

A major and much-awaited revision of an indispensable classic

Grossman's Endodontic Practice

Editors
B. Suresh Chandra • V. Gopi Krishna

Twelfth Edition

Ever since its first publication over 70 years ago, this classic has served as the foundational text on the art and science of endodontics. Successive generations of students and clinicians have not only learnt the basics of endodontics from this book but have also followed it extensively as an authoritative clinical resource. The book kept pace with the developments in the field till the mid-1980s when its last edition was published.

The Twelfth Edition of this classic now incorporates the changes that have occurred in endodontic theory and practice over the last 20 years. This edition therefore highlights:

- The gradual shift in endodontic principles from a chemomechanically centered approach to a more biologically centered and biocompatible approach
- The innovations in materials, instruments, and techniques which have revolutionized endodontic practice in recent years

Accordingly, this edition updates the coverage of the book and includes three new chapters on:

- Prosthodontic considerations of endodontically treated teeth
- Lasers in endodontics
- Procedural errors and their management

The presentation of the concepts and techniques has also been significantly revised and the book now includes:

- Over 1100 new illustrations comprising relevant line diagrams, clinical photographs, radiographs, and histological slides
- Several case reports illustrating the conceptual and clinical discussions

With its contemporary approach and comprehensive coverage, this classic is indeed an indispensable text for both students and practitioners.

Grossman Nostalgia for Enthusiasts

This edition also presents a Tribute to Grossman from Prof. Gutmann

Prefaces to previous editions

Archived photographs of Louis I. Grossman

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Grossman's Endodontic Practice

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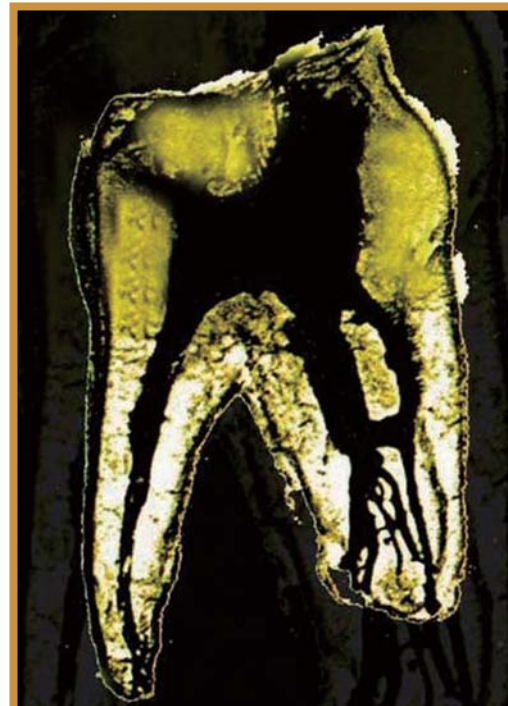
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Dedicated to my parents, wife, and children.

–B. Suresh Chandra

*Dedicated to
My beloved mother, Sulochana Velayutham, for being the
source and nurturer of my life and intellect ...
My beloved ardhangini, Lakshmi, for being the source of love,
support, and inspiration
My beloved angels, Sanjana and Sidharth, for being the source of my happiness....
And above all to the Almighty for being the source of my creativity...*

–V. Gopi Krishna



Dr. Grossman (*Reproduced with permission from AAE Archives, American Association of Endodontists, Chicago, IL.*)



Dr. Grossman presenting the AAE Louis I. Grossman Award to its first recipient Dr. Birger Nygaard-Østby
(Reproduced with permission from AAE Archives, American Association of Endodontists, Chicago, IL)

Louis I. Grossman: The Visionary Father of Modern Endodontics

Dr. Louis I. Grossman was born in a Ukrainian village near Odessa on December 16, 1901, and was brought to the United States by his family as a boy. He grew up in Philadelphia and completed his high school education at South Philadelphia High School in 1919. He earned a doctorate in dental surgery at the University of Pennsylvania in 1923 and a doctorate in medical dentistry (Dr. Med Dent) at the University of Rostock in Germany in 1928.

On December 21, 1928, he married Emma May MacIntyre and they had two children, a daughter Clara Ruth Grossman in 1939 and a son Richard Alan Grossman in 1943.

Dr. Grossman began his teaching career as an Instructor in Operative Dentistry at the University of Pennsylvania in 1927, in addition to being appointed as a Fellow in Research at the American Dental Association. In 1941 he was an Associate in Oral Medicine; he became Assistant Professor of Oral Medicine in 1947, Associate Professor of Oral Medicine in 1950, and Professor in 1954.

His achievements and honors were extensive in many sectors of dentistry with a prime focus in endodontics. He was an honorary member of the Association of Licentiates in Dental Surgery and University of Dentists of Belgium; Montreal Endodontia Society; Vancouver Endodontic Study Club, Brazilian Dental Association; Dental Association of Medellin (Colombia); and the Japanese Endodontic Association. He received an honorary Doctor of Science (ScD) from the University of Pennsylvania.

His major publication and crowning achievement was his textbook *Root Canal Therapy* published in 1940 (now known as *Endodontic Practice*) with multiple editions appearing worldwide. Subsequently translated into eight languages, the book has served as a benchmark for the development of modern endodontic philosophy and practice. Dr. Grossman also authored *Dental Formulas and Aids to Dental Practice*, first published in 1952, and the *Handbook of Dental Practice* published in 1948.

He was a chairman of the American Board of Endodontics, was a charter member of the American Association of Endodontists (AAE) and served as its President from 1948 to 1949. He was a Fellow of the American Association for the Advancement of Science.

Dr. Grossman passed away at the age of 86 in 1988. The University of Pennsylvania has honored Dr. Grossman with an endowed Professorship, usually given to the department chairperson. The AAE has honored him with the Louis I. Grossman Award that recognizes an author for cumulative publication of significant research studies that have made an extraordinary contribution to endodontology. This award is given at the AAE meeting when warranted.

A study club was formed in Philadelphia in the honor of Dr. Louis I. Grossman for his unyielding dedication and commitment towards facilitating the recognition of endodontics as a specialty in the field of dentistry. The purpose of the Louis I. Grossman Study Club was to provide an opportunity to the endodontists as well as other interested dentists to meet, share ideas, and expand and update our knowledge in the field of endodontics and dental medicine.

x ➤ Louis I. Grossman – The Visionary Father of Modern Endodontics

Dr. Louis I. Grossman was the founder of the first Root Canal Study Club. It was established in 1939 in Philadelphia, Pennsylvania, at a time when the Focal Infection Theory threatened the future of endodontics. The purpose of the Root Canal Study Club as stated in the original letter compiled by Dr. Grossman was “to study problems connected with root canal therapy and to present clinics so as to help others in practicing this important phase of dentistry more adequately.” Endodontists from as far away as Massachusetts chose Philadelphia as the hub for scientific and educational learning in the field of endodontics.

James L. Gutman

Preface to Twelfth Edition

"If I have seen further it is by standing on the shoulders of giants."

—Isaac Newton

Dr. Louis I. Grossman was one such giant in the field of endodontics. His textbook *Endodontic Practice* has over the past 70 years been not only the reporter but also the harbinger of changes sweeping through the field of endodontics. It truly deserves the title "Bible of Endodontics" as it had consistently set a benchmark of excellence in the teaching and understanding of the art and science of endodontics.

The last edition of *Endodontic Practice* (eleventh edition) was published in 1987 and tremendous changes have occurred since then, both in our understanding as well as in our practice of endodontic therapy. The focus of this current twelfth, edition is twofold; primarily, it is to update this classic book and incorporate all the advances in materials, instruments, and techniques which have revolutionized endodontics in the past two decades.

The other objective is to highlight the gradual shift in the philosophy of endodontics from being chemo-mechanically centered to a more biologically centered and biocompatible approach. This approach, coupled with a better appreciation of the microbial dynamics and complex root canal variations, has made endodontic prognosis more predictable.

In this edition, we have included three new chapters: Chapter 18, Prosthodontic Considerations of Endodontically Treated Teeth; Chapter 19, Lasers in Endodontics; and Chapter 20, Procedural Errors and Their Management. This edition contains over 1100 new figures, radiographs, and illustrations, many of which are contributions from clinicians and academicians from across the world. We have tried to keep the spirit of Grossman alive by retaining most of the line illustrations which were the hallmark of the earlier editions.

In the previous edition, Grossman stated "there are concerns that endodontics does not become more technologic than biologic ..." Rest assured, the future of endodontics would be a combination of technological advancement in instruments and techniques for diagnosis, cleaning, shaping, and obturation of the pulp space. At the same time, this would go hand in hand with the development of more biomimetic and biocompatible materials like MTA, which should herald a new era of Endodontic Practice.

B. Suresh Chandra • V. Gopi Krishna

From Prefaces to Previous Editions

Ninth Edition

Endodontic Practice has now been translated into six languages—one of the translations “pirated”—which in itself speaks well for the reception the book has been accorded. Through the years the author has tried to keep abreast of the steady progress made in the field of endodontics, and has revised the book accordingly. In this ninth edition the chapter on pulp capping has been omitted because it rightly belongs in a text on operative dentistry rather than endodontics, and the extensive section on antibiotics has been reduced because the dental profession is now well acquainted with these agents and their use in endodontic practice. A new chapter, Endodontic Emergencies, should serve well the harried general dentist or endodontist, and many new illustrations have been included to supplement the text. Moreover, current developments and reference sources have been inserted in every chapter.

In retrospect, endodontic practice has come far in its development since the first edition (called *Root Canal Therapy*) was published in 1940. The dental and medical professions had to shed the focal infection theory before root canal treatment could be accepted as an effective and safe procedure. In the early development of endodontic practice, treatment was focused first on antiseptics, then on asepsis, later on debridement, and finally on obturation of the canal. Today it is generally agreed that each of these is an important link in the chain of treatment.

Current endodontic practice encompasses a much broader field than did root canal treatment of the past. In addition to being a recognized part of dental practice in its own right, it plays a special role in helping to retain teeth that are periodontally involved, teeth that are used for fixed or removable partial denture abutments, and even teeth used for support of overdentures.

Louis I. Grossman
Philadelphia, Pennsylvania

Tenth Edition

When one looks in retrospect at the forty years since the first edition of this book appeared, there is much to be grateful for. Endodontics is given equal recognition with other branches of dentistry today, both as a part of everyday general practice and as a specialty. It was not always so. Biologic studies during the last two or three decades and clinical observation have contributed much to our knowledge and understanding of pulpal disease and endodontic practice. On this biologic foundation modern endodontics has been developed. However, there is still much to be learned, and much that is already known needs to be applied clinically. In this era of fast communication, fast transportation and even fast foods, one has the impression that short-cuts are being taken at times in endodontic practice, with less than successful results.

Throughout a lifetime of teaching, I have always stressed prevention of pulpal damage. The modes of damage and the means of prevention are succinctly outlined in the chapter on diseases of the pulp. However, despite our best efforts, at times the pulp does become involved and treatment is necessary. In this regard, the chapter on emergency endodontic treatment should be helpful. In this book, an attempt has been made to describe every phase of endodontic therapy and surgery as comprehensively and as clearly as possible. I hope the reader, whether student or practitioner, will find it so.

Louis I. Grossman
Philadelphia

Eleventh Edition

In a previous edition, I stated, "It is expected that the next great advance in endodontics will be in obturation of the root canal." During the last decade there has been a proliferation of both filling materials and methods of filling the root canal. This has given dentists a greater choice of materials and methods for obturating root canals. Their success in sealing the canal will depend upon their intelligent use of the materials and the care and time taken to obturate the canal. They must be concerned, however, that endodontics does not become more technologic than biologic, and the individual is still the key to good endodontic practice.

Louis I. Grossman
Philadelphia

Preface to First Edition

THE last several years have witnessed a gradual return to the practice of root canal therapy. The dental profession has slowly come to realize that extraction is not the solution to the root canal problem. The physician, too, has learned that extraction is not the solution he had hoped it might be to those baffling cases in which oral focal infection was suspected. And the public has awakened to the realization that dental substitutes do not always function as well as natural teeth.

We have not yet arrived at that stage in dental education of the public when preventive dentistry is an accomplished fact. Despite our sincerest efforts to practise preventive dentistry, exposure or death of the pulp will nevertheless occur from decay under a filling, from trauma, from accidental exposure during cavity preparation, etc. The need for intelligent practice of root canal therapy is therefore evident if one is to do the right thing, rather than the most expedient.

Recent studies have given support to clinical observation that non-vital teeth, in selected cases and with careful treatment, may be retained with safety to the patient. The reader will find a brief account of these studies in the chapter on Pulpless Teeth and Focal Infection.

It is the hope of the author that root canal work will be practised with greater care and thoroughness than in the past, and that this book, by acting as a guide, may have a share in pointing the way to more successful root canal therapy. In preparing its text the author has kept in mind the necessity for a practical treatise, but basic principles have also been discussed in order to establish fundamentals of practice. Methods of conserving the pulp after exposure and of treating non-vital teeth are presented. Careful selection of

root canal cases has been stressed, an aseptic technic has been emphasized, and bacteriologic control has been urged. The importance of treating pulpless teeth as an integral part of the body, not as a separate entity, has also been emphasized. For the aim of root canal therapy should be to retain the tooth not only with comfort, but also with safety to the patient. And, if we are to avoid the criticism of the past, root canal work must be done more adequately than in the past.

It is hoped the dentist who is already practising root canal work will find in this book sufficient aids and “pointers” to make it worth his perusal; the dentist who is returning to root canal work, or who practises it only occasionally, will find in it much food for thought as well as a guide for everyday practice; the student will find the text sufficiently adequate to give him a good understanding of the root canal problem, yet practical enough to apply in the college clinic, or later in his office.

The author wishes to acknowledge his indebtedness to Dr. J. L. T. Appleton for the privilege of pursuing research studies in his laboratory, for constant help and advice, and for reviewing three of the chapters; to Dr. Hermann Prinz for early training and guidance; to Dr. H. R. Churchill for reviewing two of the chapters and for the use of several photomicrographs; to Dr. Elsie Gerlach for contributing the chapter on Root Canal Therapy in Deciduous Teeth; to his wife, without whose help, both material and spiritual, this book would probably not have been written; and to that large number of dentists who, by their laboratory research or clinical studies have enriched our knowledge of the science and practice of the art of root canal therapy. He desires also to thank Mr. Leon E. Lewis for correcting the manuscript and reading proof, and Mr. W. D. Wilcox of Lea & Febiger for his coöperation in seeing the book through press.

Louis I. Grossman
Philadelphia, PA

Acknowledgments

This, twelfth, edition, after nearly 23 years of the previous edition, continues the work and legacy of Dr. L.I. Grossman, the legendary endodontist from the University of Pennsylvania. I have toiled for several months along with Dr. Gopi Krishna to bring out this updated edition. I would like this edition to be my tribute to Thai Universal mother god almighty, my spiritual guru Sri Swami Narendranath Kotekar, and Amma Shakuntala Kotekar for all their blessings, inspiration, and guidance for this project.

I am grateful to my teachers as well as my beloved students and colleagues for all that they have taught me. They have truly made me what I am today. A special note of gratitude to Sir Prof. A. Parameshwaran and his beloved wife Mrs. Seetha Parameshwaran who have continued to be everything for me in my professional and personal life. In Dr. Parameshwaran, I have always found a teacher *par excellence* and a friend, philosopher, and guide. Thanks to Mr. A.J. Shetty and Mr. Prashanth Shetty, President and Vice President, respectively, A.J. Institute of Dental Sciences, Mangalore, for all their encouragement.

I would like to express my gratitude to my coeditor Dr. Gopi Krishna for all his efforts, dedication, and perseverance.

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My special thanks to my wife Suryakanthi, daughter Sowmya, and son Shravan for their wonderful patience and support throughout the development of this prestigious project.

B. Suresh Chandra

Thank you are two little words which would probably never completely convey the sense of gratitude and regards which I feel for each of the following wonderful people who have made *Grossmans Endodontic Practice*, – twelfth edition, a reality.

First and foremost, my sincere gratitude to Dr. Suresh Chandra, P. Sangeetha, and Rajiv Banerji for inviting me to be a part of this monumental work. At the same time, I would not have been part of this book but for the kind permission granted by Dr. Anil Kohli, President, DCI, and Ritu Sharma, Reed Elsevier.

I would like to take this opportunity to thank each one of my teachers who have helped in my growth as an endodontist. My *pranams* to my *Gurus* Dr. A. Parameswaran, Dr. B. Suresh Chandra, Dr. E. Munirathnam Naidu, Dr. D. Kandaswamy, and Dr. L. Lakshmi Narayanan.

I would like to specially thank two people who have been instrumental in my growth as an academician and as a clinician: *James “Jim” Gutmann*, for being a perennial source of inspiration, motivation, and support in my academic endeavors; *Dr. Vijailakshmi Acharya*, for motivating me to give the very best to our patients and inspiring me to be a quality conscious clinician.

The true soul of this edition has been the numerous images and clinical contributions by eminent researchers and clinicians from across the world. I thank each one for accepting my invitation to contribute and for their kindness and generosity in sharing their knowledge and expertise.

I would like to compliment the wonderful team at Wolters Kluwer Health for showing genuine passion and professionalism in giving life and body to this edition. Thank you Rajiv Banerji, P. Sangeetha, Eti Dinesh, Munish Khanna, and Honey Pal for your support. Many thanks to Dr. Harini Swaminathan for her meticulous editing of the manuscript.

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V. Gopi Krishna



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The Dental Pulp and Periradicular Tissues

The beginning of all things are small....

PART 1: EMBRYOLOGY

The pulp and dentin are different components of a tooth which remain closely integrated, both functionally and anatomically, throughout the life of the tooth. The two tissues are referred to as the pulp–dentin organ or the pulp–dentin complex.

Development of the Dental Lamina and Dental Papilla

The dental pulp has its genesis at about the sixth week of the intrauterine life, during the initiation of tooth development (Fig. 1.1). The oral stratified squamous epithelium covers the primordia of the future maxillary and mandibular processes in a horseshoe-shaped pattern.

Formation of Dental Lamina

Tooth development starts when stratified squamous epithelium begins to thicken and forms the dental lamina. The cuboidal basal layer of the dental lamina begins to multiply and to thicken in five specific areas in each quadrant of the jaw to mark the position of the future primary teeth.

Formation of Ectomesenchyme

The stratified squamous oral epithelium covers an embryonic connective tissue that is called the

ectomesenchyme because of its derivation from the neural crest cells. By a complex interaction with the epithelium, this ectomesenchyme initiates and controls the development of the dental structures. The ectomesenchyme below the thickened epithelial areas proliferates and begins to form a capillary network to support further nutrient activity of the ectomesenchyme–epithelium complex. This condensed area of ectomesenchyme forms the future dental papilla and subsequently the pulp (Figs. 1.2 and 1.3).

Bud Stage (Formation of Enamel Organ)

The thickened epithelial areas continue to proliferate and to migrate into the ectomesenchyme and in the process forms a bud enlargement called the enamel organ. This point is considered the bud stage of tooth development (Fig. 1.4).

Cap Stage (Outer and Inner Enamel Epithelium)

The enamel organ continues to proliferate into the ectomesenchyme with an uneven rhythmic cell division producing a convex and a concave surface characteristic of the cap stage of tooth development (Fig. 1.5).

The convex surface consists of the cuboidal epithelial cells and is called the outer enamel epithelium. The concave surface, called the inner enamel epithelium, consists of elongated epithelial cells with polarized nuclei that later differentiate

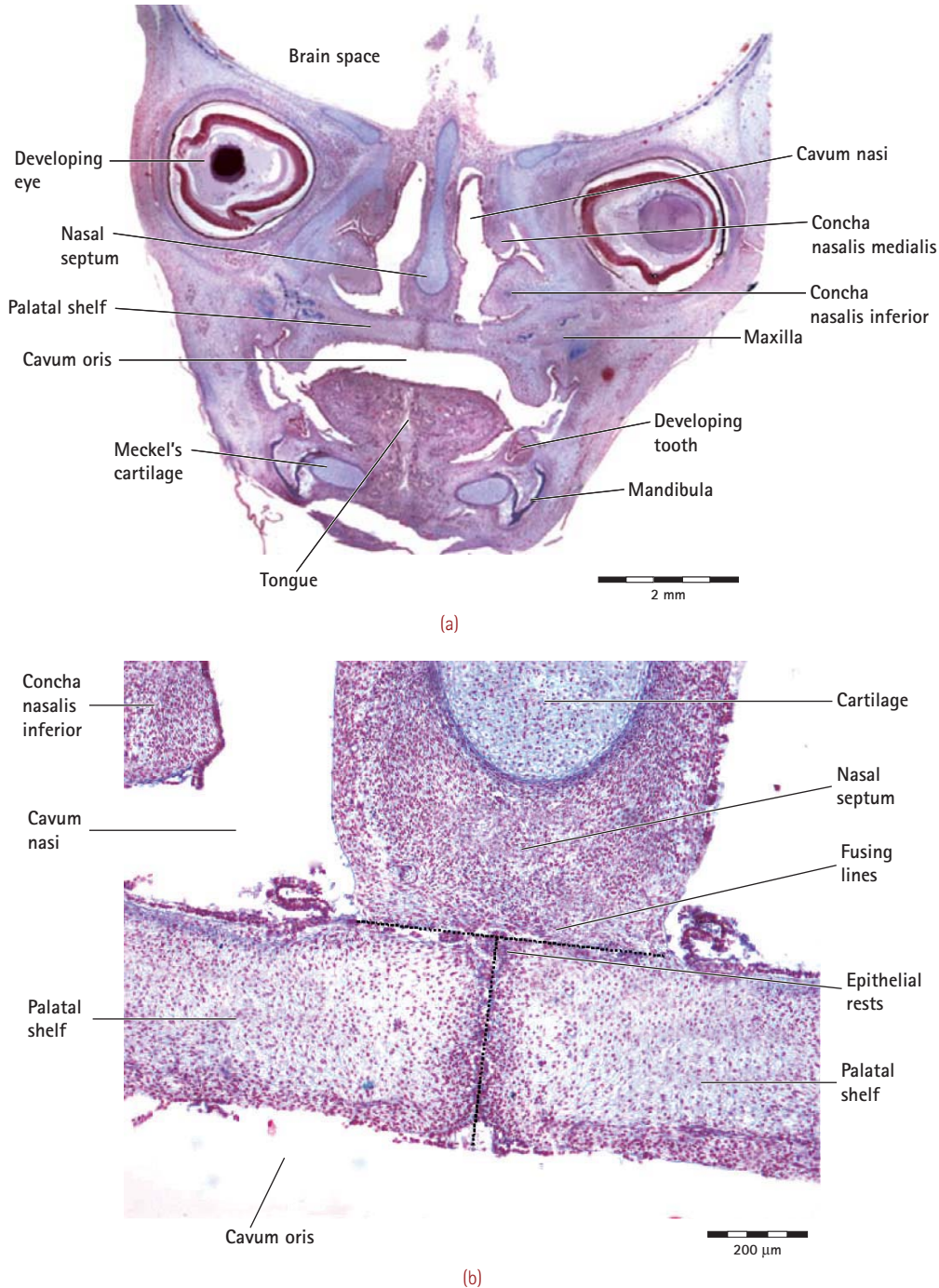


Fig. 1.1 (a) Human fetus, head. This is a frontal section of the head of a human fetus. You can see the maxilla and the mandible taking shape. You can also see Meckel's cartilage in the mandible. The mandible also contains two dental buds in this section (stain: Azan). (b) At higher magnification, you can see the fusing lines between the nasal septum and the palatal shelf. If something goes wrong during this process, the fetus may develop a cleft palate (stain: Azan). (Courtesy: Mathias Nordvi, University of Oslo, Norway.)

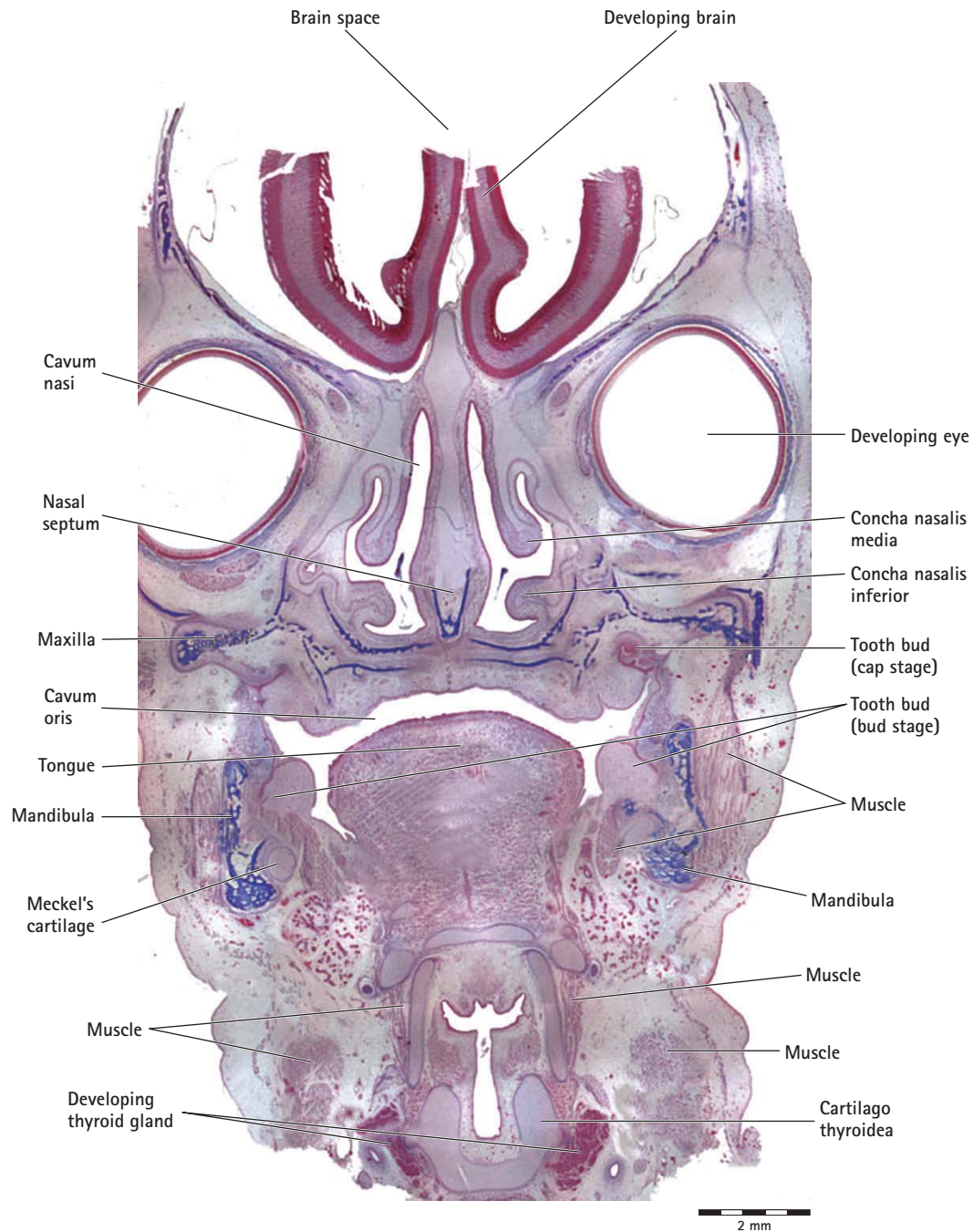


Fig. 1.2 Human fetus, head. This is a frontal section of the head of a human fetus. The nasal cavity (Latin *cavum nasi*) is divided into two by the nasal cartilage within the nasal septum. At both sides of the septum, you can see the nasal conchae (Latin *concha nasalis media et inferior*). They are made up of cartilage at this stage of development. The palate and the maxilla also contain a few spicules of bone. (Courtesy: Mathias Nordvi, University of Oslo, Norway.)

Microbiology

That it will never come again is what makes life so sweet.

—Emily Dickinson

Microorganisms virtually cause all the pathoses of the pulpal and periapical tissues. Endodontic infection is the infection of the root canal system and is the major etiologic factor of apical periodontitis. The root canal infection usually develops after pulpal necrosis, which can occur as a sequel of caries, trauma, and periodontal diseases or operative procedures.

The role of microbiology in endodontic practice, although clearly important, has remained controversial through most of the twentieth century. Onderdenk suggested the need for bacteriologic examination of the root canal in 1901. Shortly thereafter, in 1910, Hunter made his historic address in Montreal, in which he condemned the “golden traps of sepsis,” the ill-fitting crowns and bridgework of his day that inexplicably resulted in the extraction of countless numbers of treated pulpless teeth and the inception of the “focal infection theory.”

Within 25 years, nearly 2000 papers on focal infection were published, many concerned with oral focal infection. During this period, a few voices were raised to stem the hysterical tide and to return endodontic care to its proper role in the healing arts. La Roche and Coolidge suggested that bacteriologic examination be used in treating the root canal. Histologic studies of repair were reported by Blayney in 1932, Coolidge in 1931, Kronfeld in 1939, Aisenberg in 1931, Hatton and associates in 1928, Orban in 1932, Gottlieb and colleagues in 1928, and others.

Another study was published in 1936 by Fish and MacLean, who demonstrated that the pulp and periapical tissues of vital healthy teeth are invariably free of the evidence of microorganisms when examined histologically. In 1935, Okell and Elliot reported a transient bacteremia following extraction; Appleton suggested that without bacteria no need would exist for endodontic treatment, a hypothesis supported by the classic study of Kakehashi and colleagues, who reported that exposed pulps in gnotobiotic rats healed without treatment in a germ-free environment.

In the last few decades, many reports have been published on the bacterial flora of the pulp and periapical and periodontal tissues, the pathways of infection, the immunologic reactions, and the inflammatory responses. Although treatment procedures have changed radically, they reflect a better understanding of the host–parasite relationship and of the way in which such reactions are managed more effectively.

Bacterial Pathways into the Pulp

Bacteria enter the pulp in various ways:

- Through dentinal tubules following carious invasion
- Through crown or root following traumatic exposure of the pulp
- Coronal leakage following restorative procedures and restorations

TABLE 2.3 *Microorganisms Detected in Root-Filled Teeth Associated with Persistent Apical Peridontitis*

Taxonomy

Enterococcus faecalis
Pseudoramibacter alactolyticus
Propionibacterium propionicum
Filifactor alocis
Dialister pneumosintes
Streptococcus spp.
T. forsythia
Dialister invisus
Campylobacter rectus
P. gingivalis
Treponema denticola
Fusobacterium nucleatum
P. intermedia
Candida albicans

antimicrobial procedures. These microbes endure periods of nutrient deprivation in a prepared canal. However, fewer species are present than primary infections. Higher frequencies

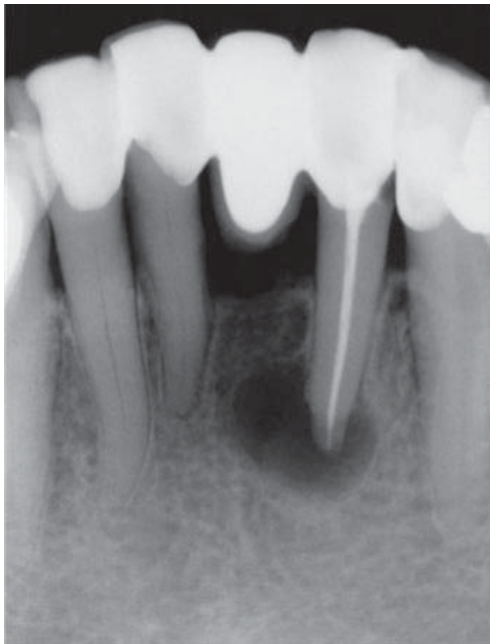


Fig. 2.2 Radiographic appearance of a secondary intraradicular infection in a root-filled tooth.



Fig. 2.3 *Enterococcus faecalis*.

of fungi are present than in primary infections. Gram-positive facultative bacteria, particularly, *E. faecalis* (Fig. 2.3), are predominant in such cases.

E. faecalis is a persistent organism that, despite making up a small proportion of the flora in untreated canals, plays a major role in the etiology of persistent periradicular lesions after root canal treatment. It is commonly found in a high percentage of root canal failures and is able to survive in the root canal as a single organism or as a major component of the flora. *E. faecalis* is also more commonly associated with asymptomatic cases than with symptomatic ones. Although *E. faecalis* possesses several virulence factors, its ability to cause periradicular disease stems from its ability to survive the effects of root canal treatment and persist as a pathogen in the root canals and dentinal tubules of teeth (Table 2.4).

Persistent and secondary infections are clinically indistinguishable and are responsible for persistent exudation, persistent symptoms, interappointment exacerbations, and failure of endodontic treatment characterized by persistent apical periodontitis.

TABLE 2.4 *Survival and Virulence Factors of E. faecalis*

- Endures prolonged periods of nutritional deprivation
- Binds to dentin and proficiently invades dentinal tubules
- Alters host responses
- Suppresses the action of lymphocytes
- Possesses lytic enzymes, cytotoxin, aggregation substance, pheromones, and lipoteichoic acid
- Utilizes serum as a nutritional source
- Resists intracanal medicaments (i.e., $\text{Ca}(\text{OH})_2$)
 - Maintains pH homeostasis
 - Properties of dentin lessen the effect of calcium hydroxide
- Competes with other cells
- Forms a biofilm

Extraradicular Infections

Microbial invasion of the inflamed periradicular tissue is invariably a sequel of intraradicular infection. Acute alveolar abscess is an example of extraradicular extension or a sequel to intraradicular infection (Fig. 2.4).

Sometimes extraradicular infection can be independent of intraradicular infection. For example,

apical actinomycosis caused by *Actinomyces* sp. and *P. propionicum* is a pathological disease which can be treated only by periapical surgery. Other pathogens implicated in such infections are as follows:

- *Treponema* spp.
- *T. forsythia*
- *P. endodontalis*
- *P. gingivalis*
- *F. nucleatum*

Biofilms (Fig. 2.5)

Biofilm is defined as a community of microcolonies of microorganisms in an aqueous solution that is surrounded by a matrix made of glycocalyx, which also attaches the bacterial cells to a solid substratum. A biofilm is one of the basic survival methods employed by bacteria in times of starvation. According to Caldwell et al., a biofilm has the following attributes:

- *Autopoiesis*. Ability to self-organize
- *Homeostasis*. Ability to resist environmental disturbances
- *Synergy*. Effective in association with fellow microorganisms than in isolation

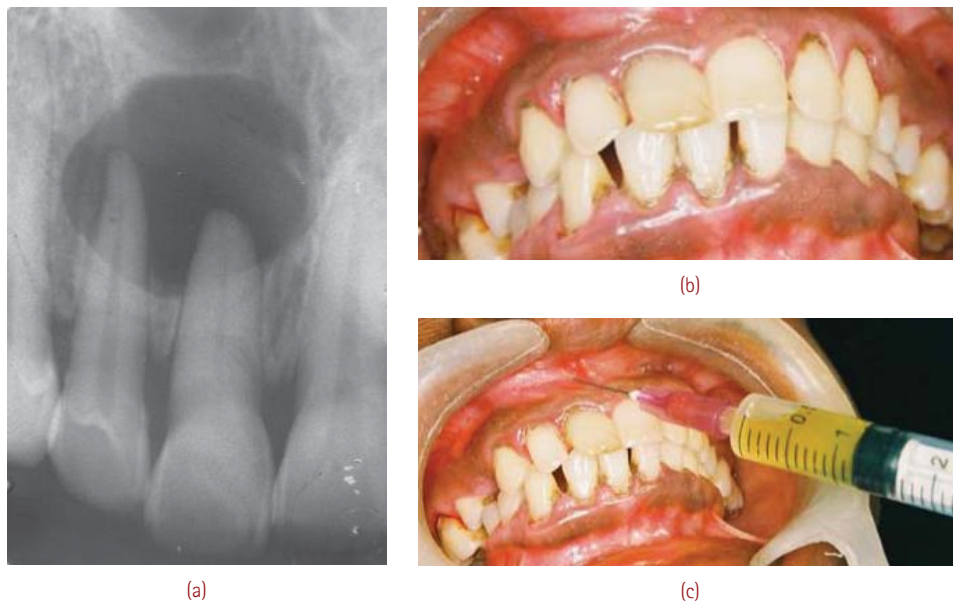


Fig. 2.4 Radiographic appearance, clinical view, and aspiration of serous exudate from an extraradicular infection. (Courtesy: S. Karthiga Kannan, India.)

Clinical Diagnostic Methods

Listen to your patient The patient will give you the diagnosis.

—Sir William Osler

Diagnosis is the correct determination, discriminative estimation, and logical appraisal of conditions found during examination as evidenced by distinctive signs, marks, and symptoms. Diagnosis is also defined as the art of distinguishing one disease from another. Correct treatment begins with a correct diagnosis. Arriving at a correct diagnosis requires knowledge, skill, and art: knowledge of the diseases and their symptoms, skill to apply proper test procedures, and the art of synthesizing impressions, facts, and experience into understanding.

Diagnostic procedures should follow a consistent, logical order which includes comprehensive medical and dental history, radiographic examination, extraoral and intraoral clinical examination including histopathological examination to arrive at the final diagnosis when required.

The process begins with the initial call requesting an appointment for some specific reason, usually a complaint of pain. Subjective information is supplied by the written history or questionnaire that each patient completes and signs. Further information is obtained by the clinician, who reviews the questionnaire and asks specific questions regarding the patient's chief complaint, past medical history, past dental history, and present medical and dental status. The clinician should not hesitate to consult the patient's physician whenever the patient appears

to be medically compromised or when the gained information is inadequate or unclear. More often than not, a patient's medical problem affects the course of treatment, especially concerning the use of anesthetics, antibiotics, and analgesics. Occasionally, the patient's medical status bears a direct relation to the clinical diagnosis. For example, diffuse pain in the mandibular left molars may be a referred pain caused by angina pectoris, or bizarre symptoms may be the result of psychogenic or neurologic disorders.

History and Record

Case history is defined as the data concerning an individual and his or her family and environment, including the individual medical history that may be useful in analyzing and diagnosing his or her case or for instructional purposes. Because many diseases have similar symptoms, the clinician must be astute in determining the correct diagnosis. Differential diagnosis is the most common procedure. This technique distinguishes one disease from several other similar disorders by identifying their differences. Diagnosis by exclusion, on the other hand, eliminates all possible diseases under consideration until one remaining disease correctly explains the patient's symptoms. Although proper clinical diagnosis may appear to be simple, it can tax the most experienced clinician.



(a)

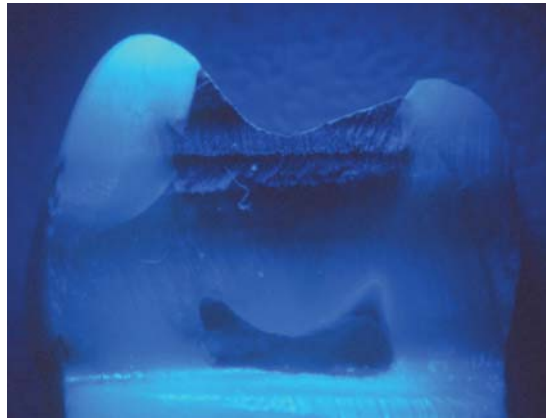


(b)

Fig. 3.5 (a) Translucency and appearance of a normal vital tooth. (b) Loss of translucency and change of color in a nonvital tooth.



Fig. 3.6 Tooth discoloration due to old amalgam filling.



(a)



(b)

Fig. 3.7 (a) Section of a tooth with cracked tooth syndrome after extraction due to poor periodontal prognosis. A clearly visible incomplete fracture, at a distance from the pulp. (b) Same tooth sectioned from the other approximate surface. Another crack visible, close to the dental pulp. (Courtesy: Niek Opdam, Radboud University, Netherlands.)

Abou-Rass recommends endodontic treatment and crown restoration once a cracked tooth develops symptoms because tooth cracks can become tooth fractures (Figs. 3.7a and 3.7b).

The consistency of the hard tissue relates to the presence of caries and internal or external resorption. Obviously, an exposed pulp will require some kind of treatment if the tooth is to be retained. Therefore, pulp exposure, initially recognized on the radiograph, should be confirmed by exploration and excavation.

The technique of visual and tactile examination is simple. One uses one's eyes, fingers, an explorer, and the periodontal probe. The patient's teeth and periodontium should be examined in good light under dry conditions. For example, a sinus tract (fistula) might escape detection if it is covered by saliva or an interproximal cavity may escape notice if it is filled with food.

Loss of translucency, slight color changes, and cracks may not be apparent in poor light. In fact, a transilluminator may aid in detecting enamel cracks or crown fractures.

Visual examination should include the soft tissue adjacent to the involved tooth, for detection of swelling. The periodontal probe should be used routinely to determine the periodontal status of the suspected tooth and adjacent teeth (Fig. 3.8). Sinus tracts opening into the gingival crevice or deep infrabony pockets may go undetected because of failure to use the periodontal probe.

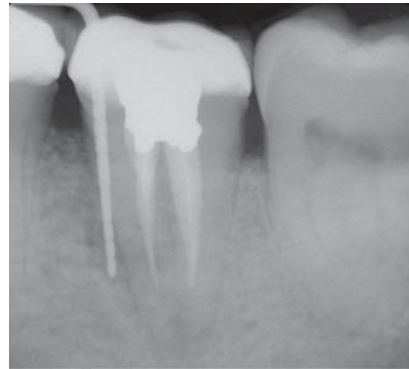
Periodontal pocket probing depths must be measured and recorded. A significant pocket in the absence of periodontal disease may indicate root fracture (Fig. 3.9). Poor periodontal prognosis may be a contraindication to root canal therapy.

Glickman's classification for furcation defects is as follows:

- **Grade I.** Incipient lesion when the pocket is suprabony involving soft tissue and there is slight bone loss.



Fig. 3.8 Periodontal probing to ascertain the health of the periodontium.



(a)



(b)

Fig. 3.9 (a) Deep periodontal probing on tooth #37 indicates that a vertical fracture is present. (b) A 45-year-old female patient presented with pain to biting on mandibular first molar. There was a history of root canal treatment and extensive restorative procedures. Radiographically, the patient has a J-shaped lesion on the mesial root. Probing confirmed the presence of a fracture. (Courtesy: James L. Gutmann, United States of America.)

- **Grade II.** Bone is destroyed on one or more aspects of the furcation but probe can only penetrate partially into the furcation.
- **Grade III.** Intraradicular bone is completely absent but the tissue covers the furcation.
- **Grade IV.** Through and through furcation defect.

The crown of the tooth should be carefully evaluated to determine whether it can be restored properly after the completion of endodontic treatment. Finally, a rapid survey of the entire mouth should be made to ascertain whether the tooth requiring treatment is a strategic tooth.

CHAPTER

4

Diseases of the Dental Pulp

For there was never yet a philosopher who could endure the toothache patiently.

—William Shakespeare, *Much Ado About Nothing*, Act V

The pulp is the formative organ of the tooth. It builds primary dentin during the development of the tooth, secondary dentin after tooth eruption, and reparative dentin in response to stimulation as long as the odontoblasts remain intact (Figs. 4.1a and 4.1b). The pulp responds to hot and cold stimuli which are perceived only as pain. Heat at temperatures between 60°F (16°C) and 130°F (55°C) when applied directly to an intact tooth surface is usually well tolerated by the pulp, but foodstuffs and beverages above and below this temperature range can also be endured. Cavity preparation also produces temperature changes, with an increase of 20°C in temperature during dry cavity preparation 1 mm from the pulp and a 30°C increase 0.5 mm from the pulp. A theoretical model has shown that the sensory reaction to thermal stimulation is registered before a temperature change occurs at the pulpodentinal junction, where the nerve endings are located. The sensation of pain, a warning signal that the pulp is endangered, is a protective reaction, as it is elsewhere in the body.

The pulp has been described as a highly resistant organ and as an organ with little resistance or recuperating ability. Its resistance depends on cellular

activity, nutritional supply, age, and other metabolic and physiologic parameters. This variability has led to the remark that “*some pulps will die if you look crossly at them, while others can’t be killed with an axe.*” On the whole, the resistance of the pulp to injury is slight in certain cases, but evidence of unusual persistence of vitality following injury has also been observed.

The desirability of maintaining a vital pulp and protecting it from injury was recognized by the earliest practitioners of dentistry. The value of the pulp as an integral part of the tooth, both anatomic and functional, should be recognized and every effort made to conserve it.

Accurate pulpal diagnosis is the key to all endodontic treatments. Unfortunately, there has been poor correlation between the clinical symptoms and histopathology of the pulp. Previous attempts made to diagnose the condition of the pulp based on either clinical signs (or symptoms), electric pulp test, and/or thermal tests and radiography have not always been successful. The endodontist is expected to understand the various causative factors of pulpal diseases, collect information about the presentation and history of symptoms, and conduct many practical tests before formulating the final diagnosis.

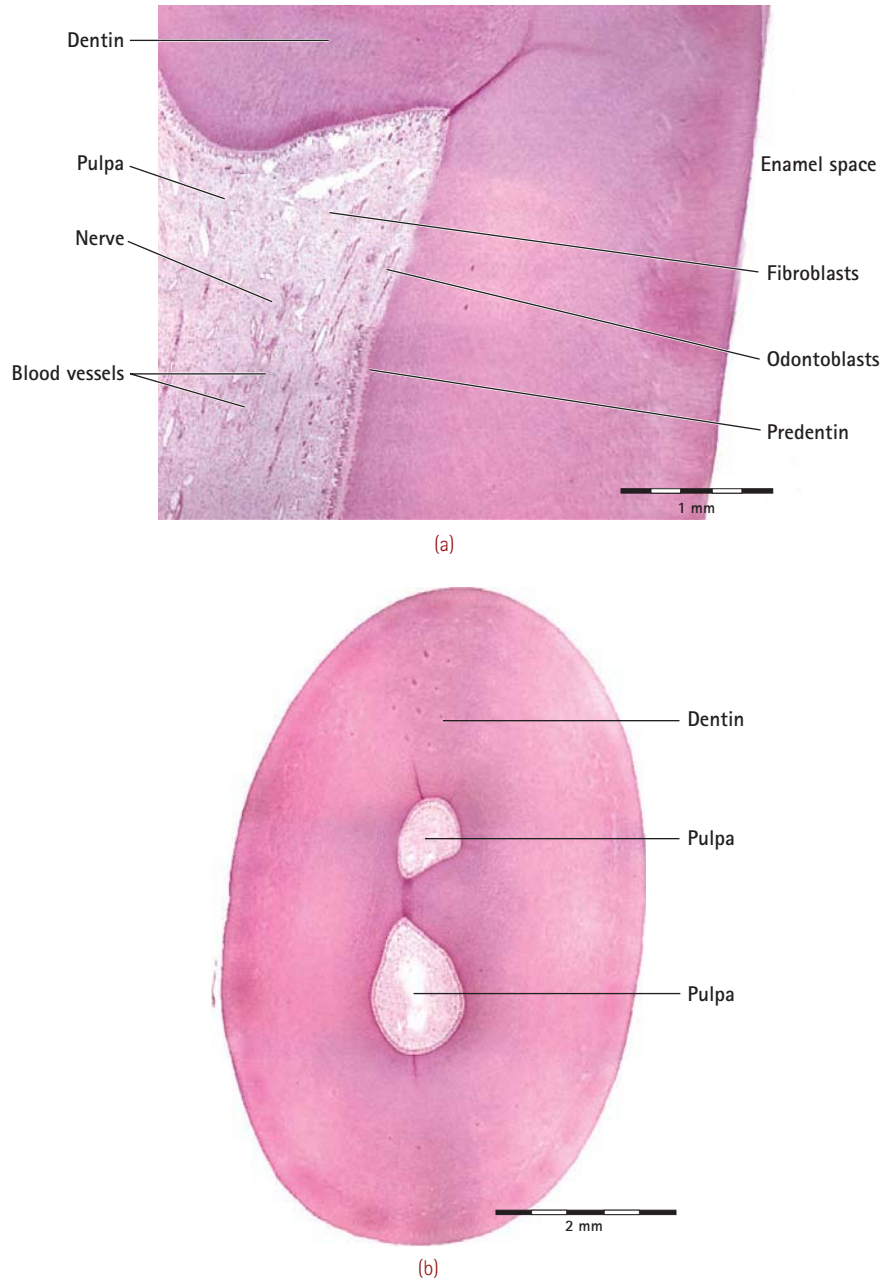


Fig. 4.1 (a) Demineralized tooth, longitudinal section: this is a section through a premolar. The pulp (Latin *pulpa*) comprises loose connective tissue, blood vessels, and nerves (stain: H + E). (b) Demineralized tooth, cross-section: this is a section through the coronal part of a premolar. The enamel has been lost during the preparation of the section. The pulp (Latin *pulpa*) is visible at the center of the tooth (stain: H + E). (Courtesy: Mathias Nordvi, University of Oslo, Norway.)

Diseases of the Periradicular Tissues

Life tells you nothing ... it shows you everything.

—Richard Bach

Pulpal disease is only one of several possible causes of diseases of the periradicular tissues. Because of the inter-relationship between the pulp and the periradicular tissues, pulpal inflammation causes inflammatory changes in the periodontal ligament even before the pulp becomes totally necrotic. Bacteria and their toxins, immunologic agents, tissue debris, and products of tissue necrosis from the pulp reach the periradicular area through the various foramina of the root canals and give rise to inflammatory and immunologic reactions. Neoplastic disorders, periodontal conditions, developmental factors, and trauma can also cause periradicular diseases.

The diseases of periradicular tissues can be classified on the basis of the etiology, symptoms, and histopathological features. The WHO has classified diseases of periradicular tissues into various categories (Table 5.1).

Periradicular diseases of pulpal origin may also be classified as acute or chronic periradicular diseases based on symptoms, etiology, and histopathology (Table 5.2).

Acute Periradicular Diseases

These disorders include acute apical periodontitis, acute alveolar abscess, and acute exacerbation of a chronic lesion.

TABLE 5.1 *WHO Classification of Periradicular Diseases*

Code Number	Category
K04.4	Acute apical periodontitis
K04.5	Chronic apical periodontitis (apical granuloma)
K04.6	Periapical abscess with sinus (dentoalveolar abscess with sinus, periodontal abscess of pulpal origin)
K04.60	Periapical abscess with sinus to maxillary antrum
K04.61	Periapical abscess with sinus to nasal cavity
K04.62	Periapical abscess with sinus to oral cavity
K04.63	Periapical abscess with sinus to skin
K04.7	Periapical abscess without sinus (dental abscess without sinus, dentoalveolar abscess without sinus, periodontal abscess of pulpal origin without sinus)
K04.8	Radicular cyst (apical periodontal cyst, periapical cyst)
K04.80	Apical and lateral cyst
K04.81	Residual cyst
K04.82	Inflammatory periodontal cyst

may be present. The root surface may show external root resorption due to cementoclastic activity or hypercementosis due to cementoblast activity.

Treatment

Root canal therapy may suffice for the treatment of a chronic apical periodontitis. Removal of the cause of inflammation is usually followed by resorption of the granulomatous tissue and repair with trabeculated bone (Fig. 5.13).

Prognosis

The prognosis for long-term retention of the tooth is excellent.

Chronic Alveolar Abscess

A chronic alveolar abscess is a long-standing, low-grade infection of the periradicular alveolar bone characterized by the presence of an abscess draining through a sinus tract.

Synonym

Chronic suppurative apical periodontitis.

Causes

The source of the infection is in the root canal. Chronic alveolar abscess is a natural sequela of death of the pulp with extension of the infective

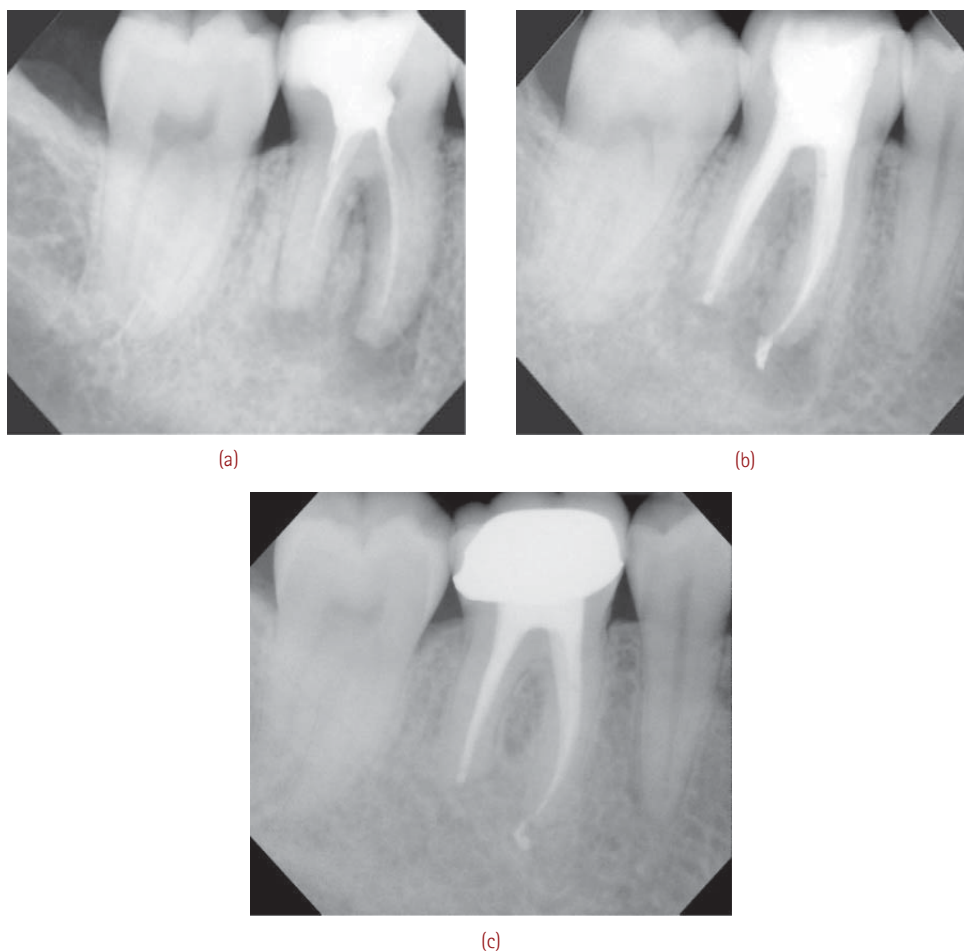


Fig. 5.13 (a) Chronic apical periodontitis in an asymptomatic poorly obturated mandibular molar. (b) Radiographic view after completion of retreatment and obturation of the root canals. (c) Two-year follow-up shows complete resolution of the periradicular pathology. (Courtesy: Julian Webber, England.)

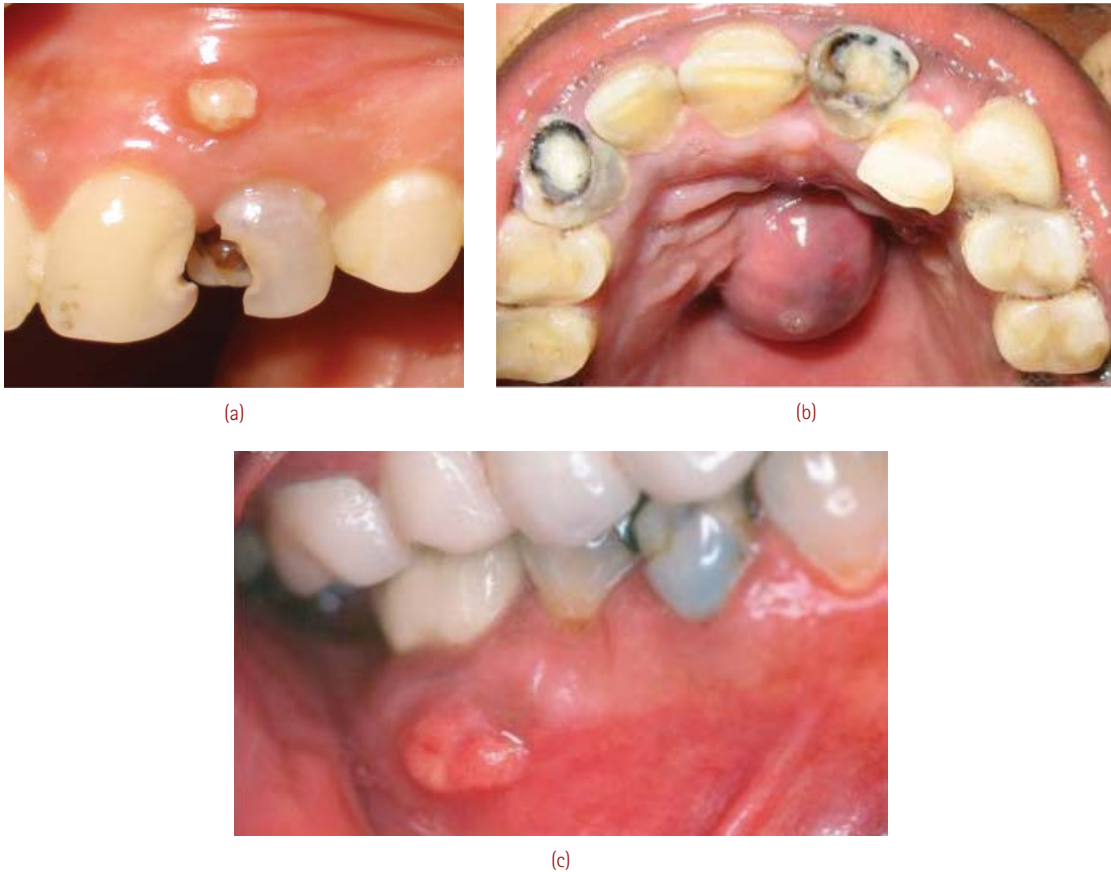


Fig. 5.14 (a) Intraoral labial sinus opening in relation to the carious maxillary lateral incisor. (b) Intraoral palatal sinus opening in relation to the carious maxillary central incisor. (c) Intraoral buccal sinus opening in relation to the carious mandibular premolar.

process periapically, or it may result from a pre-existing acute abscess.

Symptoms

A tooth with a chronic alveolar abscess is generally asymptomatic, or only mildly painful. At times, such an abscess is detected only during routine radiographic examination or because of the presence of a sinus tract, which can be either intraoral (Figs. 5.14a–5.14c) or extraoral (Fig. 5.15). The sinus tract usually prevents exacerbation or swelling by providing continual drainage of the periradicular lesion.

Diagnosis

The first sign of osseous breakdown is radiographic evidence seen during routine examination or



Fig. 5.15 Extraoral sinus opening. (Courtesy: Karthiga Kannan, India.)

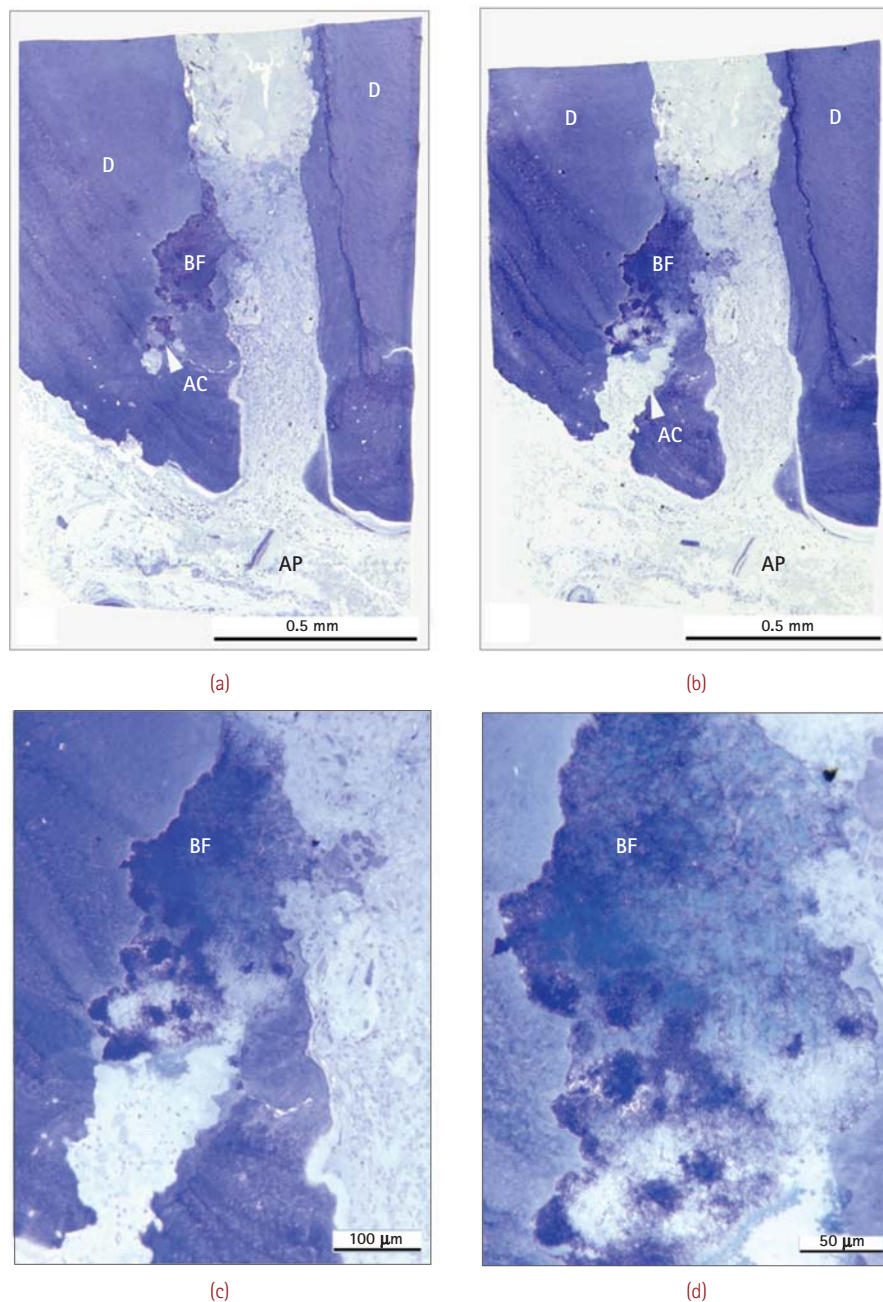


Fig. 5.26 Photomicrographs of axial semithin sections through the surgically removed apical portion of the root with a persistent apical periodontitis. Note the adhesive biofilm (BF) in the root canal. Consecutive sections [(a)–(b)] reveal the emerging widened profile of an accessory canal (AC) that is clogged with the biofilm. The AC and the biofilm are magnified in (c) and (d), respectively. Magnifications: (a) $\times 75$, (b) $\times 70$, (c) $\times 110$, and (d) $\times 300$. (Adapted from Nair, P.N.R., et al.: Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: A long-term light and electron microscopic follow-up study. *J. Endod.*, **16**:580–88, 1990.)

Rationale of Endodontic Treatment

CHAPTER

6

When the only tool you own is a hammer, every problem begins to resemble a nail.

—Abraham Maslow

Injury to the calcified structure of teeth and to the supporting tissues by noxious stimuli may cause changes in the pulp and the periradicular tissues. Noxious stimuli can be physical, chemical, or bacterial. They can produce changes that are either reversible or irreversible, depending on duration, intensity, and pathogenicity of the stimulus and the host's ability to resist the stimulus and to repair tissue damage.

On the basis of these premises, we can generalize that mild-to-moderate noxious stimuli to the pulp may produce sclerosis of the dentinal tubules, formation of reparative dentin, or reversible inflammation. Irreversible inflammatory changes caused by severe injury can lead to necrosis of the pulp and subsequent pathologic changes in the periradicular tissues.

The inflammatory response of the connective tissue of the dental pulp is modified because of its milieu. Because the pulp is encased in hard tissues with limited portals of entry, it is an organ of terminal and limited circulation with no efficient collateral circulation and with limited space to expand during the inflammatory reaction. A clear concept of the fundamentals of inflammation is necessary for the understanding of the diseases of the pulp and their extension to the periradicular tissues.

Inflammation

Inflammation is the local physiologic reaction of the body to noxious stimuli or irritants. Any irritant, whether of traumatic, chemical, or bacterial origin, produces a sequence of basic physiologic and morphologic reactions in vascular, lymphatic, and connective tissues. Host-resistance factors and intensity, duration, and virulence of the irritant modify the ultimate character, extent, and severity of the tissue changes and, to some degree, the clinical manifestations.

The objective of inflammation is to remove or destroy the irritant and to repair damage to the tissue. Inflammation brings to the area phagocytic cells to digest bacteria or cellular debris, antibodies to recognize, attack, and destroy foreign matter, edema or fluid to dilute and neutralize the irritant, and fibrin to limit the spread of inflammation.

Repair of the tissues depends on the severity of injury and host resistance. The injurious agent may cause reversible or irreversible changes to the tissues. Irreversible damage leads to tissue necrosis, whereas reversible damage leads to repair. Repair, or the return of the tissue to normal structure and function, begins as the tissue becomes involved in the inflammatory process. Removal of

Zone of Stimulation

This zone is characterized by fibroblasts and osteoblasts. At the periphery, Fish noted that the toxin was mild enough to be a stimulant. In response to this stimulation, collagen fibers were laid down by the fibroblasts, which acted both as a wall of defense around the zone of irritation and as a scaffolding on which the osteoblasts built new bone. This new bone was built in irregular fashion.

By analogy, we can apply the knowledge gained in Fish's experiment to understand better the reaction of the periradicular tissues to a pulpless tooth. The root canal is the site of infection (Fig. 6.3). The microorganisms in the root canal are rarely motile and do not move from the root canal to the periradicular tissues; however, they can multiply sufficiently to grow out of the root canal, or the metabolic products of these microorganisms or the toxic products of tissue necrosis

may be diffused to the periradicular tissues. As the microorganisms gain access to the periradicular area, they are destroyed by the polymorphonuclear leukocytes. When the microorganisms are sufficiently virulent, or when enough are present, they overwhelm the defensive mechanism, and a *periradicular lesion* results. When they are of low virulence and numbers, however, a stalemate occurs. The polymorphonuclear leukocytes destroy the microorganisms as rapidly as they gain access to the periradicular tissues. The result is a *chronic abscess*. The toxic products of the microorganisms and the necrotic pulp in the root canal are irritating and destructive to the periradicular tissue and, together with the proteolytic enzymes released by the dead polymorphonuclear leukocytes, help to produce pus.

At the periphery of the destroyed area of osseous tissue, toxic bacterial products may be diluted

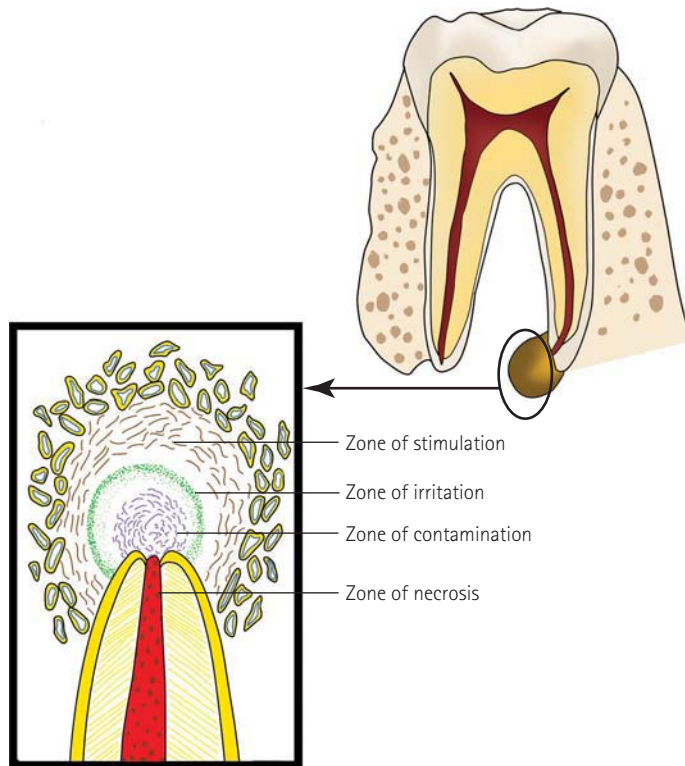


Fig. 6.3 Schematic diagram showing bacteria in the root canal and the zones of infection, contamination, irritation, and stimulation.

Selection of Cases for Treatment

Education is a progressive discovery of our own ignorance.

—Will Durant

Proper selection of cases avoids pitfalls during endodontic treatment and helps to ensure success. Not every tooth is suitable for endodontic treatment. Errors in case selection, some of which could have been avoided, constituted 22% of failures reported in a study by Ingle and Beveridge.

Many more root canals are treated today than before because of a greater interest in endodontics by the practitioner not only to save endodontically involved teeth, but also to use them as abutments for bridges or partial dentures. Unfortunately, a general practitioner's best effort may not be good enough because of a mistaken diagnosis, such as a curved canal not capable of being instrumented to the apex, with a resulting persistence of the area of rarefaction. On the other hand, certain cases formerly contraindicated for endodontic treatment, such as a sinus tract discharging into the gingival sulcus, are treated successfully today because of advances in both endodontic and periodontal therapy. To depend on root canal treatment alone for all endodontic cases is bound to meet with a certain degree of failure. If such treatment is combined with other endodontic procedures, such as apexification with mineral trioxide aggregate (MTA) to induce the formation of a calcific barrier in root canals with open apex or the use of periapical microsurgery to remove the periapical

pathological tissues, the possibilities of it being successful are high. On the other hand, microsurgery is not indicated simply because an area of rarefaction is present.

The selection of cases for endodontic treatment has been discussed by a number of authors, including Bender, Grossman and associates, and Strindberg. Advances in the understanding of endodontics, better techniques, and principles of canal preparation and obturation have led to significantly increased and predictable healing rates for endodontic treatment—95% and higher under ideal conditions according to current literature. However, before selecting a case for endodontic therapy the clinician should consider the following factors that influence the outcome of the treatment:

- Health and systemic status of the patient
- Anatomy of the root canal system
- Extent of previous tooth restoration
- Presence or absence of periradicular pathosis
- Radiographic interpretation
- Degree of difficulty in locating, cleaning, shaping, and obturating the complete root canal system
- Periodontal status of the tooth
- Presence of crown or root fractures

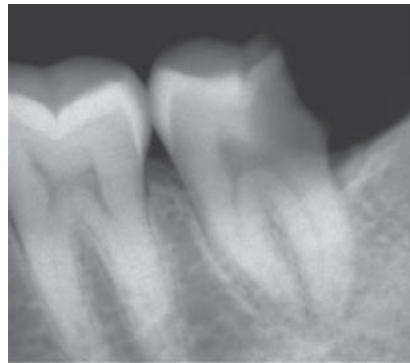
TABLE 7.2 *AHA-Recommended Antibiotic Prophylaxis Regimens for Dental Procedures*

Situation	Agent	Adults	Children
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin	2 g IM or IV	50 mg/kg IM or IV
	or cefazolin or ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
Allergic to penicillin or ampicillin—oral	Cephalexin*†	2 g	50 mg/kg
	or clindamycin	600 mg	20 mg/kg
	or azithromycin or clarithromycin	500 mg	15 mg/kg
Allergic to penicillin or ampicillin and unable to take oral medication	Cefazolin or ceftriaxone†	1g IM or IV	50 mg/kg IM or IV
	or clindamycin	600 mg IM or IV	20 mg/kg IM or IV

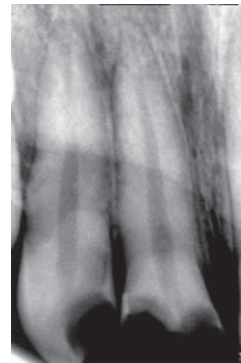
IM, intramuscular; IV, intravenous.

* Or other first or second generation in equivalent adult or pediatric dosage.

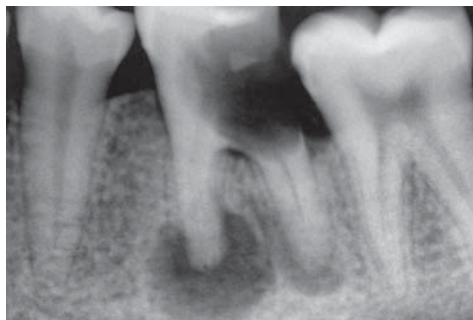
† Cephalosporins should not be used in an individual with a history of anaphylaxis, angioderma, or urticaria with penicillins or ampicillin.



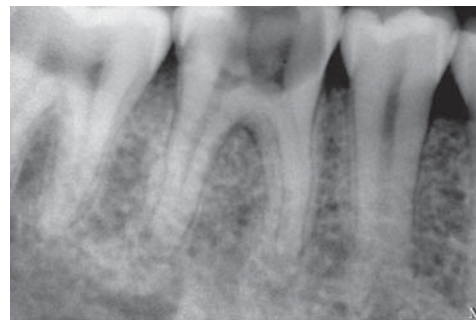
(a)



(b)



(c)



(d)

Fig. 7.1 (a) Distal carious lesion involving the pulp with periradicular changes. (b) Extensive loss of coronal tooth structure due to rapidly advancing caries lesion. (c) Extensive carious destruction of tooth with large periradicular lesion. (d) Carious involvement of the lower molar with progressive calcification of pulp chamber and canals.

Principles of Endodontic Treatment

Quality is, doing the little things right when nobody is looking.

—George Bernard Shaw

The basic principles underlying the treatment of teeth with endodontic problems are those underlying surgery in general. An aseptic technique, debridement of the wound, drainage, and gentle treatment of the tissues with both instruments and drugs—all are cardinal principles of surgery. Specifically, pain must be controlled, if present. During treatment, all pulp tissue must be removed, the root canal enlarged and irrigated, the canal surface rendered sterile as determined by bacteriologic examination, and the root canal well obturated to prevent the possibility of reinfection.

Rubber Dam Isolation

To achieve the first principle of endodontic treatment a safe and aseptic operating technique needs to be maintained. For this, application of rubber dam is imperative. It is the only sure safeguard against bacterial contamination from saliva and accidental swallowing of root canal instruments. All endodontic operations should be performed under the rubber dam. Treatment of any tooth should not be attempted under cotton rolls. The risk of losing a reamer or file down the patient's trachea or esophagus is too great to warrant this practice.

In some cases, it is first necessary to replace a missing wall with a restoration or to cement a stainless steel band to prevent the rubber dam clamp from slipping off the tooth. In other cases, a gingivectomy may need to be done, with removal of about 2-mm gingival tissue to provide enough tooth structure for application of a rubber dam clamp. Gingivectomy may be necessary in any event for restoration of the crown of the tooth.

In a survey of the use of the rubber dam in endodontic treatment by general practitioners, Going and Sawinski found that 36% of dentists never, or seldom, used the rubber dam. Their survey emphasizes the need for more general use of the rubber dam by dentists, especially in view of the more than threefold increase in ingestion or aspiration of instruments during endodontic treatment in the last decade. To risk operating without a rubber dam is to risk one's professional reputation. A variety of objects used in endodontic practice may be swallowed accidentally if the rubber dam is not applied. Root canal instruments swallowed during endodontic treatment has been reported (Fig. 8.1).

If an instrument is swallowed or aspirated during endodontic treatment without a rubber dam, one is likely to be confronted with a lawsuit. Grossman

dam is held in the left hand and is kept from obstructing the view, while the clamp is slipped over the tooth with the right hand. The forceps are then disengaged from the clamp, and the rubber dam is slipped under the anterior arms of the clamp. If a wing clamp is used, the wing of the clamp is inserted into the hole of the rubber dam, the clamp is applied to the tooth, the clamp forceps are removed, and the rubber dam is slipped under the arms of the clamp.

To facilitate slipping the rubber dam over the tooth, especially if the contact point is tight, the surface of the rubber dam adjacent to the hole should be wiped with liquid soap, or a wet finger

may be rubbed on a cake of soap and applied to the rubber dam around the punched hole. Petrolatum or cocoa butter should not be used for this purpose because these substances soften and weaken the rubber dam, and leakage may result.

The following methods are used while applying rubber dam in different teeth in the oral cavity:

- Molar tooth isolation using rubber dam (Figs. 8.7a–8.7f)
- Isolation of multiple teeth—clamp first followed by dam (Figs. 8.8a–8.8i)
- Optradam application (Figs. 8.9a–8.9c)



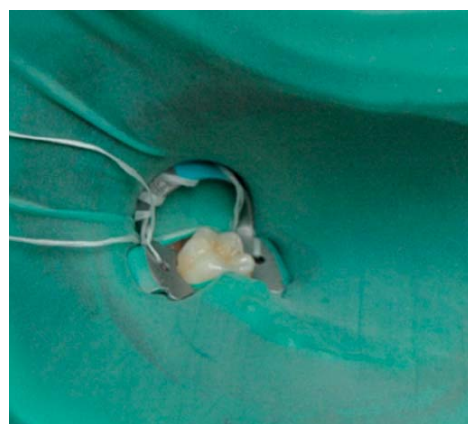
(a)



(b)



(c)



(d)

Fig. 8.7 (a) The rubber dam sheet is placed on the rubber dam template and the tooth to be isolated is marked. Note that the dull side of the dam faces the operator. (b) Hole is punched using a rubber dam punch. (c) The wings of the clamp engage the dam through the punched hole. The entire assembly can be carried to the tooth to be isolated. (d) The dam is secured with the rubber dam frame. Note the dam material is engaged on the wings of the clamp. *(continued)*

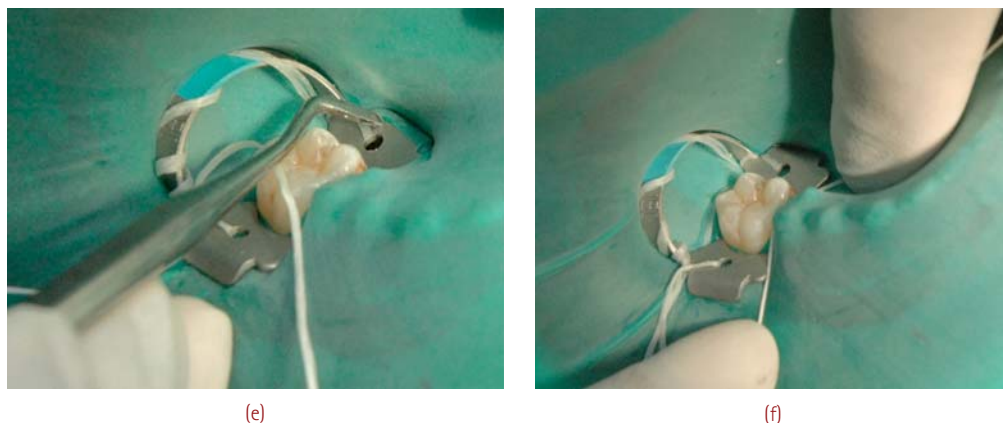


Fig. 8.7 (continued) (e) The dam is gently teased away from the wing of the clamp using a blunt hand instrument. (f) Dental floss is passed mesially and distally to ensure inversion of the dam for isolation.

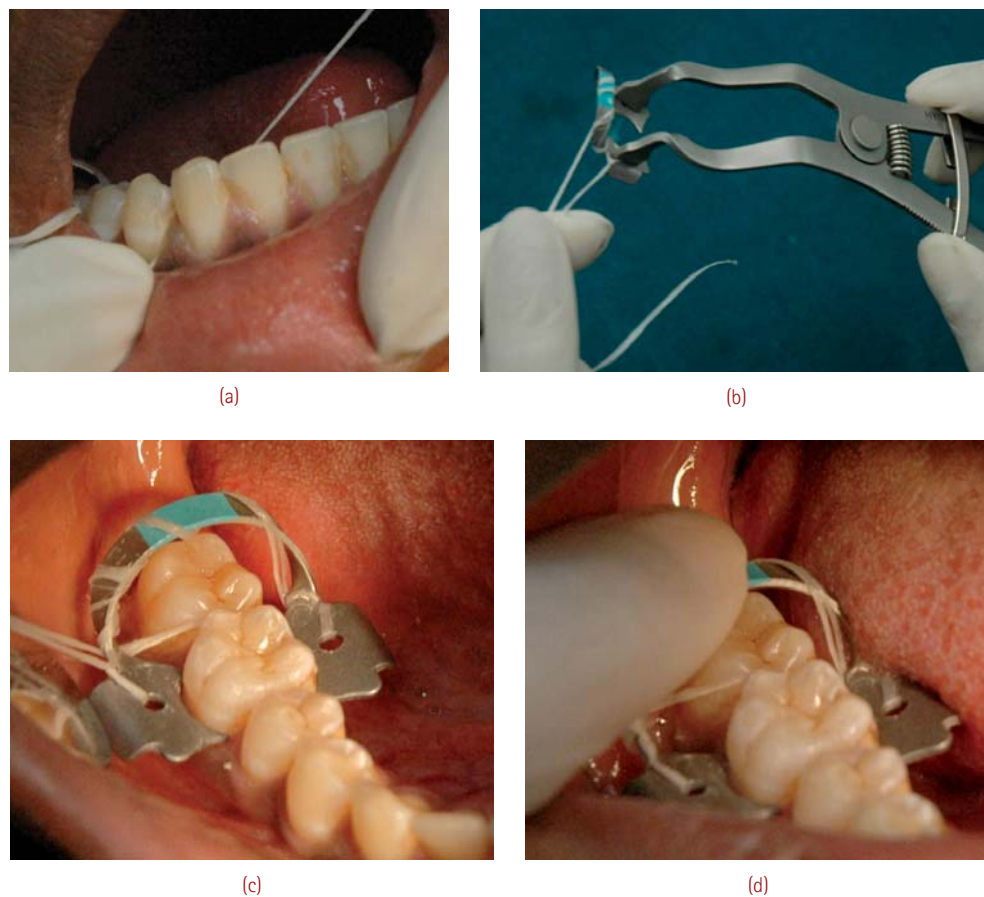


Fig. 8.8 (a) Testing and lubricating the proximal contact. (b) The clamp is engaged with the clamp forceps and the forceps is secured with the lock. (c) The clamp is transferred to the tooth. The jaws of the clamp engage the tooth gingival to the height of contour along the four axial line angles of the tooth. (d) Check the stability of the dam with index finger on the bow of the clamp. The clamp should not rock. (continued)

CHAPTER

9

Anatomy of Pulp Cavity and Its Access Opening

*Of all the phases of anatomic study in the human system,
One of the most complex is the pulp cavity morphology.*

—M.T. Barrett

The journey of a thousand miles begins with a single small step.

—Lao Tzu

The external morphologic features of the crowns of teeth vary according to the shape and size of the head. The length of the crown differs with the size and sex of the person and is generally shorter in females than in males. As the external morphology of the tooth varies from person to person, so does the internal morphology of the crown and root. Changes in pulp cavity anatomy result from age, disease, and trauma. Although morphologic variations occur, clinical experience indicates that these changes usually follow a general pattern, and thus the study of pulp cavity morphology is a feasible undertaking.

Pulp Cavity

The pulp cavity is the central cavity within a tooth and is entirely enclosed by dentin except at the apical foramen (Fig. 9.1). The pulp cavity may be divided into a coronal portion, the *pulp chamber*, and a radicular portion, the *root canal*.

Pulp Chamber

In anterior teeth, the pulp chamber gradually merges into the root canal, and this division becomes indistinct. In multirrooted teeth, the pulp cavity consists of a single pulp chamber and usually three root canals, although the number of canals can vary from one to four or more. The *roof* of the pulp chamber consists of dentin covering the pulp chamber occlusally or incisally (Fig. 9.1).

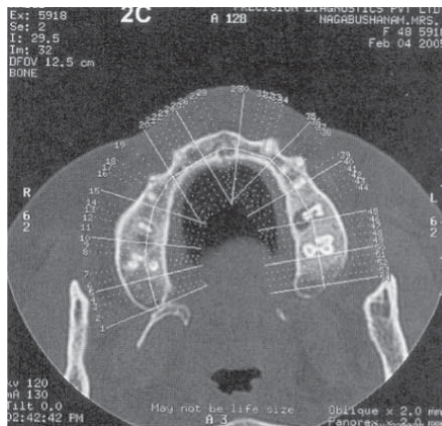
The *pulp horn* is an accentuation of the roof of the pulp chamber directly under a cusp or developmental lobe. The term refers more commonly to the prolongation of the pulp itself directly under a cusp. The *floor* of the pulp chamber runs parallel to the *roof* and consists of dentin bounding the pulp chamber near the cervical area of the tooth, particularly dentin forming the furcation area. The *canal orifices* are openings in the floor of the pulp chamber leading into the root canals. The canal orifices are not separate structures, but are continuous with both the pulp chamber and the root canals. The walls of the pulp chamber derive their names from the corresponding walls of the tooth surface, such



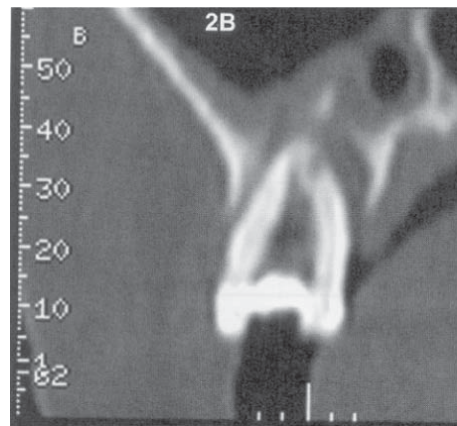
(a)



(b)

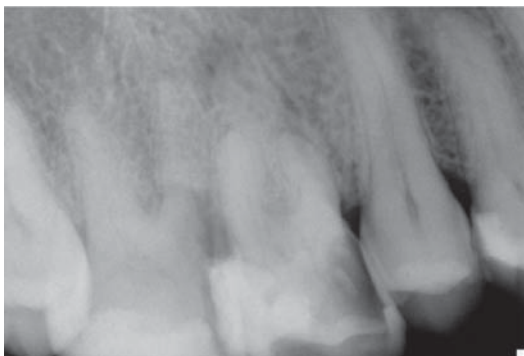


(c)

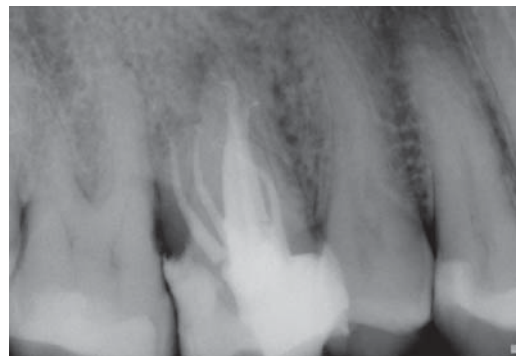


(d)

Fig. 9.22 Maxillary first molar with a single root and a single canal. (Adapted from Gopikrishna, V., Bhargavi, N., and Kandaswamy, D.: Endodontic management of a maxillary first molar with a single root and single canal diagnosed with spiral CT—a case report. *J. Endod.*, **32**:687–91, 2006.)



(a)



(b)

Fig. 9.23 Maxillary first molar with two separate mesiobuccal canals as well as two separate distobuccal canals: (a) Preoperative radiograph indicating complex and unusual root canal anatomy. (b) Postobturation radiograph showing two distinct distal canals. (Courtesy: Sashi Nalapatti, Jamaica.)



Fig. 9.37 Middle mesial canal in mesial root of mandibular first molar.

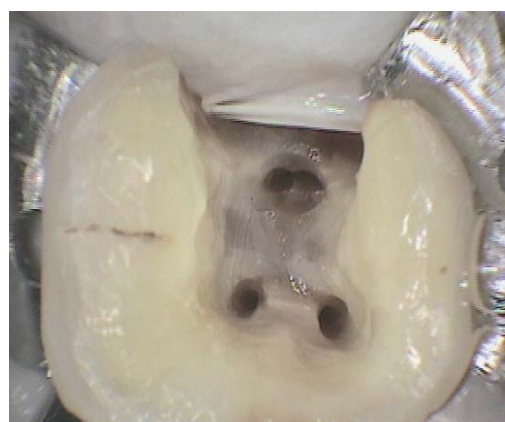
- Two canals that coalesce to exit in one foramen in 28%.
- Two canals that coalesce to form one canal and bifurcate and exit in two foramina in 13%.
- One canal that exits in one foramen in 12%.
- One canal that bifurcates and exits in two foramina in 8%.
- In rare cases, three canals exit in three foramina and this third canal which is present between the mesiobuccal and mesiolingual canal orifices is referred to as the *middle mesial canal* (Fig. 9.37).

Distal Root (Fig. 9.38)

- The distal root has one canal exiting in one foramen in 70% of cases.
- Two canals coalescing and exiting in one foramen in 15%.
- One canal bifurcating and exiting in two foramina in 8%.
- Two canals exiting in two foramina in 5%.
- Two canals coalescing to form one canal and later bifurcating to exit in two foramina in 2% of cases.
- When two canals are present in either root, they may converge towards and exit in one foramen, or they may have interconnecting lateral canals between them that form a single ribbon canal ending in one foramen.



(a)



(b)

Fig. 9.38 Variations in distal root of mandibular first molar: (a) Distal root of a mandibular first molar with one canal and one orifice. (b) Distal root of a mandibular first molar with one canal and two orifices. (*continued*)

In cross-section, all three canals are ovoid in the cervical and middle thirds and round in the apical third. Two canals present in the distal root are usually round in cross-section from the cervical third to the apical third.

Clinical Significance

The mesial root of the mandibular first molar is in close proximity to the buccal cortical plate, whereas the distal root is centrally located. The apex of the roots of mandibular first molars may be close to the mandibular canal, or they may be at some distance

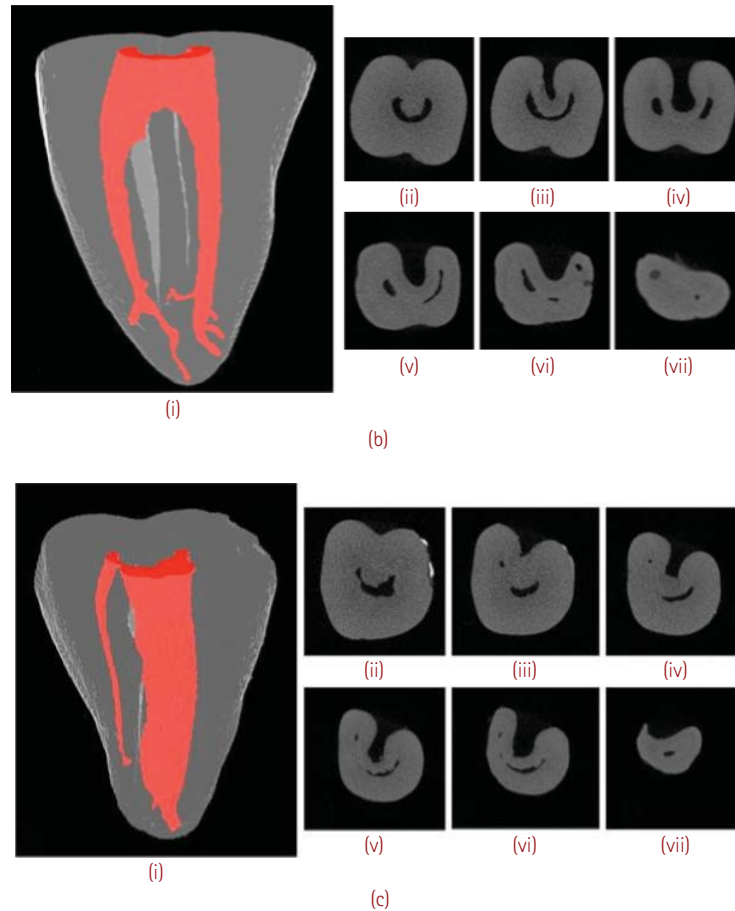


Fig. 9.43 (continued) (b) Type II C-shaped canal system (symmetrical type): (i) 3D reconstructed root canal image; (ii) cross-section 1 mm from apex; (iii) cross-section 3 mm from apex; (iv) cross-section 4 mm from apex; (v) cross-section 8 mm from apex; (vi) cross-section 11 mm from apex; (vii) cross-section 12 mm from apex (orifice). (c) Type III C-shaped canal system (asymmetrical type): (i) 3D reconstructed root canal image; (ii) cross-section 1 mm from apex; (iii) cross-section 3 mm from apex; (iv) cross-section 5 mm from apex; (v) cross-section 7 mm from apex; (vi) cross-section 9 mm from apex; (vii) cross-section 10 mm from apex (orifice). (Courtesy: Prof. Bing Fan, Wuhan University, China.)

Mandibular Third Molar

Average Tooth Length

The average length of this tooth is 18.5 mm.

Pulp Chamber

The pulp chamber of the mandibular third molar anatomically resembles the pulp chamber of the mandibular first and second molars. It is large and possesses many anomalous configurations such as C-shaped root canal orifices.

Roots and Root Canals

The mandibular third molar usually has two roots and two canals, but occasionally one root and one canal or three roots and three canals. The root canals are generally large and short.

Clinical Significance

The alveolar socket of the mandibular third molar may project on to the lingual plate of the mandible. The apex of the root may be in close proximity to the mandibular canal.

Preparation of the Radicular Space: Instruments and Techniques

CHAPTER

10

*What we remove from the pulp space,
is far more important than what we replace it with...*

Endodontic treatment can be divided into three main phases: (i) proper access preparation into the pulp space, (ii) cleaning and shaping of the root canal, and (iii) obturation. The initial step for cleaning and shaping the root canal is proper access to the chamber that leads to straight-line penetration of the root canal orifices. The next step is exploration of the root canal, extirpation of the remaining pulp tissue or gross debridement of the necrotic tissue, and verification of the instrument working length. This step is followed by proper instrumentation, irrigation and debridement, and disinfection/sterilization of the root canal. Obturation usually completes the procedure.

The importance of adequate canal cleaning and shaping, rather than reliance on antiseptics, cannot be overemphasized. Histologic examination of pulpless teeth in which root canal therapy has failed often shows that the canals were not completely cleaned. Obturation of an improperly cleaned canal would still lead to an endodontic failure (Fig. 10.1).

Cleaning and Shaping of Radicular Space

Cleaning and shaping of the root canal comprises the most important phase of endodontic treatment. Other aspects of treatment cannot be neglected, however, because they are all inter-related and contribute to the success of endodontic therapy.

The objectives of cleaning and shaping are two-fold:

1. To debride and disinfect/sterilize the root canal system
2. To shape/contour the root canal walls, for the purpose of sealing the root canal completely with a condensed, inert filling material

To help achieve these objectives, each individual root canal should be examined radiographically and explored with endodontic instruments. The examination should include an assessment of canal length, shape, size, curvature, entrance orifice, location of foramina, canal ramifications, and presence of calcifications or obstructions.

According to Schilder, the cleaning and shaping of a root canal should fulfill the following *mechanical objectives*:

- Should have a continuous tapering, conical shape, with the narrowest cross-sectional diameter apically and the widest diameter coronally.
- The walls should taper evenly towards the apex and should be confluent with the access cavity.
- To give the prepared root canal the “quality of flow,” i.e, a shape that permits plasticized gutta-percha to flow against the walls without impedance
- Should keep the apical foramen as small as practical.

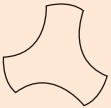
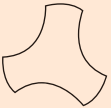
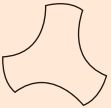
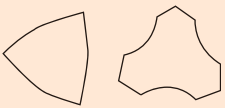
- **Pitch.** Pitch is the distance from one cutting edge to the next cutting edge. A file with short pitch will have more spirals than a file with a longer pitch.
- **Rake angle.** On perpendicular sectioning of a file, the angle which the leading edge forms with the radius of the file is known as the rake angle. If it forms an obtuse angle then the rake angle is considered to be positive. An acute angle is termed negative rake angle.

- **Helix angle.** It is the angle the cutting edge forms with the long axis of the tooth.

Table 10.4 gives an overview and salient features of the most widely used rotary nickel–titanium systems in endodontics (Fig. 10.14).

Motors for Nickel–Titanium Rotary Instrumentation The manufacturer-recommended speeds for the currently available nickel–titanium rotary instruments are in the range of 150–600 RPM with

TABLE 10.4 *Design Features of Current Rotary Ni–Ti File Systems*

Instrument System	Cross-Sectional Design	Tip Design	Taper	Other Features
ProFile (Dentsply Maillefer)	 Triple-U shape with radial lands. Neutral rake angle planes dentin walls	Noncutting	Fixed taper. 2, 4, and 6%	20-degree helix angle and constant pitch
GT Files (Dentsply Maillefer)	 Triple-U shape with radial lands	Noncutting	Fixed taper. 4, 6, 8, 10, and 12%	Files have a short cutting portion. Variable pitch
GT Series X	Variable-width lands (lands at the tip and shank region of the file are narrower than midfile lands)	Noncutting	No 10 or 12% taper	Decreased helical angle, increased pitch. Heat treatment aims to improve cyclic fatigue resistance
LightSpeed Instruments (Lightspeed, San Antonio, TX)	 Triple-U shape with radial lands	Noncutting	Specific instrument sequence produces a tapered shape	Thin, flexible noncutting shaft and short cutting head
ProTaper (Dentsply Maillefer)	 Convex triangular shape, sharp cutting edges, no radial lands. F3, F4, F5 files have U-flutes for increased flexibility	Noncutting	Variable taper along the length of each instrument	Pitch and helix angle balanced to prevent instruments screwing into the canal

(continued)

Irrigants and Intracanal Medicaments

Medicine heals doubts as well as diseases.

–Karl Marx

The cleaning and shaping of the root canal constitutes one of the most important phases of endodontic therapy. Instrumentation of the canal reduces the microbial content of the root canal to a great extent. However, the root canal anatomy provides areas in which bacteria can persist and thrive (Figs. 11.1 and 11.2).

Individual microorganisms proliferate to form populations which occur as microcolonies. The root canal system with necrotic pulp provides space for microbial colonization and affords the microorganism a moist, warm, nutritive,

and anaerobic environment, protected from host defenses due to lack of microcirculation in the necrotic pulp tissue. A wide variety of microorganisms have been identified to invade depths up to 2000 μm into the dentinal tubules (Table 11.1).

It has become increasingly clear that the largest proportion of endodontic diseases of both pulp and periradicular tissues is due to the presence of microorganisms, though mechanical and manual challenges of root canal debridement remain. Therefore, the success of treatment depends upon

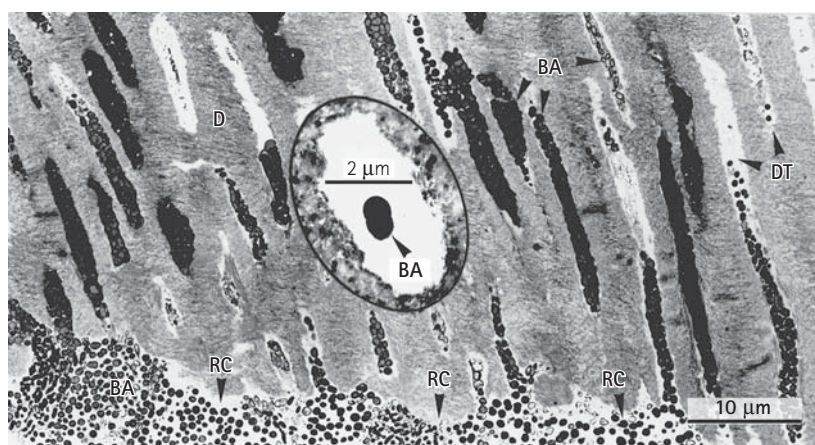


Fig. 11.1 Bacteria (BA) in dentinal tubules (DT) in the apical part of human root dentin (D). The bacteria invade the tubules from the infected root canal (RC). The presence of dividing forms (inset) is a clear sign of vitality of the microorganisms at the time of fixation (magnifications: $\times 2480$; inset $\times 9600$). (Courtesy: P.N.R. Nair, Switzerland.)

MTAD

One of the newly introduced irrigant is MTAD (Fig. 11.10) which employs a mixture of a tetracycline isomer, citric acid, and a detergent (Tween 80) as a final rinse to remove the smear layer. It is commonly employed after initial irrigation with 1.3% NaOCl. The antibacterial effect of MTAD is due to the tetracycline present in high concentration. The combination of NaOCl and MTAD has been advocated to remove the smear layer and also has substantial antimicrobial efficacy.

Apart from the chemical nature of the irrigants, the following factors determine the efficacy of irrigants (Table 11.3):

- Several investigators have shown that unless adequate irrigation is part of the canal cleaning process, debris and microorganisms will be left behind regardless of the irrigant used. Moreover, the clearance of debris from the canal is proportional to the amount of irrigant used.
- Other studies have shown that frequent irrigation of the canal is mandatory, and irrigation is more complete in properly enlarged canals.
- Penetration of the irrigant is more effective in canals with larger diameters than in smaller canal diameters.
- The irrigating solution should be constantly exchanged to maintain its efficiency.
- Increasing the temperature of NaOCl has also been demonstrated to improve its efficacy;

TABLE 11.3	Factors Affecting the Efficacy of an Irrigant
•	Volume of the irrigant used
•	Concentration of the irrigant
•	Frequency of irrigation
•	Temperature of the irrigant
•	Length and time of intracanal contact
•	Gauge of the irrigating needle
•	Depth of penetration
•	Diameter of the prepared canals
•	Age of the irrigating solution

however, temperature might not play a role for other irrigants.

- High-volume passive ultrasonic irrigation systems have been developed. These systems enhance the penetrating and debriding action of the irrigant by passive ultrasonic activation.

Irrigation Guidelines

The technique of irrigation is simple. The only instrument required is a disposable luer lock syringe (Fig. 11.11) with an endodontic blunt-ended side-vented needle (Figs. 11.12a and 11.12b).

The needle is inserted partway into the root canal. It should be passively inserted without binding the needle into the root canal. Sufficient room between needle and canal wall allows for the return



Fig. 11.10 MTAD irrigant (BioPure MTAD). (Courtesy: Dentsply Tulsa.)



Fig. 11.11 Disposable luer lock syringes: (a) 3cc and (b) 12cc.

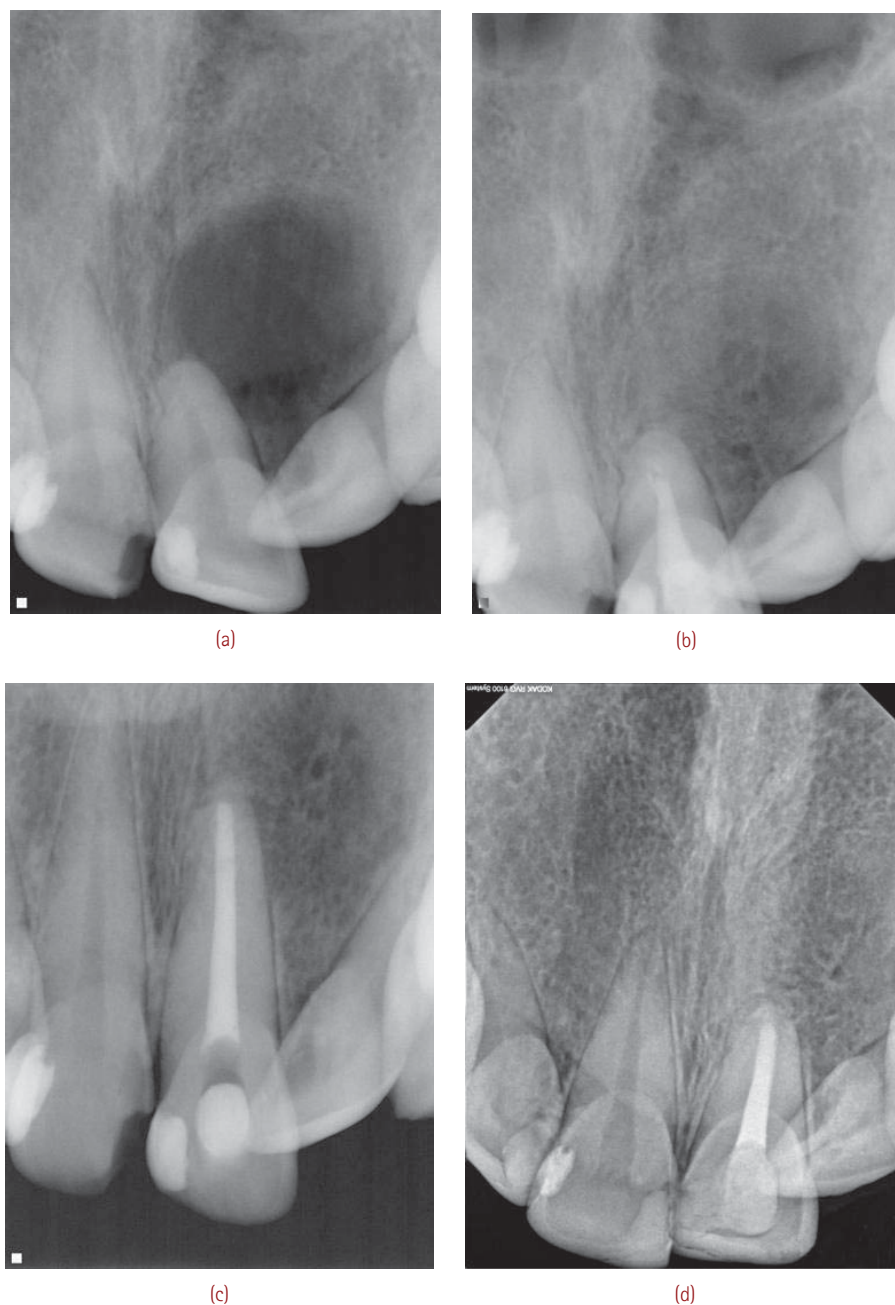


Fig. 11.16 (a) Large periradicular radiolucency associated with a nonvital maxillary central incisor. (b) Calcium hydroxide used as intracanal medicament for a period of 6 months. (c) Obturation was done after radiographic healing had taken place. (d) At 6 months recall tooth was asymptomatic and functional with complete resolution of the lesion. (Courtesy: Sashi Nalapatti, Jamaica.)

CHAPTER

12

Obturation of the Radicular Space

Perfection is not attainable, but if we chase it ... we can reach excellence.

—Vince Lombardi

The function of a root canal filling is to obturate the canal and eliminate all portals of entry between the periodontium and the root canal. The better the seal, the better the prognosis of the tooth. Achieving the ideal seal, however, is as complex as the anatomy of the root canal system itself. Because all root canal fillings must seal all foramina leading into the periodontium, an ideal filling must be well condensed, must conform and adhere to the instrumented canal walls, and must end at the juncture of the root canal and the periodontium.

The objective of obturating the root canal is the substitution of an inert filling in the space previously occupied by the pulp tissue. Naidorf has stated that inadequate obturation of the root canal exposes it to periradicular tissue fluids, which provide material for growth of microorganisms or localization of bacteria in such dead spaces. According to a study made by Ingle and Beveridge, 58% of endodontic failures can be attributed to incomplete obturation of root canals (Fig. 12.1).

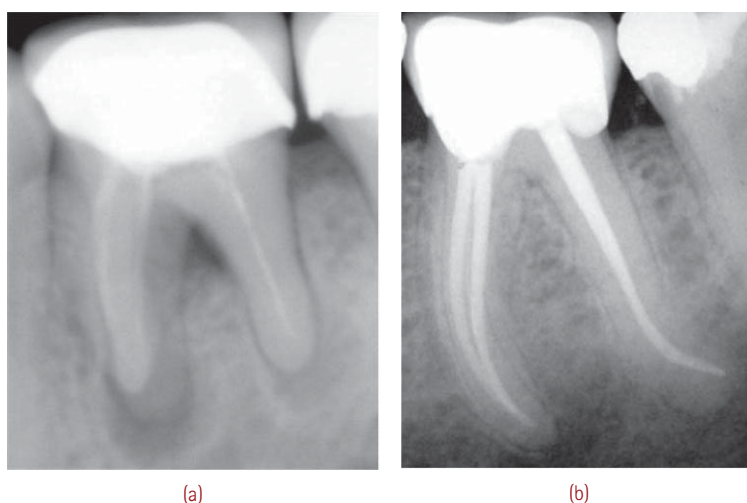


Fig. 12.1 (a) Incomplete obturation of a tooth leading to endodontic failure. (b) Proper cleaning and shaping of the root canals followed by a complete obturation ensures endodontic success.



Fig. 12.12 (a) Touch n Heat heat carrier. (b) System B heat carrier. (Courtesy: Sybron Endo.)

applied to the heated gutta-percha to force the plasticized material apically.

7. This alternate application of heat carrier and condenser is repeated until the plasticized gutta-percha seals the larger accessory canals and fills the lumen of the canal in three dimensions up to the apical foramen. The remaining portion of the canal is plugged with warm sections of additional pieces of gutta-percha.

Lifshitz and colleagues used the scanning electron microscope to determine the effectiveness of the vertical condensation method of sealing root canals in conjunction with a sealer. The investigators found a wall-to-wall adaptation of the gutta-percha in the apical area, as demonstrated by a solid interface among dentin sealer and gutta-percha. In an

in vitro study, Goodman and associates have shown that the maximum regional temperature to which gutta-percha is subjected during the vertical condensation method is 80°C, and the temperature in the apical region is between 40 and 42°C.

Advantages

- Excellent seal of the canal apically and laterally
- Obturation of the larger lateral and accessory canals

Disadvantages

- The amount of time it takes
- The risk of vertical root fracture resulting from undue force

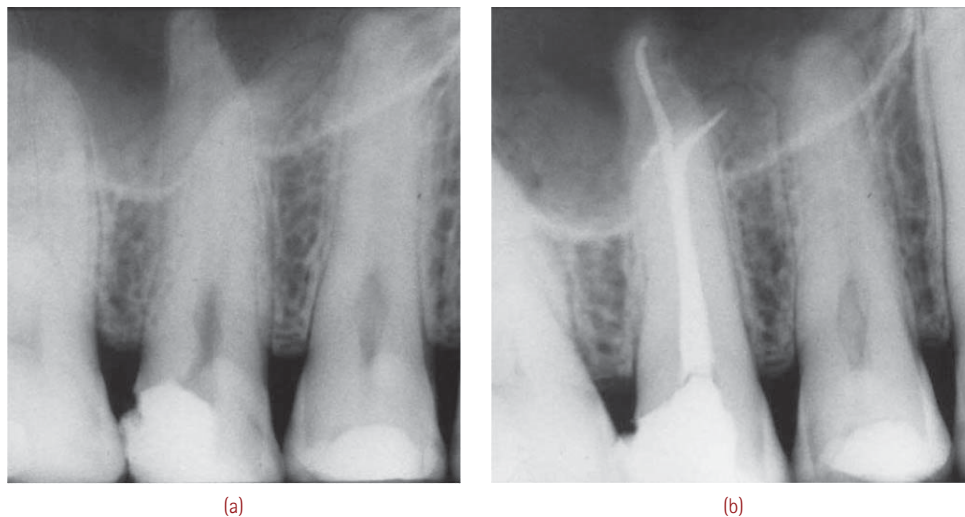


Fig. 12.13 Warm vertical compaction obturation in a maxillary premolar.

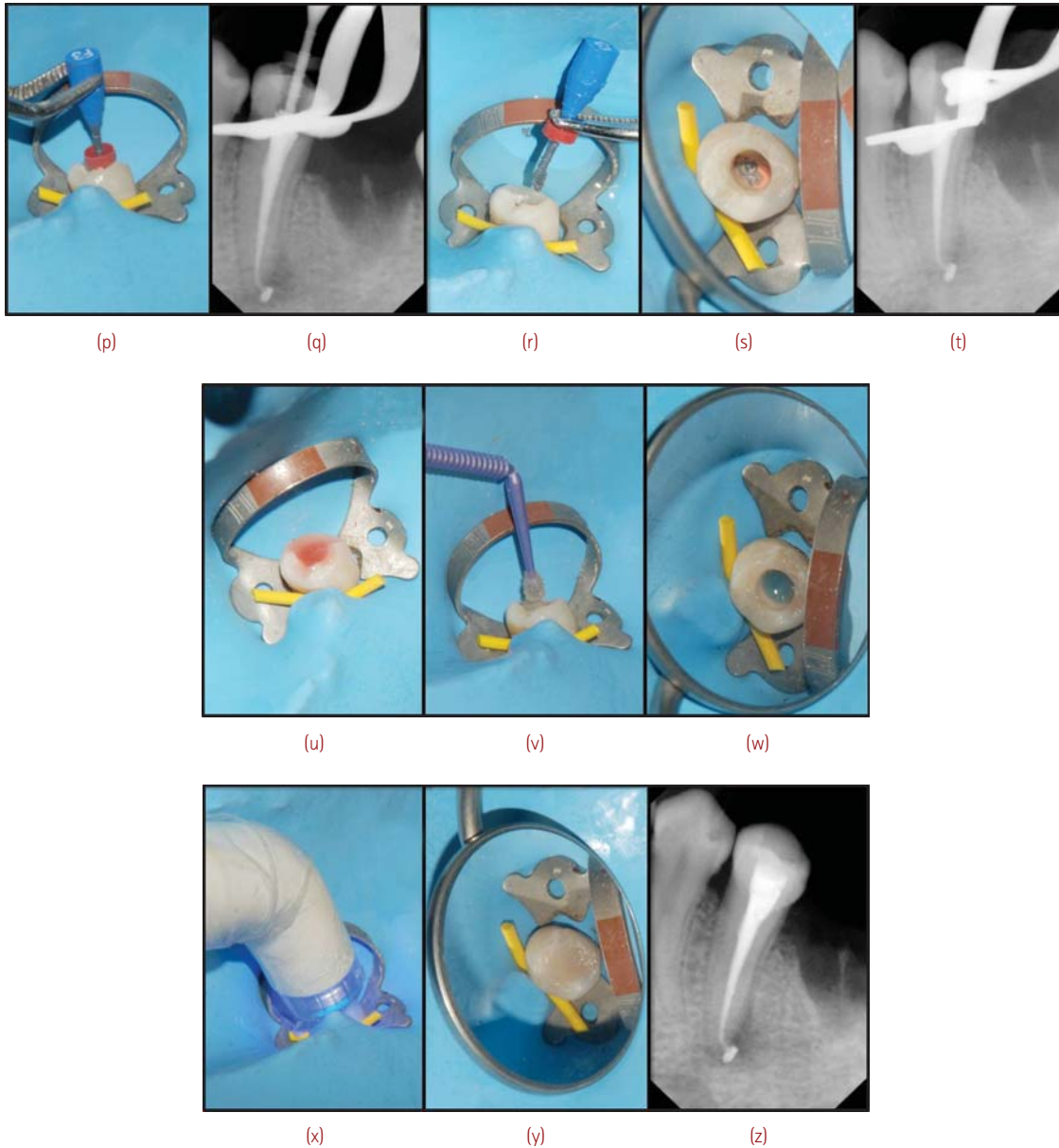


Fig. 12.23 (continued) (p) Therafil carrier placed to working length as verified by stopper placement. (q) Radiograph showing therafil carrier in place. (r) Therafil carrier handle sectioned at orifice level using high-speed carbide bur. (s) Sectioned therafil carrier at orifice level. (t) Radiographic verification of sectioned therafil carrier. (u) 40% phosphoric acid etchant (K-Etchant Gel) placed in coronal access. (v) Etchant washed and dried followed by placement of bonding agent. (w) Blue core composite placed at orifice level. (x) Curing the layers of composite resin. (y) Completed composite coronal seal. (z) Verification of complete obturation with adequate coronal and apical seal. (Courtest: Vivek Hegde and Roheet Khatavkar, India.)

Vital Pulp Therapy, Pulpotomy, and Apexification

Never must the physician say the disease is incurable. By that admission he denies God, our Creator; He doubts Nature with her profuseness of hidden powers and mysteries.

—Paracelsus

The unaffected, exposed vital pulp possesses an inherent capacity for healing through cell reorganization and bridge formation when a proper biological seal is provided and maintained against leakage of oral contaminations. Throughout the life of a tooth, vital pulp tissue contributes to the production of secondary dentin, peritubular dentin, and reparative dentin in response to biological and pathological stimuli. The pulp tissue with its circulation extending into the tubular dentin keeps the dentin moist, which in turn ensures that the dentin maintains its resilience and toughness (Fig. 13.1). The earliest account of vital pulp therapy was in 1756, when Phillip Pfaff packed a small piece of gold over an exposed vital pulp to promote healing.

By 1922, in the light of his experiences with similar antiseptic treatments, Rebel summarized his thoughts in the expression, “the exposed pulp is a doomed organ.” He concluded that recovery of the vital unaffected pulp when exposed to the oral environment was invariably doomed and that one must consider it as a lost organ. Despite Rebel’s much-quoted statements, the realization gradually evolved that the dental pulp did at times possess definite powers of recuperation and repair. Major advances in the practice of vital pulp therapy have

been made and the emphasis has shifted from the “doomed organ” concept of an exposed pulp to one of “predictable repair and recovery.”

Pulpal Inflammation and Its Sequelae

The traditional theory which explained pulpal inflammation and its sequelae was known as strangulation theory. This theory is no longer accepted and a newer current theory explains the sequelae of pulpal inflammation.

Strangulation Theory

In the accepted view on pulpal inflammation, maximum significance was diverted and focused on the influence of anatomical environment on the pulp. The assumption was that with local inflammation of the pulp there is an increased blood flow to the inflamed area. Inflammation induces vasodilatation and increased capillary pressure and permeability. This results in an increased filtration from the capillaries into the tissues leading to increased tissue pressure. The thin vessel walls are compressed as the increasing pressure outside the vessels leads to a decrease in blood flow as well as an increase in venous pressure. This results in a vicious cycle. The increase

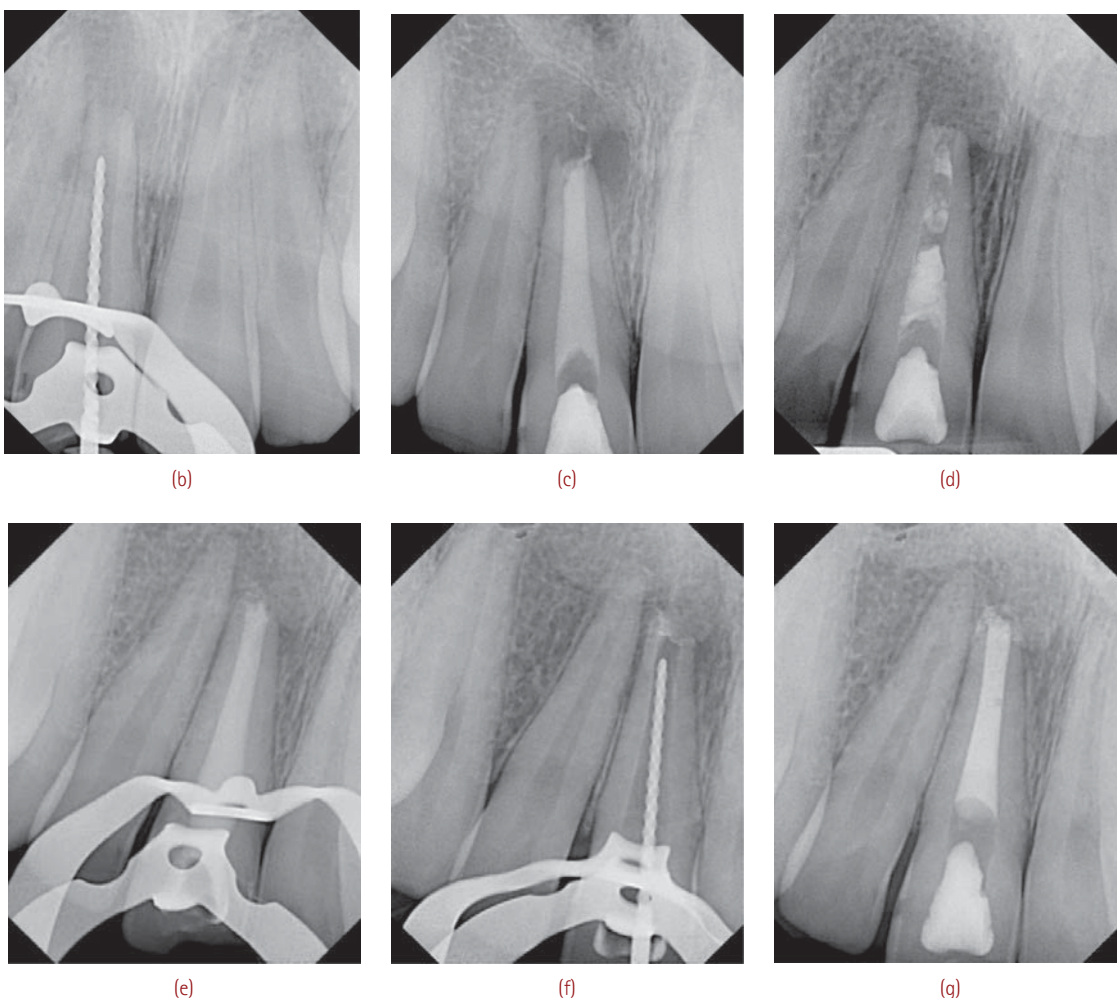


Fig. 13.24 (continued) (b) Tooth debrided 2 mm short of the apex. (c) Ca(OH)_2 placed into the canal up to the apex. (d) Three-month check shows resorption of Ca(OH)_2 , but apex still open. Apical lesion almost completely healed. (e) Ca(OH)_2 placed again. (f) Ten-month re-evaluation. Apical barrier present, so it is time to obturate. This is a great view of the apical barrier that has formed. (g) Obturation completed. (Courtesy: Jason J. Hales, United States of America.)

and root canal, and irrigation is performed with sterile water or saline solution to prevent further irritation to the periradicular tissues due to sodium hypochlorite.

- A file is inserted and the stop is set to the apparent length of the tooth and a radiograph is taken to measure the actual length of the tooth. The measured difference between the file tip and the root tip in the radiograph is used to adjust the apparent length to the actual length.
- The working length should be at least 2 mm short of the length of the tooth to prevent in-

jury to the apical tissues and the thin walls at the apical third of the root.

- Circumferential enlargement is effected by lateral pressure against the walls with a large file. The instrument should follow the natural shape and contour of the root canal, which is usually not entirely round. In some cases, the walls are thin and fragile, so care must be used to prevent a perforation or a fractured wall.
- The purpose of cleaning and shaping is to remove any necrotic pulpal tissue and to pre-

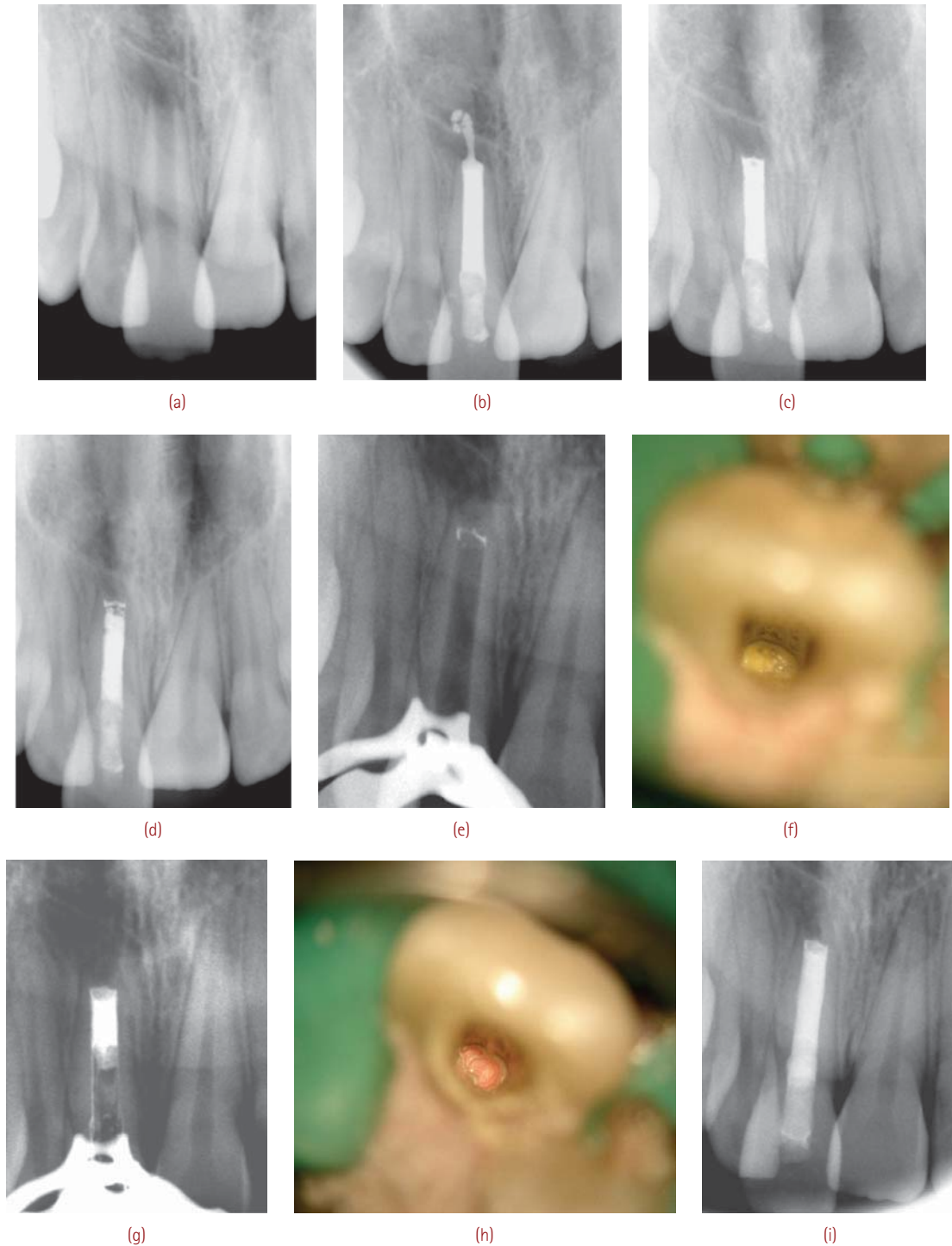


Fig. 13.25 (a) Central incisor with open apex. (b) Intracanal Ca(OH)_2 placed and GIC entrance filling given. (c) Six-month follow-up. (d) Twenty-month follow-up. (e) Calcium hydroxide removed and evidence of calcific barrier. (f) Calcific barrier seen through the microscope. (g) Thermoplasticized backfill obturation done. (h) View through the microscope. (i) Two-year recall shows good healing. (Courtesy: Siju Jacob, India.)

Bleaching of Discolored Teeth

We live only to discover beauty. All else is a form of waiting.

—Kahlil Gibran

Esthetics is an important factor in a patient's decision to undergo endodontic treatment. A frequent question is, "Will my tooth turn black?" The usual response is a "qualified no," with the explanation that modern treatment and procedures are designed to avoid crown staining and tooth discoloration. Nevertheless, teeth can and do discolor, sometimes before endodontic treatment and sometimes afterward, in spite of all precautions taken to prevent color changes. When teeth discolor, bleaching should be considered as a means of restoring tooth esthetics.

The normal color of primary teeth is bluish white. The color of permanent teeth is grayish yellow, grayish white, or yellowish white. The color of teeth is determined by the translucency and thickness of the enamel, the thickness and color of the underlying dentin, and the color of the pulp. Alterations in the color may be physiologic or pathologic and endogenous or exogenous in nature.

With age, the enamel becomes thinner because of abrasion and erosion, and the dentin becomes thicker because of the deposition of secondary and reparative dentin, which produce color changes in teeth during one's life. Teeth of elderly persons are usually more yellow or grayish yellow than those of younger persons.

Classification of Tooth Discoloration

Tooth discoloration can be classified as either extrinsic or intrinsic.

Extrinsic Discolorations

Extrinsic discolorations are found on the outer surface of teeth and are usually of local origin, such as tobacco stains. Some extrinsic discoloration, such as the green discoloration associated with the Nasmyth's membrane in children, and tea and tobacco stains (Fig. 14.1), can be removed by scaling and polishing during tooth prophylaxis. Extrinsic discolorations are stains that get bound to the tooth surface and for this reason can be easily removed. The stains deposited on the tooth surface are a result of attractive forces which are long-range interactions such as van der Waals and electrostatic forces and short-range interactions such as hydration forces, hydrophobic interactions, and hydrogen bonds. Other types of extrinsic discoloration, such as silver nitrate stains, are almost impossible to eliminate without grinding because the stains penetrate the surface of the crowns and are difficult to remove by chemical means alone.

In the past, extrinsic discoloration was classified as metallic or nonmetallic stains. The problem with this classification is that it does not explain the

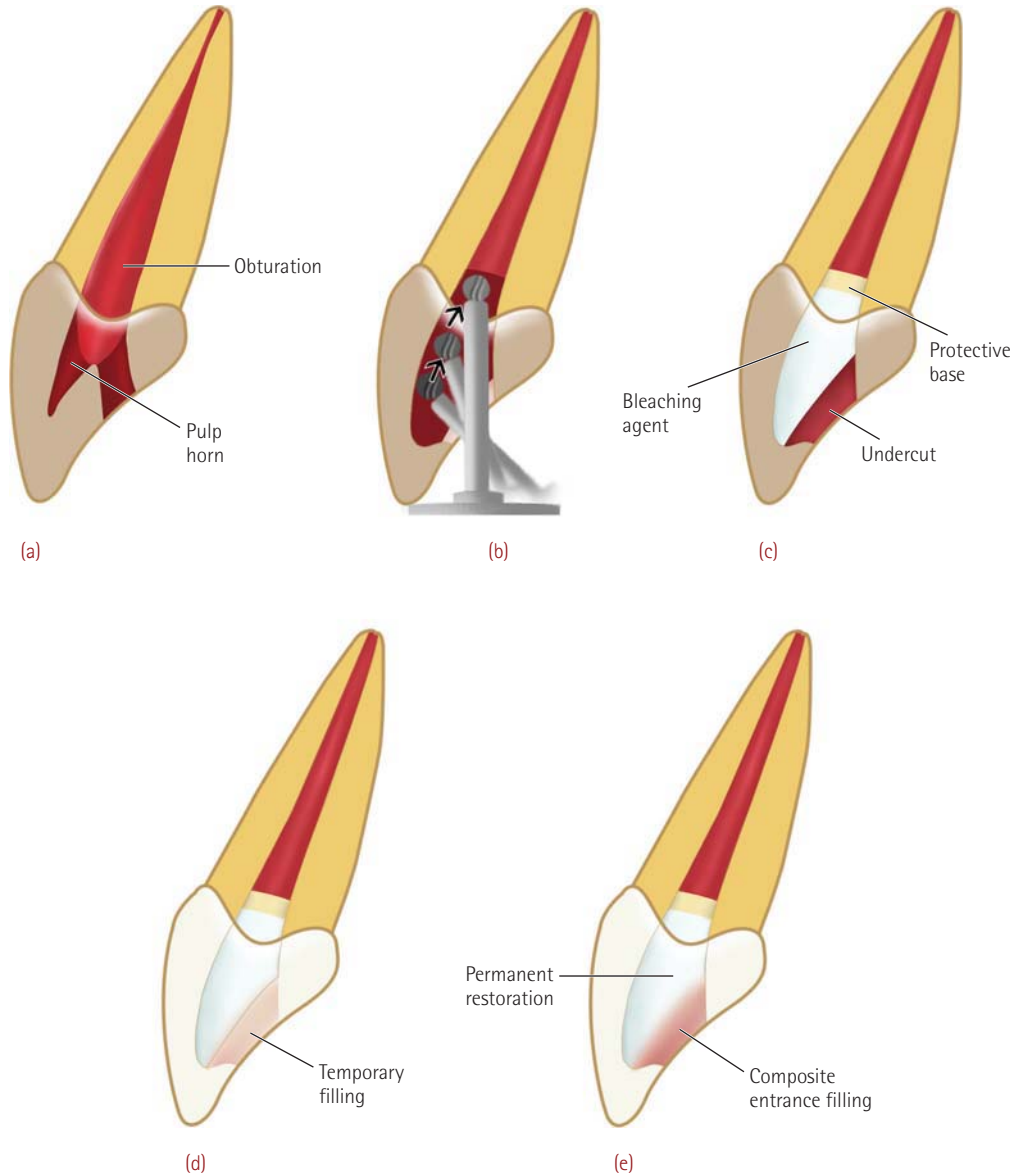


Fig. 14.9 Walking bleach: (a) Internal staining of dentin caused by remnants of obturating materials in the pulp chamber, as well as by materials and tissue debris in pulp horns. (b) Coronal restoration is removed completely, access preparation is improved, and gutta-percha is removed apically to just below the cervical margin. Next, the pulp horns are cleaned with a round bur. (Shaving a thin layer of dentin from the facial wall is optional and may be attempted at later appointments if discoloration persists.) (c) A protective cement base is placed over the gutta-percha, not extending above the cervical margin. After removal of sealer remnants and materials from the chamber with solvents, a paste composed of sodium perborate and water (mixed to the consistency of wet sand) is placed. The incisal area is undercut to retain the temporary restoration. (d) A temporary filling seals the access. (e) At a subsequent appointment, when the desired shade has been reached, a permanent restoration is placed. Acid-etched composite restores lingual access and extends into the pulp horns for retention and to support the incisal edge.



(a)



(b)



(c)

Fig. 14.15 (a) Enamel fluorosis. Isolated dark-brown stains pose a problem for tooth-veneering procedures. (b) Appearance following first sitting of microabrasion using modified McInnes solution and fine grit abrasive disks. (c) Microabrasion was repeated after 2 weeks. Note the acceptable lightening of the stains. Final esthetic appearance can be achieved by composite resin veneering. (Courtesy: Anbu, Inda.)

Tetracycline Discoloration

Classification (Jordon and Boskman, 1984)

- *First degree.* Light yellow to light gray staining without banding
- *Second degree.* Darker and more extensive yellow or gray staining without banding

- *Third degree.* Severe staining characterized with dark gray or blue discoloration with banding

Minocycline has the ability to affect permanent dentition even in adults due to its ability to form complexes with calcium in dentin. It is usually seen with long-term use for the treatment of acne.

Destaining of the yellow color is most successful, whereas brownish teeth are least successfully bleached.

The use of 30% hydrogen peroxide and a thermostatically controlled heat source for bleaching tetracycline-stained teeth has been described. Unfortunately, the decoloration is only superficial and does not affect the stained dentin. Another method of bleaching tetracycline stain has been described. In this method, the pulps of the teeth are intentionally extirpated, the root canals are cleaned, shaped and obturated, and the teeth are internally bleached as previously described. The result in humans, as well as in dogs, has been successful. We believe that labial veneers with composite resins or even porcelain-veneer full-crown restorations are indicated instead of intentional devitalization of a tooth with a normal pulp.

The application of heat to the tooth during bleaching procedures may cause some transitory reaction in the pulp. Although in one study the application of heat in the range of 115–124°F produced a slight superficial inflammation, in another study, temperatures of 114°F had no effect on the pulp. In this study, the use of hydrogen peroxide caused hemorrhage, inflammation, and destruction of the odontoblast layer of the pulp, but the reaction was reversible and healing occurred within 60 days.

Management

Teeth discolored by tetracycline may also be bleached to some extent with hydrogen peroxide, but the bleaching effect leaves something to be desired, with regard to both discoloration and permanency, because the chemical cannot reach the real cause of the discoloration, which is the incorporation of tetracycline into the dentin. The degree of staining depends on the stage of tooth development at the time when medication is begun; the greater the amount of crown developed, the less severe the stain, and vice versa.

Treatment of Traumatized Teeth

Healing is a matter of time, but it is sometimes also a matter of opportunity.

—Hippocrates

Trauma of the oral and maxillofacial region occurs frequently and comprises 5% of all injuries for which people seek treatment. Among all facial injuries, dental injuries are the most common, of which crown fractures and luxations occur most frequently. Trauma to the teeth may result either in injury of the pulp, with or without damage to the crown or root, or in displacement of the tooth from its socket. When the crown or root is fractured, the pulp may recover and survive the injury, it may succumb immediately, or it may undergo progressive degeneration and ultimately die.

Causes and Incidence of Dental Injuries

Traumatic injuries to the teeth can occur at any age. Young children learning to walk or falling from a chair are subject to anterior tooth injuries. Frequently, child abuse results in facial and dental trauma. Sports accidents and fights affect teenagers and young adults, whereas automobile accidents affect all age groups. As many dental accidents are sports related, every precaution should be taken to protect the teeth of children from such accidents by using educational programs in addition to mouth guards. The common causes of traumatic injuries to the teeth include the following:

- Direct and indirect trauma
- Sports accidents

- Automobile accidents
- Fights and assaults
- Domestic violence
- Inappropriate use of teeth
- Biting hard items

The incidence of fractures is about 5%; Ellis reports an incidence of 4.2%, and Grundy, 5.1%. Boys have about two to three times as many fractured teeth as girls. Bakland reported that children 8–12 years of age are most prone to dental accidents. The 2–4 years of age is the other age group which has a high incidence of dental traumatic injuries. The three key predisposing factors for these kinds of injuries are as follows:

- Increased overjet of the teeth
- Protrusion of the maxillary anterior teeth
- Insufficient lip closure

Fractures of Teeth

Fractures of the crowns of teeth are generally diagonal, involving a corner of the tooth, frequently the mesial. Traumatic injuries that do not cause fracture of the crown or root are just as often responsible for pulp injury as those in which the crown or root is fractured. In injuries that do not fracture the crown or root, the impact of the blow is transmitted head-on to the pulp, which receives



Fig. 15.1 Enamel infraction lines seen in a tooth with crown fracture without pulp exposure.

roughened margins to prevent laceration of the soft tissues. In more extensive enamel fractures, recontouring the roughened margins followed by esthetic composite restorations would be necessary (Fig. 15.2). If the fractured fragment is available, it can be repositioned and bonded to the tooth. Periodic assessment of the vitality status of such teeth is recommended.

Crown Fracture without Pulp Exposure

The objective in treating a tooth with a fractured crown without pulp exposure is threefold:

1. Elimination of discomfort
2. Preservation of the vital pulp
3. Restoration of the fractured crown

In an uncomplicated fracture of the crown without pulpal exposure, a remaining dentinal thickness of 2 mm is sufficient to shield the pulp and ensure a good prognosis. Inflammatory response in the form of pain on percussion is usually transient as long as the vascular supply to the pulp remains intact. Composite resin restoration is the preferred restorative procedure in such cases. In certain cases, the fractured segments can be reapproximated and bonded back with the help of dentin bonding agents and composite resins (Fig. 15.3). The use of indirect veneering procedures at a later date is another approach to improve the esthetics.

The tooth should be periodically tested with the electric pulp tester or with ice or ethylchloride spray. If the pulp tests are vital and the tooth comfortable, it should be checked again after a week, a month, 3 months, 6 months, and a year. Radiographs should be taken at 6-month intervals. If the pulp continues to respond normally during this



(a)



(b)

Fig. 15.2 (a) Enamel fracture. (b) Recontouring followed by direct composite restoration.



(a)



(b)

Fig. 15.3 (a) Crown fracture without pulpal exposure. (b) Restoration with direct composite restoration.

time, the pulp can be assumed to have recovered. If progressively more current is necessary to elicit a vitality response, the pulpal prognosis is unfavorable, and the pulp will probably become necrotic necessitating endodontic treatment.

Crown Fracture with Pulp Exposure

For a tooth with a fractured crown with pulp exposure, four kinds of treatment are possible:

1. Direct pulp capping
2. Pulpotomy (pulp is vital)
3. Apexification (pulp is necrotic)
4. Pulpectomy (endodontic treatment)

The primary aim for a fractured crown presenting with a pulpal exposure is to maintain the pulpal

vitality. Mechanical exposure of the pulp due to trauma has a better prognosis than carious exposures. Every attempt must be made to minimize bacterial contamination of the exposure. The extent of fracture and the stage of root development are the two critical factors that would determine the treatment plan.

For more details on direct pulp capping, pulpotomy, and apexification, the student is referred to Chapter 13.

Crown–Root Fractures

This kind of traumatic dental injury is characterized by an oblique fracture line that usually begins few millimeters incisal to the marginal gingiva and extends beyond the gingival crevice. They resemble a crown fracture but are more complex to treat as the fracture involves the root also. Clinically, the displacement of the coronal fracture segment is minimal as fractured segments are held together by the underlying periodontal ligament.

The fracture line is usually single but multiple fractures can occasionally be seen. In spite of the pulpal exposure, symptoms are mild and pain is due to mobility of the fractured segment during function (Fig. 15.4). The emergency care in such cases comprises bonding of the loose tooth fragments and in certain cases initiation of pulp



(a)

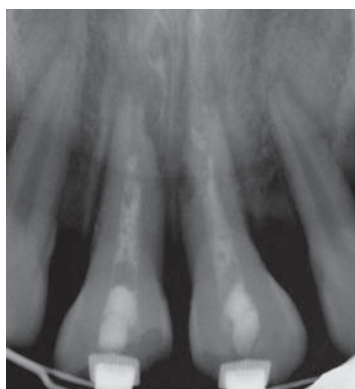
Fig. 15.4 (a) Complicated crown fracture of maxillary canine. (continued)



(d)



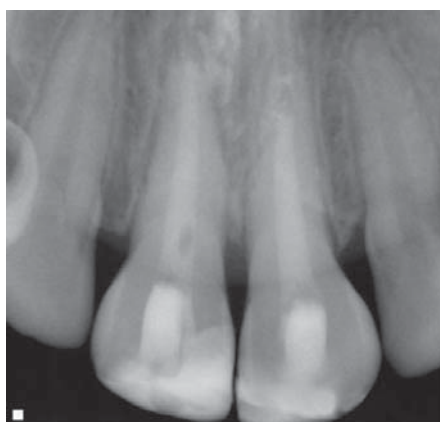
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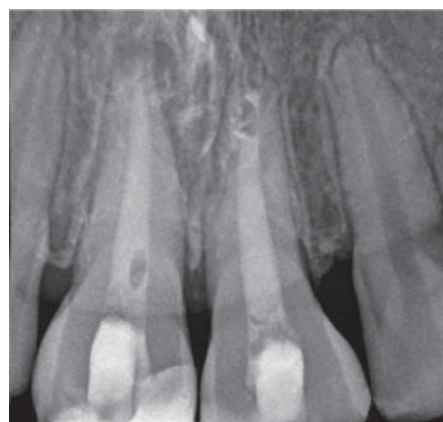
(f)



(g)



(h)



(i)

Fig. 15.16 (continued) (d) Three-month view: orthodontic extrusion initiated. (e) Three-month view: endodontic treatment commenced with the placement of intracanal calcium hydroxide. (f) Ten-month view: intracanal calcium hydroxide dressing changed. (g) One-year-and-one-month view: orthodontic treatment discontinued. (h) One-year-and-one-month view: intracanal calcium hydroxide dressing changed. (i) One-year-and-seven-month view: intracanal calcium hydroxide dressing changed. (continued)

Endodontic Surgery

We are what we repeatedly do Excellence, then, is not an act ... but a habit.

—Aristotle

The scope of endodontic surgery has extended beyond root-end resection to include other forms of periradicular surgery, fistulative surgery, corrective surgery, and intentional replantation. Root-end resection is still the most common form of periradicular surgery.

In general practice, the number of cases being indicated for root-end surgery has drastically reduced over a period of time. This may be due to the fact that today the science of endodontics has a better understanding of the biological principles of cleaning and shaping. In the last few decades, endodontics is more of a biological science than mere chemomechanical debridement. There has been a tremendous improvement in the available materials and instruments for cleaning and shaping. With the present knowledge of internal anatomy of pulp space, microbiology, disinfection of the pulp space, and also with the introduction of rotary and micro-endodontic instrumentation, clinicians are better equipped to produce a more predictable and clean pulp space.

Most of the endodontic periradicular pathologies can be managed nonsurgically. There has been a gradual paradigm shift from surgical to nonsurgical treatment over the past few decades. However, the nonsurgical management may not be always successful. Even if nonsurgical treatment is unsuccessful, the current concept is to do an introspection of the quality of nonsurgical treatment

before selecting surgical intervention. If the initial endodontic treatment of a tooth is not satisfactory then one should attempt nonsurgical retreatment of that tooth first.

The view that endodontic surgery is the last resort is based on past experience with instruments that were unsuitable. Also, the vision available at the surgical site was inadequate and incidence of postoperative complications was high. Fortunately, today the endodontist is equipped with better magnification, illumination, and instruments. The present era of microsurgery is with surgical operating microscopes, ultrasonic tips for retro-preparation, low-speed high-torque motors, and miniaturized surgical instruments for root-end surgery. The introduction of dental surgical microscopes has also resulted in better understanding of apical third anatomy, better root-end surgical resection techniques, better patient interaction, and all this has resulted in better success rates. The success rate of surgical endodontics is high, about 73–99%, depending on the criteria used for evaluating success.

Objectives and Rationale for Surgery

- **Curettage.** Effective curettage of the pathologically affected periradicular tissue which cannot be accessed in an orthograde approach. This

With DOMs, one can visualize the surgical field and evaluate the surgical technique. The magnification of a microscope is determined by:

- Power of the eyepiece
- Focal length of the binoculars
- Magnification change factor
- Focal length of the objective lens

Illumination

Illumination with DOMs is coaxial with the line of sight. This means the light is focused between the eyes in such a fashion that you can look into the surgical site without seeing any shadow. Elimination of shadow is made possible by using Galilean optics, which focuses at infinity and sends parallel beams of light to each eye. With parallel light, the operator's eyes are at rest and therefore lengthy operations can be performed without eye fatigue.

Magnification and illumination with the DOMs have helped the endodontists to introduce microsurgery into surgical endodontics. This, in turn, has helped the endodontists to minimize trauma and enhance the surgical results. The DOMs have benefitted treatment protocols in periradicular surgery.



Fig. 16.3 Dental operating microscope.

They have become a vital armamentarium in not only microsurgical endodontics, but they play a key role in enhancing the quality and precision of routine nonsurgical endodontic therapy (Figs. 16.3 and 16.4). The arrival of DOMs has also brought in revolutionary changes in instruments and instrumentation techniques. The introduction of ultrasonic instruments has made minimal and precise retropreparation feasible during root-end surgery. The introduction of



(a)



(b)

Fig. 16.4 (a) A dental operating microscope (DOM)-centered endodontic practice enhances clinical performance and outcomes. (b) Dental operating microscope with beam splitter for documentation. (Courtesy: Global Microscopes.)

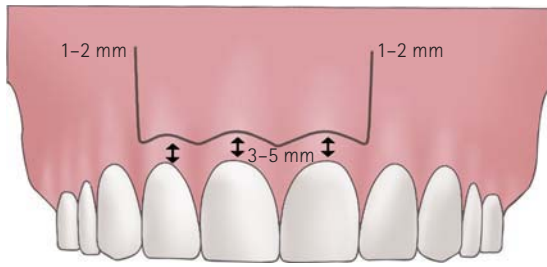


Fig. 16.11 The Luebke-Ochsenbein design connects a scalloped horizontal incision in the attached gingiva with two apically directed vertical incisions. The incisions extend from a point 1–2 mm short of entering the mucobuccal fold to a point on the attached gingiva 3–5 mm above or below the marginal gingiva and sulcus depth.

the incision follows the direction of the tissue fibers and the blood vessels. If one follows this concept, fewer fibers and blood vessels are severed, and in the post-operative phase, sutured incisions are hardly noticeable because they quickly heal.

- The submarginal scalloped rectangular flap also called Luebke-Ochsenbein flap is ideal for crowned teeth when open crown margins after surgery are an esthetic concern. The Luebke-Ochsenbein flap (Fig. 16.11) is often described as that consisting of two vertical incisions and

one horizontal scalloped incision away from the gingival tissues, i.e., around 3–5 mm away from the gingival attachment. With this scalloped horizontal incision, the attached gingiva around the gingival margins remains intact and one can be assured of preserving the existing esthetics. While designing this flap, the two vertical incisions are to remain parallel to each other so that the width of the flap at the base is same as it is at the top. It is important to observe here that the horizontal scalloped incision placed in the middle of the attached gingiva should have an angle of 45° to the cortical plate which provides the widest cut surface allowing for meticulous adaptation when the flap is repositioned.

Flap Elevation

Even though several types of elevators are available, the Molts curette no. 2–4 is suitable for both elevation and curetting with minimum trauma. One has to gently walk the elevator against the bone taking care not to tear the flap. It is necessary to reflect the flap along with the periosteum to minimize bleeding during the surgical procedure (Fig. 16.12).

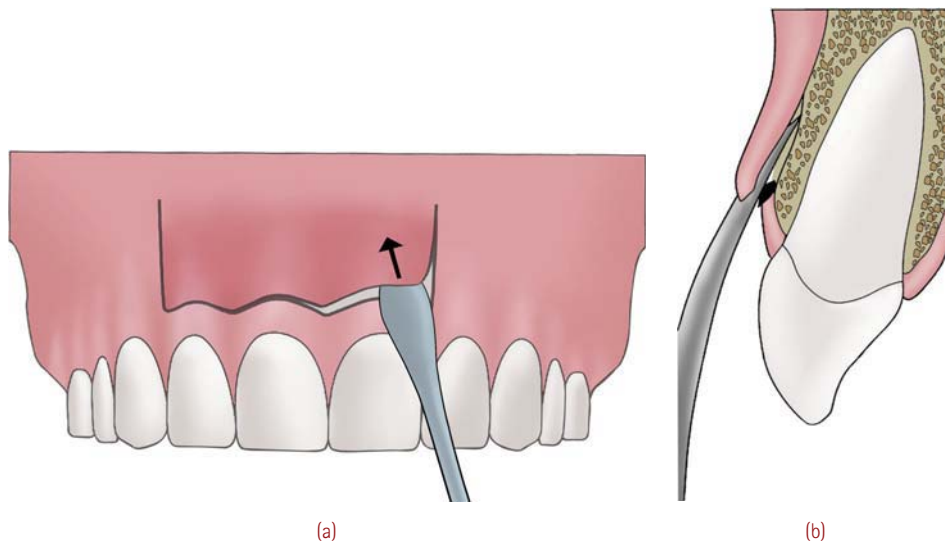
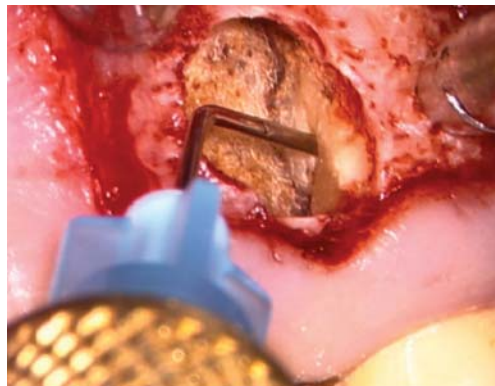


Fig. 16.12 (a) and (b) Raising a Luebke-Ochsenbein flap: the elevator edge, with its concave surface facing the bone, cleaves the periosteum from bone apically and laterally until the bone above the lesion is exposed. (continued)



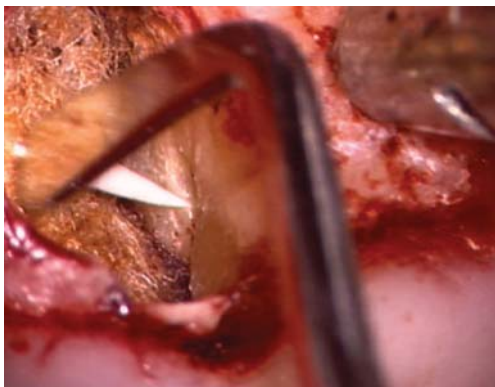
(c)



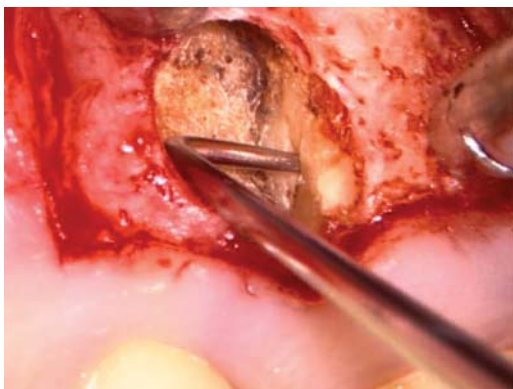
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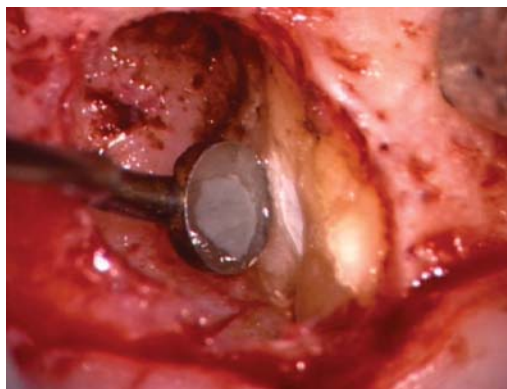
(e)



(f)



(g)



(h)

Fig. 16.24 (continued) (c) The ultrasonic tip is preparing the lateral cavity for the retrofill. (d) The cavity is now dried with the Stropko Irrigator. (e) The cavity is now ready to be obturated. (f) The retrofilling material is being carried in the cavity. (g) The microplugger is condensing the material. (h) The lateral retrofill has been completed and finished. (continued)

Endodontic–Periodontic Interrelationship

*Not everything that can be counted counts,
and not everything that counts can be counted....*

–Albert Einstein

The tooth, its pulp, and its supporting structures must be viewed as a biological unit. Because the vitality of the tooth depends on its ability to function, and not on the viability of the pulp, the health of these structures is of prime importance. The interrelations among these structures influence each other during function and disease.

Until recently, an endodontically involved tooth with an underlying periodontal pathology was considered to have a questionable prognosis. As a result, many teeth were sacrificed unnecessarily. Fortunately, the combined endodontic–periodontic lesion that affects a single tooth can now be diagnosed and treated successfully, with a predictable prognosis in many instances.

Pulpoperiodontal Pathways (Fig. 17.1)

The pulp and the periodontium have intimate embryological, anatomical, and vascular pathways of communication. Pathology in either of these two structures invariably leads to an adverse effect on the other. This is evident from the fact that pulpoperiodontal pathoses are responsible for more than 50% of tooth mortality. The following are the means of intercommunication between the pulp and the periodontal tissues:

- Apical foramen
- Dentinal tubules
- Lateral canals
- Periodontal ligament
- Alveolar bone
- Palatogingival groove
- Neural pathways
- Vasculolymphatic drainage pathways
- Pathological communications due to fractures and perforations

Etiology of Endo-Perio Lesions

Examination of the etiological factors that cause endoperio lesions indicates that these factors originate from either endodontic or periodontal or both the disease processes. For example, a progressing infrabony pocket exposes the pulp tissue to the oral environment by “uncovering” a lateral canal and resulting in irreversible pulpal inflammation. Both endodontic and periodontal therapies are required for healing to occur. Similarly, furcation bone loss can lead to pulp exposure by “uncovering” a subpulpal-floor accessory canal, with concomitant sequelae. Thus, it is possible for periodontal disease to cause secondary pulpal disease.

Conversely, pulpal disease can cause periodontal disease. A pulpal infection can spread

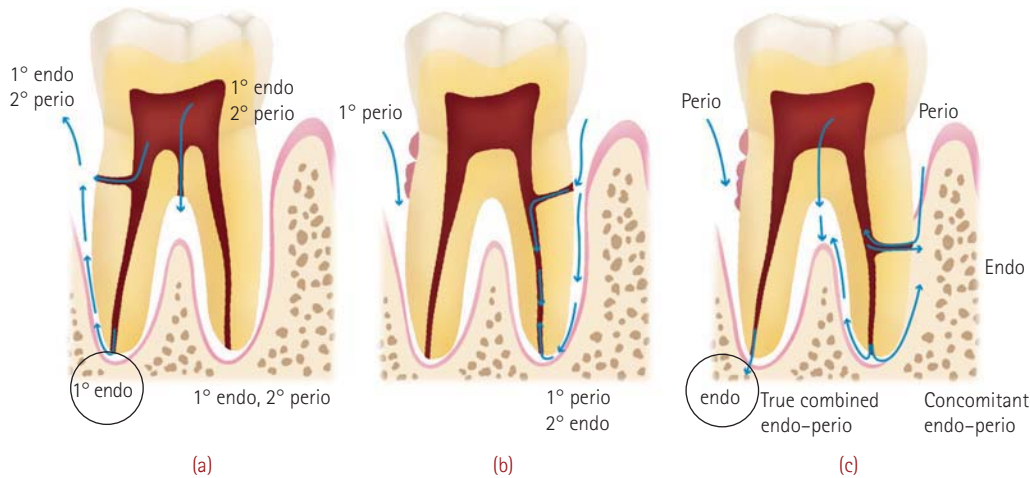


Fig. 17.1 Endodontic and periodontal pathways: (a) Endodontic lesions. The pathway of inflammation is through the apical foramen, furcation canals, and lateral accessory canals to the periodontium. This results in a primary endodontic lesion, sometimes progressing towards secondary periodontal involvement. (b) Periodontal lesions. This is the progression of periodontitis by way of lateral canal and apex to induce a secondary endodontic lesion. (c) True combined endodontic and periodontal lesion and concomitant endodontic and periodontal lesions.

through lateral and accessory canals or apical foramina and may cause furcation breakdown, infrabony pocket formation, and periapical lesions. Conceivably, a persistent sinus tract draining through the gingival crevice could become an infrabony pocket and could require combined therapy for healing to occur.

The time period of persistence of the etiological factors in a susceptible environment is directly related to the probability that combined therapy will be needed. In other words, duration can be a key factor in evaluating etiological effects.

Classification

Understanding the endodontic–periodontic relationship is essential because it frequently dictates the plan of treatment. Oliet and Pollock suggested a classification for the combined endodontic–periodontic lesion based on treatment procedures.

Oliet and Pollock's Classification

This classification is based on treatment protocol.

I. Lesions that require endodontic treatment procedures only

1. Any tooth with a necrotic pulp and periradicular pathosis, with or without a sinus tract (chronic periapical abscess).
2. Chronic periapical abscess with a sinus tract draining through the gingival crevice, thus passing through a section of the attachment apparatus in its entire length alongside the root.
3. Root fractures, longitudinal and horizontal.
4. Root perforations, pathologic and iatrogenic.
5. Teeth with incomplete apical root development and inflamed or necrotic pulps, with and without periradicular pathoses.
6. Replants, intentional or traumatic.
7. Transplants, autotransplants or allotransplants.
8. Teeth requiring hemisection or radisectomy.
9. Intentional endodontic therapy for prosthodontic consideration.

II. Lesions that require periodontal treatment procedures only

1. Occlusal trauma causing reversible pulpitis.
2. Occlusal trauma plus gingival inflammation, resulting in pocket formation.
 - (a) Reversible but increased pulpal sensitivity caused by trauma or, possibly, by exposed dentinal tubules.

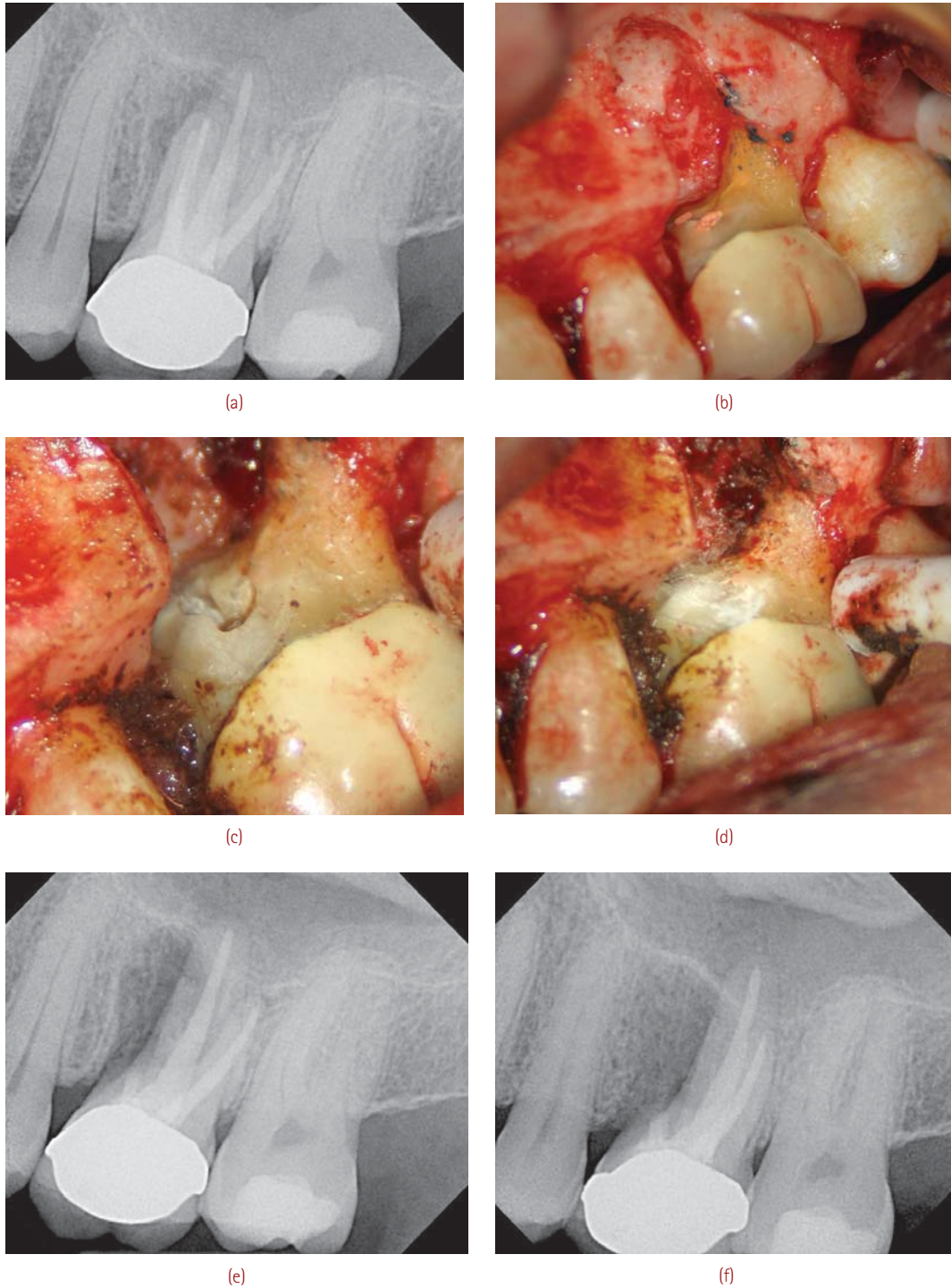


Fig. 17.10 Radisectomy of the mesiobuccal root of a maxillary molar: (a) Lesion in relation to the mesiobuccal root. (b) Osteotomy and radisection of the mesiobuccal root completed. Note the exposed gutta-percha from both the mesiobuccal canals. (c) Ultrasonic retropreparation completed in both canals. (d) MTA retrofilling done. (e) Immediate postoperative radiograph. (f) Three-month follow-up radiograph showing healing. (Courtesy: Jason Hales, United States of America.)

Prosthodontic Considerations in Endodontically Treated Teeth

CHAPTER

18

Our objective should be the perpetual preservation of what remains than the meticulous restoration of what is missing.

—M.M. De Van

A successful endodontic treatment has to be complemented with an adequate post-endodontic restoration to make the pulpless tooth function indefinitely as an integral part of the masticatory apparatus. The cumulative loss of tooth structure due to caries, trauma, and endodontic procedures combined with the loss of structural integrity contributes to the fracture of the tooth.

Careful postendodontic restoration is required as endodontically treated teeth are mainly lost due to restorative difficulties and failure of the root canal treatment. Ideally, the final restoration should be planned before root canal treatment is begun, though the restorative plan may be modified as the treatment progresses.

Assessment of Restorability

An endodontically treated tooth must be evaluated before definitive restorative procedures are initiated. Evaluation factors (Fig. 18.1) are used to determine whether the endodontically treated tooth is restorable, unrestorable, or restorable after successful retreatment. Definitive restorative treatment should not be initiated if the treated tooth exhibits any of the following:

1. Poor root canal filling
2. Active inflammation

3. Pressure sensitivity
4. Exudate
5. Fistula (or parulus)
6. Periodontal disease (moderate or severe periodontitis)
7. Severe loss of sound tooth structure (tooth would not benefit from crown lengthening or orthodontic extrusion)

In short, seven categories of infection, trauma, inflammation, unacceptable endodontics, or lack of restorability, as listed, can delay or end up in no definitive restorative treatment (Figs. 18.2 and 18.3).

The choices of treatment for such cases include the following:

1. Retreatment
 - (a) Endodontic retreatment (can reverse inflammation, permitting the tooth to receive restorative treatment)
 - (i) Nonsurgical endodontic retreatment
 - (ii) Surgical endodontic retreatment
 - (b) Periodontal retreatment (tooth will require stabilization)
2. Monitoring (time to assess progressive healing)
3. Extraction (unrestorable)

If none of the aforementioned problems exist, a definite restorative treatment may be initiated. The treatment guidelines for anterior and posterior

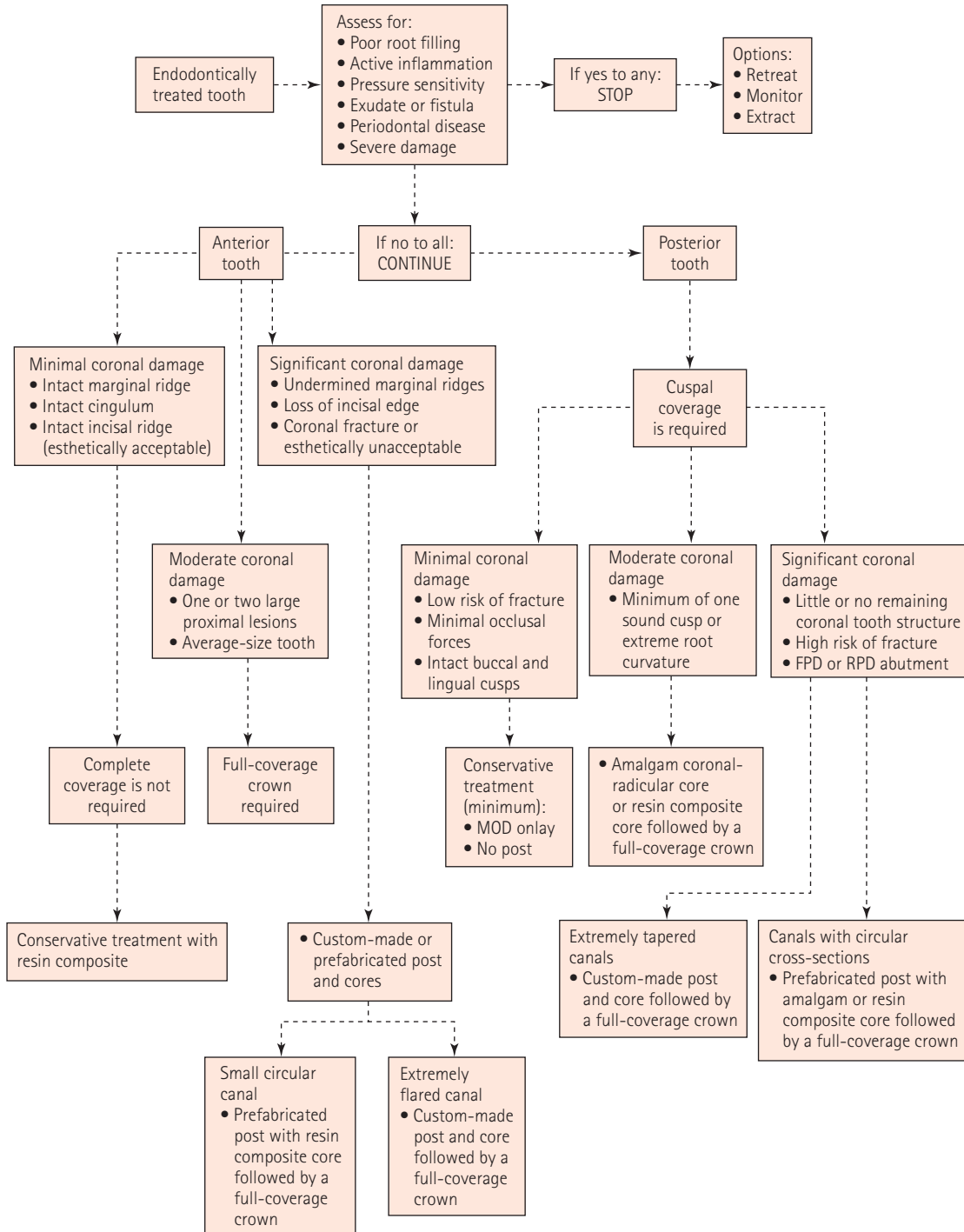


Fig. 18.1 Restorative decision-making chart. FPD, fixed partial denture; RPD, removable partial denture; MOD, mesio-occlusal-distal.

Lasers in Endodontics

*We can easily forgive a child who is afraid of the dark.
The real tragedy of life is when an adult is afraid of the light.*

–Plato

Numerous researchers have investigated laser applications in dentistry in the past five decades. Research and development has lead to a multitude of lasers both in type and means of delivery systems. With better understanding of biological tissue responses, the possibilities of a clinician using lasers in a range of endodontic procedures have increased, varying from pulpal diagnosis, pulpotomy, root canal disinfection, retreatment to root resection.

- 1988 Miserendino: Apicectomy with CO₂ laser
- 1990 Potts and Petrou: Laser-aided photopolymerization of camphoroquinone-activated resins
- 1993 Paghdiwala: Er:YAG lasers for root resection and retrograde cavity preparation
- 1994 Morita: Nd:YAG lasers in endodontics
- 1998 Mazeki et al.: Root canal shaping with Er:YAG laser

Chronology of Laser Development

- 1916 Albert Einstein: Theory of spontaneous emission of radiation
- 1953 Charles Townes: MASER (microwave amplification by the stimulated emission of radiation)
- 1960 Theodore Maiman: Ruby laser
- 1964 Stern, Sognnaes, and Goldman: Lasers in dentistry
- 1971 Weichman and Johnson: Lasers in endodontics
- 1979 Adrian and Gross: Argon laser sterilization of dental instruments
- 1985 Shoji et al.: Laser-aided pulpotomy
- 1986 Zakariasen et al.: Sterilization of root canals

Basics of Laser Physics

Light is a form of electromagnetic energy that travels at a constant velocity in the form of waves. The basic unit of light is termed *photon*, or a particle of light. Ordinary light, as produced by the sun or a table lamp, is a sum of many types of photons or light waves that are diffuse and not focused. Photons are released when an atom in its ground state absorbs energy, gets excited, and moves to a higher energy state. The release of photon is spontaneous in nature and hence termed *spontaneous emission*. This is accompanied by neighboring atoms producing waves that are not in phase with one another, thereby having varying wavelengths and amplitude. The light waves produced are noncoherent and polychromatic (Fig. 19.1).

Two photons are released if additional quantum of energy is absorbed by an already excited atom. The

- **Absorption.** This depends on tissue characteristics like water content and level of pigmentation and on laser wavelength and emission mode.
- **Transmission.** The tissue transmits the laser energy through the tissue, with no effect on target tissue. This depends on the wavelength of the laser employed.
- **Scattering.** This process weakens the energy of the beam and produces no useful biological effect. Scattering can cause heat transfer to the adjacent tissues and consequent thermal damage.

Laser Wavelengths Used in Dentistry

Nd:YAG

- Solid active medium containing crystal of yttrium–aluminum–garnet doped in neodymium



FIG. 19.7 Nd:YAG laser.

- Wavelength: 1064 nm (invisible beam in the infrared range)
- First laser designed exclusively for dentistry
- Fiberoptically delivered in a pulsed mode and is most often used in contact with the tissue
- The pulsed Nd:YAG laser is ideal for soft-tissue procedures and root canal sterilization (Fig. 19.7)

Diode

- Solid-state semiconductor laser that uses a combination of aluminum, gallium, and arsenide that converts electric energy into light energy
- Wavelength: 800–980 nm
- Fiberoptically delivered in continuous or in a pulsed mode and used commonly in contact with the tissue
- Poorly absorbed by tooth structure; hence, soft-tissue surgery can be safely performed in close proximity to dental hard tissues
- Very well absorbed by pigmented tissues and is a good soft-tissue surgical laser indicated for precision cutting and coagulation of gingiva
- Advantage of being compact, portable, and economical (Fig. 19.8)



Fig. 19.8 Diode laser.

Procedural Errors: Prevention and Management

CHAPTER

20

Experience is what we get, when we don't get what we want ...!

Procedural errors during endodontic therapy occur not only with students and beginners, but also with skilled and experienced clinicians in spite of taking all possible precautions. Such iatrogenic complications can be prevented by adhering to the fundamental biological and mechanical guidelines of endodontics. A clinician should have the ability to:

- Assess and inform the patient about the prognosis of a case before initiating the treatment.
- Identify the given clinical problem with diagnostic acumen.
- Anticipate problems in challenging cases.
- Use appropriate materials and modifications in routine techniques in order to prevent procedural errors.
- Identify clinical problems the moment they occur during the procedure and manage them positively.

Before commencing a procedure, the endodontist (or a general practitioner), trained in endodontics, must do a sincere introspection of his/her capabilities in handling an endodontic situation. If it is not within the purview of the attending clinician, then he/she should either call in a peer consultant or refer the case to one who can handle the situation.

Procedural errors can be avoided to a larger extent by following these guidelines:

- Establish a proper communication and rapport with the patient. The patient has a moral and legal right to be informed about every step of the treatment procedure.
- Complete a thorough history and meticulous clinical examination of the tooth or teeth in question before commencing the treatment.
- Ascertain the periodontal status, prosthodontic restorability, and prognosis of tooth in question.
- One must have a thorough knowledge of internal anatomy of pulp space and variations in the root canal configuration. Also required is a complete knowledge of apical terminus of root canals especially apical foramen, apical constriction, and anatomical apex. In addition, one must know approximately at what age root apex is complete in different teeth in the arch. A beginner can learn to anticipate these variations by observing cross and vertical ground sections of all teeth at various levels (see Chapter 9).
- Obtain an intraoral periapical radiograph for every step of endodontic therapy. These include one before commencing the treatment, during working length determination, during master apical gutta-percha cone selection, and one following the completion of the obturation.

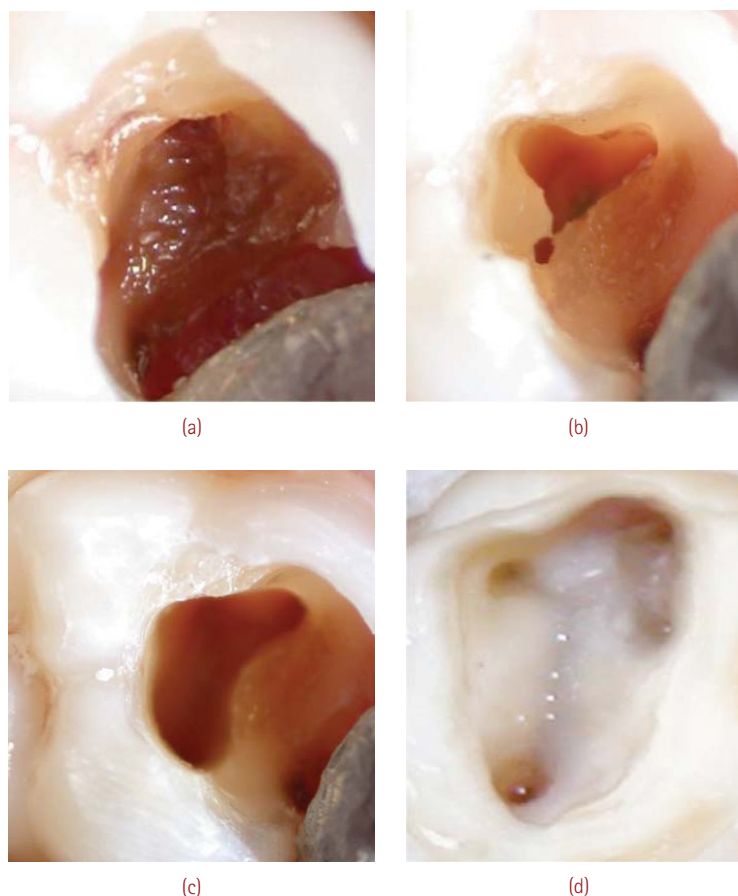


Fig. 20.5 (a) Distal carious exposure of pulp in a maxillary first molar. (b) Caries removed and the triangular opening seen is the access entry into the pulp chamber through the roof. Note the incomplete removal of the roof of the pulp chamber. (c) Access modified to remove the roof and facilitate straightline access to all the canals. (d) Access refined and the second mesiobuccal canal traced.

- Incomplete removal and shaping of lateral walls of the pulp chamber
- The access openings in both maxillary and mandibular molars are always on the mesial half of the occlusal surface rarely extending across the midline (see Figs. 9.19 and 9.36)
- In maxillary premolars, the opening is always buccolingual with one canal under buccal cusp and one under palatal cusp (see Fig. 9.14)

Clues in Locating Extra Canals (Fig. 20.6)

- Case report of mandibular first molar with a middle mesial canal (Fig. 20.7)

- Case report of mandibular second premolar with four canals (Fig. 20.8)

Prevention and Action

- Good periapical radiographs preoperatively and during root canal cleaning and shaping. Observe radiographs under magnification.
- Multiple radiographs in varying angulations help the clinician to better understand the morphology of the tooth and aid in tracing extra canals (Figs. 20.9 and 20.10).
- Use of surgical loupes and DOMs with better enhanced vision and lighting up the focus area immensely helps the clinician to handle

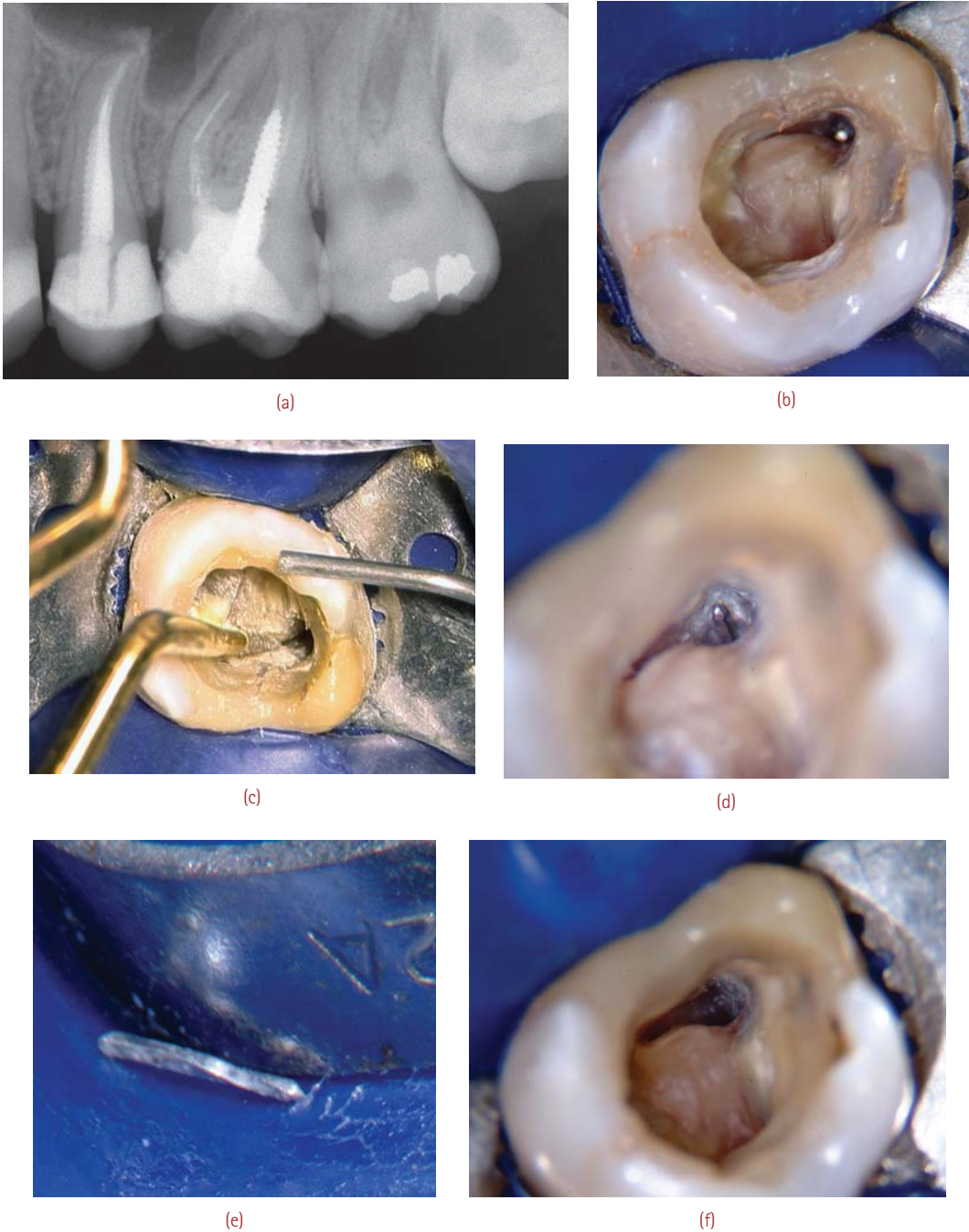


Fig. 20.31 (a) A preoperative radiograph of an endodontically failing left first molar. The tooth has a screw post in the palatal canal and a broken instrument in the mesiobuccal canal. (b) After removing the screw post and cleaning the pulp chamber, now the most coronal portion of the fragment is evident under the operating microscope. (c) The ProUltra #4 ultrasonic tip is isolating the fragment and transmitting vibrations, while the dental assistant is blowing air using the Stropko irrigator. (d) Now the fragment is more evident. (e) The fragment has been removed with ultrasonics and is now on the rubber dam, near the rubber dam clamp. (f) The clinical image confirms the removal of the fragment. *(continued)*

Radiographic Technique for Endodontics

The most pathetic person in the world is someone who has sight but no vision.

—Helen Keller

Radiographs are indispensable diagnostic and prognostic aids in endodontics and are one of the most reliable methods of monitoring endodontic treatment. Radiographs provide an important visual method of gaining clinical knowledge of teeth and periradicular tissues; therefore, they are essential to the practice of endodontics.

Proper positioning and stabilization of the radiographic film during endodontic procedures becomes difficult because of the interference from the protruding rubber dam clamp or root canal instruments or obturating material protruding from the access cavity. The visualization of the tooth for proper film positioning and cone angulation is impeded by the presence of the rubber dam. This makes the process of taking a radiograph a difficult proposition.

Radiographic Technical Requirements

1. The image of the tooth being evaluated or undergoing endodontic therapy should be in the center of the radiograph.
2. Radiographs should show at least 5 mm of bone surrounding the apex of the tooth being evaluated or undergoing endodontic therapy.

3. If a periradicular lesion is too large to fit in one periapical film, supplemental diagnostic radiographs must be made.
4. A single radiograph taken from one direction only may not provide sufficient diagnostic information in multirooted teeth or teeth with curved roots. Under these circumstances, at least two periapical radiographs should be taken to help gain a three-dimensional perspective. One radiograph should be taken at normal vertical and horizontal angulation, while the other at a 20° change in horizontal angle from either mesial or distal direction (Fig. A.1).
5. If a sinus tract is present, a tracing radiograph should be taken. This procedure is accomplished by carefully threading a gutta-percha cone into the tract and taking a radiograph to identify the origin of the tract (see Figs. 5.16 and 15.12). This technique is also useful for localization and depth marking of certain periodontal defects.
6. Correct processing of the radiographic film is essential to evaluate success or failure of the case at a later date.

In endodontics, the long-cone paralleling technique is preferred over the short-cone bisecting-angle technique because dimensional distortion is lesser, the image is sharper, and the same angulations are easily reproduced. The paralleling technique may



Fig. A.4 Correct method of assembling an endodontic film holder for anterior teeth. The assembly consists of anterior rod inserted in radiographic film holder.



Fig. A.5 Beam-alignment ring in place on an endodontic film holder.



Fig. A.6 Ensure the film is centered in the beam-alignment ring. Note that the long axis of the film is parallel to the anterior rod.

- (d) Align the X-ray tube with the rod and beam-alignment ring to obtain correct vertical and horizontal angulations (Figs. A.8 and A.9).
- (e) Make the exposure.
- (f) Replace the rubber dam frame.

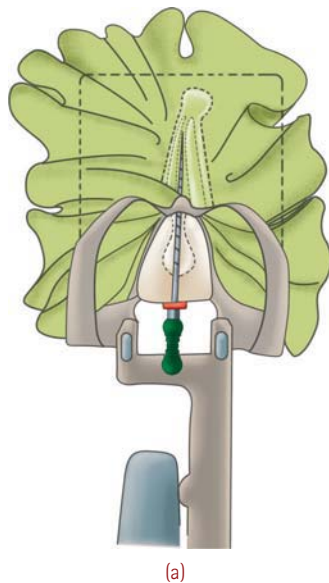


Fig. A.7 Radiographic technique for anterior teeth. (See text for details.)

Root Canal Configuration

The palest ink is better than the best memory.

—Chinese Proverb

TABLE B.1 *Root Canals and Apical Foramina in Maxillary First Premolars*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)	Three Canals (%)
Pineda and Kuttler	26.2	7.7	23.9	41.7	0.5
Green	8.0	—	26.0	66.0	—
Cams and Skidmore	9.0	—	13.0	72.0	6.0
Vertucci and Gegauff	8.0	7.0	18.0	62.0	5.0
Bellizzi and Hartwell	6.2	—	—	90.5	3.3

TABLE B.2 *Root Canals and Apical Foramina in Maxillary Second Premolars*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)	Three Canals (%)
Pineda and Kuttler	62.8	8.9	19.0	9.3	—
Green	72.0	—	24.0	4.0	—
Vertucci and colleagues	48.0	—	27.0	24.0	1.0
Bellizzi and Hartwell	40.3	—	—	58.6	1.1

TABLE B.3 *Root Canals and Apical Foramina in Maxillary First Molars: Mesibuccal Root*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)
Weine	48.5	—	37.5	14.0
Pineda and Kuttler	39.0	—	12.5	48.5
Pineda	41.0	—	17.0	42.0
Seidberg and colleagues	38.0	—	37.0	25.0
Pomeranz and Fishelberg	72.0	—	17.0	11.0
Vertucci	45.0	—	37.0	18.0

TABLE B.4 *Root Canals and Apical Foramina in Maxillary Second Molars: Mesibuccal Root*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)
Pineda and Kuttler	64.6	14.4	8.2	12.8
Pomeranz and Fishelberg	62.1	—	13.8	24.1
Vertucci	71.0	—	17.0	12.0

TABLE B.5 *Root Canals and Apical Foramina in Mandibular Incisors*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)
Green	80.0	—	7.0	13.0
Rankine-Wilson and Henry	60.0	—	35.0	5.0
Green	79.0	—	17.0	4.0
Madeira and Hetem	88.5	—	11.0	0.5
Benjamin and Dowson	59.0	—	40.0	1.0
Vertucci	92.5	—	5.0	2.5

TABLE B.6 *Root Canals and Apical Foramina in Mandibular Canine*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)
Pineda and Kuttler	81.5	—	13.5	5.0
Green	87.0	—	10.0	3.0
Vertucci	80.0	—	14.0	6.0

TABLE B.7 *Root Canals and Apical Foramina in Mandibular First Premolars*

Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)	Three Canals (%)
Pineda and Kuttler	74.2	23.4	—	1.5	0.9
Green	86.0	—	4.0	10.0	—
Zillich and Dowson	76.9	—	5.2	17.5	0.4
Vertucci	74.0	24.0	—	1.5	0.5

TABLE B.8 *Root Canals and Apical Foramina in Mandibular Second Premolars*

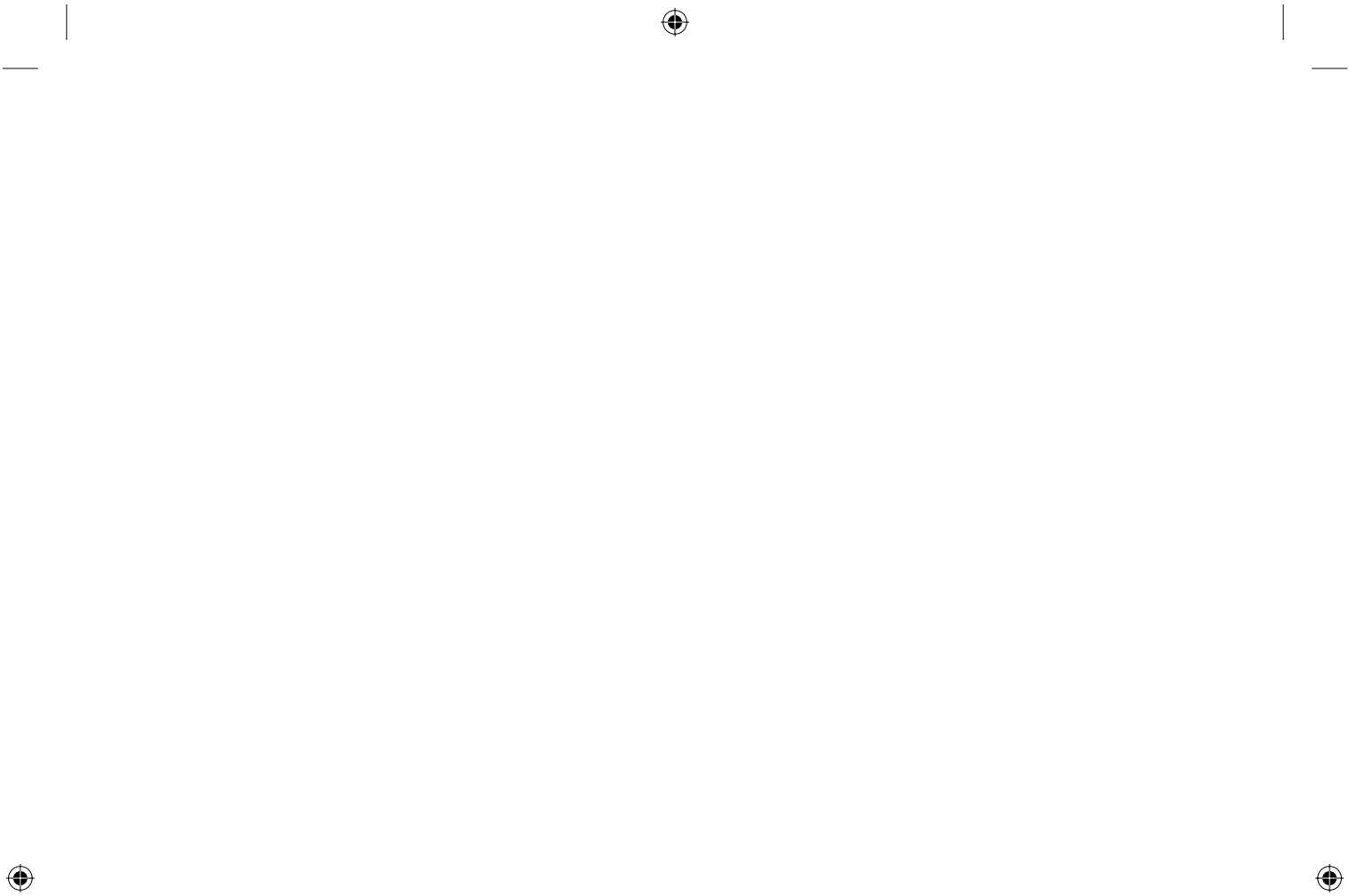
Investigator	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)	Three Canals (%)
Pineda and Kuttler	98.8	1.2	—	—	—
Green	92.0	—	4.0	4.0	—
Zillich and Dowson	87.9	—	0.9	10.8	0.4
Vertucci	97.5	2.5	—	—	—

TABLE B.9 *Root Canals and Apical Foramina in Mandibular First Molars*

Investigator	Roots	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)	Three Canals (%)
Skidmore and Bjorndahl	Mesial	6.7	—	37.8	55.5	—
	Distal	71.1	—	17.7	11.2	—
Pineda and Kuttler	Mesial	12.8	—	—	30.2	57.0
	Distal	73.0	—	—	12.7	14.3
Vertucci	Mesial	12.0	8.0	28.0	51.0	1.0
	Distal	70.0	8.0	15.0	7.0	—

TABLE B.10 *Root Canals and Apical Foramina in Mandibular Second Molars*

Investigator	Roots	One Canal and One Foramen (%)	One Canal and Two Foramina (%)	Two Canals and One Foramen (%)	Two Canals and Two Foramina (%)
Pineda and Kuttler	Mesial	58.0	—	20.6	21.4
	Distal	73.0	—	12.7	14.3
Vertucci	Mesial	27.0	9.0	38.0	26.0
	Distal	92.0	1.0	3.0	4.0



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