal irritants activate both the innate and acquired immune systems. The innate immune system does not require prior pathogen sensitization and includes functions like physical barriers, phagocytes, inflammation, nonspecific proteins, and cellular responses from macrophages, polymorphonuclear leukocytes, mononuclear cells, natural killer cells, mast cells, and basophils (Rittenhouse-Olson and De Nardin). The adaptive immune system requires prior antigen sensitization and includes all B- and T-cell responses (Rittenhouse-Olson and De Nardin). The components of the innate and adaptive immune responses are summarized in Fig 4-6. For a more complete immunology reference, please review *Contemporary Clinical Immunology and Serology* by Rittenhouse-Olsen and De Nardin.

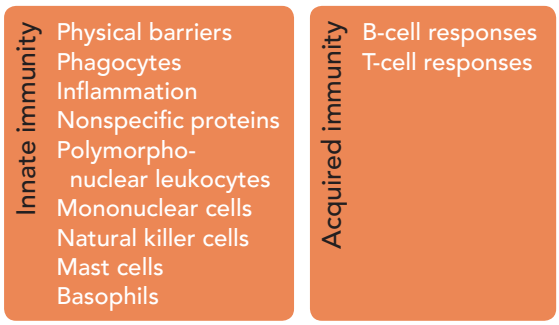

Fig 4-6 The innate and acquired immune systems.

Neurovascular responses

Strong stimuli, transferred via fluid movements, activate Aδ and C fibers contained within the pulp (Brännström). C fibers not only transport signals to the brain when stimulated but also release neuropeptides, including CGRP and SP into the local environment. These peptides have several important immune functions, including potentiation of chemotaxis, phagocytosis, lymphocyte proliferation, stimulation of IL-2 production, and expression of adhesion molecules in immunocompetent cells (Caviedes-Bucheli et al 2008). Not only are these peptides released in response to damaging stimuli, but their receptors are also upregulated in the clinical state of irreversible pulpitis (Caviedes-Bucheli et al 2005).

In addition to their immunoregulatory functions, neuropeptides also produce a profound vascular response exhibited by sustained vasodilation (Caviedes-Bucheli et al 2008). Kim found that the activation of C fibers, but not Aδ fibers, led to alterations in pulp blood flow.

Kim and Dorscher-Kim also characterized the nature of pulpal blood-flow responses to irritation as biphasic (Fig 4-7). The initial response involves an increase in blood flow due to vasodilation with an associated increase in tissue fluid. In the second phase, pulpal blood flow decreases as a result of vasodilation and the increase in tissue fluid.

Fig 4-7 The biphasic nature of pulpal blood flow following insult (Kim and Dorscher-Kim).

Pulpal immunology

Immune responses in the pulpal and periapical tissues involve both cellular and humoral responses (Fig 4-8). The role of leukocytes in pulpal inflammation is well documented in the literature. Torneck located both lymphocytes and plasma cells in the early stages of pulpal inflammation, whereas advanced inflammation was associated with the presence of polymorphonuclear leukocytes and macrophages. These cells are extravasated in the venules of the pulpal tissues where they can fulfill their immune function (Kogushi et al).

In addition to cellular responses, humoral responses occur in response to pulpal irritants and include immunoglobulin (Ig), prostaglandin (PG), and cytokine production. These responses are likely a direct result of exposure to bacteria and their by-products. Hosoya and Matsushima found that the expression of the proinflammatory cytokine IL-1β by human dental pulp cells was stimulated by lipopolysaccharide (LPS) exposure. Nakanishi et al found that inflamed pulp tissue contained significantly larger amounts of IgG, IgA, IgM, elastase, and PGE2 than normal pulp tissue.

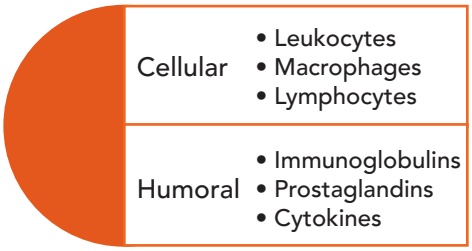

Fig 4-8 Some of the cellular and humoral immune responses observed in pulpal tissues.

The sum total of humoral and cellular responses is the development of pulpal inflammation. If inflammation persists, focal microabscesses may develop in pulp tissues. As these micro-abscesses coalesce, the pulp tissues become necrotic (Langeland). Necrotic pulp tissue becomes populated by bacteria, and the process of microbial succession occurs, by which initially aerobic species are replaced by anaerobes (Fabricius et al). As infection spreads, Lin and Langeland documented that bacteria then penetrate into the circumpulpal dentinal tubules to the edge of the necrotic front. LPS,

a portion of the cell wall of gram-negative bacteria, has also been associated with pulpal and periapical symptoms. Studies by Horiba et al, Dahlen and Bergenholtz, and Schein and Schilder all document this effect.

Signs and symptoms versus histology

Mild pulpal symptoms are associated with reversible pulpitis, whereas more severe symptoms are associated with irreversible pulpitis (Levin et al). However, clinical signs and symptoms may not correlate with the histologic pulp status. In a systematic review, Mejare et al concluded that insufficient evidence exists to determine whether the presence, nature, and duration of symptoms offer accurate information about the extent of pulpal inflammation. Several other studies support this claim. Bhaskar and Rappaport observed vital tissue in traumatized anterior teeth that did not respond to pulp vitality tests. Mumford found no correlation between pain perception thresholds and pulp condition. Similarly, Mendoza et al (1987a, 1987b) were unable to link the histologic presentation of pulpal nerves and blood vessels with clinical symptoms. Studies by Block et al, Seltzer et al, and Langeland et al also support the claim that clinical signs and symptoms may not correlate with the histologic picture.

A recent study by Ricucci et al, however, found the opposite. A correlation was noted between clinical diagnoses and histologic evaluations of normal, reversibly inflamed, or irreversibly inflamed pulps. The clinical diagnosis of normal or reversible pulpitis matched histologic diagnosis 96% of the time, and in cases of irreversible pulpitis, 84% were matched. These findings are encouraging as clinical tests are relied on for treatment planning and correlation of symptoms with histology indicates that practitioners can correctly guide their treatment choices.

Periapical Pathology

Microbial spread from the pulp into the periapical tissues causes apical periodontitis by activating both positive and negative pathways (Marton and Kiss) (Fig 4-9). Kakehashi et al elegantly demonstrated the association between bacterial infiltration in the pulp and the development of apical periodontitis. Korzen et al found more severe inflammatory responses to mixed infections than to a mono-infection. Just as these studies illustrate the relationship between bacteria and apical periodontitis, others illustrate a similar relationship with LPS (Dwyer and Torabinejad, Schonfeld et al). Lastly, evidence supports the role of viruses in periapical pathology (Sabeti et al). These pathologic entities activate both cellular and humoral immune responses, ultimately resulting in bone resorption.

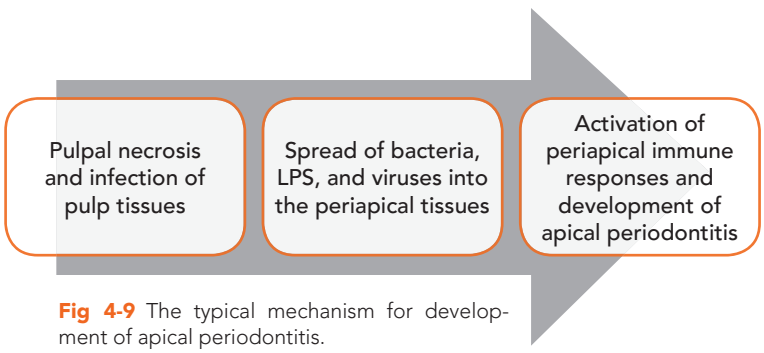

Fig 4-9 The typical mechanism for development of apical periodontitis.

Cellular responses

Just as pulpal responses include both cellular and humoral components, so do those in the periapical tissues (Torabinejad and Bakland 1978). Lymphocytes are the predominant cell population in the development of apical periodontitis (Stern et al). While both T and B cells are present, the average number of T cells is typically greater than the number of B cells (Torabinejad and Kettering). Several subpopulations of T cells have been demonstrated in periapical tissues, including helper T (TH) cells, suppressor T (TS) cells, and cytotoxic T cells. TH cells predominate during the early, active phase of the lesion development, whereas TS cells are more numerous in chronic lesions (Stashenko and Yu) (Fig 4-10). Additionally, Xiong et al found TH17 cells during all phases of rat periapical lesion development, particularly during lesion expansion. B cell deficiencies do not appear to alter lesion development (Waterman et al), whereas lesion progression appears to be slowed by deficiencies in T cell populations (Tani et al). In addition to lymphocytes, macrophage/monocyte cells, plasma cells, polymorphonuclear leukocytes (Stern et al), natural killer cells, dendritic cells (Nilsen et al), and mast cells (Ledesma-Montes et al) have been implicated in the development of apical periodontitis.

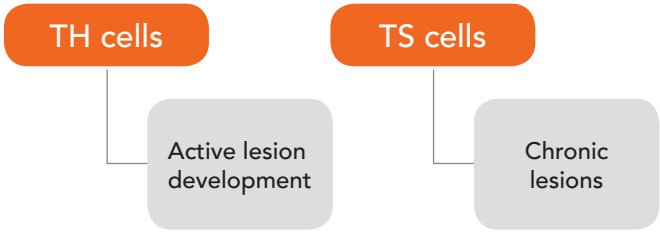

Fig 4-10 Lymphocytic responses in the periapical tissues are predominated by T-cell responses, and both TH and TS cells are involved.

Humoral responses

Immunoglobulins, PGs, and cytokines are involved in apical periodontitis, just as in pulpal inflammation. Immunoglobulins present in periapical lesions include IgG, IgA, IgE, and IgM, with IgG predominating (Pulver et al). Additionally, systemic changes in immunoglobulin levels have been reported in the presence of apical pathology. An increase in circulating immunoglobulins has been noted in patients with acute abscesses (Kettering and Torabinejad 1984) and flare-ups (Svetcov et al); however, the effect of chronic lesions on circulating numbers is controversial. While Nevins et al found higher systemic levels of IgE in patients with asymptomatic necrotic teeth, Kettering and Torabinejad (1986) did not.

PGs and cytokines compose the remaining humoral responses in apical periodontitis. PGs are metabolic byproducts of arachidonic acid formed via the cyclooxygenase pathways. They hold many functions, including gastrointestinal homeostasis, promotion of inflammation, vasodilation, chemotaxis, pain, vascular permeability, and bone resorption (Torabinejad and Bakland 1980a and 1980b). Symptomatic lesions have a significantly higher concentration of PG activity than chronic lesions or uninflamed tissues (McNicholas et al).

Cytokines, including IL-1β and IL-1α, are produced by inflammatory cells in apical periodontitis (Stashenko et al). Endodontic pathogens activate toll-like receptor 4 on macrophages leading to the expression of IL-1β, tumor necrosis factor α (TNFα), IL-6, and IL-10 (Sousa et al, Martinho et al). Sabeti et al and Hernadi et al recently discovered that viral infections promote cytokine production in periapical lesions. IL-1β is the most active bone resorptive cytokine, reported to have approximately 15 times greater potency than IL-1α (Stashenko). Genetic differences in IL-1β production have been associated with persistent apical periodontitis (Morsani et al). Cytokines, including IL-1, TNFα, and leukotriene, promote the fusion of osteoclast precursor cells and cause consequent bone resorption (Kobayashi et al) (Fig 4-11).

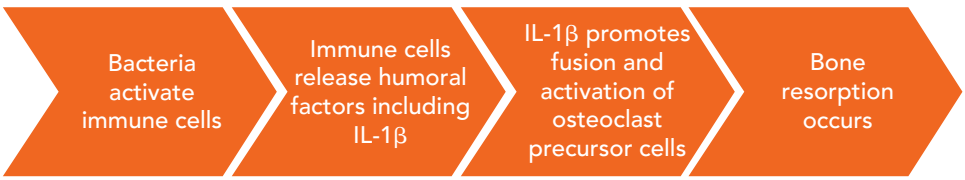

Fig 4-11 The pathway by which bacteria lead to bone loss in apical periodontitis.

The humoral responses in apical periodontitis can cause systemic effects, and systemic drug administration may mitigate the response. In a systematic review, Gomes et al found that apical periodontitis is associated with increased systemic levels of C-reactive protein, IL-6, IL-1, IL-2, IgA, IgG, and IgM in humans. Alternatively, systemic medications may mitigate the humoral response. Lin et al found that administration of simvastatin decreased the expression of pro-inflammatory cytokines and attenuated bone resorption in rats. Liu et al found that metformin had similar results.

Bone resorption

Bone resorption in apical periodontitis results from immune responses stimulated by bacterial and viral insults. Cytokines produced during inflammatory responses stimulate the production of a molecule called *receptor activator of nuclear factor κ-B ligand (RANKL)* by immune competent cells, which ultimately results in the destruction of bone by osteoclasts. Osteoblasts, rather than osteoclasts, possess the receptors for resorptive hormones and cytokines. In the functional absence of osteoblasts, osteoclasts cannot respond to resorptive mediators (Thomson et al). Bacteria and LPS may also directly activate cells, including periapical T cells, to produce RANKL, thus stimulating osteoclasts to resorb bone (Silva et al).

Bacteria in the periapical lesion

Though the role of bacteria in the progression of apical periodontitis is undeniable, controversy exists regarding the existence of living bacteria in periapical lesions (Fig 4-12). Several authors discovered bacteria in periapical tissues. A multitude of histologic evidence from

Matsumiya and Kitamura, Tronstad et al, Sundqvist and Reuterving, and Iwu et al located bacteria in periapical tissues. Wayman et al found more bacteria in lesions that communicated with the oral cavity. In addition to histologic data, newer DNA probe techniques have also been used to demonstrate bacteria in apical lesions. Saber et al used this technique to locate bacteria in symptomatic lesions. On the contrary, both Shindell as well as Walton and Ardjmand were unable to detect viable bacteria within periapical tissues. Additionally, Siqueira and Lopes noted that bacteria were restricted to the root canal space in teeth without previous root canal therapy. Despite the controversy regarding localization of live bacteria, however, a relationship between bacteria and apical periodontitis is undeniable.

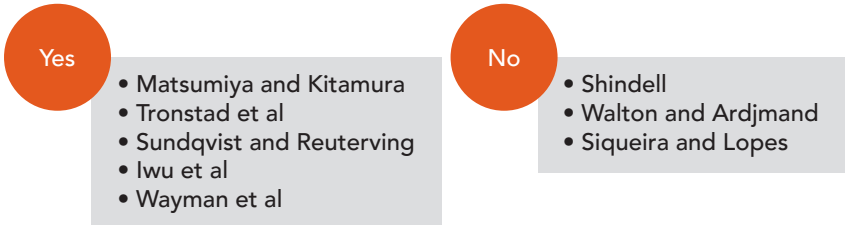

Fig 4-12 Authors who assert and deny the presence of live bacteria in periapical lesions.

The Nature of the Periapical Lesion

Once bony destruction commences, apical pathology can take several histologic forms. Though many believe that the nature of these tissues is discernable based on radiographic features alone, this assumption has proven false time and time again. Though radiographs provide necessary information for the diagnosis of dental pathology, they have proven unable to differentiate between dental cysts and granulomas. Studies by Linenberg et al, Stockdale and Chandler, Wais, Baumann and Rossman, and Lalonde all demonstrated the inability to correlate histologic diagnoses with radiographic findings. Ricucci et al found no correlation between the presence of a radiopaque lamina and the histologic diagnosis of a cyst. These findings, taken together with the small chance of locating other pathologic entities, underscore the importance of histologic examination of periapical specimens. Peters and Lau suggest that histologic evaluation of a periapical lesion is appropriate any time recoverable tissue is present.

Periapical granulomas

Periapical granulomas are composed of granulation tissue and chronic inflammation rather than granulomatous inflammation as the nomenclature suggests (Weiner et al). Humoral factors are found in granulomas, and the immunoglobulin found most frequently is IgG (Pulver et al). Cells found in granulomas include macrophages, lympohcytes, plasma cells,

polymorphonucleocytes, fibroblasts, vascular elements, and epithelial cells (Stern et al). The organization of epithelial cells in cystic lesions, rather than the simple presence of epithelial cells, is what differentiates periapical cysts from periapical granulomas (Nair et al 1996). A comparison between common granuloma and cyst features is presented in Fig 4-13.

Granulomas	Cysts
• May contain epithelial cells, though no epithelial lining is present • Contain immune cells • Humoral immunity, IgG most common	• Epithelial-lined cavity • Contain immune cells • Humoral immunity, IgG and IgA

Fig 4-13 Common features of periapical granulomas and periapical cysts.

Periapical cysts

Periapical cysts are epithelial-lined cavities that form when epithelial rests of Mallasez line a periapical lesion (Torabinejad). Several theories of cyst formation have been proposed through the years (Fig 4-14) (Nair). Ten Cate proposed the nutritional deficiency theory, asserting that cells in the center of the epithelial cavity lose their nutritional source and necrose. The abscess theory, described by Nair et al (2008), suggests that proliferating epithelial cells line an existent abscess cavity as a result of the inherent nature of epithelial cells to cover exposed connective tissue surfaces. Torabinejad's immunologic theory proposes that the inflammatory mediators present in periapical responses stimulate epithelial cells to proliferate.

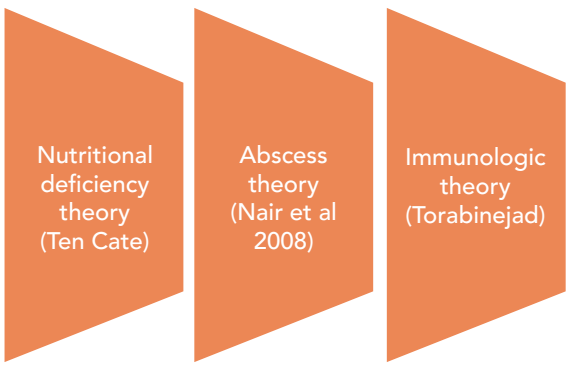

Fig 4-14 The theories of cyst formation and the authors to whom they are attributed.

Cysts and granulomas are composed of similar immune cells and humoral responses. Pulver et al asserted that IgG and IgA are present in equal amounts in periapical cysts. What differentiates cysts from granulomas is the presence of a definitive epithelial lining in the periapical cyst. Simon described two types of periapical cysts based on differences with this lining: the true cyst or the bay cyst. True cysts represent epithelial-lined lesions that do not attach to the root canal system, whereas bay cysts

communicate with the periapex. Nair et al (1996) found that true cysts were more common than bay cysts, also known as *pocket cysts*. Though Simon would dispute his claim, Bhaskar theorized that cysts can heal after nonsurgical root canal therapy, as the frequency of cysts diagnosed via biopsy is larger than failure rates examined in prognostic literature.

Other periapical lesions

In addition to commonly encountered periapical pathologic entities, several other lesions have been located in the periapical area (Fig 4-15). According to a recent review by Sirotheau Correa Pontes et al, 66% of non-endodontic lesions found at the periapex were benign, 29% were malignant, and 5% represented Stafne bone cavities. The most common benign lesions located were ameloblastomas and nasopalatine duct cysts. The most common malignancies encountered were metastatic lesions and carcinomas. Riccuci et al found the presence of ciliated columnar epithelium in 2% of all apical periodontitis lesions and 8% of all cysts and found it exclusively in the maxilla. Other entities encountered at the periapex include orthokeratinized odontogenic cysts (Silva Serato et al), central giant cell granulomas (de Carvalhosa et al), periapical giant cell granulomas (Nair et al 1990), lymphomas (Koivisto et al 2013), fungal infections, actinomycosis, central ossifying fibromas, Langerhans cell histiocytosis, osteoblastomas, central ossifying fibromas, osteosarcoma, plasma cell tumors, and metastatic lesions among others (Peters and Lau). The existence of these entities again underscores the necessity for pathologic evaluation of any tissue removed from the periapex.

Differential diagnosis for apical radiolucencies

- Periapical granuloma
- Periapical cyst
- Periapical scar
- Ameloblastoma
- Ameloblastic fibroma
- Keratocystic odontogenic tumor
- Odontogenic myxoma
- Dentigerous cyst
- Residual cyst
- Nasopalatine duct cyst
- Globulomaxillary cyst
- Lateral periodontal cyst
- Traumatic bone cyst
- Stafne bone defect
- Central giant cell lesion
- Langerhans cell histiocytosis
- Brown tumor
- Cemento-osseous dysplasia (early)
- Vitamin D–resistant rickets
- Neurofibromatosis
- Malignancy

Fig 4-15 A number of pathologic entities encountered at the periapex other than the more frequently found cysts or granulomas. The variety of conditions mentioned underscores the importance for submission of all excised periapical tissues for pathologic evaluation. For more detailed discussion of non-endodontic pathology that should be included in a differential diagnosis when radiolucencies are encountered in the jawbone, please refer to chapter 7.

Frequency of specific diagnoses

The reported frequency of periapical cysts or granulomas varies based on the publication. This variance is due in large part to the authors' definitions of what comprises a cyst or granuloma. Nair et al (1996) found that periapical biopsy specimens contained granulomas 50% of the time, abscesses 35% of the time, and cysts 15% of the time. Of the cysts, 61% represented true cysts, whereas 39% represented pocket cysts. In another study that examined 9,723 biopsy specimens, Koivisto et al (2012) diagnosed granulomas in 40% of cases and cysts in 33% of cases. The remaining lesions were comprised of other pathologic entities.

Endodontic Flare-Up

Thus far, this chapter has described preoperative endodontic pathology. Postoperatively, however, patients may experience an exacerbation of symptoms known as the *endodontic flare-up* (AAE glossary). The risk of experiencing a flare-up is rather low according to the literature. Yu et al found only a 5.8% incidence of flare-up after initiation of root canal therapy on teeth with periapical lesions. The likelihood of experiencing a flare-up appears related to several factors (Fig 4-16). Walton and Fouad associated flare-ups with female sex, complaints of preoperative pain and swelling, and pulpal necrosis. In addition to those factors previously mentioned, Torabinejad et al associated flare-ups with retreatment therapy and a history of allergies. Flare-ups can cause both discomfort and unease among patients. Unfortunately, they cannot be prevented with antibiotic administration (Walton and Fouad). Analgesics and antibiotics (Seltzer and Naidorf) may be used to control a flare-up once initiated. Though uncomfortable, flare-ups have not been associated with treatment failures (de Chevigny et al).

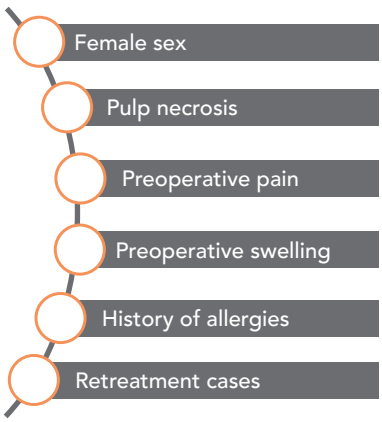

Fig 4-16 Several factors that have been associated with the development of a postoperative flare-up.

Bibliography

Pulpal Irritants

Abou-Rass M. The stressed pulp condition: An endodontic-restorative diagnostic concept. J Prosthet Dent 1982;48:264–267.

Bender IB, Byers MR, Mori K. Periapical replacement resorption of permanent, vital, endodontically treated incisors after orthodontic movement: Report of two cases. J Endod 1997;23: 768–773.

Bender IB, Seltzer S. The effect of periodontal disease on the pulp. Oral Surg Oral Med Oral Pathol 1972;33:458-474.

Cameron CE. The cracked tooth syndrome: Additional findings. J Am Dent Assoc 1976;93:971–975.

Cave SG, Freer TJ, Podlich HM. Pulp-test responses in orthodontic patients. Aust Orthod J 2002;18:27–34.

Caviedes-Bucheli J, Moreno JO, Ardila-Pinto J, et al. The effect of orthodontic forces on calcitonin gene-related peptide expression in human dental pulp. J Endod 2011;37:934–937.

Cheung GS, Lai S, Ng RP. Fate of vital pulps beneath a metal-ceramic crown or a bridge retainer. Int Endod J 2005;38:521–530.

Cotton WR, Siegel RL. Human pulpal response to citric acid cavity cleanser. J Am Dent Assoc 1978;96:639–644.

Creech JL 3rd, Walton RE, Kaltenbach R. Effect of occlusal relief on endodontic pain. J Am Dent Assoc 1984;109:64–67.

Czarnecki RT, Schilder H. A histological evaluation of the human pulp in teeth with varying degrees of periodontal disease. J Endod 1979;5:242–253.

Dawson V, Petersson K, Wolf E, Akerman S. Periapical status of non-root-filled teeth with resin composite, amalgam, or full crown restorations: A cross-sectional study of a Swedish adult population. J Endod 2014;40:1303–1308.

Dowden WE, Emmings F, Langeland K. The pulpal effect of freezing temperatures applied to monkey teeth. Oral Surg Oral Med Oral Pathol 1983;55:408–418.

Drucker DB. The role of sugar in the aetiology of dental caries. 4. The microbiological evidence. J Dent 1983;11:205–207.

Giovanella LB, Barletta FB, Felippe WT, Bruno KF, de Alencar AH, Estrela C. Assessment of oxygen saturation in dental pulp of permanent teeth with periodontal disease. J Endod 2014;40:1927–1931.

Glickman I, Smulow JB, Vogel G, Passamonti G. The effect of occlusal forces on healing following mucogingival surgery. J Periodontol 1966;37:319–325.

Hamilton AI, Kramer IR. Cavity preparation with and without waterspray. Effects on the human dental pulp and additional effects of further dehydration of the dentine. Br Dent J 1967;123:281–285.

Heithersay GS. Invasive cervical resorption: An analysis of potential predisposing factors. Quintessence Int 1999;30:83–95.

Iglesias-Linares A, Yanez-Vico RM, Ortiz-Ariza E, et al. Postorthodontic external root resorption in root-filled teeth is influenced by interleukin-1beta polymorphism. J Endod 2012;38:283–287.

Kwang S, Aminoshariae A, Harding J, Montagnese TA, Mickel A. The critical time-lapse between various restoration placements and subsequent endodontic intervention. J Endod 2014;40:1922–1926.

Landay MA, Nazimov H, Seltzer S. The effects of excessive occlusal force on the pulp. J Periodontol 1970;41:3–11.

Lazzaretti DN, Bortoluzzi GS, Torres Fernandes LF, Rodriguez R, Grehs RA, Martins Hartmann MS. Histologic evaluation of human pulp tissue after orthodontic intrusion. J Endod 2014;40:1537–1540.

Mattison GD, Delivanis HP, Delivanis PD, Johns PI. Orthodontic root resorption of vital and endodontically treated teeth. J Endod 1984;10:354–358.

Mazur B, Massler M. Influence of periodontal disease of the dental pulp. Oral Surg Oral Med Oral Pathol 1964;17:592–603.

Menezes R, Bramante CM, Letra A, Carvalho VG, Garcia RB. Histologic evaluation of pulpotomies in dog using two types of mineral trioxide aggregate and regular and white Portland cements as wound dressings. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98:376–379.

Mitchell DF, Tarplee RE. Painful pulpitis: A clinical and microscopic study. Oral Surg Oral Med Oral Pathol 1960;13:1360–1370.

Mjor IA, Tronstad L. Experimentally induced pulpitis. Oral Surg Oral Med Oral Pathol 1972;34: 102–108.

Murray PE, Smith AJ, Windsor LJ, Mjor IA. Remaining dentine thickness and human pulp responses. Int Endod J 2003;36:33–43.

Nixon CE, Saviano JA, King GJ, Keeling SD. Histomorphometric study of dental pulp during orthodontic tooth movement. J Endod 1993;19:13–16.

Parirokh M, Rekabi AR, Ashouri R, Nakhaee N, Abbott PV, Gorjestani H. Effect of occlusal reduction on postoperative pain in teeth with irreversible pulpitis and mild tenderness to percussion. J Endod 2013;39:1–5.

Reeves R, Stanley HR. The relationship of bacterial penetration and pulpal pathosis in carious teeth. Oral Surg Oral Med Oral Pathol 1966;22:59–65.

Reitan K. Initial tissue behavior during apical root resorption. Angle Orthod 1974;44:68–82.

Rickoff B, Trowbridge H, Baker J, Fuss Z, Bender IB. Effects of thermal vitality tests on human dental pulp. J Endod 1988;14:482–485.

Robertson A, Andreasen FM, Bergenholtz G, Andreasen JO, Noren JG. Incidence of pulp necrosis subsequent to pulp canal obliteration from trauma of permanent incisors. J Endod 1996;22:557–560.

Rosenberg PA, Babick PJ, Schertzer L, Leung A. The effect of occlusal reduction on pain after endodontic instrumentation. J Endod 1998;24:492–496.

Rubach WC, Mitchell DF. Periodontal disease, accessory canals and pulp pathosis. J Periodontol 1965;36:34–38.

Saunders WP, Saunders EM. Prevalence of periradicular periodontitis associated with crowned teeth in an adult Scottish subpopulation. Br Dent J 1998;185:137–140.

Schuurs AH, Gruythuysen RJ, Wesselink PR. Pulp capping with adhesive resin-based composite vs. calcium hydroxide: A review. Endod Dent Traumatol 2000;16:240–250.

Seltzer S, Bender IB, Ziontz M. The interrelationship of pulp and periodontal disease. Oral Surg Oral Med Oral Pathol 1963;16:1474–1490.

Sinai IH, Soltanoff W. The transmission of pathologic changes between the pulp and the periodontal structures. Oral Surg Oral Med Oral Pathol 1973;36:558–568.

Stanley HR. Local and systemic responses to dental composites and glass ionomers. Adv Dent Res 1991;6:55–64.

Tarim B, Hafez AA, Cox CF. Pulpal response to a resin-modified glass-ionomer material on non-exposed and exposed monkey pulps. Quintessence Int 1998;29:535–542.

Tronstad L, Mjor IA. Capping of the inflamed pulp. Oral Surg Oral Med Oral Pathol 1972;34: 477–485.

Turp JC, Gobetti JP. The cracked tooth syndrome: An elusive diagnosis. J Am Dent Assoc 1996;127:1502–1507.

Valderhaug J, Jokstad A, Ambjornsen E, Norheim PW. Assessment of the periapical and clinical status of crowned teeth over 25 years. J Dent 1997;25:97–105.

Venkatesh S, Ajmera S, Ganeshkar SV. Volumetric pulp changes after orthodontic treatment determined by cone-beam computed tomography. J Endod 2014;40:1758–1763.

Von Bohl M, Ren Y, Fudalej PS, Kuijpers-Jagtman AM. Pulpal reactions to orthodontic force application in humans: A systematic review. J Endod 2012;38:1463–1469.

Walton RE, Michelich RJ, Smith GN. The histopathogenesis of vertical root fractures. J Endod 1984;10:48–56.

Zach L, Cohen G. Pulp response to externally applied heat. Oral Surg Oral Med Oral Pathol 1965;19:515–530.

Historical Perspectives on Infection and Immunology

Delivanis PD, Snowden RB, Doyle RJ. Localization of blood-borne bacteria in instrumented unfilled root canals. Oral Surg Oral Med Oral Pathol 1981;52:430–432.

Dubrow H. Silver points and gutta-percha and the role of root canal fillings. J Am Dent Assoc 1976;93:976–980.

Easlick KA. Evaluation of the action of focal dental infections on health [in French]. Med Hyg (Genève) 1952;10:35.

Gier RE, Mitchell DF. Anachoretic effect of pulpitis. J Dent Res 1968;47:564–570.

Price WA. Dental infections and related degenerative diseases. JAMA 1925;84:254.

Rickert UG, Dixon CM. The controlling of root surgery. In: Villain G (ed). 8e Congrès Dentaire International: Paris, 2–8 Août 1931. Paris: Fédération Dentaire International, 1931:15–22.

Torneck CD. Reaction of rat connective tissue to polyethylene tube implants. I. Oral Surg Oral Med Oral Pathol 1966;21:379–387.

Wenger JS, Tsaknis PJ, del Rio CE, Ayer WA. The effects of partially filled polyethylene tube intraosseous implants in rats. Oral Surg Oral Med Oral Pathol 1978;46:88–100.

Pulpal Pathology

Bhaskar SN, Rappaport HM. Dental vitality tests and pulp status. J Am Dent Assoc 1973;86:409–411.

Block RM, Bushell A, Rodrigues H, Langeland K. A histopathologic, histobacteriologic, and radiographic study of periapical endodontic surgical specimens. Oral Surg Oral Med Oral Pathol 1976;42:656–678.

Brännström M. The hydrodynamic theory of dentinal pain: Sensation in preparations, caries, and the dentinal crack syndrome. J Endod 1986;12:453–457.

Caviedes-Bucheli J, Arenas N, Guiza O, et al. Calcitonin gene-related peptide receptor expression in healthy and inflamed human pulp tissue. Int Endod J 2005;38:712–717.

Caviedes-Bucheli J, Munoz HR, Azuero-Holguin MM, Ulate E. Neuropeptides in dental pulp: The silent protagonists. J Endod 2008;34:773–788.

Dahlen G, Bergenholtz G. Endotoxic activity in teeth with necrotic pulps. J Dent Res 1980;59:1033–1040.

Fabricius L, Dahlen G, Ohman AE, Moller AJ. Predominant indigenous oral bacteria isolated from infected root canals after varied times of closure. Scand J Dent Res 1982;90:134–144.

Horiba N, Maekawa Y, Abe Y, Ito M, Matsumoto T, Nakamura H. Correlations between endotoxin and clinical symptoms or radiolucent areas in infected root canals. Oral Surg Oral Med Oral Pathol 1991;71:492–495.

Hosoya S, Matsushima K. Stimulation of interleukin-1 beta production of human dental pulp cells by *Porphyromonas endodontalis* lipopolysaccharide. J Endod 1997;23:39–42.

Kim S, Dorscher-Kim J. Hemodynamic regulation of the dental pulp in a low compliance environment. J Endod 1989;15:404–408.

Kim S. Neurovascular interactions in the dental pulp in health and inflammation. J Endod 1990;16:48–53.

Kogushi M, Nakamura S, Kishi Y, Kim S, Takahashi K. A study of leukocyte extravasation in early inflammatory changes in the pulp. J Endod 1988;14:475–481.

Langeland K. Tissue response to dental caries. Endod Dent Traumatol 1987;3:149–171.

Langeland K, Block RM, Grossman LI. A histopathologic and histobacteriologic study of 35 periapical endodontic surgical specimens. J Endod 1977;3:8–23.

Levin LG, Law AS, Holland GR, Abbott PV, Roda RS. Identify and define all diagnostic terms for pulpal health and disease states. J Endod 2009;35:1645–1657.

Lin L, Langeland K. Light and electron microscopic study of teeth with carious pulp exposures. Oral Surg Oral Med Oral Pathol 1981;51:292–316.

Mejare IA, Axelsson S, Davidson T, et al. Diagnosis of the condition of the dental pulp: A systematic review. Int Endod J 2012;45:597–613.

Mendoza MM, Reader A, Meyers WJ, Foreman DW. An ultrastructural investigation of the human apical pulp in irreversible pulpitis. I. Nerves. J Endod 1987a;13:267–276.

Mendoza MM, Reader A, Meyers WJ, Marquard JV. An ultrastructural investigation of the human apical pulp in irreversible pulpitis. II. Vasculature and connective tissue. J Endod 1987b;13: 318–327.

Mumford JM. Pain perception threshold on stimulating human teeth and the histological condition of the pulp. Br Dent J 1967;123:427–433.

Nakanishi T, Matsuo T, Ebisu S. Quantitative analysis of immunoglobulins and inflammatory factors in human pulpal blood from exposed pulps. J Endod 1995;21:131–136.

Ricucci D, Loghin S, Siqueira JF Jr. Correlation between clinical and histologic pulp diagnoses. J Endod 2014;40:1932–1939.

Rittenhouse-Olson K, De Nardin E. Contemporary Clinical Immunology and Serology. Boston: Pearson, 2013.

Schein B, Schilder H. Endotoxin content in endodontically involved teeth. 1975. J Endod 2006;32:293–295.

Seltzer S, Bender IB, Nazimov H. Differential diagnosis of pulp conditions. Oral Surg Oral Med Oral Pathol 1965;19:383–391.

Torneck CD. Changes in the fine structure of the human dental pulp subsequent to carious exposure. J Oral Pathol 1977;6:82–95.

Periapical Pathology

Dwyer TG, Torabinejad M. Radiographic and histologic evaluation of the effect of endotoxin on the periapical tissues of the cat. J Endod 1981;7:31–35.

Gomes MS, Blattner TC, Sant'Ana Filho M, et al. Can apical periodontitis modify systemic levels of inflammatory markers? A systematic review and meta-analysis. J Endod 2013;39: 1205–1217.

Hernadi K, Gyongyosi E, Meszaros B, et al. Elevated tumor necrosis factor-alpha expression in periapical lesions infected by Epstein-Barr virus. J Endod 2013;39:456–460.

Iwu C, MacFarlane TW, MacKenzie D, Stenhouse D. The microbiology of periapical granulomas. Oral Surg Oral Med Oral Pathol 1990;69:502–505.

Kakehashi S, Stanley HR, Fitzgerald RJ. The effects of surgical exposures of dental pulps in germfree and conventional laboratory rats. Oral Surg Oral Med Oral Pathol 1965;20:340–349.

Kettering JD, Torabinejad M. Concentrations of immune complexes, IgG, IgM, IgE, and C3 in patients with acute apical abscesses. J Endod 1984;10:417–421.

Kettering JD, Torabinejad M. Concentrations of immunoglobulin E in patients with chronic periapical lesions. J Endod 1986;12:306–308.

Kobayashi K, Takahashi N, Jimi E, et al. Tumor necrosis factor alpha stimulates osteoclast differentiation by a mechanism independent of the ODF/RANKL-RANK interaction. J Exp Med 2000;191:275–286.

Korzen BH, Krakow AA, Green DB. Pulpal and periapical tissue responses in conventional and monoinfected gnotobiotic rats. Oral Surg Oral Med Oral Pathol 1974;37:783–802.

Ledesma-Montes C, Garces-Ortiz M, Rosales-Garcia G, Hernandez-Guerrero JC. Importance of mast cells in human periapical inflammatory lesions. J Endod 2004;30:855–859.

Lin LD, Lin SK, Chao YL, et al. Simvastatin suppresses osteoblastic expression of Cyr61 and progression of apical periodontitis through enhancement of the transcription factor Forkhead/ winged helix box protein O3a. J Endod 2013;39:619–625.

Liu L, Zhang C, Hu Y, Peng B. Protective effect of metformin on periapical lesions in rats by decreasing the ratio of receptor activator of nuclear factor kappa B ligand/osteoprotegerin. J Endod 2012;38:943–947.

Martinho FC, Leite FR, Chiesa WM, Nascimento GG, Feres M, Gomes BP. Signaling pathways activation by primary endodontic infectious contents and production of inflammatory mediators. J Endod 2014;40:484-489.

Marton IJ, Kiss C. Overlapping protective and destructive regulatory pathways in apical periodontitis. J Endod 2014;40:155–163.

Matsumiya S, Kitamura K. Histopathological and histobacteriological studies of the relation between the condition of sterilization of the interior of the root canal and the healing process of periapical tissues in experimentally infected root canals. Bull Tokyo Dental Coll 1960;1:1–19.

McNicholas S, Torabinejad M, Blankenship J, Bakland L. The concentration of prostaglandin E2 in human periradicular lesions. J Endod 1991;17:97–100.

Morsani JM, Aminoshariae A, Han YW, Montagnese TA, Mickel A. Genetic predisposition to persistent apical periodontitis. J Endod 2011;37:455–459.

Nevins AJ, Levine S, Faitlowicz-Gayer Y, Svetcov S. Sensitization via IgE-mediated mechanism in patients with chronic periapical lesions. J Endod 1985;11:228–230.

Nilsen R, Johannessen AC, Skaug N, Matre R. In situ characterization of mononuclear cells in human dental periapical inflammatory lesions using monoclonal antibodies. Oral Surg Oral Med Oral Pathol 1984;58:160–165.

Pulver WH, Taubman MA, Smith DJ. Immune components in human dental periapical lesions. Arch Oral Biol 1978;23:435–443.

Saber MH, Schwarzberg K, Alonaizan FA, et al. Bacterial flora of dental periradicular lesions analyzed by the 454-pyrosequencing technology. J Endod 2012;38:1484–1488.

Sabeti M, Kermani V, Sabeti S, Simon JH. Significance of human cytomegalovirus and Epstein-Barr virus in inducing cytokine expression in periapical lesions. J Endod 2012;38:47–50.

Schonfeld SE, Greening AB, Glick DH, Frank AL, Simon JH, Herles SM. Endotoxic activity in periapical lesions. Oral Surg Oral Med Oral Pathol 1982;53:82–87.

Shindell E. Studies on the possible presence of a virus in subacute and choronic periapical granulomas. Oral Surg Oral Med Oral Pathol 1962;15:1382–1384.

Silva MJ, Kajiya M, AlShwaimi E, et al. Bacteria-reactive immune response may induce RANKL-expressing T cells in the mouse periapical bone loss lesion. J Endod 2012;38:346–350.

Siqueira JF Jr, Lopes HP. Bacteria on the apical root surfaces of untreated teeth with periradicular lesions: A scanning electron microscopy study. Int Endod J 2001;34:216–220.

Sousa EL, Martinho FC, Leite FR, Nascimento GG, Gomes BP. Macrophage cell activation with acute apical abscess contents determined by interleukin-1 beta and tumor necrosis factor alpha production. J Endod 2014;40:1752–1757.

Stashenko P. Role of immune cytokines in the pathogenesis of periapical lesions. Endod Dent Traumatol 1990;6:89–96.

Stashenko P, Obernesser MS, Dewhirst FE. Effect of immune cytokines on bone. Immunol Invest 1989;18:239–249.

Stashenko P, Yu SM. T helper and T suppressor cell reversal during the development of induced rat periapical lesions. J Dent Res 1989;68:830–834.

Stern MH, Dreizen S, Mackler BF, Levy BM. Isolation and characterization of inflammatory cells from the human periapical granuloma. J Dent Res 1982;61:1408–1412.

Sundqvist G, Reuterving CO. Isolation of *Actinomyces israelii* from periapical lesion. J Endod 1980;6:602–606.

Svetcov SD, DeAngelo JE, McNamara T, Nevins AJ. Serum immunoglobulin levels and bacterial flora in subjects with acute oro-facial swellings. J Endod 1983;9:233–235.

Tani N, Kuchiba K, Osada T, Watanabe Y, Umemoto T. Effect of T-cell deficiency on the formation of periapical lesions in mice: Histological comparison between periapical lesion formation in BALB/c and BALB/c nu/nu mice. J Endod 1995;21:195–199.

Thomson BM, Mundy GR, Chambers TJ. Tumor necrosis factors alpha and beta induce osteoblastic cells to stimulate osteoclastic bone resorption. J Immunol 1987;138:775–779.

Torabinejad M, Bakland LK. Immunopathogenesis of chronic periapical lesions. A review. Oral Surg Oral Med Oral Pathol 1978;46:685–699.

Torabinejad M, Bakland LK. Prostaglandins: Their possible role in the pathogenesis of pulpal and periapical diseases, part 1. J Endod 1980a;6:733–739.

Torabinejad M, Bakland LK. Prostaglandins: Their possible role in the pathogenesis of pulpal and periapical diseases, part 2. J Endod 1980b;6:769–776.

Torabinejad M, Kettering JD. Identification and relative concentration of B and T lymphocytes in human chronic periapical lesions. J Endod 1985;11:122–125.

Tronstad L, Barnett F, Riso K, Slots J. Extraradicular endodontic infections. Endod Dent Traumatol 1987;3:86–90.

Walton RE, Ardjmand K. Histological evaluation of the presence of bacteria in induced periapical lesions in monkeys. J Endod 1992;18:216–227.

Waterman PA Jr, Torabinejad M, McMillan PJ, Kettering JD. Development of periradicular lesions in immunosuppressed rats. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85: 720–725.

Wayman BE, Murata SM, Almeida RJ, Fowler CB. A bacteriological and histological evaluation of 58 periapical lesions. J Endod 1992;18:152–155.

Xiong H, Wei L, Peng B. Immunohistochemical localization of IL-17 in induced rat periapical lesions. J Endod 2009;35:216–220.

The Nature of the Periapical Lesion

Baumann L, Rossman SR. Clinical, roentgenologic, and histopathologic findings in teeth with apical radiolucent areas. Oral Surg Oral Med Oral Pathol 1956;9:1330–1336.

Bhaskar SN. Oral surgery—Oral pathology conference No. 17, Walter Reed Army Medical Center. Periapical lesions—Types, incidence, and clinical features. Oral Surg Oral Med Oral Pathol 1966;21:657–671.

de Carvalhosa AA, Zandonade RM, de Souza C, de Araujo Estrela CR, Borges AH, Estrela C. 8-year follow-up of central giant cell lesion mimicking apical periodontitis. J Endod 2014;40:1708–1712.

Koivisto T, Bowles WR, Magajna WA, Rohrer M. Malignant lymphoma in maxilla with cystic involvement: A case report. J Endod 2013;39:935–938.

Koivisto T, Bowles WR, Rohrer M. Frequency and distribution of radiolucent jaw lesions: A retrospective analysis of 9,723 cases. J Endod 2012;38:729–732.

Lalonde ER. A new rationale for the management of periapical granulomas and cysts: An evaluation of histopathological and radiographic findings. J Am Dent Assoc 1970;80:1056–1059.

Linenberg WB, Waldron CA, Delaune GF Jr. A clinical, roentgenographic, and histopathologic evaluation of periapical lesions. Oral Surg Oral Med Oral Pathol 1964;17:467–472.

Nair PN. New perspectives on radicular cysts: Do they heal? Int Endod J 1998;31:155–160.

Nair PNR, Pajarola GF, Schroeder HE. Types and incidence of human periapical lesions obtained with extracted teeth. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:93–102.

Nair PN, Sjogren U, Krey G, Sundqvist G. Therapy-resistant foreign body giant cell granuloma at the periapex of a root-filled human tooth. J Endod 1990;16:589–595.

Nair PN, Sundqvist G, Sjogren U. Experimental evidence supports the abscess theory of development of radicular cysts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106: 294–303.

Peters E, Lau M. Histopathologic examination to confirm diagnosis of periapical lesions: A review. J Can Dent Assoc 2003;69:598–600.

Pulver WH, Taubman MA, Smith DJ. Immune components in human dental periapical lesions. Arch Oral Biol 1978;23:435–443.

Ricucci D, Loghin S, Siqueira JF Jr, Abdelsayed RA. Prevalence of ciliated epithelium in apical periodontitis lesions. J Endod 2014;40:476–483.

Ricucci D, Mannocci F, Ford TR. A study of periapical lesions correlating the presence of a radiopaque lamina with histological findings. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:389–394.

Silva Servato JP, Cardoso SV, Parreira da Silva MC, Cordeiro MS, Rogerio de Faria P, Loyola AM. Orthokeratinized odontogenic cysts presenting as a periapical lesion: Report of a case and literature review. J Endod 2014;40:455–458.

Simon JH. Incidence of periapical cysts in relation to the root canal. J Endod 1980;6:845–848.

Sirotheau Correa Pontes F, Paiva Fonseca F, Souza de Jesus A, et al. Nonendodontic lesions misdiagnosed as apical periodontitis lesions: Series of case reports and review of literature. J Endod 2014;40:16–27.

Stern MH, Dreizen S, Mackler BF, Selbst AG, Levy BM. Quantitative analysis of cellular composition of human periapical granuloma. J Endod 1981;7:117–122.

Stockdale CR, Chandler NP. The nature of the periapical lesion—A review of 1108 cases. J Dent 1988;16:123–129.

Ten Cate AR. The epithelial cell rests of Malassez and the genesis of the dental cyst. Oral Surg Oral Med Oral Pathol 1972;34:956–964.

Torabinejad M. The role of immunological reactions in apical cyst formation and the fate of epithelial cells after root canal therapy: A theory. Int J Oral Surg 1983;12:14–22.

Wais FT. Significance of findings following biopsy and histologic study of 100-periapical lesions. Oral Surg Oral Med Oral Pathol 1958;11:650–653.

Weiner S, McKinney RV Jr, Walton RE. Characterization of the periapical surgical specimen. A morphologic and histochemical study of the inflammatory patterns. Oral Surg Oral Med Oral Pathol 1982;53:293–302.

Endodontic Flare-Up

American Association of Endodontists. Glossary of Endodontic Terms. dev.aae.org/glossary/. Accessed 1 October 2015.

de Chevigny C, Dao TT, Basrani BR, et al. Treatment outcome in endodontics: The Toronto study—Phase 4: Initial treatment. J Endod 2008;34:258–263.

Seltzer S, Naidorf IJ. Flare-ups in endodontics: II. Therapeutic measures. J Endod 1985;11:559–567.

Torabinejad M, Kettering JD, McGraw JC, Cummings RR, Dwyer TG, Tobias TS. Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps. J Endod 1988;14:261–266.

Walton R, Fouad A. Endodontic interappointment flare-ups: A prospective study of incidence and related factors. J Endod 1992;18:172–177.

Yu VS, Messer HH, Yee R, Shen L. Incidence and impact of painful exacerbations in a cohort with post-treatment persistent endodontic lesions. J Endod 2012;38:41–46.

5

Medicine and Pharmacology

Both the daily practice of endodontics and the successful completion of the American Board of Endodontics (ABE) examination process require an intimate familiarity with a broad scope of medications, medical conditions, and potential medical emergencies. Rather than an exhaustive review of medicine, the purpose of this chapter is to briefly review common pharmacologic agents used in the practice of endodontics and to cover the endodontic literature as it relates to medical conditions. A thorough review of an appropriate source, such as *Little and Falace's Dental Management of the Medically Compromised Patient,* is encouraged prior to the examination.

Pharmacology

Local anesthetics

Local anesthetic agents are used in the practice of dentistry to prevent the transmission of pain impulses from peripheral neurons to the brain. Malamed explained that these agents achieve their effect by blocking sodium channels in peripheral neurons, thus preventing depolarization of the nerve and the propagation of the action potential. Malamed also provides a thorough review of anesthetic action. Common anesthetics used in dentistry as well as their maximum dosages are listed in Table 5-1.

Table 5-1 Local anesthetic preparations available in the United States[a]

Local anesthetic	Maximum dose (mg/kg)	Maximum dose for adults (mg)	Dose of local anesthetic per cartridge (mg)	Dose of vaso-constrictor per cartridge (mg)
4% articaine 1:100,000 epinephrine	7.0	500	72	0.017
0.5% bupivacaine 1:200,000 epinephrine	1.3	90	8.5	0.0085
2% lidocaine 1:100,000 epinephrine	6.6	500	34	0.017
3% mepivacaine	6.6	400	51	NA
4% prilocaine 1:200,000 epinephrine	6.0	400	72	0.0085

[a]Data from Little et al.
NA, not applicable.

The properties of the local anesthetic depend on several factors. The speed of onset depends on the logarithmic acid dissociation contant (pK_a), with a lower pK_a causing a more rapid onset of action. The potency of the drug results from its lipid solubility. More lipid-soluble anesthetics diffuse more quickly through the neural sheath. Most commercially available local anesthetic drugs are amides and are metabolized in the liver (Malamed). Articaine has an additional ester linkage, thus permitting its metabolism by plasma esterases in addition to normal liver metabolism (Oertel et al).

The duration of action depends on the diffusion of the local anesthetic away from the site of action and is influenced both by the ability of the anesthetic to block sodium channels and the presence of a vasoconstrictor in the local anesthetic solution. All local anesthetics are vasodilators, thus vasoconstrictors like epinephrine are added in solution to slow diffusion

from the site of action (Malamed). Phentolamine mesylate [OraVerse, Septodont] acts as a vasodilator and, when administered, can reduce the duration of local anesthesia (Laviola et al).

While allergies to local anesthetics themselves are rare, allergies to sulfite preservatives in solutions containing epinephrine are more common. Additional issues with epinephrine are encountered in patients under the care of a physician for cardiac issues. Though the maximum dosage of epinephrine in a healthy patient is 0.2 mg, the maximum dosage that may be used in a cardiac patient is 0.04 mg, according to Haas. Lastly, epinephrine is subject to a number of drug-drug interactions, including tricyclic antidepressants (eg, amitryptiline, doxepin), nonselective beta blockers (eg, propranolol, nadolol), cocaine, general anesthetics (Haas), and digitalis (Becker). A thorough review of a patient's medical history is necessary prior to the administration of any local anesthetic.

Antibiotics

According to Harrison and Svec (1998a and 1998b), the use of antibiotics in the treatment of endodontic disease should be limited to those infections with rapidly increasing signs or symptoms, evidence of systemic involvement, in immunocompromised patients, and with the involvement of anatomical danger zones. The American Association of Endodontists (AAE) Colleagues for Excellence (2012) further recommended the use of antibiotic therapy in cases with a fever greater than 100°F, lymphadenopathy, trismus, osteomyelitis, and in persistent infections. The AAE Colleagues for Excellence (2006) recommended avoiding antibiotic therapy in cases of pain without signs or symptoms of infection, with an asymptomatic radiolucency, with drainage through sinus tracts, or with fluctuant swelling. Keenan et al suggested that antibiotic therapy should not be employed in the treatment of irreversible pulpitis. Controversy exists regarding the use of antibiotics in flare-up prevention, defined by the AAE glossary as an acute exacerbation of periapical pathosis after the initiation or continuation of nonsurgical root canal therapy. While Torabinejad et al recommended antibiotic therapy for the prevention of flare-ups, Pickenpaugh et al and Walton and Chiappinelli advised against their use as preventative agents. Indications for antibiotic use are summarized in Fig 5-1, and contraindications are presented in Fig 5-2.

Pathogen susceptibility must be considered when selecting an antibiotic. Using the Etest [BioMérieux], Baumgartner and Xia found that of the 98 species of bacteria collected from endodontic abscesses, 85% were susceptible to penicillin VK, 45% to metronidazole, 91% to amoxicillin, 100% to amoxicillin/clavulanate, and 96% to clindamycin. Projected values for combined therapy suggested that 93% of bacteria would be susceptible to a combination of penicillin VK and metronidazole, and 99% to a combination of amoxicillin and metronidazole.

According to the American Dental Association (ADA) Council on Scientific Affairs, antibiotic resistance is an increasing problem, both in hospitals and in the community, because of inappropriate prescription and consumption. Poeschl et al sampled bacteria from deep neck space infections and found that 10% of bacteria were resistant to penicillin G, 9% to amoxicillin, 24% to clindamycin, 24% to erythromycin, and 0% to clavulanic acid. Jungermann et al assessed the prevalence of antibiotic resistance genes in bacteria sampled from endodontic infections and found that β-lactam resistance genes were more prevalent in primary than persistent infections and were significantly reduced or eliminated after treatment. Tetracycline resistance genes were identified in a smaller number of cases; however, these were more resistant to alternative treatment methods. No vancomycin resistance genes were identified in the study.

Rapidly increasing signs or symptoms	Evidence of systemic involvement	Involvement of anatomical danger zones
Immunocompromised patients	Fever > 100°F	Lymphadenopathy
Trismus	Osteomyelis	Persistent infection

Fig 5-1 The appropriate uses for antibiotic therapy in endodontic treatment (Harrison and Svec 1998a, 1998b; AAE 2012).

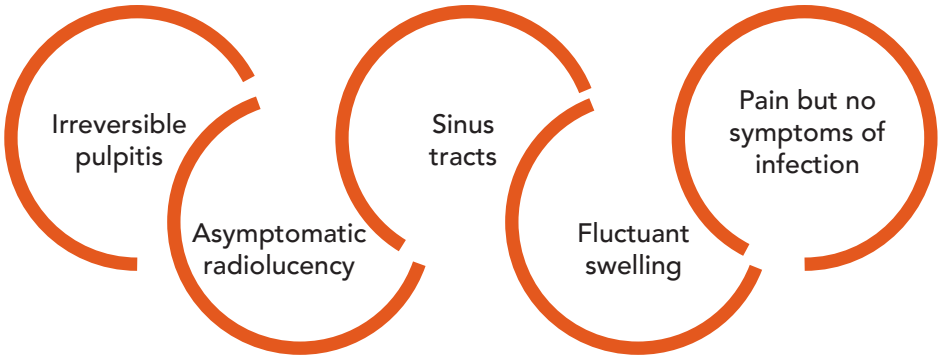

Fig 5-2 A list of contraindications for the use of antibiotic therapy in endodontic therapy (AAE 2006, Keenan et al).

Following antibiotic selection, the appropriate dosage must be administered. According to Pallasch, antibiotic therapy should be prescribed for the shortest duration possible to reduce the risk of toxicity and allergy but for long enough so that host defenses can return to fight the infection. Furthermore, doses need to be high enough to reach the minimum inhibitory concentration (MIC) and should be given at intervals of three to four times the serum half-life of the drug. Pallasch also recommended a loading dose of two times the maintenance dose to reach faster serum concentrations.

In addition to those concerns mentioned above, one must select an antibiotic appropriate for the patient's medical history, including specific medical conditions, medications, and allergies. Table 5-2 describes commonly employed antibiotics in the treatment of endodontic infections, and Table 5-3 presents a number of common interactions with antibiotics.

Table 5-2 Commonly used antibiotics in the treatment of endodontic infections[a]

Drug	Common adult dosage	Antimicrobial activity	Mechanism of action
Penicillin VK	250/500 mg 4 times daily	Bacteriocidal	Inhibits cell wall synthesis
Amoxicillin	250/500 3 times daily	Bacteriocidal	Inhibits cell wall synthesis
Augmentin	500 mg 3 times daily, 875 mg twice daily	Bacteriocidal	Inhibits cell wall synthesis
Cephalexin	250/500 mg 4 times daily	Bacteriocidal	Inhibits cell wall synthesis
Clindamycin	150/300 mg 3 times daily	Bacteriostatic	Inhibits protein synthesis
Azithromycin	500 mg on day 1 and 250 mg daily for days 2–5, or 500 mg daily for 3 days	Bacteriostatic	Inhibits protein synthesis
Metronidazole	250/500 mg 4 times daily	Bacteriocidal	Inhibits DNA synthesis

[a]Data from Ganda.

Table 5-3 Common drug-drug interactions with antibiotics[a]

Antibiotic	Interacting drug
Antibiotics (general)	Oral contraceptives
ß-lactams (penicillins and cephalosporins)	Allopurinol β blockers (tenormin, lopressor) Bacteriostatic antibiotics and tetracyclines
Tetracyclines	Antacids Insulin
Metronidazole	Ethanol Lithium
Macrolides	Benzodiazepines Carbamazepine Cyclosporine H1 histamine blockers Statins Prednisone, methylprednisolone Theophylline
Erythromycin and tetracyclines	Digoxin
Cephalosporins, erythromycin, clarithromycin, metronidazole	Warfarin

[a]Data from Little et al.

Antibiotic prophylaxis

Prophylactic antibiotics are administered to at-risk patients without evidence of infection to prevent complications as a result of bacteremias that might result from treatment (AAE 2012). Bacteremia, or the often transient presence of bacteria in the blood stream (AAE glossary), may result from dental treatment performed by endodontists. The occurrence of bacteremias following nonsurgical root canal therapy is controversial. Baumgartner et al (1976) was unable to detect evidence of bacteremias in patients undergoing nonsurgical root canal therapy when instrumentation was confined to the tooth. Conversely, Debilian et al demonstrated the occurrence of bacteremias during standard nonsurgical root canal therapy. The occurrence of bacteremias following soft tissue surgery, however, is irrefutable. Baumgartner et al (1977) demonstrated bacteremias in patients undergoing surgical root canal therapy.

The prescription of prophylactic antibiotics in dentistry has traditionally been limited to two groups of patients, those at risk for infective endocarditis and those with a history of prosthetic joint replacement. In their 2007 guidelines, the American Heart Association (Wilson et al) recommended antibiotic prophylaxis prior to dental procedures for those patients at risk for adverse outcomes if infective endocarditis were to develop (Fig 5-3). These include patients with prosthetic heart valves, a history of infective endocarditis, unrepaired or incompletely repaired cyanotic congenital heart defects, completely repaired cyanotic congenital heart defects within 6 months of the repair, and repaired congenital cyanotic heart defects with residual defects at the surgical site (Wilson et al).

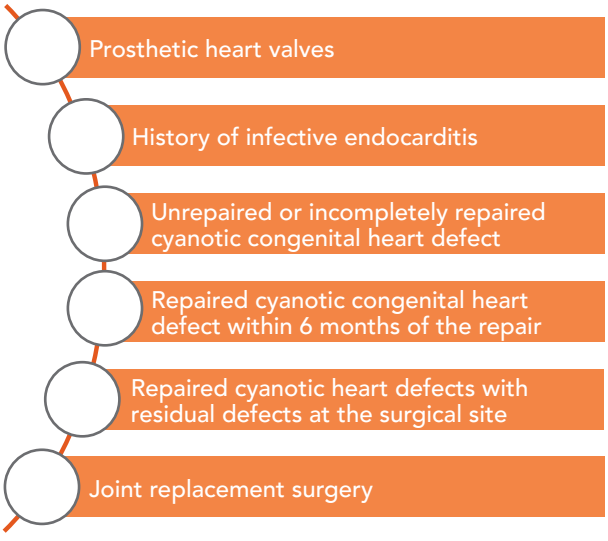

Fig 5-3 A list of conditions for which antibiotic premedication may be indicated prior to dental treatment (AAOS 2009, Wilson et al).

In addition to those patients at risk for adverse outcomes as a result of infective endocarditis, antibiotic prophylaxis may be indicated for patients with prosthetic joints. In 2009, the American Association of Orthopedic Surgeons (AAOS) recommended that clinicians consider prophylactic antibiotic therapy for joint replacement patients prior to any invasive procedure. In response, the American Association of Oral Medicine reviewed the literature and concluded that the risks of antibiotic prophylaxis do not justify the practice of using prophylactic antibiotics routinely on patients with prosthetic joints. The authors of this statement called for a future multidisciplinary review of the literature. Consequently in 2012, a joint systematic review of the literature by the ADA and the

AAOS (Jevsevar and Abt) found dental procedures are unrelated to prosthesis infection and that antibiotic prophylaxis, though reducing the incidence of bacteremias during dental procedures, does not reduce the risk of subsequent prosthesis infection. Consequently, the organizations recommend that practitioners may discontinue the practice of routinely prescribing prophylactic antibiotics for patients with prosthetic joints undergoing joint prophylaxis. As a result, the decision to premedicate a patient prior to dental procedures should not be considered an absolute recommendation but should rather be determined by thoughtful consideration by the dentist, patient, and surgeon (Jevsevar and Abt).

Analgesics

According to Nusstein and Beck, 81% to 84% of patients reported taking analgesics prior to their initial endodontic appointment. Read et al suggested that this may prove problematic clinically because analgesics, including ibuprofen, can affect endodontic diagnostic testing results including cold, percussion, and palpation. However, the combination of hydrocodone and acetaminophen may not affect the reliability of pulp tests (Kardelis et al). Consequently, prior to any endodontic assessment, one must inquire about a patient's recent analgesic usage.

Unfortunately, in symptomatic patients, the use of preoperative analgesics does not increase the success rates of local anesthetic injections. Ianiro et al found no significant difference between the success rates of inferior alveolar nerve blocks in patients given acetaminophen or a combination of ibuprofen and acetaminophen before treatment. Oleson et al found similar results.

Postoperative analgesics are often necessary in clinical endodontic practice. The prevalence of postoperative pain has been reported between 3% and 58% (Sathorn et al). Yu et al reported a 5.8% risk of more acute pain related to flare-up following endodontic treatment of persistent periapical lesions. More commonly, Yu et al found a lower degree of pain with a minimal impact on activities of daily living in 45% of patients. The majority of postoperative pain occurred within the first 24 hours after treatment. Furthermore, Torabinejad et al found that the incidence of postoperative pain is typically higher following cleaning and shaping as opposed to obturation. Mattscheck et al reported that the degree of postoperative discomfort is significantly influenced by the pretreatment pain level.

Analgesic use should be appropriate to control expected postoperative pain. According to Mickel et al, a survey of endodontists found that 600 mg ibuprofen four times a day was the most preferred analgesic prescribed for patients regardless of their perceived level of pain, endodontic diagnosis, or treatment rendered. Furthermore, educators and board-certified AAE members were less likely than non-board-certified AAE members to manage their patients' perceived severe pain with a narcotic.

Analgesic drugs available to treat endodontic discomfort exert their effects by different mechanisms. Ibuprofen, a nonsteroidal anti-inflammatory drug (NSAID), asserts its effects by blocking the cyclooxygenase 1 and 2 enzymes, thus preventing the production of prostaglandins (Hargreaves et al). Acetaminophen has antipyretic and analgesic activity comparable with aspirin. The presumed mechanism of action proposed by Yagiela et al is inhibition of prostaglandin synthesis as well as interactions with both the cannabinoid and serotonergic systems. Narcotics, which assert their effects on central μ and κ opioid receptors, are sometimes advocated in the treatment of severe odontogenic pain (Hargreaves and Keiser).

Menhinick et al found that the combination of ibuprofen and acetaminophen was superior to either drug alone in the alleviation of postoperative discomfort. Wells et al refuted that claim, however, stating that both the combination and ibuprofen alone are equally as effective. Hargreaves and Keiser advocated maximizing the use of NSAIDs prior to the administration of a narcotic pain reliever. The maximum recommended dose of ibuprofen over 24 hours is 3.2 grams, according to Gage and Pickett. In a recent study by Parirokh et al, no differences were noted between medications taken on a time schedule versus on-demand medication for 48 hours after treatment. Table 5-4 lists commonly prescribed analgesic agents used in dentistry, and Table 5-5 presents some commonly reported drug interactions with analgesic medications.

Table 5-4 Commonly used analgesics in dentistry[a]

Drug	Prescription
Ibuprofen	200–600 mg every 4–6 h
Acetaminophen	325–650 mg every 4–6 h
Vicodin (hydrocodone plus acetaminophen)	5/325-mg tablets, 1–2 every 4–6 h
Percocet (oxycodone plus acetaminophen)	5/325-mg tablets, 1–2 every 4–6 h

[a]Data from Ganda.

Table 5-5 Common drug-drug interactions with analgesics[a]

Analgesic	Interacting drug
Acetaminophen	Alcohol
Aspirin	Oral hypoglycemic agents (sulfonylureas, glyburide, chlorpropamide)
Aspirin and NSAIDS	Alcohol Anticoagulants
NSAIDS	ß-blockers ACE inhibitors Lithium Methotrexate

[a]Data from Little et al.
ACE, angiotensin-converting enzyme.

Anxiolytics

The thought of endodontic treatment may cause anxiety for some patients. Consequently, clinician familiarity with anxiolytic medications may prove useful. Oral anxiolytics are easily administered and often very effective. Benzodiazepines are commonly prescribed oral anxiolytics that work via binding to γ-aminobutyric acid (GABA) channels to assert their effects (Page et al). Kaufman et al found that triazolam was safe and effective for dental treatment.

Ehrich et al found that 0.25 mg of triazolam was more effective than 5 mg of diazepam in relieving dental anxiety. Unfortunately, the use of these medications does not increase the success rates of local anesthetic injections in symptomatic patients. Lindemann et al found no significant increase in anesthetic success rate in those patients given sublingual triazolam prior to the administration of local anesthetics.

Inhalational anxiolytics may also prove useful for treatment of dental anxiety. In addition to its effect as an anxiolytic, nitrous oxide may also have analgesic effects, according to Becker and Rosenberg. These effects are the result of the release of endogenous opioid peptides and the inhibition of N-methyl-D-aspartate (NMDA) receptors (Emmanouil and Quock). Furthermore, Stanley et al demonstrated that nitrous oxide may increase the success rates of inferior alveolar nerve blocks in patients with irreversible pulpitis.

Medical Conditions Referenced in the Endodontic Literature

Though a thorough review of medical conditions is not permissible within the scope of this chapter, a review of medical conditions recently referenced in the endodontic literature is included. Conditions are presented in alphabetic order.

Abscess

Dental abscesses are frequently but not exclusively caused by endodontic infections. Campanelli and Walton found that the presence of a dental abscess did not affect patient vital signs.

Anemia

Several types of anemia have been described in the medical literature, including sickle cell anemia, which results from mutations in the hemoglobin A gene. Costa et al (2013) found that the presence of sickle cell anemia is a potential risk factor for spontaneous pulp necrosis, with an odds ratio of 8.3. In other words, a patient with sickle cell anemia is 8.3 times more likely to experience spontaneous pulp necrosis than a patient without the disease.

Bisphosphonate use

Bisphosphonates slow bone turnover and are used in the treatment of osteoporosis and bone-associated cancers. High-dose intravenous bisphosphonates and traumatic dental surgery put patients at risk for developing osteonecrosis, an entity termed *bisphosphonate-related osteonecrosis of the jaw (BRONJ).* According to Ruggiero et al, patients receiving oral therapy are at a lower risk. The incidence of BRONJ has been reported between 1% and 10% in different population groups, as reviewed by McLeod et al. To prevent BRONJ, a thorough dental examination and treatment of any pathology is recommended prior to the start of bisphosphonate therapy. Should patients require dental treatment after the start of bisphosphonate therapy, root canal therapy is preferable to dental extractions and apical surgery, as suggested by Katz. Furthermore, patients having undergone bisphosphonate therapy may experience pain that resembles odontogenic pain (Katz). A

recent report by Alsalleeh et al found that co-administration of immune suppressant medications like methotrexate may complicate the management of BRONJ.

Chemotherapeutics and radiation

Radiation therapy for oropharyngeal malignancies in doses greater than 30 to 35 grays can reduce the number of teeth responding to pulp sensitivity tests, according to Kataoka et al. Despite the effects on pulp vitality tests, Faria et al showed that pulp histology is not altered by radiation. No changes in pulpal microvasculature, innervation, or extracellular matrix proteins were noted in patients subject to radiation therapy.

Chemotherapeutic and antibiologic drugs, like those used to treat rheumatoid arthritis and similar diseases, target tumor necrosis factor α, interleukin-1, interleukin-6, T cells, or B cells and block the activity of these targets. As apical periodontitis is an inflammatory disease resulting from interactions between microbes and host immune functions, biologic medications may alter the progression of apical periodontitis. According to Cotti et al (2014), the majority of published studies examining the interaction between the biologics and oral diseases focus on periodontal disease. The results of these studies indicate that biologic medications may influence both disease severity and patient response to treatment.

Cardiovascular disease

The interplay between oral and systemic health often centers on interactions between oral and systemic diseases, particularly cardiovascular disease. Consequently, many published studies have investigated the link between cardiovascular disease and endodontic lesions. Pasqualini et al correlated the presence of apical periodontitis with cardiovascular disease. Similarly, Costa et al (2014) demonstrated in a cross-sectional study that patients with chronic apical periodontitis had a 2.8 times higher risk of developing coronary artery disease than those without chronic apical disease. The mechanism may be a spread of inflammation from the periapex to the systemic circulation. Cotti et al (2011) suggested that low-grade chronic inflammation and associated systemic interleukin-2 (IL-2) increases, like that seen in apical periodontitis, may be a risk factor for the development of atherosclerosis because of IL-2's ability to induce endothelial dysfunction.

Just as endodontic disease may affect cardiovascular disease, the converse may also be true, including both preoperative and postoperative conditions. Edds et al found a greater prevalence of pulp stones in patients with cardiovascular disease than in those without it. Su et al found that both hypertension and coronary artery disease were significant risk factors for extraction of teeth after nonsurgical root canal therapy.

Diabetes

Diabetes has been associated with an increased prevalence of endodontic disease. Marotta et al found that apical periodontitis was more prevalent in untreated teeth in patients with type 2 diabetes. Lopez-Lopez et al (2011) found that, although the presence of types 1 and 2 diabetes was associated with an increase in the incidence of apical periodontitis among all teeth, no difference was noted in the incidence of apical periodontitis in previously root canal–treated teeth.

Other authors, however, have found an association between apical periodontitis in previously treated teeth and the presence of diabetes. Fouad and Burleson found not only an increase in the rate of flare-ups in diabetic patients but also a reduced likelihood of success following root canal therapy. Wang et al found that diabetes was a significant risk factor for extraction following root canal therapy, supporting Fouad's findings.

Hepatitis

Hepatitis can result from viral infections, toxin exposure, contaminated food and water, and other causes. Grawish et al found that the presence of hepatitis C has been associated with coronal dental pulp abnormalities, including inappropriate cellularity, deranged vasculature, and altered extracellular matrix proteins.

Lyme disease

Lyme disease is an infection caused by the bacteria *Borrelia burgdorferi* and its interaction with the immune system. Heir and Fein described the occurrence of orofacial pain, temporomandibular disorder pain, and dental pain in patients with lyme disease.

Lymphoma

Periapical lesions may present without endodontic cause. Koivisto et al and Mendonca et al presented several cases of periapical lesions that were revealed to be lymphoma on biopsy. Hodgkin lymphoma has a predilection for the neck and mediastinum and is often found in the lymph nodes, whereas non-Hodgkin lymphoma is frequently extranodal. The oral cavity is cited as the location of 2% to 3% of these non-Hodgkin lesions (Koivisto et al). According to Kemp et al, these lesions present more often in the maxilla than the mandible.

Multiple myeloma

Multiple myeloma is a tumor caused by proliferation of plasma cells. It often causes punched-out lesions in the jaw, which may resemble apical periodontitis. Further association with root resorption was reported in a recent case report and literature review by Troeltzsch et al.

Smoking

Smoking undoubtedly has an effect on systemic immunity and microvasculature. Nociti et al reviewed its associations with periodontal disease. Several recent studies have found associations between smoking and the presence of apical periodontitis. Lopez-Lopez et al (2012) confirmed this finding in a retrospective case-control study. Segura-Egea et al found that the prevalence of both apical periodontitis and a history of endodontic treatment were significantly higher in smokers with hypertension when compared with non-smoking hypertensive patients.

Bibliography

Introduction

Little JW, Miller D, Rhodus NL, Falace D (eds). Little and Falace's Dental Management of the Medically Compromised Patient, ed 8. St Louis: Mosby, 2012.

Pharmacology

American Academy of Orthopaedic Surgeons. Information Statement: Antibiotic Prophylaxis for Bacteremia in Patients with Joint Replacements. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2009.

American Academy of Orthopaedic Surgeons. Prevention of Orthopaedic Implant Infection in Patients Undergoing Dental Procedures; Evidence-Based Guideline and Evidence Report. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2012.

American Association of Endodontists. Endodontics Colleagues for Excellence: Antibiotics and the Treatment of Endodontic Infections. Chicago: American Association of Endodontists, 2006.

American Association of Endodontists. Endodontics Colleagues for Excellence: Use and Abuse of Antibiotics. Chicago: American Association of Endodontists, 2012.

American Association of Endodontists. Glossary of Endodontic Terms. dev.aae.org/glossary/. Accessed 20 January 2016.

American Dental Association Council on Scientific Affairs. Combating antibiotic resistance. J Am Dent Assoc 2004;135:484–487.

Baumgartner JC, Heggers JP, Harrison JW. Incidence of bacteremias related to endodontic procedures. II. Surgical endodontics. J Endod 1977;3:399–402.

Baumgartner JC, Heggers JP, Harrison JW. The incidence of bacteremias related to endodontic procedures. I. Nonsurgical endodontics. J Endod 1976;2:135–140.

Baumgartner JC, Xia T. Antibiotic susceptibility of bacteria associated with endodontic abscesses. J Endod 2003;29:44–47.

Becker DE. Cardiovascular drugs: Implications for dental practice part 1—Cardiotonics, diuretics, and vasodilators. Anesth Prog 2007;54:178–185.

Becker DE, Rosenberg M. Nitrous oxide and the inhalation anesthetics. Anesth Prog 2008;55:124–130.

Debelian GJ, Olsen I, Tronstad L. Bacteremia in conjunction with endodontic therapy. Endod Dent Traumatol 1995;11:142–149.

Ehrich DG, Lundgren JP, Dionne RA, Nicoll BK, Hutter JW. Comparison of triazolam, diazepam, and placebo as outpatient oral premedication for endodontic patients. J Endod 1997;23:181–184.

Emmanouil DE, Quock RM. Advances in understanding the actions of nitrous oxide. Anesth Prog 2007;54:9–18.

Gage TW, Pickett FA. Mosby's Dental Drug Reference, ed 7. St Louis: Mosby, 2005.

Ganda KM. Dentist's Guide to Medical Conditions and Complications. Ames, Iowa: Wiley-Blackwell, 2008.

Haas DA. An update on local anesthetics in dentistry. J Can Dent Assoc 2002;68:546–551.

Hargreaves KM, Keiser K. Development of new pain management strategies. J Dent Educ 2002;66:113–121.

Hargreaves KM, Troullos ES, Dionne RA. Pharmacologic rationale for the treatment of acute pain. Dent Clin N Am 1987;31:675–694.

Harrison JW, Svec TA. The beginning of the end of the antibiotic era? Part I. The problem: Abuse of the "micracle drugs." Quintessence Int 1998a;29:151–162.

Harrison JW, Svec TA. The beginning of the end of the antibiotic era? Part II. Proposed solutions to antibiotic abuse. Quintessence Int 1998b;29:223–229.

Ianiro SR, Jeansonne BG, McNeal SF, Eleazer PD. The effect of preoperative acetaminophen or a combination of acetaminophen and ibuprofen on the success of inferior alveolar nerve block for teeth with irreversible pulpitis. J Endod 2007;33:11–14.

Jevsevar DS, Abt E. The new AAOS-ADA clinical practice guideline on prevention of orthopaedic implant infection in patients undergoing dental procedures. J Am Acad Orthop Surg 2013;21:195–197.

Jungermann GB, Burns K, Nandakumar R, Tolba M, Venezia RA, Fouad AF. Antibiotic resistance in primary and persistent endodontic infections. J Endod 2011;37:1337–1344.

Kardelis AC, Meinberg TA, Sulte HR, Gound TG, Marx DB, Reinhardt RA. Effect of narcotic pain reliever on pulp tests in women. J Endod 2002;28:537–539.

Kaufman E, Hargreaves KM, Dionne RA. Comparison of oral triazolam and nitrous oxide with placebo and intravenous diazepam for outpatient premedication. Oral Surg Oral Med Oral Pathol 1993;75:156–164.

Keenan JV, Farman AG, Fedorowicz Z, Newton JT. Antibiotic use for irreversible pulpitis. Cochrane Database Syst Rev 2005:CD004969.

Laviola M, McGavin SK, Freer GA, et al. Randomized study of phentolamine mesylate for reversal of local anesthesia. J Dent Res 2008;87:635–639.

Lindemann M, Reader A, Nusstein J, Drum M, Beck M. Effect of sublingual triazolam on the success of inferior alveolar nerve block in patients with irreversible pulpitis. J Endod 2008;34:1167–1170.

Little JW, Miller D, Rhodus NL, Falace D (eds). Little and Falace's Dental Management of the Medically Compromised Patient, ed 8. St Louis: Mosby, 2012.

Malamed SF (ed). Handbook of Local Anesthesia, ed 6. St Louis: Mosby, 2012.

Mattscheck DJ, Law AS, Noblett WC. Retreatment versus initial root canal treatment: Factors affecting posttreatment pain. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92:321–324.

Menhinick KA, Gutmann J, Regan JD, Taylor SE, Buschang PH. The efficacy of pain control following nonsurgical root canal treatment using ibuprofen or a combination of ibuprofen and acetaminophen in a randomized, double-blind, placebo-controlled study. Int Endod J 2004;37:531–541.

Mickel AK, Wright AP, Chogle S, Jones JJ, Kantorovich I, Curd F. An analysis of current analgesic preferences for endodotnic pain management. J Endod 2006;32:1146–1154.

Nusstein JM, Beck M. Comparison of preoperative pain and medication use in emergency patients presenting with irreversible pulpitis or teeth with necrotic pulps. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96:207–214.

Oertel R, Rahn R, Kirch W. Clinical pharmacokinetics of articaine. Clin Pharmacokinet 1997;33:417–425.

Oleson M, Drum M, Reader A, Nusstein J, Beck M. Effect of preoperative ibuprofen on the success of the inferior alveolar nerve block in patients with irreversible pulpitis. J Endod 2010;36:379–382.

Page C, Curtis M, Sutter M, Walker M, Hoffman B (eds). Integrated Pharmacology, ed 2. St Louis: Mosby, 2002.

Pallasch TJ. Pharmacokinetic principles of antimicrobial therapy. Periodontol 2000;10:5–111.

Parirokh M, Sadr S, Nakhaee N, Abbott PV, Manochehrifar H. Comparison between prescription of regular or on-demand ibuprofen on postoperative pain after single-visit root canal treatment of teeth with irreversible pulpitis. J Endod 2014;40:151–154.

Pickenpaugh L, Reader A, Beck M, Meyers WJ, Peterson LJ. Effect of prophylactic amoxicillin on endodontic flare up in asymptomatic necrotic teeth. J Endod 2001;27:53–56.

Poeschl PW, Crepaz V, Russmueller G, Seemann R, Hirschl AM, Ewers R. Endodontic pathogens causing deep neck space infections: Clinical impact of different sampling techniques and antibiotic susceptibility. J Endod 2011;37:1201–1205.

Read JK, McClanahan SB, Khan AA, Lunos S, Bowles WR. Effect of ibuprofen on masking endodontic diagnosis. J Endod 2014;40:1058–1062.

Sathorn C, Parashos P, Messer H. The prevalence of postoperative pain and flare-up in single- and multiple-visit endodontic treatment: A systematic review. Int Endod J 2008;41:91–99.

Stanley W, Drum M, Nusstein J, Reader A, Beck M. Effect of nitrous oxide on the efficacy of the inferior alveolar nerve block in patients with symptomatic irreversible pulpitis. J Endod 2012;38:565–569.

Torabinejad M, Kettering JD, McGraw JC, Cummings RR, Dwyer TG, Tobias TS. Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps. J Endod 1994;14:261–266.

Walton RE, Chiappinelli J. Prophylactic penicillin: Effect on posttreatment symptoms following root canal treatment of asymptomatic periapical pathosis. J Endod 1993;19:466–470.

Wells LK, Drum M, Nusstein J, Reader A, Beck M. Efficacy of ibuprofen and ibuprofen/acetaminophen on postoperative pain in symptomatic patients with a pulpal diagnosis of necrosis. J Endod 2011;37:1608–1612.

Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: Guidelines from the American Heart Association. Circulation 2007;116:1736–1754.

Yagiela JA, Neidle EA, Dowd FJ. Pharmacology and therapeutics for dentistry. St Louis: Mosby, 1998.

Yu VSH, Messer HH, Yee R, Shen L. Incidence and impact of painful exacerbations in a cohort with post-treatment persistent endodontic lesions. J Endod 2012;38:41–46.

Medical Conditions Referenced in the Endodontic Literature

Alsalleeh F, Keippel J, Adams L, Bavitz B. Bisphosphonate-associated osteonecrosis of jaw reoccurrence after methotrexate therapy: A case report. J Endod 2014;40:1505–1507.

Campanelli CA, Walton RE, Williamson AE, Drake DR, Qian F. Vital signs of the emergency patient with pulpal necrosis and localized acute apical abscess. J Endod 2008;34:264–267.

Costa CP, Thomaz EB, Souza Sde F. Association between sickle cell anemia and pulp necrosis. J Endod 2013;39:177–181.

Costa TH, de Figueiredo Neto JA, de Oliveira AE, Lopes e Maia Mde F, de Almeida AL. Association between chronic apical periodontitis and coronary artery disease. J Endod 2014;40:164–167.

Cotti E, Dessi C, Piras A, et al. Association of endodontic infection with detection of an initial lesion to the cardiovascular system. J Endod 2011;37:1624–1629.

Cotti E, Schirru E, Acquas E, Usai P. An overview on biologic medications and their possible role in apical periodontitis. J Endod 2014;40:1902–1911.

Edds AC, Walden JE, Scheetz JP, Goldsmith LJ, Drisko CL, Eleazer PD. Pilot study of correlation of pulp stones with cardiovascular disease. J Endod 2005;31:504–506.

Faria KM, Brandao TB, Ribeiro AC, et al. Micromorphology of the dental pulp is highly preserved in cancer patients who underwent head and neck radiotherapy. J Endod 2014;40:1553–1559.

Fouad AF, Burleson J. The effect of diabetes mellitus on endodontic treatment outcome: Data from an electronic patient record. J Am Dent Assoc 2003;134:43–51.

Grawish MA, Khounganian R, Hamam MK, et al. Altered coronal tissue of the human dental pulp in chronic hepatitis C virus infected patients. J Endod 2013;39:752–758.

Heir GM, Fein LA. Lyme disease: Considerations for dentistry. J Orofac Pain 1996;10:74–86.

Kataoka SH, Setzer FC, Fregnani ER, Pessoa OF, Gondim E Jr, Caldeira CL. Effects of 3-dimensional conformal or intensity-modulated radiotherapy on dental pulp sensitivity during and after the treatment of oral or oropharyngeal malignancies. J Endod 2012;38:148–152.

Katz H. Endodontic implications of bisphosphonate-associated osteonecrosis of the jaws: A report of three cases. J Endod 2005;31:831–834.

Kemp S, Gallagher G, Kabani S, Noonan V, O'Hara C. Oral non-Hodgkin's lymphoma: Review of the literature and World Health Organization classification with reference to 40 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105:194–201.

Koivisto T, Bowles WR, Magajna WA, Rohrer M. Malignant lymphoma in maxilla with cystic involvement: A case report. J Endod 2013;39:935–938.

Lopez-Lopez J, Jane-Salas E, Estrugo-Devesa A, Velasco-Ortega E, Martin-Gonzalez J, Segura-Egea JJ. Periapical and endodontic status of type 2 diabetic patients in Catalonia, Spain: A cross-sectional study. J Endod 2011;37:598–601.

Lopez-Lopez J, Jane-Salas E, Martin-Gonzalez J, et al. Tobacco smoking and radiographic periapical status: A retrospective case-control study. J Endod 2012;38:584–588.

Marotta PS, Fontes TV, Armada L, Lima KC, Rôças IN, Siqueira JF Jr. Type 2 diabetes mellitus and the prevalence of apical periodontitis and endodontic treatment in an adult Brazilian population. J Endod 2012;38:297–300.

McLeod NM, Brennan PA, Ruggiero SL. Bisphosphonate osteonecrosis of the jaw: A historical and contemporary review. Surgeon 2012;10:36–42.

Mendonca EF, Sousa TO, Estrela C. Non-Hodgkin lymphoma in the periapical region of a mandibular canine. J Endod 2013;39:839–842.

Nociti FH Jr, Casati MZ, Duarte PM. Current perspective of the impact of smoking on the progression and treatment of periodontitis. Periodontol 2000 2015;67:187–210.

Pasqualini D, Bergandi L, Palumbo L, et al. Association among oral health, apical periodontitis, CD14 polymorphisms, and coronary heart disease in middle-aged adults. J Endod 2012;38:1570–1577.

Ruggiero SL, Dodson TB, Assael LA, et al. American Association of Oral and Maxillofacial Surgeons position paper on bisphosphonate-related osteonecrosis of the jaws—2009 update. J Oral Maxillofac Surg 2009;67:2–12.

Segura-Egea JJ, Castellanos-Cosano L, Velasco-Ortega E, et al. Relationship between smoking and endodontic variables in hypertensive patients. J Endod 2011;37:764–767.

Su Y, Wang C, Ye L. Healing rate and post-obturation pain of single- versus multiple-visit endodontic treatment for infected root canals: A systematic review. J Endod 2011;37:125–132.

Troeltzsch M, Oduncu F, Mayr D, Ehrenfeld M, Pautke C, Otto S. Root resorption caused by jaw infiltration of multiple myeloma: Report of a case and literature review. J Endod 2014;40:1260–1264.

Wang CH, Chueh LH, Chen SC, Feng YC, Hsiao CK, Chiang CP. Impact of diabetes mellitus, hypertension, and coronary artery disease on tooth extraction after nonsurgical endodontic treatment. J Endod 2011;37:1–5.

6

Diagnosis

Prior to endodontic treatment, a definitive diagnosis must be established. In practice and in the board examination process, a systematic approach to patient care is necessary to ensure a proper, thorough work-up and an accurate diagnosis. This chapter discusses diagnostic protocols and tests as well as the research on which these are based. Furthermore, radiographic adjuncts to clinical diagnostic procedures, including cone beam computed tomography (CBCT) technology, are covered. Lastly, this chapter concludes with a review of common endodontic diagnoses. Figure 6-1 outlines a comprehensive, step-wise patient evaluation.

Clinical examination

Subjective examination

- Chief complaint
- Dental history
  - Localization
  - Commencement
  - Duration
  - Intensity
  - Provocation/relief
  - Frequency
  - Progression
  - Quality
  - Associated symptoms
  - History
- Medical history
  - Problems/diseases
  - Associated laboratory tests
  - Medications
  - Allergies
  - Adverse events
  - Last visit with PCP
  - Social history

Objective examination

- Vital signs
- Extraoral examination
- Intraoral examination
  - Full-mouth soft and hard tissue examination
  - Focused view on tooth or teeth in question
    - Associated swelling or sinus tracts
    - Pulp sensitivity testing
    - PDL (percussion, palpation, biting)
    - Periodontal examination
    - Other: transillumination, selective anesthesia

Radiographic examination

- Periapical radiograph
- Bitewing radiograph
- CBCT, when indicated

Fig 6-1 Suggested elements of an examination. PCP, primary care physician; PDL, periodontal ligament.

Clinical Examination

The clinical examination can be separated into a subjective examination that involves a patient interview and an objective examination that involves a physical inspection by the practitioner.

Subjective examination

The clinical examination should begin with a subjective examination of the patient, involving a patient interview. The patient's chief complaint should be recorded in the patient's own words. A dental history should be obtained consisting of the following elements, as outlined by Berman and Hartwell: localization of the problem, symptom commencement, symptom duration, pain intensity, things that provoke symptoms, things that relieve symp-

toms, frequency of symptoms, progression of symptoms, quality of symptoms, any other associated symptoms, and history of symptoms or treatment in the same tooth or region.

At this time, a comprehensive medical history should be obtained including the following elements: medical problems or diseases, associated laboratory values, prescription and nonprescription medications and supplements (as well as the patient's compliance with these medications), allergies, any history of adverse events with dental or medical procedures, date of the patient's last visit with a primary care or other monitoring physician, and a social history, including use of tobacco, alcohol, or recreational drugs. For a more detailed discussion of medical conditions germane to endodontic treatment, please refer to chapter 5.

Objective examination

The objective examination involves the physical examination of the patient, as outlined by Berman and Hartwell. Vital signs should be recorded, including blood pressure, pulse readings, and respiratory rate for all patients. Additionally, for those patients with a suspected infection, temperature should be measured. Following vital sign measurement, the extraoral examination should be completed. Particularly close attention should be paid to symmetry with a patient's mouth open and closed, extraoral swelling or sinus tracts, and cervicofacial lymphadenopathy.

An intraoral examination should follow, including a full examination of oral soft tissues with particular attention to symmetry, swelling, and sinus tracts. A thorough oral cancer screening should be performed. Oral hard tissues should be examined to determine areas of caries, defective restorations, color changes, or other variations from normal structure.

A focused examination should then follow on the tooth or teeth in question. According to Bender, odontogenic pain can refer discomfort to neighboring teeth and between jaws, so it is important to examine not only the suspected tooth but several adjacent teeth as well as teeth in the opposing jaw. During this examination, the diagnosing clinician should consider any factors that might influence the accuracy of diagnosis. For example, Read et al found that ibuprofen taken by patients prior to diagnostic testing could significantly affect the results of percussion, palpation, and cold testing. On the other hand, Fowler et al showed that a combination of acetaminophen and hydrocodone had no effect on cold testing in patients with symptomatic irreversible pulpitis.

The focused examination should include pulp sensitivity testing, the key element in the establishment of a pulpal diagnosis of normal pulp vitality, pulpitis, or pulpal necrosis. The most commonly available pulp sensitivity tests are thermal and electric pulp testing (EPT). Pulp sensitivity testing is of course a conduit. No test currently available provides an absolutely reliable indicator of the pulp's histologic status, a concept first described by Seltzer et al (1963). Regardless, Ricucci et al found an 84% to 97% agreement between clinical and histologic diagnoses using currently available diagnostic criteria. These results indicate that the clinical tests on which treatment is recommended can often accurately discern the true diagnosis.

Cold testing is generally considered the first-line pulp sensitivity test. According to Trowbridge et al, cold testing relies on outward hydrodynamic fluid flow to stimulate Aδ fibers. Cold testing poses no threats to pulp vitality. Rickoff et al found no pathologic changes

in the pulp as a result of cold testing. The recommended method of cold testing utilizes a topical refrigerant spray, such as Endo Ice [Hygenic], containing tetrofluoroethane or a similar chemical because of its ease of use and availability. White and Cooley recommended Endo Ice based on convenience and demonstrated its superiority over a cold-water bath or ice stick. Miller et al found refrigerant sprays to be the superior method of cold testing in teeth restored with full-coverage porcelain-fused-to-metal restorations. Carbon dioxide snow and ethyl chloride are alternative methods for cold testing.

Jespersen et al compared the accuracy of pulp sensitivity testing to direct visualization of the dental pulp. They reported a positive predictive value of 0.86, meaning that a lack of response to cold testing using Endo Ice corresponded to true pulpal necrosis 86% of the time. They also reported a negative predictive value of 0.94, meaning that a response to cold indicated pulp vitality 94% of the time. In total, Jespersen et al found that cold testing was 90% accurate.

Heat testing is generally reserved as a second-line pulp sensitivity test or when a patient's chief complaint includes heat sensitivity. Heat testing is safe for patients. Rickoff et al found no pathologic changes in the pulp as a result of heat testing. Bierma et al found that a special tip on a System B endodontic fill device [Kerr Dental], with a reservoir to heat a small piece of gutta-percha, was the most consistent and safe method of heat testing. However, heated gutta-percha was also considered safe. The authors advised against use of a heated ball burnisher or hot water because these methods may create temperatures high enough to cause pulpal damage. Although one often assumes that previously treated teeth should not respond to pulp sensitivity tests, Keir et al presented a case series of a positive heat response in previously treated teeth as a result of untreated anatomy.

Petersson et al compared heat testing to direct visualization of the dental pulp. They reported a positive predictive value of 0.48, meaning that a lack of response to heat testing using heated gutta-percha corresponded to true pulpal necrosis only 48% of the time, which indicates a high incidence of false positives. Petersson et al reported a negative predictive value of 0.83, indicating that a response to heat correctly identified pulp vitality 83% of the time.

EPT offers an additional means for pulp sensitivity testing. Bender described ionic changes in dentinal fluid resulting from the EPT, which lead to stimulation of Aδ fibers (Fig 6-2). Fulling and Andreasen found that EPT was not accurate in immature teeth due to late development of the responsive Aδ nerve fibers. Ketterl reported that EPT might

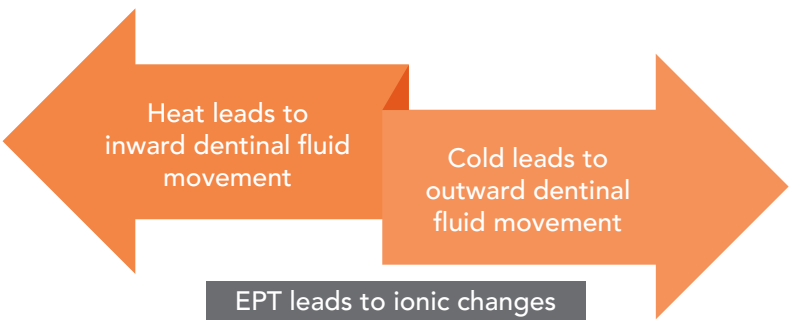

Fig 6-2 Pulp sensitivity testing leads to fluid changes causing Aδ fiber stimulation (Bender).

be more useful than thermal testing in calcified teeth due to loss of the fluid needed for hydrodynamic Aδ fiber stimulation. Because orthodontic tooth movement may alter EPT thresholds, Alomari et al suggested thermal sensitivity tests might be more accurate during orthodontic treatment. Based on a clinical study by Bender et al, ideal placement of the electrode for testing is on the incisal edge or cusp tip.

Some have proposed that EPT should not be used in patients with a pacemaker due to possible interference with the pacemaker's functioning. However, Wilson et al found that EPT does not interfere with pacemaker function and affirm its safety. As a precaution, though, it is best to check with the manufacturer of the EPT device as well as the patient's physician prior to use.

EPT is most useful to rule out pulpal necrosis. Jespersen et al compared pulp sensitivity testing to direct visualization of the dental pulp and reported a positive predictive value of 0.58, meaning that a lack of response to EPT corresponded to true pulpal necrosis 58% of the time. On the other hand, they reported a negative predictive value of 0.90, indicating a positive response to EPT represented pulp vitality 90% of the time. They found that EPT was 75% accurate. As a result of the relatively high occurrence of false positives, EPT is generally reserved as a secondary measure to confirm suspected cases of pulpal necrosis. A summary of the epidemiologic findings can be found in Table 6-1.

Table 6-1 Epidemiology of pulp sensitivity testing

	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
Cold[a]	0.92	0.90	0.86	0.94	90%
Heat[b]	0.86	0.41	0.48	0.83	NA
EPT[a]	0.84	0.74	0.58	0.90	75%

[a]Data from Jespersen et al.
[b]Data from Petersson et al.
NA, not available.

It is important to note that the currently available pulp tests actually test pulp sensitivity, and not pulp vitality. These measures, though frequently correlated, are not always one and the same. For example, transient loss of pulp sensitivity following traumatic dental injuries is common, and the pulp may fully recover over time. Bhaskar and Rappaport found vital tissue in traumatized teeth nonresponsive to traditional pulp sensitivity testing. They advised a delay in the diagnosis of necrosis when relying on these methods alone because of proposed transient sensory deficiencies. Future pulp sensitivity testing methodologies will likely focus on true measures of vitality, including representation of blood flow. Mesaros et al proposed the use of laser Doppler flowmetry, whereas Schnettler and Wallace suggested

pulse oximetry as a means of true pulp vitality testing. Levin discussed the potential utility of dual wavelength spectrophotometry as well as thermography. None of these methodologies have been adapted for clinical use at the time of publication. A full summary of sensitivity and vitality tests is presented in Fig 6-3.

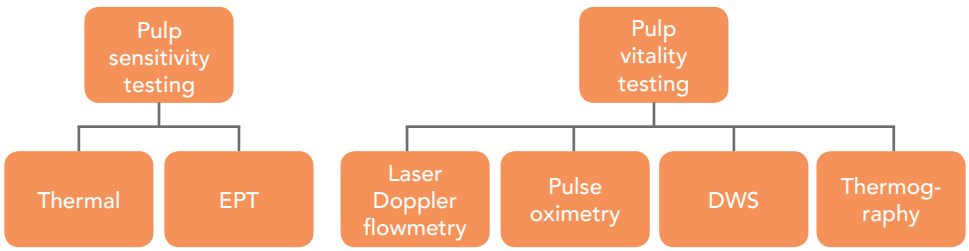

Fig 6-3 Pulp sensitivity and vitality testing. DWS, dual wavelength spectrophotometry.

Assessment of the periodontal ligament (PDL) occurs by proxy via measurement of percussion, palpation, and biting because direct examination is not possible in a clinical setting. Owatz et al equated percussion tenderness to mechanical allodynia, a painful response to a normally nonnoxious stimulant. Percussion tenderness may result from endodontic disease beyond the apex, periodontal disease, occlusal disease, or secondary to physical trauma (Seltzer et al 1965). Palpation tenderness evaluates soft tissue swelling, bony expansion, or subjective sensitivity indicative of endodontic disease or other alveolar damage. Biting pain can be assessed using a plastic bite stick [ie, Tooth Slooth, Professional Results], cotton roll, or other device capable of isolating pain to a particular cusp or tooth. Seltzer et al (1965) reported that biting pain can indicate extension of endodontic disease beyond the apex or tooth fracture.

A limited periodontal examination should be performed as part of any diagnostic workup for endodontic pathology. Periodontal probing depths should be measured around the teeth in question because deep pockets can be associated with localized periodontal disease or gingivitis, periodontal-endodontic lesions, or root fractures. Furcation involvement should be recorded and classified (Glickman and Carranza) (Figs 6-4 and 6-5). Mobility should be measured and classified (Glickman and Carranza) (Fig 6-6).

Class 1	Just noticeable
Class 2	Not quite through and through
Class 3	Through and through

Fig 6-4 Glickman's furcation classification.

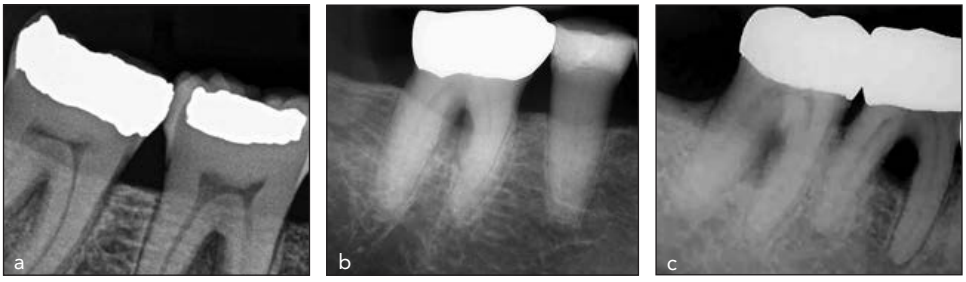

Fig 6-5 *(a to c)* Radiographic examples of furcation involvement. (Courtesy of Dr Stephen Fucini, Hanover, New Hampshire.)

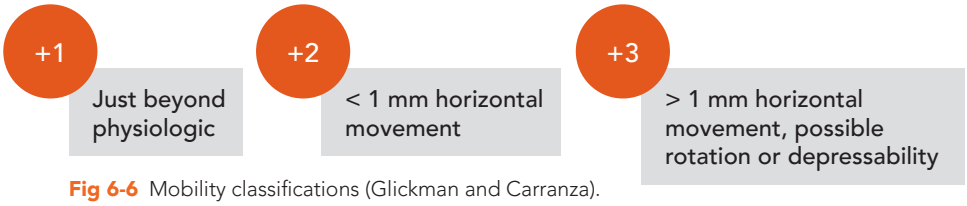

Fig 6-6 Mobility classifications (Glickman and Carranza).

Additional diagnostic testing may be useful in case-specific circumstances. With suspected fractures, Friedman and Marcus recommended transillumination with a fiber-optic light source to illuminate coronal fracture elements. As an alternative, Berman and Hartwell suggested staining of fractures with methylene blue dye for visualization. When pulp sensitivity testing is inconclusive or referred pain is suspected, Berman and Hartwell also recommended selective anesthesia testing to isolate the pain source. For the sake of efficiency, this technique should begin with PDL anesthesia in the maxilla with extension to broader techniques and the mandible if needed.

Radiographic Examination

For successful completion of the American Board of Endodontics (ABE) examination, basic understanding of oral radiology is essential. One should be familiar with radiation physics and techniques to improve images, including adjustments to milliamperes to alter radiation density, and peak voltage to adjust contrast. For a complete radiology reference, please refer to White and Pharaoh's *Oral Radiology: Principles and Interpretation*.

Although they provide essential information for diagnosis, radiographs are not comprehensive in their diagnostic scope. Radiographs must be taken together with the clinical examination to provide a definitive diagnosis. Evaluation and description of radiographic findings should follow a systematic approach. White and Pharoah suggest the following categorical approach to radiographic description (Fig 6-7): localization, periphery and shape, internal structure, and the effects on surrounding structures.

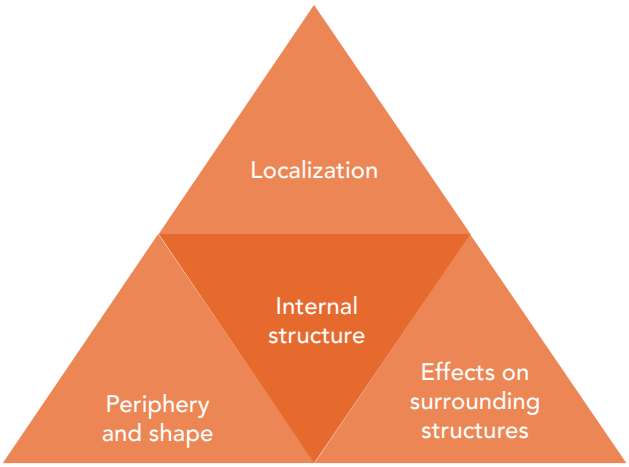

Fig 6-7 A systematic approach for description of radiographic findings (White and Pharoah).

Radiographic changes should be characterized as localized or generalized, and the position of the defect within the jaw should be noted. The borders of the lesion should be described as well or ill defined. Lesions may present with a regular or irregular shape and may have scalloped borders. The internal structure should be described as radiolucent, radiopaque or a mixed lesion. The effects of the radiographic changes on neighboring teeth, lamina dura, and the PDL should be described, including displacement, widening, or resorption. This is particularly important in lesions of non-endodontic origin. Similarly, the presence of corticated or sclerotic bone adjacent to lesions or periosteal reactions should be noted. These findings are associated with reactive processes and are often indicative of slow-growing lesions. Lastly, any effect on the inferior alveolar nerve canal or mental foramen should be noted, including displacement or erosion of border architecture.

Periapical films are the most commonly examined radiographs for endodontic diagnosis. Bitewing radiographs are additionally useful to evaluate caries, existing restorations, periodontal involvement and previously initiated endodontic therapy. In addition, Robinson et al found that bitewing radiographs provide an accurate representation of the location and size of the pulp chamber and can aid in the design of the endodontic access preparation. Panoramic radiographs are often recommended to assess trauma cases, particularly to rule out the presence of alveolar fractures. CBCT is increasingly useful in all facets of endodontic diagnosis and treatment and is covered in greater detail later in this chapter. Radiographic modalities are compared in Fig 6-8.

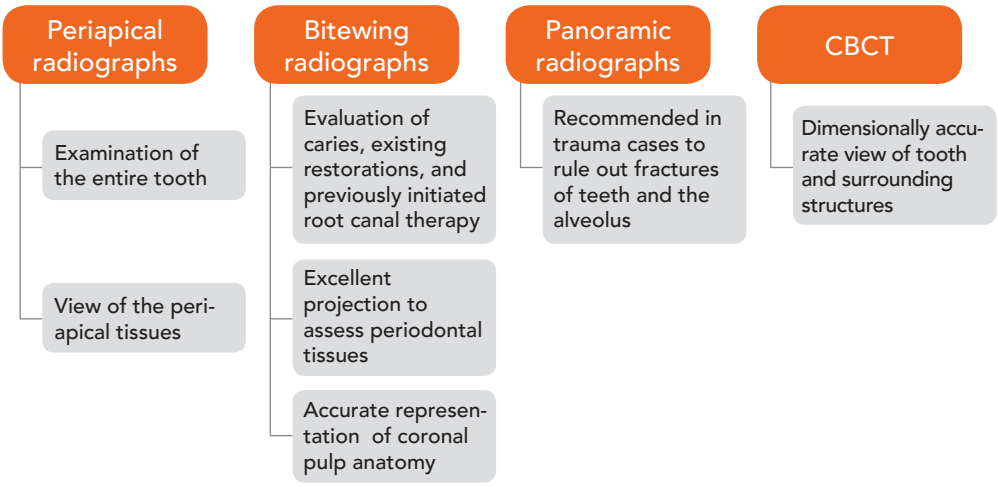

Fig 6-8 Common uses for each radiographic modality in endodontic diagnosis.

Digital radiography has long been accepted for use in endodontics. A study by Soh et al in 1993 showed that digital radiographs reduced radiation doses to patients because they require 22% less radiation exposure than traditional radiographic films. According to Barbat and Messer, digital radiographs offer no improvement in diagnostic yield over traditional radiographs. However, the reduction in radiation is a clear benefit. Professional societies and the United States Food and Drug Administration advise following the ALARA, or "as low as reasonably achievable," principle in considering any radiation exposure.

Though radiographs are useful diagnostic aids in the determination of a definitive diagnosis, they do not provide an accurate representation of pulpal or periapical histology. Brynolf (1967) demonstrated that histologic inflammation can be present in the absence of radiographic changes. Similarly, Lalonde found that cysts and granulomas could not be differentiated based on radiographic features alone. The defnitive diagnosis of a periapical lesion requires histologic evaluation (Peters and Lau).

Two-dimensional dental radiography

Two-dimensional (2D) dental radiography generally refers to periapical, bitewing, and panoramic radiographs. Though useful, these images are subject to structural overlap, limited spatial determination, and geometric distortion. Bender and Seltzer introduced the concept of anatomical noise to endodontics in their study showing that periapical lesions confined to cancellous bone cannot be seen because of the projection of overlying anatomy. To compensate, they recommend taking multiple angles of periapical radiographs based on Brynolf's (1970) classic study for improved diagnostics. She found that diagnostic accuracy increased from 74% to 90% when using one versus three radiographs taken from multiple angles.

In addition to issues with structural overlap, 2D radiographs are limited in their ability to determine the location of radiographic changes in space. Examination of multiple radiographic angles, though, can provide spatial information. Gutmann and Endo reviewed the application of Clark's rule, also known as the *buccal object rule*, to provide spatial information. This rule asserts that objects closer to the film move less with angled radiographs and thus structures located farther lingually/palatally move less with tube head angulation changes.

Lastly, a certain degree of geometric distortion is expected with 2D radiography, the extent of which can vary depending on technique. Vande Voorde and Bjorndahl found at least 5% magnification due to divergence between the x-ray beam and the distance between the object and receptor.

As a result of structural overlap, limited spatial determination, and geometric distortion, 2D radiography is subject to several limitations (Fig 6-9). Rud and Ommell reported that fracture lines could only be visualized if the x-ray beam was within four degrees of the fractured plane. Additionally, all radiographs are subject to variations in reader interpretation. Goldman et al found only 46% interexaminer reliability or agreement between two different examiners. Furthermore, only marginal improvement was reported with repeated reading by the same examiner, with only 70% intra-examiner reliability reported. Some of these limitations may be overcome by CBCT imaging.

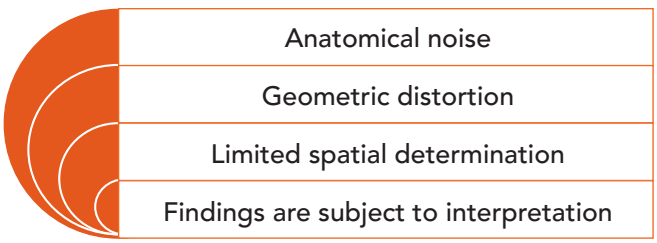

Fig 6-9 Commonly reported issues with 2D radiography. Several of these issues are improved with three-dimensional radiographic techniques, namely CBCT.

Cone beam computed tomography

CBCT has become more prevalent in endodontic diagnosis and treatment in recent years and may eventually become the standard of care in certain cases. Even if an endodontist does not have a CBCT machine on-site, the technology is available in most communities on a referral basis. Practitioners must familiarize themselves with CBCT because its importance is certain to increase in the future.

Unlike traditional CT scans that use a fan-shaped x-ray beam, CBCT images are obtained by a cone-shaped beam. CBCTs capture a large volume of data with a single rotation around the patient, whereas traditional scans require several rotations. Computer algorithms reconstruct high-resolution images, and the data are compiled into a volume for viewing. CBCT images are considered superior to traditional CT scans for dental purposes because of the reduced time needed for imaging, the ability to take the image with the

patient seated upright, the reduced radiation, and the fact that scans are dimensionally accurate as a result of cubic voxels (Low et al). MicroCT, cited as the gold standard in endodontic research imaging by Rhodes et al, uses similar technology to CBCT; however, these machines are not large enough to image human subjects.

CBCT imaging holds several advantages over 2D radiography. CBCT imaging essentially eliminates anatomical noise and geometric distortion, and obviates Brynolf's (1970) recommendations for multiple radiographic angles. Furthermore, CBCT imaging is more sensitive in terms of its ability to detect apical pathology. In a study essentially replicating Bender and Seltzer's work from 1961, Patel et al (2009) showed that CBCT images could detect lesions confined to cancellous bone. Lofthag-Hansen et al reported identification of significantly more lesions by CBCT than 2D radiographs. This holds true even in the maxilla, where lesions are often obscured by the maxillary sinus and zygomatic process. Low et al found that, when compared with CBCT, traditional radiographs missed 34% of lesions in the posterior maxilla.

The major disadvantage associated with CBCT imaging is increased radiation dosages. In a recent meta-analysis, Ludlow et al reported adult effective dosages of 46 to 1,073 μSv for large field of view (FOV) machines that visualize the entire maxilla or mandible. For limited FOV machines that visualize only a few teeth at a time, Ludlow et al reported effective dosages of 5 to 652 μSv. For comparison, one digital periapical film exposes a patient to 2 to 8 μSv (Ludlow and Ivanovic), and a digital panoramic radiograph exposes 14 to 24 μSv (Pauwels et al). Table 6-2 summarizes effective dosages for various types of radiographic images. It is clear that huge variations in radiation exposure can occur depending on the particular machine used as well as patient factors; however, a clear trend of increasing amounts of radiation arises as one moves beyond 2D radiography.

Table 6-2 Radiation dosages

	Effective dose (μSv)
Periapical radiograph (digital)	2–8
Panoramic radiograph (digital)	14–24
Limited FOV CBCT	5–652
Large FOV CBCT	46–1,073

An additional disadvantage associated with CBCT machines is their substantial cost, which must in turn be passed along to the patient. Furthermore, scans take 20 to 40 seconds, which subjects the image to movement-based artifacts. Consequently, CBCT imaging is not ideal for patients that cannot hold still, including some pediatric patients or patients with a tremor.

Though CBCT images offer improvements over 2D radiographs in many respects, some issues with image quality have been reported. CBCT images are known to have poor contrast resolution. Scarfe et al reported that bone is difficult to differentiate from dentin. CBCT images traditionally have limited spatial resolution. According to Tsai et al, lesions must be greater than 1.4 mm in diameter for adequate detection. On the other hand, Domark et al showed that second mesiobuccal canals in maxillary molars, oftentimes quite

small, could be identified equally as well by CBCT as by the gold standard micro-CT. Lastly, Scarfe et al described artifacts, including beam hardening secondary to metallic restorations, that can render images nondiagnostic (Fig 6-10).

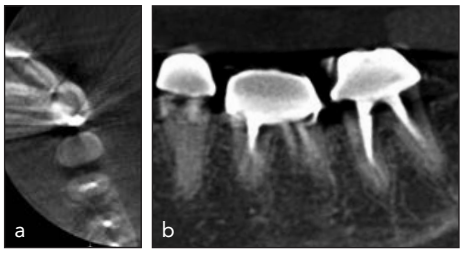

Fig 6-10 *(a and b)* Examples of the beam-hardening artifact encountered adjacent to metallic restorations.

An additional potential disadvantage of CBCT imaging is the liability risk of nondiagnosis imparted by the imaging of nondental structures. Incidental, particularly extragnathic findings are common, including sinus, temporomandibular joint, vertebral, or brain lesions, as reviewed by Edwards et al. Although the use of focused-field images reduces the area of responsibility, the prescribing clinician is responsible for recognition of potential anomalies and referral for further diagnosis and treatment when appropriate. Secondary "over-reads" by licensed oral radiologists minimize liability exposure of the prescribing clinician. Advantages and disadvantages of CBCT imaging are summarized in Fig 6-11.

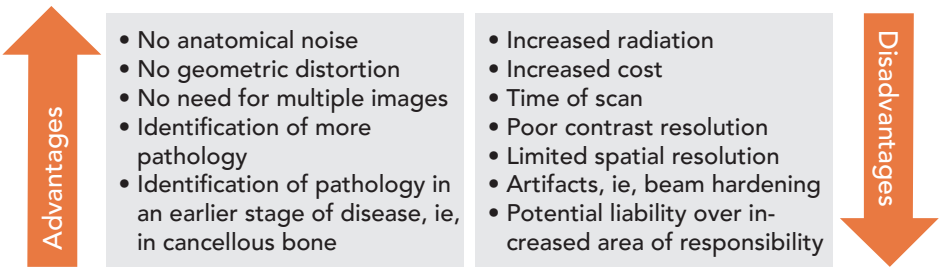

Fig 6-11 The advantages and disadvantages of CBCT imaging compared with 2D dental imaging techniques.

The American Association of Endodontists (AAE) and the American Academy of Oral and Maxillofacial Radiology (AAOMR) released a joint position statement summarizing recommended indications for CBCT imaging (Fig 6-12). Following the principle of ALARA, radiation dose should be kept as low as reasonably achievable (Farman). CBCT imaging is not appropriate for every patient, and it is important to consider the need for CBCT imaging on a case-by-case basis. The suggested indications for CBCT are as follows: to aid in difficult diagnoses, as a treatment aid, in the identification of complications, and assessing cases of trauma, resorption, planned dental implants, and planned surgery. The literature abounds with research supporting these indications. Interestingly, research indicates that CBCT images may alter clinicians' treatment recommendations. Ee et al reported that when clinicians evaluated cases, first using traditional periapical radiographs and 2 weeks later by CBCT imaging, their diagnoses and treatment plans changed 62% of the time.

CBCT imaging can be especially helpful to diagnose pathology associated with previously treated teeth. Liang et al found CBCT imaging useful to assess the quality of a root canal fill. Patel et al (2009) discussed the use of CBCT imaging in the diagnosis and treatment of resorptive defects. Ball et al and the 2013 AAE Guidelines for the Treatment of Traumatic Dental Injuries discussed the benefits and potential indications for CBCT imaging in the diagnosis and management of these injuries. Von Arx et al recommended CBCT imaging in treatment planning surgical cases, particularly in the identification of the mandibular nerve.

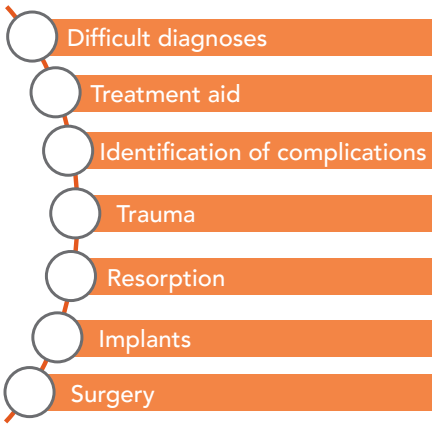

Fig 6-12 The seven indications for CBCT imaging in endodontics recommended by the AAE and the AAOMR.

Neves et al and Brady et al reported that CBCT imaging can, in a limited capacity, detect root fractures. Brady et al noted increased diagnostic potential with fractures displaying more than 50 µm of separation. This detection appears to be accurate, as Edlund et al found that CBCT correctly identified fractures in 84% of cases. However, CBCT imaging may not be as useful in the presence of a metal post. Costa et al found that accuracy of fracture detection decreased by 20% because of the influence of post-retained restorations. Though CBCTs may not be able to directly visualize fractures, Fayad et al described several findings often associated with fractures, including loss of bone midroot with intact bone coronal and apical to the defect, absence of the entire buccal plate, a midroot radiolucency in the area of termination of the post, and space between the buccal or lingual plate and the root surface.

CBCT imaging is often useful to aid in the diagnosis and treatment of intraoperative complications. Shemesh et al discussed identification of perforations by CBCT imaging. Ball et al presented a case series on the use of CBCT imaging to manage intraoperative complications, particularly in the management of complex anatomy, dystrophic calcifications, root resorption, perforations, and root fractures. Lastly, CBCT images may also be useful as a treatment aid. Patel presented a case using CBCT imaging for conservative management of a dens invaginatus in which mapping of the pulp spaces allowed for conservation of significant amounts of tooth structure. Lenzi and Trope suggested the use of CBCT imaging in regenerative endodontic therapy. Jeger et al discussed the accuracy of CBCT imaging in working length determination.

CBCT images are more sensitive than traditional radiographs in terms of their ability to detect apical pathology. Consequently, future prognostic studies using this imaging modality are likely to present a more accurate, though less optimistic, view of outcomes of endodontic treatment. Several studies illustrating this point are discussed in chapter 11.

Image interpretation

Assessment of periapical radiographs requires familiarity with both normal dental anatomy and surrounding anatomical structures. In the mandible, these structures include the mental foramen and the inferior alveolar nerve canal. Moiseiwitsch reported that the mental foramen was most often located between the mandibular first and second premolars, with vertical localization averaging 16 mm from the cementoenamel junction of the nearest tooth. However,

great variation was found in both horizontal and vertical placement of the mental foramen. Kim et al found that the inferior alveolar nerve approached the root apices as it moved posteriorly. On average, the nerve structure was 4.7 mm from the second premolar apex and 3.7 mm from the second molar apex. Radiographic examples of the mental foramen and mandibular canal are presented in Figs 6-13 and 6-14, respectively.

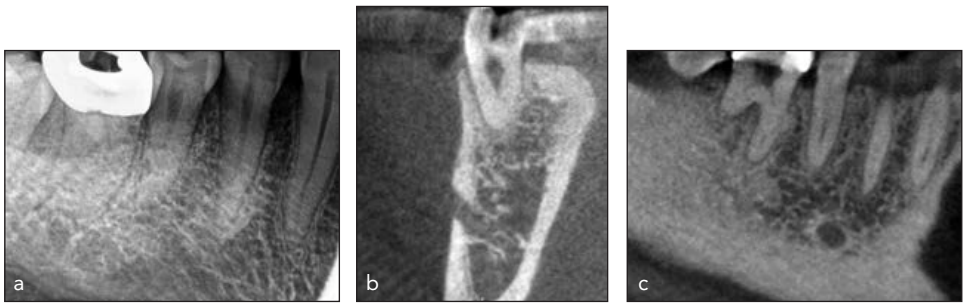

Fig 6-13 Radiographic depictions of mental foramen between mandibular first and second premolars: *(a)* periapical radiograph, *(b)* coronal section of CBCT, and *(c)* sagittal section of CBCT.

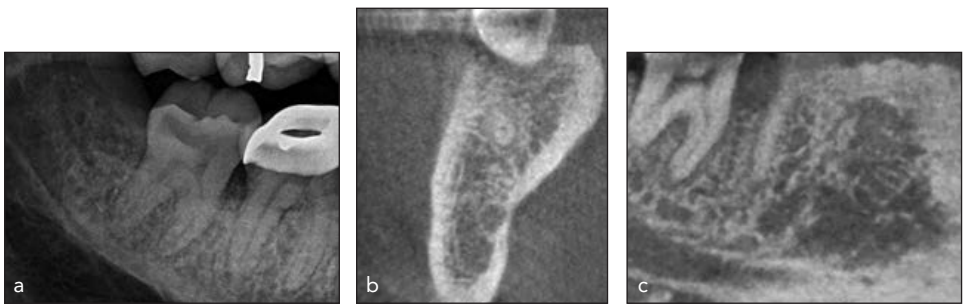

Fig 6-14 Radiographic depictions of inferior alveolar nerve canal adjacent to mandibular molars: *(a)* periapical radiograph, *(b)* coronal section of CBCT, and *(c)* sagittal section of CBCT.

In the maxilla, nondental anatomy is often visualized including the maxillary sinus walls and zygomatic process. It is important to note the potential proximity and involvement of the maxillary sinus with any endodontic disease. Pagin et al found that 14% of maxillary molar roots perforate the sinus. Shanbhag et al found that maxillary sinus mucosal thickening was nine times more likely adjacent to maxillary molars with apical periodontitis. CBCT imaging is especially useful in this region (Fig 6-15).

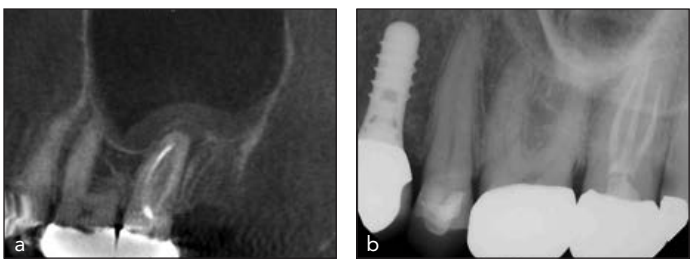

Fig 6-15 CBCT showing maxillary sinus mucositis *(a)*, not seen on a periapical radiograph *(b)*.

Pulpal and Periapical Diagnostic Terminology

The results of the clinical and radiographic examinations lead clinicians to the definitive diagnosis. The ABE and AAE have agreed on standardized diagnostic terminology, as defined in the AAE glossary. These terms include a list of standardized pulpal and periapical diagnoses (Figs 6-16 and 6-17, respectively). Board candidates should familiarize themselves with this list because terminology has changed in recent years.

Pulpal diagnoses

- Normal pulp
- Reversible pulpitis
- Symptomatic irreversible pulpitis
- Asymptomatic irreversible pulpitis
- Pulp necrosis
- Previously treated
- Previously initiated therapy

Fig 6-16 Diagnostic terms for pulpal diagnoses (AAE glossary).

Periapical diagnoses

- Normal apical tissue
- Symptomatic apical periodontitis
- Asymptomatic apical periodontitis
- Chronic apical abscess
- Acute apical abscess
- Condensing osteitis

Fig 6-17 Diagnostic terms for periapical diagnoses (AAE glossary).

Pulpal diagnoses

- **Normal pulp:** pulp is symptom-free and normally responsive to pulp sensitivity testing.
- **Reversible pulpitis:** subjective and objective findings indicate that inflammation should resolve and the pulp should return to normal.
- **Symptomatic irreversible pulpitis:** subjective and objective findings indicate that the vital inflamed pulp is incapable of healing; symptoms include lingering thermal pain, spontaneous pain, or referred pain.
- **Asymptomatic irreversible pulpitis:** subjective and objective findings indicate that the vital inflamed pulp is incapable of healing; no clinical symptoms but inflammation is present and attributed to caries, caries excavation, or trauma.
- **Pulp necrosis:** indicates death of the dental pulp; pulp is usually nonresponsive to pulp sensitivity testing.
- **Previously treated:** tooth has been endodontically treated, and the canals are obturated with various filling materials other than intracanal medicaments.
- **Previously initiated therapy:** tooth has been previously treated by partial endodontic therapy, such as pulpotomy or pulpectomy.

Periapical diagnoses

- **Normal apical tissue:** teeth not sensitive to percussion or palpation testing; the lamina dura surrounding the root is intact, and the PDL space is uniform.
- **Symptomatic apical periodontitis:** inflammation usually of the apical periodontium producing clinical symptoms, including a painful response to biting and/or percussion or palpation; it may or may not be associated with an apical radiolucent area.

- **Asymptomatic apical periodontitis:** inflammation and destruction of the apical periodontium that is of pulpal origin, appears as an apical radiolucent area, and does not produce clinical symptoms.
- **Chronic apical abscess:** an inflammatory reaction to pulpal infection and necrosis characterized by gradual onset, little or no discomfort, and the intermittent discharge of pus through an associated sinus tract.
- **Acute apical abscess:** an inflammatory reaction to pulpal infection and necrosis characterized by rapid onset, spontaneous pain, tenderness of the tooth to pressure, pus formation, and swelling of associated tissues.
- **Condensing osteitis:** diffuse radiopaque lesion representing a localized bony reaction to a low-grade inflammatory stimulus, usually seen at the apex of the tooth.

Adjunctive Diagnostic Terminology

In addition to the pulpal and periapical diagnosis, there are several other diagnostic categories often associated with endodontically involved teeth. These include fractures, periodontal-endodontic lesions, pulp polyps, resorption, and traumatic dental injuries.

Fractures

Fractures can coexist with endodontic pathology or present independently. Furthermore, they may or may not cause symptoms. The AAE divided fractures into the following categories: craze lines, fractured cusp, cracked tooth, split tooth, and vertical root fracture (Fig 6-18).

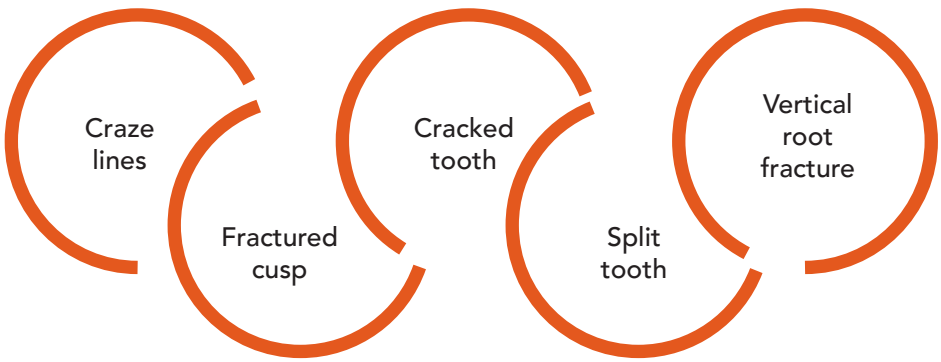

Fig 6-18 Commonly discussed fracture terminology (AAE).

Craze lines affect only enamel and are often nonpathogenic. Fractured cusps, cracked teeth, and split teeth involve enamel and dentin and can extend to involve the pulp. The AAE favored the term *cracked tooth* versus the older term *cracked tooth syndrome*, described by Cameron. Vertical root fractures begin in the root. According to Cameron, fractures most often involve the mandibular second molar, followed by maxillary premolars and maxillary molars.

The diagnosis of fracture type is aided by bite tests described earlier, transillumination, or methylene blue dyes. Radiographs are generally not useful to identify fractures because, according to Rud and Ommell, the x-ray beam must be within four degrees of the fracture itself for visualization, and fractures must have adequate separation to be discerned. CBCT imaging may prove more useful but again has limitations based on the separation of fracture line (Brady et al).

Vertical root fractures often have a characteristic presentation, described by Tsesis et al. Radiographically, these fractures present with a J-shaped or halo radiolucency extending apically from the marginal periodontium. Clinically, they often present with a deep periodontal probing depth that is narrow and localized as well as multiple sinus tracts often within close proximity to the gingival margin. Most often, vertical root fractures are noted in teeth treated previously with root canal therapy and posts (Tsesis et al).

Brady et al suggested the use of CBCT imaging to aid in the diagnosis of vertical root fractures, but these images do not always visualize the fractures unless the size of the fracture is larger than the voxel size. CBCT images are most useful in the visualization of associated patterns of bone loss. According to a recent study by Neves et al, metal posts and gutta-percha present in the root can result in beam-hardening artifacts that can negatively influence the diagnostic potential of CBCT imaging. Kajan and Taromsari suggested that beam-hardening artifacts themselves might even be misdiagnosed as fractures because of their similar appearance. Overall, dental radiography, including traditional periapical radiographs and CBCT imaging, has higher specificity than sensitivity for the detection of vertical root fractures; according to Chavda et al, they are better at ruling out fractures than determining their presence. Radiographic examples of vertical root fractures visualized by traditional and CBCT imaging are shown in Figs 6-19 and 6-20.

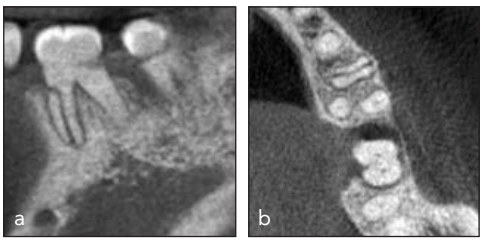

Fig 6-19 *(a and b)* Vertical root fracture diagnosed incidentally on the mandibular right first molar during consultation regarding the adjacent second molar.

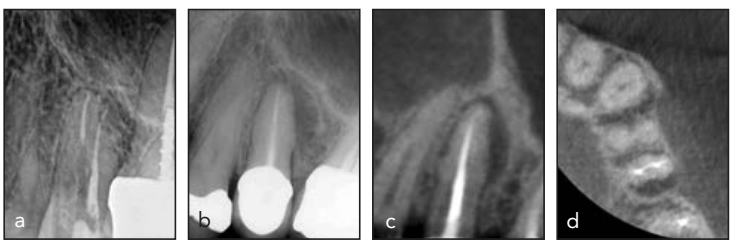

Fig 6-20 Initial root canal therapy on the maxillary left second premolar *(a)*, with failure due to vertical root fracture noted 5 years later by periapical radiograph *(b)*, sagittal section on CBCT *(c)*, and axial section on CBCT *(d)*.

While teeth with vertical root fractures or coronal fractures extending into the root structure require extraction, the other fracture categories are often treatable. In the case of shallow fractures without pulpal involvement, cuspal coverage may provide sufficient treatment. Pulpal involvement, whether determined by direct visualization of the fracture or symptoms of irreversible pulpitis, necessitates endodontic therapy (AAE). A summary of fracture types and the recommendations for treatment are summarized in Fig 6-21.

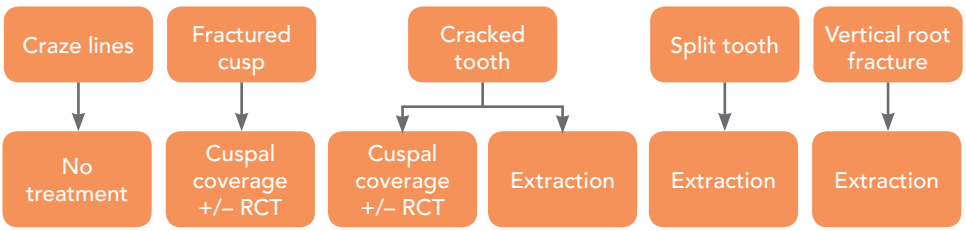

Fig 6-21 Types of fracture and associated treatment recommendations (AAE). RCT, root canal treatment.

Periodontal-endodontic lesions

Endodontic lesions that create an area of drainage through the periodontal pocket are often dubbed "perio-endo" lesions, though in fact these are a version of a chronic apical abscess where the apical infection has established a pathway of drainage. Harrington et al described these lesions generally as a sinus tract through the PDL space that originates from a periapical or lateral lesion and is detected as a narrow, deep probing depth. Since these lesions arise from an endodontic source, they can be treated with endodontic therapy alone. Simon et al described these lesions as "primary endo, secondary perio."

These primary endodontic lesions require a necrotic pulp to form. Harrington et al suggested pathways of narrow, sinus tract–type probing depths that might mimic a periodontal-endodontic lesion, despite the presence of a vital pulp. The drainage could originate from a neighboring endodontically involved tooth or localized periodontal defects because of developmental grooves, fused roots, incomplete coronal fractures, crown-root fractures, or enamel spurs.

Theoretically, extensive periodontal involvement could precipitate development of endodontic disease secondary to the periodontal disease, sometimes referred to as "primary perio, secondary endo" (Simon et al). Langeland et al, however, surmised that periodontal pathology is unlikely to cause endodontic involvement unless the periodontal lesion reaches the apex.

Controversy exists over the existence of a true combined endodontic and periodontal infection. Harrington et al suggested that, although these true combined lesions are rare, they do likely exist when independent endodontic and periodontal infections extend enough to coalesce. Although spontaneous vertical root fractures or perforations may present in a way that mimics these lesions, they should not be considered periodontal-endodontic lesions.

Although periodontal disease can lead to unrelated failures of endodontically treated teeth, Harrington et al found no reason to support reduced rates of success of periodontal procedures in teeth previously treated by root canal. A summary of potential periodontal-endodontic relationships is pictured in Fig 6-22.

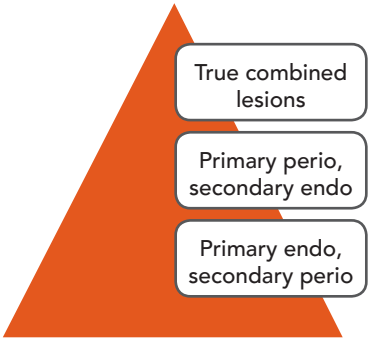

Fig 6-22 The possible periodontal-endodontic relationships (Simon et al).

Pulp polyps

Also known as *hyperplastic pulpitis*, the AAE glossary defines a *pulp polyp* as a form of chronic pulpal inflammation usually following carious or traumatic exposure in a young patient. The pulp polyp is characterized by a proliferation of dental pulp tissue from the exposed pulp chamber that fills the cavity with a pedunculated or sessile, pinkish-red, fleshy mass, usually covered with epithelium.

Resorption

Resorption describes a heterogenous diagnostic category including internal root resorption, external inflammatory root resorption, invasive cervical root resorption, replacement resorption, and pressure resorption. Each has some degree of pulpal involvement or potential pulpal involvement, but all differ in etiology, pathogenesis, and clinical presentation. A more detailed discussion of resorption can be found in chapter 10.

Traumatic dental injuries

A full description of traumatic dental injuries, including crown fractures, root fractures, alveolar fractures, luxation injuries, and avulsions, is found in chapter 9.

Bibliography

Clinical Examination

Alomari FA, Al-Habahbeh R, Alsakarna BK. Responses of pulp sensibility tests during orthodontic treatment and retention. Int Endod J 2011;44:635–643.

Bender IB. Pulpal pain diagnosis—A review. J Endod 2000;26:175–179.

Bender IB, Landau MA, Fonsecca S, Trowbridge HO. The optimum placement-site of the electrode in electric pulp testing of the 12 anterior teeth. J Am Dent Assoc 1989;118:305–310.

Berman LH, Hartwell GR. Diagnosis. In: Hargreaves KM, Cohen S, Berman LH (eds). Cohen's Pathways of the Pulp. St Louis: Mosby, 2011:2–39.

Bhaskar SN, Rappaport HM. Dental vitality tests and pulp status. J Am Dent Assoc 1973;86:409–411.

Bierma MM, McClanahan S, Baisden MK, Bowles WR. Comparison of heat-testing methodology. J Endod 2012;38:1106–1109.

Fowler S, Fullmer S, Drum M, Reader A. Does acetaminophen/hydrocodone affect cold pulpal testing in patients with symptomatic irreversible pulpitis? A prospective, randomized, double-blind, placebo-controlled study. J Endod 2014;40:1958–1960.

Friedman J, Marcus MI. Transillumination of the oral cavity with use of fiber optics. J Am Dent Assoc 1970;80:801–809.

Fulling HJ, Andreasen JO. Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing. Scand J Dent Res 1976;84:286–290.

Glickman I, Carranza FA. Glickman's Clinical Periodontology, ed 7. Philadelphia: Saunders, 1990.

Jespersen JJ, Hellstein J, Williamson A, Johnson WT, Qian F. Evaluation of dental pulp sensibility tests in a clinical setting. J Endod 2014;40:351–354.

Keir DM, Walker WA 3rd, Schindler WG, Dazey SE. Thermally induced pulpalgia in endodontically treated teeth. J Endod 1991;17:38–40.

Ketterl W. Age-induced changes in the teeth and their attachment apparatus. Int Dent J 1983;33:262–271.

Levin LG. Pulp and periradicular testing. Pediatr Dent 2013;35:113–119.

Mesaros S, Trope M, Maixner W, Burkes EJ. Comparison of two laser Doppler systems on the measurement of blood flow of premolar teeth under different pulpal conditions. Int Endod J 1997;30:167–174.

Miller SO, Johnson JD, Allemang JD, Strother JM. Cold testing through full-coverage restorations. J Endod 2004;30:695–700.

Owatz CB, Khan AA, Schindler WG, Schwartz SA, Keiser K, Hargreaves KM. The incidence of mechanical allodynia in patients with irreversible pulpitis. J Endod 2007;33:552–556.

Petersson K, Soderstrom C, Kiani-Anaraki M, Levy G. Evaluation of the ability of thermal and electrical tests to register pulp vitality. Endod Dent Traumatol 1999;15:127–131.

Read JK, McClanahan SB, Khan AA, Lunos S, Bowles WR. Effect of ibuprofen on masking endodontic diagnosis. J Endod 2014;40:1058–1062.

Rickoff B, Trowbridge H, Baker J, Fuss Z, Bender IB. Effects of thermal vitality tests on human dental pulp. J Endod 1988;14:482–485.

Ricucci D, Loghin S, Siqueira JF Jr. Correlation between clinical and histologic pulp diagnoses. J Endod 2014;40:1932–1939.

Schnettler JM, Wallace JA. Pulse oximetry as a diagnostic tool of pulpal vitality. J Endod 1991;17:488–490.

Seltzer S, Bender IB, Nazimov H. Differential diagnosis of pulp conditions. Oral Surg Oral Med Oral Pathol 1965;19:383–391.

Seltzer S, Bender IB, Ziontz M. The dynamics of pulp inflammation: Correlations between diagnostic data and actual histologic findings in the pulp. Oral Surg Oral Med Oral Pathol 1963;16:969–977.

Trowbridge HO, Franks M, Korostoff E, Emling R. Sensory response to thermal stimulation in human teeth. J Endod 1980;6:405–412.

White J, Cooley R. A quantitative evaluation of thermal pulp testing. J Endod 1977;3:453–457.

Wilson BL, Broberg C, Baumgartner JC, Harris C, Kron J. Safety of electronic apex locators and pulp testers in patients with implanted cardiac pacemakers or cardioverter/defibrillators. J Endod 2006;32:847–852.

Radiographic Examination

American Association of Endodontists; American Academy of Oral and Maxillofacial Radiology. Use of cone-beam computed tomography in endodontics. Joint Position Statement of the American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;111:234–237.

American Association of Endodontists. The Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. Revised 2013. www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 21 January 2016.

Ball RL, Barbizam JV, Cohenca N. Intraoperative endodontic applications of cone-beam computed tomography. J Endod 2013;39:548–557.

Barbat J, Messer HH. Detectability of artificial periapical lesions using direct digital and conventional radiography. J Endod 1998;24:837–842.

Bender IB, Seltzer S. Roentgenographic and direct observation of experimental lesions in bone: II. 1961. J Am Dent Assoc 1961;62:708–716.

Brady E, Mannocci F, Brown J, Wilson R, Patel S. A comparison of cone beam computed tomography and periapical radiography for the detection of vertical root fractures in nonendodontically treated teeth. Int Endod J 2014;47:735–746.

Brynolf I. A histological and roentgenological study of the periapical region of upper incisors. Odont Revy 1967;18:lZ97.

Brynolf I. Roentgenologic periapical diagnosis. II. One, two or more roentgenograms? Sven Tandlak Tidskr 1970;63:345–350.

Costa FF, Gaia BF, Umetsubo OS, Cavalcanti MG. Detection of horizontal root fracture with small-volume cone-beam computed tomography in the presence and absence of intracanal metallic post. J Endod 2011;37:1456–1459.

Domark JD, Hatton JF, Benison RP, Hildebolt CF. An ex vivo comparison of digital radiography and cone-beam and micro computed tomography in the detection of the number of canals in the mesiobuccal roots of maxillary molars. J Endod 2013;39:901–905.

Edlund M, Nair MK, Nair UP. Detection of vertical root fractures by using cone-beam computed tomography: A clinical study. J Endod 2011;37:768–772.

Edwards R, Altalibi M, Flores-Mir C. The frequency and nature of incidental findings in cone-beam computed tomographic scans of the head and neck region: A systematic review. J Am Dent Assoc 2013;144:161–170.

Ee J, Fayad MI, Johnson BR. Comparison of endodontic diagnosis and treatment planning decisions using cone-beam volumetric tomography versus periapical radiography. J Endod 2014;40:910–916.

Farman AG. ALARA still applies. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100:395–397.

Fayad MI, Ashkenaz PJ, Johnson BR. Different representations of vertical root fractures detected by cone-beam volumetric tomography: A case series report. J Endod 2012;38:1435–1442.

Goldman M, Pearson AH, Darzenta N. Reliability of radiographic interpretations. Oral Surg Oral Med Oral Pathol 1974;38:287–293.

Gutmann JL, Endo C. Clark's rule vis a vis the buccal object rule: Its evolution & application in endodontics. J Hist Dent 2011;59:12–15.

Jeger FB, Janner SF, Bornstein MM, Lussi A. Endodontic working length measurement with preexisting cone-beam computed tomography scanning: A prospective, controlled clinical study. J Endod 2012;38:884–888.

Kim TS, Caruso JM, Christensen H, Torabinejad M. A comparison of cone-beam computed tomography and direct measurement in the examination of the mandibular canal and adjacent structures. J Endod 2010;36:1191–1194.

Lalonde ER. A new rationale for the management of periapical granulomas and cysts: An evaluation of histopathological and radiographic findings. J Am Dent Assoc 1970;80:1056–1059.

Lenzi R, Trope M. Revitalization procedures in two traumatized incisors with different biological outcomes. J Endod 2012;38:411–414.

Liang YH, Yuan M, Li G, Shemesh H, Wesselink PR, Wu MK. The ability of cone-beam computed tomography to detect simulated buccal and lingual recesses in root canals. Int Endod J 2012;45:724–729.

Lofthag-Hansen S, Huumonen S, Grondahl K, Grondahl HG. Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:114–119.

Low KM, Dula K, Burgin W, Von Arx T. Comparison of periapical radiography and limited cone-beam tomography in posterior maxillary teeth referred for apical surgery. J Endod 2008;34:557–562.

Ludlow JB, Ivanovic M. Comparative dosimetry of dental CBCT devices and 64-slice CT for oral and maxillofacial radiology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106:106–114.

Ludlow JB, Timothy R, Walker C, et al. Effective dose of dental CBCT—A meta analysis of published data and additional data for nine CBCT units. Dentomaxillofac Radiol 2015;44:20140197.

Moiseiwitsch JR. Position of the mental foramen in a North American, white population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:457–460.

Neves FS, Freitas DQ, Campos PS, Ekestubbe A, Lofthag-Hansen S. Evaluation of cone-beam computed tomography in the diagnosis of vertical root fractures: The influence of imaging modes and root canal materials. J Endod 2014;40:1530–1536.

Pagin O, Centurion BS, Rubira-Bullen IR, Alvares Capelozza AL. Maxillary sinus and posterior teeth: Accessing close relationship by cone-beam computed tomographic scanning in a Brazilian population. J Endod 2013;39:748–751.

Patel S. The use of cone beam computed tomography in the conservative management of dens invaginatus: A case report. Int Endod J 2010;43:707–713.

Patel S, Dawood A, Mannocci F, Wilson R, Pitt Ford T. Detection of periapical bone defects in human jaws using cone beam computed tomography and intraoral radiography. Int Endod J 2009;42:507–515.

Patel S, Dawood A, Wilson R, Horner K, Mannocci F. The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography—An in vivo investigation. Int Endod J 2009;42:831–838.

Pauwels R, Beinsberger J, Collaert B, et al. Effective dose range for dental cone beam computed tomography scanners. Eur J Radiol 2012;81:267–271.

Peters E, Lau M. Histopathologic examination to confirm diagnosis of periapical lesions: A review. J Can Dent Assoc 2003;69:598–600.

Rhodes JS, Ford TR, Lynch JA, Liepins PJ, Curtis RV. Micro-computed tomography: A new tool for experimental endodontology. Int Endod J 1999;32:165–170.

Robinson D, Goerig AC, Neaverth EJ. Endodontic access: An update, Part I. Compendium 1989;10:290–292, 294–296, 298.

Rud J, Ommell K. Root fracture due to corrosion. Scand J Dent Res 1970;78:397–403.

Scarfe WC, Levin MD, Gane D, Farman AG. Use of cone beam computed tomography in endodontics. Int J Dent 2009;2009:634567.

Shanbhag S, Karnik P, Shirke P, Shanbhag V. Association between periapical lesions and maxillary sinus mucosal thickening: A retrospective cone-beam computed tomographic study. J Endod 2013;39:853–857.

Shemesh H, Cristescu RC, Wesselink PR, Wu MK. The use of cone-beam computed tomography and digital periapical radiographs to diagnose root perforations. J Endod 2011;37:513–516.

Soh G, Loh FC, Chong YH. Radiation dosage of a dental imaging system. Quintessence Int 1993;24:189–191.

Tsai P, Torabinejad M, Rice D, Azevedo B. Accuracy of cone-beam computed tomography and periapical radiography in detecting small periapical lesions. J Endod 2012;38:965–970.

Vande Voorde HE, Bjorndahl AM. Estimating endodontic "working length" with paralleling radiographs. Oral Surg Oral Med Oral Pathol 1969;27:106–110.

Von Arx T, Hanni A, Sendi P, Buser D, Bornstein MM. Radiographic study of the mandibular retromolar canal: An anatomic structure with clinical importance. J Endod 2011;37:1630–1635.

White SC, Pharoah MJ. Oral Radiology: Principles and Interpretation, ed 7. St Louis: Mosby, 2013.

Pulpal and Periapical Diagnostic Terminology

American Association of Endodontists. Glossary of Endodontics Terms. dev.aae.org/glossary/. Accessed 20 January 2016.

Adjunctive Diagnostic Terminology

American Association of Endodontists. Endodontics: Colleauges for Excellence. Cracking the Cracked Tooth Code: Detection and Treatment of Various Longitudinal Tooth Fractures. Chicago: American Association of Endodontists, 2008.

Brady E, Mannocci F, Brown J, Wilson R, Patel S. A comparison of cone beam computed tomography and periapical radiography for the detection of vertical root fractures in nonendodontically treated teeth. Int Endod J 2014;47:735–746.

Cameron CE. Cracked-tooth syndrome. J Am Dent Assoc 1964;68:405–411.

Chavda R, Mannocci F, Andiappan M, Patel S. Comparing the in vivo diagnostic accuracy of digital periapical radiography with cone-beam computed tomography for the detection of vertical root fracture. J Endod 2014;40:1524–1529.

Harrington GW, Steiner DR, Ammons WF. The periodontal-endodontic controversy. Periodontol 2000 2002;30:123–130.

Kajan ZD, Taromsari M. Value of cone beam CT in detection of dental root fractures. Dentomaxillofac Radiol 2012;41:3–10.

Langeland K, Rodrigues H, Dowden W. Periodontal disease, bacteria, and pulpal histopathology. Oral Surg Oral Med Oral Pathol 1974;37:257–270.

Neves FS, Freitas DQ, Campos PS, Ekestubbe A, Lofthag-Hansen S. Evaluation of cone-beam computed tomography in the diagnosis of vertical root fractures: The influence of imaging modes and root canal materials. J Endod 2014;40:1530–1536.

Rud J, Ommell K. Root fracture due to corrosion. Scand J Dent Res 1970;78:397–403.

Simon JH, Glick DH, Frank AL. The relationship of endodontic-periodontic lesions. J Periodontol 1972;43:202–208.

Tsesis I, Rosen E, Tamse A, Taschieri S, Kfir A. Diagnosis of vertical root fractures in endodontically treated teeth based on clinical and radiographic indices: A systematic review. J Endod 2010;36:1455–1458.

7

Diagnosis of Non-Endodontic Disease Entities

Although pulpal and periapical disease are the common diagnostic entities to consider when dental pain, infection, or radiographic changes are encountered, an array of pathologic and nonpathologic conditions exist and should be included in a comprehensive differential diagnosis. This chapter discusses pain syndromes, non-endodontic infections, and unusual radiographic findings.

Pain

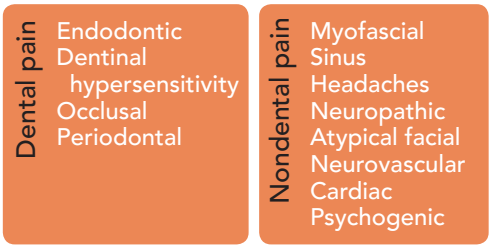

Fig 7-1 Sources of pain in the maxillofacial region.

Pain attributed to the teeth can arise from dental or nondental origins (Fig 7-1). Pain of dental origin may not necessarily arise from endodontic pathology. Entities of non-endodontic dental pain include dentinal hypersensitivity, occlusal pain, or periodontal pain. Brännström attributed dentinal hypersensitivity to hydrodynamic effects on the intratubular fluid of the dentinal tubules, oftentimes secondary to increased tubule patency or adjacent low-grade inflammation. Thermal stimuli may cause inward or outward fluid movement resulting in Aδ fiber stimulation and pain, which is usually transient in nature as long as pulpal inflammation is not progressive. This entity is often equivalent to reversible pulpitis, described in chapter 6. This dentinal hypersensitivity can proceed until the dentinal tubules are occluded either by normal physiologic processes or with interventions, including sensitivity toothpastes, fluoride varnishes, or bonded restorative agents. Brännström suggested that eccentric occlusal loads can also result in dentinal hypersensitivity.

Dental pain may also arise as a result of referred pain. Sessle et al described referred, or *heterotopic,* pain as that arising due to convergence of afferent neurons from different areas to the same projection neuron. In the oral cavity, these afferents arise from the trigeminal nerve. In this respect, referred pain can result in the perception of pain in a tooth other than the source of discomfort or pain from a nondental source presenting as dental pain.

Typical sources of nondental pain include muscular or myofascial pain, sinus-related pain, headaches, neuropathic pain, atypical facial pain, neurovascular pain, pain of cardiac origin, or psychogenic pain.

Myofascial pain

Fricton et al defined *myofascial pain* as that originating from small, tender trigger points within myofascial structures, often a distance from the area of perceived pain. Wright found that the masseter was the muscle that most often referred pain to teeth, and referral occurred most often to molars with a predilection for the mandible. Diagnosis as well as treatment of myofascial pain often involves injection of local anesthetic at these trigger points, though additional treatment aids include splint therapy, jaw exercises, massage, trigger point compression or myotherapy (wherein pressure is applied to the trigger point), acupuncture, relaxation therapy, stress management, biofeedback, and pharmacologic management with muscle relaxers, as described by Wright and Schiffman.

Sinus pain

Inflammation or infection of the maxillary sinuses can elicit pain in the adjacent maxillary molars as a result of close anatomical proximity. Mattscheck et al describe symptoms associated with sinus involvement as "fullness" or "pressure beneath the eyes." If the nasal

mucosa is involved, dull and aching pain may also be present. Other typical symptoms of maxillary sinusitis are generally present, including congestion and nasal drainage, and common etiologies are allergic, viral, or bacterial infections. Pain of sinus origin is not relieved by local anesthesia of the adjacent teeth. Chen et al presented a case of perceived dental pain secondary to maxillary sinusitis.

Headaches

Headaches comprise a broad diagnostic entity with a variety of presentations and predilections. The International Classification of Headache Disorders described *primary headaches* as those without other discernible causes, such as trauma, and offered diagnostic guidelines (Levin). The classification includes four different categories of primary headaches (Fig 7-2): *(1)* migraines, *(2)* tension-type headaches, *(3)* trigeminal autonomic cephalalgias, and *(4)* other primary headaches.

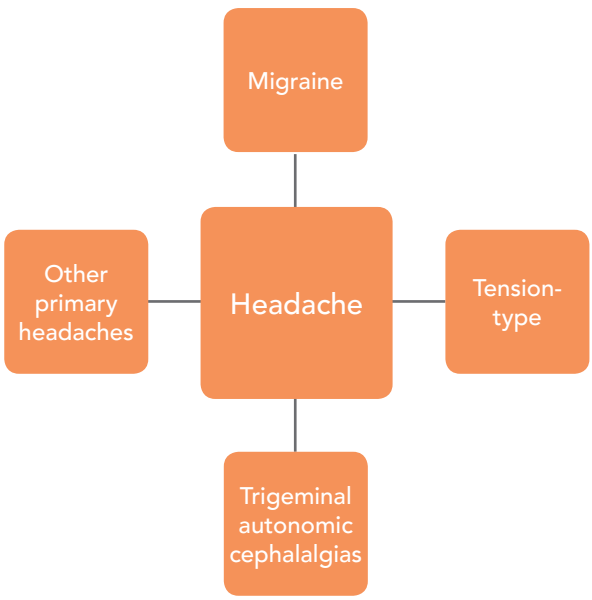

Fig 7-2 Headaches based on the International Classification of Headache Disorders diagnostic guidelines (Levin).

Migraine headaches may or may not present with a preceding aura, consisting of visual, sensory, or dysphasic symptoms. They are characterized by their unilateral location, pulsating quality, moderate or severe pain intensity, and possible aggravation by routine physical activity. Migraines are often associated with nausea or vomiting and photophobia or phonophobia. Attacks may last from 4 to 72 hours. Triptans are the primary treatment for migraine headaches (Levin).

Tension-type headaches are distinguished from migraine headaches by their milder intensity, lack of aura, and lack of nausea, vomiting, photophobia, or phonophobia. They tend to be bilateral and are characterized by a pressing or tightening rather than pulsating quality (Levin).

Trigeminal autonomic cephalalgias generally involve autonomic symptoms, such as conjunctival inflammation, tearing, nasal congestion, rhinorrhea, eyelid edema, facial sweating, miosis, and ptosis. This category includes cluster headache, paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks, and hemicrania continua. Complete relief with indomethacin is diagnostic for paroxysmal hemicranias. With the exception of hemicrania continua, the trigeminal autonomic cephalalgias occur episodically and repeatedly (Levin).

Other primary headaches include those attributed to certain activities, such as exertion, as well as headaches of otherwise unknown origin.

Neuropathic pain

Neuropathic pain often occurs secondary to a central lesion, including vascular compression of the trigeminal ganglion. Trigeminal neuralgia (TN) is the classic example of neuropathic pain and was recently reviewed by Zakrzewska. TN presents with recurrent episodes of sudden, sharp, stabbing pain in the distribution of the trigeminal nerve. A particularly hypersensitive trigger zone may or may not be present. Though it is often associated with vascular compression, Goh et al reported TN secondary to a tumor or multiple sclerosis. Neuropathic pain, including diagnoses other than TN, may also arise secondary to a neuroma (a proliferative mass of tissue at the site of prior trigeminal nerve transection) or neuritis due to inflammation oftentimes secondary to injury or an infection such as herpes zoster. A recent case report by Mehrkhodavandi et al showcased the complexity of neuropathic facial pain in a case of an acoustic neuroma resulting in TN and presenting as a toothache. Figure 7-3 summarizes types of neuropathic pain.

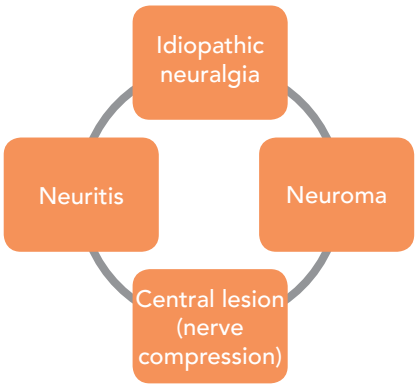

Fig 7-3 Types of neuropathic pain.

Treatment of neuropathic pain is by neurologic mediators, including carbamazepine, gabapentin, or pregabalin. Narcotic pain relievers are considered ineffective for TN.

Atypical facial pain

Atypical facial pain is a poorly understood pain entity that has been described extensively in the literature using varying terminology. The second edition of the International Classification of Headache Disorders renamed atypical facial pain. This pain that lacks the classical characteristics of cranial neuralgias and for which there is no obvious cause was instead termed *persistent idiopathic facial pain* (PIFP). It is essentially a diagnosis of exclusion made after

ruling out other conceivable diagnostic entities. PIFP generally refers to moderate to severe, nonanatomically distributed pain of unknown origin that does not follow typical diagnostic patterns. Often, patients describe pain of a burning quality that moves around. PIFP may develop following surgery or injury to the facial structures, though it persists without any obvious cause (International Headache Society).

The term *phantom tooth pain* was first used by Marbach to describe a similar disease entity to deafferentation pain. This pain was likened to phantom limb pain, wherein poorly understood pain persisted following extraction or removal of the dental pulp. Rees and Harris later coined the term *atypical odontalgia*. The International Classification of Headache Disorders mentioned this terminology as a subset of PIFP involving persistent pain in the teeth or tooth socket following dental work without any obvious dental cause. It appears that many use the term *atypical odontalgia* to refer to pain involving the teeth, and *atypical facial pain* or PIFP when the pain extends beyond the tooth into the orofacial structures. In any case, all of the terms mentioned are acceptable, including atypical facial pain and PIFP for pain involving the facial structures, and atypical odontalgia or phantom tooth pain for pain involving the dentition and related structures (Fig 7-4).

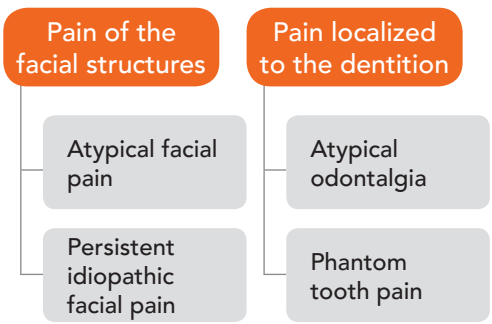

Fig 7-4 Various terminologies used to describe atypical facial pain.

Treatment of the PIFP entities can be partially diagnostic, in that local anesthetics are ineffective against this type of pain. Classically, treatment is pharmacologic. Denucci et al reviewed the use of tricyclic antidepressants (ie, amitriptyline) in the treatment of atypical facial pain.

Neuralgia-inducing cavitational osteonecrosis (NICO), also known as *Ratner bone cavities*, is a controversial diagnostic entity often classified within the atypical facial pains. Bouquot et al suggested its association with TN-like facial pain. The American Association of Endodontists released a position paper in 2012 that questioned its existence and suggested referral of suspected cases to an orofacial pain specialist.

Neurovascular pain

Neurovascular pain can occur in relation to the prominent neurovasculature in the head and neck. Temporal arteritis (TA) falls under the category of neurovascular pain. Friedlander and Runyon described TA as a systemic granulomatous disease, often affecting the carot-

id arteries, presenting with ocular symptoms, burning tongue, and headache. It is often associated with polymyalgia rheumatica, classically identified by an elevated erythrocyte sedimentation rate. TA is a serious disease with a high potential for subsequent blindness. Consequently, treatment with indomethacin should begin immediately on suspicion of TA, with a secondary biopsy to confirm the diagnosis.

Cardiac pain

Pain of cardiac origin, secondary to angina or myocardial infarction, classically presents as left posterior mandibular pain on exertion. Kreiner et al (2010) found that cardiac pain was more often described as pressure and burning, rather than the throbbing and aching frequently used to describe odontogenic pain. Cardiac pain in the orofacial area is not relieved by local anesthesia, but relief can occur with administration of nitroglycerin in early stages. Natkin et al published the first case report of dental pain of cardiac origin, and Kreiner et al (2007) presented an extensive case series.

Psychogenic pain

The psychogenic toothache, described by Dworkin and Burgess, should be a diagnosis of exclusion made in concert with a mental health professional. It should not be confused with malingering or factitious disease.

Infection

Though many infections of the orofacial complex are of endodontic origin, several other sources of infection exist and must be ruled out during the clinical examination. Periodontal disease is the most likely non-endodontic source of orofacial infections. Acute periodontal infections are associated with clinical and radiographic signs of periodontal disease and, usually, vital pulp tissue. They may present in patients with generalized signs of periodontitis or in localized areas in otherwise periodontally healthy patients, especially in cases where the etiology is a developmental groove or other anatomical anomaly. According to Berman and Hartwell, infections of periodontal origin generally do not present with diffuse facial swelling as endodontic infections are prone to do.

Additional non-endodontic infections include those caused by cemental tears. Cemental tears are generally classified as a type of root fracture that often results in periodontal, and occasionally periapical, infection. Haney et al described the cemental tear as a complete or partial detachment of the cementum from the underlying dentin, usually attributed to trauma from occlusion. In a study by Lin et al (2012), they were found most often in incisors of older patients and may or may not be associated with a vital pulp. Often, they present with a sinus tract tracing to root structure. Their diagnosis can only be confirmed surgically, as Stewart and McClanahan described in a case report. Although the lesions themselves do not often cause endodontic pathology, endodontic therapy is often completed due to initial misdiagnosis or may be required as a result of apical bony destruction.

Surgical removal of the cemental tear can result in a good prognosis for the tooth. Ultimately, the goal in treatment is the regeneration of periodontal and apical tissues lost

based on sound clinical reasoning. Treatments can range from nonsurgical scaling and root planing to complete surgical removal or even extraction. Lin et al (2014) performed a retrospective analysis of 71 cases of cemental tears treated by nonsurgical and surgical methods. Their group reported that a surgical approach was more often successful than a purely nonsurgical approach. Furthermore, the location of the cemental tear was a significant factor in the likelihood for healing, with tears located in the apical or middle portions of the root having better outcomes. An example of a cemental tear is presented in Fig 7-5.

Other possible etiologies of non-endodontic orofacial infections include osteomyelitis, infections secondary to osteonecrosis, or secondary infections of other pathologic bony or soft tissue lesions, such as malignant or nonmalignant tumors. An example of osteomyelitis is presented in Fig 7-6.

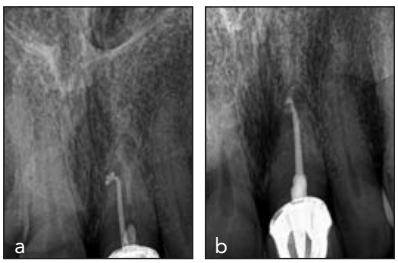

Fig 7-5 Case of a cemental tear involving the maxillary left central incisor in an 80-year-old woman. *(a)* Nonhealing sinus tract found following pulpectomy with 2 weeks of intracanal calcium hydroxide. *(b)* Completing root canal therapy and surgical access to remove the cemental tear resulted in healing of the sinus tract.

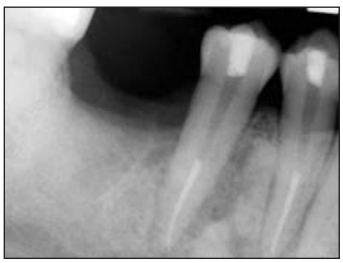

Fig 7-6 A case of osteomyelitis of the mandible presenting as a diffuse radiolucency surrounding the mandibular right first and second premolars.

Noninfectious swelling may also occur in orofacial structures and should be included in the differential diagnosis of any swelling without other obvious signs of infection, such as purulent drainage, lymphadenopathy, or fever. Hard, expansile lesions may be a nonpathogenic exostosis or torus or a malignancy such as a Kaposi sarcoma, osteosarcoma, or lymphoma. More fluctuant lesions may be salivary gland tumors, hemangiomas, angioedema, neurofibromas, or irritation fibromas.

Radiographic Entities Resembling Endodontic Pathology

Radiographic findings may mimic endodontic disease when pathology presents in or around the tooth. Radiographic changes may occur within the pulp chamber or pulp space, in the periodontal ligament (PDL), or periapically. In this text, we limit our discussion to those commonly encountered entities with examples prevalent in the literature. For a com-

prehensive review of radiographic findings that might mimic endodontic disease, please review an oral and maxillofacial radiology textbook, such as White and Pharaoh's *Oral Radiology: Principles and Interpretation*, as well as an oral pathology textbook, such as Neville et al's *Oral and Maxillofacial Pathology*.

Pulp chamber anomalies include calcifications or variations from normal anatomy. Calcifications may occur secondary to deep caries, restorative procedures, excessive occlusal forces, or trauma (Sener et al) (Fig 7-7). Certain systemic drugs are associated with generalized calcifications of the pulp, including statins (Pettiette et al 2013) and corticosteroids (Gold). Certain medical conditions are also associated with calcifications, including dentinogenesis imperfecta (Pettiette et al 1998), gout, hypercalcemia, end-stage renal disease (Sayegh and Reed), and cardiovascular disease (Edds et al).

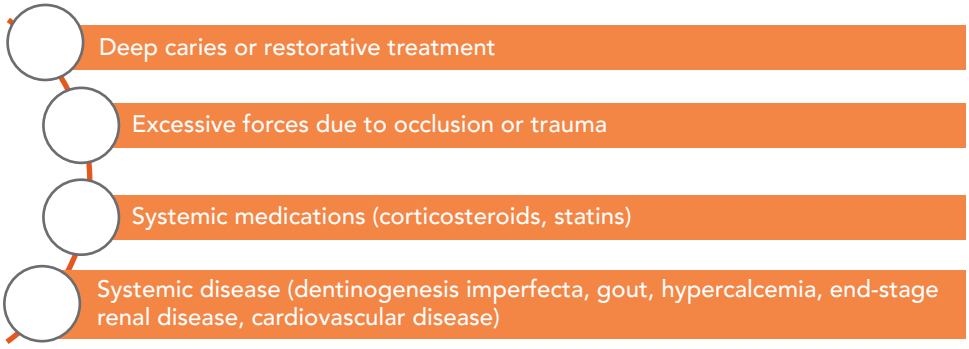

Fig 7-7 Factors associated with pulpal calcifications.

Other genetic conditions manifest with particular variants of pulpal anatomy. Dentin dysplasia, first presented in the endodontic literature by Tidwell and Cummingham, can be associated with a constricted pulp chamber shape and an increased prevalence of pulpal disease. Ehlers-Danlos syndrome may result in an increased number of pulp stones in molars as well as dilacerated roots (Hoff). An example of Ehlers-Danlos-associated pulp stones is presented in Fig 7-8. Vitamin D–resistant rickets may be associated with a taurodont-like shape to the tooth with an unusually high extension of the pulp horns to the dentinoenamel junction as well as thinned areas of enamel increasing the susceptibility to pulpal disease; this was described in a case report by Beltes and Zachou.

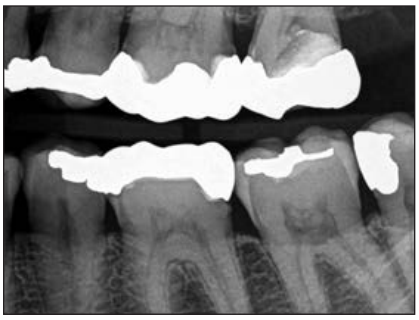

Fig 7-8 A radiographic depiction of multiple pulp stones in a patient with Ehlers-Danlos syndrome.

Widening of the PDL in a generalized fashion may be associated with scleroderma (Hasan et al) or related to certain malignancies of the jaw, such as osteosarcoma (Samraj et al).

In addition to radiographic changes evident within the dentition in response to pathologic conditions, surrounding tissues may also present with changes. Periapical radiolucencies often present secondary to endodontic pathology. Endodontic lesions fall into the categories of periapical granulomas, cysts, or apical scars. In a study of periapical biopsies taken during root-end surgery, Carrillo et al found that 66% of lesions were histologically granulomas, 9% were cysts, and 26% were scars.

Several other pathologic entities have been described in the periapical area, and this text describes several conditions mentioned in the endodontic literature.

Ameloblastomas are unicystic or multilocular radiolucencies generally found in the posterior mandible. Some have been found in the anterior mandible and may cross the midline. They are generally asymptomatic. Gondak et al presented a case of a unicystic ameloblastoma mimicking apical periodontitis.

Nasopalatine duct cysts may arise adjacent to maxillary incisors, presenting as a heart-shaped radiolucency at the maxillary midline. They are often associated with vital teeth, though in cases of previously treated teeth or associated with a history of traumatic dental injuries, their presentation may be confusing. Hilfer et al presented a case of misdiagnosis of a nasopalatine duct cyst associated with a previously treated tooth. Figure 7-9 presents a case of a nasopalatine duct cyst that is particularly apparent in a cone beam computed tomography (CBCT) section showing communication of the lesion with the nasopalatine duct.

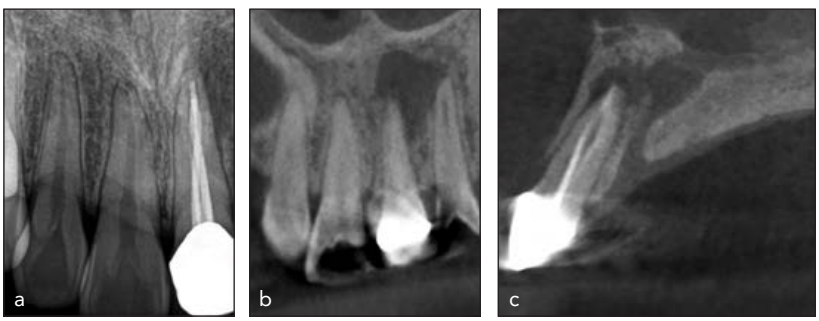

Fig 7-9 *(a to c)* Nasopalatine duct cyst associated with a previously treated tooth. Initial endodontic treatment may have been due to a misdiagnosis.

Periapical cemento-osseous dysplasia (PCOD) is a commonly encountered nonpathogenic entity comprising multiple radiolucencies in the periapical areas in early stages and radiopacities in the later stages (Fig 7-10). PCOD will often arise in the periapical areas of mandibular incisors mimicking apical pathology. PCOD can be differentiated from endodontic pathology by vitality testing and requires no treatment other than radiographic follow-up.

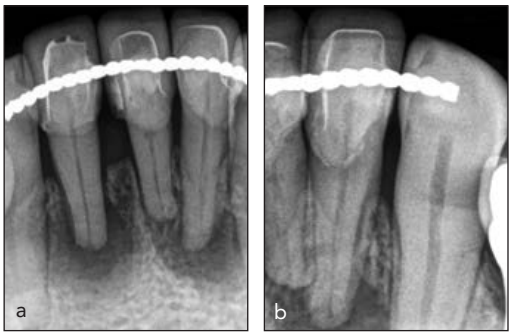

Fig 7-10 *(a and b)* PCOD associated with vital pulps.

Malignancies that cause bony resorption may present as radiolucencies in the jaw and have been reported in the periapical tissues. Malignancies generally occur in conjunction with other findings, including spontaneous paresthesias, pain, and swelling. Due to their fast-growing and aggressive nature, they may degrade the cortical bone without an opportunity for reactive sclerosis. Displacement of teeth is often not observed as rapid lesion expansion causes either PDL widening or the appearance of teeth floating in space. Malignant lesions have the potential to cause root resorption. Malignancies of the jaw that might cause radiographic changes include squamous cell carcinoma, osteosarcoma, multiple myeloma, non-Hodgkin lymphoma, and metastatic cancers. Troeltzsch et al presented a case of periapical radiolucencies and aggressive root resorption of mandibular molars secondary to jaw infiltration of multiple myeloma. Selden et al presented a case of metastatic pancreatic cancer mimicking periapical pathology. Figures 7-11 and 7-12 present malignancies mimicking apical periodontitis.

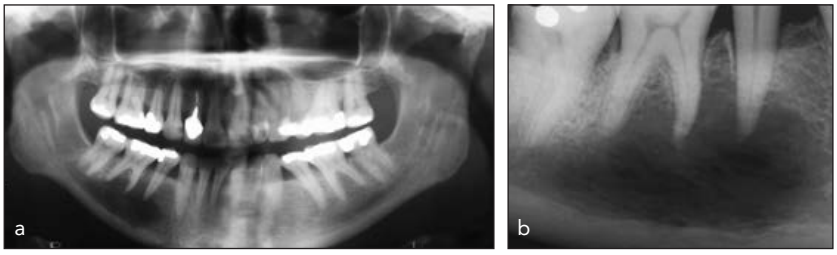

Fig 7-11 *(a and b)* Metastasis of breast cancer to the mandible mimicking apical periodontitis.

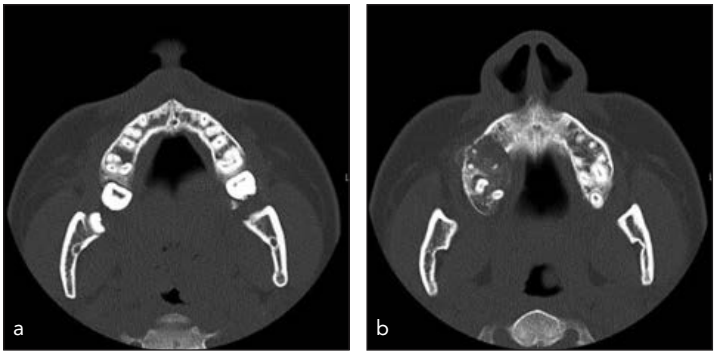

Fig 7-12 *(a and b)* CBCT depicting a case of osteosarcoma of the maxilla. (Courtesy of Dr Andrea Chung Shah, Peabody, Massachusetts.)

Other radiolucencies of the jaw include ameloblastic fibromas, keratocystic odontogenic tumors, odontogenic myxomas, globulomaxillary cysts, lateral periodontal cysts, and giant cell lesions, including the central giant cell granuloma, Langerhans cell histiocytosis, and the Brown tumor of hyperparathyroidism.

The complete list of radiolucencies of the jaw can be found in Fig 7-13. For a more comprehensive overview of jaw radiolucencies, including further diagnostic criteria, please refer to an oral pathology text.

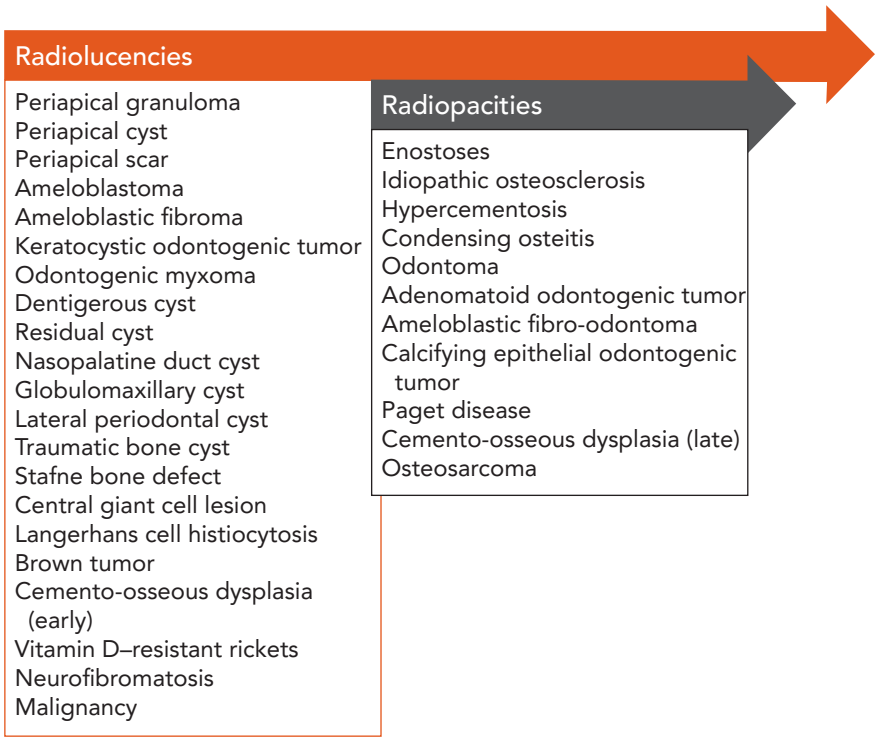

Fig 7-13 Disease entities to include in a differential diagnosis for radiographic periapical changes.

Radiopacities of the jaw may include nonpathologic and pathologic entities. Nonpathologic entities include enostoses (ie, areas of increased bone density) and idiopathic osteosclerosis. Pathologic entities include hypercementosis, condensing osteitis, and several other conditions. Hypercementosis may be a reactionary finding caused by occlusal or other trauma, creating a bulbous cementum area with an intact PDL and lamina dura surrounding. Hypercementosis may also occur secondary to Paget disease or acromegaly. According to Burklein et al, hypercementosis is not progressive and requires no intervention. Furthermore, they found a relatively low incidence of hypercementosis in a German population at 0.12%. Condensing osteitis is considered a pathologic condition associated with a nonvital tooth and should be treated endodontically (Eliasson et al).

Other radiopacities of the jaw include odontomas, adenomatoid odontogenic tumors, ameloblastic fibro-odontomas, calcifying epithelial odontogenic or Pindborg tumors, Paget disease of bone, late-stage cemento-osseous disease (Fig 7-14), fibrous dysplasia, and late-stage osteosarcomas. A comprehensive list of radiopacities of the jaw can be found in Fig 7-13. Readers are encouraged to review pathology and/or radiology texts for further details on these conditions.

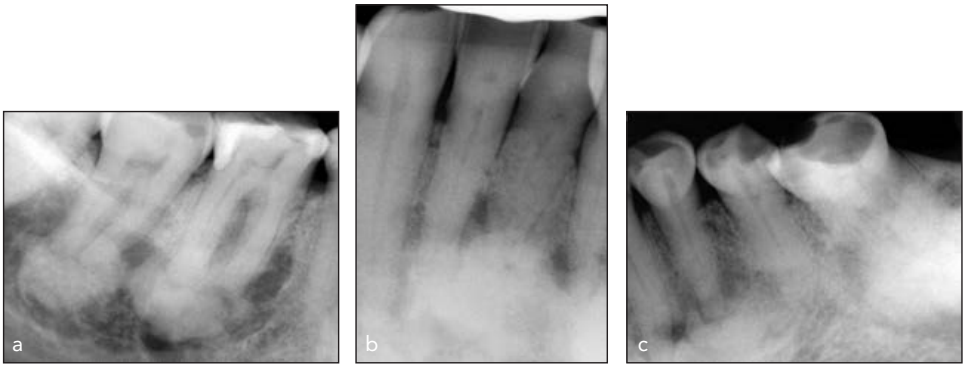

Fig 7-14 Florid cemento-osseous dysplasia presenting with mixed radiolucencies and radiopacities in the right *(a)*, anterior *(b)*, and left *(c)* mandible.

Certain diseases may cause endodontic disease in multiple teeth without obvious dental etiology. Costa et al found that sickle cell anemia was a risk factor for spontaneous pulpal necrosis. As described earlier in the discussion of pulpal changes, vitamin D–resistant rickets is associated with defects in enamel resulting in pulpal disease described by Beltes and Zachou. Rauckhorst and Baumgartner reported a case of spontaneous pulpal necrosis secondary to zoster involving the trigeminal nerve. Figure 7-15 summarizes disease entities associated with multiple periapical radiolucencies on nonvital teeth.

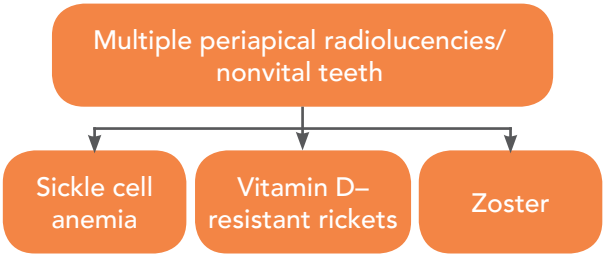

Fig 7-15 Differential diagnoses for multiple endodontically involved teeth without obvious dental etiology.

Other conditions are particularly associated with the presence of multiple periapical radiolucencies around vital teeth. As discussed earlier, PCOD and malignancies as well as neurofibromatosis and Brown tumors may present with multiple periapical radiolucencies associated with vital teeth. Neurofibromatosis is a genetic disease associated with multiple

neurofibromas, café au lait spots of the skin, and multiple periapical radiolucencies associated with vital teeth. The Brown tumor, associated with hyperparathyroidism, classically presents with a ground-glass appearance of bone, loss of the lamina dura, and multiple periapical radiolucencies. Brown tumors are associated with both primary hyperparathyroidism due to a pituitary adenoma and secondary hyperparathyroidism due to low serum calcium originating from vitamin D deficiency or chronic renal disease. Figure 7-16 summarizes disease entities associated with multiple periapical radiolucencies on vital teeth.

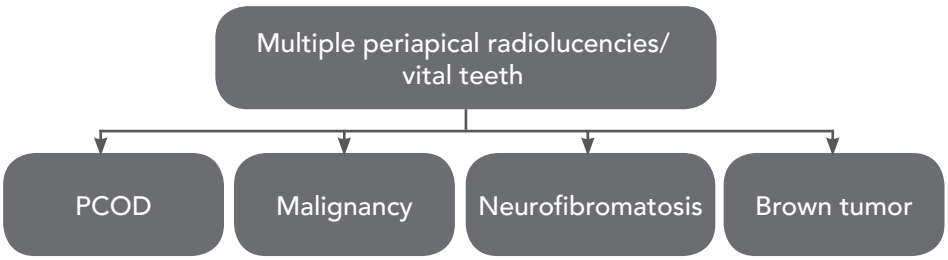

Fig 7-16 Differential diagnoses for multiple periapical radiolucencies associated with vital teeth.

Bibliography

Pain

American Association of Endodontists Position Statement 2012. NICO Lesions: Neuralgia-Inducing Cavitational Osteonecrosis. https://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/nicolesionsnew.pdf. Accessed 7 December 2015.

Bouquot JE, Roberts AM, Person P, Christian J. Neuralgia-inducing cavitational osteonecrosis (NICO). Osteomyelitis in 224 jawbone samples from patients with facial neuralgia. Oral Surg Oral Med Oral Pathol 1992;73:307–320.

Brännström M. Etiology of dentin hypersensitivity. Proc Finn Dent Soc 1992;88(Suppl 1):7–13.

Chen YH, Tseng CC, Chao WY, Harn WM, Chung SF. Toothache with a multifactorial etiology: A case report. Endod Dent Traumatol 1997;13:245–247.

Denucci DJ, Dionne RA, Dubner R. Identifying a neurobiologic basis for drug therapy in TMDs. J Am Dent Assoc 1996;127:581–593.

Dworkin SF, Burgess JA. Orofacial pain of psychogenic origin: Current concepts and classification. J Am Dent Assoc 1987;115:565–571.

Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: A review of clinical characteristics of 164 patients. Oral Surg Oral Med Oral Pathol 1985;60:615–623.

Friedlander AH, Runyon C. Polymyalgia rheumatica and temporal arteritis. Oral Surg Oral Med Oral Pathol 1990;69:317–321.

Goh BT, Poon CY, Peck RH. The importance of routine magnetic resonance imaging in trigeminal neuralgia diagnosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92:424–429.

Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia 2004;24(Suppl 1):9–160.

Kreiner M, Falace D, Michelis V, Okeson JP, Isberg A. Quality difference in craniofacial pain of cardiac vs dental origin. J Dent Res 2010;89:965–969.

Kreiner M, Okeson JP, Michelis V, Lujambio M, Isberg A. Craniofacial pain as the sole symptom of cardiac ischemia: A prospective multicenter study. J Am Dent Assoc 2007;138:74–79.

Levin M. The International Classification of Headache Disorders, 3rd edition (ICHD III)—Changes and challenges. Headache 2013;53:1383–1395.

Marbach JJ. Phantom tooth pain. J Endod 1978;4:362–372.

Mattscheck D, Law A, Nixdorf D. Diagnosis of nonodontogenic toothache. In: Hargreaves KM, Cohen S, Berman LH (eds). Cohen's Pathways of the Pulp, ed 10. St Louis: Mosby, 2011:49–70.

Mehrkhodavandi N, Green D, Amato R. Toothache caused by trigeminal neuralgia secondary to vestibular schwannoma: A case report. J Endod 2014;40:1691–1694.

Natkin E, Harrington GW, Mandel MA. Anginal pain referred to the teeth. Report of a case. Oral Surg Oral Med Oral Pathol 1975;40:678–680.

Rees RT, Harris M. Atypical odontalgia. Br J Oral Surg 1979;16:212–218.

Sessle BJ, Hu JW, Amano N, Zhong G. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurones in trigeminal subnucleus caudalis (medullary dorsal horn) and its implications for referred pain. Pain 1986;27:219–235.

Wright EF. Referred craniofacial pain patterns in patients with temporomandibular disorder. J Am Dent Assoc 2000;131:1307–1315.

Wright EF, Schiffman EL. Treatment alternatives for patients with masticatory myofascial pain. J Am Dent Assoc 1995;126:1030–1039.

Zakrzewska JM. Diagnosis and differential diagnosis of trigeminal neuralgia. Clin J Pain 2002;18:14–21.

Infection

Berman LH, Hartwell GR. Diagnosis. In: Hargreaves KM, Cohen S, Berman LH (eds). Cohen's Pathways of the Pulp, ed 10. St Louis: Mosby, 2011:2–39.

Haney JM, Leknes KN, Lie T, Selvig KA, Wikesjo UM. Cemental tear related to rapid periodontal breakdown: A case report. J Periodontol 1992;63:220–224.

Lin HJ, Chang MC, Chang SH, et al. Treatment outcome of the teeth with cemental tears. J Endod 2014;40:1315–1320.

Lin HJ, Chang SH, Chang MC, et al. Clinical fracture site, morphologic and histopathologic characteristics of cemental tear: Role in endodontic lesions. J Endod 2012;38:1058–1062.

Stewart ML, McClanahan SB. Cemental tear: A case report. Int Endod J 2006;39:81–86.

Radiographic Entities Resembling Endodontic Pathology

Beltes C, Zachou E. Endodontic management in a patient with vitamin D-resistant Rickets. J Endod 2012;38:255–258.

Burklein S, Jansen S, Schafer E. Occurrence of hypercementosis in a German population. J Endod 2012;38:1610–1612.

Carrillo C, Penarrocha M, Bagan JV, Vera F. Relationship between histological diagnosis and evolution of 70 periapical lesions at 12 months, treated by periapical surgery. J Oral Maxillofac Surg 2008;66:1606–1609.

Costa CP, Thomaz EB, Souza Sde F. Association between sickle cell anemia and pulp necrosis. J Endod 2013;39:177–181.

Edds AC, Walden JE, Scheetz JP, Goldsmith LJ, Drisko CL, Eleazer PD. Pilot study of correlation of pulp stones with cardiovascular disease. J Endod 2005;31:504–506.

Eliasson S, Halvarsson C, Ljungheimer C. Periapical condensing osteitis and endodontic treatment. Oral Surg Oral Med Oral Pathol 1984;57:195–199.

Gold SI. Root canal calcification associated with prednisone therapy: A case report. J Am Dent Assoc 1989;119:523–525.

Gondak RO, Rocha AC, Neves Campos JG, et al. Unicystic ameloblastoma mimicking apical periodontitis: A case series. J Endod 2013;39:145–148.

Hasan S, Khan MR, Haroon A, Khwaja KJ, Tarannum F, Hussain AR. Scleroderma—A case report and review of literature. Int J Contemp Dent 2011;2:46–53.

Hilfer PB, Bergeron BE, Ozgul ES, Wong DK. Misdiagnosis of a nasopalatine duct cyst: A case report. J Endod 2013;39:1185–1188.

Hoff M. Dental manifestations in Ehlers-Danlos syndrome. Report of a case. Oral Surg Oral Med Oral Pathol 1977;44:864–871.

Neville B, Damm D, Allen C, Chi A. Oral and Maxillofacial Pathology, ed 4. Philadelphia: Saunders, 2015.

Pettiette MT, Wright JT, Trope M. Dentinogenesis imperfecta: Endodontic implications. Case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86:733–737.

Pettiette MT, Zhong S, Moretti AJ, Khan AA. Potential correlation between statins and pulp chamber calcification. J Endod 2013;39:1119–1123.

Rauckhorst AJ, Baumgartner JC. Zebra. XIX. Part 2. Oral herpes zoster. J Endod 2000;26:469–471.

Samraj L, Kaliamoorthy S, Venkatapathy R, Oza N. Osteosarcoma of the mandible: A case report with an early radiographic manifestation. Imaging Sci Dent 2014;44:85–88.

Sayegh FS, Reed AJ. Calcification in the dental pulp. Oral Surg Oral Med Oral Pathol 1968;25:873–882.

Selden HS, Manhoff DT, Hatges NA, Michel RC. Metastatic carcinoma to the mandible that mimicked pulpal/periodontal disease. J Endod 1998;24:267–270.

Sener S, Cobankara FK, Akgunlu F. Calcifications of the pulp chamber: Prevalence and implicated factors. Clin Oral Investig 2009;13:209–215.

Tidwell E, Cummingham CJ. Dentinal dysplasia: Endodontic treatment, with case report. J Endod 1979;5:372–376.

Troeltzsch M, Oduncu F, Mayr D, Ehrenfeld M, Pautke C, Otto S. Root resorption caused by jaw infiltration of multiple myeloma: Report of a case and literature review. J Endod 2014;40:1260–1264.

White S, Pharoah M. Oral Radiology: Principles and Interpretation, ed 7. St Louis: Mosby, 2013.

8

Treatment of Endodontic Disease

The treatment of endodontic pathology follows careful diagnostic procedures and a thorough review of a patient's medical history. This chapter covers a wide range of endodontic treatment modalities, from nonsurgical root canal therapy to nonsurgical retreatment to surgical endodontic therapy and beyond. Furthermore, the reader will find information regarding postendodontic restorative care. The chapter concludes with a review of ethics and workplace safety issues.

Local Anesthesia

The foundation of endodontic treatment is the successful attainment of profound local anesthesia. The selection of both the proper anesthetic solution and administration technique permits comfortable delivery of endodontic therapy for the practitioner as well as patient. This section reviews several clinical anesthesia studies. A discussion of anesthetic pharmacology can be found in chapter 5, and orofacial anatomy relevant to anesthesia can be found in chapter 3. For those readers in search of a more comprehensive anesthesia text, please review *Successful Local Anesthesia for Restorative Dentistry and Endodontics* by Reader et al.

Maxillary anesthesia

Infiltrations, rather than nerve blocks, are often effective when treating maxillary teeth. In a recent study by Aggarwal et al (2011), no difference was noted between infiltrations and posterior superior alveolar blocks in their ability to anesthetize maxillary first molars. Furthermore, the addition of palatal anesthesia does not appear to increase anesthetic success rates. In the same study by Aggarwal et al (2011), no difference was found between buccal infiltrations alone or when combined with palatal infiltrations. Similarly, Guglielmo et al found that anesthesia success rates did not improve when palatal infiltrations were added; however, the duration of local anesthesia increased.

Based on the literature, both lidocaine and articaine provide successful anesthesia for the treatment of maxillary teeth. Controversy exists as to whether one provides superior anesthesia. Srinivasan et al found 4% articaine was superior to 2% lidocaine for posterior teeth, whereas Evans et al found that articaine was superior in the anterior but not the posterior regions. On the other hand, Kanaa et al (2012a) found no difference between the anesthetics. Consequently, the selection of either anesthetic appears justified.

Mandibular anesthesia

For treatment of mandibular anterior and premolar teeth, infiltration techniques often provide sufficient anesthesia. Dressman et al found that a single infiltration of 4% articaine in the mandibular premolar region provided successful pulpal anesthesia 80% to 87% of the time, and an additional infiltration increased success rates to 92% to 94%. Currie et al found that local infiltration in the molar area works via a combined mental and incisive nerve block.

The standard inferior alveolar nerve (IAN) block is the technique of choice for anesthesia of mandibular posterior teeth. Goldberg et al found no advantage of the Gow-Gates or Akinosi techniques over the standard approach. Malamed, on the other hand, suggested that the Gow-Gates technique is superior to the standard block.

The choice of anesthetic appears to have little effect on IAN block success. McLean et al found no differences between 3% mepivacaine, 2% lidocaine, or 4% prilocaine using this technique. Fernandez et al additionally found no advantage of using 0.5% bupivacaine over 2% lidocaine. However, Whitworth et al found that, in healthy teeth, 4% articaine is more effective than 2% lidocaine in achieving anesthesia of the mandibular first molar. Cau-

tion must be exercised with its use though: Haas and Lennon as well as Gaffen and Haas found a five-fold increase in paresthesias when articaine was implemented for IAN blocks. Similarly, Garisto et al reported that prilocaine and articaine used for dental local anesthesia were associated with an increased risk of paresthesia at 7.3 and 3.6 times, respectively.

Just as anesthetic formulation has little effect on the success of the IAN block, the volume of anesthetic also appears to have little effect on outcomes. In a study of 55 patients, Aggarwal et al (2012b) found that more profound anesthesia was achieved with 3.6 mL than with 1.8 mL of 2% lidocaine. In a similar, larger-scale study, Fowler and Reader found no differences between anesthetic volumes. Lastly, just as volume has no effect on anesthetic success, injection speed does not influence outcomes. Both Kanaa et al (2006) and Aggarwal et al (2012a) found no differences in anesthetic success between fast and slow injections.

Recently, some authors have suggested that buccal infiltration techniques may provide anesthetic success rates similar to those obtained by IAN blocks for the treatment of mandibular molars. Corbett et al and Poorni et al reported no difference between infiltrations and IAN blocks with 4% articaine in their ability to achieve pulpal anesthesia. Recently, Nydegger et al found that though 4% articaine was statistically more effective than lidocaine or prilocaine for buccal infiltrations of the mandibular first premolar in asymptomatic teeth, its success rate was only 55%. These findings suggest that the success of infiltration anesthesia is not predictable enough to support its use as a primary technique in the posterior mandible.

Adjunctive anesthetic techniques

When common anesthetic techniques fail to provide sufficient anesthesia for endodontic therapy, adjunctive anesthetic techniques become necessary. Commonly cited reasons for local anesthetic failure, summarized in Fig 8-1, include lower pH of inflamed tissue, unsuccessful techniques, inflamed nerves with altered resting potentials and decreased excitability thresholds, upregulation of anesthetic- and tetrodotoxin-resistant sodium channels, and patient apprehension (Hargreaves and Keiser). Furthermore, difficulty obtaining anesthesia of mandibular molars with the standard IAN block is well documented in the literature. In their text, Reader et al report that the success rate of the standard IAN block is between 15% and 57%. Consequently, supplementary anesthesia is often required.

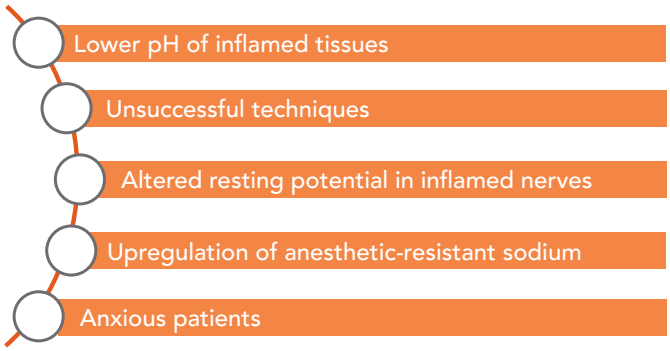

Fig 8-1 Commonly cited reasons for anesthetic failures in dentistry (Hargreaves and Keiser).

Supplemental anesthetic techniques include infiltrations, intraosseous anesthesia, periodontal ligament (PDL) injections, and intrapulpal injections (Fig 8-2). Infiltration, particularly with articaine, may be the most effective supplemental anesthetic technique. According to a randomized controlled trial by Kanaa et al (2012b), after a failed IAN block, additional articaine infiltrations provided successful anesthesia 84% of the time, intraosseous anesthesia 68% of the time, PDL injections 48% of the time, and repeat IAN blocks 32% of the time. Rogers et al found that articaine was significantly more effective than lidocaine when given as a supplementary buccal infiltration in symptomatic molars. Intraosseous anesthesia, though effective, has been associated with an increase in heart rate (Wood et al), and thus caution must be exercised with coexisting cardiac disease. Lastly, PDL injections do not damage periodontal tissues (Lin et al) and provide a safe alternative.

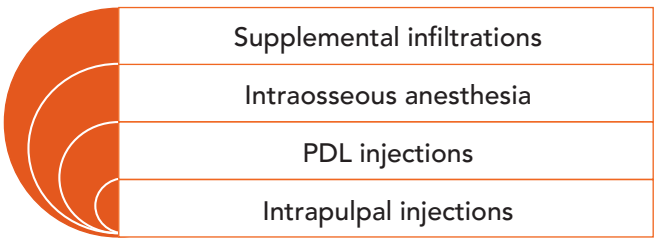

Fig 8-2 Available adjunctive anesthetic techniques for endodontic practice.

When all other techniques fail, intrapulpal anesthesia may be required. VanGheluwe and Walton found that this technique is successful 92% of the time. Furthermore, the effect appears to be independent of solutions employed, namely saline or anesthetic. These results suggest that anesthesia obtained by intrapulpal injection is due to intrapulpal pressure rather than anesthetic pharmacology.

Nonsurgical Root Canal Therapy

Isolation

The American Association of Endodontists (AAE 2010) asserts that the use of rubber dams during endodontic treatment is the standard of care. The aim of root canal therapy is to render the root canal system free of microbes. The rubber dam is the only device capable of preventing contamination of the root canal system with oral flora during treatment (Cochran et al). Furthermore, the rubber dam also aids in visualization during treatment and reduces the risk of aspiration of irrigants or instruments (Ahmad). Lastly, survival of endodontically treated teeth appears to be influenced by the use of rubber dam isolation during treatment. In a recent study, Lin et al found that the survival probability of endodontically treated teeth was significantly enhanced by rubber dam isolation.

Magnification

The use of magnification, including that provided by the surgical operating microscope (Fig 8-3), is essential in the practice of endodontics. Microscopy serves to aid the practitioner in the location of normal anatomical structures during nonsurgical or surgical endodontic therapy (Rubinstein and Kim), detection of cracks or fractures (Slaton et al), removal of obstructions, and management of treatment complications (Carr and Murgel). Given the number of advantages provided by the use of magnification during treatment, the AAE position (2012) is that the microscope is an integral and important part of the performance of modern endodontic techniques.

Fig 8-3 Surgical operating microscope. Magnification aids the practitioner in locating anatomical structures, detecting of cracks or fractures, removing obstructions, and managing treatment complications effectively.

Access

The shape and location of the access preparation should reflect pulpal anatomy. Krasner and Rankow found that the cementoenamel junction (CEJ) provides the most consistent landmark for the pulp chamber. Consequently, this landmark should be used when designing the access preparation to reduce the risks of underextension and untreated anatomy as well as overextension and perforation. Radiographic landmarks may also be useful to successfully locate the pulp chamber. Robinson et al (1989a, 1989b) recommended bitewing radiography to assess coronal pulp anatomy. Recently, Azim et al found that cone beam computed tomography (CBCT) images may be used for precise measurement of pulp chamber landmarks prior to access cavity preparation.

Working length determination

Following coronal access, working lengths (WLs) for each canal can be determined by radiographs, pre-existing CBCT scans, and electronic apex locators (EALs). Historically, radiographs were the only means by which WL could be determined. The accuracy of WL determination using this method varies by film type and radiographic technique. Lozano et al found that conventional film was more accurate than digital radiographs for WL determination when smaller file sizes were used; however, with files larger than a no.15 K file (0.15 mm in apical diameter), digital radiographs performed similarly. Forsberg found that the paralleling technique more accurately determined WL than the bisecting angle technique. New imaging techniques, namely CBCT, have been shown to provide accurate WL measurements. Jeger et al found a high degree of correlation between WLs measured with CBCTs and EALs.

EALs, like radiographs, provide accurate WL measurements. These instruments were developed based on the work of Suzuki, who showed that a constant value of electrical resistance, 6.4 kilo-ohms, exists between the PDL and the oral mucosa. Sunada confirmed Suzuki's findings in humans and applied this principle to electronic apex determination. Kobayashi and Suda developed the Root ZX apex locator [J. Morita] based on these findings. Typical EALs like the Root ZX measure impedance values that represent a ratio of resistances (Kobayashi and Suda) (Fig 8-4).

Fig 8-4 A Root ZX electronic apex locator. These units utilize impedance values to accurately measure root canal lengths. Their accuracy is not diminished by apical periodontitis, root resorption, or the presence of irrigants.

Research indicates that these devices are not only accurate but also useful under many clinical conditions. Shabahang et al found that EALs accurately located the apical foramen 96% of the time. However, Ounsi and Naaman found that they were more useful to determine the major diameter than the minor diameter. Their accuracy does not differ when used in vital or necrotic cases (Dunlap et al), in the presence of apical root resorption (Goldberg et al), or in the presence of apical periodontitis (Saatchi et al). Lastly, their accuracy is not affected by the presence of solutions, including lidocaine, sodium hypochlorite, RC-Prep [Premier Dental], ethylenediaminetetraacetic acid (EDTA), hydrogen peroxide, and chlorhexidine (Jenkins et al). Their accuracy may be diminished when apical diameters are larger than 0.6 mm (Herrera et al). Just as their accuracy is similar to radiographs, no differences in postoperative pain were noted when WLs were measured with either method (Kara Tuncer and Gerek).

Not only are EALs an accurate means to measure WL, they provide a safe means for WL determination in patients with pacemakers. Historically, EALs were considered unsafe based on work by Wooley et al. However, recent research has failed to demonstrate an effect of these units on pacemaker function or safety. An in vitro study by Garofalo et al showed no effect on pacemaker function when EALs were directly connected to the units. An in vivo study by Wilson et al further supported these results. Similarly, Idzahi et al found that these units are safe to use in patients with implantable cardiac defibrillators (ICD). The same study, however, indicated that electrosurgical units might alter ICD function.

Guide path maintenance and patency

With WLs established, a glide path to the apex must be maintained. Some authors advocate the maintenance of apical patency at the WL by passing small files through the cementodentinal junction (CDJ). Vera et al (2012a) found that maintaining apical patency decreased irrigant vapor lock in large canals by a significant margin, thus increasing irrigant

efficiency at the apex. In addition to an increase in irrigation efficiency, Arias et al found less postoperative pain when patency was maintained in nonvital teeth. Silva et al, on the other hand, found no significant differences in postoperative discomfort whether or not patency was maintained.

Regardless of the effect of patency maintenance on irrigation efficiency or postoperative discomfort, traversing the apical foramen with small files may have a detrimental effect on apical anatomy. Goldberg and Massone found that passing files through the periapex caused transportation of the apical foramen regardless of file size or instrument type. Adorno et al demonstrated a significant increase in the presence of apical cracks as a result of foraminal enlargement. Because of such demonstrable damage, one must carefully consider the practice of patency maintenance. Arguments both for and against the maintenance of apical patency are summarized in Fig 8-5.

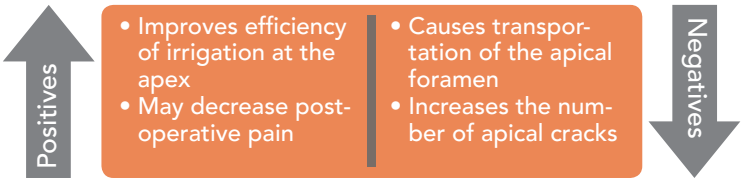

Fig 8-5 Positive and negative findings associated with the maintenance of apical patency by passing instruments through the apical foramen.

Instrumentation

Endodontic instrumentation serves to remove debris (Dalton et al), permit irrigant penetration to the apex (Salzgeber and Brilliant), and prepare the canal for obturation (Schilder 1974). Several root canal instrumentation techniques have been described in the literature. These techniques include the step-down (Goerig et al), passive step-back (Torabinejad), anticurvature filing (Abou-Rass et al), balanced force (Roane et al 1985), and crown-down techniques (Morgan and Montgomery) (Fig 8-6). According to Wu and Wesselink, all of these techniques leave residual debris behind to a similar degree. Consequently, technique selection should be based on operator experience and preference.

In addition to the multitude of instrumentation techniques described, several types of instruments, including both hand and rotary instruments, are available to practitioners. Hand instruments are often fabricated out of stainless steel, whereas rotary

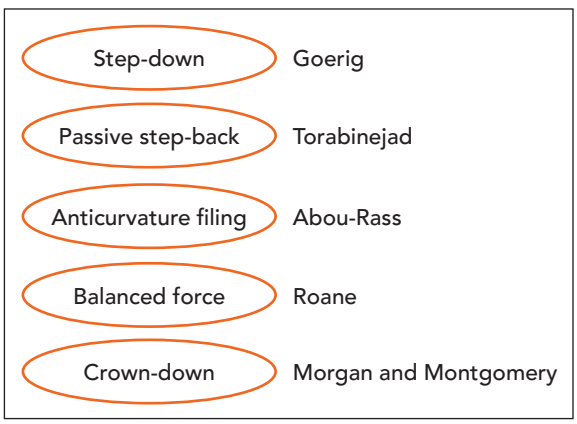

Fig 8-6 Commonly described instrumentation techniques and the authors to whom they are attributed.

instruments are frequently constructed from nickel titanium (NiTi) alloys (Hargreaves et al). NiTi alloys cycle through several temperature-dependent crystalline structures, including the stiffer austenite phase, the intercrystalline R phase, and the more flexible low-temperature martensite phase (Shen et al 2013). Newer instruments are fabricated from controlled memory (CM) wire, a heat-treated NiTi substance in which the austenite finish temperature is higher than body temperature, thus keeping the instrument in the more flexible martensite and R phases (Shen et al 2013). This technology improves NiTi fatigue resistance over conventional instruments (Shen et al 2011). According to Dalton et al, both hand and rotary instruments reduce intracanal bacterial levels to a similar degree. However, Short et al found that rotary instruments remain better centered in the root canal system.

With instruments and techniques selected, cleaning and shaping of the entire root canal system can commence. Instrumentation of the coronal portion of the canal, referred to as *coronal flaring*, has several functions (Fig 8-7). It provides straight-line access to the apical portion of the canal (Schroeder et al), and allows the apical foramen to be reached more consistently when read with EALs (Ibarrola et al). Furthermore, Roland et al found that preflaring decreases the incidence of rotary instrument separation. However, this process leads to a change in WL measurements; although, according to Schroeder et al, this change is clinically insignificant.

Fig 8-7 Functions of coronal flaring.

Instrumentation of the apical portion of the canal is often described with respect to the master apical file (MAF). Salzgeber and Brilliant found that a minimum MAF size of a no. 30 K file allowed penetration of irrigants to the apex. Other authors, including Mickel et al, have suggested that the MAF size should reflect the size of the apex. They found that by using a crown-down technique to assess apical size, followed by an increase in three file sizes to final instrumentation, greater bacterial reduction was noted than if only one file size greater than the initial was selected. Similarly, Saini et al found that outcomes were significantly improved when the MAF was three sizes larger than the initial apical size and that further enlargement did not provide any additional benefit. Consequently, absolute master apical file size recommendations may be inappropriate.

Instrumentation, though effective in shaping the root canal system and removing debris, produces a smear layer. Mader et al found that the smear layer consisted of two confluent components: the smeared layer on the surface of the canal wall and debris packed in dentinal tubules. McComb et al found that all standard instrumentation techniques produced this layer. Controversy exists as to whether or not this layer needs to be removed prior to

root canal obturation. Those who advocate for its removal include Taylor et al, who found less coronal leakage with its removal, and Sen et al, who found that the smear layer blocks the disinfecting properties of both sodium hypochlorite and chlorhexidine. Those who recommend leaving it intact include Madison and Krell, who found that the layer did not affect an apical seal, and Clark-Holke et al and Drake et al, who found more bacteria with the layer removed.

Irrigation

Endodontic instrumentation alone cannot render root canal systems free of debris. According to Peters et al, endodontic instrumentation leaves 35% of canal walls untouched. Furthermore, the existence of isthmuses between canals and lateral canal anatomy has been well documented in the literature (Senia et al), and often, instruments do not reach these areas. Consequently, irrigation is necessary to flush the root canal system of debris and eradicate microbes. According to a review by Zehnder, the ideal irrigant possesses broad antimicrobial properties, is highly effective against anaerobic and facultative microorganisms, dissolves both vital and necrotic tissue, inactivates lipopolysaccharide (LPS), and either prevents the formation of the smear layer during instrumentation or dissolves it once formed. Ideal irrigant properties are summarized in Fig 8-8.

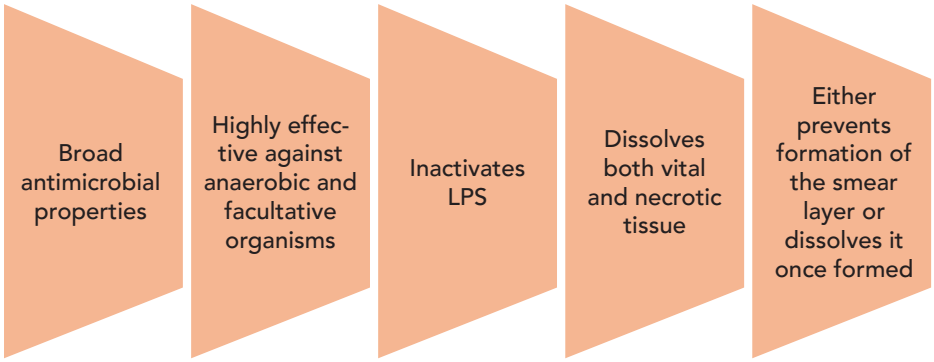

Fig 8-8 Properties of the ideal endodontic irrigant. Though many irrigants possess one or several of these capabilities, no one irrigant available today fulfills all criteria (Zehnder).

Sodium hypochlorite is the most commonly used endodontic irrigant, as it fulfills many of Zehnder's criteria. Its properties are summarized in Fig 8-9. Baumgartner and Mader found that sodium hypochlorite dissolves necrotic tissue and the organic component of the smear layer, while Rosenfeld et al found that it dissolves vital tissue. Sodium hypochlorite effectively eradicates endodontic pathogens, including planktonic bacteria (Haapasalo et al), those in established biofilms (Del Carpio-Perochena et al), as well as bacteria that have penetrated into the dentinal tubules up to 0.3 mm (Wong and Cheung).

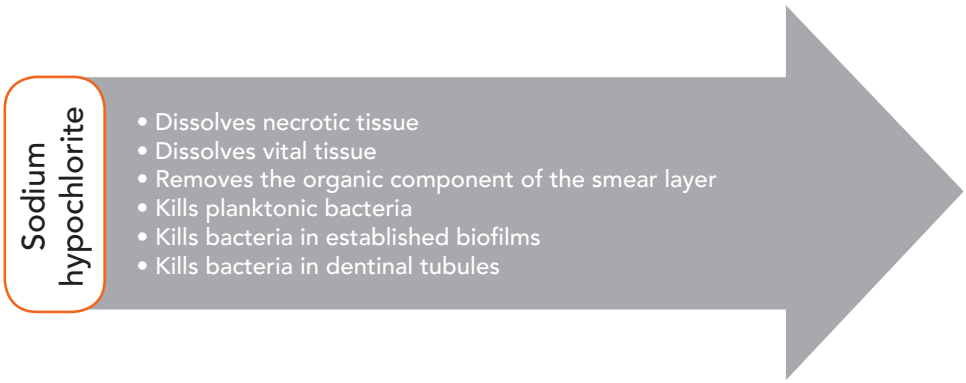

Fig 8-9 Properties of sodium hypochlorite that make it an effective endodontic irrigant.

Most AAE members report using sodium hypochlorite at concentrations of 5.25% or greater (Dutner et al). Several literature justifications exist to support the use of this concentration. Hand et al found that 5.25% sodium hypochlorite was most effective at dissolving necrotic tissue, and Senia et al found it was the best concentration for removing vital tissue. Harrison et al found that this concentration was safe for clinical use and did not increase postoperative pain. Morgental et al found that 5.25% sodium hypochlorite was more effective than either chlorhexidine or QMix irrigation solution [Dentsply]—a product containing chlorhexidine, EDTA, and surfactant—at eradicating *Enterococcus faecalis*. Despite reports on concentration effectiveness, it is clear that great variation exists in sodium hypochlorite concentrations obtained commercially, and less free chlorine is often available than is reported on the label (van der Waal et al). Consequently, practitioners must pay close attention to the products they purchase.

Although sodium hypochlorite effectively disinfects the root canal system and dissolves tissue, it lacks the ability to dissolve the mineralized component of the smear layer. As a result, many practitioners use chelating solutions as part of their irrigation protocols. EDTA chelates calcium ions, effectively targeting dental hard tissue debris (Calt and Serper). Calt and Serper found that a 1-minute rinse with EDTA removed the smear layer in its entirety, although a 10-minute application resulted in excessive dentin erosion. Additionally, Dai et al found that QMix was as effective as 17% EDTA in removing the smear layer. Consequently, practitioners now possess several choices in demineralization solutions.

Caution must be exercised when both EDTA and sodium hypochlorite are used in the same procedure, as their combined use can result in excessive demineralization of tooth structure. Qian et al found that sodium hypochlorite, if used as a final irrigant after EDTA, caused marked erosion of root canal dentin. Baumgartner and Mader found that when both solutions were alternated, excessive loss of intertubular dentin occurred. Furthermore, when solutions are combined, EDTA can reduce the efficacy of sodium hypochlorite. Clarkson et al found that the active chlorine content of sodium hypochlorite was reduced when mixed with EDTA. Consequently, practitioners must design their irrigation protocols carefully.

In addition to sodium hypochlorite and EDTA, chlorhexidine gluconate (CHX), a biguanide, has gained popularity as an endodontic irrigant. Jeansonne and White recommended its use in cases of allergy to sodium hypochlorite or with open apices where sodium hypochlorite extrusion would pose a risk. They found it was as effective as 5.25% sodium hypochlorite in terms of its antibacterial activity. Cook et al found that a 10-minute CHX soak was the most effective means to eliminate *E faecalis* from the root canal system. CHX also possesses the property of substantivity; it binds to dentin, allowing its antibacterial activity to persist for as many as 48 days after exposure (Baca et al). Despite these overwhelmingly positive findings, CHX has several drawbacks. First, it lacks the ability to dissolve organic substances, namely vital and necrotic tissue (Okino et al). Second, as Basrani et al found, it reacts with sodium hypochlorite to form a red-brown, carcinogenic precipitate called *parachloroaniline* (Fig 8-10). This precipitate has the ability to occlude dentinal tubules (Bui et al). Recent research by Barbin et al suggested that this precipitate may be present in solutions of CHX alone and with its use in combination with calcium hydroxide. Consequently, CHX should be used cautiously.

Fig 8-10 The chemical reaction between CHX and sodium hypochlorite solutions to produce the red-brown precipitate parachloroaniline (Basrani et al).

Adjunctive irrigation techniques

Just as several irrigant solutions are available to practitioners, so are adjunctive irrigation techniques, including passive ultrasonic activation, sonic activation, photodynamic therapy, and the EndoVac [Kerr] irrigation system (Fig 8-11). At this time, 45% of AAE members report using adjunctive irrigation techniques (Dutner et al). All techniques appear to offer improvements in disinfection, debridement, or both.

The EndoVac system and photodynamic therapy offer improvements in debridement or bacterial reduction, but not both. According to Nielsen and Baumgartner, the EndoVac system offers significantly better debridement than needle irrigation at a level 1 mm from the apex, most likely because it improves delivery of the irrigant to the apex (Munoz and Camacho-Cuadra). It may also improve sealer penetration at the apex (Kara Tuncer and Unal). Despite these improvements, Beus et al found that the EndoVac offers no improvement in bacterial reduction. Photodynamic therapy, according to Chrepa et al, is effective at reducing microbial loads; however, limited clinical studies exist.

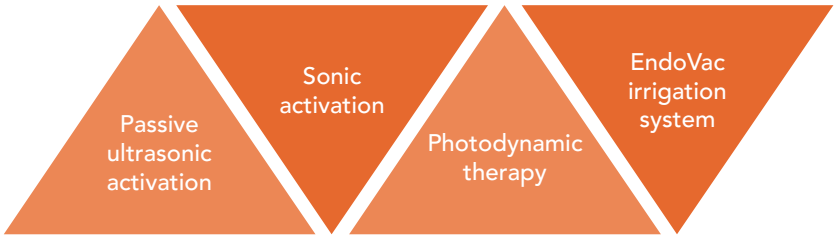

Fig 8-11 Commonly researched adjunctive irrigation techniques. These offer improvements in debridement, disinfection, or both.

Passive ultrasonic irrigation (PUI) improves both debridement and disinfection. Furthermore, according to Jensen et al, sonic activation offers debridement equivalent to PUI. By creating acoustic streaming patterns (Ahmad et al), PUI improves the cleanliness of both the main canal and isthmuses (Gutarts et al). It is more effective than syringe irrigation in removing debris from depressions in the canal space (Malki et al). PUI is also as effective as a final rinse with CHX in eliminating bacteria (Beus et al). Grundling et al, however, assert that bacterial elimination during PUI is a function of the irrigant rather than the activation. Despite the improvements PUI has over standard needle irrigation, Liang et al found no significant differences in periapical healing when this technique was employed.

Single-visit therapy versus multiple-visit therapy

Endodontic therapy can be completed in a single visit or over the course of multiple visits. Arguments both for and against single-visit endodontic therapy often focus on one of three factors (Fig 8-12): the eradication of bacteria, the effect on postoperative pain, and the effect on prognosis. A thorough review of the literature reveals that both single- and multiple-visit treatment can be supported based on those three subjects. Consequently, treatment either in a single visit or in multiple visits is justifiable, and the choice lies at the discretion of the practitioner. Vela et al found that though many patients would prefer single-visit therapy, most would follow their dentist's recommendation for two-visit therapy if an improved outcome was expected.

While several reports indicate that the root canal system cannot be reliably cleaned of

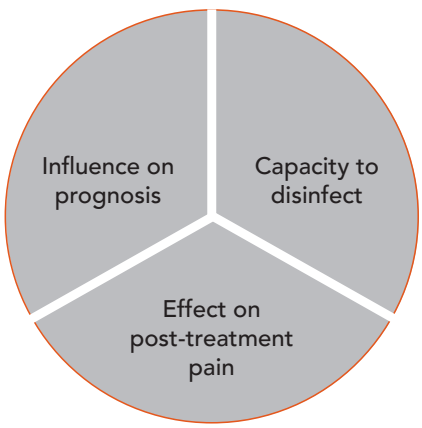

Fig 8-12 Three factors used to justify single-visit endodontic therapy versus multiple-visit therapy. Positions supporting either treatment can be defended based on any of these considerations.

bacteria in a single visit without use of an interappointment, intracanal medicament, other research found that disinfection was not improved with medicaments. Sjogren et al (1991) found that thorough disinfection of the root canal system required 7 days of treatment with calcium hydroxide (CH), and bacteria were not sufficiently eliminated after 10 minutes or 24 hours of exposure to the medicament. Law and Messer found that interappointment treatment with CH increased the number of root canals with undetectable levels of bacteria. Xavier et al interestingly found no difference in bacterial reduction in single versus multiple visits but found that two-visit treatment with CH eliminated more LPS than root canal therapy completed in a single visit.

On the other hand, several studies found that two-visit therapy does not increase bacterial disinfection. Peters and Wesselink found that, in two-visit therapy, bacterial regrowth was not prevented by interappointment CH, and no further disinfection was evident. Vera et al (2012b) found that, after two-visit treatment with CH dressing, bacteria were still evident in the isthmuses and other canal ramifications but not within the main canal or dentinal tubules.

Pain outcomes are also cited as reasons both for and against single-visit treatment. Like those for bacterial reduction, literature justifications exist to support either position. Soltanoff and Figini et al found that patients experienced more pain following single-visit treatment. Conversely, both Roane et al (1983) and Eleazer and Eleazer found more pain and more frequent flare-ups in multiple-visit treatment. Between these two extremes are authors, namely Pekruhn as well as Walton and Fouad, who found no differences in pain experienced by either group.

The last area often discussed in reference to single- and multiple-visit therapy is the effect on prognosis. Like bacterial reduction and pain, evidence for and against single-visit treatment based on prognosis is present in the literature. Trope et al found that two-visit therapy improved outcomes in necrotic cases. Similarly, Peters and Wesselink found a tendency for increased healing when two-visit endodontic therapy was performed, though results were not statistically significant. However, several studies rebuff these findings. Studies by Penesis et al, Molander et al, and Paredes-Vieyra and Enriquez and a meta-analysis by Su et al found no difference in outcomes between teeth treated in a single visit or over multiple visits.

Intracanal medicaments

If the decision to perform multiple-visit root canal therapy is made, the use of an intracanal medicament is recommended. The purpose of the medicament is to aid in disinfection and prevent recolonization of the root canal space with bacteria. Bystrom and Sundqvist demonstrated that, without the use of an intracanal medicament, canals rendered bacteria free during instrumentation exhibited culture reversals. Several medications are available to practitioners including CH, CHX gel, and antibiotic pastes. For further information regarding intracanal antibiotic pastes, please refer to the section in this chapter on regenerative endodontics.

CH has a basic pH between 11 and 12 and diffuses into dentinal tubules, causing an increase in the pH of outer root dentin after 2 to 3 weeks (Nerwich et al). These effects do not extend into cementum or the adjacent PDL space, indicating that the cementum may act as a buffer (Tronstad et al). The most effective means of delivering CH into the root canal space is the lentulo spiral, followed by injection and K files (Sigurdsson et al). CH eliminates

the majority of bacterial species in the root canal system, though some anaerobic species (Sjogren et al 1991) and *E faecalis* may be resistant to its effects (Siren et al). In addition to its antibacterial effects, CH reduces the cytotoxic response to LPS by destroying its lipid A moiety (Safavi and Nichols). CH may also dissolve tissue remaining in the root canal space after instrumentation (Hasselgren et al). Furthermore, it is the favored intracanal medicament for regenerative endodontic therapy as it is less cytotoxic than antibiotic pastes or CHX against apical papilla stem cells (Ruparel et al). Interestingly, the combination of CH and omeprazole resulted in superior repair of rat periapical lesions when compared with conventional CH (Wagner et al).

Despite its positive effects, care must be exercised with CH as it may negatively impact the physical properties of both teeth and root canal filling materials. Blomlof et al demonstrated that long-term applications could cause PDL necrosis. Additionally, Andreasen et al found that long-term CH decreased dentinal fracture resistance. CH can also negatively impact surrounding anatomical structures. Care should be taken to prevent its overextension into tissue spaces as it has been associated with devastating tissue necrosis (Lindgren et al) and paresthesia (Ahlgren et al). Lastly, CH may influence obturation because it inhibits setting of eugenol-based sealers (Margelos et al). Remaining CH can affect the penetration of sealers into dentinal tubules and increase apical leakage (Kim and Kim). Consequently, its thorough removal is imperative prior to obturation. Passive ultrasonic irrigation (Capar et al) or rotary instrumentation (Kenee et al) are more effective than syringe irrigation at removing CH from the canal system. Peracetic acid was found to remove more CH than sodium hypochlorite or EDTA (Sagsen et al).

CHX gel is often suggested as an alternative to CH. Like CH, it possesses broad antibacterial abilities. Wang et al found that 2% CHX gel is an effective root canal disinfectant. It is effective against gram-positive and gram-negative bacteria, both aerobic and anaerobic species, and fungi (Waltimo et al). Furthermore, it exhibited greater antibacterial activity against *E faecalis* than CH (Buck et al). Like CH, CHX also reduces intracanal LPS (Oliveira et al). Lastly, CHX alters periapical healing responses. Not only does its application between appointments prevent the increase of pro-inflammatory and immunoregulatory cytokines (Tavares et al), but more favorable healing of periapical lesions was noted with CHX application than with CH (Leonardo et al). Properties of CH and CHX are summarized in Fig 8-13.

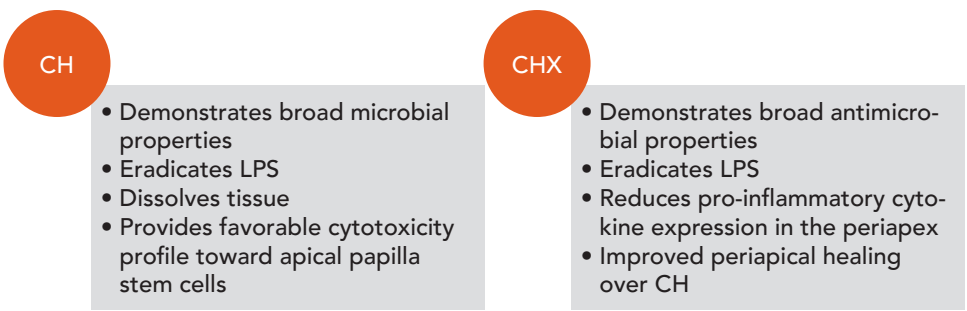

Fig 8-13 Properties associated with CH and CHX intracanal medicaments that make them useful for endodontic therapy.

Obturation

Following cleaning and shaping of the root canal system, placement of a three-dimensionally adapted filling is indicated (Schilder 1967). Several materials and techniques are available to accomplish this goal. Traditional root canal filling material is composed of gutta-percha (GP), described in Fig 8-14. GP is closely related to rubber and is a naturally occurring polymer of isoprene (Goodman et al 1974). GP cones are composed of zinc oxide (65%), GP (20%), and other materials including waxes, resins, and metals (Goodman et al 1974). GP is in its beta crystalline state when in cone form and undergoes a change to the alpha crystalline state when heated between 42°C and 49°C (Goodman et al 1981). GP designed for thermoplastic applications shrinks quickly and extensively upon cooling and differs significantly between brands (Lottanti et al). As GP is chemically close to rubber, care should be used in patients with a severe type I allergy to latex (Costa et al, Orstavik).

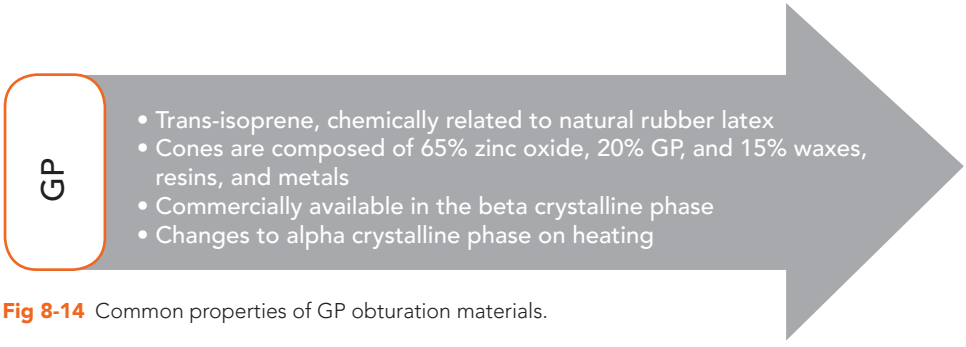

Fig 8-14 Common properties of GP obturation materials.

Filling techniques with this material include lateral condensation and vertical condensation. Neither leakage nor outcomes appear to be influenced by filling technique. Jacobson et al found that, though teeth obturated by lateral condensation leaked microbes faster than those filled by vertical condensation, no differences in the numbers of teeth that leaked were noted. No differences were noted in voids between the two techniques (Reader et al). Lastly, no differences in outcomes were noted between teeth treated with either technique (Peng et al).

Resilon [Resilon Research] offers an alternative to GP fillings; it is a polyester core material used with a resin-based sealer (Orstavik). Though previous research suggested that Resilon offered superior fills to GP (Teixeira et al), other work suggested that fills offer no improvements over GP in terms of leakage (Biggs et al) or movement into three-dimensional anatomy (Karr et al). Although Resilon was thought to create a "mono block" filling material whereby the sealer bonded both to dentin and to the filling material (Teixeira et al), research does not support these claims (Gesi et al).

Carrier-based systems provide an alternative to GP, though the filling quality they provide may be inferior to other techniques. These are marketed as offering significant time savings and ease over other obturation systems. However, research shows that the carrier-based filling materials result in fills that leak significantly more than laterally or vertically condensed GP (Baumgardner et al).

No matter which technique is used to obturate the root canal system, sealer is an essential component (Marshall and Massler). Several sealers are commercially available, including eugenol-based products, resin/epoxy–based products, glass-ionomer–based products, silicone-based products, and CH-based products. Recently, sealers-based on mineral trioxide aggregate (MTA) have become available (Vitti et al). Sealers are often compared in terms of biocompatibility, ease of use, antibacterial activity, adherence to dentin, and other properties (Orstavik). It behooves the practitioner to select an appropriate product based on these properties and one's personal preferences. No matter which sealer is selected, placement can be completed with K files, lentulo spirals, or GP cones with no effect on fill quality (Wiemann and Wilcox).

Instrumentation and obturation should terminate at the CDJ (Ricucci), as overinstrumenation and overfilling cause periapical inflammation (Seltzer et al). Obturation materials, including particulate GP, have been associated with robust inflammatory responses (Sjogren et al 1995). Though Augsburger and Peters found that sealers expressed into the periapical tissues resorb radiographically over time, care should be taken to avoid doing so because these materials have been associated with chronic inflammation (Seltzer et al), sinus infection with *Aspergillus* (Giardino et al), and paresthesia (Gonzalez-Martin et al).

Temporary restorations

Following completion of root canal therapy, and prior to placement of the definitive coronal restoration, temporary restorative materials are used to seal the coronal access, thus preventing contamination of the root canal filling with oral microbes (Swanson and Madison). Several temporary filling materials are commercially available, including but not limited to Cavit [3M ESPE], Intermediate Restorative Material (IRM) [Dentsply], and glass-ionomer cements. In a study by Turner et al, all of the aforementioned materials adequately prevented microleakage. For adequate prevention of leakage with Cavit, Webber et al found that a 3.5-mm-thick seal was necessary. Lamers et al showed that Cavit leaked after 42 days, supporting its use only in the short term. Barthel et al showed that both Cavit and IRM leaked significantly more than glass ionomers. Consequently, glass ionomers may be the materials of choice for temporary restorations.

Eugenol is found in several temporary restorations and in root canal cements. This clove oil derivative exhibits several biologic activities worth mentioning (Fig 8-15). Eugenol alters neurotransmission (Kozam) via increased potassium permeability and decreased sodium influx in nerves, thus decreasing the rate at which action potentials fire (Trowbridge et al). Furthermore, eugenol blocks the expression of neuropeptides associated with inflammation (Trowbridge). Lastly, it decreases the vasoconstriction response to epinephrine (Mjor).

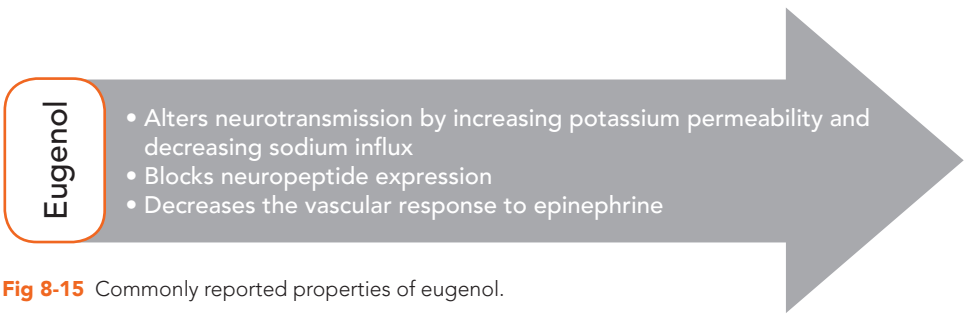

Fig 8-15 Commonly reported properties of eugenol.

Nonsurgical Retreatment

Nonsurgical retreatment is indicated when symptoms or radiographic pathology are evident in endodontically treated teeth. Factors associated with persistent or recurrent apical periodontitis include coronal leakage (Ricucci and Siqueira), intraradicular infection (Vieira et al), extraradicular infection, untreated canal anatomy, fractures, foreign body reactions, and true cystic lesions (Nair). With proper case selection, the prognosis of nonsurgical retreatment is favorable (Salehrabi and Rotstein). Despite the often-favorable outcomes, many general dentists prefer to pursue extraction of teeth with recurrent apical periodontitis in favor of implant placement (Azarpazhooh et al). Consequently, specialists must educate their referral base regarding both treatment possibilities and their prognosis.

Post removal

Posts are often the first obstacles encountered in a retreatment case. According to Johnson et al, 16 minutes of ultrasonic vibration is an effective means to remove metal posts. In addition to their efficacy, ultrasonics are safe for use, even in patients with pacemaker units (Gomez et al). Care must be taken, though because ultrasonics produce significant amounts of heat and are thus capable of causing bony damage. Dominici et al found that temperature increases approaching 10°C occurred in as little as 15 seconds without irrigation. Thankfully, Huttula et al found that the use of an irrigant maintained the temperature well below that which could cause bony damage. The removal of fiber posts can be accomplished most effectively with either burs or ultrasonic vibration, though these methods are slower than commercially available kits (Lindemann et al).

Gutta-percha removal

The ability of retreatment techniques to remove GP fillings depends not only on the composition of the sealer (Neelakantan et al, Hess et al) but also the type of instruments used (Xu et al). Retreatment of GP root canal filings often employs the use of solvents. Kaplowitz found that of five tested solvents, chloroform was the only solvent able to completely dissolve the GP. Not only is chloroform safe for both patients (Chutich et al) and providers (McDonald and Vire) if used properly, it also aids in disinfection of the root canal space (Edgar et al). Ferreira et al found that the combination of chloroform and either rotary or hand instrumentation produced similarly clean canals; however, rotary methods were significantly faster. Care must be taken no matter which method is chosen as both can cause defects in canal walls (Shemesh et al).

"Russian Red" removal

A combination of formaldehyde and resorcinol used in conjunction with a sodium hydroxide catalyst produces the paste-like filling material often referred to as "Russian Red." These components solidify following their introduction into the root canal space with an appropriate instrument. Retreatment of Russian Red is often difficult, as the fillings produced by this material range in consistency from sap-like to rock hard (Schwandt and Gound). Unlike GP fillings, solvents do not affect Russian Red endodontic pastes. Vranas et al found that both

chloroform and Endosolv R [Septodont] had no effect on the material, while both sodium hypochlorite and sodium chloride had only marginal efficacy. Ultrasonics may provide an effective means for removal of the paste (Krell and Neo), though if the fill is solidified to the apex, one may not be able to gain patency. Another technique involves bypassing with files, though as with ultrasonics, success is dependent on filling consistency (Schwandt and Gound). A retreatment case of Russian Red is illustrated in Fig 8-16.

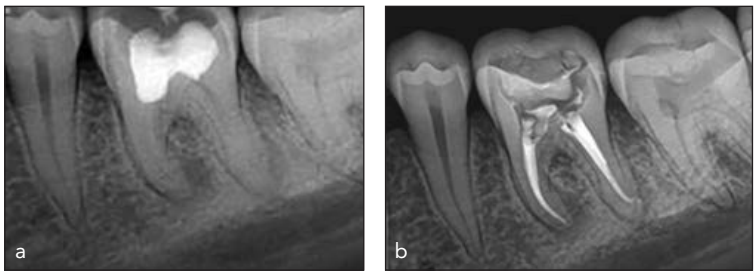

Fig 8-16 Preoperative *(a)* and postoperative *(b)* radiographs of a retreatment procedure on a tooth previously treated with the Russian Red technique. Judicious use of an ultrasonic instrument allowed the clinician to bypass the hardened material, and small files were used to reach the apex.

Silver points

Silver points were historically used to obturate the root canal system. The AAE recommends against the further use of this material due to its inferiority to modern techniques. Not only do silver points represent less effective obturation materials, but Seltzer et al found that they corrode in the presence of tissue fluid, producing highly toxic corrosion byproducts. The AAE holds that, while prophylactic revision of silver points is not indicated, retreatment is recommended in the presence of pathology or when their presence complicates proper restorative care. Retreatment techniques effective at removing these materials include ultrasonic removal of surrounding sealer, bypassing with instruments, or the use of a Masserann technique involving trephination around the coronal portion of the fill followed by insertion of a tube to extract the piece (Krell et al). A silver point retreatment case is illustrated in Fig 8-17.

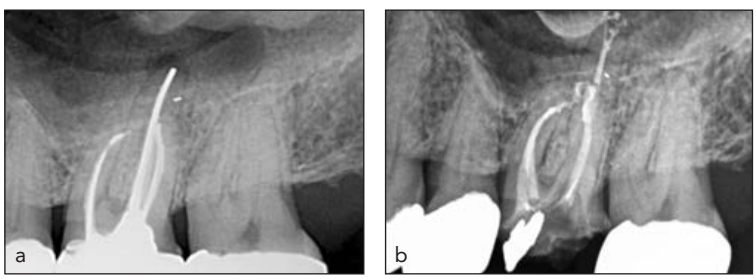

Fig 8-17 Preoperative *(a)* and postoperative *(b)* radiographs of a retreatment procedure on a tooth initially obturated with silver points. Bypassing and the Masserann technique were implemented to remove the points.

Carrier-based system removal

Carrier-based obturation techniques have become more widely used, necessitating the development of effective retreatment strategies for these materials. Both metal and plastic carriers are impervious to chloroform (Wilcox). Consequently, an effective technique for their retreatment involves the combination of chloroform to soften the surrounding GP and hand files to remove the carrier (Bertrand et al). Rotary instruments have also been advocated for the removal of these fillings (Royzenblat and Goodell), though instrument speed must be kept at manufacturers' recommended levels to decrease the incidence of instrument separation. Lastly, heated instruments, like the System B endodontic fill device [Kerr], may be effective for removal of carrier-based fillings. According to Wolcott et al, temperature settings must be maintained below 300°C to avoid melting the plastic carrier. Retreatment of a carrier-based root canal filling is illustrated in Fig 8-18.

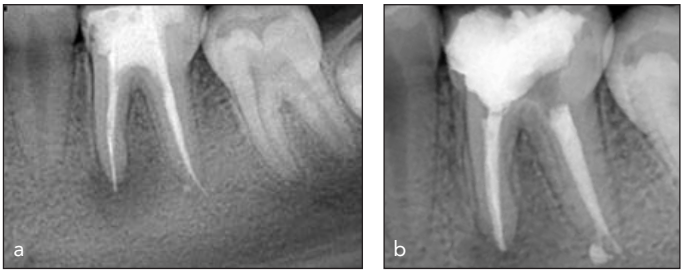

Fig 8-18 Preoperative *(a)* and postoperative *(b)* radiographs of a retreatment procedure on a tooth initially obturated with a carrier-based material.

Surgical Endodontics

Surgical endodontic therapy seeks to resolve periapical pathology when orthograde endodontic treatment is not feasible (Iqbal et al). Tools and techniques have evolved significantly since surgical therapy was developed, and with these advancements have come improved outcomes (Setzer et al). Modern advances in surgical techniques include magnification with loupes or microscopes (Von Arx et al 2010), ultrasonic retropreparations (de Lange et al), and improved filling materials (Song and Kim). With proper case selection and the use of modern techniques, surgical endodontics provides patients with a predictable means of eradicating periapical pathology (Tsesis et al 2013). Lastly, as financial concerns may play a role in patients' pursuit of dental treatment, it should be noted that surgery is often more cost effective than nonsurgical retreatment (Kim and Solomon). This section covers basic surgical literature. A more complete surgical reference can be found in Kim et al's *Color Atlas of Microsurgery in Endodontics*.

Hemostasis

Effective hemostasis is essential during endodontic surgery to maintain visibility in the operative field (Gutmann). Furthermore, hemostatic measures curb the amount of blood loss during surgical intervention. According to Selim et al, an average of 9.5 mL of blood is lost during surgery. Several measures are available to the clinician to curb intraoperative blood loss. The first measure involves a thorough review of a patient's medical history including assessment of medication and supplement use (Witherspoon and Gutmann). Doing so allows the clinician to anticipate difficulties with hemostasis prior to surgical intervention and develop an appropriate plan. In certain cases following consultation with the prescribing physician, alterations in medication use may be made preoperatively to minimize bleeding risk.

During surgery, clinicians have several hemostatic measures at their disposal. Historically, bone wax was employed for hemostasis, though it has fallen out of favor because it causes a robust inflammatory response (Ibarrola et al). Epinephrine is an effective hemostatic agent via its interaction with blood vessel alpha receptors, producing vasoconstriction (Gutmann). Local anesthetics containing a 1:50,000, rather than the more commonly used 1:100,000, concentration of epinephrine reduce blood loss by half and offer better visibility, reduced surgical time, and improved postoperative hemostasis (Buckley et al). Racellet hemostatic cotton pellets [Pascal], containing 0.55 mg of racemic epinephrine per pellet, also promote surgical hemostasis without risking cardiovascular effects (Vickers et al). Ferric sulfate also produces excellent hemostasis in the surgical field (Vickers et al), though it requires curettage prior to wound closure to prevent the formation of a foreign body reaction (Jeansonne et al). Other local hemostatic measures include collagen-based agents like CollaCote [Zimmer], gelatin-based products like Gelfoam [Pfizer], and cellulose-based products like Surgicel [Ethicon] (Witherspoon and Gutmann). Lastly, calcium sulfate was recently investigated for use in endodontic surgery and was found to provide effective hemostasis (Scarano et al). Hemostatic measures for endodontic surgery are summarized in Fig 8-19.

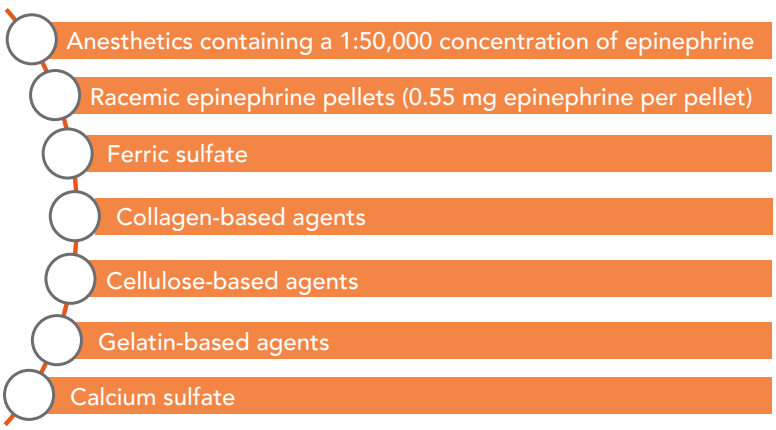

Fig 8-19 Common hemostatic agents available during endodontic surgery.

Exposure of the surgical site

Soft tissue mobilization permits access to the periapical tissues, and both horizontal and vertical incisions are used during this process. Several incision types are available for practitioners and must be carefully selected; the choice of incision technique may affect the patient's postoperative quality of life (Del Fabbro et al). Commonly used types of horizontal incisions for endodontic surgical applications include the intrasulcular, submarginal, semilunar, and papilla-based incisions. Intrasulcular incisions extend through the gingival sulcus and expose the entirety of the root structure for inspection (Velvart and Peters). They are associated with more postoperative recession and scarring than other incision types (Kramper et al). Submarginal incisions, also known as the *Oschenbein-Luebke technique*, require a minimum of 2 mm of attached gingiva and do not expose the cervical third of the root (Velvart and Peters). Scalloping of this type of incision is often recommended to provide landmarks during flap closure (Vreeland and Tidwell). The scalloped submarginal incisions are associated with better epithelial closure than submarginal or semilunar incisions but do pose a risk for scar formation (Kramper et al). Semilunar flaps incise into the alveolar mucosa and expose a minimal amount of root structure for visualization; consequently, they are infrequently used in endodontic surgery (Velvart and Peters). Lastly, the papilla-based incision represents a modification of the intrasulcular technique and permits visibility of the entire root surface, while mitigating the risk for recession by preserving the interdental papillae (Velvart et al). Horizontal incision techniques are summarized in Fig 8-20.

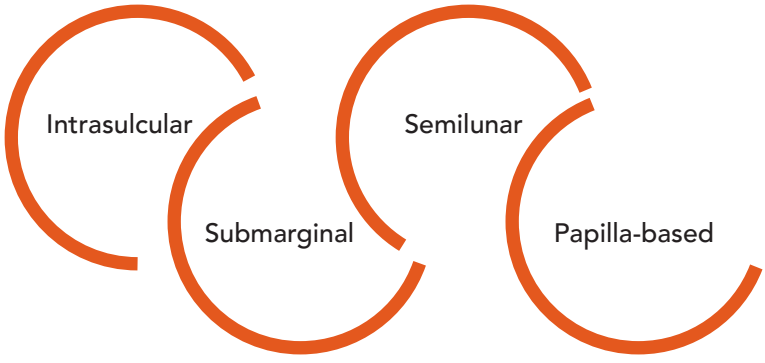

Fig 8-20 Commonly used horizontal incision types for surgical endodontics.

Horizontal incisions are connected to vertical incisions to create the soft tissue flap. The use of a single vertical releasing incision creates a triangular flap, whereas two vertical incisions create a rectangular or trapezoidal flap (Velvart and Peters). When creating a rectangular flap, a length-to-width ratio of 2:1 is recommended (Velvart and Peters).

The osteotomy exposes the root apex for inspection and can be created with the use of burs under coolant spray (Kim et al). The osteotomy should be large enough to permit manipulation of ultrasonic tips. Its size, however, should be limited, as smaller osteotomy preparations are associated with more favorable outcomes (Barone et al).

Resection and retropreparation

Apical surgery involves both resection of the root apex and preparation into the root canal system and should be completed with the use of a coolant to avoid overheating of the bone (Nicoll and Peters). Resection of the root apex can be quite simply accomplished by burs in a high-speed handpiece (Kim et al). Block et al advocated 3 mm of apical resection to remove the majority of apical deltas. However, Weller et al reported that a 4-mm resection of the mesiobuccal root of the maxillary first molar may be necessary to expose intercanal isthmuses. Historically, resection involved beveling to improve visualization. However, recent trends shy away from bevels because they are associated with leakage (Gilheany et al). The number of dentinal tubules increases from the CDJ to the canal wall, and consequently retrofillings must extend to the most coronal aspect of the bevel, lest potentially contaminated open tubules be exposed (Tidmarsh and Arrowsmith).

Following resection of the root surface, it should be inspected for fractures, additional anatomy, and other findings. Methylene blue dye is often utilized during the inspection process. Methylene blue dye is a biocompatible reducing agent, notable for its use in the treatment of methemoglobinemia. In endodontics, it outlines root anatomy, delineates dentin from bone, stains isthmuses, and can aid in visualization of fractures (Cambruzzi et al).

On resection and inspection of the root surface, retropreparation into the canal system can commence. Matisson et al recommended a 3-mm depth for all retropreparations, and historically, burs were utilized for this purpose (Wuchenich et al). Burs in high-speed handpieces have largely been replaced by surgical ultrasonic tips. Several types of surgical ultrasonic tips are available, including diamond-coated tips and those with microprojections; both are equally effective for surgical applications (Liu et al). According to Wuchenich et al, apical preparations with ultrasonic tips followed the direction of the root canal system, had parallel walls, and had a minimum depth of 2.5 mm. Bur preparations, on the other hand, had an average depth of 1 mm and were covered in a smear layer. Additionally, according to Rainwater et al, ultrasonics are no more likely to crack root structure than burs. Overall, ultrasonic retropreparations are associated with more favorable outcomes (de Lange et al). Just as ultrasonics effectively clean the root canal space, they are useful for cleaning the isthmuses often located between canals (Engel and Steiman).

Retrofilling

Practitioners have a variety of retrofill choices available to them, including amalgam, Super EBA [Bosworth], MTA, and the more recently introduced bioactive cements. Amalgam represents the historical retrofill of choice. However, amalgam is associated with more leakage (Torabinejad et al 1995), poorer biocompatibility, more inflammation (Baek et al 2010, 2005), and poorer outcomes (Setzer et al) than newer materials. Super EBA represents an improvement over amalgam in terms of both sealing capabilities (Oynick and Oynick) and toxicity (Keiser et al). Furthermore, outcomes with Super EBA are as favorable as those with newer filling materials including MTA (Song and Kim).

MTA represents the current material of choice for many practitioners. It contains tricalcium silicate, dicalcium silicate, tricalcium aluminate, bismuth oxide, and calcium dihydrate. The grey formulation additionally contains tetracalcium aluminoferrite (Camilleri et al). Upon setting, MTA forms calcium silicate hydrate and calcium hydroxide (Camilleri et al). MTA

provides a marked improvement over older materials. It provides a better apical seal than amalgam, IRM, or Super EBA (Fischer et al), even in the presence of blood (Torabinejad et al 1994). Additionally, MTA promotes cementum and bone coverage, unlike amalgam or Super EBA (Baek et al 2005). Despite its many improvements, MTA possesses several drawbacks, including difficult handling and long setting time (Parirokh and Torabinejad). Newer iterations of MTA promise to address these undesirable properties.

Recently, several bioactive materials have become available to practitioners. They represent an improvement over MTA in terms of handling ease (Damas et al). These bioactive products are composed of calcium silicate and calcium phosphate (Damas et al). They are similar to MTA in terms of both in vitro (Damas et al) and in vivo biocompatibility (Ma et al). Furthermore, they create a mechanical bond to dentin, theoretically providing an excellent seal (Damas et al). Retrofiling materials are compared in Table 8-1.

Table 8-1 Comparison of retrofill materials

Material	Biocompatible	Seals well	Ease of use
Amalgam	No	No	Yes
Super EBA	Moderate	Yes	Yes
MTA	Yes	Yes	No
Bioactive cements	Yes	Unknown	Yes

While the majority of endodontic surgeries involve retropreparation, several authors contend that this practice is unnecessary for both MTA apical plugs and GP fillings. Andelin et al found that resection of set MTA does not diminish its sealing abilities. Harrison and Todd found that the resection of root ends with well-condensed GP does not adversely affect the seal, though the same is not true of silver point fillings. Kaplan et al advocated that GP fillings should be burnished after resection. Minnich et al suggested that even burnishing of well-sealed fillings is unnecessary, though poorly condensed restorations may benefit from burnishing.

Suturing

Sutures immobilize the surgical flap and promote healing by primary intention (Harrison). Flaps should be reapproximated without tension to avoid impairment of flap circulation. Monofilament synthetic sutures are recommended for this purpose, as they are less traumatic, discourage inflammation, and promote less bacterial adhesion than multifilament sutures (Velvart and Peters). Smaller suture gauges, particularly the 6-0 and 8-0 sutures, are recommended to avoid tissue necrosis and minimize scarring. The suture technique selected, either simple-interrupted or continuous, should be appropriate for the clinical situation. Sutures should be removed 48 to 96 hours following surgery (Velvart and Peters).

Grafts and membranes

Bone graft materials and membranes are available for practitioners who wish to implement them during surgical intervention. Grafts fall under several broad categories, including osteogenic, osteoinductive, and osteoconductive grafts (Bashutski and Wang). Osteogenic grafts contain cells capable of depositing bony matricies. Osteoinductive grafts release mediators that signal the host to induce new bone formation. Lastly, osteoconductive grafts serve as scaffolds on which bone can grow. Grafts can be further categorized by source, including autogenous grafts, allografts, xenografts, and alloplasts (Bashutski and Wang). Autogenous grafts are derived from the host, allografts from a genetically dissimilar member of the same species, and xenografts from another species altogether. On the other hand, alloplasts are inert materials that serve as a scaffold for new bone but are not derived from biologic donors. Graft types are described in Fig 8-21.

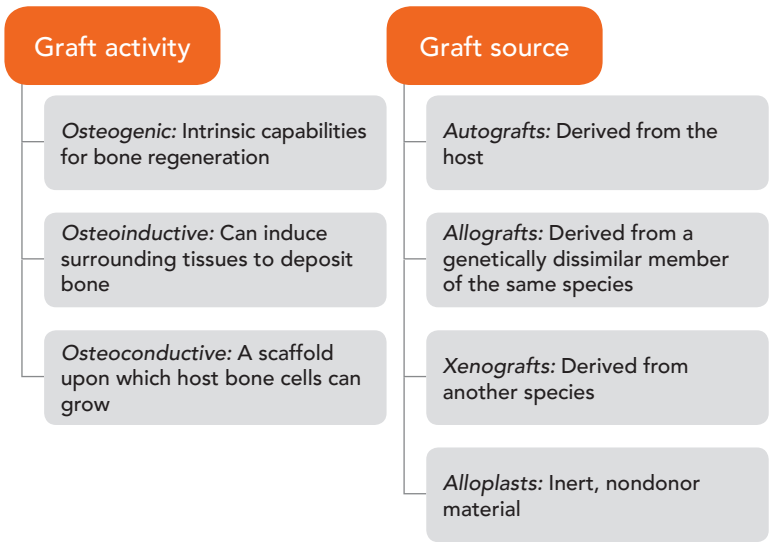

Fig 8-21 Two means by which grafts can be categorized (Bashutski and Wang).

Bone grafts are often used with membranes and are available in both resorbable and nonresorbable forms (Bashutski and Wang). The concomitant use of both products is often referred to as *guided tissue regeneration*, a procedure that facilitates tissue regeneration to its original form (Caffesse and Quinones). This procedure is often used in periodontal surgery and is effective in the treatment of two- and three-wall intrabony defects and Class II furcation invasions (Caffesse and Quinones). For the majority of apical surgical procedures with loss of only one cortical plate, the use of membranes or grafts provides no advantages over traditional surgical techniques (Garrett et al). However, it may improve outcomes for treatment of through-and-through lesions (Lin et al 2010), large periapical defects (Tsesis et al 2011), or apicomarginal defects (Douthitt et al). For these indications, resorbable membranes are favored over nonresorbable membranes, and bone grafts may not be necessary except to hold the membrane in place (Tsesis et al 2011).

Postoperative management

Discomfort following surgery is often of short duration, with its maximum intensity in the early postoperative period (Chong and Pitt Ford), and is correlated with preoperative pain (Tsesis et al 2003). A long-acting postoperative anesthetic, such as bupivacaine, can markedly reduce postoperative discomfort in the initial period (Hargreaves and Keiser). Additionally, for a large number of patients, nonprescription analgesics are adequate and effective for management of postoperative pain (Chong and Pitt Ford). Antibiotics are not believed effective in managing postoperative discomfort.

To aid in soft tissue healing after surgical procedures, Kim et al recommended the use of both pre- and postoperative chlorhexidine rinses to reduce soft tissue inflammation. Shahan et al advised avoidance of chlorhexidine for 48 hours immediately postoperatively as it may reduce fibroblastic attachment to root surfaces and negatively affect tensile wound strength.

Surgical complication management

A common complication encountered in apical surgery on maxillary posterior teeth is a perforation of the maxillary sinus (Rud and Rud) because of the close proximity of the maxillary posterior teeth to the sinus (Von Arx et al 2014). Thankfully, data indicate that these areas tend to self-repair, regardless of size, with a limited bony covering and a fibrous scar (Tataryn et al). Furthermore, the use of resorbable membranes does not appear to improve osseous repair (Tataryn et al). Postoperatively, the use of "sinus precautions," including avoidance of nose blowing and other activities involving forceful air production, as well as the prescription of a decongestant, is recommended whenever a sinus exposure is observed or suspected (Lin et al 1985). The prophylactic prescription of antibiotics is controversial. While Kim et al recommends this practice, Lin et al (1985), Rud and Rud, and Walton disagree.

The mandibular posterior teeth lie in close proximity to the inferior alveolar nerve canal space (Denio et al). Several strategies are available to practitioners to aid in avoiding this structure, including preoperative CBCT to assess for of proximity (Kovisto et al), careful vertical incision placement, and grooving the bone to stabilize retractor placement (Moiseiwitsch). That being said, sensory disturbance of a variable duration in the lower lip is evident in approximately 20% of all patients following mandibular molar surgery (Wesson and Gale). Thankfully, only 1% of patients retain a permanent deficit.

Surgical healing

Andreason and Rud characterized healing after surgical intervention into three main types: healing with either reformation of the PDL or ankylosis and little inflammation, healing with a fibrous scar, or moderate to severe inflammation without the presence of scar tissue. Radiographic findings associated with scar tissue formation include reduction in size of the apical radiolucency, formation of an irregular outline, and angular extension into the PDL space (Andreason and Rud). Regardless of outcome, healing progresses through several stages, including clotting and inflammation, epithelial healing, connective tissue healing, maturation, and remodeling (Harrison).

Harrison and Jurosky (1991a) described the common histologic findings throughout these stages (Fig 8-22). Twenty-four hours postoperatively, they noted a thin epithelial seal, evidence of blood clotting, and a predominantly polymorphonucleocyte (PMN) infiltrate. Furthermore, necrosis of the periostum was evident (Harrison and Jurosky 1991b). Forty-eight hours postoperatively, Harrison and Jurosky (1991a) observed a multilayered epithelial seal, early type III collagen production, and macrophages replacing PMNs as the dominant inflammatory cell. Seventy-two hours postoperatively, collagen production continued (Harrison and Jurosky 1991a). Four days postoperatively, the clot was replaced by granulation tissue, type I collagen production began (Harrison and Jurosky 1991a), and osteocyte proliferation from the endosteum was noted (Harrison and Jurosky 1991b). Two weeks postoperatively, woven bony trabeculae occupied 80% of the wounded area, and new periosteum was evident (Harrison and Jurosky 1991b). Furthermore, normal sulcular epithelium was noted at this time (Harrison and Jurosky 1991a). Twenty-eight days postoperatively, the bony wound was populated by maturing bony trabeculae (Harrison and Jurosky 1991b).

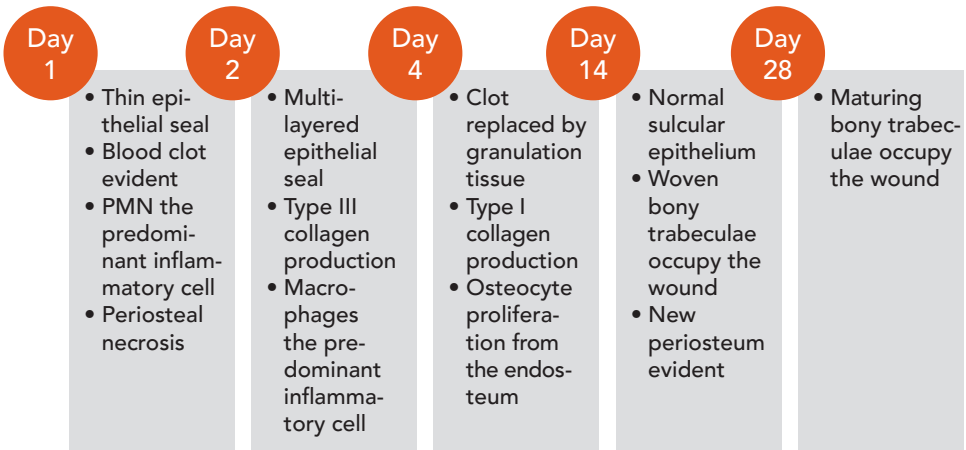

Fig 8-22 Harrison and Jurosky's (1991a, 1991b) observations of postoperative healing after endodontic surgical intervention. PMN, polymorphonucleocyte.

Unconventional surgical approaches

While the majority of practicing endodontists regularly perform apical microsurgery, two often-unmentioned surgical procedures within the scope of the specialty include intentional replantation and autotransplantation. *Intentional replanatation* was defined by Grossman as "removal of a tooth and its almost immediate replacement, with the object of obturating the canals apically while the tooth is out of the socket." While this procedure is not indicated when conventional endodontic therapy or apical surgery provides a reasonable treatment choice, it may be a reasonable alternative to extraction in the presence of failed nonsurgical endodontics with unfavorable anatomical features for traditional apical surgery (Cotter and Panzarino). Key treatment recommendations for this procedure include atraumatic extraction, minimal handling of the root surface, an extraoral time of less than 10 minutes, biocompatible apical fills, and nonrigid postoperative splinting (Kratchman). Bender and Rossman reported a success rate of 81% for 31 intentional replantation cases.

Autotransplantation provides a replacement option for missing teeth under proper circumstances. Though often used to replace lost teeth in the early permanent dentition (Cardona et al), a case report of autotransplantation of an incompletely formed, unerupted mandibular premolar into an artificial socket in a patient with agenesis of a maxillary lateral incisor was recently published (Intra et al). Considerations for this therapy include patient age, occlusion, space available in the recipient site, and the anatomy of the donor tooth (Jonsson and Sigurdsson). Frequently, mandibular premolars are selected as donor teeth due to favorable anatomical features (Jonsson and Sigurdsson). Ideally, the donor tooth should have a partially developed root with an open apex (Lundberg and Isaksson). For successful autotransplantation, one must atraumatically extract the donor tooth to preserve the Hertwig epithelial root sheath and maintain the PDL cells for the recipient site (Andreasen et al). Successful autotransplantation permits development of the donor tooth and continued growth of the alveolar bone and associated soft tissue (Lundberg and Isaksson).

Vital Pulp Therapy

Vital pulp therapy serves to maintain the vitality of the radicular pulp tissue in cases of trauma, deep caries, iatrogenic damage, or in the presence of developmental anomalies. Maintaining pulp vitality, particularly in permanent teeth with open apices, is the treatment of choice as it promotes completion of root development. This process is often referred to as *apexogenesis* (AAE glossary). Several pulp therapies are available to practitioners to accomplish this goal, including pulp capping and both partial and full pulpotomies. This section will cover both of these treatment modalities.

Pulp capping

Pulp capping, according to the AAE glossary, involves "treatment of the exposed vital pulp by sealing with a dental material to facilitate the formation of reparative dentin and maintenance of the vital pulp." According to Bergenholtz, pulp caps should be placed as soon as an exposure occurs for the best prognosis. Other prognostic factors include patient age; exposure site, namely whether an axial or occlusal exposure occurs; and pulp capping material (Cho et al).

Several pulp cap materials are available for practitioners, including MTA, CH, and Biodentine [Septodont]. Histologic responses to MTA and CH are similar (Iwamoto et al). The typical histologic response to both is coagulative necrosis followed by hard tissue bridging (Fig 8-23) (Torabinejad and Parirokh). These similar responses are likely observed because MTA forms CH upon setting (Parirokh and Torabinejad). Biodentine has recently been advocated as an alternative to both materials. Nowicka et al found that Biodentine-capped pulps had a similar histologic appearance to those treated with MTA. Conversely, composite restorative materials and their bonding agents are unacceptable pulp-capping agents. Silva et al found that leakage occurs when these materials are used due to bonding failures.

Recent research highlights the ability of additional medicaments to facilitate healing after pulp capping procedures. Karanxha et al found that simvastatin induced differentiation of human dental pulp cells when placed over pulp exposures. Furthermore, the same authors found that simvastatin also promoted odontogenesis. Additional research may push this medication from the realm of cholesterol treatment to vital pulp therapy.

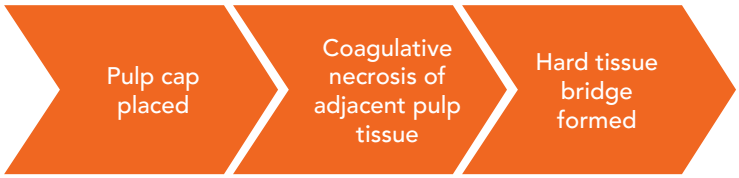

Fig 8-23 Healing of pulp tissues following pulp-capping procedures with MTA or CH (Torabinejad and Parirokh).

Pulpotomy

Pulpotomy treatment involves removal of the coronal portion of a vital pulp to preserve the vitality of the radicular pulp and can be either complete or partial, depending on the extent of coronal pulp tissue removed (AAE glossary). Pulpotomy treatment facilitates continued root development in immature permanent teeth (Cvek), or may offer effective emergency care in mature teeth until definitive care can be delivered. Asgary and Eghbal found that pain relief following pulpotomy treatment was greater than with a full pulpectomy in vital cases. However, this treatment is not without risk. Cvek found that common postoperative complications included calcification, internal root resorption, and complete pulpal necrosis.

After removing pulp tissue with a water-cooled high-speed handpiece (Cvek), several medicaments and restorative materials are available to cover the pulp tissue remaining in the roots. Historically, formocresol was often used during pulpotomy treatment, although this material is problematic for several reasons. Pashley et al found that formaldehyde enters the systemic circulation following formocresol pulpotomies, and Block et al found that formaldehyde-containing medications antigenically alter tissues and stimulate cell-mediated responses. The AAE recommends avoiding formocresol and other products containing formaldehyde or paraformaldehyde, as they are both unsafe and ineffective. Consequently, the continued use of this material in future care is unjustifiable.

Cvek used CH to cover pulp tissue stumps in his original work and found that it achieved predictable results. Bakland recommended instead that MTA be used for this purpose. Aguilar and Linsuwanont found that both materials provided predictable treatment results. Lastly, research by Keswani et al found that platelet-rich fibrin was as effective a pulpotomy restorative material as MTA. A case illustrating an MTA pulpotomy is depicted in Fig 8-24.

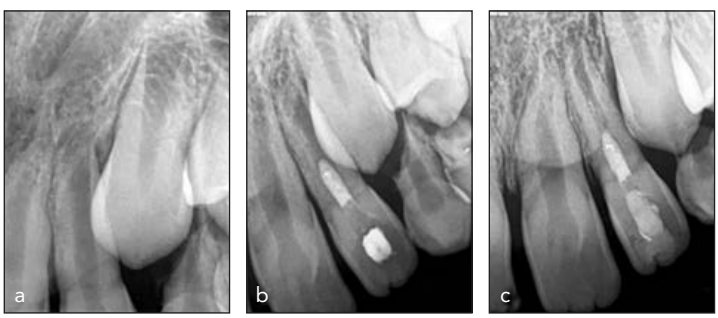

Fig 8-24 Preoperative *(a)* and immediate postoperative *(b)* radiographs of an MTA pulpotomy in a 10-year-old girl. *(c)* A 6-month follow-up radiograph displays root thickening and continued apical closure.

Apexification

Pulpal necrosis in an immature permanent tooth poses decided treatment challenges for practitioners. While a fully formed root apex instrumented properly creates a sufficient apical barrier for obturation, the open apices of incompletely developed roots require additional treatment to create a proper barrier. The treatment aimed at barrier creation is referred to as *apexification* (AAE glossary).

The first apexification technique described in the literature involved placement of CH into the root canal system allowing the Hertwig epithelial root sheath to generate an apical barrier (Frank). Kleier and Barr found that barriers typically form in an average of 1 year with a standard deviation of 7 months. In a review of the literature, Shabahang found that barrier formation may take as long as 24 months to form. Finucane and Kinirons recommended that CH be changed either every 3 months, every 6 to 8 months, or never during the apexification process. A CH apexification case is illustrated in Fig 8-25.

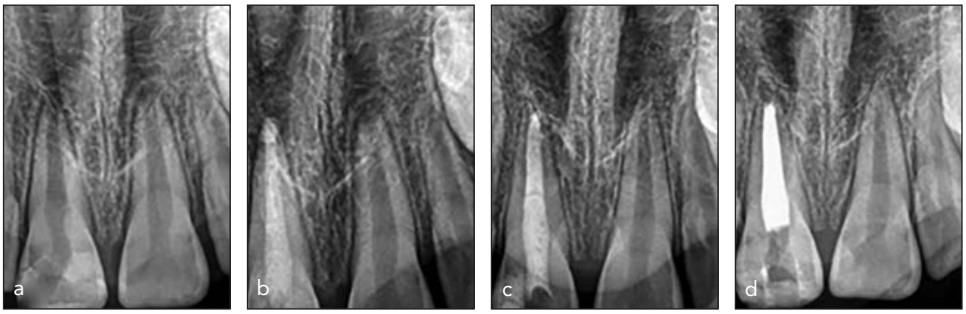

Fig 8-25 A CH apexification case. *(a)* A 12-year-old boy presented with a necrotic maxillary right central incisor with an open apex. A CH hard pack was placed *(b)*, and a calcific barrier formed in 9 months *(c)*. *(d)* Obturation of the canal space was completed with GP once barrier formation was complete. (Courtesy of Dr John Dresser, White River Junction, Vermont.)

Though CH apexification achieves relatively predictable results (Jeeruphan et al), long-term CH apexification has been associated with an increased risk of cervical root fracture (Cvek). According to Cvek, the incidence of cervical root fracture following CH apexification is between 28% and 77%, depending on the extent of root development. This may result from the effect of CH on the physical properties of teeth. Andreason et al found that extended CH application in sheep teeth significantly weakened root structures.

To combat the problematic effects of CH on dental structures, practitioners proposed the development of artificial apical barriers. Given MTA's biocompatibility and ability to seal in the presence of blood (Torabinejad and Parirokh), it was a natural choice for this application (Rafter). The use of artificial barriers also significantly decreases treatment times for patients. Simon et al found that even single-visit apexification treatment with MTA was possible. Witherspoon and Ham found that outcomes for MTA apexification completed in a single visit or over multiple visits were statistically equivalent. When MTA apexification is compared with CH apexification, outcomes are equivalent (Chala et al) or better (Jeeruphan et al). Consequently, MTA barrier placement offers an excellent alternative for patients in which follow-up may be problematic. An MTA apexification case is illustrated in Fig 8-26.

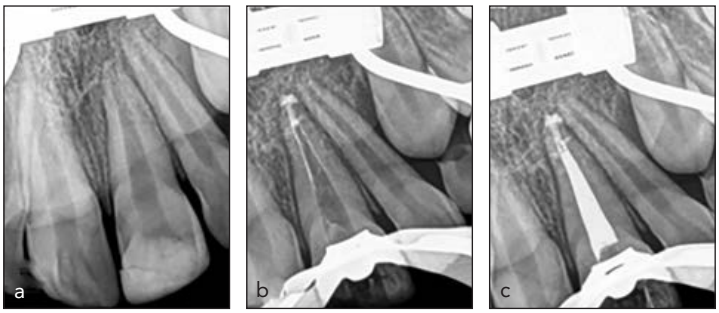

Fig 8-26 An MTA apexification case. *(a)* A 10-year-old girl presented with a necrotic maxillary left central incisor. An apical MTA plug was placed *(b)*, and GP filling was inserted *(c)* at the same visit.

Though apexification treatment preserves an immature permanent tooth, the treatment modality is limited by its inability to increase root thickness. Consequently, apexified teeth are more likely to experience root fractures than fully mature permanent teeth (Cvek). As a result of this risk, several strategies for reinforcement of apexified teeth have been suggested in the literature. Katebzadeh et al found that metal posts, opaque posts, and resin-bonded posts all improved fracture resistance over unrestored teeth. Goldberg et al found that resin-modified glass ionomers reinforced tooth structure.

Regenerative Endodontics

Necrotic immature permanent teeth pose specific challenges to practitioners, including the risk for fracture of thin root walls (Cvek). Consequently, alternatives to the apexification procedure have been investigated for a number of years. *Regenerative endodontics* refers to the "biologically based procedures designed to physiologically replace damaged tooth structures including dentin and root structures, as well as cells of the pulp-dentin complex" (AAE glossary). While early work by Nygaard-Ostby and Hjortdal brought this idea to the endodontic world, a case report by Banchs and Trope reignited excitement surrounding this treatment modality. According to Law, regenerative endodontics requires a necrotic pulp, an immature apex, no need for a post and core restoration, and parental compliance. Law asserted that the etiology of pulp necrosis is unimportant when considering this treatment modality. Recently, Colombo et al found that rapid attenuation of inflammation is additionally necessary to facilitate repair. With proper case selection and adherence to scientifically backed treatment protocols, regenerative endodontics may provide a reasonable alternative to apexification.

Regenerative endodontics is, in essence, a tissue engineering procedure that facilitates development of dental hard tissues. According to Langer and Vacanti, requirements for tissue engineering include stem cells, growth factors, and a scaffold (Fig 8-27). Huang suggested that the scaffold and growth factors in regenerative endodontics are derived from the dentin, fibrin clot, and sterilized pulp tissue remnants. Lovelace et al showed that the mesenchymal stem cells that infiltrate the canal space originate in the apical papilla.

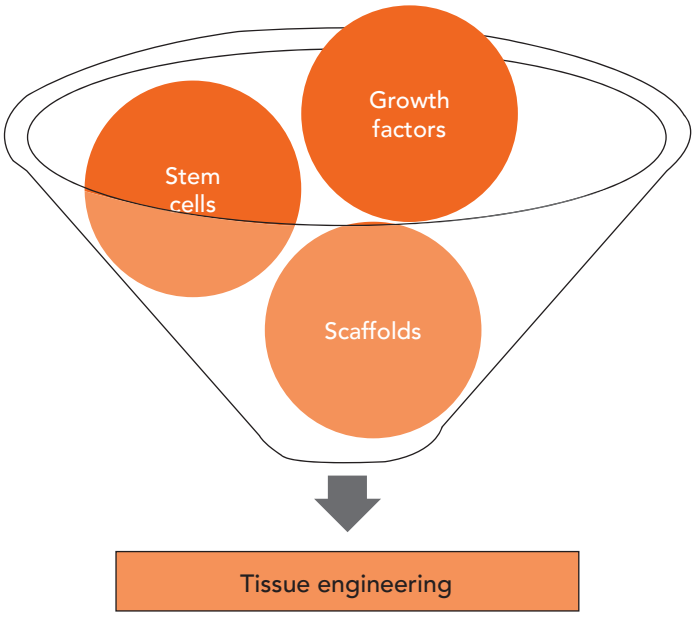

Fig 8-27 The three factors necessary for tissue engineering to occur (Langer and Vacanti).

Regenerative endodontic techniques facilitate both disinfection of the root canal space and ingrowth of stem cells capable of regenerating dental hard tissues. On access into the necrotic pulp space, Hargreaves et al recommended minimal instrumentation and the use of gentle irrigation to begin disinfection. Nagata et al found that the microbial profile of infected immature permanent teeth is similar to that found in mature permanent teeth. Consequently, irrigants like CHX and sodium hypochlorite are effective antimicrobials in these cases. Despite their efficacy, practitioners must balance antimicrobial efficacy and toxicity to stem cells. Trevino et al found that CHX reduced stem cell viability. Consequently, avoidance of CHX as an irrigant or intracanal medicament is indicated. High concentrations of sodium hypochlorite also reduce stem cell viability and differentiation (Martin et al 2014). Martin et al (2014) found that by using a lower concentration of 1.5% sodium hypochlorite solution followed by a rinse with 17% EDTA, cell viability improved. Other authors recommend the use of EDTA in irrigation protocols, including Trevino et al and Pang et al. Galler et al suggested that EDTA promotes release of growth factors in dentin. Consequently, EDTA aids in two of the three requirements for tissue engineering, namely the maintenance of stem cells and growth factor release, and its use is indicated in regenerative endodontic procedures.

Following minimal instrumentation and irrigation, the placement of an intracanal medicament is indicated for a period of 2 to 4 weeks (Law). A similar balance between antimicrobial efficacy and stem cell toxicity must be achieved with medicaments as with irrigants. Hoshino et al found a triple antibiotic paste, combining ciprofloxacin, metronidazole, and minocycline, eradicated endodontic microbes in vitro. Windley et al confirmed these

findings in vivo in dogs. Clinical issues with this formulation, including staining of dental tissues attributed to minocycline, a tetracycline derivative (Kim et al), led to research into other antibiotic formulations. Thibodeau and Trope advocated a substitution of cefaclor for minocycline. Iwaya et al recommended a double antibiotic paste of metronidazole and ciprofloxacin. Recently, Nosrat et al (2013) recommended Augmentin [GlaxoSmithKline] for disinfection purposes.

Though antibiotic pastes are effective antimicrobials (Ordinola-Zapata et al), their negative effect on stem cell viability makes their use in regeneration protocols problematic (Ruparel et al). A promising alternative to these formulations is CH. Nagata et al found no difference in bacterial reduction between triple antibiotic pastes and CH. Ruparel et al found that while both double and triple antibiotic pastes in clinically useful concentrations reduced stem cell viability, CH did not. Furthermore, Althumairy et al found that CH promoted stem cell survival and proliferation. Consequently, CH may be better suited than antibiotic pastes as intracanal, interappointment medicaments for regenerative endodontic therapy.

Disinfection of the root canal system by irrigants and intracanal medicaments precedes the introduction of stem cells, growth factors, and scaffolds into the root canal space. This is achieved by overinstrumentation into the apical papilla to stimulate bleeding (Banchs and Trope). Ideally, blood clots below the CEJ and is covered with an MTA fill. MTA is the cap material of choice; Mente et al found that it facilitates migration and proliferation of apical papilla stem cells. The blood clot beneath the MTA plug serves as the scaffold for migration of stem cells and development of hard tissues (Hargreaves et al). Recently, additional scaffolds have been investigated, including cross-linked collagen (Yamauchi et al 2011b), polylactic acid (Chandrahasa et al), and platelet-rich plasma (Jadhav et al). Petrino et al recommended placement of a collagen matrix such as CollaPlug [Zimmer] to prevent overextension of the MTA.

A successful outcome after regenerative endodontic treatment includes both elimination of apical periodontitis and increases in both root length and width (Lenzi and Trope). Flake et al recommended that radiographic follow-up should include measurement of the radiographic root area. Recent prognostic investigations indicate that regenerative endodontics may predictably achieve these outcomes (Jeeruphan et al) (Fig 8-28).

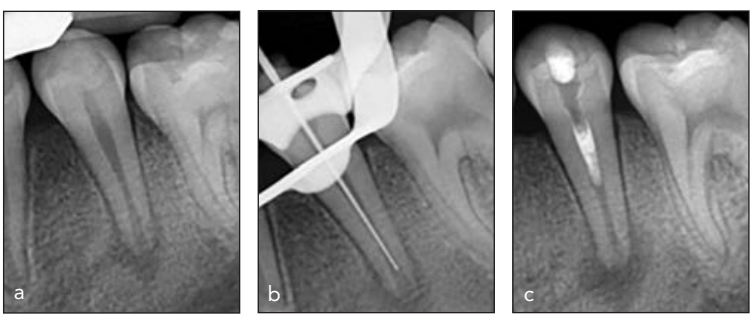

Fig 8-28 *(a)* A regenerative endodontic procedure on the mandibular left second premolar in a 12-year-old boy with a history of a fractured dens evaginatus resulting in pulpal necrosis with symptomatic apical periodontitis. *(b)* Minimal instrumentation to the WL was performed during the first visit, and bleeding into the canal space was achieved during the second visit. *(c)* An MTA plug was placed over the blood clot.

Several authors have investigated the nature of the intracanal hard tissues formed following regenerative endodontic procedures. Wang et al found that they resemble cementum, PDL, and bone. Likewise, Becerra et al found fibrous connective tissues, cementum, and PDL. Yamauchi et al (2011a) found that hard tissue is found both associated with the dentin and separately in the canal space. Similarly appearing tissues are produced when platelet-rich plasma scaffolds are used (Martin et al 2013, Torabinejad et al).

Though promising, regenerative endodontic therapy is not without risk (Fig 8-29). Nosrat et al (2012) found that postoperative issues may include caries, short roots, discoloration, and canal calcification. The discoloration can result from either the use of minocycline (Kim et al) or the MTA plug (Felman and Parashos). Further study is indicated to mitigate these risks.

A recent survey by Manguno et al indicates that dentists are becoming more accepting of regenerative endodontics, and many wish for additional training. Consequently, it behooves the endodontic specialist to both maintain current knowledge of this therapy and educate their referral base regarding treatment possibilities.

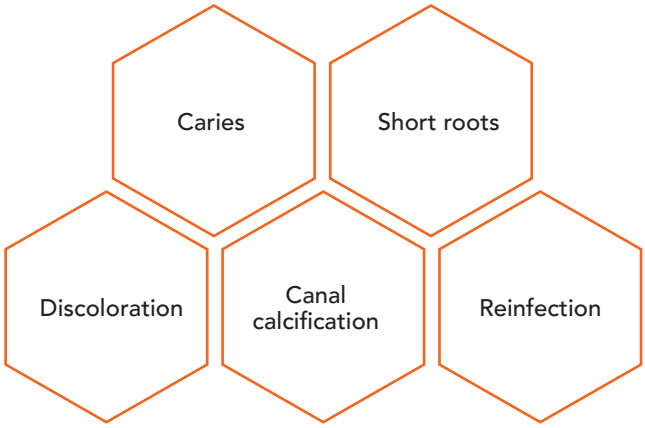

Fig 8-29 Potential complications following regenerative endodontic therapy.

Internal Bleaching

Internal bleaching techniques can effectively eliminate dentinal staining caused by intrapulpal hemolysis and byproducts of endodontic infections (Glockner et al). Walton et al found successful removal of stains caused by tetracycline use during dentinogenesis. Furthermore, this technique may be effective for stains caused by MTA (Belobrov and Parashos).

Spasser originally described the sodium perborate walking bleach technique. Nutting and Poe recommended the addition of Superoxol [Miltex], a 30% hydrogen peroxide solution, to sodium perborate for added efficacy. However, Madison and Walton found that both Superoxol and heat were associated with an increased risk of resorption. Friedman recommended that water be used in place of Superoxol to minimize these risks. Rotstein et al advocated the placement of a 2-mm base at the CEJ to further reduce the risk of resoprtion, as the presumed pathway for irritants to create the feared invasive cervical root resorption would be through exposed tubules at the level of the junctional epithelium and CEJ.

Follow-Up Care

The majority of published studies, including those by Friedman and Mor and also Orstavik, indicate that patients receiving nonsurgical endodontic therapy should have, at minimum, a 1-year postoperative follow-up examination. Orstavik recommended using a standardized technique, particularly the periapical index (PAI) score, for determining radiographic healing. One year, though, may prove insufficient for definitive determination of success or failure, as many lesions require additional time to heal. Murphy et al found that 70% of lesions take more than 1 year to heal. Molven et al echoed these findings by suggesting that lesions may take between 10 and 20 years to heal. For lesions unhealed at the 1-year mark, Yu et al found that 57% improved over time, while 31% worsened. Predictors for worsening included recall lesion size, pain on biting at recall, history of a postobturation flare, and nonideal root canal length.

Just as nonsurgical endodontic treatment should receive follow-up care, so should surgical treatment. According to Rud et al, surgical patients should be examined 1 year postoperatively. Echoing Rud et al's statements, Song et al found no differences in outcomes when teeth were examined 1 or 4 years postoperatively. Rubenstein and Kim found that, for those teeth healed at early examination periods, the periapical status remained stable when followed for several years.

Restoration of Endodontically Treated Teeth

Definitive restorative treatment following endodontic therapy is essential. The placement of a well-sealed restoration prevents percolation of oral fluids and bacteria around the obturation material. Swanson and Madison found that oral fluids reached the apex of endodontically treated teeth in as little as 3 days with exposure to the oral environment. However, Torabinejad et al found that leakage down the root canal space required 3 weeks. These findings illustrate the necessity of both temporary and permanent restorations.

Restorative treatment is also indicated because endodontic therapy may affect the physical properties of the teeth, including stiffness and moisture content. These alterations may make teeth more susceptible to fracture. Reeh et al found that endodontic therapy reduced tooth stiffness by 5%, exclusively related to the access opening. Restorative procedures resulted in further loss of stiffness by as much as 60% with preparation across both marginal ridges. Helfer et al found that the calcified tissues of pulpless teeth contain 9% less moisture than healthy teeth. Though stiffness and moisture content is affected by endodontic treatment, a review by Cheron et al concluded that dental hardness is not. Regardless of these findings, protection of the teeth with adequate restorations is essential to mitigate the effects of endodontic therapy on the physical properties of teeth.

Restorative care is not only necessary to mitigate the effects of endodontic treatment on dental physical properties, but also because outcomes of endodontically treated teeth are significantly influenced by restorative care (Fig 8-30). Chugal et al demonstrated a significant reduction in failures in permanently versus temporarily restored teeth, again illustrating the importance of definitive restorative care. The type of definitive restoration placed can influence treatment outcomes based on tooth type. Sorensen and Martinoff found that outcomes of pulpless maxillary and mandibular premolars and molars were improved

by full coronal coverage. Outcomes of anterior teeth, however, were not significantly improved by full-coverage restorations.

Restorative treatment must respect the biologic width, and full-coverage restorations should encompass a ferrule of tooth structure. Restorations that violate either principle may cause early periodontal or restorative failures. Biologic width, defined by Gargiulo et al as the minimally acceptable distance between the crown margin and alveolar crest, should total 3 mm. The biologic width comprises the combined gingival sulcus depth, epithelial attachment, and connective tissue attachment (Fig 8-31). Restoring clinicians should maintain this buffer between restoration margins and the alveolar bone. Violation of the biologic width leads to periodontal inflammation and bone loss. The ferrule refers to a 1.5- to 2-mm circumferential vertical wall of tooth structure encompassed by full-coverage restorations. This feature, described in a review by Juloski et al, decreases the fracture rate of endodontically treated teeth by providing both retention and resistance of the crown to displacement. Respecting both the biologic width and the ferrule effect can dramatically improve restorative outcomes of endodontically treated teeth.

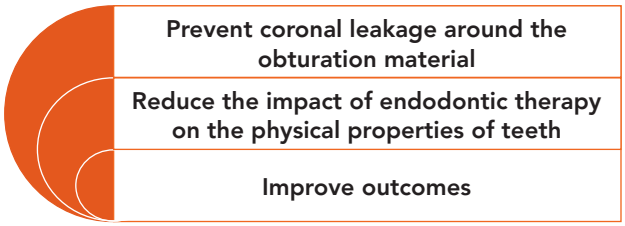

Fig 8-30 Common reasons cited to restore endodontically treated teeth.

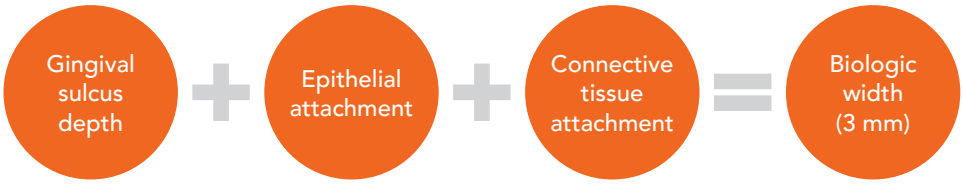

Fig 8-31 The anatomical features that contribute to the biologic width. This is the distance that the restoring dentist should maintain between the restoration margin and the alveolar bone (Gargiulo et al).

Posts

Posts enhance retention of the coronal restoration and may be used in the restorative treatment of endodontically treated teeth. Posts should either equal the length of the clinical crown or measure two-thirds of the root length, whichever is longest (Shillingburg). Furthermore, posts should measure no wider than the size of a no. 3 Gates Glidden drill to prevent excessive dentin thinning (Kuttler et al). When placing a post, rubber dam isolation should always be used, as Goldfein et al demonstrated that outcomes are significantly influenced by their use.

Posts should be used only when necessary as they neither improve the strength of endodontically treated teeth nor long-term outcomes. Their weakening effects may result from the destruction of tooth structure caused by post preparations. Katz and Tamse found that

when post spaces were prepared in the buccal roots of premolars, the remaining dentin thickness was often less than 1 mm. Sagsen et al found that fiber post systems, theoretically expected to improve root strength, did not reinforce the endodontically treated teeth under study. Just as posts do not buttress endodontically treated teeth, they do not improve long-term outcomes. In a clinical study of teeth restored with posts, Sorensen and Martinoff found that intracoronal reinforcement did not significantly increase the clinical success rate of endodontic therapy. Doyle et al (2007) found that placement of a post actually decreased the success rates of endodontically treated teeth.

Not only do posts fail to improve tooth strength or outcomes of endodontically treated teeth, the removal of root canal filling material for their placement increases leakage of the obturation material. Abramovitz et al found that leakage was significantly increased in roots prepared for posts versus intact root canal fillings. Mattison et al found that 5 to 7 mm of remaining GP exhibited significantly less leakage than 3 mm. Consequently, they recommended that at least 5 to 7 mm of GP remain in the root canal space when posts are to be placed. Common issues associated with post-retained restorations are summarized in Fig 8-32.

Fig 8-32 Often-reported issues associated with post placement in endodontically treated teeth.

Despite the associated risks, post placement may be necessary to improve retention of coronal restorations in certain cases. Controversy exists regarding both the timing and the method of preparing space for posts. Though Bourgeois and Lemon found no significant difference between initial and delayed preparation, Fan et al found that delayed post preparations resulted in greater leakage than those prepared immediately. Madison and Zakariasen found no differences in leakage when post spaces were prepared with reamers, heat, or chloroform on files. Mattison et al found that mechanical and thermal post preparations resulted in significantly less leakage than those prepared with chloroform. Without a clear consensus in the literature, it is clear that the timing and method of post placement should be at the discretion of the provider.

Endodontic treatment versus implants

Advances in implant technology provide dentists with a predictable option for replacing missing teeth. Implants provide patients and clinicians with an excellent option when faced with heroic and risky dental treatment. Studies show that both implants and endodontic

treatment provide satisfactory results for patients. Gatten et al found high satisfaction rates in patients having undergone either implant therapy or root canal therapy.

As both treatment options are agreeable to patients, treatment decisions should consider the prognosis of both therapies. In a systematic review, Iqbal and Kim found no differences in survival between root canal therapy and implant-retained restorations. These results echo those of Doyle et al (2006), who found no differences between the failure rates of matched pairs of endodontically treated and restored teeth and single-unit implants. The absolute failure rate of both therapies was 6.1%. Several factors influence the prognosis of both treatment options. Doyle et al (2007) found a significant increase in the failure rate of both implants and endodontically treated teeth in smokers. An increase in failures, though nonsignificant, was found in diabetic patients. Interestingly, however, the presence of an endodontically treated tooth next to an implant did not alter the success rate of either treatment modality.

As success rates and reported patient satisfaction are similar for both treatment modalities, the decision to restore or extract a compromised tooth must be based on other factors. The AAE states that, when considering implant therapy or endodontic treatment, the decision to proceed with either treatment option must be based on factors other than outcome. Restorative choices must be based on case complexity or the patient's individual health and preferences.

Treatment of cracked teeth

The diagnosis and treatment of cracked teeth can prove vexing for clinicians and patients alike. Cameron described *cracked tooth syndrome*, including both chewing discomfort on biting and release as well as unexplained pain in response to cold. Cracked teeth are often minimally restored or unrestored (Turp and Gobetti). Hiatt found a nearly equal distribution of cracked teeth in unrestored teeth and those with Class 1 and 2 restorations. Seo et al associated cracked teeth with a history of nonbonded restorations, such as amalgams. Hallmarks of cracked tooth syndrome are summarized in Fig 8-33.

Unlike many entities responsible for pulpal symptoms, cracked teeth are often best treated by proper restorative care rather than endodontic therapy. Guthrie and DiFiore found that 89% of patients with cracked teeth treated with full-coverage restorations exhibited symptom elimination following placement of the temporary restoration. In a long-term study, Krell and Rivera found that only 21% of teeth with cracked tooth syndrome that were crowned ever developed irreversible pulpitis or pulpal necrosis. Consequently, in patients presenting with cracked tooth syndrome, referral to the restorative dentist for a full-coverage restoration is the initial treatment of choice.

Fig 8-33 Commonly encountered symptoms associated with cracked tooth syndrome.

Ethics and the Law

Dental professionals are tasked with providing excellent care to patients, all the while upholding stringent ethical and legal standards. The American Dental Association (ADA) code of ethics notes: "Although ethics and the law are closely related, they are not the same. Ethical obligations may and often do exceed legal duties." The ethical principles on which the ADA's code is based include patient autonomy, nonmaleficence, beneficence, justice, and veracity (Fig 8-34). These principles represent the "aspirational goals of the profession" and should guide the practitioner in every patient interaction (ADA).

Fig 8-34 Ethical principles outlined in the ADA Principles of Ethics and Code of Professional Conduct.

Dentists also must abide by both state and federal laws. Every dentist must be familiar with their individual state laws, which can often be obtained from state dental societies. Furthermore, dentists must uphold federal laws, including those that fall under the Occupational Health and Safety Administration (OSHA) and the Department of Health and Human Services. OSHA regulates every workplace and establishes blood-borne pathogen standards, hazard communication regulations, ionizing radiation safety, the reporting of occupational injuries, electrical safety, and establishment of safety routes among other standards. Every dentist shares the responsibility of ensuring that these standards are upheld in their place of business.

Dentists must protect the confidentiality of patient records. This issue becomes increasingly complex with the development of new technology. The Health Insurance Portability and Accountability Act (HIPAA), under the US Department of Health and Human Services, establishes guidelines for the protections of protected health information (PHI) and outlines punishments for violations. Every dentist is responsible for ensuring compliance with these statutes by themselves and their employees.

Bibliography

Local Anesthesia

Aggarwal V, Singla M, Miglani S, Ansari I, Kohli S. A prospective, randomized, single-blind comparative evaluation of anesthetic efficacy of posterior superior alveolar nerve blocks, buccal infiltrations, and buccal plus palatal infiltrations in patients with irreversible pulpitis. J Endod 2011;37:1491–1494.

Aggarwal V, Singla M, Miglani S, Kohli S, Irfan M. A prospective, randomized single-blind evaluation of effect of injection speed on anesthetic efficacy of inferior alveolar nerve block in patients with symptomatic irreversible pulpitis. J Endod 2012a;38:1578–1580.

Aggarwal V, Singla M, Miglani S, Kohli S, Singh S. Comparative evaluation of 1.8 mL and 3.6 mL of 2% lidocaine with 1:200,000 epinephrine for inferior alveolar nerve block in patients with irreversible pulpitis: A prospective, randomized single-blind study. J Endod 2012b;38:753–756.

Corbett IP, Kanaa MD, Whitworth JM, Meechan JG. Articaine infiltration for anesthesia of mandibular first molars. J Endod 2008;34:514–518.

Currie CC, Meechan JG, Whitworth JM, Corbett IP. Is mandibular molar buccal infiltration a mental and incisive nerve block? A randomized controlled trial. J Endod 2013;39:439–443.

Dressman AS, Nusstein J, Drum M, Reader A. Anesthetic efficacy of a primary articaine infiltration and a repeat articaine infiltration in the incisive/mental nerve region of mandibular premolars: A prospective, randomized, single-blind study. J Endod 2013;39:313–318.

Evans G, Nusstein J, Drum M, Reader A, Beck M. A prospective, randomized, double-blind comparison of articaine and lidocaine for maxillary infiltrations. J Endod 2008;34:389–393.

Fernandez C, Reader A, Beck M, Nusstein J. A prospective, randomized, double-blind comparison of bupivacaine and lidocaine for inferior alveolar nerve blocks. J Endod 2005;31:499–503.

Fowler S, Reader A. Is a volume of 3.6 mL better than 1.8 mL for inferior alveolar nerve blocks in patients with symptomatic irreversible pulpitis? J Endod 2013;39:970–972.

Gaffen AS, Haas DA. Retrospective review of voluntary reports of nonsurgical paresthesia in dentistry. J Can Dent Assoc 2009;75:579.

Garisto GA, Gaffen AS, Lawrence HP, Tenenbaum HC, Haas DA. Occurrence of paresthesia after dental local anesthetic administration in the United States. J Am Dent Assoc 2010;141:836–844.

Goldberg S, Reader A, Drum M, Nusstein J, Beck M. Comparison of the anesthetic efficacy of the conventional inferior alveolar, Gow-Gates, and Vazirani-Akinosi techniques. J Endod 2008;34:1306–1311.

Guglielmo A, Drum M, Reader A, Nusstein J. Anesthetic efficacy of a combination palatal and buccal infiltration of the maxillary first molar. J Endod 2011;37:460–462.

Haas DA, Lennon D. A 21 year retrospective study of reports of paresthesia following local anesthetic administration. J Can Dent Assoc 1995;61:319–320, 323–316, 329–330.

Hargreaves KM, Keiser K. Local anesthetic failure in endodontics. Mechanisms and management. Endod Topics 2002;1:26–39.

Kanaa MD, Meechan JG, Corbett IP, Whitworth JM. Speed of injection influences efficacy of inferior alveolar nerve blocks: A double-blind randomized controlled trial in volunteers. J Endod 2006;32:919–923.

Kanaa MD, Whitworth JM, Meechan JG. A comparison of the efficacy of 4% articaine with 1:100,000 epinephrine and 2% lidocaine with 1:80,000 epinephrine in achieving pulpal anesthesia in maxillary teeth with irreversible pulpitis. J Endod 2012a;38:279–282.

Kanaa MD, Whitworth JM, Meechan JG. A prospective randomized trial of different supplementary local anesthetic techniques after failure of inferior alveolar nerve block in patients with irreversible pulpitis in mandibular teeth. J Endod 2012b;38:421–425.

Lin L, Lapeyrolerie M, Skribner J, Shovlin F. Periodontal ligament injection: Effects on pulp tissue. J Endod 1985;11:529–534.

Malamed SF. Handbook of Local Anesthesia, ed 6. St Louis: Mosby, 2013.

McLean C, Reader A, Beck M, Meryers WJ. An evaluation of 4% prilocaine and 3% mepivacaine compared with 2% lidocaine (1:100,000 epinephrine) for inferior alveolar nerve block. J Endod 1993;19:146–150.

Nydegger B, Nusstein J, Reader A, Drum M, Beck M. Anesthetic comparisons of 4% concentrations of articaine, lidocaine, and prilocaine as primary buccal infiltrations of the mandibular first molar: A prospective randomized, double-blind study. J Endod 2014;40:1912–1916.

Poorni S, Veniashok B, Senthilkumar AD, Indira R, Ramachandran S. Anesthetic efficacy of four percent articaine for pulpal anesthesia by using inferior alveolar nerve block and buccal infiltration techniques in patients with irreversible pulpitis: A prospective randomized double-blind clinical trial. J Endod 2011;37:1603–1607.

Reader A, Nusstein J, Drum M. Successful Local Anesthesia for Restorative Dentistry and Endodontics. Chicago: Quintessence, 2011.

Rogers BS, Botero TM, McDonald NJ, Gardner RJ, Peters MC. Efficacy of articaine versus lidocaine as a supplemental buccal infiltration in mandibular molars with irreversible pulpitis: A prospective, randomized, double-blind study. J Endod 2014;40:753–758.

Srinivasan N, Kavitha M, Loganathan CS, Padmini G. Comparison of anesthetic efficacy of 4% articaine and 2% lidocaine for maxillary buccal infiltration in patients with irreversible pulpitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107:133–136.

VanGheluwe J, Walton R. Intrapulpal injection: Factors related to effectiveness. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:38–40.

Whitworth JM, Kanaa MD, Corbett IP, Meechan JG. Influence of injection speed on the effectiveness of incisive/mental nerve block: A randomized, controlled, double-blind study in adult volunteers. J Endod 2007;33:1149–1154.

Wood M, Reader A, Nusstein J, Beck M, Padgett D, Weaver J. Comparison of intraosseous and infiltration injections for venous lidocaine blood concentrations and heart rate changes after injection of 2% lidocaine with 1:100,000 epinephrine. J Endod 2005;31:435–438.

Nonsurgical Root Canal Therapy

Abou-Rass M, Frank AL, Glick DH. The anticurvature filing method to prepare the curved root canal. J Am Dent Assoc 1980;101:792–794.

Adorno CG, Yoshioka T, Suda H. Crack initiation on the apical root surface caused by three different nickel-titanium rotary files at different working lengths. J Endod 2011;37:522–525.

Ahlgren FK, Johannessen AC, Hellem S. Displaced calcium hydroxide paste causing inferior alveolar nerve paraesthesia: Report of a case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96:734–737.

Ahmad IA. Rubber dam usage for endodontic treatment: A review. Int Endod J 2009;42: 963–972.

Ahmad M, Pitt Ford TJ, Crum LA. Ultrasonic debridement of root canals: Acoustic streaming and its possible role. J Endod 1987;13:490–499.

American Association of Endodontists. AAE Position Statement 2010. Dental Dams. http://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/dentaldamstatement.pdf. Accessed 22 December 2015.

American Association of Endodontists. AAE Position Statement 2012. Use of Microscopes and Other Magnification Techniques. Https://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/microscopesstatement.pdf. Accessed 11 February 2016.

American Association of Endodontists. Endodontics Colleagues for Excellence. Use and Abuse of Antibiotics. Chicago: American Association of Endodontists, 2012.

Andreasen JO, Farik B, Munksgaard EC. Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture. Dent Traumatol 2002;18:134–137.

Arias A, Azabal M, Hidalgo JJ, de la Macorra JC. Relationship between postendodontic pain, tooth diagnostic factors, and apical patency. J Endod 2009;35:189–192.

Augsburger RA, Peters DD. Radiographic evaluation of extruded obturation materials. J Endod 1990;16:492–497.

Azim AA, Azim KA, Deutsch AS, Huang GT. Acquisition of anatomic parameters concerning molar pulp chamber landmarks using cone-beam computed tomography. J Endod 2014;40: 1298–1302.

Baca P, Junco P, Arias-Moliz MT, Castillo F, Rodriguez-Archilla A, Ferrer-Luque CM. Antimicrobial substantivity over time of chlorhexidine and cetrimide. J Endod 2012;38:927–930.

Barbin LE, Estrela C, Guedes DF, Spano JC, Sousa-Neto MD, Pecora JD. Detection of para-chloroaniline, reactive oxygen species, and 1-chloro-4-nitrobenzene in high concentrations of chlorhexidine and in a mixture of chlorhexidine and calcium hydroxide. J Endod 2013;39: 664–668.

Barthel CR, Strobach A, Briedigkeit H, Gobel UB, Roulet JF. Leakage in roots coronally sealed with different temporary fillings. J Endod 1999;25:731–734.

Basrani BR, Manek S, Sodhi RN, Fillery E, Manzur A. Interaction between sodium hypochlorite and chlorhexidine gluconate. J Endod 2007;33:966–969.

Baumgardner KR, Taylor J, Walton R. Canal adaptation and coronal leakage: Lateral condensation compared to Thermafil. J Am Dent Assoc 1995;126:351–356.

Baumgartner JC, Mader CL. A scanning electron microscopic evaluation of four root canal irrigation regimens. J Endod 1987;13:147–157.

Beus C, Safavi K, Stratton J, Kaufman B. Comparison of the effect of two endodontic irrigation protocols on the elimination of bacteria from root canal system: A prospective, randomized clinical trial. J Endod 2012;38:1479–1483.

Biggs SG, Knowles KI, Ibarrola JL, Pashley DH. An in vitro assessment of the sealing ability of resilon/epiphany using fluid filtration. J Endod 2006;32:759–761.

Blomlof L, Lindskog S, Hammarstrom L. Influence of pulpal treatments on cell and tissue reactions in the marginal periodontium. J Periodontol 1988;59:577–583.

Buck RA, Cai J, Eleazer PD, Staat RH, Hurst HE. Detoxification of endotoxin by endodontic irrigants and calcium hydroxide. J Endod 2001;27:325–327.

Bui TB, Baumgartner JC, Mitchell JC. Evaluation of the interaction between sodium hypochlorite and chlorhexidine gluconate and its effect on root dentin. J Endod 2008;34:181–185.

Bystrom A, Sundqvist G. Bacteriologic evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy. Scand J Dent Res 1981;89:321–328.

Calt S, Serper A. Time-dependent effects of EDTA on dentin structures. J Endod 2002;28:17–19.

Capar ID, Ozcan E, Arslan H, Ertas H, Aydinbelge HA. Effect of different final irrigation methods on the removal of calcium hydroxide from an artificial standardized groove in the apical third of root canals. J Endod 2014;40:451–454.

Carr GB, Murgel CA. The use of operating microscopes in endodontics. Dent Clin N Am 2004;54:191–214.

Chrepa V, Kotsakis GA, Pagonis TC, Hargreaves KM. The effect of photodynamic therapy in root canal disinfection: A systematic review. J Endod 2014;40:891–898.

Clark-Holke D, Drake D, Walton R, Rivera E, Guthmiller JM. Bacterial penetration through canals of endodontically treated teeth in the presence or absence of the smear layer. J Dent 2003;31:275–281.

Clarkson RM, Podlich HM, Moule AJ. Influence of ethylenediaminetetraacetic acid on the active chlorine content of sodium hypochlorite solutions when mixed in various proportions. J Endod 2011;37:538–543.

Cochran MAM, Miller CH, Sheldrake MA. The efficacy of the rubber dam as a barrier to the spread of microorganisms during dental treatment. J Am Dent Assoc 1989;119:141–144.

Cook J, Nandakumar R, Fouad AF. Molecular- and culture-based comparison of the effects of antimicrobial agents on bacterial survival in infected dentinal tubules. J Endod 2007;33: 690–692.

Costa GE, Johnson JD, Hamilton RG. Cross-reactivity studies of gutta-percha, gutta-balata, and natural rubber latex (*Hevea brasiliensis*). J Endod 2001;27:584–587.

Dai L, Khechen K, Khan S, et al. The effect of QMix, an experimental antibacterial root canal irrigant, on removal of canal wall smear layer and debris. J Endod 2011;37:80–84.

Dalton BC, Orstavik D, Phillips C, Pettiette M, Trope M. Bacterial reduction with nickel-titanium rotary instrumentation. J Endod 1998;24:763–767.

Del Carpio-Perochena AE, Bramante CM, Duarte MA, et al. Biofilm dissolution and cleaning ability of different irrigant solutions on intraorally infected dentin. J Endod 2011;37:1134–1138.

Drake DR, Wiemann AH, Rivera EM, Walton RE. Bacterial retention in canal walls in vitro: Effect of smear layer. J Endod 1994;20:78–82.

Dunlap CA, Remeikis NA, BeGole EA, Rauschenberger CR. An in vivo evaluation of an electronic apex locator that uses the ratio method in vital and necrotic canals. J Endod 1998;24:48–50.

Dutner J, Mines P, Anderson A. Irrigation trends among American Association of Endodontists members: A web-based survey. J Endod 2012;38:37–40.

Eleazer PD, Eleazer KR. Flare-up rate in pulpally necrotic molars in one-visit versus two-visit endodontic treatment. J Endod 1998;24:614–616.

Figini L, Lodi G, Gorni F, Gagliani M. Single versus multiple visits for endodontic treatment of permanent teeth: A Cochrane systematic review. J Endod 2008;34:1041–1047.

Forsberg J. Radiographic reproduction of endodontic 'working length' comparing the paralleling and the bisecting-angle techniques. Oral Surg Oral Med Oral Pathol 1987;64:353–360.

Garofalo RR, Ede EN, Dorn SO, Kuttler S. Effect of electronic apex locators on cardiac pacemaker function. J Endod 2002;28:831–833.

Gesi A, Raffaelli O, Goracci C, Pashley DH, Tay FR, Ferrari M. Interfacial strength of Resilon and gutta-percha to intraradicular dentin. J Endod 2005;31:809–813.

Giardino L, Pontieri F, Savoldi E, Tallarigo F. Aspergillus mycetoma of the maxillary sinus secondary to overfilling of a root canal. J Endod 2006;32:692–694.

Goerig AC, Michelich RJ, Schultz HH. Instrumentation of root canals in molar using the step-down technique. J Endod 1982;8:550–554.

Goldberg F, De Silvio AC, Manfre S, Nastri N. In vitro measurement accuracy of an electronic apex locator in teeth with simulated apical root resorption. J Endod 2002;28:461–463.

Goldberg F, Massone EJ. Patency file and apical transportation: An in vitro study. J Endod 2002;28:510–511.

Gonzalez-Martin M, Torres-Lagares D, Gutierrez-Perez JL, Segura-Egea JJ. Inferior alveolar nerve paresthesia after overfilling of endodontic sealer into the mandibular canal. J Endod 2010;36:1419–1421.

Goodman A, Schilder H, Aldrich W. The thermomechanical properties of gutta-percha. II. The history and molecular chemistry of gutta-percha. Oral Surg Oral Med Oral Pathol 1974;37: 954–961.

Goodman A, Schilder H, Aldrich W. The thermomechanical properties of gutta-percha. Part IV. A thermal profile of the warm gutta-percha packing procedure. Oral Surg Oral Med Oral Pathol 1981;51:544–551.

Grundling GL, Zechin JG, Jardim WM, de Oliveira SD, de Figueiredo JA. Effect of ultrasonics on *Enterococcus faecalis* biofilm in a bovine tooth model. J Endod 2011;37:1128–1133.

Gutarts R, Nusstein J, Reader A, Beck M. In vivo debridement efficacy of ultrasonic irrigation following hand-rotary instrumentation in human mandibular molars. J Endod 2005;31:166–170.

Haapasalo M, Shen Y, Qian W, Gao Y. Irrigation in endodontics. Dent Clin North Am 2010;54: 291–312.

Hand RE, Smith ML, Harrison JW. Analysis of the effect of dilution on the necrotic tissue dissolution property of sodium hypochlorite. J Endod 1978;4:60–64.

Hargreaves KM, Cohen S, Berman LH. Cohen's Pathways of the Pulp. St. Louis: Mosby, 2011.

Harrison JW, Baumgartner CJ, Zielke DR. Analysis of interappointment pain associated with the combined use of endodontic irrigants and medicaments. J Endod 1981;7:272–276.

Hasselgren G, Olsson B, Cvek M. Effects of calcium hydroxide and sodium hypochlorite on the dissolution of necrotic porcine muscle tissue. J Endod 1988;14:125–127.

Herrera M, Abalos C, Lucena C, Jimenez-Planas A, Llamas R. Critical diameter of apical foramen and of file size using the Root ZX apex locator: An in vitro study. J Endod 2011;37:1306–1309.

Ibarrola JL, Chapman BL, Howard JH, Knowles KI, Ludlow MO. Effect of preflaring on Root ZX apex locators. J Endod 1999;25:625–626.

Idzahi K, de Cock CC, Shemesh H, Brand HS. Interference of electronic apex locators with implantable cardioverter defibrillators. J Endod 2014;40:277–280.

Jacobson HL, Xia T, Baumgartner JC, Marshall JG, Beeler WJ. Microbial leakage evaluation of the continuous wave of condensation. J Endod 2002;28:269–271.

Jeansonne MJ, White RR. A comparison of 2.0% chlorhexidine gluconate and 5.25% sodium hypochlorite as antimicrobial endodontic irrigants. J Endod 1994;20:276–278.

Jeger FB, Janner SF, Bornstein MM, Lussi A. Endodontic working length measurement with preexisting cone-beam computed tomography scanning: A prospective, controlled clinical study. J Endod 2012;38:884–888.

Jenkins JA, Walker WA 3rd, Schindler WG, Flores CM. An in vitro evaluation of the accuracy of the Root ZX in the presence of various irrigants. J Endod 2001;27:209–211.

Jensen SA, Walker TL, Hutter JW, Nicoll BK. Comparison of the cleaning efficacy of passive sonic activation and passive ultrasonic activation after hand instrumentation in molar root canals. J Endod 1999;25:735–738.

Kara Tuncer A, Gerek M. Effect of working length measurement by electronic apex locator or digital radiography on postoperative pain: A randomized clinical trial. J Endod 2014;40:38–41.

Kara Tuncer A, Unal B. Comparison of sealer penetration using the EndoVac irrigation system and conventional needle root canal irrigation. J Endod 2014;40:613–617.

Karr AN, Baumgartner JC, Marshall JG. A comparison of gutta-percha and Resilon in the obturation of lateral grooves and depressions. J Endod 2007;33:749–752.

Kenee DM, Allemang JD, Johnson JD, Hellstein J, Nichol BK. A quantitative assessment of efficacy of various calcium hydroxide removal techniques. J Endod 2006;32:563–565.

Kim SK, Kim YO. Influence of calcium hydroxide intracanal medication on apical seal. Int Endod J 2002;35:623–628.

Kobayashi C, Suda H. New electronic canal measuring device based on the ratio method. J Endod 1994;20:111–114.

Kozam G. The effect of eugenol on nerve transmission. Oral Surg Oral Med Oral Pathol 1977;44:799–805.

Krasner P, Rankow HJ. Anatomy of the pulp-chamber floor. J Endod 2004;30:5–16.

Lamers AC, Simon M, van Mullem PJ. Microleakage of Cavit temporary filling material in endodontic access cavities in monkey teeth. Oral Surg Oral Med Oral Pathol 1980;49:541–543.

Law A, Messer H. An evidence-based analysis of the antibacterial effectiveness of intracanal medicaments. J Endod 2004;30:689–694.

Leonardo MR, da Silva LA, Filho MT, Bonifacio KC, Ito IY. In vitro evaluation of the antimicrobial activity of a castor oil-based irrigant. J Endod 2001;27:717–719.

Liang YH, Jiang LM, Jiang L, et al. Radiographic healing after a root canal treatment performed in single-rooted teeth with and without ultrasonic activation of the irrigant: A randomized controlled trial. J Endod 2013;39:1218–1225.

Lin PY, Huang SH, Chang HJ, Chi LY. The effect of rubber dam usage on the survival rate of teeth receiving initial root canal treatment: A nationwide population-based study. J Endod 2014;40:1733–1737.

Lindgren P, Eriksson KF, Ringberg A. Severe facial ischemia after endodontic treatment. J Oral Maxillofac Surg 2002;60:576–579.

Lottanti S, Taubock TT, Zehnder M. Shrinkage of backfill gutta-percha upon cooling. J Endod 2014;40:721–724.

Lozano A, Forner L, Llena C. In vitro comparison of root-canal measurements with conventional and digital radiology. Int Endod J 2002;35:542–550.

Mader CL, Baumgartner JC, Peters DD. Scanning electron microscopic investigation of the smeared layer on root canal walls. J Endod 1984;10:477–483.

Madison S, Krell KV. Comparison of ethylenediamine tetraacetic acid and sodium hypochlorite on the apical seal of endodontically treated teeth. J Endod 1984;10:499–503.

Malki M, Verhaagen B, Jiang LM, et al. Irrigant flow beyond the insertion depth of an ultrasonically oscillating file in straight and curved root canals: Visualization and cleaning efficacy. J Endod 2012;38:657–661.

Margelos J, Eliades G, Verdelis C, Palaghias G. Interaction of calcium hydroxide with zinc oxide-eugenol type sealers: A potential clinical problem. J Endod 1997;23:43–48.

Marshall FJ, Massler M. The sealing of pulpless teeth evaluated with radio-isotopes. J Dent Med 1961:172–184.

McComb D, Smith DC, Beagrie GS. The results of in vivo endodontic chemomechanical instrumentation—A scanning electron microscopic study. J Br Endod Soc 1976;9:11–18.

Mickel AK, Chogle S, Liddle J, Huffaker K, Jones JJ. The role of apical size determination and enlargement in the reduction of intracanal bacteria. J Endod 2007;33:21–23.

Mjor IA. Pulp-dentin biology in restorative dentistry. Part 7: The exposed pulp. Quintessence Int 2002;33:113–135.

Molander A, Warfvinge J, Reit C, Kvist T. Clinical and radiographic evaluation of one- and two-visit endodontic treatment of asymptomatic necrotic teeth with apical periodontitis: A randomized clinical trial. J Endod 2007;33:1145–1148.

Morgan LF, Montgomery S. An evaluation of the crown-down pressureless technique. J Endod 1984;10:491–498.

Morgental RD, Singh A, Sappal H, Kopper PM, Vier-Pelisser FV, Peters OA. Dentin inhibits the antibacterial effect of new and conventional endodontic irrigants. J Endod 2013;39:406–410.

Munoz HR, Camacho-Cuadra K. In vivo efficacy of three different endodontic irrigation systems for irrigant delivery to working length of mesial canals of mandibular molars. J Endod 2012;38:445–448.

Nerwich A, Figdor D, Messer HH. pH changes in root dentin over a 4-week period following root canal dressing with calcium hydroxide. J Endod 1993;19:302–306.

Nielsen BA, Baumgartner JC. Comparison of the EndoVac system to needle irrigation of root canals. J Endod 2007;33:611–615.

Okino LA, Siqueira EL, Santos M, Bombana AC, Figueiredo JA. Dissolution of pulp tissue by aqueous solution of chlorhexidine digluconate and chlorhexidine digluconate gel. Int Endod J 2004;37:38–41.

Oliveira LD, Carvalho CA, Carvalho AS, Alves Jde S, Valera MC, Jorge AO. Efficacy of endodontic treatment for endotoxin reduction in primarily infected root canals and evaluation of cytotoxic effects. J Endod 2012;38:1053–1057.

Orstavik D. Materials used for root canal obturation: Technical, biological, and clinical testing. Endod Topics 2005:25–38.

Ounsi HF, Naaman A. In vitro evaluation of the reliability of the Root ZX electronic apex locator. Int Endod J 1999;32:120–123.

Paredes-Vieyra J, Enriquez FJ. Success rate of single- versus two-visit root canal treatment of teeth with apical periodontitis: A randomized controlled trial. J Endod 2012;38:1164–1169.

Pekruhn RB. The incidence of failure following single-visit endodontic therapy. J Endod 1986;12:68–72.

Penesis VA, Fitzgerald PI, Fayad MI, Wenckus CS, BeGole EA, Johnson BR. Outcome of one-visit and two-visit endodontic treatment of necrotic teeth with apical periodontitis: A randomized controlled trial with one-year evaluation. J Endod 2008;34:251–257.

Peng L, Ye L, Tan H, Zhou X. Outcome of root canal obturation by warm gutta-percha versus cold lateral condensation: A meta-analysis. J Endod 2007;33:106–109.

Peters LB, Wesselink PR. Periapical healing of endodontically treated teeth in one and two visits obturated in the presence or absence of detectable microorganisms. Int Endod J 2002;35:660–667.

Peters OA, Schonenberger K, Laib A. Effects of four Ni-Ti preparation techniques on root canal geometry assessed by micro computed tomography. Int Endod J 2001;34:221–230.

Qian W, Shen Y, Haapasalo M. Quantitative analysis of the effect of irrigant solution sequences on dentin erosion. J Endod 2011;37:1437–1441.

Reader CM, Himel VT, Germain LP, Hoen MM. Effect of three obturation techniques on the filling of lateral canals and the main canal. J Endod 1993;19:404–408.

Ricucci D. Apical limit of root canal instrumentation and obturation, part 1. Literature review. Int Endod J 1998;31:384–393.

Roane JB, Dryden JA, Grimes EW. Incidence of postoperative pain after single- and multiple-visit endodontic procedures. Oral Surg Oral Med Oral Pathol 1983;55:68–72.

Roane JB, Sabala CL, Duncanson MG Jr. The "balanced force" concept for instrumentation of curved canals. J Endod 1985;11:203–211.

Robinson D, Goerig AC, Neaverth EJ. Endodontic access: An update, Part I. Compendium 1989a;10:290–292, 294–296, 298.

Robinson D, Goerig AC, Neaverth EJ. Endodontic access: An update, Part II. Compendium 1989b;10:328–330, 332–323.

Roland DD, Andelin WE, Browning DF, Hsu GH, Torabinejad M. The effect of preflaring on the rates of separation for 0.04 taper nickel titanium rotary instruments. J Endod 2002;28: 543–545.

Rosenfeld EF, James GA, Burch BS. Vital pulp tissue response to sodium hypochlorite. J Endod 1978;4:140–146.

Rubinstein RA, Kim S. Short-term observation of the results of endodontic surgery with the use of a surgical operation microscope and Super-EBA as root-end filling material. J Endod 1999;25:43–48.

Ruparel NB, Teixeira FB, Ferraz CC, Diogenes A. Direct effect of intracanal medicaments on survival of stem cells of the apical papilla. J Endod 2012;38:1372–1375.

Saatchi M, Aminozarbian MG, Hasheminia SM, Mortaheb A. Influence of apical periodontitis on the accuracy of 3 electronic root canal length measurement devices: An in vivo study. J Endod 2014;40:355–359.

Safavi KE, Nichols FC. Alteration of biological properties of bacterial lipopolysaccharide by calcium hydroxide treatment. J Endod 1994;20:127–129.

Sagsen B, Ustun Y, Aslan T, Canakci BC. The effect of peracetic acid on removing calcium hydroxide from the root canals. J Endod 2012;38:1197–1201.

Saini HR, Tewari S, Sangwan P, Duhan J, Gupta A. Effect of different apical preparation sizes on outcome of primary endodontic treatment: A randomized controlled trial. J Endod 2012;38:1309–1315.

Salzgeber RM, Brilliant JD. An in vivo evaluation of the penetration of an irrigating solution in root canals. J Endod 1977;3:394–398.

Schilder H. Cleaning and shaping the root canal. Dent Clin North Am 1974;18:269–296.

Schilder H. Filling root canals in three dimensions. Dent Clin North Am 1967:723–744.

Schroeder KP, Walton RE, Rivera EM. Straight line access and coronal flaring: Effect on canal length. J Endod 2002;28:474–476.

Seltzer S, Soltanoff W, Smith J. Biologic aspects of endodontics. V. Periapical tissue reactions to root canal instrumentation beyond the apex and root canal fillings short of and beyond the apex. Oral Surg Oral Med Oral Pathol 1973;36:725–737.

Sen BH, Safavi KE, Spangberg LS. Antifungal effects of sodium hypochlorite and chlorhexidine in root canals. J Endod 1999;25:235–238.

Senia ES, Marshall FJ, Rosen S. The solvent action of sodium hypochlorite on pulp tissue of extracted teeth. Oral Surg Oral Med Oral Pathol 1971;31:96–103.

Shabahang S, Goon WW, Gluskin AH. An in vivo evaluation of Root ZX electronic apex locator. J Endod 1996;22:616–618.

Shen Y, Qian W, Abtin H, Gao Y, Haapasalo M. Fatigue testing of controlled memory wire nickel-titanium rotary instruments. J Endod 2011;37:997–1001.

Shen Y, Zhou HM, Zheng YF, Peng B, Haapasalo M. Current challenges and concepts of the thermomechanical treatment of nickel-titanium instruments. J Endod 2013;39:163–172.

Short JA, Morgan LA, Baumgartner JC. A comparison of canal centering ability of four instrumentation techniques. J Endod 1997;23:503–507.

Sigurdsson A, Stancill R, Madison S. Intracanal placement of Ca(OH)2: A comparison of techniques. J Endod 1992;18:367–370.

Silva EJ, Menaged K, Ajuz N, Monteiro MR, Coutinho-Filho Tde S. Postoperative pain after foraminal enlargement in anterior teeth with necrosis and apical periodontitis: A prospective and randomized clinical trial. J Endod 2013;39:173–176.

Siren EK, Haapasalo MP, Waltimo TM, Orstavik D. In vitro antibacterial effect of calcium hydroxide combined with chlorhexidine or iodine potassium iodide on *Enterococcus faecalis*. Eur J Oral Sci 2004;112:326–331.

Sjogren U, Figdor D, Spangberg L, Sundqvist G. The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing. Int Endod J 1991;24:119–125.

Sjogren U, Sundqvist G, Nair PN. Tissue reaction to gutta-percha particles of various sizes when implanted subcutaneously in guinea pigs. Eur J Oral Sci 1995;103:313–321.

Slaton C, Loushine R, Weller R, Parker M, Kimbrough W, Pashley D. Identification of resected root-end dentinal cracks: A comparative study of visual magnification. J Endod 2003;29:519–522.

Soltanoff W. Pain in endodontics. Bull Newark Dent Club 1968;43:4.

Su Y, Wang C, Ye L. Healing rate and post-obturation pain of single- versus multiple-visit endodontic treatment for infected root canals: A systematic review. J Endod 2011;37:125–132.

Sunada I. New method for measuring the length of the root canal. J Dent Res 1962:375–387.

Suzuki K. Experimental study on ionophoresis. J Jap Stomatol Soc 1942:411–429.

Swanson K, Madison S. An evaluation of coronal microleakage in endodontically treated teeth. Part I. Time periods. J Endod 1987;13:56–59.

Tavares WL, de Brito LC, Henriques LC, et al. The impact of chlorhexidine-based endodontic treatment on periapical cytokine expression in teeth. J Endod 2013;39:889–892.

Taylor JK, Jeansonne BG, Lemon RR. Coronal leakage: Effects of smear layer, obturation technique, and sealer. J Endod 1997;23:508–512.

Teixeira FB, Teixeira EC, Thompson J, Leinfelder KF, Trope M. Dentinal bonding reaches the root canal system. J Esthet Restor Dent 2004;16:348–354; discussion 354.

Torabinejad M. Passive step-back technique. Oral Surg Oral Med Oral Pathol 1994;77:398–401.

Tronstad L, Andreasen JO, Hasselgren G, Kristerson L, Riis I. pH changes in dental tissues after root canal filling with calcium hydroxide. J Endod 1981;7:17–21.

Trowbridge HO. Intradental sensory units: Physiological and clinical aspects. J Endod 1985;11:489–498.

Trope M, Delano EO, Orstavik D. Endodontic treatment of teeth with apical periodontitis: Single vs. multivisit treatment. J Endod 1999;25:345–350.

Trowbridge H, Edwall L, Panopoulos P. Effect of zinc oxide-eugenol and calcium hydroxide on intradental nerve activity. J Endod 1982;8:403–406.

Turner JE, Anderson RW, Pashley DH, Pantera EA Jr. Microleakage of temporary endodontic restorations in teeth restored with amalgam. J Endod 1990;16:1–4.

Van der Waal SV, van Dusseldorp NE, de Soet JJ. An evaluation of the accuracy of labeling of percent sodium hypochlorite on various commercial and professional sources: Is sodium hypochlorite from these sources equally suitable for endodontic irrigation? J Endod 2014;40:2049–2052.

Vela KC, Walton RE, Trope M, Windschitl P, Caplan DJ. Patient preferences regarding 1-visit versus 2-visit root canal therapy. J Endod 2012a;38:1322–1325.

Vera J, Hernandez EM, Romero M, Arias A, van der Sluis LW. Effect of maintaining apical patency on irrigant penetration into the apical two millimeters of large root canals: An in vivo study. J Endod 2012b;38:1340–1343.

Vera J, Siqueira JF Jr, Ricucci D, et al. One- versus two-visit endodontic treatment of teeth with apical periodontitis: A histobacteriologic study. J Endod 2012;38:1040–1052.

Vitti RP, Prati C, Silva EJ, et al. Physical properties of MTA Fillapex sealer. J Endod 2013;39:915–918.

Wagner C, Barth CV Jr, de Olveira SD, Campos MM. Effectiveness of the proton pump inhibitor omeprazole associated with calcium hydroxide as intracanal medication: An in vivo study. J Endod 2011;37:1253–1257.

Waltimo TM, Orstavik D, Siren EK, Haapasalo MP. In vitro susceptibility of *Candida albicans* to four disinfectants and their combinations. Int Endod J 1999;32:421–429.

Walton R, Fouad A. Endodontic interappointment flare-ups: A prospective study of incidence and related factors. J Endod 1992;18:172–177.

Wang CS, Arnold RR, Trope M, Teixeira FB. Clinical efficiency of 2% chlorhexidine gel in reducing intracanal bacteria. J Endod 2007;33:1283–1289.

Webber RT, del Rio CE, Brady JM, Segall RO. Sealing quality of a temporary filling material. Oral Surg Oral Med Oral Pathol 1978;46:123–130.

Wiemann AH, Wilcox LR. In vitro evaluation of four methods of sealer placement. J Endod 1991;17:444–447.

Wilson BL, Broberg C, Baumgartner JC, Harris C, Kron J. Safety of electronic apex locators and pulp testers in patients with implanted cardiac pacemakers or cardioverter/defibrillators. J Endod 2006;32:847–852.

Wong DT, Cheung GS. Extension of bactericidal effect of sodium hypochlorite into dentinal tubules. J Endod 2014;40:825–829.

Wooley LH, Woodworth J, Dobbs JL. A preliminary evaluation of the effects of electrical pulp testers on dogs with artificial pacemakers. J Am Dent Assoc 1974:1099–1101.

Wu MK, Wesselink PR. Efficacy of three techniques in cleaning the apical portion of curved root canals. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:492–496.

Xavier AC, Martinho FC, Chung A, et al. One-visit versus two-visit root canal treatment: Effectiveness in the removal of endotoxins and cultivable bacteria. J Endod 2013;39:959–964.

Zehnder M. Root canal irrigants. J Endod 2006;32:389–398.

Nonsurgical Retreatment

American Association of Endodontists. AAE Position Statement 2013. Use of Silver Points. https://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/silverpointsstatement.pdf. Accessed 22 December 2015.

Azarpazhooh A, Dao T, Figueiredo R, Krahn M, Friedman S. A survey of dentists' preferences for the treatment of teeth with apical periodontitis. J Endod 2013;39:1226–1233.

Bertrand MF, Pellegrino JC, Rocca JP, Klinghofer A, Bolla M. Removal of Thermafil root canal filling material. J Endod 1997;23:54–57.

Chutich MJ, Kaminski EJ, Miller DA, Lautenschlager EP. Risk assessment of the toxicity of solvents of gutta-percha used in endodontic retreatment. J Endod 1998;24:213–216.

Dominici JT, Clark S, Scheetz J, Eleazer PD. Analysis of heat generation using ultrasonic vibration for post removal. J Endod 2005;31:301–303.

Edgar SW, Marshall JG, Baumgartner JC. The antimicrobial effect of chloroform on *Enterococcus faecalis* after gutta-percha removal. J Endod 2006;32:1185–1187.

Ferreira JJ, Rhodes JS, Ford TR. The efficacy of gutta-percha removal using ProFiles. Int Endod J 2001;34:267–274.

Gomez G, Jara F, Sanchez B, Roig M, Duran-Sindreu F. Effects of piezoelectric units on pacemaker function: An in vitro study. J Endod 2013;39:1296–1299.

Hess D, Solomon E, Spears R, He J. Retreatability of a bioceramic root canal sealing material. J Endod 2011;37:1547–1549.

Huttula AS, Tordik PA, Imamura G, Eichmiller FC, McClanahan SB. The effect of ultrasonic post instrumentation on root surface temperature. J Endod 2006;32:1085–1087.

Johnson WT, Leary JM, Boyer DB. Effect of ultrasonic vibration on post removal in extracted human premolar teeth. J Endod 1996;22:487–488.

Kaplowitz GJ. Evaluation of gutta-percha solvents. J Endod 1990;16:539–540.

Krell KV, Fuller MW, Scott GL. The conservative retrieval of silver cones in difficult cases. J Endod 1984;10:269–273.

Krell KV, Neo J. The use of ultrasonic endodontic instrumentation in the re-treatment of a paste-filled endodontic tooth. Oral Surg Oral Med Oral Pathol 1985;60:100–102.

Lindemann M, Yaman P, Dennison JB, Herrero AA. Comparison of the efficiency and effectiveness of various techniques for removal of fiber posts. J Endod 2005;31:520–522.

McDonald MN, Vire DE. Chloroform in the endodontic operatory. J Endod 1992;18:301–303.

Nair PN. On the causes of persistent apical periodontitis: A review. Int Endod J 2006;39:249–281.

Neelakantan P, Grotra D, Sharma S. Retreatability of 2 mineral trioxide aggregate-based root canal sealers: A cone-beam computed tomography analysis. J Endod 2013;39:893–896.

Ricucci D, Siqueira JF Jr. Recurrent apical periodontitis and late endodontic treatment failure related to coronal leakage: A case report. J Endod 2011;37:1171–1175.

Royzenblat A, Goodell GG. Comparison of removal times of Thermafil plastic obturators using ProFile rotary instruments at different rotational speeds in moderately curved canals. J Endod 2007;33:256–258.

Salehrabi R, Rotstein I. Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment. J Endod 2010;36:790–792.

Schwandt NW, Gound TG. Resorcinol-formaldehyde resin "Russian Red" endodontic therapy. J Endod 2003;29:435–437.

Seltzer S, Green DB, Weiner N, DeRenzis F. A scanning electron microscope examination of silver cones removed from endodontically treated teeth. Oral Surg Oral Med Oral Pathol 1972;33:589–605.

Shemesh H, Roeleveld AC, Wesselink PR, Wu MK. Damage to root dentin during retreatment procedures. J Endod 2011;37:63–66.

Vieira AR, Siqueira JF Jr, Ricucci D, Lopes WS. Dentinal tubule infection as the cause of recurrent disease and late endodontic treatment failure: A case report. J Endod 2012;38:250–254.

Vranas RN, Hartwell GR, Moon PC. The effect of endodontic solutions on resorcinol-formalin paste. J Endod 2003;29:69–72.

Wilcox LR. Thermafil retreatment with and without chloroform solvent. J Endod 1993;19:563–566.

Wolcott JF, Himel VT, Hicks ML. Thermafil retreatment using a new "System B" technique or a solvent. J Endod 1999;25:761–764.

Xu LL, Zhang L, Zhou XD, Wang R, Deng YH, Huang DM. Residual filling material in dentinal tubules after gutta-percha removal observed with scanning electron microscopy. J Endod 2012;38:293–296.

Surgical Endodontics

Andelin WE, Browning DF, Hsu GH, Roland DD, Torabinejad M. Microleakage of resected MTA. J Endod 2002;28:573–574.

Andreasen JO, Paulsen HU, Yuz, Bayer T. A long-term study of 370 autotransplanted premolars. Part IV. Root development subsequent to transplantation. Eur J Orthod 1990;12:38–50.

Andreasen JO, Rud J. Correlation between histology and radiography in the assessment of healing after endodontic surgery. Int J Oral Surg 1972;1:161–173.

Baek SH, Lee WC, Setzer FC, Kim S. Periapical bone regeneration after endodontic microsurgery with three different root-end filling materials: Amalgam, SuperEBA, and mineral trioxide aggregate. J Endod 2010;36:1323–1325.

Baek SH, Plenk H Jr, Kim S. Periapical tissue responses and cementum regeneration with amalgam, SuperEBA, and MTA as root-end filling materials. J Endod 2005;31:444–449.

Barone C, Dao TT, Basrani BB, Wang N, Friedman S. Treatment outcome in endodontics: The Toronto study—Phases 3, 4, and 5: Apical surgery. J Endod 2010;36:28–35.

Bashutski JD, Wang HL. Periodontal and endodontic regeneration. J Endod 2009;35:321–328.

Bender IB, Rossman LE. Intentional replantation of endodontically treated teeth. Oral Surg Oral Med Oral Pathol 1993;76:623–630.

Block RM, Bushell A, Rodrigues H, Langeland K. A histopathologic, histobacteriologic, and radiographic study of periapical endodontic surgical specimens. Oral Surg Oral Med Oral Pathol 1976;42:656–678.

Buckley JA, Ciancio SG, McMullen JA. Efficacy of epinephrine concentration in local anesthesia during periodontal surgery. J Periodontol 1984;55:653–657.

Caffesse RG, Quinones CR. Guided tissue regeneration: Biologic rationale, surgical technique, and clinical results. Compendium 1992;13:166–170.

Cambruzzi JV, Marshall FJ, Pappin JB. Methylene blue dye: An aid to endodontic surgery. J Endod 1985;11:311–314.

Camilleri J, Montesin FE, Brady K, Sweeney R, Curtis RV, Ford TR. The constitution of mineral trioxide aggregate. Dent Mater 2005;21:297–303.

Cardona JL, Caldera MM, Vera J. Autotransplantation of a premolar: A long-term follow-up report of a clinical case. J Endod 2012;38:1149–1152.

Chong BS, Pitt Ford TR. Postoperative pain after root-end resection and filling. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100:762–766.

Cotter MR, Panzarino J. Intentional replantation: A case report. J Endod 2006;32:579–582.

Damas BA, Wheater MA, Bringas JS, Hoen MM. Cytotoxicity comparison of mineral trioxide aggregates and EndoSequence bioceramic root repair materials. J Endod 2011;37:372–375.

de Lange J, Putters T, Baas E, van Ingen JM. Ultrasonic root-end preparation in apical surgery: A prospective randomized study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:841–845.

Del Fabbro M, Taschieri S, Weinstein R. Quality of life after microscopic periradicular surgery using two different incision techniques: A randomized clinical study. Int Endod J 2009;42:360–367.

Denio D, Torabinejad M, Bakland LK. Anatomical relationship of the mandibular canal to its surrounding structures in mature mandibles. J Endod 1992;18:161–165.

Douthitt JC, Gutmann JL, Witherspoon DE. Histologic assessment of healing after the use of a bioresorbable membrane in the management of buccal bone loss concomitant with periradicular surgery. J Endod 2001;27:404–410.

Engel TK, Steiman HR. Preliminary investigation of ultrasonic root end preparation. J Endod 1995;21:443–445.

Fischer EJ, Arens DE, Miller CH. Bacterial leakage of mineral trioxide aggregate as compared with zinc-free amalgam, intermediate restorative material, and Super-EBA as a root-end filling material. J Endod 1998;24:176–179.

Garrett K, Kerr M, Hartwell G, O'Sullivan S, Mayer P. The effect of a bioresorbable matrix barrier in endodontic surgery on the rate of periapical healing: An in vivo study. J Endod 2002;28:503–506.

Gilheany PA, Figdor D, Tyas MJ. Apical dentin permeability and microleakage associated with root end resection and retrograde filling. J Endod 1994;20:22–26.

Grossman LI. Intentional replantation of teeth: A clinical evaluation. J Am Dent Assoc 1982;104:633–639.

Gutmann JL. Parameters of achieving quality anesthesia and hemostasis in surgical endodontics. Anesth Pain Control Dent 1993;2:223–226.

Hargreaves KM, Keiser K. Building effective strategies for the management of endodontic pain. Endod Topics 2002;3:93–105.

Harrison JW. Healing of surgical wounds in oral mucoperiosteal tissues. J Endod 1991;17:401–408.

Harrison JW, Jurosky KA. Wound healing in the tissues of the periodontium following periradicular surgery. 1. The incisional wound. J Endod 1991a;17:425–435.

Harrison JW, Jurosky KA. Wound healing in the tissues of the periodontium following periradicular surgery. 2. The dissectional wound. J Endod 1991b;17:544–552.

Harrison JW, Todd MJ. The effect of root resection on the sealing property of root canal obturations. Oral Surg Oral Med Oral Pathol 1980;50:264–272.

Ibarrola JL, Bjorenson JE, Austin BP, Gerstein H. Osseous reactions to three hemostatic agents. J Endod 1985;11:75–83.

Intra JB, Roldi A, Brandao RC, de Araujo Estrela CR, Estrela C. Autogenous premolar transplantation into artificial socket in maxillary lateral incisor site. J Endod 2014;40:1885–1890.

Iqbal MK, Kratchman SI, Guess GM, Karabucak B, Kim S. Microscopic periradicular surgery: Perioperative predictors for postoperative clinical outcomes and quality of life assessment. J Endod 2007;33:239–244.

Jeansonne BG, Boggs WS, Lemon RR. Ferric sulfate hemostasis: Effect on osseous wound healing. II. With curettage and irrigation. J Endod 1993;19:174–176.

Jonsson T, Sigurdsson TJ. Autotransplantation of premolars to premolar sites. A long-term follow-up study of 40 consecutive patients. Am J Orthod Dentofacial Orthop 2004;125:668–675.

Kaplan SD, Tanzilli JP, Raphael D, Moodnik RM. A comparison of the marginal leakage of retrograde techniques. Oral Surg Oral Med Oral Pathol 1982;54:583–585.

Keiser K, Johnson CC, Tipton DA. Cytotoxicity of mineral trioxide aggregate using human periodontal ligament fibroblasts. J Endod 2000;26:288–291.

Kim S, Pecora G, Rubinstein RA. Color Atlas of Microsurgery in Endodontics. Philadelphia: Saunders, 2001.

Kim SG, Solomon C. Cost-effectiveness of endodontic molar retreatment compared with fixed partial dentures and single-tooth implant alternatives. J Endod 2011;37:321–325.

Kovisto T, Ahmad M, Bowles WR. Proximity of the mandibular canal to the tooth apex. J Endod 2011;37:311–315.

Kramper BJ, Kaminski EJ, Osetek EM, Heuer MA. A comparative study of the wound healing of three types of flap design used in periapical surgery. J Endod 1984;10:17–25.

Kratchman S. Intentional replantation. Dent Clin North Am 1997;41:603–617.

Lemon RR, Steele PJ, Jeansonne BG. Ferric sulfate hemostasis: Effect on osseous wound healing. Left in situ for maximum exposure. J Endod 1993;19:170–173.

Lin L, Chance K, Shovlin F, Skribner J, Langeland K. Oroantral communication in periapical surgery of maxillary posterior teeth. J Endod 1985;11:40–44.

Lin L, Chen MY, Ricucci D, Rosenberg PA. Guided tissue regeneration in periapical surgery. J Endod 2010;36:618–625.

Liu Z, Zhang D, Li Q, Xu Q. Evaluation of root-end preparation with a new ultrasonic tip. J Endod 2013;39:820–823.

Lundberg T, Isaksson S. A clinical follow-up study of 278 autotransplanted teeth. Br J Oral Maxillofac Surg 1996;34:181–185.

Ma J, Shen Y, Stojicic S, Haapasalo M. Biocompatibility of two novel root repair materials. J Endod 2011;37:793–798.

Mattison GD, von Fraunhofer JA, Delivanis PD, Anderson AN. Microleakage of retrograde amalgams. J Endod 1985;11:340–345.

Minnich SG, Hartwell GR, Portell FR. Does cold burnishing gutta-percha create a better apical seal? J Endod 1989;15:204–209.

Moiseiwitsch JR. Avoiding the mental foramen during periapical surgery. J Endod 1995;21: 340–342.

Nicoll BK, Peters RJ. Heat generation during ultrasonic instrumentation of dentin as affected by different irrigation methods. J Periodontol 1998;69:884–888.

Oynick J, Oynick T. A study of a new material for retrograde fillings. J Endod 1978;4:203–206.

Parirokh M, Torabinejad M. Mineral trioxide aggregate: A comprehensive literature review—Part III: Clinical applications, drawbacks, and mechanism of action. J Endod 2010;36:400–413.

Rainwater A, Jeansonne BG, Sarkar N. Effects of ultrasonic root-end preparation on microcrack formation and leakage. J Endod 2000;26:72–75.

Rud J, Rud V. Surgical endodontics of upper molars: Relation to the maxillary sinus and operation in acute state of infection. J Endod 1998;24:260–261.

Scarano A, Artese L, Piattelli A, Carinci F, Mancino C, Iezzi G. Hemostasis control in endodontic surgery: A comparative study of calcium sulfate versus gauzes and versus ferric sulfate. J Endod 2012;38:20–23.

Selim HA, el Deeb ME, Messer HH. Blood loss during endodontic surgery. Endod Dent Traumatol 1987;3:33–36.

Setzer FC, Shah SB, Kohli MR, Karabucak B, Kim S. Outcome of endodontic surgery: A meta-analysis of the literature—Part 1: Comparison of traditional root-end surgery and endodontic microsurgery. J Endod 2010;36:1757–1765.

Shahan MH, Chuang AH, Brennan WA, Dirksen TR, Van Dyke TE, McPherson JC. The effect of chlorhexidine irrigation on tensile wound strength. J Periodontol 1993;64:719–722.

Song M, Kim E. A prospective randomized controlled study of mineral trioxide aggregate and super ethoxy–benzoic acid as root-end filling materials in endodontic microsurgery. J Endod 2012;38:875–879.

Tataryn RW, Torabinejad M, Boyne PJ. Healing potential of osteotomies of the nasal sinus in the dog. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84:196–202.

Tidmarsh BG, Arrowsmith MG. Dentinal tubules at the root ends of apicected teeth: A scanning electron microscopic study. Int Endod J 1989;22:184–189.

Torabinejad M, Higa RK, McKendry DJ, Pitt Ford TR. Dye leakage of four root end filling materials: Effects of blood contamination. J Endod 1994;20:159–163.

Torabinejad M, Hong CU, Lee SJ, Monsef M, Pitt Ford TR. Investigation of mineral trioxide aggregate for root-end filling in dogs. J Endod 1995;21:603–608.

Tsesis I, Fuss Z, Lin S, Tilinger G, Peled M. Analysis of postoperative symptoms following surgical endodontic treatment. Quintessence Int 2003;34:756–760.

Tsesis I, Rosen E, Tamse A, Taschieri S, Del Fabbro M. Effect of guided tissue regeneration on the outcome of surgical endodontic treatment: A systematic review and meta-analysis. J Endod 2011;37:1039–1045.

Tsesis I, Rosen E, Taschieri S, Telishevsky Strauss Y, Ceresoli V, Del Fabbro M. Outcomes of surgical endodontic treatment performed by a modern technique: An updated meta-analysis of the literature. J Endod 2013;39:332–339.

Velvart P, Ebner-Zimmermann U, Ebner JP. Comparison of long-term papilla healing following sulcular full thickness flap and papilla base flap in endodontic surgery. Int Endod J 2004;37:687–693.

Velvart P, Peters CI. Soft tissue management in endodontic surgery. J Endod 2005;31:4–16.

Vickers FJ, Baumgartner JC, Marshall G. Hemostatic efficacy and cardiovascular effects of agents used during endodontic surgery. J Endod 2002;28:322–323.

Von Arx T, Fodich I, Bornstein MM. Proximity of premolar roots to maxillary sinus: A radiographic survey using cone-beam computed tomography. J Endod 2014;40:1541–1548.

Von Arx T, Penarrocha M, Jensen S. Prognostic factors in apical surgery with root-end filling: A meta-analysis. J Endod 2010;36:957–973.

Vreeland DL, Tidwell E. Flap design for surgical endodontics. Oral Surg Oral Med Oral Pathol 1982;54:461–465.

Walton RE. Iatrogenic maxillary sinus exposure during maxillary posterior root-end surgery. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97:3; author reply 3–4.

Weller RN, Niemczyk SP, Kim S. Incidence and position of the canal isthmus. Part 1. Mesiobuccal root of the maxillary first molar. J Endod 1995;21:380–383.

Wesson CM, Gale TM. Molar apicectomy with amalgam root-end filling: Results of a prospective study in two district general hospitals. Br Dent J 2003;195:707–714.

Witherspoon DE, Gutmann JL. Haemostasis in periradicular surgery. Int Endod J 1996;29: 135–149.

Wuchenich G, Meadows D, Torabinejad M. A comparison between two root end preparation techniques in human cadavers. J Endod 1994;20:279–282.

Vital Pulp Therapy

Aguilar P, Linsuwanont P. Vital pulp therapy in vital permanent teeth with cariously exposed pulp: A systematic review. J Endod 2011;37:581–587.

American Association of Endodontists. AAE Position Statement 2013. Concerning Paraformaldehyde-Containing Endodontic Filling Materials and Sealers. https://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/paraformaldehydefillingmaterials.pdf. Accessed 22 December 2015.

American Association of Endodontists. Glossary of Endodontic Terms. dev.aae.org/glossary. Accessed 20 January 2016.

Andreasen JO, Paulsen HU, Yu Z, Bayer T. A long-term study of 370 autotransplanted premolars. Part IV. Root development subsequent to transplantation. Eur J Orthod 1990;12:38–50.

Asgary S, Eghbal MJ. The effect of pulpotomy using a calcium-enriched mixture cement versus one-visit root canal therapy on postoperative pain relief in irreversible pulpitis: A randomized clinical trial. Odontology 2010;98:126–133.

Bakland LK. Management of traumatically injured pulps in immature teeth using MTA. J Calif Dent Assoc 2000;28:855–858.

Bergenholtz G. Advances since the paper by Zander and Glass (1949) on the pursuit of healing methods for pulpal exposures: Historical perspectives. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100:S102–S108.

Block RM, Lewis RD, Sheats JB, Burke SH. Antibody formation to dog pulp tissue altered by 6.5 percent paraformaldehyde via the root canal. J Pedod 1977;2:3–15.

Cho SY, Seo DG, Lee SJ, Lee J, Lee SJ, Jung IY. Prognostic factors for clinical outcomes according to time after direct pulp capping. J Endod 2013;39:327–331.

Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture. J Endod 1978;4:232–237.

Iwamoto CE, Adachi E, Pameijer CH, Barnes D, Romberg EE, Jefferies S. Clinical and histological evaluation of white ProRoot MTA in direct pulp capping. Am J Dent 2006;19:85–90.

Karanxha L, Park SJ, Son WJ, Nor JE, Min KS. Combined effects of simvastatin and enamel matrix derivative on odontoblastic differentiation of human dental pulp cells. J Endod 2013;39:76–82.

Keswani D, Pandey RK, Ansari A, Gupta S. Comparative evaluation of platelet-rich fibrin and mineral trioxide aggregate as pulpotomy agents in permanent teeth with incomplete root development: A randomized controlled trial. J Endod 2014;40:599–605.

Nowicka A, Lipski M, Parafiniuk M, et al. Response of human dental pulp capped with biodentine and mineral trioxide aggregate. J Endod 2013;39:743–747.

Parirokh M, Torabinejad M. Mineral trioxide aggregate: A comprehensive literature review—Part I: Chemical, physical, and antibacterial properties. J Endod 2010;36:16–27.

Pashley EL, Myers DR, Pashley DH, Whitford GM. Systemic distribution of 14C-formaldehyde from formocresol-treated pulpotomy sites. J Dent Res 1980;59:602–608.

Silva GA, Gava E, Lanza LD, Estrela C, Alves JB. Subclinical failures of direct pulp capping of human teeth by using a dentin bonding system. J Endod 2013;39:182–189.

Torabinejad M, Parirokh M. Mineral trioxide aggregate: A comprehensive literature review—Part II: Leakage and biocompatibility investigations. J Endod 2010;36:190–202.

Apexification

American Association of Endodontists. Glossary of Endodontic Terms. dev.aae.org/glossary. Accessed 20 January 2016.

Andreasen JO, Farik B, Munksgaard EC. Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture. Dent Traumatol 2002;18:134–137.

Chala S, Abouqal R, Rida S. Apexification of immature teeth with calcium hydroxide or mineral trioxide aggregate: Systematic review and meta-analysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112:e36–e42.

Cvek M. Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta percha. Endod Dent Traumatol 1992;8:45–55.

Finucane D, Kinirons MJ. Non-vital immature permanent incisors: Factors that may influence treatment outcome. Endod Dent Traumatol 1999;15:273–277.

Frank AL. Therapy for the divergent pulpless tooth by continued apical formation. J Am Dent Assoc 1966;72:87–93.

Goldberg F, De Silvio AC, Manfre S, Nastri N. In vitro measurement accuracy of an electronic apex locator in teeth with simulated apical root resorption. J Endod 2002;28:461–463.

Jeeruphan T, Jantarat J, Yanpiset K, Suwannapan L, Khewsawai P, Hargreaves KM. Mahidol study 1: Comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: A retrospective study. J Endod 2012;38:1330–1336.

Katebzadeh N, Dalton BC, Trope M. Strengthening immature teeth during and after apexification. J Endod 1998;24:256–259.

Kleier DJ, Barr ES. A study of endodontically apexified teeth. Endod Dent Traumatol 1991;7:112–117.

Rafter M. Apexification: A review. Dent Traumatol 2005;21:1–8.

Shabahang S. Treatment options: Apexogenesis and apexification. J Endod 2013;39:S26–S29.

Simon S, Rilliard F, Berdal A, Machtou P. The use of mineral trioxide aggregate in one-visit apexification treatment: A prospective study. Int Endod J 2007;40:186–197.

Torabinejad M, Parirokh M. Mineral trioxide aggregate: A comprehensive literature review—Part II: Leakage and biocompatibility investigations. J Endod 2010;36:190–202.

Witherspoon DE, Ham K. One-visit apexification: Technique for inducing root-end barrier formation in apical closures. Pract Proced Aesthet Dent 2001;13:455–460.

Regenerative Endodontics

Althumairy RI, Teixeira FB, Diogenes A. Effect of dentin conditioning with intracanal medicaments on survival of stem cells of apical papilla. J Endod 2014;40:521–525.

Banchs F, Trope M. Revascularization of immature permanent teeth with apical periodontitis: New treatment protocol? J Endod 2004;30:196–200.

Becerra P, Ricucci D, Loghin S, Gibbs JL, Lin LM. Histologic study of a human immature permanent premolar with chronic apical abscess after revascularization/revitalization. J Endod 2014;40:133–139.

Chandrahasa S, Murray PE, Namerow KN. Proliferation of mature ex vivo human dental pulp using tissue engineering scaffolds. J Endod 2011;37:1236–1239.

Colombo JS, Moore AN, Hartgerink JD, D'Souza RN. Scaffolds to control inflammation and facilitate dental pulp regeneration. J Endod 2014;40:S6–S12.

Cvek M. Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta percha. Endod Dent Traumatol 1992;8:45–55.

Felman D, Parashos P. Coronal tooth discoloration and white mineral trioxide aggregate. J Endod 2013;39:484–487.

Flake NM, Gibbs JL, Diogenes A, Hargreaves KM, Khan AA. A standardized novel method to measure radiographic root changes after endodontic therapy in immature teeth. J Endod 2014;40:46–50.

Galler KM, D'Souza RN, Federlin M, et al. Dentin conditioning codetermines cell fate in regenerative endodontics. J Endod 2011;37:1536–1541.

Hargreaves KM, Giesler T, Henry M, Wang Y. Regeneration potential of the young permanent tooth: What does the future hold? J Endod 2008;34:S51–S56.

Hoshino E, Kurihara-Ando N, Sato I, et al. In-vitro antibacterial susceptibility of bacteria taken from infected root dentine to a mixture of ciprofloxacin, metronidazole and minocycline. Int Endod J 1996;29:125–130.

Huang GT. A paradigm shift in endodontic management of immature teeth: Conservation of stem cells for regeneration. J Dent 2008;36:379–386.

Iwaya S, Ikawa M, Kubota M. Revascularization of an immature permanent tooth with periradicular abscess after luxation. Dent Traumatol 2011;27:55–58.

Jadhav G, Shah N, Logani A. Revascularization with and without platelet-rich plasma in nonvital, immature, anterior teeth: A pilot clinical study. J Endod 2012;38:1581–1587.

Jeeruphan T, Jantarat J, Yanpiset K, Suwannapan L, Khewsawai P, Hargreaves KM. Mahidol study 1: Comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: A retrospective study. J Endod 2012;38:1330–1336.

Kim JH, Kim Y, Shin SJ, Park JW, Jung IY. Tooth discoloration of immature permanent incisor associated with triple antibiotic therapy: A case report. J Endod 2010;36:1086–1091.

Langer R, Vacanti JP. Tissue engineering. Science 1993;260:920–926.

Law AS. Considerations for regeneration procedures. J Endod 2013;39:S44–S56.

Lenzi R, Trope M. Revitalization procedures in two traumatized incisors with different biological outcomes. J Endod 2012;38:411–414.

Lovelace TW, Henry MA, Hargreaves KM, Diogenes A. Evaluation of the delivery of mesenchymal stem cells into the root canal space of necrotic immature teeth after clinical regenerative endodontic procedure. J Endod 2011;37:133–138.

Manguno C, Murray PE, Howard C, Madras J, Mangan S, Namerow KN. A survey of dental residents' expectations for regenerative endodontics. J Endod 2012;38:137–143.

Martin DE, De Almeida JF, Henry MA, et al. Concentration-dependent effect of sodium hypochlorite on stem cells of apical papilla survival and differentiation. J Endod 2014;40:51–55.

Martin G, Ricucci D, Gibbs JL, Lin LM. Histological findings of revascularized/revitalized immature permanent molar with apical periodontitis using platelet-rich plasma. J Endod 2013;39:138–144.

Mente J, Leo M, Panagidis D, et al. Treatment outcome of mineral trioxide aggregate in open apex teeth. J Endod 2013;39:20–26.

Nagata JY, Soares AJ, Souza-Filho FJ, et al. Microbial evaluation of traumatized teeth treated with triple antibiotic paste or calcium hydroxide with 2% chlorhexidine gel in pulp revascularization. J Endod 2014;40:778–783.

Nosrat A, Homayounfar N, Oloomi K. Drawbacks and unfavorable outcomes of regenerative endodontic treatments of necrotic immature teeth: A literature review and report of a case. J Endod 2012;38:1428–1434.

Nosrat A, Li KL, Vir K, Hicks ML, Fouad AF. Is pulp regeneration necessary for root maturation? J Endod 2013;39:1291–1295.

Nygaard-Ostby B, Hjortdal O. Tissue formation in the root canal following pulp removal. Scand J Dent Res 1971;79:333–349.

Ordinola-Zapata R, Bramante CM, Minotti PG, et al. Antimicrobial activity of triantibiotic paste, 2% chlorhexidine gel, and calcium hydroxide on an intraoral-infected dentin biofilm model. J Endod 2013;39:115–118.

Pang NS, Lee SJ, Kim E, et al. Effect of EDTA on attachment and differentiation of dental pulp stem cells. J Endod 2014;40:811–817.

Petrino JA, Boda KK, Shambarger S, Bowles WR, McClanahan SB. Challenges in regenerative endodontics: A case series. J Endod 2010;36:536–541.

Ruparel NB, Teixeira FB, Ferraz CC, Diogenes A. Direct effect of intracanal medicaments on survival of stem cells of the apical papilla. J Endod 2012;38:1372–1375.

Thibodeau B, Trope M. Pulp revascularization of a necrotic infected immature permanent tooth: Case report and review of the literature. Pediatr Dent 2007;29:47–50.

Torabinejad M, Faras H, Corr R, Wright KR, Shabahang S. Histologic examinations of teeth treated with 2 scaffolds: A pilot animal investigation. J Endod 2014;40:515–520.

Trevino EG, Patwardhan AN, Henry MA, et al. Effect of irrigants on the survival of human stem cells of the apical papilla in a platelet-rich plasma scaffold in human root tips. J Endod 2011;37:1109–1115.

Wang X, Thibodeau B, Trope M, Lin LM, Huang GT. Histologic characterization of regenerated tissues in canal space after the revitalization/revascularization procedure of immature dog teeth with apical periodontitis. J Endod 2010;36:56–63.

Windley W 3rd, Teixeira F, Levin L, Sigurdsson A, Trope M. Disinfection of immature teeth with a triple antibiotic paste. J Endod 2005;31:439–443.

Yamauchi N, Nagaoka H, Yamauchi S, Teixeira FB, Miguez P, Yamauchi M. Immunohistological characterization of newly formed tissues after regenerative procedure in immature dog teeth. J Endod 2011a;37:1636–1641.

Yamauchi N, Yamauchi S, Nagaoka H, et al. Tissue engineering strategies for immature teeth with apical periodontitis. J Endod 2011b;37:390–397.

Internal Bleaching

Belobrov I, Parashos P. Treatment of tooth discoloration after the use of white mineral trioxide aggregate. J Endod 2011;37:1017–1020.

Friedman S. Internal bleaching: Long-term outcomes and complications. J Am Dent Assoc 1997;128(suppl):51S–55S.

Glockner K, Hulla H, Ebeleseder K, Stadtler P. Five-year follow-up of internal bleaching. Braz Dent J 1999;10:105–110.

Madison S, Walton R. Cervical root resorption following bleaching of endodontically treated teeth. J Endod 1990;16:570–574.

Nutting EB, Poe GS. Chemical bleaching of discolored endodontically treated teeth. Dent Clin North Am 1967:655–662.

Rotstein I, Mor C, Friedman S. Prognosis of intracoronal bleaching with sodium perborate preparation in vitro: 1-year study. J Endod 1993;19:10–12.

Spasser H. A simple bleaching technique using sodium perborate. NYS Dent J 1961:332–334.

Walton RE, O'Dell NL, Lake FT, Shimp RG. Internal bleaching of tetracycline-stained teeth in dogs. J Endod 1983;9:416–420.

Follow-Up Care

Friedman S, Mor C. The success of endodontic therapy—Healing and functionality. J Calif Dent Assoc 2004;32:493–503.

Molven O, Halse A, Fristad I, MacDonald-Jankowski D. Periapical changes following root-canal treatment observed 20-27 years postoperatively. Int Endod J 2002;35:784–790.

Murphy WK, Kaugars GE, Collett WK, Dodds RN. Healing of periapical radiolucencies after nonsurgical endodontic therapy. Oral Surg Oral Med Oral Pathol 1991;71:620–624.

Orstavik D. Time-course and risk analyses of the development and healing of chronic apical periodontitis in man. Int Endod J 1996;29:150–155.

Rubinstein RA, Kim S. Long-term follow-up of cases considered healed one year after apical microsurgery. J Endod 2002;28:378–383.

Rud J, Andreasen JO, Jensen JE. Radiographic criteria for the assessment of healing after endodontic surgery. Int J Oral Surg 1972;1:195–214.

Song M, Nam T, Shin SJ, Kim E. Comparison of clinical outcomes of endodontic microsurgery: 1 year versus long-term follow-up. J Endod 2014;40:490–494.

Yu VS, Messer HH, Shen L, Yee R, Hsu CY. Lesion progression in post-treatment persistent endodontic lesions. J Endod 2012;38:1316–1321.

Restoration of Endodontically Treated Teeth

Abramovitz L, Lev R, Fuss Z, Metzger Z. The unpredictability of seal after post space preparation: A fluid transport study. J Endod 2001;27:292–295.

American Association of Endodontists. AAE Position Statement 2007. Implants. https://www.aae.org/uploadedfiles/publications_and_research/guidelines_and_position_statements/implantsstatement.pdf. Accessed 22 December 2015.

Bourgeois RS, Lemon RR. Dowel space preparation and apical leakage. J Endod 1981;7:66–69.

Cameron CE. The cracked tooth syndrome: Additional findings. J Am Dent Assoc 1976;93:971–975.

Cheron RA, Marshall SJ, Goodis HE, Peters OA. Nanomechanical properties of endodontically treated teeth. J Endod 2011;37:1562–1565.

Chugal NM, Clive JM, Spangberg LS. Endodontic treatment outcome: Effect of the permanent restoration. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:576–582.

Doyle SL, Hodges JS, Pesun IJ, Baisden MK, Bowles WR. Factors affecting outcomes for single-tooth implants and endodontic restorations. J Endod 2007;33:399–402.

Doyle SL, Hodges JS, Pesun IJ, Law AS, Bowles WR. Retrospective cross sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants. J Endod 2006;32:822–827.

Fan B, Wu MK, Wesselink PR. Coronal leakage along apical root fillings after immediate and delayed post space preparation. Endod Dent Traumatol 1999;15:124–126.

Gargiulo A, Krajewski J, Gargiulo M. Defining biologic width in crown lengthening. CDS Rev 1995;88:20–23.

Gatten DL, Riedy CA, Hong SK, Johnson JD, Cohenca N. Quality of life of endodontically treated versus implant treated patients: A university-based qualitative research study. J Endod 2011;37:903–909.

Goldfein J, Speirs C, Finkelman M, Amato R. Rubber dam use during post placement influences the success of root canal-treated teeth. J Endod 2013;39:1481–1484.

Guthrie RC, DiFiore PM. Treating the cracked tooth with a full crown. J Am Dent Assoc 1991;122:71–73.

Helfer AR, Melnick S, Schilder H. Determination of the moisture content of vital and pulpless teeth. Oral Surg Oral Med Oral Pathol 1972;34:661–670.

Hiatt WH. Incomplete crown-root fracture in pulpal-periodontal disease. J Periodontol 1973;44:369–379.

Iqbal MK, Kim S. For teeth requiring endodontic treatment, what are the differences in outcomes of restored endodontically treated teeth compared to implant-supported restorations? Int J Oral Maxillofac Implants 2007;22(suppl):96–116.

Juloski J, Radovic I, Goracci C, Vulicevic ZR, Ferrari M. Ferrule effect: A literature review. J Endod 2012;38:11–19.

Katz A, Tamse A. A combined radiographic and computerized scanning method to evaluate remaining dentine thickness in mandibular incisors after various intracanal procedures. Int Endod J 2003;36:682–686.

Krell KV, Rivera EM. A six year evaluation of cracked teeth diagnosed with reversible pulpitis: Treatment and prognosis. J Endod 2007;33:1405–1407.

Kuttler S, McLean A, Dorn S, Fischzang A. The impact of post space preparation with Gates-Glidden drills on residual dentin thickness in distal roots of mandibular molars. J Am Dent Assoc 2004;135:903–909.

Madison S, Zakariasen KL. Linear and volumetric analysis of apical leakage in teeth prepared for posts. J Endod 1984;10:422–427.

Mattison GD, Delivanis PD, Thacker RW Jr, Hassell KJ. Effect of post preparation on the apical seal. J Prosthet Dent 1984;51:785–789.

Reeh ES, Messer HH, Douglas WH. Reduction in tooth stiffness as a result of endodontic and restorative procedures. J Endod 1989;15:512–516.

Sagsen B, Zortuk M, Ertas H, Er O, Demirbuga S, Arslan H. In vitro fracture resistance of endodontically treated roots filled with a bonded filling material or different types of posts. J Endod 2013;39:1435–1437.

Seo DG, Yi YA, Shin SJ, Park JW. Analysis of factors associated with cracked teeth. J Endod 2012;38:288–292.

Shillingburg HT. Fundamentals of Fixed Prosthodontics, ed 3. Chicago: Quintessence, 1997.

Sorensen JA, Martinoff JT. Intracoronal reinforcement and coronal coverage: A study of endodontically treated teeth. J Prosthet Dent 1984;51:780–784.

Swanson K, Madison S. An evaluation of coronal microleakage in endodontically treated teeth. Part I. Time periods. J Endod 1987;13:56–59.

Torabinejad M, Ung B, Kettering JD. In vitro bacterial penetration of coronally unsealed endodontically treated teeth. J Endod 1990;16:566–569.

Turp JC, Gobetti JP. The cracked tooth syndrome: An elusive diagnosis. J Am Dent Assoc 1996;127:1502–1507.

Ethics and the Law

American Dental Association. Principles of Ethics and Code of Professional Conduct. With official advisory opinions revised to April 2012. https://www.ada.org/~/media/ADA/About%20the%20ADA/Files/code_of_ethics_2012.ashx. Accessed 22 December 2015.

Occupational Safety and Health Administration. Training Requirements in OSHA Standards. https://www.osha.gov/Publications/osha2254.pdf. Accessed 22 December 2015.

US Department of Health & Human Services. Health Information Privacy. http://www.hhs.gov/hipaa/index.html. Accessed 22 December 2015.

9

Traumatic Dental Injuries

The abilities to diagnose and properly treat traumatic dental injuries are essential skills for the practicing endodontist. A joint symposium in 2012 between the American Association of Endodontists (AAE) and the American Academy of Pediatric Dentistry resulted in the release of a new set of guidelines on the diagnosis and treatment of traumatic dental injuries. The American Board of Endodontics (ABE) exam requires intimate knowledge of these guidelines in addition to general familiarity with the literature on which these guidelines are based. A 2013 supplement in the *Journal of Endodontics* was published with a specific focus on dental trauma in conjunction with the release of these guidelines. This chapter presents a review of traumatic dental injuries and their management and will largely focus on the permanent dentition.

Epidemiology

Trauma to the orofacial region is a common finding in young patients, as reviewed by Andersson (Fig 9-1). Five percent of all bodily injuries are to the orofacial region, and this percentage increases substantially in preschool-aged children, where as many as 17% of all bodily injuries are to the orofacial region. Population-based studies indicate that the incidence of traumatic dental injuries is between 1% and 3%, and the prevalence is from 20% in the permanent dentition to 30% in the primary dentition. Of all patients seeking treatment for injuries to the orofacial region, 92% have experienced dental trauma, 28% have been affected by soft tissue injuries, and 6% have suffered jaw fractures. Injuries occur most frequently during the first 10 years of life and are rarely encountered after the age of 30. Males are more frequently affected by traumatic injuries than females. Furthermore, children known to participate in risk-related behaviors are more frequently affected than those who abstain. A summary of risk factors for dental injuries is pictured in Fig 9-2.

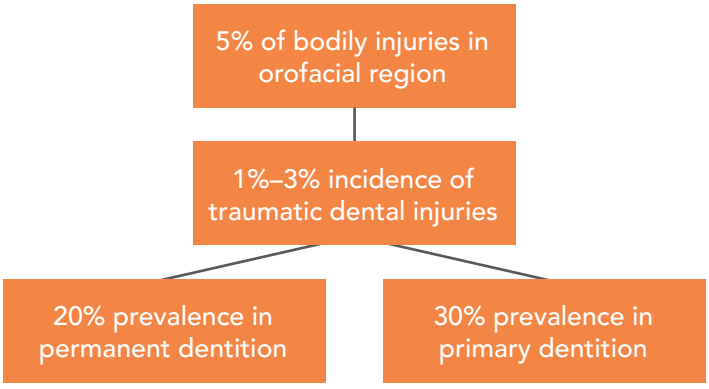

Fig 9-1 Epidemiology of traumatic dental injuries (Andersson).

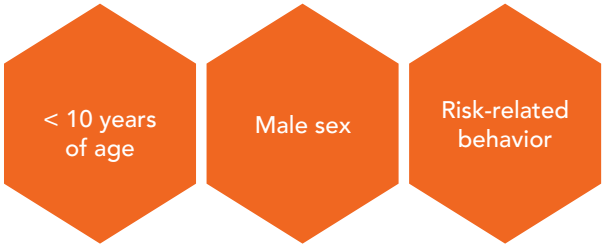

Fig 9-2 Factors associated with increased risk of traumatic dental injury (Andersson).

The etiologic factors related to traumatic injuries are largely a function of patient age, according to Andersson (Fig 9-3). In preschool-aged children, falls are the most common cause of traumatic injuries. School-aged children experience injuries most frequently in relation to sports incidents. In adolescents and young adults, trauma most frequently occurs in relation to assaults and traffic incidents and is often related to alcohol use.

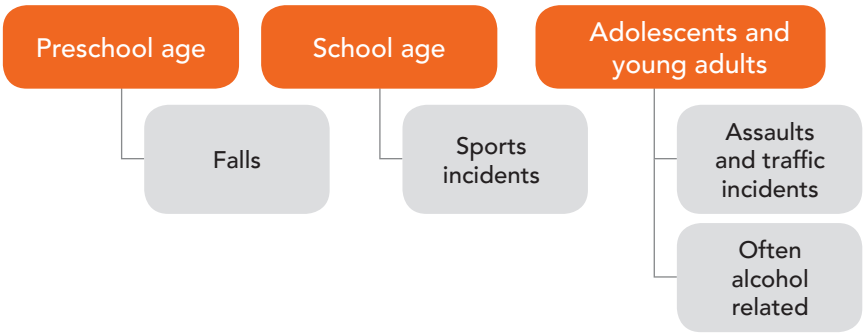

Fig 9-3 Typical injury etiology by age (Andersson).

Pathophysiology

Pulpal inflammation may occur following trauma, and spontaneous healing can occur in the absence of bacteria without progression to endodontic infection. Kakehashi et al's classic experiment on germ-free rats demonstrated that bacteria are necessary for the progression of pulpal disease to apical periodontitis (Fig 9-4). Without bacterial ingress, spontaneous healing of the pulp can occur (Andreasen). Bergenholtz proposed that microcracks in tooth structure might allow progression of bacteria into the necrotic pulps of traumatized teeth without direct exposure. Nagaoka et al suggested some immune protection is offered by vital pulp whose loss, in the case of pulpal necrosis, might allow ingress of bacteria via dentinal tubules. Once the key combination of bacteria and inflammation is present in the pulp, pulpal necrosis is inevitable.

Fig 9-4 Requirements for apical periodontitis (Kakehashi et al).

Diagnosis

The first step in appropriate management of traumatic orofacial injuries is to perform a rapid physical assessment. Steelman advised a primary survey following airway, breathing, circulation, disability, and exposure (ABCDE). This primary survey ensures appropriate airway maintenance and cervical spine protection, adequate breathing and ventilation, intact circulation without evidence of shock, evaluation for neurological disability, and that the patient is exposed for a full examination. A secondary survey delves further into the medical history, requiring a patient interview for allergies, medications, past illnesses, last meal, the events and environment leading to trauma, and tetanus vaccination history.

Following the rapid physical assessment and secondary survey, an oral examination should be performed. An early assessment should triage for appropriate treatment timing. Bakland and Andreasen proposed division of traumatic dental injuries into those with *acute priority* requiring treatment within hours, those with *subacute priority* where delaying treatment several hours should not affect the prognosis, and those with *delayed priority* where a delay—even beyond 24 hours—should not affect the prognosis. Acute priority injuries include root fractures, alveolar fractures, lateral luxation injuries, extrusive luxation injuries, and avulsion injuries. Subacute priority injuries include complicated crown fractures with pulp exposures, concussions, subluxations, and intrusions. Delayed priority injuries include uncomplicated crown fractures without pulp exposures. A summary of Bakland and Andreasen's recommendations for prioritizing dental injuries can be found in Fig 9-5.

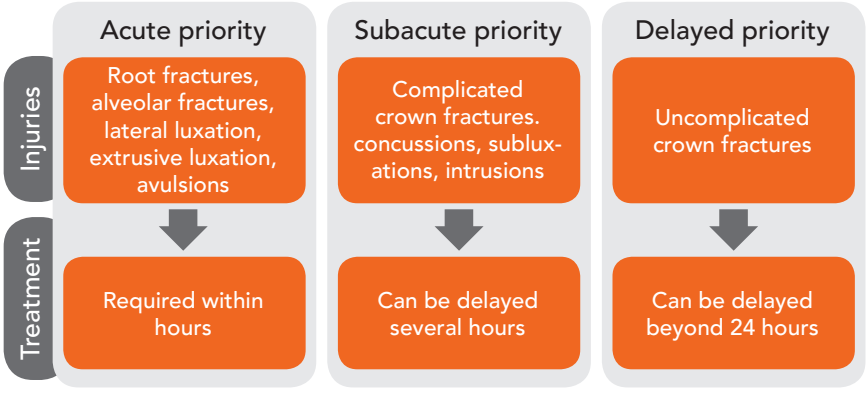

Fig 9-5 Prioritization treatment (Bakland and Andreasen).

Diagnosis of traumatic dental injuries requires the accurate collection of both clinical and radiographic data. Clinical data include both pulp sensitivity tests and periradicular tests. Levin reviewed specific pulpal and periradicular testing that should be performed to come to a definitive diagnosis. Radiographic data should include periapical radiographs from multiple angulations, as recommended by the AAE guidelines. If an alveolar fracture is suspected, Levin suggested the use of a panoramic radiograph. Ball et al suggested that cone beam computed tomography (CBCT) should be considered depending on the severity of injury, as this may provide a more reliable assessment of the extent of the injury. Figure 9-6 summarizes the recommended systematic approach to evaluating patients presenting with traumatic dental injuries.

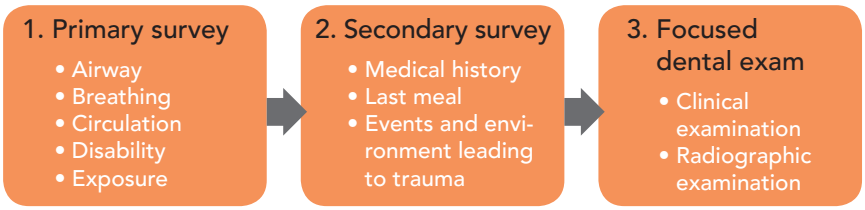

Fig 9-6 Recommended approach to the emergency patient presenting with a traumatic dental injury (Steelman).

Clinical exam

Baseline pulp sensitivity testing should occur at the earliest possible time following the injury. The testing modality selected, namely thermal or electric pulp test (EPT), should take into consideration the type of injury and the age of the patient.

In a tooth with a mature apex, the AAE guidelines consider a lack of response to pulp sensitivity testing 3 months post-trauma as an indication of pulpal necrosis, and they advise that pulpal necrosis should be diagnosed by at least two signs or symptoms. Bhaskar and Rappaport found vital tissue in traumatized teeth nonresponsive to traditional pulp sensitivity testing and advised a delay in diagnosis when relying on these methods alone due to proposed transient sensory deficiencies. Ozcelik et al's histologic analysis showed intramyelin edema, axonal swelling, and partial loss of the myelin sheath in the neurons of pulps exposed in complicated coronal fractures, supporting this theory of neuronal injury. Although not yet available to the clinician, Levin suggested that, in the future, true pulp vitality tests that evaluate the presence of vital tissue rather than sensitivity of the tissue, such as laser Doppler flowmetry, pulse oximetry, dual wavelength spectrophotometry, and thermography, may bypass these limitations.

Levin recommended pulp sensitivity testing immediately post-trauma and again at 2 weeks, 4 weeks, 6 to 8 weeks, 6 months, and 1 year. Thermal testing is considered the gold standard among available tests. EPT should be considered a secondary test, as its accuracy depends on the circumstances. For example, Fulling and Andreasen found that EPT was not accurate in immature teeth due to late development of the responsive Aδ nerve fibers. EPT is considered most useful to confirm suspected necrosis, based on reports by Peters et al and Gopikrishna et al of its high positive predictive value. No response to EPT is highly predictive of a necrotic pulp. Conversely, Ketterl reported an age-related reduction in dentinal tubule size and therefore the fluid important in thermal testing, increasing the utility of EPT in the older patient.

Levin further suggested that periradicular testing should include an assessment of mobility, percussion, and palpation testing. Single-tooth mobility can assess the degree of dislodgement of the tooth from the socket or a cervically located root fracture, whereas mobility of several teeth in unison is often indicative of alveolar fracture. Percussion sensitivity in the acute presentation of trauma can indicate recent attachment damage, whereas protracted or new percussion sensitivity at follow-up oftentimes indicates infection or the presence of an alveolar fracture. A metallic tone on percussion can indicate that a tooth is locked in bone related to lateral or intrusive luxation injury or, in late stages of healing, that ankylosis has occurred. Palpation allows one to feel alveolar fractures or the dislocation of a luxation injury. Palpation sensitivity at follow-up can indicate infection or the presence of a nonhealing alveolar fracture.

Clinical findings

Clinical findings are discussed by diagnostic entity as defined by the AAE guidelines.

Fractures (Fig 9-7)

Crown fractures can be divided into uncomplicated and complicated. For these injuries, pulp sensitivity testing and mobility are generally normal, and teeth are not sensitive to percussion. The exposed pulp can be sensitive to stimuli. If percussion tenderness is noted during examination of a crown fracture, it is important to assess the patient for other traumatic injuries, namely concomitant luxation injuries, crown/root fractures and root fractures.

- **Uncomplicated fractures** involve enamel and dentin without pulp exposure.
- **Complicated fractures** involve enamel, dentin, and an exposed pulp.
- **Crown/root fractures** involve enamel, dentin, and cementum and extend subgingivally. A pulp exposure may or may not be noted. However, normal responses to pulp sensitivity testing are expected. Mobility and percussion tenderness are often present.
- **Root fractures** involve root structure and may be located in the apical, middle, or cervical thirds of the root. The coronal fragment may be displaced and is often mobile, whereas the apical segment is not often displaced. Percussion tenderness is common. Pulp sensitivity testing may initially be nonresponsive, indicative of transient pulpal damage.
- **Alveolar fractures** involve the fracture and mobility of a bony segment containing a single tooth or several teeth. Often, if the fractured segment includes more than one tooth, mobility of several teeth in concert is common. The fractured segment may be displaced, creating occlusal interference.

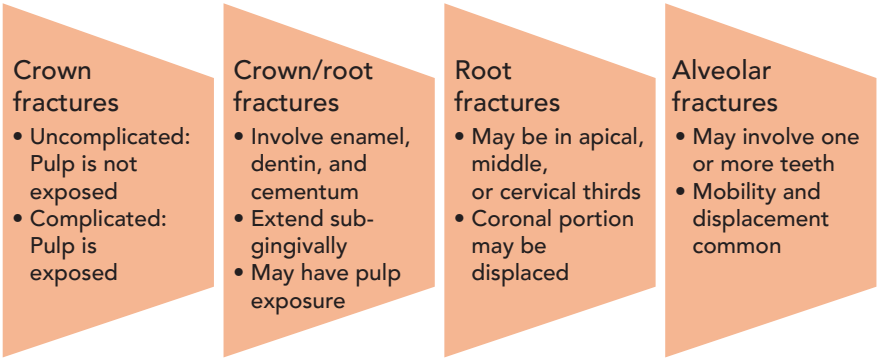

Fig 9-7 Classification of fractures (AAE trauma guidelines).

Luxation-type injuries (Fig 9-8)

- **Concussion injuries** present with percussion tenderness but lack displacement or mobility. Pulp sensitivity testing is usually normal.
- **Subluxation injuries** present with percussion tenderness and mobility but lack displacement. Pulp sensitivity testing may initially be nonresponsive, indicative of transient pulpal damage, but is typically normal.

- **Extrusive luxation** injuries present with outward or incisal displacement and percussion tenderness. Pulp sensitivity testing is often nonresponsive. Mobility of the extruded tooth is often noted.
- **Lateral luxation** injuries present with lateral displacement, oftentimes associated with a fracture of the facial cortical bone that can be palpable. The tooth may appear immobile or locked in bone and is typically percussion tender. Pulp sensitivity testing is often nonresponsive.
- **Intrusive luxation** injuries present with displacement of the tooth into the alveolar bone, oftentimes associated with palpable fracture of the alveolar process. The tooth may appear immobile or locked and is typically percussion tender. Pulp sensitivity testing is often nonresponsive.

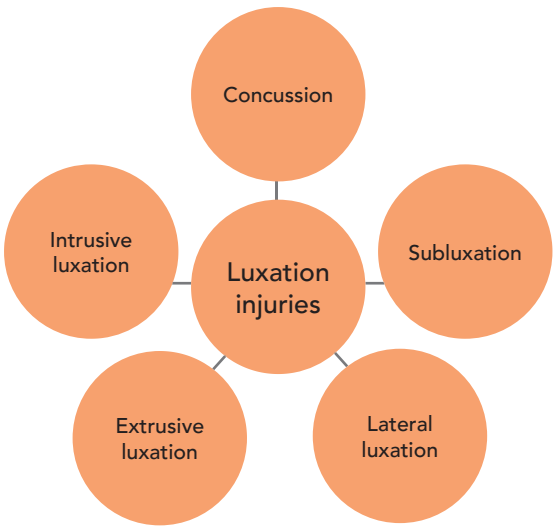

Fig 9-8 Luxation-type injuries (AAE trauma guidelines).

Avulsions

Avulsion involves complete loss of a tooth from its socket, with possible fracture of the associated alveolar bone. Treatment plans must take into account the stage of root maturation, extraoral dry time prior to replantation, and particular extraoral storage media used.

Radiographic examination

Radiographic examination is essential following dental trauma. The AAE guidelines generally advise taking at least two periapical radiographs from different horizontal angulations. Depending on the type of injury suspected based on the history and clinical and preliminary radiographic exams, additional radiographs are often suggested as outlined below. Ball et al suggested the addition of CBCT imaging in many cases of traumatic dental injuries.

Radiographic findings

Radiographic findings are unique to each diagnostic entity, according to the AAE guidelines. Examples of traumatic injuries are shown in Fig 9-9.

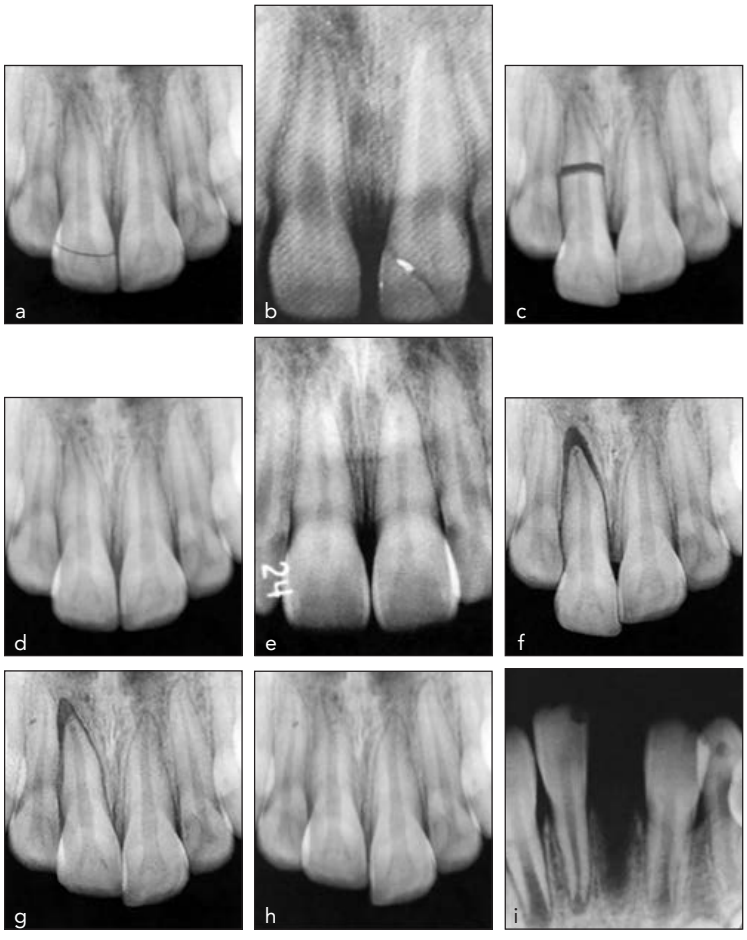

Fig 9-9 Radiographic presentations of traumatic dental injuries. *(a)* Uncomplicated crown fracture. *(b)* Complicated crown fracture. *(c)* Horizontal root fracture. *(d)* Concussion. *(e)* Subluxation. *(f)* Extrusive luxation. *(g)* Lateral luxation. *(h)* Intrusive luxation. *(i)* Avulsion.

Fractures

In cases of suspected tooth or alveolar fracture, one occlusal and at least two periapical radiographs of varying horizontal angulation should be taken. When a root fracture is suspected, Bender and Freedland suggested that periapical radiographs of several varying

vertical angulations can be helpful. If soft tissue lacerations are noted, radiographs should be taken of the lacerations to identify any tooth fragments or foreign material contained within the soft tissue wound. CBCT images are suggested when there is suspicion of a root fracture; however, the resolution of CBCT images may not identify certain fractures if fragment separation is insufficient. Brady et al found that vertical root fractures smaller than 50 μm were not detectable in the particular CBCT machines studied. CBCT or panoramic radiographic images are suggested when there is suspicion of alveolar fracture.

Luxation-type injuries

For all luxation-type injuries, two periapical radiographs from varying horizontal angulations are recommended to evaluate for displacement. For extrusive, lateral, and intrusive luxation injuries, an occlusal film should be added to the radiographic exam. The periodontal ligament (PDL) should be closely evaluated, as extrusive and lateral luxation injuries often present with an enlarged PDL space, whereas intrusive luxation injuries may present with either full or partial loss of the PDL. A CBCT image is suggested depending on the severity of the injuries, specifically to evaluate the PDL and the alveolar bone for the presence or absence of fracture. If a tooth is completely intruded, a lateral cephalogram is recommended to evaluate for penetration into the nasal cavity.

Avulsions

For all avulsion injuries, two periapical radiographs from mesial and distal angulations are recommended as well as a CBCT image to confirm proper repositioning and evaluate for any alveolar fractures.

Treatment Protocols

Splinting

The International Association of Dental Traumatology (IADT) states that, following trauma, splinting is used to maintain the correct tooth position, provide patient comfort, and improve function (Diangelis et al). The AAE guidelines exclusively advise the use of flexible splints when splinting is indicated, with wire diameter not to exceed 0.016 inches or 0.4 mm. Von Arx et al provided an argument for nonrigid splinting. In studies on nonhuman primates, PDL damage and replacement resorption were noted when rigid splints were used following traumatic injuries. In addition to the use of flexible splints, shorter splinting times are indicated. Nasjleti et al found that a splinting time of 7 days resulted in significantly less replacement resorption than 30 days, following experimental avulsive injuries in monkeys. The guidelines for splinting are described in Fig 9-10. Table 9-1 summarizes recommended splint times for each diagnostic entity.

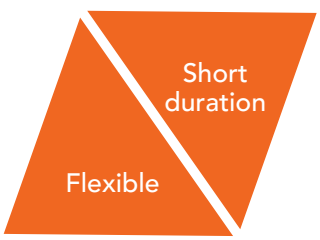

Fig 9-10 Requirements for splinting (AAE trauma guidelines).

Table 9-1 Summary of treatment protocols for traumatic dental injuries

Injury	Splinting time	Follow-up times
Crown fractures	NA	6–8 w, 1 y
Crown/root fractures	NA	6–8 w, 1 y
Root fractures	4 w if in the middle or apical third; 4 m if in the coronal third	4 w, 6–8 w, 4 m, 6 m, 1 y, yearly for 5 y
Alveolar fracture	4 w	4 w, 6–8 w, 4 m, 6 m, 1 y, yearly for 5 y
Concussion	NA	2 w, 4 w, 6–8 w, 6 m, 1 y, yearly for 5 y
Subluxation	2 w	2 w, 4 w, 6–8 w, 6 m, 1 y, yearly for 5 y
Extrusive luxation	2 w	2 w, 4 w, 6–8 w, 6 m, 1 y, yearly for 5 y
Lateral luxation	2 w; if extensive luxation, can splint 4 w	2 w, 4 w, 6–8 w, 6 m, 1 y, yearly for 5 y
Intrusive luxation	2 w; if extensive luxation, can splint 4 w	2 w, 4 w, 6–8 w, 6 m, 1 y, yearly for 5 y
Avulsed tooth, mature apex	1–2 w	2 w, 4 w, 3 m, 6 m, 1 y, yearly for 5 y
Avulsed tooth, immature apex	1–2 w; if extraoral dry time > 60 min, splint 4 w	2 w, 4 w, 3 m, 6 m, 1 y, yearly for 5 y

NA, not applicable; min, minute; w, weeks; m, months; y, years.

Fractures

- **Crown fractures** should be treated with appropriate restorative materials or rebonding of available tooth fragments. If a definitive restoration cannot be placed immediately, sensitive areas of exposed dentin in uncomplicated fractures can be covered with glass ionomer or bonded resins. In complicated fractures, it is important to preserve pulp vitality with pulp capping or partial pulpotomy using calcium hydroxide or mineral trioxide aggregate.
- **Crown/root fracture** management depends on the extent of injury. Without a pulp exposure, the fractured segment can be removed with or without a gingivectomy to restore. If pulp exposure is present, immature teeth should be managed with a partial pulpotomy to attempt to maintain vitality of the root pulp. Mature teeth should be treated with root canal therapy and restored appropriately. More extensive crown/root fractures may require orthodontic or surgical extrusion for restoration or may even require extraction.
- **Root fractures** may present with full loss of the coronal segment, in which case they should be managed as avulsive injuries, as described later in this chapter. Repositioning of a displaced coronal segment should occur as soon as possible, and following radiographic confirmation of correct positioning, a flexible splint should be placed for 4 weeks

or as long as 4 months for cervically located root fractures. If pulpal necrosis develops, root canal therapy should be completed on the coronal segment only to the level of the fracture with the use of calcium hydroxide as an intracanal medicament. Cvek et al (2001) reported markedly improved success rates when root canal therapy was completed only to the level of the fracture rather than beyond.

- **Alveolar fractures** should be managed by repositioning and stabilization with a flexible splint for 4 weeks.

Clinical and radiographic follow-ups for crown and crown/root fractures should be performed at 6 to 8 weeks and 1 year post-trauma. Root and alveolar fractures should have clinical and radiographic follow-ups at 4 weeks, 6 to 8 weeks, 4 months, 6 months, 1 year, and yearly thereafter for 5 years. Complications requiring intervention can develop at any time. Andreasen and Hjorting-Hansen described the types of healing following root fracture: calcified tissue, connective tissue wherein the pulp space is often obliterated, a combination of these two tissues, or nonhealing cases with granulation tissue at the site of fracture and associated pulpal necrosis (Fig 9-11). At follow-up, it is important to note the type of healing occurring for root fractures. Table 9-1 summarizes follow-up times for all diagnostic entities.

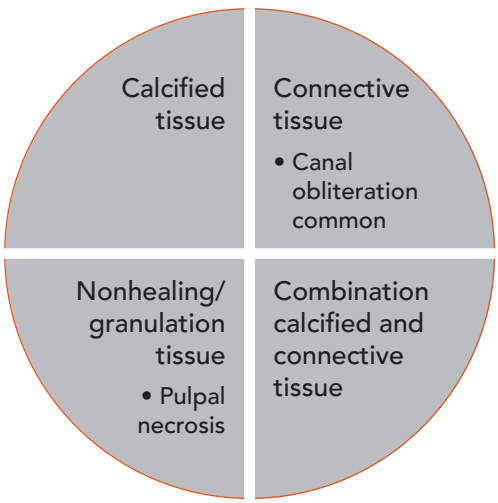

Fig 9-11 Types of healing following root fracture (Andreasen and Hjorting-Hansen).

Luxation-type injuries

- **Concussion injuries** do not require immediate treatment.
- **Subluxation injuries** may be splinted for up to 2 weeks for patient comfort.
- **Extrusive luxation injuries** should be immediately repositioned and splinted for up to 2 weeks.
- **Lateral luxation injuries** should be repositioned, which may involve disengagement from a bony lock, followed by splinting for 2 weeks or up to 4 weeks for more extensive displacement.
- **Intrusive luxation injuries** are managed differently based on the stage of root development and the extent of intrusion (Fig 9-12):

– Teeth with incompletely formed apices intruded up to 7 mm should be allowed to spontaneously re-erupt. If re-eruption does not occur within 3 weeks, orthodontic repositioning should be initiated. With intrusion greater than 7 mm, surgical or orthodontic repositioning should be initiated within 3 weeks.
– Mature teeth intruded up to 3 mm should be allowed to spontaneously re-erupt without intervention. If re-eruption does not occur within 2 to 3 weeks, surgical or orthodontic repositioning should be initiated before ankylosis develops. For intrusion between 3 and 7 mm, surgical or orthodontic repositioning should be initiated within 3 weeks. For intrusion greater than 7 mm, the tooth should be surgically repositioned and splinted for 2 weeks or up to 4 weeks for extensive displacement.

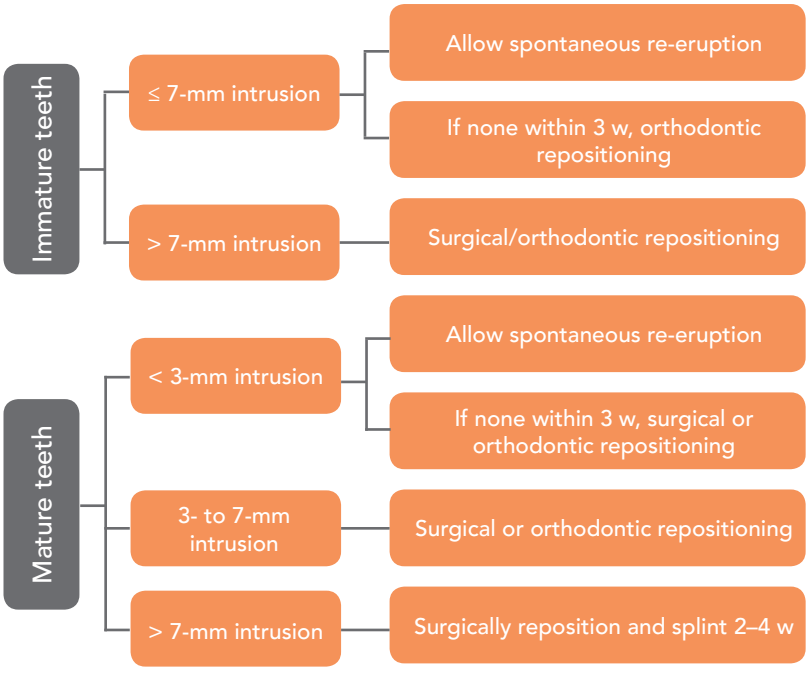

Fig 9-12 Treatment guidelines for intrusive luxations (AAE trauma guidelines). w, weeks.

For concussion, subluxation, extrusive, and lateral luxation injuries, the pulpal response to sensitivity and vitality testing should be monitored for 3 months before a definitive pulpal diagnosis and decision to initiate endodontic therapy is made, unless pulpal necrosis is confirmed sooner with at least two signs or symptoms. Root canal therapy is the treatment of choice for mature teeth with pulpal necrosis, whereas pulp revascularization therapy or apexification should be considered for immature teeth. Intrusive luxation injuries of mature teeth are likely to result in pulpal necrosis, and root canal therapy should be initiated within 2 weeks of injury with 4 weeks of intracanal calcium hydroxide following pulpectomy. Immature teeth with intrusive luxation injuries may escape pulpal necrosis and should be monitored the same as the other luxation-type injuries. There is currently no strong evidence to support orthodontic versus surgical repositioning of intrusively luxated teeth that do not spontaneously re-erupt. In a systematic review, Al Khalifa and Al Azemi reported

only 5% to 12% failure of spontaneous re-eruption and no difference in adverse outcomes between orthodontic versus surgical repositioning of these teeth.

Clinical and radiographic follow-ups for all luxation-type injuries are recommended at 2 weeks, 4 weeks, 6 to 8 weeks, 6 months, 1 year, and yearly thereafter for 5 years. Complications requiring intervention can develop at any time. Table 9-1 summarizes follow-up times for all diagnostic entities.

Avulsions

The goal in treatment of avulsed teeth is to maintain the PDL, as most complications result from injury to this structure. Van Hassel et al showed that removal of the PDL resulted in severe, progressive root resorption. Though the dental pulp is anticipated to undergo necrosis following avulsive injuries, particularly in mature teeth, pulpal infection should be minimized to prevent progression of apical periodontitis. According to Andreasen et al, the prognosis of avulsed teeth is highly dependent on the status of root maturation, the time to replantation, and the extraoral storage media. Therefore, treatment protocols differ depending on these variables.

Historically, because avulsed teeth with immature roots or open apices had a poor prognosis following replantation, older guidelines advised that these not be replanted in all cases. More recently, thought has shifted, and modifications to treatment protocols have been made in the new AAE guidelines, including the replantation of these teeth despite the poor prognosis. This recommendation was made in order to maintain bone volume in the area of the avulsion and improve the feasibility of dental implant placement in the fully grown patient.

Replantation of avulsed teeth is recommended as soon as possible because extraoral dry time should ideally fall under 60 minutes. Extraoral storage should be in a physiologic storage media because dry storage leads to rapid PDL cell death and inevitable root resorption, and nonphysiologic solutions, such as water, can lead to cell lysis via osmosis. Hank's balanced salt solution (HBSS), saline (supported by Trope and Friedman), and milk (supported by Blomlof) are the recommended storage media (Fig 9-13). More recently, novel storage media including soy milk and coconut water have been proposed due to their rich nutrient content. In a study by de Paula Reis et al, coconut water showed promise, but increased levels of replacement resorption were noted with soy milk storage.

Fig 9-13 Ideal storage media for avulsed teeth (AAE trauma guidelines).

Following avulsion injuries, systemic antibiotics are recommended. Doxycycline is the drug of choice in patients over 12 years old. Due to the risks of staining the developing dentin that are associated with doxycycline, amoxicillin is recommended for patients under 12 years old. The recommendation of doxycycline is extrapolated from Sae-Lim et al's report of tetracycline's antiresorptive properties due to reduced osteoclast motility and collagenase function. Hammarström et al supported the general use of systemic antibiotics to prevent external root resorption, and his work is the basis for the suggested use of amoxicillin when doxycycline is contraindicated. If tetanus coverage is uncertain, the patient should be referred to their physician for a tetanus booster.

Specific treatment recommendations vary according to the status of root maturation, the extraoral dry time, and the particular extraoral storage media used (Fig 9-14). Following are the guidelines based on these variables:

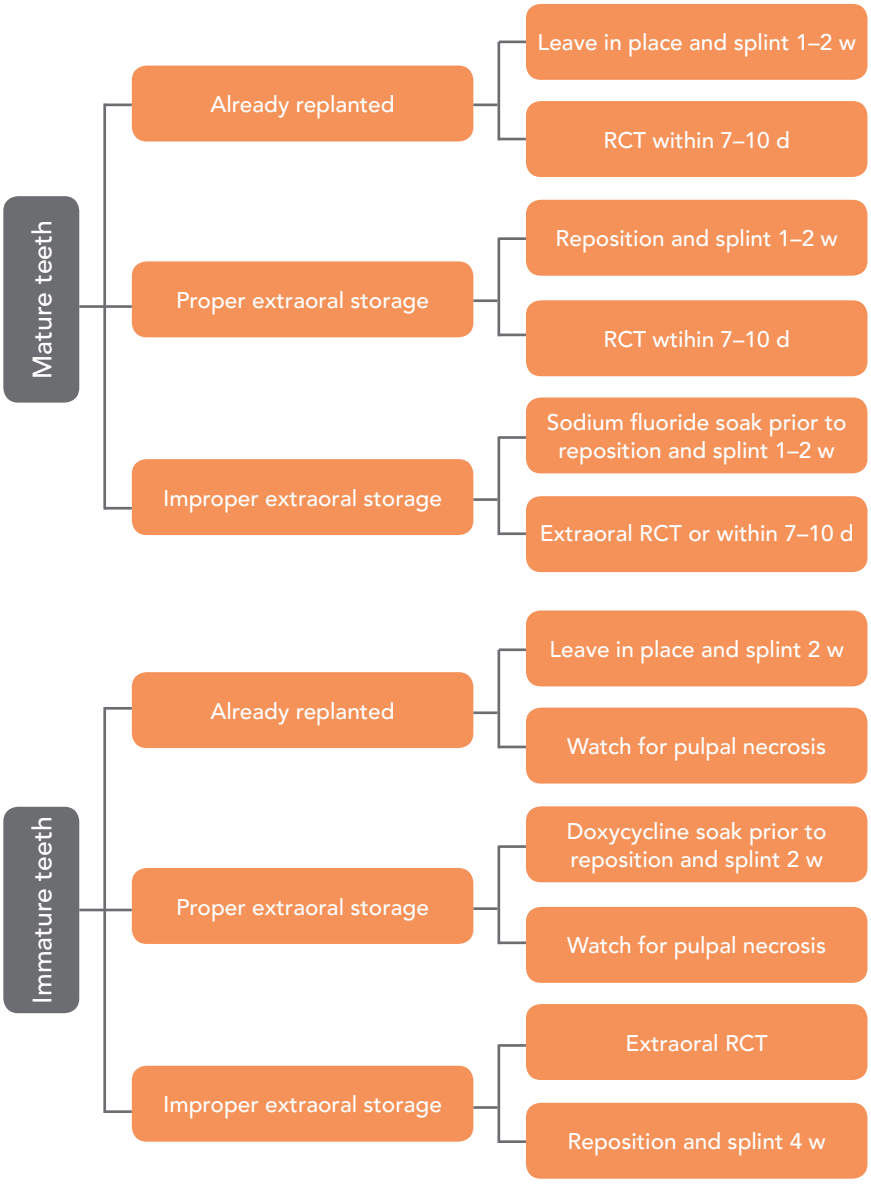

Fig 9-14 Treatment guidelines for avulsed teeth (AAE trauma guidelines) d, days; RCT, root canal treatment; w, weeks.

Mature teeth with closed apex

- If the tooth has already been replanted, it should be left in place, and its position should be confirmed radiographically. A flexible splint should be placed for 1 to 2 weeks. Root canal therapy should be initiated between 7 and 10 days postreplantation while the splint is still in place. Interappointment intracanal calcium hydroxide should be placed for at least 4 weeks. Trope et al reported markedly reduced external inflammatory root resorption when long-term calcium hydroxide was used following avulsive injuries. Alternatively, root canal therapy could be initiated immediately following replantation using a corticosteroid as the intracanal medication for at least 2 weeks. Bryson et al reported reduced resorption with intracanal Ledermix cement [Dentsply].
- If the tooth has been kept in a physiologic storage medium, such as HBSS, saline, or milk, and/or stored dry for less than 60 minutes, the root and socket should be cleaned gently with a saline rinse and the tooth replanted under local anesthesia. Placement should be confirmed radiographically before a flexible splint is placed for 1 to 2 weeks. Root canal therapy is indicated similarly to the previously replanted tooth.
- If the tooth has been kept dry and out of the mouth for more than 60 minutes, the root should be cleaned gently using dry gauze. Prior to replantation, a 20-minute soak in a 2% sodium fluoride solution can be considered as a means of potentially slowing replacement resorption, based on the work of Coccia, but is not an absolute recommendation. Replantation should occur as above, followed by placement of a flexible splint for 1 to 2 weeks. Root canal therapy could be completed extraorally prior to replantation or based on the same methodology recommended for the tooth that was already replanted on presentation. Replacement resorption is considered an inevitable complication with prolonged extraoral dry time because of PDL cell death, and decoronation according to the protocol proposed by Malmgren is advised once the tooth is greater than 1 mm infrapositioned. The decoronation procedure involves removal of the coronal tooth structure, with possible use of this fragment as a bonded temporary, followed by surgical access to remove any root filling material, contouring of the root structure, and reapproximation of the flap. Once replacement resorption is complete, a dental implant can be placed.

Immature teeth with open apex

- If the tooth has already been replanted, it should be left in place, and its position should be confirmed radiographically. A flexible splint should be placed for 2 weeks. Revascularization will ideally occur without endodontic intervention. Kling et al found an 18% incidence of revascularization following avulsion injuries in immature teeth with apical diameter greater than 1 mm. If evidence of pulpal necrosis is found before root maturation is complete, regenerative endodontic therapy or apexification is recommended.
- If the tooth has been kept in a physiologic storage medium, such as HBSS, saline, or milk, or stored dry for less than 60 minutes, the root and socket should be cleaned gently with a saline rinse and the tooth replanted under local anesthesia. Prior to replantation, a 20-minute soak in 1 mg/mL minocycline or doxycycline solution is indicated based on the work of Cvek et al (1990), which suggested its ability to improve revascularization. Placement should be confirmed radiographically before a flexible splint is placed for 2 weeks. As with the tooth that has already been replanted, revascularization will ideally occur without endodontic intervention, but if evidence of pulpal necrosis is found before root maturation is complete, regenerative endodontic therapy or apexification is recommended.

- If the tooth has been kept dry and out of the mouth for more than 60 minutes, the root should be cleaned gently using dry gauze. Root canal therapy should be completed extraorally prior to replantation. Replantation should occur as above, followed by placement of a flexible splint for 4 weeks. Replacement resorption is considered an inevitable complication with prolonged extraoral dry time due to PDL cell death, and decoronation according to the protocol proposed by Malmgren is advised once the tooth is greater than 1 mm infrapositioned. Since tooth loss is an anticipated eventuality, growth monitoring via height and weight tracking is suggested to determine when dental implant placement is advisable.

Clinical and radiographic follow-ups for all avulsion injuries are recommended at 4 weeks, 3 months, 6 months, 1 year, and yearly thereafter for 5 years. Complications requiring intervention can develop at any time. Table 9-1 summarizes follow-up times for all diagnostic entities.

Postoperative instructions

The AAE guidelines recommend a soft diet for 1 to 2 weeks following traumatic dental injuries, depending on the severity of injury. Good oral hygiene with the use of a soft-bristled toothbrush should be maintained, and a 0.12% chlorhexidine mouthrinse should be used twice daily for 2 weeks following luxation-type and avulsion injuries. Contact sports should be avoided for at least 2 weeks following avulsion injuries, and on resuming contact sports, mouth guards should be worn.

Primary dentition

The goal in management of traumatic injuries to the primary dentition is to minimize any damage to or lasting effects on the underlying permanent dentition, as the primary and permanent dentition are situated in extremely close proximity during development. The IADT generally advises restorative repair of more superficial fractures and potential pulp cap or at most extreme pulpotomy as the only endodontic treatment recommended (Malmgren). Should evidence of pulpal involvement necessitating greater intervention be present, extraction is advised. For luxation injuries where repositioning will not damage the successor tooth, careful repositioning can be considered; however, when the permanent tooth is at risk with repositioning, extraction is advised. Similarly, if postoperative complications arise requiring more invasive treatment, extraction of the primary tooth is recommended to preserve the health of its permanent successor. Splinting is advised for alveolar fractures but not for fracture or luxation injuries. Systemic antibiotics are not recommended. Otherwise, postoperative instructions involving maintenance of good hygiene and avoidance of further injury are similar to that of the permanent dentition. The IADT advises against replantation of avulsed primary teeth because of the high risk of damaging the permanent successors.

Prognosis

Outcomes for the traumatically injured tooth are a function of the extent and type of the injury, the stage of root development, and appropriate treatment at the time of injury, according to Levin. Table 9-2 presents commonly reported values for the percentages of teeth that maintain their vitality following traumatic dental injuries.

Table 9-2 Prognosis of traumatically injured teeth

Injury	Teeth that maintain vitality (%)	Reference
Crown fracture	94	Ravn
Root fracture	78	Andreasen et al
Concussion	97	Andreasen and Pedersen
Subluxation	94	Andreasen and Pedersen
Extrusive luxation	74	Andreasen and Pedersen
Lateral luxation	42	Andreasen and Pedersen
Intrusive luxation	15	Andreasen and Pedersen
Avulsion (mature tooth)	0	Kling et al
Avulsion (immature tooth)	18	Kling et al

Complications

The complications encountered following traumatic dental injuries to the permanent dentition are a function of the stage of tooth development and the extent and type of injury. The incidence of necrosis following injury ranges from as little as 3% in concussions to 100% in the instance of an avulsed mature tooth, as seen in Table 9-2. Hecova et al reported a higher incidence of pulpal necrosis following traumatic injuries of mature rather than immature teeth. The diagnosis of necrosis following trauma requires at least two signs or symptoms as false negative results to pulp sensitivity tests are frequently encountered, especially in the early stages following traumatic dental injuries. Pulpal necrosis requires treatment appropriate for the stage of root development, namely root canal therapy or apexification or regenerative procedures.

Another complication frequently encountered following traumatic dental injuries is pulp canal obliteration (PCO), which presents as narrowing of the canal space following trauma. Andreasen and Pedersen described PCO as occurring mainly in teeth with open apices and may be a result of revascularization. Histologically, Lundberg and Cvek described PCO tissues as osteoid with little inflammation or bacterial contamination. Jacobsen and Kerekes found PCO in as many as 40% of luxated teeth and as few as 8% of root-fractured teeth. PCO itself does not necessitate dental treatment, as Andreasen and Pedersen found that only 7% of teeth exhibiting PCO develop pulp necrosis. No higher frequency of pulp necrosis was noted in teeth with PCO when compared with normal teeth when subjected to caries, new trauma, orthodontic treatment, or complete crown restoration. Although the incidence of pulp necrosis in teeth displaying PCO seems to increase over time, Robertson et al advised against prophylactic endodontic treatment due to the relatively low lifetime risk of pulpal necrosis.

Resorption may also be encountered following traumatic injuries to the dentition. Internal, invasive cervical, external, and replacement resorption have all been noted following traumatic dental injuries. According to Tronstad, internal resorption is encountered following coronal necrosis of pulp tissue. Gabor et al reported histologic signs of internal resorption in as many as 50% of teeth with irreversible pulpitis and 77% of teeth with pulp

necrosis, even in the absence of clinical evidence. Invasive cervical root resorption is generally a late complication noted incidentally on clinical or radiographic examination.

Van Hassel et al pointed to PDL damage resulting from traumatic dental injuries as the major etiologic factor in external resorption. According to Trope, clastic cells from the PDL may be stimulated by necrotic pulp tissue to resorb dental hard tissues that have lost their protective, unmineralized layer. External inflammatory root resorption is a frequent complication encountered following avulsions but may occur following any traumatic injury. After an avulsion, maintenance of the PDL cells with prompt replantation and appropriate extraoral storage media, including HBSS, saline, and milk, may limit external resorption. Some evidence exists to support the use of systemic antibiotics following replantation as well. Hammarström et al suggested a preventive action of systemic antibiotics at the time of injury but not weeks later. Once resorption is initiated, however, Trope et al recommended root canal therapy with long-term calcium hydroxide to prevent progression of the resorptive defect. Alternatively, an intracanal corticosteroid such as Ledermix may be used according to Bryson et al.

Replacement resorption, otherwise known as *ankylosis*, is yet another potential complication following traumatic dental injuries. Tronstad described replacement resorption as osteoblastic repopulation of resorptive lacunae causing the defect to fill in with bone rather than cementum or PDL tissues. This process continues, leading to the replacement of root structure with bone. Common clinical findings with replacement resorption include a metallic tone to percussion, a lack of physiologic mobility, and infrapositioning in a growing patient. Like external resorption, this may occur following any traumatic injury. Following avulsion injuries with extraoral dry times beyond 60 minutes, the AAE guidelines suggest that replacement resorption is inevitable. It is therefore necessary to discuss this likely outcome with patients and parents to prepare them for its occurrence. Tronstad reported that replacement resorption itself is untreatable and will continue until the entire root structure is replaced by bone. In the growing patient where infrapositioning is a likely occurrence, the AAE guidelines advise decoronation once the position is 1 mm apical to the neighboring teeth. Malmgren described a useful protocol for decoronation, discussed previously with the treatment of avulsive injuries.

External apical root resorption (EARR), also referred to as *pressure resorption*, is associated with orthodontic movement of previously traumatized teeth (Brin et al). Obviously, orthodontic movement cannot be avoided for a patient's lifetime following traumatic injuries, but there is no consensus in the literature on the appropriate amount of time to wait to resume or commence orthodontic movement following trauma. Kindelan et al suggested a range of between 3 months for minor injuries and up to 1 year following severe injuries; however, they point out that these recommendations are based on empirical rather than scientific data. Pereira et al, based on experimental work on a rat model, noted that a delay of 15 to 30 days should be sufficient to resume orthodontic movement following a subluxation injury.

Figure 9-15 summarizes the potential complications following traumatic dental injuries, and Fig 9-16 shows radiographic examples of such complications. For additional information on resorption, please refer to chapter 10.

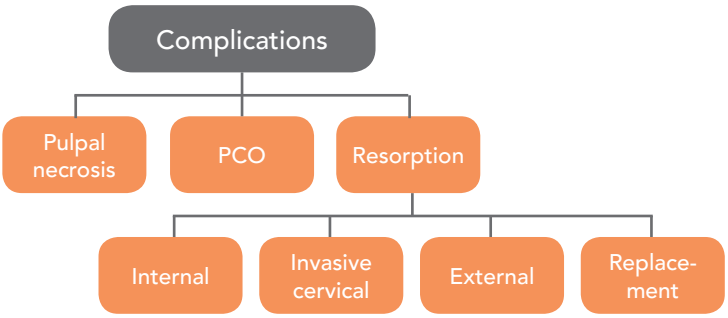

Fig 9-15 Potential complications following traumatic dental injuries.

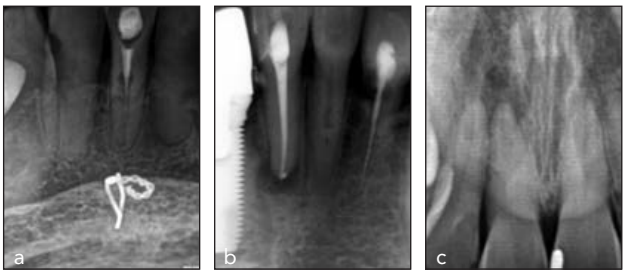

Fig 9-16 Examples of complications following traumatic dental injuries. *(a)* Internal root resorption, *(b)* external inflammatory root resorption and replacement resorption, and *(c)* PCO.

Prevention

Mouth guards are the best form of protection from traumatic dental injuries during contact sports. The AAE suggests fabricating custom-made mouth guards for proper extension ensuring adequate protection and comfort. Sigurdsson speculated that these mouth guards are helpful in the protection of hard tissue but not soft tissue injuries. Although some historic literature suggested that mouth guards might also prevent concussions and other brain injuries, Sigurdsson found no support for this.

In society, a wide range of actions can be implemented in the prevention of traumatic dental injuries. Andersson advised that the focus of these actions should be on personal and social education aimed at developing life skills and social policies against bullying and violence, physical environment changes, school health policies, alcohol policies, school provision of mouth guards, and links with health services.

Bibliography

Introduction

American Association of Endodontists. Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. http://www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 8 January 2016.

Epidemiology

Andersson L. Epidemiology of traumatic dental injuries. J Endod 2013;39:S2–S5.

Pathophysiology

Andreasen FM. Pulpal healing after luxation injuries and root fracture in the permanent dentition. Endod Dent Traumatol 1989;5:111–131.

Bergenholtz G. Micro-organisms from necrotic pulp of traumatized teeth. Odontol Revy 1974;25:347–358.

Kakehashi S, Stanley HR, Fitzgerald RJ. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. Oral Surg Oral Med Oral Pathol 1965;20:340–349.

Nagaoka S, Miyazaki Y, Liu HJ, Iwamoto Y, Kitano M, Kawagoe M. Bacterial invasion into dentinal tubules of human vital and nonvital teeth. J Endod 1995;21:70–73.

Diagnosis

American Association of Endodontists. Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. http://www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 8 January 2016.

Bakland LK, Andreasen JO. Dental traumatology: Essential diagnosis and treatment planning. Endod Topics 2004;7:14–34.

Ball RL, Barbizam JV, Cohenca N. Intraoperative endodontic applications of cone-beam computed tomography. J Endod 2013;39:548–557.

Bender IB, Freedland JB. Clinical considerations in the diagnosis and treatment of intra-alveolar root fractures. J Am Dent Assoc 1983;107:595–600.

Bhaskar SN, Rappaport HM. Dental vitality tests and pulp status. J Am Dent Assoc 1973;86:409–411.

Brady E, Mannocci F, Brown J, Wilson R, Patel S. A comparison of cone beam computed tomography and periapical radiography for the detection of vertical root fractures in nonendodontically treated teeth. Int Endod J 2014;47:735-746.

Fulling HJ, Andreasen JO. Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing. Scand J Dent Res 1976;84:286–290.

Gopikrishna V, Tinagupta K, Kandaswamy D. Comparison of electrical, thermal, and pulse oximetry methods for assessing pulp vitality in recently traumatized teeth. J Endod 2007;33:531–535.

Ketterl W. Age-induced changes in the teeth and their attachment apparatus. Int Dent J 1983;33:262–271.

Levin LG. Pulp and periradicular testing. Pediatr Dent 2013;35:113–119.

Ozcelik B, Kuraner T, Kendir B, Asan E. Histopathological evaluation of the dental pulps in crown-fractured teeth. J Endod 2000;26:271–273.

Peters DD, Baumgartner JC, Lorton L. Adult pulpal diagnosis. I. Evaluation of the positive and negative responses to cold and electrical pulp tests. J Endod 1994;20:506–511.

Steelman R. Rapid physical assessment of the injured child. J Endod 2013;39:S9–S12.

Treatment Protocols

Al Khalifa JD, Al Azemi AA. Intrusive luxation of permanent teeth: A systematic review of factors important for treatment decision-making. Dent Traumatol 2014;30:169–175.

American Association of Endodontists. Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. http://www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 8 January 2016.

Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed permanent incisors. 1. Diagnosis of healing complications. Endod Dent Traumatol 1995;11:51–58.

Andreasen JO, Hjorting-Hansen E. Intraalveolar root fractures: Radiographic and histologic study of 50 cases. J Oral Surg 1967;25:414–426.

Blomlof L. Milk and saliva as possible storage media for traumatically exarticulated teeth prior to replantation. Swed Dent J Suppl 1981;8:1–26.

Bryson EC, Levin L, Banchs F, Abbott PV, Trope M. Effect of immediate intracanal placement of Ledermix Paste on healing of replanted dog teeth after extended dry times. Dent Traumatol 2002;18:316–321.

Coccia CT. A clinical investigation of root resorption rates in reimplanted young permanent incisors: A five-year study. J Endod 1980;6:413–420.

Cvek M, Andreasen JO, Borum MK. Healing of 208 intra-alveolar root fractures in patients aged 7-17 years. Dent Traumatol 2001;17:53–62.

Cvek M, Cleaton-Jones P, Austin J, Lownie J, Kling M, Fatti P. Effect of topical application of doxycycline on pulp revascularization and periodontal healing in reimplanted monkey incisors. Endod Dent Traumatol 1990;6:170–176.

de Paula Reis MV, Moura CC, Soares PB, et al. Histologic and micro-computed tomographic analyses of replanted teeth stored in different kind of media. J Endod 2014;40:665–669.

Diangelis AJ, Andreasen JO, Ebeleseder KA, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of permanent teeth. Dent Traumatol 2012;28:2–12.

Hammarström L, Blomlöf L, Feiglin B, Andersson L, Lindskog S. Replantation of teeth and antibiotic treatment. Endod Dent Traumatol 1986;2:51–57.

Kling M, Cvek M, Mejare I. Rate and predictability of pulp revascularization in therapeutically reimplanted permanent incisors. Endod Dent Traumatol 1986;2:83–89.

Malmgren B. Ridge preservation/decoronation. Pediatr Dent 2013;35:164–169.

Malmgren B, Andreasen JO, Flores MT, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 3. Injuries in the primary dentition. Dent Traumatol 2012;28:174–182.

Nasjleti CE, Castelli WA, Caffesse RG. The effects of different splinting times on replantation of teeth in monkeys. Oral Surg Oral Med Oral Pathol 1982;53:557–566.

Sae-Lim V, Wang CY, Choi GW, Trope M. The effect of systemic tetracycline on resorption of dried replanted dogs' teeth. Endod Dent Traumatol 1998;14:127–132.

Trope M, Friedman S. Periodontal healing of replanted dog teeth stored in Viaspan, milk and Hank's balanced salt solution. Endod Dent Traumatol 1992;8:183–188.

Trope M, Moshonov J, Nissan R, Buxt P, Yesilsoy C. Short vs. long-term calcium hydroxide treatment of established inflammatory root resorption in replanted dog teeth. Endod Dent Traumatol 1995;11:124–128.

Van Hassel HJ, Oswald RJ, Harrington GW. Replantation 2. The role of the periodontal ligament. J Endod 1980;6:506–508.

Von Arx T, Filippi A, Buser D. Splinting of traumatized teeth with a new device: TTS (Titanium Trauma Splint). Dent Traumatol 2001;17:180–184.

Prognosis

Andreasen FM, Andreasen JO, Bayer T. Prognosis of root-fractured permanent incisors—Prediction of healing modalities. Endod Dent Traumatol 1989;5:11–22.

Andreasen FM, Pedersen BV. Prognosis of luxated permanent teeth--the development of pulp necrosis. Endod Dent Traumatol 1985;1:207–220.

Kling M, Cvek M, Mejare I. Rate and predictability of pulp revascularization in therapeutically reimplanted permanent incisors. Endod Dent Traumatol 1986;2:83–89.

Levin LG. Pulp and periradicular testing. Pediatr Dent 2013;35:113–119.

Ravn JJ. Follow-up study of permanent incisors with enamel fractures as a result of an acute trauma. Scand J Dent Res 1981;89:213–217.

Complications

American Association of Endodontists. Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. http://www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 8 January 2016.

Andreasen FM, Pedersen BV. Prognosis of luxated permanent teeth—The development of pulp necrosis. Endod Dent Traumatol 1985;1:207–220.

Brin I, Ben-Bassat Y, Heling I, Engelberg A. The influence of orthodontic treatment on previously traumatized permanent incisors. Eur J Orthod 1991;13:372–377.

Bryson EC, Levin L, Banchs F, Abbott PV, Trope M. Effect of immediate intracanal placement of Ledermix Paste on healing of replanted dog teeth after extended dry times. Dent Traumatol 2002;18:316–321.

Gabor C, Tam E, Shen Y, Haapasalo M. Prevalence of internal inflammatory root resorption. J Endod 2012;38:24–27.

Hammarström L, Blomlöf L, Feiglin B, Andersson L, Lindskog S. Replantation of teeth and antibiotic treatment. Endod Dent Traumatol 1986;2:51–57.

Hecova H, Tzigkounakis V, Merglova V, Netolicky J. A retrospective study of 889 injured permanent teeth. Dent Traumatol 2010;26:466–475.

Jacobsen I, Kerekes K. Long-term prognosis of traumatized permanent anterior teeth showing calcifying processes in the pulp cavity. Scand J Dent Res 1977;85:588–598.

Kindelan SA, Day PF, Kindelan JD, Spencer JR, Duggal MS. Dental trauma: An overview of its influence on the management of orthodontic treatment. Part 1. J Orthod 2008;35:68–78.

Lundberg M, Cvek M. A light microscopy study of pulps from traumatized permanent incisors with reduced pulpal lumen. Acta Odontol Scand 1980;38:89–94.

Malmgren B. Ridge preservation/decoronation. Pediatr Dent 2013;35:164–169.

Pereira AL, de Mendonca MR, Sonoda CK, Bussato MC, Cuoghi OA, Fabre AF. Microscopic evaluation of induced tooth movement in traumatized teeth: An experimental study in rats. Dent Traumatol 2012;28:114–120.

Robertson A, Andreasen FM, Bergenholtz G, Andreasen JO, Noren JG. Incidence of pulp necrosis subsequent to pulp canal obliteration from trauma of permanent incisors. J Endod 1996;22:557–560.

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

Trope M. Root resorption due to dental trauma. Endod Topics 2002;1:79–100.

Trope M, Moshonov J, Nissan R, Buxt P, Yesilsoy C. Short vs. long-term calcium hydroxide treatment of established inflammatory root resorption in replanted dog teeth. Endod Dent Traumatol 1995;11:124–128.

Van Hassel HJ, Oswald RJ, Harrington GW. Replantation 2. The role of the periodontal ligament. J Endod 1980;6:506–508.

Prevention

American Association of Endodontists. Use of Mouth Guards to Prevent Injury. AAE Position Statement. Chicago: American Association of Endodontics, 2013.

Andersson L. Epidemiology of traumatic dental injuries. J Endod 2013;39:S2–5.

Sigurdsson A. Evidence-based review of prevention of dental injuries. J Endod 2013;39:S88–S93.

10

Resorption

Resorption may present as a primary diagnostic entity or as a secondary complication resulting in failure of endodontically treated teeth. Resorption falls into the following entities: internal root resorption (IRR), invasive cervical root resorption (ICRR), external inflammatory root resorption (EIRR), replacement resorption (RR), and pressure resorption (Fig 10-1). Resorption pathogenesis varies based on type, although inflammation is a component of each (Fig 10-2).

Odontoclasts in the pulp and osteoclasts in the periodontal ligament (PDL) cannot adhere to unmineralized tissue like the predentin and odontoblasts internally and the precementam and cementoblasts externally. For resorption to occur, Tronstad found that loss or damage of the protective, unmineralized layers must occur.

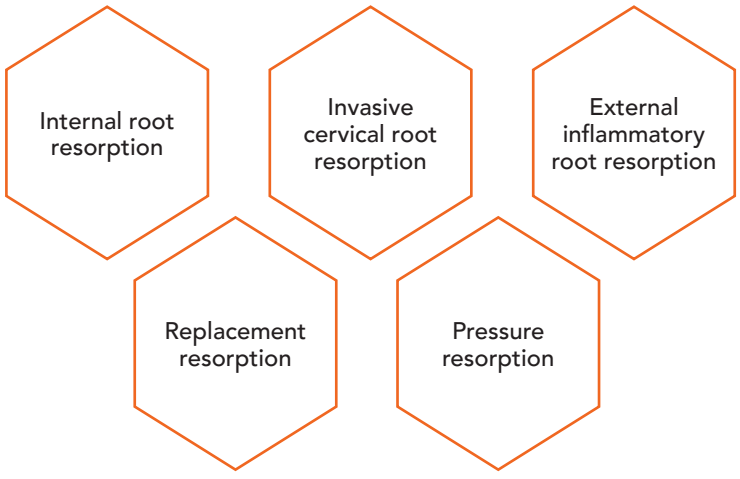

Fig 10-1 Types of resorption.

Fig 10-2 Pathogenesis of resorption (Tronstad).

Internal Root Resorption

Frank first described internal root resorption in 1974. Tronstad later reviewed the pathophysiologic circumstances crucial to progression of IRR. As a result of pulpal injury, some degree of partial coronal pulp necrosis occurs and induces inflammation of the remaining apical vital tissue. This inflamed tissue must abut an area of exposed dentinal tubules, secondary to loss of the protective predentin and odontoblastic layer, for resorption to occur. If the apical tissue necroses, the resorptive process stops.

In an animal study, Wedenberg and Lindskog discovered a further requirement for infection of the necrotic tissue apical to the resorptive lesion. Although transient internal resorption occurs commonly following dental trauma, it will not progress in the absence of infection. Even in cases of pulpal infection, progressive IRR is rare, as vital inflamed tissue is necessary for continued resorption. Gabor et al found that IRR, whether stable or progressive, is a common histologic finding in the absence of clinical or radiographic changes. They noted IRR defects in 50% of extracted teeth with a preoperative diagnosis of pulpitis and 77% of those with pulpal necrosis.

Patel et al reviewed potential etiologic factors for IRR, including trauma, caries, periodontal infections, excessive heat generated during restorative procedures, calcium hydroxide procedures such as pulp caps or pulpotomy, vital root resections, anachoresis, orthodontic treatment, cracked teeth, or idiopathic dystrophic changes.

Gartner et al described the classic radiographic appearance of IRR as an oval-shaped enlargement of uniform density within the pulp space (Fig 10-3). Clinical symptoms generally do not develop until the lesion expands to create a perforation or pulpal involvement progresses to symptoms of pulpitis or pulpal necrosis with infection. Clinically, a pinkish hue may be observed in cases of coronally located IRR, as described by the classic study by Mummery. However, this is an uncommon finding.

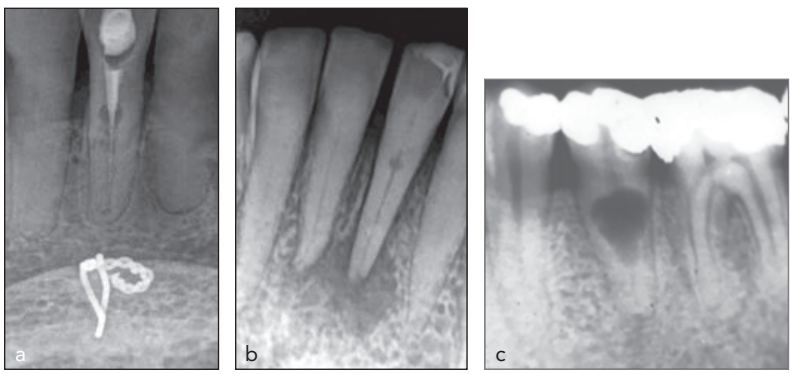

Fig 10-3 *(a to c)* Clinical examples of IRR.

If detected early, treatment of IRR is predictable. Caliskan and Turkun reported a 100% success rate of root canal therapy of nonperforating lesions at 2 to 4 years postoperatively. In cases of perforating lesions, this success rate dropped to 25%. It is important to note, however, that a very small sample size of only four cases was followed, and the study was performed using long-term calcium hydroxide for remineralization. Failures were approached surgically with limited success. With the introduction of mineral trioxide aggregate (MTA) as a repair material, success rates for perforating resorptive defects may improve for both surgical and nonsurgical cases. Although no studies have been published examining MTA's ability to treat perforating IRR, Mente et al demonstrated that MTA can successfully repair iatrogenic perforations.

Invasive Cervical Root Resorption

ICRR has been reviewed extensively by Heithersay (1999a). For this type of resorptive defect to occur, a developmental or iatrogenic defect must be present in the cementum/cementoid layer of the root, so that the PDL, with the potential for inflammatory invasion, is in direct contact with dentin. Clinically, ICRR is generally painless because complete pulpal involvement does not occur except in advanced lesions (Heithersay 1999a). Pulpitis can occur secondary to this late-stage pulpal involvement. Many report that ICRR causes pink coronal discoloration, though Heithersay (1999a) refuted this, as it occurs only in very advanced lesions and may also indicate IRR.

As the majority of ICRR lesions are asymptomatic and not clinically apparent, most cases are diagnosed incidentally on routine radiographs. Early lesions may present as subcrestal radiolucencies resembling class V caries. More extensive lesions present with irregularly shaped radiolucencies at the same location and often extend into radicular dentin. Although lesions will approach the pulp space, the outline of the pulp space usually remains well defined. Adjacent alveolar bone is usually intact. Figure 10-4 depicts some examples of ICRR.

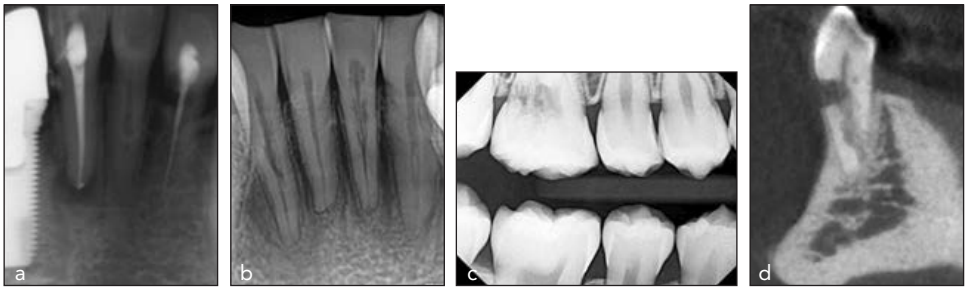

Fig 10-4 *(a to d)* Clinical examples of ICRR.

Histologically, Heithersay (1999a) found ICRR is composed of fibro-osseous and fibrovascular tissue associated with clastic cells. Multiple tunneling channels into dentin were noted, and these increased in density and extension as the lesions progressed apically.

Theories on the potential etiologies of ICRR abound, and the true etiology of ICRR is likely multifactorial. Often, an obvious cause is unknown. Heithersay (1999b) investigated the potential etiologic factors of 250 teeth with ICRR and found that orthodontics was the most commonly identified factor, followed by trauma, intracoronal bleaching, dentoalveolar surgery, periodontal therapy such as deep scaling and root planing, bruxism, developmental defects involving cementum, and intracoronal restorations. No identifiable potential etiologic factor was evident in 16% of teeth. The occurrence of more than one of the potential risk factors resulted in an increased risk of ICRR. The potential risk factors for ICRR are summarized in Fig 10-5.

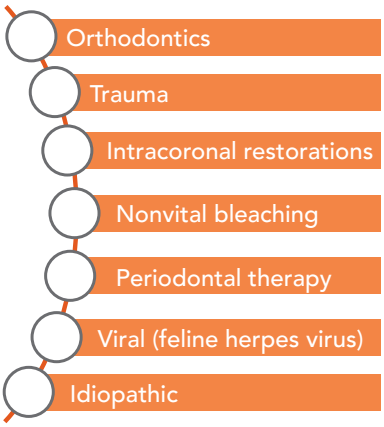

Fig 10-5 Proposed etiologies of ICRR.

Although Heithersay (1999b) reported that orthodontics is the most common etiologic factor for ICRR, other research indicates that orthodontic causation of ICRR is relatively infrequent. Thonen et al found a low incidence of ICRR at 0.9% in molars subject to orthodontic forces, with increased associations in areas of long-duration or larger movement. Consequently, the likelihood of developing ICRR appears low.

Harrington and Natkin first reported the association between ICRR and intracoronal bleaching. Rotstein et al found that superoxyl and heat were particularly associated with ICRR following intracoronal bleaching, and cemental defects found at the cementoenamel junction provided the likely pathway of irritation. Intracoronal bleaching is often performed on previously traumatized teeth, and Heithersay (1999b) suggested that these two factors might act in concert to increase the risk of ICRR.

Periodontal therapy, including deep scaling, root planing, and surgical debridement can theoretically interrupt the protective unmineralized precementum and cementoblastic layer on root structure. Tronstad theorized that sulcular bacteria could extend through the S-shaped dentinal tubules to induce ICRR. This finding is relatively infrequent, as the rapid downgrowth of epithelium and re-establishment of the junctional epithelium attachment should prevent its occurrence.

Among cases of idiopathic ICRR, several etiologic theories have been proposed. Heithersay (1999b) presumed that subclinical factors, such as histologic developmental defects in cervical precementum, might play a role. Von Arx et al proposed a viral etiology in his case series of four patients testing positive for feline herpes virus, known to cause a similar clinical entity in wild and domestic cats.

Heithersay (1999b) classified ICRR lesions based on their size and extension into root structure as follows (Fig 10-6):

- **Class 1:** Small, well-defined lesion localized to cervical area and involving dentin only.
- **Class 2:** Slightly larger, well-defined lesion localized to cervical area but penetrating further into dentin close to the coronal pulp.
- **Class 3:** Larger, less defined lesion extending into the coronal third of the root.
- **Class 4:** Large lesion extending beyond the coronal third of the root.

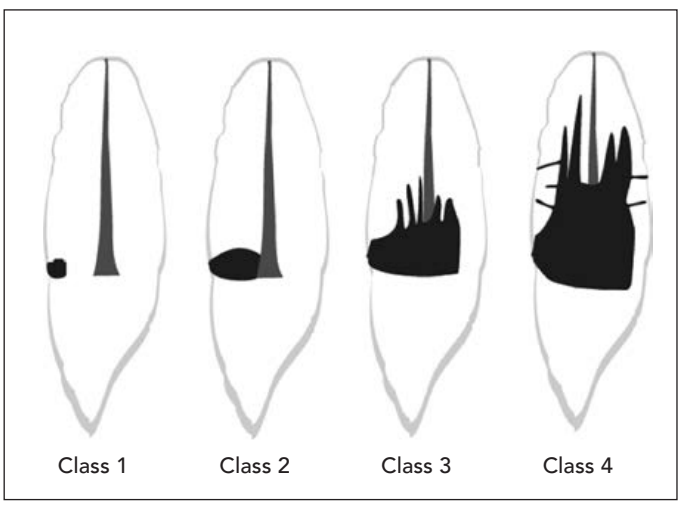

Fig 10-6 Heithersay's (1999b) classifications of ICRR.

Treatment of ICRR involves debridement of the resorptive defect, placement of glycerol on the gingival tissues, application of 90% trichloroacetic acid for 1 to 4 minutes, and restoration with glass-ionomer cements (Heithersay 1999c). To access the defect, a surgical flap is often necessary. Root canal therapy is indicated in class 2 and 3 defects because of inevitable pulpal involvement. Trichloroacetic acid deactivates the resorptive tissue via coagulative necrosis. Glass-ionomer cements are advised for restoration of resorptive defects because Dragoo demonstrated the ability of periodontal tissue to reattach to these materials. While generally an external approach is recommended for treatment, an internal approach for repair can be undertaken for class 2 lesions and involves the use of intracoronal trichloroacetic acid or multiple applications of calcium hydroxide to assure clearance of resorptive soft tissue. Additional potential methods for treating ICRR lesions include the use of guided tissue regeneration, orthodontic extrusion (possibly followed by reintrusion), and intentional replantation.

Heithersay (1999c) recommends treatment only for lesions falling into classes 1, 2, and 3 based on poor outcomes of class 4 lesions. At 3 years postoperative, Heithersay (1999c) reported 100% success for treated class 1 and 2 lesions, 78% success for class 3 lesions, and only 12.5% success for class 4 lesions. Recurrence of resorption or development of periradicular pathology were indicators of failure in their study.

External Inflammatory Root Resorption

EIRR is generally endodontic or orthodontic in origin according to Tronstad. Apical resorption secondary to periradicular pathology is generally transient. Vier and Figueiredo found that 86% of cases with apical periodontitis had associated EIRR due to infectious stimuli. As long as endodontic therapy is initiated in these cases, resorption will arrest.

Orthodontic resorption can occur apically or laterally, dependent on the vector of force. Cwyk et al showed that nearly 30% of orthodontically treated incisors had some degree of EIRR, compared with less than 5% in controls. Massler and Malone reported some degree of root resorption in 86% of orthodontic patients, related to factors including patient age, sex, and systemic conditions. In a recent systematic review, Zahrowski and Jeske found a high correlation between EIRR and intrusive- and rotation-type orthodontic movements. A predisposition to endodontic disease can also increase the risk of EIRR. Brin et al reported an increased prevalence of resorption following orthodontic movement of previously traumatized teeth. Following cessation of orthodontic movement, resorption generally ceases.

Traumatic dental injuries, including luxation and avulsion injuries, are associated with more extensive injuries to the precementum and cementoblastic layer (Tronstad). This, coupled with pulpal necrosis and infection, can result in more extensive EIRR despite endodontic intervention. If vital tissue remains or spontaneous revascularization occurs in the case of avulsion and replantation of immature teeth, Hammarström et al reported transient EIRR. With persistent inflammation or infection, EIRR progresses. Following resolution of the inflammatory component, osseous replacement occurs with commencement of replacement resorption. Consequently, initiation of endodontic therapy is recommended at the first signs of EIRR, and preventive measures are indicated in cases where inflammation and pulpal necrosis and infection are inevitable, such as avulsive injuries. Minimal extraoral dry time and proper storage media are advised, following the recommendations of the American Association of Endodontists trauma guidelines. On replantation, systemic antibiotics are recommended,

and prompt endodontic therapy should commence. In cases of prolonged extraoral dry time, a sodium fluoride soak should be considered. Examples of EIRR can be seen in Fig 10-7. Further information is available in chapter 9.

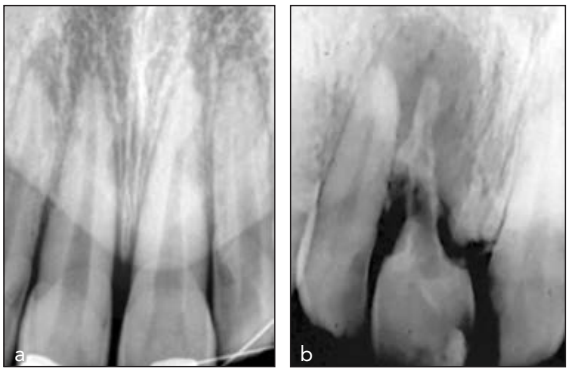

Fig 10-7 Clinical examples of EIRR, including apical *(a)* and lateral *(b)* forms.

Replacement Resorption

Tronstad described RR as a secondary complication of widespread EIRR following extensive PDL loss as a result of traumatic dental injuries with resultant loss of the corresponding protective precementum and cementoblastic layer. Direct contact between cementum and bone prevents the odontoclasts from distinguishing between bone and tooth structures, leading to further degradation. Odontoblasts replace the area of degraded cementum and dentin in a progressive manner. According to Andreasen and Kristerson, if less than 20% of the root surface is involved, reversal of resorption may occur and ankylosis may be avoided. With greater involvement, progressive replacement resorption is considered inevitable, and no known interventions halt its progression.

Clinically, Tronstad described a pathognomonic metallic tone on percussion and progressive infrapositioning of the tooth as the root structure is replaced with bone, particularly in young patients. Figure 10-8 depicts examples of RR. Malmgren recommended decoronation once infrapositioning greater than 1 mm is noted. Decoronation involves crown sectioning with splinting to the neighboring teeth for an esthetic temporary, surgical flap and removal of any prior root canal filling material, and close observation until complete replacement with bone has occurred.

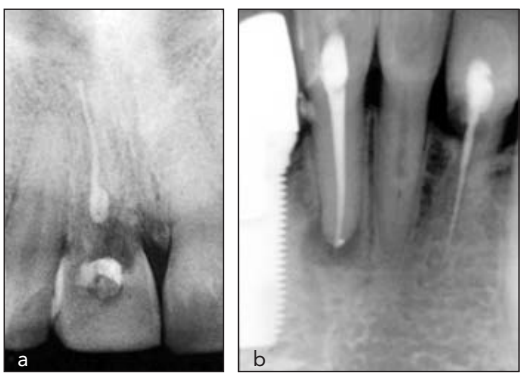

Fig 10-8 *(a and b)* Clinical examples of RR.

Pressure Resorption

Pressure resorption occurs in response to direct damage to the precementum. The dental pulp is not involved, and some outside factor must be present to create this physical damage. According to Tronstad, pressure resorption can occur secondary to misaligned tooth eruption, slow-growing tumors or cysts (such as ameloblastomas, giant cell lesions, or fibro-osseous disease), and orthodontic movement. Pressure resorption does not generally occur secondary to aggressive or malignant tumors due to their rapid nature of expansion. Orthodontically induced pressure resorption is generally limited to the apical third of the roots (Fig 10-9). Although it is not reversible once occurred, progression will cease once orthodontic movement stops (Tronstad).

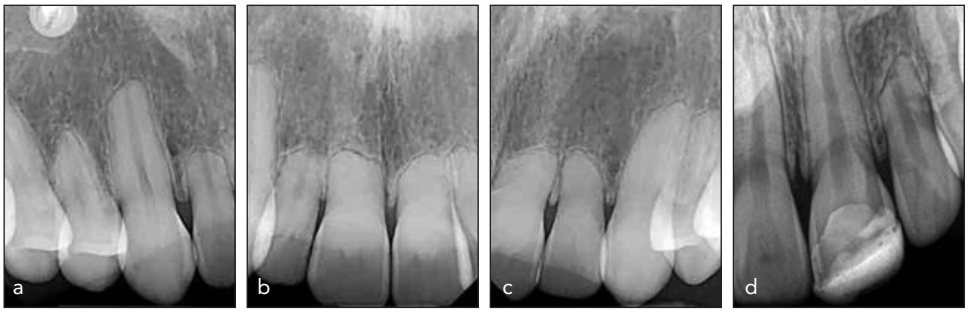

Fig 10-9 *(a to d)* Clinical examples of pressure resorption attributed to orthodontics.

Bibliography

Introduction

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

Internal Root Resorption

Caliskan MK, Turkun M. Prognosis of permanent teeth with internal resorption: A clinical review. Endod Dent Traumatol 1997;13:75–81.

Frank AL. Resorption, perforations, and fractures. Dent Clin North Am 1974;18:465–487.

Gabor C, Tam E, Shen Y, Haapasalo M. Prevalence of internal inflammatory root resorption. J Endod 2012;38:24–27.

Gartner AH, Mack T, Somerlott RG, Walsh LC. Differential diagnosis of internal and external root resorption. J Endod 1976;2:329–334.

Mente J, Leo M, Panagidis D, Saure D, Pfefferle T. Treatment outcome of mineral trioxide aggregate: Repair of root perforations-long-term results. J Endod 2014;40:790–796.

Mummery J. The pathology of "pink-spots" on teeth. Br Dent J 1920;41:301–311.

Patel S, Ricucci D, Durak C, Tay F. Internal root resorption: A review. J Endod 2010;36:1107–1121.

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

Wedenberg C, Lindskog S. Experimental internal resorption in monkey teeth. Endod Dent Traumatol 1985;1:221–227.

Invasive Cervical Root Resorption

Dragoo MR. Resin-ionomer and hybrid-ionomer cements: Part II, human clinical and histologic wound healing responses in specific periodontal lesions. Int J Periodontics Restorative Dent 1997;17:75–87.

Harrington GW, Natkin E. External resorption associated with bleaching of pulpless teeth. J Endod 1979;5:344–348.

Heithersay GS. Clinical, radiologic, and histopathologic features of invasive cervical resorption. Quintessence Int 1999a;30:27–37.

Heithersay GS. Invasive cervical resorption: An analysis of potential predisposing factors. Quintessence Int 1999b;30:83–95.

Heithersay GS. Treatment of invasive cervical resorption: An analysis of results using topical application of trichloracetic acid, curettage, and restoration. Quintessence Int 1999c;30:96–110.

Rotstein I, Friedman S, Mor C, Katznelson J, Sommer M, Bab I. Histological characterization of bleaching-induced external root resorption in dogs. J Endod 1991;17:436–441.

Thonen A, Peltomaki T, Patcas R, Zehnder M. Occurrence of cervical invasive root resorption in first and second molar teeth of orthodontic patients eight years after bracket removal. J Endod 2013;39:27–30.

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

Von Arx T, Schawalder P, Ackermann M, Bosshardt DD. Human and feline invasive cervical resorptions: The missing link? Presentation of four cases. J Endod 2009;35:904–913.

External Inflammatory Root Resorption

American Association of Endodontists. Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. http://www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 8 January 2016.

Brin I, Ben-Bassat Y, Heling I, Engelberg A. The influence of orthodontic treatment on previously traumatized permanent incisors. Eur J Orthod 1991;13:372–377.

Cwyk F, Saint-Pierre F, Tronstad L. Endodontic implications of orthodontic tooth movement. J Dent Res 1984;63:IADR abstract no. 1039.

Hammarström L, Pierce A, Blomlöf L, Feiglin B, Lindskog S. Tooth avulsion and replantation—A review. Endod Dent Traumatol 1986;2:1–8.

Massler M, Malone A. Root resorption in human permanent teeth: A roentgenographic study. Am J Orthod Dentofacial Orthop 1954;40:619–633.

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

Vier FV, Figueiredo JA. Prevalence of different periapical lesions associated with human teeth and their correlation with the presence and extension of apical external root resorption. Int Endod J 2002;35:710–719.

Zahrowski J, Jeske A. Apical root resorption is associated with comprehensive orthodontic treatment but not clearly dependent on prior tooth characteristics or orthodontic techniques. J Am Dent Assoc 2011;142:66–68.

Replacement Resorption

American Association of Endodontists. Recommended Guidelines of the American Association of Endodontists for the Treatment of Traumatic Dental Injuries. http://www.aae.org/clinical-resources/trauma-resources.aspx. Accessed 8 January 2016.

Andreasen JO, Kristerson L. The effect of limited drying or removal of the periodontal ligament. Periodontal healing after replantation of mature permanent incisors in monkeys. Acta Odontol Scand 1981;39:1–13.

Malmgren B. Ridge preservation/decoronation. Pediatr Dent 2013;35:164–169.

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

Pressure Resorption

Tronstad L. Root resorption—Etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–252.

11

Prognosis

Published success rates for endodontic therapies are often cited as justification for treatment decisions both in clinical practice and during the American Board of Endodontics (ABE) examination process. Outcomes are influenced not only by the quality of endodontic treatment but also by a number of periodontal, restorative, microbial, and patient-centered factors. When evaluating published success rates, it is important to consider not only the published success rate but also the criteria used to judge treatment success versus failure. This chapter presents published success rates for endodontic therapies and important prognostic factors that influence treatment outcomes. Table 11-1 includes a summary of prognostic data for all treatment modalities and the publishing author of the study. For complete references, please review the individual sections within this chapter.

Table 11-1 Prognostic values for endodontic treatment modalities

Treatment	Successful outcome
Nonsurgical root canal therapy	97% (Salehrabi and Rotstein 2004) 88% (de Chevigny et al)
Nonsurgical retreatment	89% (Salehrabi and Rotstein 2010) 86% (Fristad et al) 83% (de Chevigny et al)
Surgical root canal therapy	89% (Tsesis et al) 96% (Rubinstein and Kim)
MTA pulp capping	37% (Barthel et al) 80% (Mente et al)
Calcium hydroxide apexification	77% (Jeeruphan et al)
MTA apexification	95% (Jeeruphan et al) 93% (Witherspoon and Ham)
Regenerative endodontics	100% (Jeeruphan et al)

Nonsurgical Root Canal Therapy

Based on available long-term studies, evidence suggests that nonsurgical root canal therapy provides predictable outcomes for patients. In an 8-year epidemiologic study of 1,462,936 teeth, Salehrabi and Rotstein found that 97% of endodontically treated teeth were retained in the oral cavity. Furthermore, 96% of those teeth required no additional intervention. De Chevigny et al found that after 5 to 10 years, 88% of endodontically treated teeth were radiographically healed, and 94% of teeth were clinically functional.

The endodontic literature cites several prognostic factors that may influence reported outcomes. These factors include pretreatment conditions, dental conditions, intratreatment factors, and post-treatment restorative care. Pretreatment conditions capable of influencing root canal therapy outcomes include periodontal attachment loss and apical periodontitis. Setzer et al demonstrated that clinical attachment loss negatively impacts root canal therapy outcomes. Similarly, the presence of apical periodontitis negatively influences treatment outcomes. De Chevigny et al found that in teeth with normal periapices preoperatively, 93% of teeth were considered healed, and 97% were functional. However, teeth with demonstrable preoperative apical periodontitis were radiographically healed only 82% of the time, and 94% were clinically functional.

Though the presence of apical periodontitis clearly influences treatment outcomes to a small degree, the presence of a positive bacteriologic culture immediately prior to the placement of a root canal filling has not been shown to consistently influence treatment outcomes. On the one hand, Sjögren et al found that, while cases with a negative culture prior to root canal filling enjoyed a success rate of 94%, those that had a positive culture prior to root filling had only a 68% success rate. Other authors were unable to confirm this finding. Peters et al found that the presence or absence of a positive culture at the time of root filling had no effect on lesion healing. Similarly, Molander et al failed to demonstrate the effects of positive bacterial cultures at the time of fill on treatment outcomes.

As is the case with positive bacteriologic cultures, the number of treatment visits does not influence treatment outcomes. Penesis et al found similar outcomes for necrotic teeth treated in a single visit versus those treated in two visits. These results were confirmed by Paredes-Vieyra and Enriquez who found no significant difference between single- and multiple-visit therapy for necrotic teeth with apical periodontitis. The reported success rates were 96% for single-visit therapy and 89% for two-visit therapy.

Following completion of root canal therapy, restorative treatment is necessary. The quality of restorative treatment following root canal therapy has long been discussed as a possible prognostic factor. Ray and Trope found that the quality of restorative treatment was more influential on the outcome of endodontic therapy than the quality of the root canal itself. On the contrary, Moreno et al found that the quality of root canal therapy was more influential on periapical status than the quality of restorative care. Gillen et al, however, found that the presence of adequate root canal fillings with inadequate restorations had equal odds of suboptimal clinical outcomes as inadequate root canal fillings with adequate coronal restorations. Taken together, the results of these three studies demonstrate the importance of both quality endodontic treatment and excellent restorative care in the maintenance of diseased teeth.

Though the quality of restorative treatment is an arguable factor in the influence of treatment outcomes, irrefutable evidence exists regarding the necessity of permanent restorative treatment following root canal therapy. Ng et al demonstrated that both the presence of a full-coverage crown and the presence of mesial and distal contacts positively influence success rates. In addition to these findings, several retrospective studies indicate that the majority of teeth removed following root canal therapy are not properly restored. Salehrabi and Rotstein found that, of those teeth extracted following root canal therapy, 85% did not have a definitive coronal restoration. Similarly, Touré et al found that 94% of teeth extracted following root canal therapy were not restored with full-coverage restorations. Recently, in a panoramic radiographic screening study of US Air Force recruits, Winward et al found that, of endodontically treated teeth deemed hopeless, 97% had no cuspal coverage. Clearly, definitive restorative care following root canal treatment is required for positive treatment outcomes.

Patients' immune responses may also play a role in outcomes after nonsurgical root canal therapy. Marending et al found that a significant predictor of the outcome of nonsurgical root canal therapy was the integrity of the nonspecific immune system. Interestingly, this factor was found to be as influential as the technical quality of endodontic treatment. Prognostic factors associated with nonsurgical root canal therapy are listed in Fig 11-1, and examples of post-treatment apical periodontitis are presented in Fig 11-2.

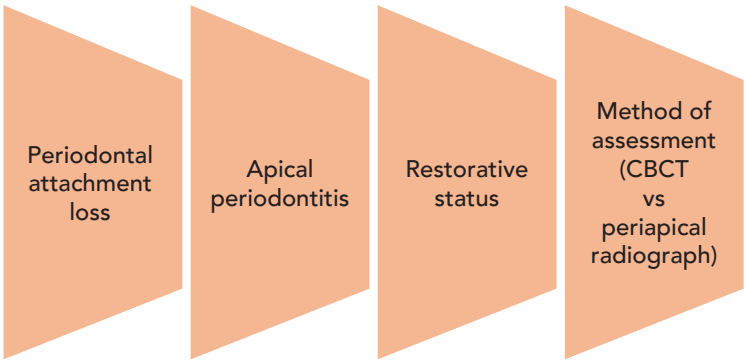

Fig 11-1 Commonly cited factors influencing the reported outcomes of nonsurgical root canal therapy. CBCT, cone beam computed tomography.

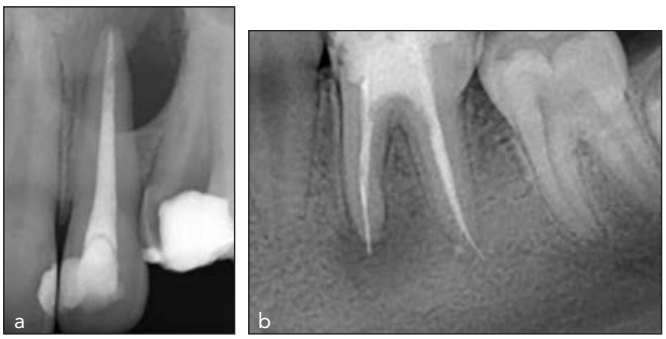

Fig 11-2 Examples of post-treatment apical periodontitis. *(a)* A large apical radiolucency is visible at 2 years following initial root canal therapy on the maxillary left lateral incisor in a 25-year-old man. *(b)* A radiograph taken 1 year after initial root canal therapy with Thermafil filling material [Dentsply] in a 14-year-old boy.

Intraoperative complications, including instrument separation, may not influence outcomes to a great degree. Panitvisai et al found no significant decline in healing rates in teeth with an irretrievable file segment following intraoperative file separation when compared with teeth in which file separations did not occur or in which the separated fragment was successfully removed. Crump and Natkin found that the odds of an unfavorable outcome increased only when the separated instrument prevented disinfection of the root canal system in the presence of a preoperative lesion.

With the advent of new diagnostic technologies including cone beam computed tomography (CBCT) imaging, it has become clear that the method of outcome assessment influences reported success rates. Liang et al found that when teeth with apical periodontitis were assessed by conventional radiography, 87% were considered healed. However, when CBCT scanning was performed on the same group, only 74% of the cases were considered healed. Similarly, Van der Borden et al found that, while the radiographic success rate at 10 to 37 months postoperatively was 88%, when the same teeth were examined by CBCT, the success rate decreased to 77%.

Nonsurgical Retreatment

Nonsurgical retreatment, like initial nonsurgical root canal therapy, provides predictable outcomes for patients. In an epidemiologic study, Salehrabi and Rotstein found that after 5 years, 89% of teeth were retained in the oral cavity. Fristad et al found that 85.7% of cases were successful after 10 to 17 years. De Chevigny et al found that after 5 to 10 years, the radiographic success rate for nonsurgical retreatment was 83%, and 94% of teeth remained clinically functional.

Outcomes for nonsurgical retreatment are influenced by several factors. As with initial nonsurgical root canal therapy, nonsurgical retreatment outcomes are negatively influenced by the presence of preoperative apical periodontitis. De Chevigny et al found that with demonstrable apical periodontitis, the radiographic success rate of nonsurgical retreatment is 80%, and 93% of teeth are clinically functional. With normal periapices preoperatively, the success rate of retreatment is 93%, and 96% of teeth are clinically functional.

In addition to the influence of apical periodontitis, nonsurgical retreatment outcomes are influenced by the quality of the initial treatment, albeit in a somewhat paradoxical manner. Gorni and Gagliani isolated the influence of previous instrumentation on treatment outcomes. They found that if the initial treatment altered the anatomy, success rates were negatively affected. In other words, if initial instrumentation led to the inadvertent alteration of root canal anatomy, nonsurgical retreatment is unlikely to improve treatment outcomes. The opposite effect was noted for initial fill quality. De Chevigny et al found that outcomes were more favorable for teeth that, on initial evaluation, demonstrated a poor-quality root canal filling. Those cases that were filled well initially suffered from poorer retreatment outcomes. Prognostic factors associated with nonsurgical retreatment are summarized in Fig 11-3 and an example of post-retreatment apical periodontitis is presented in Fig 11-4.

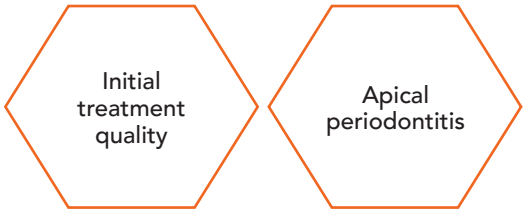

Fig 11-3 Commonly cited factors influencing the reported outcomes of nonsurgical retreatment.

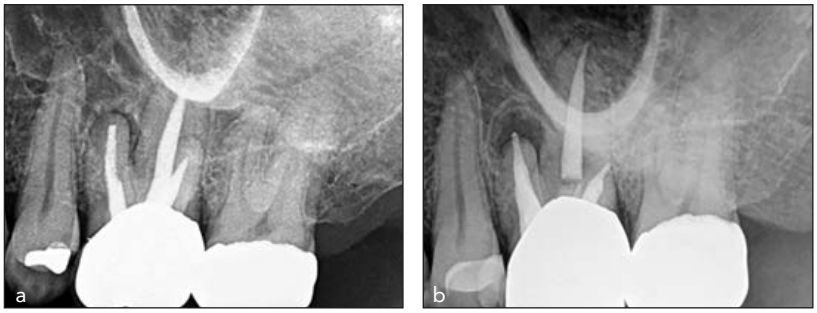

Fig 11-4 Example of failure following nonsurgical root canal retreatment. *(a)* Evidence of persistent periapical pathology after nonsurgical root canal therapy. Retreatment was completed on the tooth, and additional anatomy was located. *(b)* Evidence of continued apical pathology was noted 1 year postoperatively.

Surgical Root Canal Therapy

Like nonsurgical root canal therapy or retreatment, surgical root canal therapy performed using modern treatment techniques offers patients predictable outcomes, according to short- and long-term studies. Tsesis et al found that the success rate of surgical endodontic therapy was 89% 1 year postoperatively. Rubinstein and Kim (1999) found a 1-year success rate of 96.8%. When the cases classified as healed at 1 year were followed for an additional 5 to 7 years, 91% of the teeth remained healed (Rubinstein and Kim 2002).

Outcomes of surgical root canal therapy are influenced by surgical technique. While surgery performed using modern technologies, including the surgical operating microscope and ultrasonic instruments, are often successful, those using traditional surgical approaches offer less predictable solutions for patients. De Lange et al found a higher success rate for surgeries using ultrasonic retropreparations than those using burs in a high-speed drill. Setzer et al (2012) found more favorable outcomes for molars using the surgical operating microscope versus loupes. In a study that quantified the effects of surgical technique, Setzer et al (2010) found a 59% success rate when traditional techniques were employed versus a 94% success rate when modern techniques were implemented.

Just as surgical techniques have improved, so have filling materials. These improvements have led to superior patient outcomes. Historically, amalgam retrofills were used and reported success rates were low. Dorn and Gartner found a success rate of 75% for apical surgery with amalgam retrofillings as compared to a 95% success rate for Super EBA [Bosworth] retrofills. Recently, mineral trioxide aggregate (MTA) has been recommended for use as a retrofilling material due to improved outcomes. MTA retrofills were associated with better 5-year healing rates than composite retrofills in a recent study by Von Arx et al (2014). MTA may not offer improved success over Super EBA, however. Song and Kim found a 95% success rate for MTA retrofills and 93% for Super EBA retrofills with no statistically significant difference noted between filling types.

Preoperative periodontal status, like material and technique selection, influences treatment outcomes. According to Setzer et al (2011), preoperative attachment loss negatively impacts surgical outcomes. More specifically, Von Arx et al (2012) found that mesiodistal bone levels were a significant predictor of surgical outcomes. Song et al (2013) demonstrated that a buccal cortical plate height greater than 3 mm significantly improved clinical outcomes. Periodontal probing depth, in addition to bone height, was shown to influence outcomes, with probing depths of less than 3 mm associated with more favorable outcomes (Lui et al). The communication of endodontic pathology with periodontal attachment loss also significantly impacts outcomes. Kim et al found that isolated endodontic lesions had significantly better outcomes, with a success rate of 95%, compared with endodontic-periodontal lesions, which had success rates of 78%. Prognostic factors associated with surgical root canal therapy are presented in Fig 11-5, an example of postsurgical apical periodontitis in Fig 11-6, and a successful surgical case in Fig 11-7.

The influence of initial surgery versus revision is often discussed in the literature. While historically revisions of apical surgery were thought to have a diminished prognosis, a recent study found that resurgery may achieve excellent outcomes if performed with modern techniques. Studies by Peterson and Gutmann as well as Gagliani et al illustrate the historical perspective. Peterson and Gutmann found that surgical revisions achieved a success

rate of only 36%, while Gagliani et al found a slightly higher 59% success rate. Recently, however, Song et al (2011) found that revision surgery had a 92% success rate if modern techniques were used during the revision procedure.

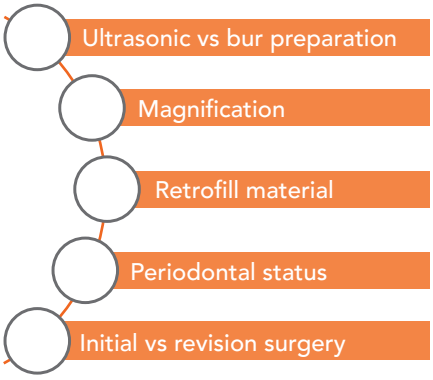

Fig 11-5 Commonly cited factors influencing the reported outcomes of surgical root canal therapy.

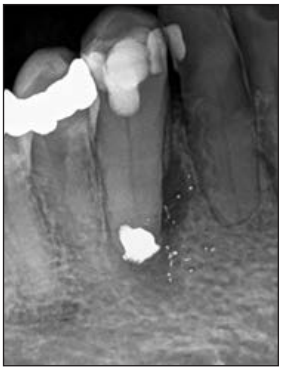

Fig 11-6 Six years postoperatively, a patient was referred for evaluation of recurrent apical pathology following apical surgery with an amalgam retrofill.

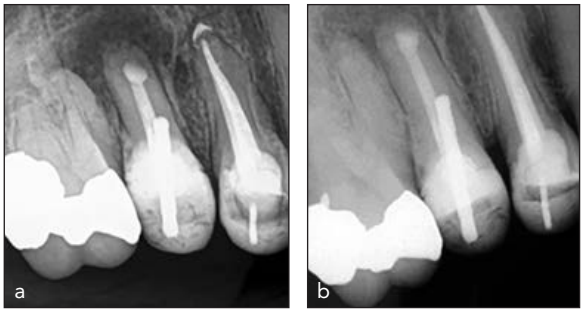

Fig 11-7 Radiographic success after surgical endodontic therapy. *(a)* Immediate postoperative radiograph. *(b)* One-year postoperative follow-up showing reformation of the periodontal ligament space and the absence of apical pathology.

Vital Pulp Therapy

Vital pulp therapies include pulp capping and both full and partial pulpotomies. These therapies maintain the vitality of the radicular pulp tissue, thus permitting further development of radicular dentin in immature permanent teeth. Studies comparing the two treatment modalities exhibit a large range of reported outcomes. Bjørndal et al reported that the success rate for vital pulp therapy was between 32% and 35%. On the other hand, Aguilar and Linsuwanont reported that the success rate of vital pulp therapy, particularly on carious exposures, was between 73% and 99%. Results clearly vary depending on the study.

The reported outcomes for pulp capping, which involves coverage of presumed healthy pulp tissue without its removal, vary in the literature. Barthel et al reported that, following

pulp capping of carious pulp exposures, regardless of the material used, 45% of teeth became necrotic after 5 years and 80% after 10 years. They also reported that the success rate of pulp capping with MTA after 3 years was 37% and after 10 years was 13%. Mente et al, on the other hand, reported that the long-term success rates for direct pulp caps with calcium hydroxide were 58% and with MTA were 80%.

Reported success rates for pulpotomy treatment, which involves amputation of the exposed pulp tissue, are also favorable. Swift et al found that 90% of teeth treated by pulpotomy using eugenol were pain free at 6 months postoperatively. Murray et al found that 78% of teeth treated by pulpotomy maintained the vitality of the radicular pulp tissue at 1 year postoperatively. An MTA pulptomy case is depicted in Fig 11-8.

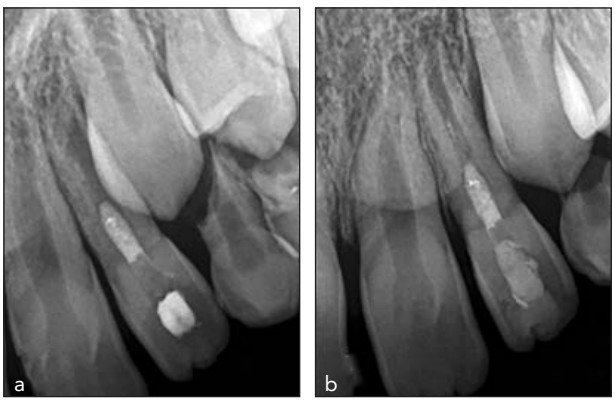

Fig 11-8 Apexogenesis performed on the maxillary left lateral incisor of a 10-year-old girl. *(a)* Immediate postoperative view. *(b)* Six-month radiographic follow-up showing continued root development and the absence of apical pathology.

Treatment of Immature Necrotic Teeth

Treatments for immature necrotic teeth include apexification and regenerative endodontic therapy. Studies on both treatment modalities report favorable success rates. A recent long-term study by Pace et al found that 94% of immature teeth treated with MTA apexification were healed 10 years postoperatively. Jeeruphan et al looked at survival, which measures the mere presence of a tooth in the mouth, as compared to success, which looks at healing (see chapter 1). They found that the radiographic survival of MTA apexification was 95%, whereas for calcium hydroxide apexification, the survival rate was 77%. However, both treatments presented a long-term risk of cervical fracture. Outcomes for teeth being treated by apexification methods are influenced by preoperative apical periodontitis and the number of treatment visits. Mente et al reported that success rates for apexification are lower in the presence of preoperative apical periodontitis. Additionally, Mente et al reported that success rates were less favorable if treatment were performed over several visits. Witherspoon and Ham found that the success rate of MTA apexification treatment was 93.5% if performed in a single visit and 90.5% if performed in two visits. An MTA apexification case is presented in Fig 11-9.

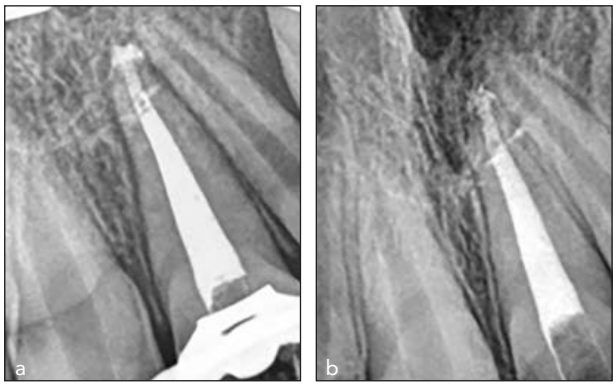

Fig 11-9 MTA apexification performed on a 10-year-old boy. *(a)* Immediate postoperative radiograph with evidence of periodontal ligament (PDL) widening. *(b)* Two-year follow-up radiograph with evidence of normal PDL structures and the absence of apical pathology.

Regenerative endodontics provides an alternative to apexification in the treatment of immature necrotic teeth. This treatment encourages further development of radicular dentin, thus decreasing the risk of cervical fracture. The majority of publications regarding regenerative endodontic therapy are lower-level-of-evidence studies, including case series and case reports, with the exception of two cohort studies (Kontakiotis et al). In one of the cohort studies, Jeeruphan et al reported a 100% survival rate for teeth treated with regenerative endodontic therapy. In a recent study comparing outcomes of apexification and regenerative endodontic therapy, Alobaid et al found the two treatments to provide statistically equivalent results. A successful regenerative endodontics case is presented in Fig 11-10.

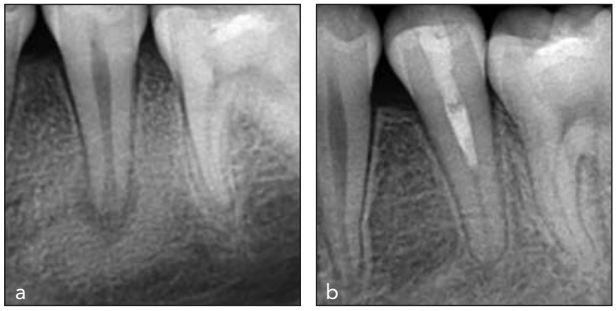

Fig 11-10 Successful regenerative endodontic therapy. *(a)* Preoperative view. *(b)* Follow-up radiograph taken 1 year postoperatively. An increase in root length and width as well as apical closure is evident in addition to the healing of the apical radiolucency.

Nonvital Bleaching

Little data on nonvital bleaching outcomes can be found in the literature. In a subjective study on patient and practitioner satisfaction 5 years postbleaching, Glockner et al found that 98% of patients were satisfied with the results. Dentists, however, were slightly less pleased with the long-term results, as only 80% of practitioners reported satisfaction with long-term outcomes.

Treatment of Endodontic Disease in the Primary Dentition

The treatment for diseased primary teeth can involve both pulpotomy and pulpectomy treatment, depending on the extent of pulpal involvement. In vital primary teeth with carious pulp exposures, pulpotomies are the treatment of choice. Fuks et al reported high success rates for pulpotomies with diformocresol and ferric sulfate at 83% and 93%, respectively. Success rates may be higher for teeth treated with MTA. Holan et al found that pulpotomies performed with MTA achieved a success rate of 97% at 2 years. Necrotic primary teeth may be treated by pulpectomy and maintained to prevent space loss in the primary dentition. Coll and Sadrian reported a 78% success rate for pulpectomies in primary teeth. This treatment, however, is not without risk. Enamel defects, over-retention, and alteration of the path of eruption of the permanent successor have been noted (Coll and Sadrian).

Bibliography

Introduction

Barthel CR, Rosenkranz B, Leuenberg A, Roulet JF. Pulp capping of carious exposures: Treatment outcome after 5 and 10 years: A retrospective study. J Endod 2001;26:525–528.

de Chevigny C, Dao TT, Basrani BR, et al. Treatment outcome in endodontics: The Toronto study—Phase 4: Initial treatment. J Endod 2008;34:258–263.

Fristad I, Molven O, Halse A. Nonsurgically retreated root filled teeth—Radiographic findings after 20-27 years. Int Endod J 2004;37:12–18.

Jeeruphan T, Jantarat J, Yanpiset K, Suwannapan L, Khewsawai P, Hargreaves KM. Mahidol study 1: Comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: A retrospective study. J Endod 2012;38:1330–1336.

Mente J, Hufnagel S, Leo M, et al. Treatment outcome of mineral trioxide aggregate or calcium hydroxide direct pulp capping: Long-term results. J Endod 2014;40:1746–1751.

Rubinstein RA, Kim S. Short-term observation of the results of endodontic surgery with the use of a surgical operation microscope and super-EBA as root-end filling material. J Endod 1999;25:43–48.

Salehrabi R, Rotstein I. Endodontic treatment outcomes in a large patient population in the USA: An epidemiological study. J Endod 2004;30:846–850.

Salehrabi R, Rotstein I. Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment. J Endod 2010;36:790–792.

Tsesis I, Rosen E, Taschieri S, Telishevsky Strauss Y, Ceresoli V, Del Fabbro M. Outcomes of surgical endodontic treatment performed by a modern technique: An updated meta-analysis of the literature. J Endod 2013;39:332–339.

Witherspoon DE, Ham K. One-visit apexification: Technique for inducing root-end barrier formation in apical closures. Pract Proced Aesthet Dent 2001;13:455–460.

Nonsurgical Root Canal Therapy

Crump MC, Natkin E. Relationship of broken root canal instruments to endodontic case prognosis: A clinical investigation. J Am Dent Assoc 1970;80:1341–1347.

de Chevigny C, Dao TT, Basrani BR, et al. Treatment outcome in endodontics: The Toronto study—Phase 4: Initial treatment. J Endod 2008;34:258–263.

Gillen BM, Looney SW, Gu LS, et al. Impact of the quality of coronal restoration versus the quality of root canal fillings on success of root canal treatment: A systematic review and meta-analysis. J Endod 2011;37:895–902.

Liang Y-H, Li G, Wesselink PR, Wu M-K. Endodontic outcome predictors identified with periapical radiographs and cone-beam computed tomography scans. J Endod 2011;37:326–331.

Marending M, Peters OA, Zehnder M. Factors affecting the outcome of orthograde root canal therapy in a general dentistry hospital practice. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:119–124.

Molander A, Warfvinge J, Reit C, Kvist T. Clinical and radiographic evaluation of one- and two-visit endodontic treatment of asymptomatic necrotic teeth with apical periodontitis: A randomized clinical trial. J Endod 2007;33:1145–1148.

Moreno JO, Alves FRF, Gonçalves LS, Martinez AM, Rôças IN, Siqueira JF Jr. Periradicular status and quality of root canal fillings and coronal restorations in an urban colombian population. J Endod 2013;39:600–604.

Ng YL, Mann V, Rahbaran S, Lewsey J, Gulabivala K. Outcome of primary root canal treatment: Systematic review of the literature—Part 1. Effects of study characteristics on probability of success. Int Endod J 2007;40:921–939.

Panitvisai P, Parunnit P, Sathorn C, Messer HH. Impact of a retained instrument on treatment outcome: A systematic review and meta-analysis. J Endod 2010;36:775–780.

Paredes-Vieyra J, Enriquez FJJ. Success rate of single- versus two-visit root canal treatment of teeth with apical periodontitis: A randomized controlled trial. J Endod 2012;38:1164–1169.

Penesis VA, Fitzgerald PI, Fayad MI, Wenckus CS, BeGole EA, Johnson BR. Outcome of one-visit and two-visit endodontic treatment of necrotic teeth with apical periodontitis: A randomized controlled trial with one-year evaluation. J Endod 2008;34:251–257.

Peters LB, Wesselink PR, Moorer WR. The fate and the role of bacteria left in root dentinal tubules. Int Endod J 1995;28:95–99.

Ray HA, Trope M. Periapical status of endodontically treated teeth in relation to the technical quality of the root filling and the coronal restoration. Int Endod J 1995;28:12–18.

Salehrabi R, Rotstein I. Endodontic treatment outcomes in a large patient population in the USA: An epidemiological study. J Endod 2004;30:846–850.

Setzer FC, Boyer KR, Jeppson JR, Karabucak B, Kim S. Long-term prognosis of endodontically treated teeth: A retrospective analysis of preoperative factors in molars. J Endod 2011;37:21–25.

Sjögren U, Figdor D, Persson S, Sundqvist G. Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. Int Endod J 1997;30:297–306.

Touré B, Faye B, Kane AW, Lo CM, Niang B, Boucher Y. Analysis of reasons for extraction of endodontically treated teeth: A prospective study. J Endod 2011;37:1512–1515.

Van der Borden WG, Wang X, Wu M-K, Shemesh H. Area and 3-dimensional volumetric changes of periapical lesions after root canal treatments. J Endod 2013;39:1245–1249.

Winward BJ, Yaccino JM, Kirkpatrick TC. A panoramic survey of air force basic trainees: How research translates into clinical practice. J Endod 2014;40:1332–1337.

Nonsurgical Retreatment

de Chevigny C, Dao TT, Basrani BR, et al. Treatment outcome in endodontics: The Toronto study—Phases 3 and 4: Orthograde retreatment. J Endod 2008;34:131–137.

Fristad I, Molven O, Halse A. Nonsurgically retreated root filled teeth—Radiographic findings after 20-27 years. Int Endod J 2004;37:12–18.

Gorni FG, Gagliani MM. The outcome of endodontic retreatment: A 2-yr follow-up. J Endod 2004;30:1–4.

Salehrabi R, Rotstein I. Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment. J Endod 2010;36:790–792.

Surgical Root Canal Therapy

de Lange J, Putters T, Baas E, van Ingen JM. Ultrasonic root-end preparation in apical surgery: A prospective randomized study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:841–845.

Dorn SO, Gartner AH. Retrograde filling materials: A retrospective success-failure study of amalgam, EBA, and IRM. J Endod 1990;16:391–393.

Gagliani MM, Gorni F, Strohmenger L. Periapical resurgery versus periapical surgery: A 5-year longitudinal comparison. Int Endod J 2005;35:320–327.

Kim E, Song J-S, Jung I-Y, Lee S-J, Kim S. Prospective clinical study evaluating endodontic microsurgery outcomes for cases with lesions of endodontic origin compared with cases with lesions of combined periodontal–endodontic origin. J Endod 2008;34:546–551.

Lui JN, Khin MM, Krishnaswamy G, Chen NN. Prognostic factors relating to the outcome of endodontic microsurgery. J Endod 2014;40:1071–1076.

Peterson J, Gutmann JL. The outcome of endodontic resurgery: A systematic review. Int Endod J 2001;34:169–175.

Rubinstein RA, Kim S. Long-term follow-up of cases considered healed one year after apical microsurgery. J Endod 2002;28:378–383.

Rubinstein RA, Kim S. Short-term observation of the results of endodontic surgery with the use of a surgical operation microscope and super-EBA as root-end filling material. J Endod 1999;25:43–48.

Setzer FC, Boyer KR, Jeppson JR, Karabucak B, Kim S. Long-term prognosis of endodontically treated teeth: A retrospective analysis of preoperative factors in molars. J Endod 2011;37:21–25.

Setzer FC, Kohli MR, Shah SB, Karabucak B, Kim S. Outcome of endodontic surgery: A meta-analysis of the literature—Part 2: Comparison of endodontic microsurgical techniques with and without the use of higher magnification. J Endod 2012;38:1–10.

Setzer FC, Shah SB, Kohli MR, Karabucak B, Kim S. Outcome of endodontic surgery: A meta-analysis of the literature—Part 1: Comparison of traditional root-end surgery and endodontic microsurgery. J Endod 2010;36:1757–1765.

Song M, Kim E. A prospective randomized controlled study of mineral trioxide aggregate and super ethoxy–benzoic acid as root-end filling materials in endodontic microsurgery. J Endod 2012;38:875–879.

Song M, Kim SG, Shin S-J, Kim H-C, Kim E. The influence of bone tissue deficiency on the outcome of endodontic microsurgery: A prospective study. J Endod 2013;39:1341–1345.

Song M, Shin S-J, Kim E. Outcomes of endodontic micro-resurgery: A prospective clinical study. J Endod 2011;37:316–320.

Tsesis I, Rosen E, Taschieri S, Telishevsky Strauss Y, Ceresoli V, Del Fabbro M. Outcomes of surgical endodontic treatment performed by a modern technique: An updated meta-analysis of the literature. J Endod 2013;39:332–339.

Von Arx T, Hanni S, Jensen SS. 5-year results comparing mineral trioxide aggregate and adhesive resin composite for root-end sealing in apical surgery. J Endod 2014;40:1077–1081.

Von Arx T, Jensen SS, Hänni S, Friedman S. Five-year longitudinal assessment of the prognosis of apical microsurgery. J Endod 2012;38:570–579.

Vital Pulp Therapy

Aguilar P, Linsuwanont P. Vital pulp therapy in vital permanent teeth with cariously exposed pulp: A systematic review. J Endod 2011;37:581–587.

Barthel CR, Rosenkranz B, Leuenberg A, Roulet JF. Pulp capping of carious exposures: Treatment outcome after 5 and 10 years: A retrospective study. J Endod 2001;26:525–528.

Bjørndal L, Reit C, Bruun G, et al. Treatment of deep caries lesions in adults: Randomized clinical trials comparing stepwise vs. direct complete excavation, and direct pulp capping vs. partial pulpotomy. Eur J Oral Sci 2010;118:290–297.

Mente J, Hufnagel S, Leo M, et al. Treatment outcome of mineral trioxide aggregate or calcium hydroxide direct pulp capping: Long-term results. J Endod 2014;40:1746–1751.

Murray PE, About I, Franquin J, Remusat M, Smith AJ. Restorative pulpal and repair responses. J Am Dent Assoc 2001;132:482–491.

Swift EJ, Trope M, Ritter AV. Vital pulp therapy for the mature tooth—Can it work? Endod Topics 2003;5:49–56.

Treatment of Immature Necrotic Teeth

Alobaid AS, Cortes LM, Lo J, et al. Radiographic and clinical outcomes of the treatment of immature permanent teeth by revascularization or apexification: A pilot retrospective cohort study. J Endod 2014;40:1063–1070.

Jeeruphan T, Jantarat J, Yanpiset K, Suwannapan L, Khewsawai P, Hargreaves KM. Mahidol study 1: Comparison of radiographic and survival outcomes of immature teeth treated with either regenerative endodontic or apexification methods: A retrospective study. J Endod 2012;38:1330–1336.

Kontakiotis EG, Filippatos CG, Agrafioti A. Levels of evidence for the outcome of regenerative endodontic therapy. J Endod 2014;40:1045–1053.

Mente J, Leo M, Panagidis D, et al. Treatment outcome of mineral trioxide aggregate in open apex teeth. J Endod 2013;39:20–26.

Pace R, Giuliani V, Nieri M, Di Nasso L, Pagavino G. Mineral trioxide aggregate as apical plug in teeth with necrotic pulp and immature apices: A 10-year case series. J Endod 2014;40: 1250–1254.

Witherspoon DE, Ham K. One-visit apexification: Technique for inducing root-end barrier formation in apical closures. Pract Proced Aesthet Dent 2001;13:455–460.

Nonvital Bleaching

Glockner K, Hulla H, Ebeleseder K, Stadtler P. Five-year follow-up of internal bleaching. Braz Dent J 1999;10:105–110.

Treatment of Endodontic Disease in the Primary Dentition

Coll JA, Sadrian R. Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition. Pediatr Dent 1996;18:57–63.

Fuks AB, Holan G, Davis J, Eidelman E. Ferric sulfate versus dilute formocresol in pulpotomized primary molars: Long-term follow up. Pediatr Dent 1997;19:327–330.

Holan G, Eidelman E, Fuks AB. Long-term evaluation of pulpotomy in primary molars using mineral trioxide aggregate or formocresol. Pediatr Dent 2005;27:129–136.

12

Complications

The identification and management of complications are integral skills for any practicing endodontist. Furthermore, the importance of treatment complications in the American Board of Endodontics (ABE) examination process cannot be overemphasized as discussion of complications comprises one-tenth of the oral examination. Complications can range in severity from nuisances requiring a few extra minutes for management to life-threatening emergencies requiring immediate medical care. In addition, complications may not affect treatment prognosis or may result in the need for extraction of an otherwise viable tooth. This chapter discusses both intraoperative and postoperative complications.

Intratreatment Complications

Instrument separation

Instrument separation is a commonly reported complication in endodontic therapy that all clinicians will likely encounter at some point in their careers. Though the majority of the published literature focuses on file separations, other instruments, including burs, ultrasonic tips, and passively used intracanal instruments like spreaders and lentulo spirals can separate. The introduction of nickel titanium (NiTi) instruments has increased the incidence of instrument separation. In a retrospective clinical study of nearly 5,000 cases treated by residents over 4 years, Iqbal et al reported a 0.25% incidence of stainless steel hand instrument separation compared with a 1.68% incidence of NiTi rotary instrument separation (Fig 12-1). In other words, rotary instruments were seven times more likely to separate than hand instruments. Separation was most commonly reported in the apical one-third of canal spaces. Torque control motors and clinician experience did not affect the likelihood of instrument separation in Iqbal et al's study. Although Iqbal et al found that certain instruments in the ProTaper system [Dentsply] were more likely to fracture than other systems, Ankrum et al reported no difference in the likelihood of instrument separation among instrument systems studied, including Protaper, K3 [Kerr], and ProFile [Dentsply].

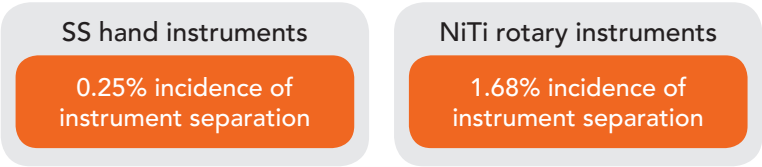

Fig 12-1 Instrument type as a risk factor for instrument separation (Iqbal et al). SS, stainless steel.

Both Crump and Natkin's study on stainless steel instruments and Spili et al's study on NiTi instruments found that separated instruments had no statistically significant effect on the prognosis. However, both authors argued that the stage of treatment when the instrument separation occurs, the preoperative diagnosis, and the ability to remove or bypass the instrument fragment may influence outcomes based on clinical empiricism. Essentially, they suggested that greater bacterial contamination caused by the inability to disinfect the canal space as a result of the retained instrument fragment might result in a less favorable prognosis. A retrospective clinical study by Fu et al found that the adequacy of the root canal filling surrounding an instrument fragment did affect prognosis following instrument separation. Fu et al also noted a trend toward better healing in the absence of perforations created during attempts at instrument removal. Figure 12-2 depicts the potential factors that might affect prognosis following instrument separation.

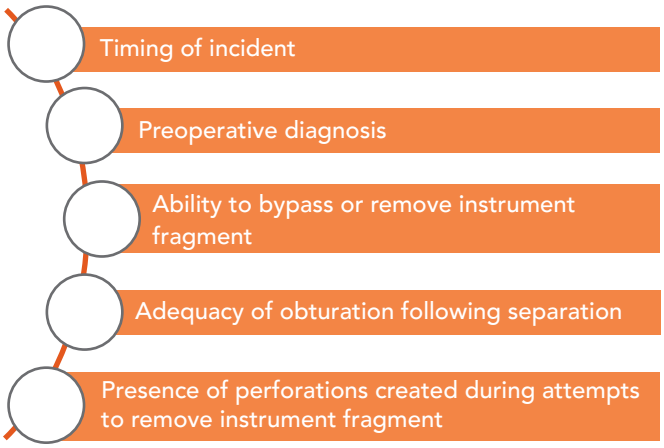

Fig 12-2 Potential factors affecting prognosis following instrument separation.

Madarati et al reviewed techniques for management of separated instrument fragments (Fig 12-3) with the primary goal being instrument removal via a variety of techniques, most notably ultrasonic instrumentation. If instrument fragments cannot be removed using traditional orthograde techniques, options remain to bypass the instrument, leave it in place, or approach its removal or entombment surgically. Figure 12-4 depicts examples of different management techniques.

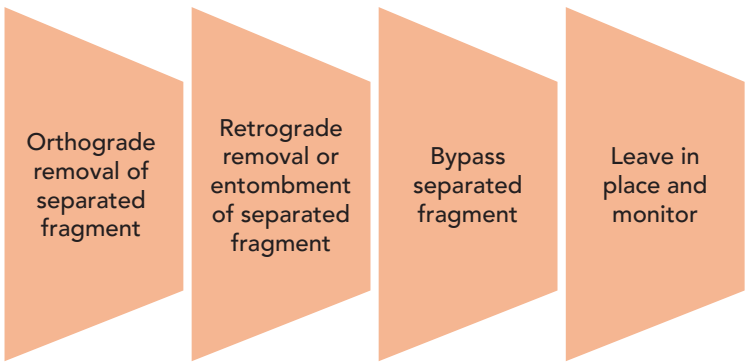

Fig 12-3 Options for management of separated instruments (Madarati et al).

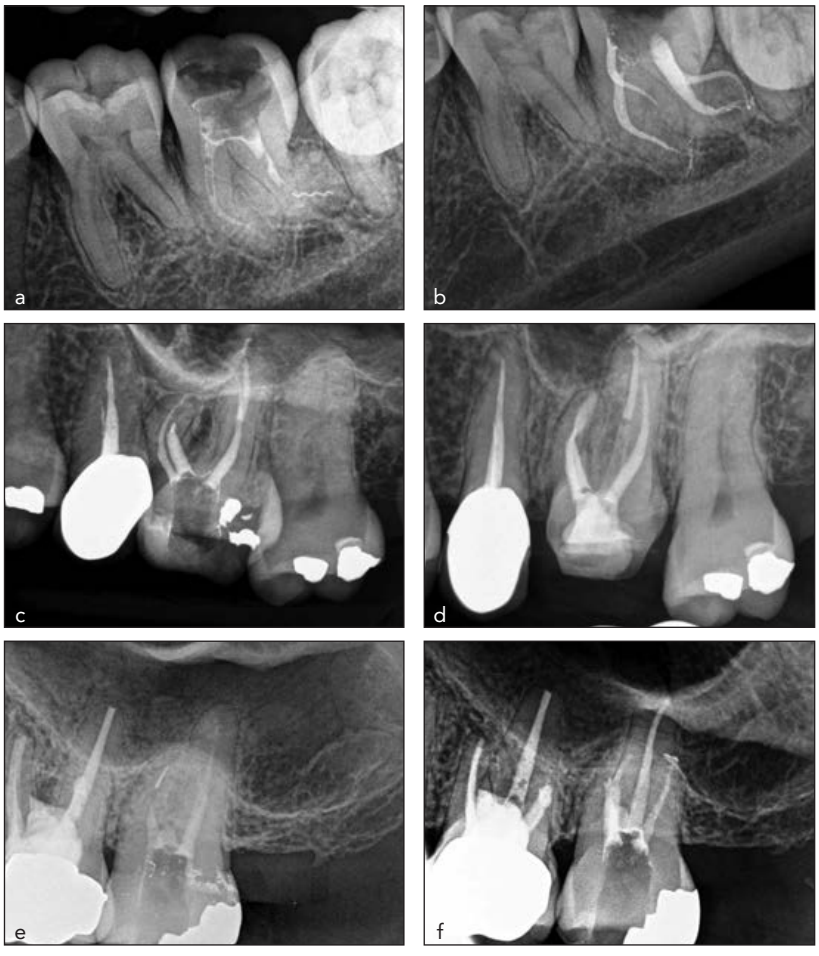

Fig 12-4 Examples of management of separated instruments. *(a and b)* A lentulo spiral was successfully bypassed. *(c and d)* A NiTi instrument was addressed surgically. *(e and f)* A NiTi instrument was left in place.

Visualization with the surgical operating microscope offers an obvious advantage when managing an instrument separation. In a clinical study, Suter et al found that 87% of separated instruments could be removed, and all cases of removal involved the use of a surgical operating microscope. In the future, they predict that laser irradiation or electrochemical dissolution techniques may be developed to remove separated instruments. Ultimately, the primary goal should be to use caution during instrumentation to prevent separation complications altogether.

Perforations

Iatrogenic perforations are a common complication during endodontic therapy, and recent advances in endodontics have markedly improved clinicians' management abilities. Endodontists must acknowledge that patients may arrive in their office with teeth that have been previously perforated and should be prepared to diagnose and manage perforations that occurred prior to their intervention. Signs of prior perforations, including bone loss adjacent to the perforation site, can be recognized preoperatively, particularly with the use of cone beam computed tomography (CBCT) imaging (Shemesh et al). For this reason, new radiographs are always advised during treatment consultations, particularly in a previously accessed tooth. Noniatrogenic perforations due to caries or resorption are further complicated by their respective causative pathology and contamination, and the following recommendations may not necessarily apply in those instances.

Iatrogenic perforations can be recognized clinically by active bleeding into the canal space, or radiographically, by close proximity of the preparation to the radicular border or extraradicular instrument placement (Fuss and Trope). Fuss et al suggested using an electronic apex locator in the early intratreatment identification of perforations.

Early recognition and repair are keys to successful outcomes when treating perforations. Fuss and Trope reported that size, location, and time of repair significantly impact the prognosis, as small intrabony perforations that are sealed immediately have a generally positive prognosis. Mente et al reported that provider experience and post placement following perforation repair may also play a role. Seltzer et al (1970) cited both the clinician's ability to seal the defect and any prior microbial contamination as potential prognostic factors. These authors described failures following unrepaired perforation due to epithelial downgrowth and periodontal involvement followed by adjacent bone loss. Figure 12-5 summarizes the potential factors that affect prognosis following a perforation repair. Figure 12-6 illustrates the pathogenesis of failure for unrepaired perforations. Figure 12-7 presents an example of failure secondary to perforation by off-angle placement of a post.

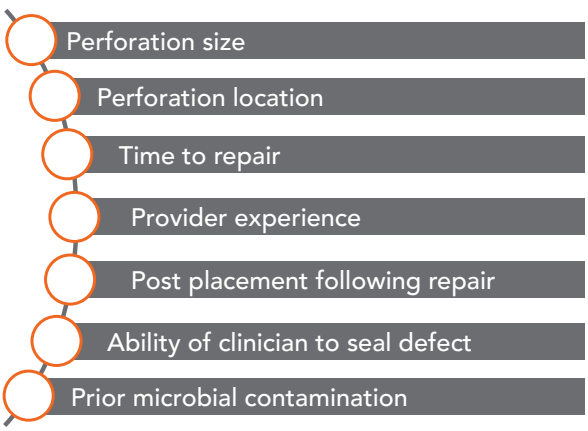

Fig 12-5 Potential prognostic factors following perforation and repair.

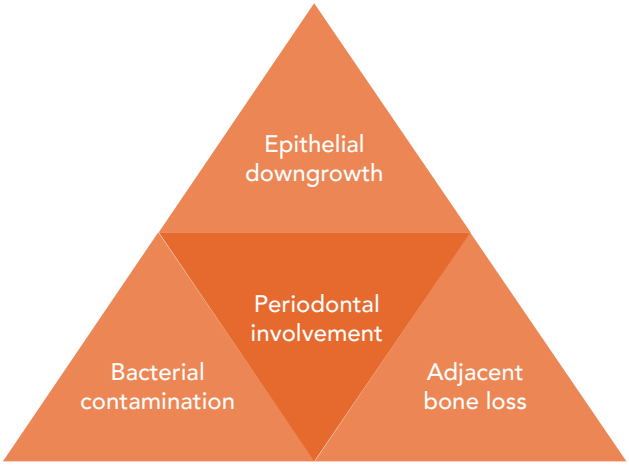

Fig 12-6 Pathogenesis of failure of unrepaired perforations (Seltzer et al 1970).

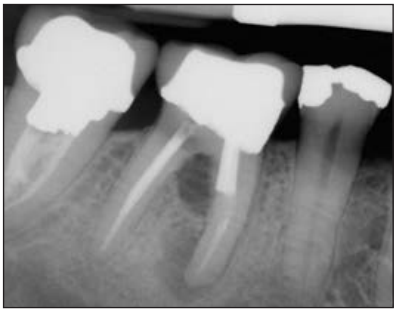

Fig 12-7 Periodontal involvement secondary to a post perforation in the mesial root of a mandibular molar.

The development and implementation of mineral trioxide aggregate (MTA) as a repair material has significantly impacted practitioners' abilities to treat iatrogenic perforations. Early on, Lee et al (1993) recommended MTA for lateral root repairs, and Pitt Ford et al found it effective for furcal perforation repairs. Since then, MTA has become the gold standard material in perforation repair due to its positive impact on histologic repair and treatment success rates. Pitt Ford et al described cementum formation beneath MTA followed by reformation of the periodontal ligament and normal bony architecture. These results have been replicated by several studies in various applications of MTA use. According to Mente et al, the success rate of MTA perforation repair with a minimum of a 1-year follow-up was approximately 86%. Such positive outcomes are likely related to MTA's biocompatibility and superior ability to seal defects. An example of a perforation successfully repaired with MTA is shown in Fig 12-8. Further information about MTA can be found in chapter 8.

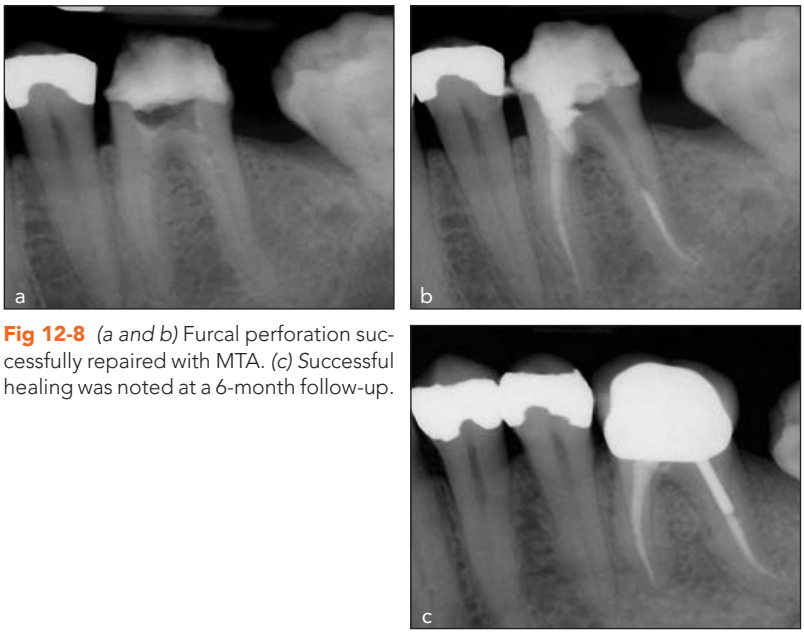

Fig 12-8 *(a and b)* Furcal perforation successfully repaired with MTA. *(c)* Successful healing was noted at a 6-month follow-up.

Despite both excellent biocompatibility and positive outcomes, MTA has the limitation of a long setting time. Consequently, in areas of heavy saliva exposure, such as adjacent to the gingival sulcus, alternative materials have been suggested to prevent washout. Dragoo recommended glass ionomers as an alternative to MTA for sulcular perforations. Figure 12-9 illustrates the recommended materials for management of perforations.

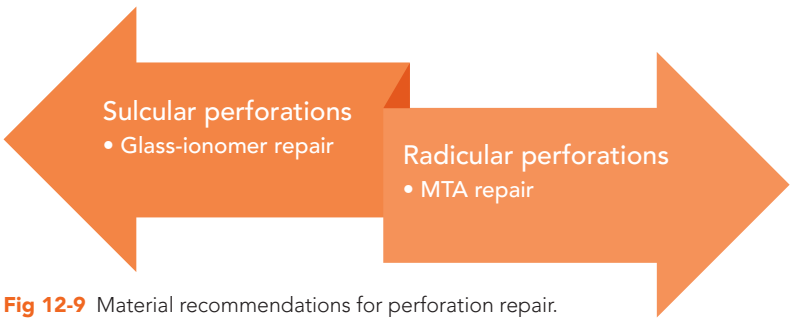

Fig 12-9 Material recommendations for perforation repair.

Sodium hypochlorite accident

Sodium hypochlorite is an effective antimicrobial and efficiently dissolves both vital and necrotic tissue. As a result of its ability to dissolve tissue combined with its causticity, it poses a risk to patients when expressed into the periapical tissues. A sodium hypochlorite accident can occur when the irrigant solution is injected beyond the apical foramen, resulting in tissue necrosis. Sodium hypochlorite accidents are often associated with open apices or extreme pressure during irrigation. Hulsmann and Hahn reviewed common symptoms associated with sodium hypochlorite accidents, including immediate severe pain, immediate edema of the neighboring soft tissue with possible extension through the fascial planes, profuse intracanal bleeding, ecchymosis, and possible taste of bleach and irritation if the solution was injected into the maxillary sinus. Delayed findings can include secondary infection and either anesthesia or paresthesia. Vital signs can be compromised by the rapid swelling associated with a sodium hypochlorite accident, and immediate, emergent medical attention should be sought if there are any signs of vital sign compromise. Figure 12-10 summarizes the clinical signs associated with a sodium hypochlorite accident.

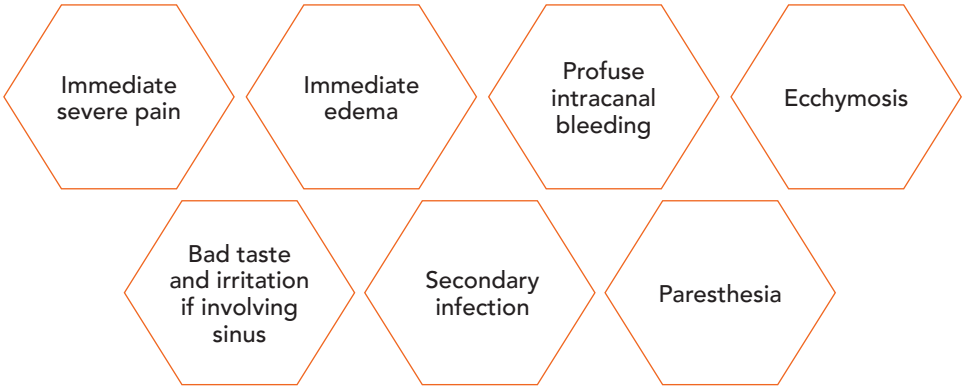

Fig 12-10 Potential clinical signs of a sodium hypochlorite accident (Hulsmann and Hahn).

Several case reports have been presented in the endodontic literature illustrating the potential outcomes of a sodium hypochlorite accident. Sabala and Powell presented a case involving severe immediate pain, dramatic swelling, bleeding, and ecchymosis due to interstitial bleeding. Reeh and Messer described a case with a similar immediate course, in addition to postoperative infection and paresthesia lasting 15 months following the initial sodium hypochlorite accident. Matthews and Merrill presented a case of chronic neuropathic pain and long-lasting paresthesia 7 months following a sodium hypochlorite accident.

Several authors have described appropriate treatment regimens to address a sodium hypochlorite accident (Fig 12-11). Hulsmann and Hahn recommended palliative care, including pain control via local anesthesia and analgesics as well as initial use of cold compresses for 24 hours followed by warm compresses afterward to stimulate local circulation. They recommended daily monitoring for improvement. Antibiotics are only recommended with evidence of secondary infection or prophylactically in high-risk cases. The use of corticosteroids is controversial. Any further endodontic treatment should use alternative irrigants, such as saline or chlorhexidine.

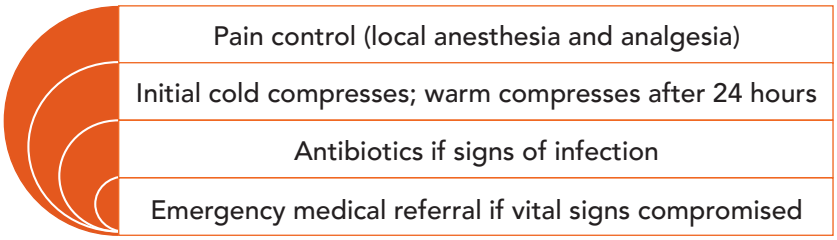

Fig 12-11 Suggested management protocol for a sodium hypochlorite accident (Hulsmann and Hahn).

Extrusion of materials beyond the apex

As the root canal space is continuous with the periapical tissues, the risk of overextension of medicament or obturation material is inherent to endodontic therapy and increases in the absence of adequate length control or in cases of open or wide apices. Although overextension of material can theoretically cause a foreign body reaction no matter its location, extension into important anatomical spaces including the maxillary sinus, mental foramen, and mandibular canal are generally considered most risky.

Calcium hydroxide (CH) is a common intracanal medicament, and though it is generally considered safe, there are several reported cases of its overextension causing complications. Generally, extension of CH into the periapical tissues in noncritical areas does not cause lasting defects. Although they do not advocate deliberate delivery of CH to the periapical spaces, De Moor and De Witte presented 11 cases with CH extrusion that did not negatively impact healing or prognosis. Similarly, a case report by Fava found no pain or pathology resulting from extrusion of CH into the maxillary sinus following endodontic therapy on a maxillary premolar. Figure 12-12 shows an example of CH extrusion associated with clinical healing in a case of chronic apical abscess with a sinus tract and periodontal-endodontic lesion. Conversely, Lindgren et al presented a case of hemifacial ischemia and necrosis following extrusion of CH into the mandibular canal and the surrounding capillary bed of the face, palate, and ear. Similarly, Ahlgren et al presented a case of lower lip paresthesia due to extrusion of CH into the mandibular canal. Following surgical excision of the material and reactionary material, the paresthesia resolved.

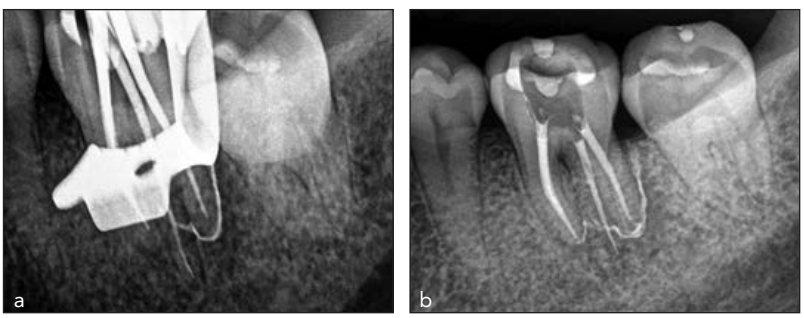

Fig 12-12 CH extruded into a periodontal-endodontic lesion at time of pulpectomy associated with clinical closure of prior sinus tract and periodontal pocket. Radiographs taken at time of cone fit *(a)* and postobturation *(b)*.

Overextension of obturation materials is an area of controversy in endodontics. Schilder advocated the classic "sealer puff" for all root canal fills (Fig 12-13). This assumes that a slight extrusion of endodontic sealer will not result in pathology. Augsburger and Peters supported this claim when they found that eugenol-based sealers were resorbed over time. In an animal study, Kawakami et al injected Vista-Cal [Vista Dental], a sealer consisting of CH, iodoform, and silicone oil, into the mandibular canal and found that macrophages phagocytosed the material, leading to time-dependent resorption. On the other hand, Seltzer et al (1973) showed that extrusion of obturation material beyond the apex caused a foreign body reaction, and Sjogren et al (1995, 1998) found chronic inflammation and bone resorption resulting from tissue exposure to gutta-percha (GP).

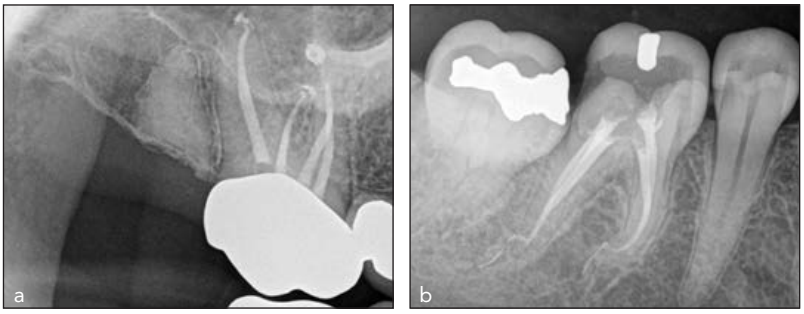

Fig 12-13 *(a and b)* "Puffs" of sealer are a common, usually nonpathologic occurrence following root canal therapy.

Though controversy exists regarding overextension of obturation material into the periapical tissues, the proximity of root apices to critical anatomical structures can make overextension problematic. Knowles et al demonstrated that the apices of the mandibular molars and premolars lie in close proximity to the mental foramen and mandibular nerve. Overextension of material out of the apices of posterior mandibular teeth can put these structures at risk. Gluskin cited possible mechanical, compression, and chemical injuries to the mandibular nerve as potential complicating factors in overextension. Rowe reported that extrusion of sealer into the mandibular canal could cause paresthesia due to either direct pressure of the material on the neurovascular bundle or neurotoxicity. Tilotta-Yasukawa et al presented a case series showing a correlation between proximity of obturation materials and the mandibular canal with an increased likelihood of paresthesias.

The composition of the extruded material plays a role in the severity of any foreign body reaction. As discussed earlier, GP can be cytotoxic, but root canal sealers are generally considered to be the more noxious agents. Pascon, Langeland, et al reported that all sealer materials are inherently cytotoxic in their freshly mixed state. Kozam demonstrated significant neurotoxic effects of eugenol on the sciatic nerves of bullfrogs. Presumably, similar neurotoxicity might occur if eugenol-based sealers contact the mandibular nerve. Tamse et al presented two cases of paresthesias following extrusion of an AH 26 [Dentsply] sealer into the mandibular canal. Kleier and Averbach presented a case of painful dysesthesia following extrusion of a paraformaldehyde N2-type paste into the mandibular canal.

Just as mandibular overextensions have been associated with the development of pathology, so have maxillary extrusions. The proximity of the apices of the maxillary molars to the maxillary sinus can increase the risk for development of sinus pathology. In a CBCT study, Pagin et al showed that 22% of maxillary posterior teeth were located in close proximity to the maxillary sinus, with 14% of root apices protruding into the sinus. Similarly, Rigolone et al found that 25% of maxillary sinuses extend between the buccal and palatal roots of maxillary molars, again based on CBCT imaging studies. Giardino et al reported a case of *Aspergillus* infection in the maxillary sinus following extrusion of a zinc oxide–eugenol sealer beyond the apex of an endodontically treated maxillary molar, presumably related to the zinc requirement for *Aspergillus* metabolism. Kaplowitz presented a case of chronic maxillary sinusitis following overextension of a GP fill in the palatal root of a maxillary molar. Paraformaldehyde-containing sealers, though no longer commercially available, are known cytotoxins. Orlay presented a case of severe pain following overextension of an N2-type paraformaldehyde-containing sealer into the maxillary sinus.

Above all, the development of signs or symptoms of pathology, including sinusitis when foreign materials reach the maxillary sinus or paresthesias or dysesthesias when materials reach the mandibular nerve, should be recognized and managed or referred for proper treatment when appropriate. Particularly in cases of nerve injury, time is of the essence. Pogrel reported that microsurgical treatment of mandibular nerve injuries should occur within 48 hours of the injury for maximal success.

Thermal injury

Thermal injuries to the periodontium can result from a multitude of heat-producing instruments used during endodontic therapy. Devices—including ultrasonic and high- and slow-speed handpieces, obturation aids including down-packing systems, and thermoplasticized GP delivery systems—create significant amounts of heat that can traumatize living structures. In order to prevent injury to the periodontal ligament, Eriksson and Albrektsson showed that root surface temperatures should not increase more than 10°C (Fig 12-14). Floren et al showed that maintaining the System B endodontic fill device [Kerr] at less than 250°C prevented said increases. According to Lee et al (1998), the Touch 'n Heat 5400 [Kerr] and flame-heated methods of down-packing GP for vertical condensation provided limited temperature control and could therefore involve root surface temperature increases of more than 10°C; this makes them less desirable as obturation aids. Based on a clinical study on dogs, Gutmann et al reported that thermoplasticized GP was safe in terms of its effects on the periodontium, with very little risk of soft tissue thermal injury.

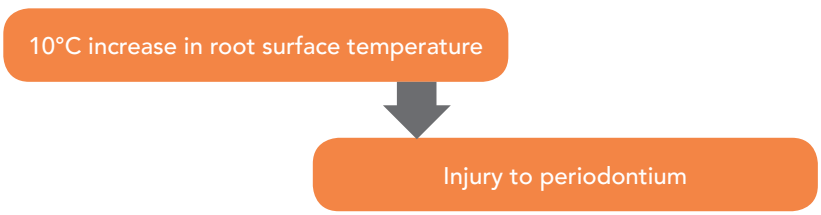

Fig 12-14 Threshold for thermal injury (Eriksson and Albrektsson).

Ultrasonic instruments, including those used to remove posts or create apical preparations during surgical endodontic therapy, create heat, particularly when used without adequate coolant. Davis et al suggested that post removal with ultrasonic instruments is particularly risky due to the relatively short amount of time during which increases to root temperature can occur that are high enough to cause periodontal damage. They found that only 20 seconds of dry ultrasonic instrumentation raised external root surface temperatures 10°C. Gluskin et al suggested that the use of adequate water or air coolant could prevent damaging temperature increases. Suggestions for methods to reduce the risk of thermal injury by ultrasonic instrumentation are summarized in Fig 12-15.

Fig 12-15 Methods to reduce thermal injury with the use of ultrasonic instruments.

Root-end surgery involves ultrasonic root-end preparations and osteotomy creation with either ultrasonic handpieces or high-speed burs. Nicoll and Peters reported that water irrigation protected slabs of dentin from temperature increases with ultrasonic scaling. This work was applied to surgical endodontics, and consequently, coolants are recommended during ultrasonic or high-speed drill manipulation of bone and surrounding tissues.

Like surgical endodontics, delivery of intraosseous anesthesia requires osteotomy preparation. Woodmansey et al reported a case of osteonecrosis following osteotomy preparation for intraosseous anesthesia without coolant. Although this particular case may have involved increased risk for nonhealing due to surgical access to remove a separated perforator and medical compromise of the patient, the authors suggested that short-duration use of the perforator could minimize the frictional heat production and therefore the risk of thermal injury to the periodontium.

Air emphysema

Air emphysema can occur during endodontic treatment when air is forced through the root canal space into the periapical tissue and beyond, creating swelling and crepitus. Classically, Shovelton presented 13 cases of air emphysema secondary to endodontic therapy, including one case in which a clarinet player introduced air via an open endodontic access. Eleazer and Eleazer found that air syringes created significant air pressure in periapical spaces in an in vitro model. A review by Hulsmann and Hahn found that air emphysema was self limiting and generally resolved without intervention. However, dramatic sequelae including fatal pneumomediastinum can result. An et al presented a case of orbital, cervicofacial, and mediastinal emphysema following endodontic retreatment of a mandibular premolar.

As swelling involving the fascial planes is commonly noted immediately following endodontic procedures, a differential diagnosis should include postoperative flare-up, necrotizing fasciitis, allergic reactions, angioedema, hematoma, or air emphysema (Fig 12-16).

Fig 12-16 Differential diagnoses for swelling noted postoperatively.

Brain abscess

A brain abscess is perhaps one of the most feared complications following untreated or recurrent endodontic disease. Periapical abscesses are generally considered encapsulated, with little risk of associated bacteremia unless overinstrumentation occurs during treatment (Bender et al). However, Li et al, reviewed the direct and indirect pathways for spread of root canal pathology to the brain (Fig 12-17). Direct extension can occur via fascial planes, hematogenous spread via the valveless facial veins or systemic circulation, or lymphatic spread. Indirect extension can occur via seeding from secondary extraoral infections; for example, a secondary brain abscess can develop following bacterial endocarditis of dental origin.

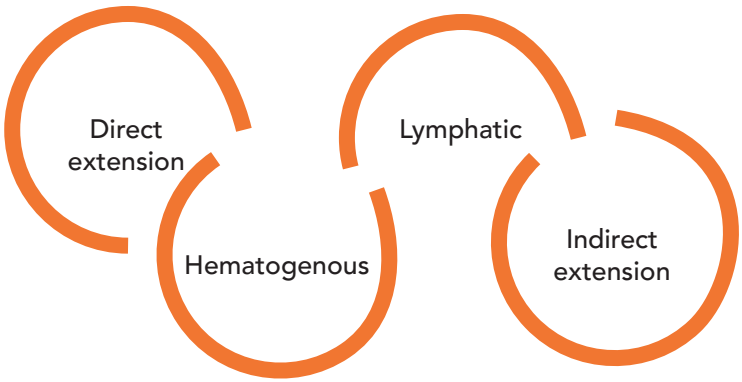

Fig 12-17 Proposed pathways for brain abscess from endodontic infection (Li et al).

Allergy

Allergic hypersensitivity reactions can occur to materials used in endodontic therapy. These reactions can be classified into four types (Fig 12-18). Types I and IV create the traditional allergic reactions experienced secondary to contact and ingested allergens commonly encountered in endodontics. Type I hypersensitivity reactions occur immediately on exposure to the allergen and result in varying physiologic responses dependent on the target tissue. Type I reactions can include urticaria, bronchospasm, vomiting, diarrhea, and anaphylaxis. Type IV hypersensitivity reactions are delayed and usually occur between 48 and 72 hours postexposure. These represent an antibody response to an allergen to which the patient had been previously exposed. Contact dermatitis is the classic type IV hypersensitivity reaction. For a full review of allergies, please review a textbook on inflammation, such as Trowbridge and Emling's *Inflammation: A Review of the Process.*

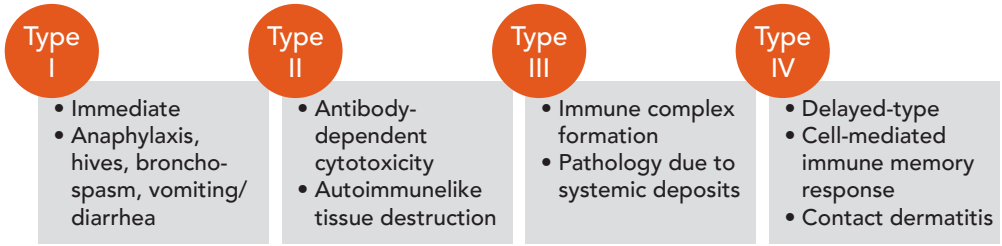

Fig 12-18 The hypersensitivity reactions (Trowbridge and Emling).

During any new patient encounter, any history of allergy must be recorded. Certain allergies, such as to latex rubber and certain antibiotics, are relatively common and are relevant in the diagnosis and treatment planning of endodontic disease. Less common allergens may still be relevant, including other pharmacologic agents (eg, analgesics and anesthetics) and certain types of dental materials. Knowledge of medication sensitivities, especially in patients who have had particularly adverse reactions to certain medications or materials in the past, is also relevant and should be accounted for in patient-specific planning. In some cases, it may be necessary to coordinate treatment with a patient's physician to mitigate allergy-associated risks.

Allergies to endodontic dental materials are rare, but several have been reported in the literature. Case reports exist of more severe reactions than anticipated given the exposure. Gazelius et al presented a case report of confirmed contact allergy with GP in a patient with latex skin allergy, resulting in prolonged pain following obturation that resolved once the offending GP was removed. Barkin et al presented a case of severe allergic reaction to eugenol in an endodontic sealer.

These reports, however, appear to be the exception rather than the norm. Although latex rubber allergies are relatively common, the risk of cross-allergenicity with commercially available GP is low. Costa et al investigated this cross-reactivity and found only potential cross-allergenicity between latex rubber and raw GP, which is only rarely added to commercial GP products. As a result, they advise that clinicians should avoid the use of GP only in patients with a type I hypersensitivity reaction to natural rubber latex. If the patient exhibits a type IV hypersensitivity to latex, avoidance of GP is not necessary.

Adverse reactions to local anesthesia

Besides the rarely reported allergic reaction, administration of local anesthesia for endodontic therapy does pose several risks to patients. Infections and paresthesia can occur following the injection of local anesthetics in the oral cavity.

The introduction of any material in violation of the oral mucosa introduces the risk of infection, and needle tract infections are a risk following administration of local anesthesia. Connor and Edelson presented a case report of a facial cellulitis attributed to the introduction of pathologic oral bacteria via local infiltration dental anesthesia.

Reports exist in the literature of long-lasting paresthesias following administration of local anesthetics, potentially due to physical trauma or neurotoxicity. Pogrel and Thamby reported a low incidence of permanent paresthesia following inferior alveolar nerve block anesthesia. Because paresthesia represents an immensely unfavorable reaction and occurs in response to a commonly performed injection, efforts to reduce this incidence based on risk factors are of interest. Haas and Lennon as well as Gaffen and Haas found a five-fold increase in paresthesias when articaine was implemented for IAN blocks. More recently, Garisto et al reported that prilocaine and articaine used for dental local anesthesia were associated with an increased risk of paresthesia at 7.3 and 3.6 times, respectively (Fig 12-19). Most cases involved paresthesia of the lingual nerve following inferior alveolar nerve block anesthesia. The authors proposed that this increased risk of paresthesia may be due to the 4% concentration of commercially available prilocaine and articaine, which is higher than that of other commercially available local anesthetics like lidocaine, bupivacaine, and mepivacaine, resulting in greater potential for neurotoxicity. In addition, they proposed that the lingual nerve generally has simpler architecture at the level at which the mandibular block is traditionally administered, differentiating it from the main branch of the inferior alveolar nerve at that location. As a result of these findings, as well as the lack of evidence to support superior effectiveness of these higher concentrations, Garisto et al suggested that local anesthetics in concentrations of 4% might be avoided for inferior alveolar block anesthesia.

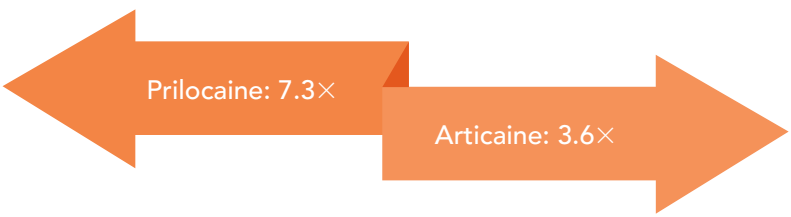

Fig 12-19 Prilocaine and articaine have an increased risk of paresthesia when used for local anesthesia, particularly for inferior alveolar nerve blocks (Garisto et al).

Paresthesia following root-end surgery

Elevation of a soft tissue flap for any dentoalveolar surgical procedure involves severance of superficial nerves, and short- or long-duration paresthesia can result, depending on the extent of injury. According to Kim et al, as long as complete severance of a major nerve bundle has not occurred, normal sensation should recur in approximately 4 weeks, or in rare cases, within a few months. Wesson and Gale reported that transient paresthesia is a

common complication in mandibular molar surgery, with 20% of patients experiencing some sensory disturbance. The majority of patients experienced resolution over time, as only 1% of patients reported a permanent deficit.

Post-Treatment Complications

Flare-ups

The American Association of Endodontists (AAE) Glossary of Endodontic Terms defines a *flare-up* as "an acute exacerbation of periradicular pathosis after initiation or continuation of root canal treatment." In a meta-analysis, Tsesis et al reported the incidence of postoperative flare-up as relatively low at 8.4%. The authors proposed that the etiology is likely multifactorial, comprising chemical, mechanical, and microbial factors inherent in the endodontic disease and treatment (Fig 12-20). Despite this proposal, no consensus on causation has been achieved, and the heterogeneity of data included in the meta-analysis made statistical analysis impossible. Siqueira reviewed the potential causes of flare-up and proposed that the altered apical environment created by endodontic therapy might allow for a changed balance in host defenses, facilitating increased microbial aggression.

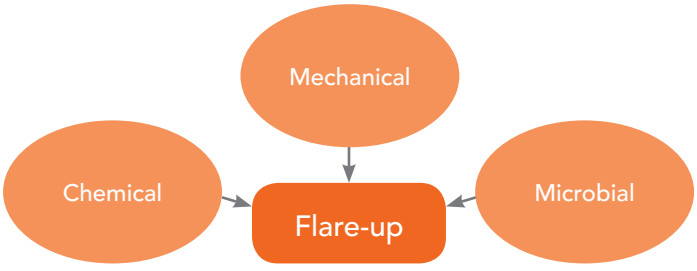

Fig 12-20 Potential etiologies of flare-up (Tsesis et al).

The literature is replete with potential predictors for flare-ups. In a retrospective chart review, Torabinejad et al found that age greater than 40 years old, female sex, mandibular teeth, allergies, preoperative pain, and analgesic use, as well as retreatment, were associated with an increased risk of flare-ups. Although Torabinejad et al found that the absence of periapical lesions was associated with an increased risk of flare-up, Iqbal et al found the opposite and associated preoperative periapical radiolucencies with an increased risk of flare-up. The presence of preoperative pain and analgesic use seems to be the most reported risk factor in the literature, with additional support in studies by Imura and Zuolo as well as Walton and Fouad. Figure 12-21 summarizes potential risk factors for flare-ups.

Several authors proposed that multiple-visit therapy and the use of an antimicrobial intracanal medicament might reduce the incidence of flare-up in necrotic, infected teeth. In a prospective study, Trope reported an increased incidence of flare-ups when retreatment procedures were performed on teeth with apical periodontitis in a single visit. However, a systematic review by Sathorn et al found no evidence to support an increased incidence of flare-up in single- versus multiple-visit therapy. Eleazer and Eleazer actually reported a lower incidence of flare-up in single-visit compared with multiple-visit treatment in a retrospective analysis of necrotic teeth.

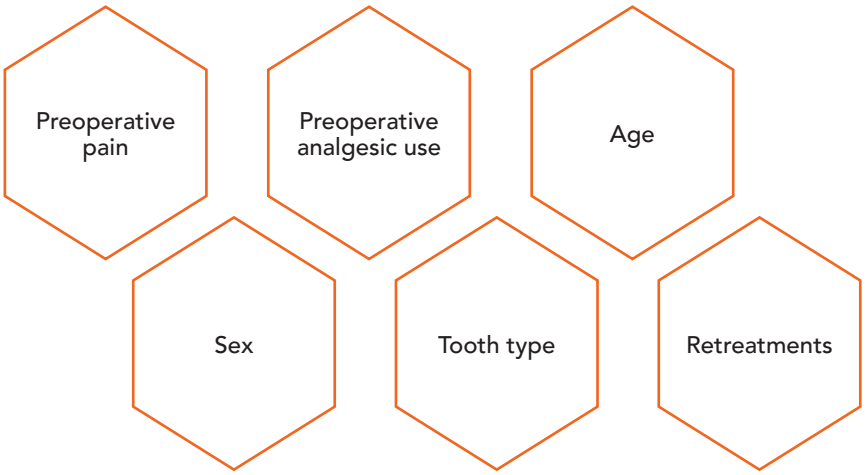

Fig 12-21 Risk factors for flare-up (Torabinejad et al).

Clinically, Walton defined a *flare-up* as an increase in pain or swelling, usually within a few hours to a few days following treatment, that prompts an unscheduled visit to the treating clinician. He suggested that treatment should involve psychologic management to reassure the patient that the condition is treatable and should not affect the prognosis, localized treatment to reclean and medicate the canal or incise and drain swelling, and pharmacologic management (Fig 12-22). Pharmacologic management might involve the use of long-acting local anesthetics, such as bupivacaine (Dunsky and Moore), or systemic drugs, including analgesics, steroids, and antibiotics, where indicated. For a review of relevant medications as well as their indications for use, please see chapter 5.

Fig 12-22 Management of flare-ups (Walton).

Some have proposed the use of prophylactic antibiotics to prevent the occurrence of postoperative flare-ups. Abbott et al reported that preoperative antibiotics reduced the incidence of flare-ups following treatment of asymptomatic necrotic, infected teeth. This study was flawed, however, as it used an outside control group. Other literature has failed to support the practice of routine prophylactic antibiotic prescription. A prospective randomized controlled trial by Pickenpaugh et al found no reduction in the incidence of flare-up by prophylactic amoxicillin in asymptomatic, necrotic teeth. Similarly, a prospective study by Walton and Chiappinelli found no effects with preoperative penicillin.

Thankfully, the occurrence of a flare-up does not affect the overall prognosis. Sjogren et al and Friedman et al each found no statistically significant effect of postoperative flare-ups on the overall prognosis of nonsurgical root canal therapy.

Failures

Though not traditionally thought of as a complication, failure of endodontic therapy, either in the short or long term, is an adverse event that both the provider and patient hope to avoid. Failures occur for a multitude of reasons but can be generally classified as persistent infections or secondary reinfection of a previously cleaned space (Fig 12-23).

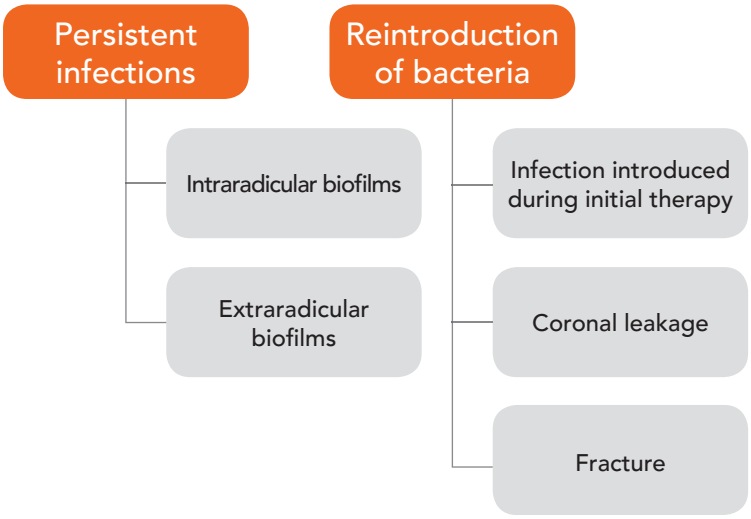

Fig 12-23 Possible etiologies of failing endodontic therapy.

Persistent infections may result from intraradicular or extraradicular factors. Siqueira et al defined *persistent infections* as those due to the originally infective microbes. This may be due to persistent intradicular or extraradicular biofilms that both the antimicrobial treatment methods and immune system could not completely address. Happonen also suggested that extraradicular periapical actinomycosis may be a causative agent for persistent infections due to its particular resistance to traditional treatment modalities. Happonen recommended surgical removal of the apical lesion to treat persistent periapical actinomycosis. Although true cysts and foreign bodies are theoretically nonmicrobial, Siqueira et al raised skepticism that an intraradicular or extraradicular biofilm component could ever be completely ruled out as a contributor to persistent pathology in these cases. Similarly, they ruled out that procedural errors such as instrument separation or overfilling directly resulted in failure without a persistent intraradicular or extraradicular infection as the more likely etiologic agent.

Secondary infections, unlike persistent infections, occur due to reintroduction of bacteria from the oral cavity. Siqueira et al defined *secondary infections* as those occurring due to microbes different from those present with the inciting endodontic disease. These microbes may be introduced during endodontic therapy, as when adequate rubber dam isolation is not used, or due to breakdown of the coronal seal following treatment. Swan-

son and Madison performed an in vitro study of coronal leakage and showed that only 3 days of saliva exposure following loss of a temporary filling led to significant microbial contamination. The AAE glossary described *fractures* as a pathway for recurrent infection and failure of root canal therapy. Vertical root fractures in particular most often occur in previously endodontically treated teeth and provide a direct pathway for reinfection. Figures 12-24 and 12-25 depict examples of failures attributed to caries and vertical root fracture, respectively.

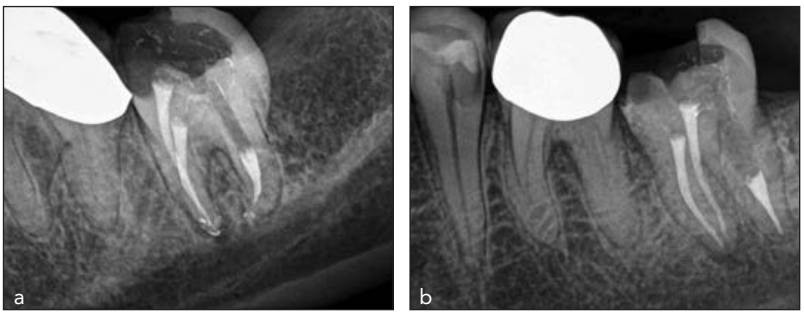

Fig 12-24 Failure due to coronal leakage. *(a)* The patient failed to have a permanent restoration placed following root canal therapy. *(b)* The result was recurrence of symptoms 1 year later due to lost temporary fill and recurrent caries.

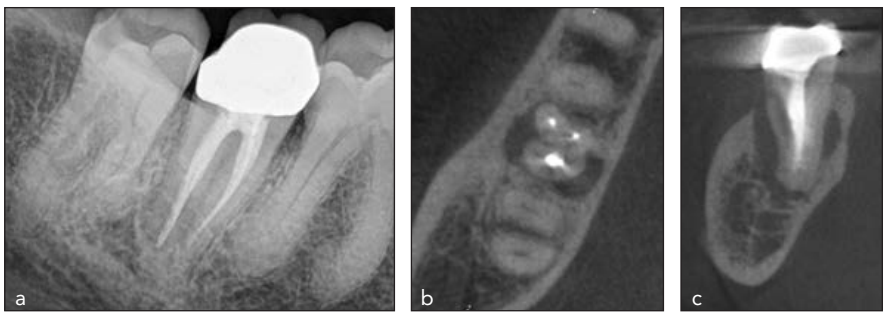

Fig 12-25 Failure due to vertical fracture in the distal root of the mandibular right second molar. The vertical fracture and resultant bone loss is less apparent in the periapical radiograph *(a)* than in either the axial *(b)* or coronal *(c)* CBCT images.

Persistent pain

Similar to failures, persistent pain following endodontic therapy is something both the provider and patient prefer to avoid. Short-term pain following endodontic treatment is relatively common. Law et al found that 19% of patients reported severe pain following endodontic therapy lasting an average of 2 days. Long-term pain, however, is relatively uncommon. In a systematic review, Nixdorf et al (2010a) found that 5% of patients experienced persistent pain more than 6 months following endodontic therapy. Polycarpou et al found that female sex, a history of chronic pain, preoperative mechanical allodynia (measured by percussion tenderness), and preoperative pain were all risk factors for persistent pain.

Persistent pain may be of endodontic origin or in other cases may arise from other sources. Using the same data from their earlier systematic review, Nixdorf et al (2010b) found that, of the 5% of patients with long-term discomfort, 3.4% of patients' pain was of nonodontogenic origin (Fig 12-26). Put another way, the frequency of long-term odontogenic pain was 1.6%. Sources of nonodontogenic pain included musculoskeletal or neuropathic pain, headaches, and pathology related to nearby structures such as the maxillary sinuses, salivary glands, vasculature, brain tumors, angina, or throat cancer (Nixdorf et al 2010b). Unfortunately, many episodes of nonodontogenic persistent pain may indicate that the original endodontic treatment may have been performed due to a misdiagnosis. Odontogenic causes of persistent pain may be difficult to ascertain and can include refractory infections, infraction, periodontal disease, and traumatic occlusion among other sources. For more information on non-endodontic pain, please refer to chapter 7.

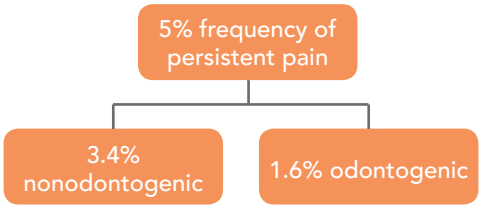

Fig 12-26 The frequency of persistent pain following endodontic treatment, and its origins (Nixdorf et al 2010a, 201b).

Whether odontogenic or not, persistent pain oftentimes comprises a grab bag diagnosis, and a definitive diagnosis may elude the provider. In a study that also found a 5% frequency of persistent pain, Vena et al found that two-thirds of those patients reporting pain did not have an identifiable cause. Obviously, a lack of definitive diagnosis comes with its own psychosocial consequences as well as a high likelihood of ineffective treatment. As diagnostic tools improve, most notably CBCT imaging, many patients that had previously fallen into this diagnostic category may be moved elsewhere. For example, CBCT imaging can often clearly diagnose early endodontic failures due to reinfection, whereas traditional two-dimensional imaging might not show early signs of apical pathology rendering an inconclusive diagnosis.

Bibliography

Intratreatment Complications

Ahlgren FK, Johannessen AC, Hellem S. Displaced calcium hydroxide paste causing inferior alveolar nerve paraesthesia: Report of a case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;96:734–737.

An GK, Zats B, Kunin M. Orbital, mediastinal, and cervicofacial subcutaneous emphysema after endodontic retreatment of a mandibular premolar: A case report. J Endod 2014;40:880–883.

Ankrum MT, Hartwell GR, Truitt JE. K3 Endo, ProTaper, and ProFile systems: Breakage and distortion in severely curved roots of molars. J Endod 2004;30:234–237.

Augsburger RA, Peters DD. Radiographic evaluation of extruded obturation materials. J Endod 1990;16:492–497.

Barkin ME, Boyd JP, Cohen S. Acute allergic reaction to eugenol. Oral Surg Oral Med Oral Pathol 1984;57:441–442.

Bender IB, Seltzer S, Yermish M. The incidence of bacteremia in endodontic manipulation: Preliminary report. Oral Surg Oral Med Oral Pathol 1960;13:353–360.

Connor JP, Edelson JG. Needle tract infection. A case report. Oral Surg Oral Med Oral Pathol 1988;65:401–403.

Costa GE, Johnson JD, Hamilton RG. Cross-reactivity studies of gutta-percha, gutta-balata, and natural rubber latex (*Hevea brasiliensis*). J Endod 2001;27:584–587.

Crump MC, Natkin E. Relationship of broken root canal instruments to endodontic case prognosis: A clinical investigation. J Am Dent Assoc 1970;80:1341–1347.

Davis S, Gluskin AH, Livingood PM, Chambers DW. Analysis of temperature rise and the use of coolants in the dissipation of ultrasonic heat buildup during post removal. J Endod 2010;36:1892–1896.

De Moor RJ, De Witte AM. Periapical lesions accidentally filled with calcium hydroxide. Int Endod J 2002;35:946–958.

Dragoo MR. Resin-ionomer and hybrid-ionomer cements: Part II, human clinical and histologic wound healing responses in specific periodontal lesions. Int J Periodontics Restorative Dent 1997;17:75–87.

Eleazer PD, Eleazer KR. Air pressures developed beyond the apex from drying root canals with pressurized air. J Endod 1998;24:833–836.

Eriksson AR, Albrektsson T. Temperature threshold levels for heat-induced bone tissue injury: A vital-microscopic study in the rabbit. J Prosthet Dent 1983;50:101–107.

Fava LR. Calcium hydroxide paste in the maxillary sinus: A case report. Int Endod J 1993;26:306–310.

Floren JW, Weller RN, Pashley DH, Kimbrough WF. Changes in root surface temperatures with in vitro use of the system B HeatSource. J Endod 1999;25:593–595.

Fu M, Zhang Z, Hou B. Removal of broken files from root canals by using ultrasonic techniques combined with dental microscope: A retrospective analysis of treatment outcome. J Endod 2011;37:619–622.

Fuss Z, Assooline LS, Kaufman AY. Determination of location of root perforations by electronic apex locators. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82:324–329.

Fuss Z, Trope M. Root perforations: Classification and treatment choices based on prognostic factors. Endod Dent Traumatol 1996;12:255–264.

Gaffen AS, Haas DA. Retrospective review of voluntary reports of nonsurgical paresthesia in dentistry. J Can Dent Assoc 2009;75:579.

Garisto GA, Gaffen AS, Lawrence HP, Tenenbaum HC, Haas DA. Occurrence of paresthesia after dental local anesthetic administration in the United States. J Am Dent Assoc 2010;141:836–844.

Gazelius B, Olgart L, Wrangsjo K. Unexpected symptoms to root filling with gutta-percha. A case report. Int Endod J 1986;19:202–204.

Giardino L, Pontieri F, Savoldi E, Tallarigo F. Aspergillus mycetoma of the maxillary sinus secondary to overfilling of a root canal. J Endod 2006;32:692–694.

Gluskin AH. Anatomy of an overfill: A reflection on the process. Endod Topics 2009;16:64–81.

Gluskin AH, Ruddle CJ, Zinman EJ. Thermal injury through intraradicular heat transfer using ultrasonic devices: Precautions and practical preventive strategies. J Am Dent Assoc 2005;136:1286–1293.

Gutmann JL, Rakusin H, Powe R, Bowles WH. Evaluation of heat transfer during root canal obturation with thermoplasticized gutta-percha. Part II. In vivo response to heat levels generated. J Endod 1987;13:441–448.

Haas DA, Lennon D. A 21-year retrospective study of reports of paresthesia following local anesthetic administration. J Can Dent Assoc 1995;61:319–320, 323–316, 329–330.

Hulsmann M, Hahn W. Complications during root canal irrigation—Literature review and case reports. Int Endod J 2000;33:186–193.

Iqbal MK, Kohli MR, Kim JS. A retrospective clinical study of incidence of root canal instrument separation in an endodontics graduate program: A PennEndo database study. J Endod 2006;32:1048–1052.

Kaplowitz GJ. Penetration of the maxillary sinus by overextended gutta percha cones. Report of two cases. Clin Prev Dent 1985;7:28–30.

Kawakami T, Nakamura C, Eda S. Effects of the penetration of a root canal filling material into the mandibular canal. 1. Tissue reaction to the material. Endod Dent Traumatol 1991;7:36–41.

Kim S, Pecora G, Rubinstein RA. Color Atlas of Microsurgery in Endodontics. Philadelphia: Saunders, 2001.

Kleier DJ, Averbach RE. Painful dysesthesia of the inferior alveolar nerve following use of a paraformaldehyde-containing root canal sealer. Endod Dent Traumatol 1988;4:46–48.

Knowles KI, Jergenson MA, Howard JH. Paresthesia associated with endodontic treatment of mandibular premolars. J Endod 2003;29:768–770.

Kozam G. The effect of eugenol on nerve transmission. Oral Surg Oral Med Oral Pathol 1977;44:799–805.

Lee FS, Van Cura JE, BeGole E. A comparison of root surface temperatures using different obturation heat sources. J Endod 1998;24:617–620.

Lee SJ, Monsef M, Torabinejad M. Sealing ability of a mineral trioxide aggregate for repair of lateral root perforations. J Endod 1993;19:541–544.

Li X, Tronstad L, Olsen I. Brain abscesses caused by oral infection. Endod Dent Traumatol 1999;15:95–101.

Lindgren P, Eriksson KF, Ringberg A. Severe facial ischemia after endodontic treatment. J Oral Maxillofac Surg 2002;60:576–579.

Madarati AA, Hunter MJ, Dummer PM. Management of intracanal separated instruments. J Endod 2013;39:569–581.

Matthews J, Merrill RL. Sodium hypochlorite-related injury with chronic pain sequelae. J Am Dent Assoc 2014;145:553–555.

Mente J, Leo M, Panagidis D, Saure D, Pfefferle T. Treatment outcome of mineral trioxide aggregate: Repair of root perforations—Long-term results. J Endod 2014;40:790–796.

Nicoll BK, Peters RJ. Heat generation during ultrasonic instrumentation of dentin as affected by different irrigation methods. J Periodontol 1998;69:884–888.

Orlay HG. Overfilling in root canal treatment. Two accidents with N2. Br Dent J 1966;120:376.

Pagin O, Centurion BS, Rubira-Bullen IR, Alvares Capelozza AL. Maxillary sinus and posterior teeth: Accessing close relationship by cone-beam computed tomographic scanning in a Brazilian population. J Endod 2013;39:748–751.

Pascon EA, Leonardo MR, Safavi K, Langeland K. Tissue reaction to endodontic materials: Methods, criteria, assessment, and observations. Oral Surg Oral Med Oral Pathol 1991;72:222–237.

Pitt Ford TR, Torabinejad M, McKendry DJ, Hong CU, Kariyawasam SP. Use of mineral trioxide aggregate for repair of furcal perforations. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:756–763.

Pogrel MA. Damage to the inferior alveolar nerve as the result of root canal therapy. J Am Dent Assoc 2007;138:65–69.

Pogrel MA, Thamby S. Permanent nerve involvement resulting from inferior alveolar nerve blocks. J Am Dent Assoc 2000;131:901–907.

Reeh ES, Messer HH. Long-term paresthesia following inadvertent forcing of sodium hypochlorite through perforation in maxillary incisor. Endod Dent Traumatol 1989;5:200–203.

Rigolone M, Pasqualini D, Bianchi L, Berutti E, Bianchi SD. Vestibular surgical access to the palatine root of the superior first molar: "Low-dose cone-beam" CT analysis of the pathway and its anatomic variations. J Endod 2003;29:773–775.

Rowe AH. Damage to the inferior dental nerve during or following endodontic treatment. Br Dent J 1983;155:306–307.

Sabala CL, Powell SE. Sodium hypochlorite injection into periapical tissues. J Endod 1989;15:490–492.

Schilder H. Filling root canals in three dimensions. Dent Clin North Am 1967:723–744.

Seltzer S, Sinai I, August D. Periodontal effects of root perforations before and during endodontic procedures. J Dent Res 1970;49:332–339.

Seltzer S, Soltanoff W, Smith J. Biologic aspects of endodontics. V. Periapical tissue reactions to root canal instrumentation beyond the apex and root canal fillings short of and beyond the apex. Oral Surg Oral Med Oral Pathol 1973;36:725–737.

Shemesh H, Cristescu RC, Wesselink PR, Wu MK. The use of cone-beam computed tomography and digital periapical radiographs to diagnose root perforations. J Endod 2011;37:513–516.

Shovelton D. Surgical emphysema as a complication of dental operations. Br Dent J 1957;102:125–129.

Sjogren U, Ohlin A, Sundqvist G, Lerner UH. Gutta-percha-stimulated mouse macrophages release factors that activate the bone resorptive system of mouse calvarial bone. Eur J Oral Sci 1998;106:872–881.

Sjogren U, Sundqvist G, Nair PN. Tissue reaction to gutta-percha particles of various sizes when implanted subcutaneously in guinea pigs. Eur J Oral Sci 1995;103:313–321.

Spili P, Parashos P, Messer HH. The impact of instrument fracture on outcome of endodontic treatment. J Endod 2005;31:845–850.

Suter B, Lussi A, Sequeira P. Probability of removing fractured instruments from root canals. Int Endod J 2005;38:112–123.

Tamse A, Kaffe I, Littner MM, Kozlovsky A. Paresthesia following overextension of AH-26: Report of two cases and review of the literature. J Endod 1982;8:88–90.

Tilotta-Yasukawa F, Millot S, El Haddioui A, Bravetti P, Gaudy JF. Labiomandibular paresthesia caused by endodontic treatment: An anatomic and clinical study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102:e47–e59.

Trowbridge HO, Emling RC. Inflammation: A Review of the Process, ed 5. Chicago: Quintessence, 1997.

Wesson CM, Gale TM. Molar apicectomy with amalgam root-end filling: Results of a prospective study in two district general hospitals. Br Dent J 2003;195:707–714.

Woodmansey KF, White RK, He J. Osteonecrosis related to intraosseous anesthesia: Report of a case. J Endod 2009;35:288–291.

Post-Treatment Complications

Abbott AA, Koren LZ, Morse DR, Sinai IH, Doo RS, Furst ML. A prospective randomized trial on efficacy of antibiotic prophylaxis in asymptomatic teeth with pulpal necrosis and associated periapical pathosis. Oral Surg Oral Med Oral Pathol 1988;66:722–733.

American Association of Endodontists. Endodontics Colleagues for Excellence: Cracking the Cracked Tooth Code: Detection and Treatment of Various Longitudinal Tooth Fractures. Chicago: American Association of Endodontists, 2008.

American Association of Endodontists. Glossary of Endodontic Terms. dev.aae.org/glossary/. Accessed 20 January 2016.

Dunsky JL, Moore PA. Long-acting local anesthetics: A comparison of bupivacaine and etidocaine in endodontics. J Endod 1984;10:457–460.

Eleazer PD, Eleazer KR. Flare-up rate in pulpally necrotic molars in one-visit versus two-visit endodontic treatment. J Endod 1998;24:614–616.

Friedman S, Abitbol S, Lawrence HP. Treatment outcome in endodontics: The Toronto Study. Phase 1: Initial treatment. J Endod 2003;29:787–793.

Happonen RP. Periapical actinomycosis: A follow-up study of 16 surgically treated cases. Endod Dent Traumatol 1986;2:205–209.

Imura N, Zuolo ML. Factors associated with endodontic flare-ups: A prospective study. Int Endod J 1995;28:261–265.

Iqbal M, Kurtz E, Kohli M. Incidence and factors related to flare-ups in a graduate endodontic programme. Int Endod J 2009;42:99–104.

Law AS, Nixdorf DR, Rabinowitz I, et al. Root canal therapy reduces multiple dimensions of pain: A national dental practice-based research network study. J Endod 2014;40:1738–1745.

Nixdorf DR, Moana-Filho EJ, Law AS, McGuire LA, Hodges JS, John MT. Frequency of persistent tooth pain after root canal therapy: A systematic review and meta-analysis. J Endod 2010a;36:224–230.

Nixdorf DR, Moana-Filho EJ, Law AS, McGuire LA, Hodges JS, John MT. Frequency of nonodontogenic pain after endodontic therapy: A systematic review and meta-analysis. J Endod 2010b;36:1494–1498.

Pickenpaugh L, Reader A, Beck M, Meyers WJ, Peterson LJ. Effect of prophylactic amoxicillin on endodontic flare-up in asymptomatic, necrotic teeth. J Endod 2001;27:53–56.

Polycarpou N, Ng YL, Canavan D, Moles DR, Gulabivala K. Prevalence of persistent pain after endodontic treatment and factors affecting its occurrence in cases with complete radiographic healing. Int Endod J 2005;38:169–178.

Sathorn C, Parashos P, Messer H. The prevalence of postoperative pain and flare-up in single- and multiple-visit endodontic treatment: A systematic review. Int Endod J 2008;41:91–99.

Siqueira JF Jr. Microbial causes of endodontic flare-ups. Int Endod J 2003;36:453–463.

Siqueira JF Jr, Rôças IN, Ricucci D, Hulsmann M. Causes and management of post-treatment apical periodontitis. Br Dent J 2014;216:305–312.

Sjogren U, Hagglund B, Sundqvist G, Wing K. Factors affecting the long-term results of endodontic treatment. J Endod 1990;16:498–504.

Swanson K, Madison S. An evaluation of coronal microleakage in endodontically treated teeth. Part I. Time periods. J Endod 1987;13:56–59.

Torabinejad M, Kettering JD, McGraw JC, Cummings RR, Dwyer TG, Tobias TS. Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps. J Endod 1988;14:261–266.

Trope M. Flare-up rate of single-visit endodontics. Int Endod J 1991;24:24–26.

Tsesis I, Faivishevsky V, Fuss Z, Zukerman O. Flare-ups after endodontic treatment: A meta-analysis of literature. J Endod 2008;34:1177–1181.

Vena DA, Collie D, Wu H, et al. Prevalence of persistent pain 3 to 5 years post primary root canal therapy and its impact on oral health-related quality of life: PEARL Network findings. J Endod 2014;40:1917–1921.

Walton R, Fouad A. Endodontic interappointment flare-ups: A prospective study of incidence and related factors. J Endod 1992;18:172–177.

Walton R. Interappointment flare-ups: Incidence, related factors, prevention, and management. Endod Topics 2002;3:67–76.

Walton RE, Chiappinelli J. Prophylactic penicillin: Effect on posttreatment symptoms following root canal treatment of asymptomatic periapical pathosis. J Endod 1993;19:466–470.

Index

Page numbers followed by "f" indicate figures; those followed by "t" indicate tables

B

C

D

E

J

L

M

N

Q

R

S

T

U

V

W

X

Z

Contents

ISBN 978-0-86715-696-6
90000>
9 780867 156966