GROSSMAN'S ENDODONTIC PRACTICE

14TH EDITION

Editor

V. Gopikrishna, BDS, MDS, PhD

Founder-Director
Root Canal Foundation
www.rootcanalfoundation.com
Chennai, India

and

Adjunct Professor
Department of Conservative Dentistry & Endodontics
Sri Ramachandra Institute of Higher Education and
Research University
Chennai, India

Wolters Kluwer

Sr. Publisher: Dr. Binny Mathur
Sr. Technical Writer: Dr. Richa Sharma
Sr. Content Mgmt Analyst: Amit Rai
Asstt. Manager Manufacturing: Sumit Johry

10th Floor, Tower C
Building No. 10
Phase – II
DLF Cyber City
Gurgaon
Haryana - 122002

Twelfth Edition, 2010
Thirteenth Edition, 2014
Fourteenth edition, 2021

ISBN-13: 978-93-89859-92-8

Published by Wolters Kluwer (India) Pvt. Ltd., New Delhi
Compositor: Design Modus, New Delhi (www.designmodus.in)

For product enquiry, please contact – Marketing Department (marketing@wolterskluwerindia.co.in) or log on to our website www.wolterskluwerindia.co.in.

Cover page image: Micro CT image showing complex root canal anatomy of a mandibular first molar. (*Courtesy:* Prof. Marco A. Versiani, DDS, MSc, PhD)

This labor of love is dedicated to

Late Prof Dr. B. Suresh Chandra

for having the faith in me to carry forward the legacy of
this monumental textbook...

My Parents

Sulochana & Ambuja ... for all the love you bestow upon us...
M Velayutham ... for teaching me integrity and humility in life...
Late *VG Sivasubramanian* ... for showing me the path of caring and giving...

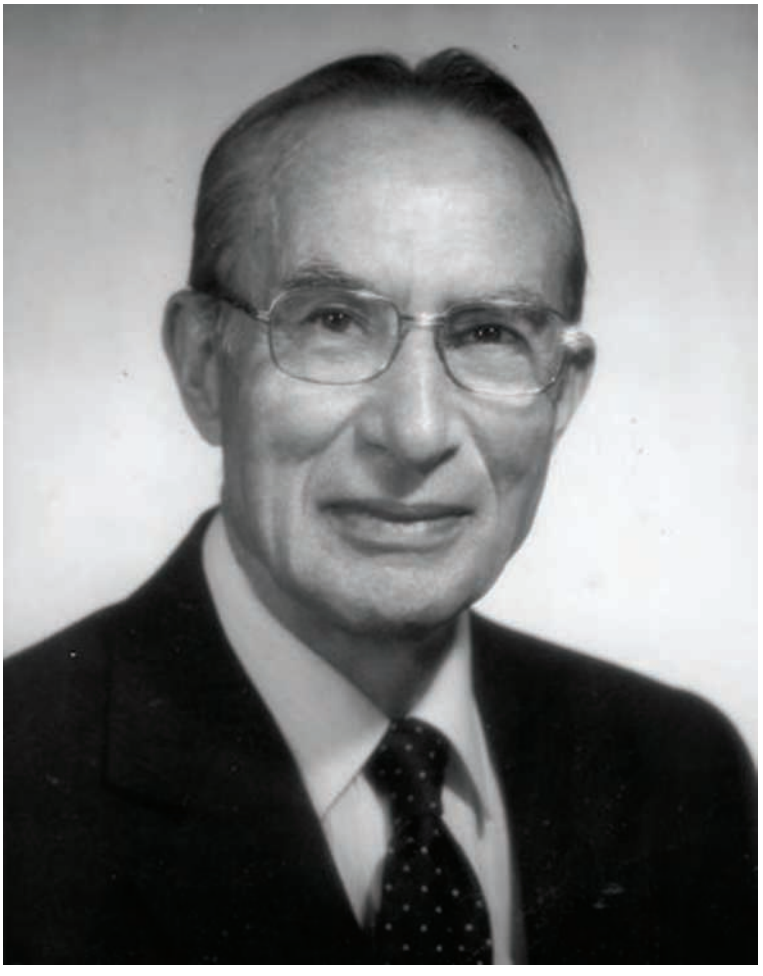
My Teachers

Dr. A Parameswaran ... for instilling in me the drive to learn, teach and research...
Dr. E. Munirathnam Naidu ... for teaching me the attributes of discipline,
hardwork & perseverance...
Dr. James "Jim" Gutmann ... for being my inspiration and all weather mentor...
Dr. K Sridhar ... for making me aware that water always finds its level...

My Family

Dr. Lakshmi S ... for being my ever-supportive and caring wife...
Sanjana & Sidharth ... for being our perennial source of happiness !!

—V. Gopikrishna



Dr. Louis I. Grossman (*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 Ukranian 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 *Handbook of Dental Practice*, published in 1948.

He was the 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 endodontists as well as other interested dentists to meet, share ideas, and expand and update their knowledge in the field of endodontics and dental medicine.

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. Gutmann

Preface to the Fourteenth Edition

It is much simpler to buy books than to read them
and easier to read them than to absorb their contents.

—Sir William Osler

It has been a great learning experience to have the opportunity to edit three editions of *Grossman's Endodontic Practice* over the past twelve years. I would be forever indebted to my teacher Dr. B Suresh Chandra who gave me the opportunity to be his co-editor for the previous two editions. The twelfth edition (2010) and thirteenth edition (2014) re-established this textbook as the premier teaching and clinical textbook for students across South Asia. Dr. Suresh Chandra's untimely demise leaves a great void in life and I have humbly tried my best to carry forward his and Grossman's legacy with this current fourteenth edition of *Grossman's Endodontic Practice*.

The ultimate goal of a textbook is in bringing clarity of thought and ease of understanding. As exemplified by the above given quote by Sir Osler, this textbook strives to bring complex concepts and techniques in a reader friendly manner for the student and clinician in you. I have added two new chapters in this edition—"Magnification in Endodontics" and "Nonsurgical Endodontic Retreatment" while comprehensively revising the remaining chapters to make the learning experience enjoyable to the student and clinician in you. This edition has more than 1,200 clinical images, x-rays, flowcharts, tables and boxes to make this a precise yet concise textbook cum reference guide for an astute practitioner of endodontics.

—V. Gopikrishna

Preface to the Thirteenth Edition

He who studies Medicine without books sails an uncharted sea,
but he who studies Medicine without patients does not go to sea at all.

—Sir William Osler

It has personally been an intellectual evolution in bringing out this thirteenth edition of the evergreen classic *Grossman's Endodontic Practice*. The process necessitates oneself to be a student in assimilating the sweeping changes that are happening in the specialty of endodontics. It was as much a learning and enriching process as it was enlightening.

The twelfth edition brought out by us in 2010 re-established this textbook as the premier teaching and clinical textbook for students across South Asia. The current edition builds up on this platform by updating and revising concepts, materials, and techniques. The increased awareness and research in biological concepts of treating the pulp tissue has made us revisit the chapter on vital pulp therapy, thereby updating it according to the current clinical guidelines. We have incorporated two new chapters into this edition: Chapter 7, *Endodontic Emergencies*, and Chapter 11, *Regenerative Endodontics*. We have also included *"Clinical Notes"* in each chapter that highlight the pertinent important clinical aspects of the topic being discussed. This book contains over 1100 figures, radiographs, and illustrations, many of which are contributions from clinicians and academicians from across the world. The format and style of presentation has also been changed to make it reader friendly. Accompanying the text is a *"Visual Masterclass" DVD* presenting videos of important clinical procedures.

We have strived to live up to the legacy of Louis I. Grossman by ensuring that this edition of *Grossman's Endodontic Practice* continues to be an evidence-based resource for students and practitioners in the field of endodontics.

B. Suresh Chandra • V. Gopikrishna

Preface to the 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 practice 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 practiced with greater care and thoroughness that 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, and 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 e4mphasized. 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 practicing 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 practices 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 advise, 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

"Thank you" are two little words which would probably never completely convey the sense of gratitude and regards which I have for each one of the following wonderful people who have made *Grossman's Endodontic Practice*, fourteenth edition, a reality.

I would 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, and Dr. E. Munirathnam Naidu.

I would like to specially thank two people who have been instrumental in my growth as an academician and a clinician: *James "Jim" Gutmann*, for being a perennial source of inspiration, motivation, and support in my academic endeavors; and *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 of you for accepting my invitation to contribute and for your kindness and generosity in sharing your knowledge and expertise.

I would like to compliment the wonderful team at Wolters Kluwer India for showing genuine passion and professionalism in giving life and body to this edition. Thank you Dr. Binny Mathur and team, Dr. Richa Sharma and Mr. Amit Rai, and Ms P. Sangeetha for your support.

Last but not the least, my indebtedness to my parents and family for being understanding and supportive during this journey.

V. Gopikrishna

The publisher thanks the following reviewers for providing valuable suggestions:

Faculty Reviewers: Deepika Chandhok, VSPM Dental College, Nagpur, Maharashtra; Dildeep Bali, Consultant Endodontist, New Delhi; Ekta Choudhary, Sharda University, Greater Noida; Jyoti Sachdeva, Fortis Memorial Research Institute, Gurugram; Pravin Kumar, All India Institute of Medical Sciences, Jodhpur; Shibani Grover, ESIC Dental College, Delhi; Vineeta Nikhil, Subharti Dental College, Meerut; Shalini Aggarwal, Dr. D. Y. Patil Dental College and Hospital, Pimpri; Jyoti Mandlik, Bharati Vidyapeeth (Deemed to be) University Dental College and Hospital, Pune; Kiran Keswani, Dr. D. Y. Patil Dental School, Lohegaon; Balaram Naik, SDM College of Dental Sciences and Hospital, Dharwad; Prahlad Saraf, PMNM Dental College and Hospital, Bagalkot, Karnataka; Vibha Hegde, G.D. Pol Foundations YMT Dental College & Hospital, Navi Mumbai; Leena Padhye, Dr. D.Y. Patil University School of Dentistry, Navi Mumbai; S. Sujatha Gopal, MNR Dental College, Sangareddy; Sita Rama Rao, Care Dental College, Guntur; Ch. N. V. Murali Krishna, Lenora Dental College, Rajahmundry; Zaheer Shaik, Care Dental College, Guntur; V. L. Deepa, Lenora Dental College, Rajahmundry; Anmol Bagaria, Mumbai.

Student Reviewers: Vishal Garg, Maulana Azad Institute of Dental Sciences, New Delhi; Shreya Aggarwal, Intern, ITS Dental College, Ghaziabad.

Contributors

The Editor, Dr. V. Gopikrishna acknowledges the following contributors for sharing their valuable case reports, inputs and clinical images.

AUSTRALIA

- **Geoff Young**, *BDS (Syd.), DCD (Melb.)*
  University of Melbourne
- **Peter Parashos**, *BDSC, LDS, MDSc, FRACDS*

BRAZIL

- **Alessandra Sverberi Carvalho**
  São Paulo State University
- **Carlos Estrela**, *DDS, MSc, PhD*
  Federal University of Goiás
- **Carlos Jose Soares**
  Federal University of Uberlândia
- **Jesus D. Pecora**, *DDS, MSc, PhD*
  University of Sao Paulo
- **Manoel D. Sousa-Neto**, *DDS, MSc, PhD*
  University of Sao Paulo
- **Marco A. Versiani**, *DDS, MSc, PhD*
  University of Sao Paulo

CANADA

- **Anil Kishen**, *MDS, PhD*
  University of Toronto

CHINA

- **Bing Fan**, *DDS, PhD*
  University of Wuhan

ENGLAND

- **Julian Webber**, *BDS, MSc, DGDP, FICD*
  The Harley Street Centre For Endodontics

EGYPT

- **Ahmed Ibrahim Salim**
  Private Practice

FRANCE

- **Wilhem J. Pertot**, *DDS*
  Private Practice

GREECE

- **Antonis Chaniotis**, *DDS, MDSc*
  Private Practice

GERMANY

- **Domonkos Horvath**, *Dr. Med. Dent*
  University Hospital Freiburg
- **Sebastian Horvath**, *Dr. Med. Dent*
  University Hospital Freiburg
- **Till Dammaschke**, *Prof. Dr. Med. Dent*
  Westphalian Wilhelms-University

INDIA

- **A.R. Pradeep Kumar**, *MDS, FDSRCS*
  Thai Moogambigai Dental College
- **Abarajithan**, *MDS*
  Private Practice
- **Ahendita Bhowmik**, *MDS*
  Private Practice
- **Arathi Ganesh**, *MDS*
  Sri Ramachandra Institute of Higher Education & Research
- **Arvind Shenoy**, *MDS*
- **B. Sivapathasundharam**, *MDS*
  Meenakshi Ammal Dental College
- **C. Praveen Kumar**, *MDS*
  Private Practice
- **Gnanavi**, *BDS*
  Private Practitioner
- **Hannah Rosaline**, *MDS*
  Private Practitioner
- **Harsh Vyas**, *MDS*
  Paediatric Dentist
- **Hemalatha Hiremath**, *MDS*
  Sri Aurobindo College of Dentistry

- **K. Manjunath**, *MDS*
  Meenakshi Ammal Dental College
- **Krithika Datta**, *MDS*
  Saveetha Dental College
- **Mahima Tilakchand**, *MDS*
  SDM College of Dental Sciences & Hospital
- **Nandini S.**, *MDS*
  Meenakshi Ammal Dental College
- **Priya Ramani**, *MDS*
  Thai Moogambigai Dental College
- **Priyanka Ashok**, *MDS*
  Private Practice
- **Ravi Shankar**, *MDS*
  Thai Moogambigai Dental College
- **Reuben Joseph**, *MDS*
  Private Practice
- **Roheet Khatavkar**, *MDS*
  Private Practice
- **S. Karthiga Kannan**, *MDS*
- **Sanjay Miglani**, *MDS*
  Jamia Millia Islamia
- **Siju Jacob**, *MDS*
  Private Practice
- **T. Sarumathi**, *MDS*
  Adhiparasakthi Dental College and Hospital
- **Tarek Frank Fessali**
- **Vivek Hegde**, *MDS*
  Rangoonwala Dental College

IRAN

- **Saeed Asgary**, *DDS, MS*
  Shahid Beheshti University of Medical Sciences

ISRAEL

- **Zvi Metzger**, *DMD*
  Tel Aviv University

ITALY

- **Arnaldo Castellucci**, *MD, DDS*
  Private Practice
- **Filippo Cardinali**
  Private Practice
- **Gianluca Plotino**, *DDS, PhD*
  University of Rome

JAMAICA

- **Sashi Nallapati**, *BDS*
  Cert. Endo, Private Practice & Nova Southeastern University

NETHERLANDS

- **Niek Opdam**
  Radboud University

NEW ZEALAND

- **Venkat Canakapalli**, *MDS*
  Private Practice

NORWAY

- **Mathias Nordvi**
  University of Oslo
- **Randi F. Klinge**
  University of Oslo

SWITZERLAND

- **Frank Paque**, *DMD*
  Univeristy of Zurich
- **P.N.R. Nair**, *BVSc, DVM, PhD (Hon.)*
  University of Zurich

THAILAND

- **Jeeraphat Jantarat**, *DDS, MS, PhD*
  Mahidol University

UNITED STATES OF AMERICA

- **Bekir Karabucak**, *DMD, MS*
  University of Pennsylvania
- **Clifford J. Ruddle**, *DDS*
  Advanced Endodontics
- **Dean Baugh**, *DDS*
  Private Practice
- **J.M. Brady**
  Private Practice
- **James L. Gutmann**, *DDS, PhD (Honoris Causa)*
  Cert. Endo, FACD, FICD, FADI
- **Jason J. Hales**, *DDS, MS*
  Private Practice
- **Louis H. Berman**, *DDS, FACD*
  Private Practice
- **Martin S. Spiller**, *DMD*
  Private Practice
- **Meetu Kohli**, *DMD*
  University of Pennsylvania
- **Samuel I. Kratchman**, *DMD*
  University of Pennsylvania
- **Syngcuk Kim**, *DDS, PhD, MD (Hon.)*
  University of Pennsylvania

UKRAINE

- **Volodomyr Kovtun**
  Private Practice

Contents

LIST OF ONLINE VIDEOS

1. Vitality assessment using an Electric Pulp Tester
2. Rubber dam placement
3. Access opening of a maxillary molar
4. Access opening of a maxillary premolar
5. Access opening of a mandibular molar
6. Working length measurement using an electronic apex locator
7. Shaping protocol using rotary Ni-Ti files
8. Removal of pulp stones using ultrasonic tips
9. Activation of NaOCl irrigation
10. Activation of intracanal $Ca(OH)_2$ medicament

1 The Dental Pulp and Periradicular Tissues

The beginning of all things is small....

PART 1: EMBRYOLOGY

The pulp and dentin are different components of a tooth that 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 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 form 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 into ameloblasts. A distinct basement membrane separates the outer and the inner enamel epithelium from the ectomesenchyme. In the region of the inner enamel epithelium, a cell-free or acellular zone also separates the enamel organ from the ectomesenchyme. This acellular zone contains the extracellular matrix, where the future predentin will be deposited. Between the inner and the outer enamel epithelium, the cells begin to separate due to the deposition of intercellular mucoid fluid rich in glycogen that forms a branch reticular arrangement called the *stellate reticulum*.

FORMATION OF DENTAL PAPILLA

The ectomesenchyme, which is partially enclosed by the inner enamel epithelium, continues to increase its cellular density. The cells are large and round or polyhedral with a pale cytoplasm and large nuclei. This structure is the *dental papilla* (**Fig. 1.6**) which differentiates into the dental pulp.

FORMATION OF DENTAL FOLLICLE (OR DENTAL SAC)

When the ectomesenchyme surrounding the dental papilla and the enamel organ condenses and becomes more fibrous, it is called the *dental follicle* or the *dental sac*—the precursor of the cementum, the periodontal ligament (PDL), and the alveolar bone (**Fig. 1.6**). The dental lamina continues to proliferate at the point where it joins the deciduous enamel organ and thereby produces the permanent bud lingual to the primary tooth germ.

BELL STAGE (CERVICAL LOOP)

The cells of the inner enamel epithelium continue to divide and thus increase the size of the tooth germ. During this

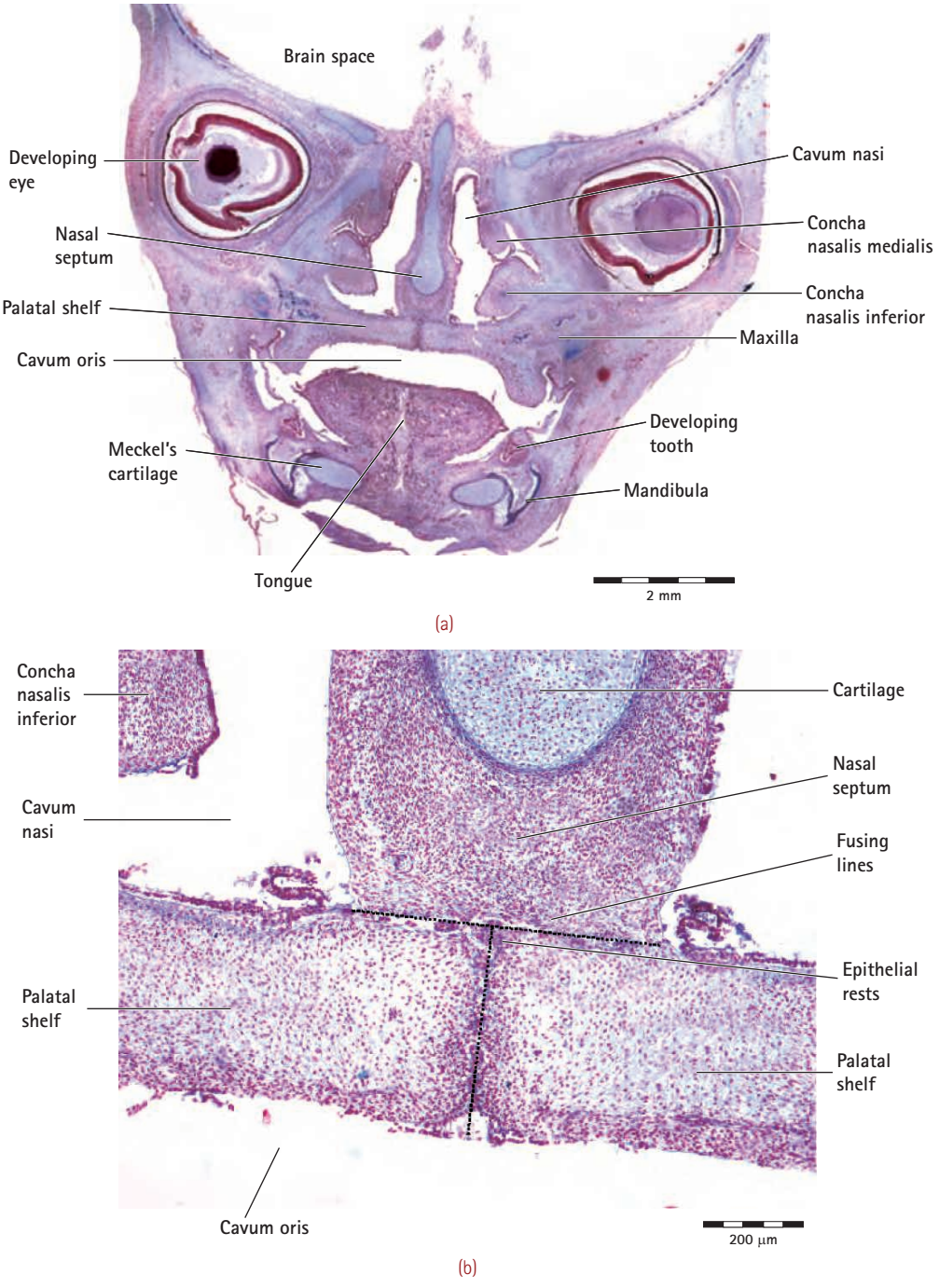


Figure 1.1 (a) Human fetus, head. This is a frontal section of the head of a human fetus. You can see the maxilla and the mandible taking shape. You can also see Meckel's cartilage in the mandible. The mandible also contains two dental buds in this section (stain: Azan). (b) At higher magnification, 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.*)

growth, the inner enamel epithelium invaginates deeper into the enamel organ, and the junction of the outer and the inner enamel epithelium at the rim of the enamel organ becomes a distinct zone called the *cervical loop*. The deep invagination of the inner enamel epithelium and the growth of the cervical loop partially enclosing the dental papilla begins to give the crown its form. This point is called the bell stage of development (**Fig. 1.7**).

During this stage, the dental lamina that migrated into the ectomesenchyme degenerates, the primary and permanent buds are thus separated from the oral epithelium, and the distal portion of the dental lamina proliferates to form

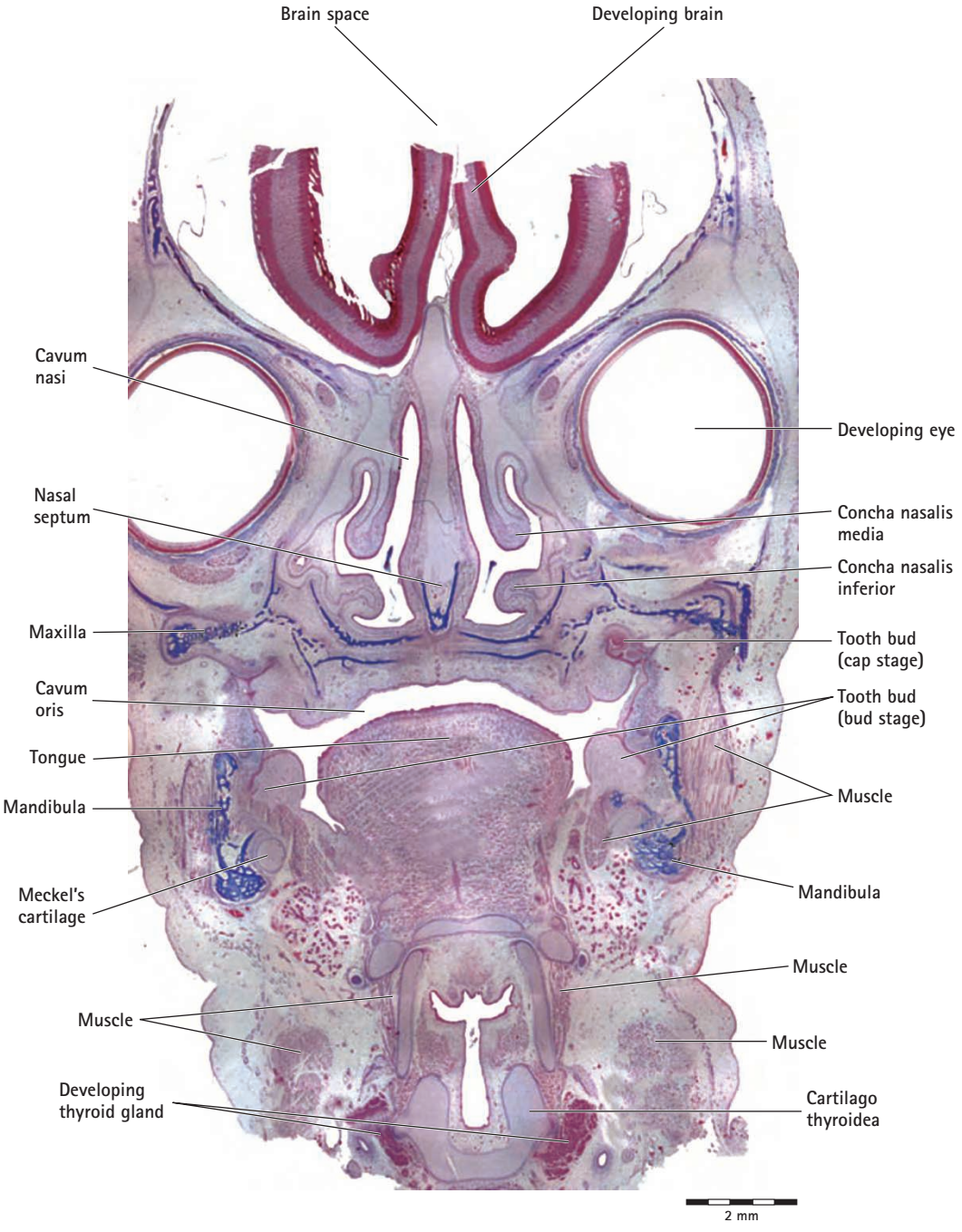


Figure 1.2 Human fetus, head. This is a frontal section of the head of a human fetus. The nasal cavity (Latin *cavum nasi*) is divided into two by the nasal cartilage within the nasal septum. At both sides of the septum, you can see the nasal conchae (Latin *concha nasalis media et inferior*). They are 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.*)

the buds of the permanent molars, which have no primary predecessors.

As the development progresses, several layers of the squamous cells between the stellate reticulum and the inner enamel epithelium form the *stratum intermedium*. This layer of cells is limited to the area of the inner enamel epithelium and seems to be involved with enamel formation.

Clinical Note

- *Stratum intermedium* → Enamel
- *Ectomesenchyme* → Dentin
- *Dental papilla* → Pulp
- *Dental follicle* or *dental sac* → Cementum, the periodontal ligament (PDL), and the alveolar bone

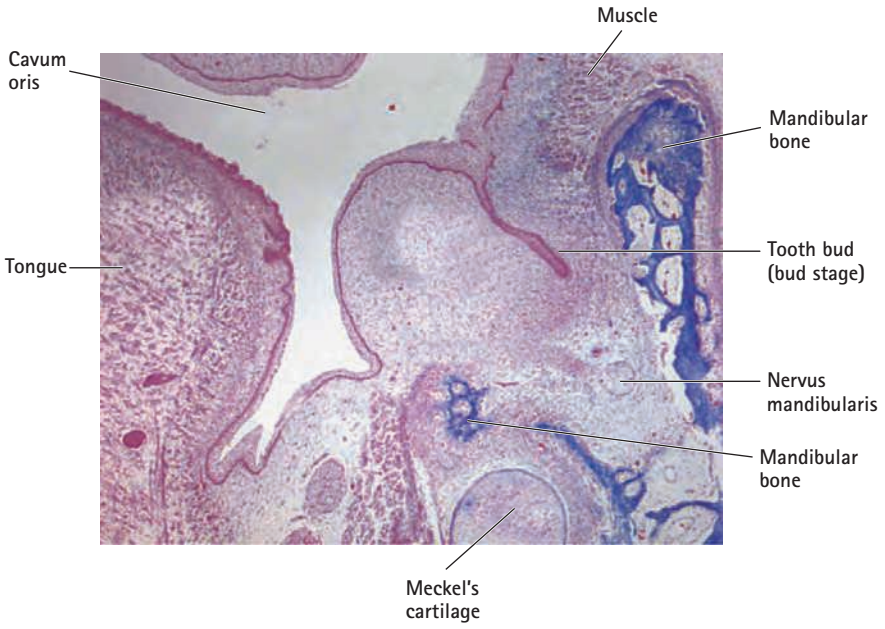


Figure 1.3 Dental lamina with its tooth bud. Around the bud, the mesenchyme is condensated. Just below the tooth bud in the mandible, you can see the alveolar nerve (Latin *n. alveolaris*). Meckel's cartilage can also easily be spotted. The tongue is also developing. It consists of muscular fibers oriented in different directions. At both sides of the tongue, you can see salivary glands. Cartilage comprising parts of the larynx can be seen below the tongue. (*Courtesy: Mathias Nordvi, University of Oslo, Norway.*)

Clinical Note

Primary dentin is formed in increments of *4–8 µm per day* and is continually deposited until the end of tooth development.

DENTINOGENESIS

In a complex series of events, the inner enamel epithelium exerts an inductive influence on the ectomesenchyme to begin dentinogenesis, and consequently, dentinogenesis has an inductive influence on the inner enamel epithelium to start amelogenesis. This series of events begins in the area of the future cusp tips and continues to the cervical loop, the future cementoenamel junction.

PREODONTOBLASTS

The periphery of the adjacent dental papilla consists of the polymorphic mesenchymal cells that develop into the cuboidal cells and, align themselves parallel to the basement membrane of the inner enamel epithelium and the acellular zone. These cuboidal cells stop dividing and develop into the columnar cells with polarized nuclei away from the basement membrane of the inner enamel epithelium. At this stage, these cells are called *preodontoblasts*.

MANTLE DENTIN FORMATION

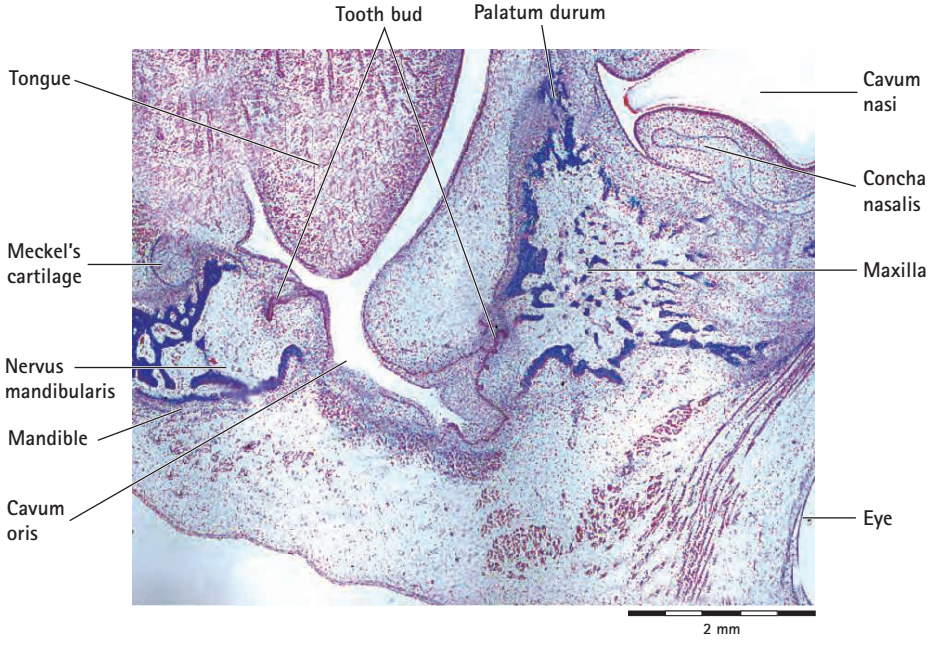
The preodontoblasts mature into odontoblasts by elongating themselves, by contacting adjacent odontoblasts through an increase in size, and by sending the cytoplasmic processes into the acellular zone. These odontoblastic processes continue to elongate and move the odontoblast cell body toward the center of the dental papilla. During this movement, large-diameter collagen fibers known as von Korff fibers are deposited at right angles to the basement membrane in the extracellular matrix of the acellular zone. This process creates the organic matrix of the first-formed dentin or *mantle dentin*. As more collagen fibrils are deposited, the inner enamel epithelium basement membrane starts to disintegrate. The vesicles carrying apatite crystals bud off from the odontoblastic processes and the crystals are deposited in the organic matrix for the initiation of mineralization. The dental papilla becomes the pulp at the moment of the mantle dentin formation.

PRIMARY DENTIN

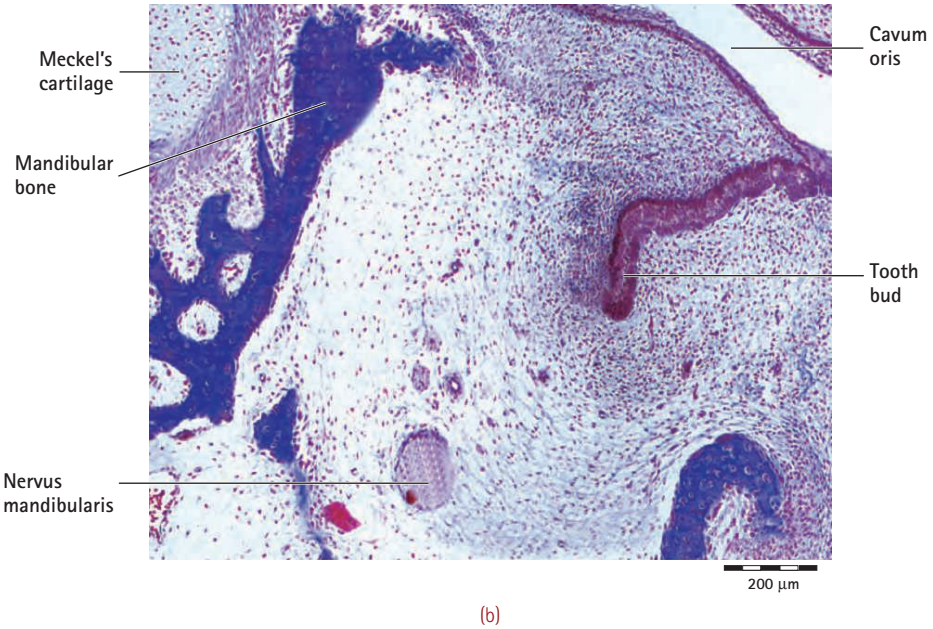
After the deposition of mantle dentin, the odontoblasts continue to move toward the center of the pulp and to leave the odontoblastic processes behind. The organic matrix or *predentin* (**Fig. 1.8a** and **1.8b**) is deposited around the odontoblastic processes. The predentin later calcifies and thereby forms the dentinal tubules. Primary dentin differs from the mantle dentin in which the matrix originates solely in the odontoblasts. The collagen fibers are smaller, are more closely packed, are at right angles to the tubules, and are interwoven. The mineralization of primary dentin originates from the previous mineralized dentin.

PERITUBULAR DENTIN

As the incremental deposition of dentin continues toward the center of the pulp, the diameter of the odontoblastic processes is reduced peripherally. Along with this, there is a reduction in size due to the circumferential deposition of dentin in the walls of the dentinal tubules. This dentin, which is more mineralized and is harder than primary dentin, is called *peritubular dentin*.



(a)



(b)

Figure 1.4 Tooth development, bud stage: (a and b) This is a frontal section of the head of a human fetus (tilted 90° to the right). The bone has started to develop in the maxilla as well as in the mandible. Because of the stain used to color this tissue sample, the bone has a blue color. Within the two quadrants seen here, there are dental laminae, and encircling these laminae, a condensation of the mesenchyme takes place. In between the spicules of bone in the mandible, you can see a cross-section of the alveolar nerve (Latin *n. alveolaris inferior*). Meckel's cartilage is situated medially to the mandibular bone. If you look closely, you can see the downgrowth of the parenchyma of the salivary glands and the developing muscular fibers of the tongue. (*Courtesy: Mathias Nordvi, University of Oslo, Norway.*) *(continued)*

II. CHEMICALS

Chemical causes of pulp injury are probably the least common.

The key factors which determine the pulpal reaction to a restorative filling material are as follows:

- Acidity (pH of the material)
- Heat generated during the setting reaction
- Absorption of water during the setting reaction
- Remaining dentin thickness (RDT)
- Poor marginal adaptation of the material which might contribute to bacterial leakage

Fillings made of silver amalgam, or composites, may produce some pulpal reaction when they are inserted in cavities with very less RDT. The deeper the cavity, the greater the damage caused, but in most cases the pulp recovers from these injuries.

Clinical Note

The long-term prognosis of a restorative filling would be determined by its ability to inhibit microleakage and pulpal bacterial contamination.

III. BACTERIA

In 1894, W.D. Miller suggested that bacteria were a possible cause of inflammation in the pulp. *The most common cause of pulp injury is bacterial.* Bacteria or their products may enter the pulp through a break in the dentin, from caries or accidental exposure, from developmental grooves (**Figs 4.9** and **4.10**), from percolation around a restoration, from extension of infection, from the gingiva, or by way of the blood.

Microorganisms play an important role in the genesis of pulpal disease (**Fig. 4.11**). Despite food impaction, dentinal bridging occurs in the pulps of gnotobiotic (germ-free) rats after pulp exposure. On the other hand, pulpal necrosis, abscess formation, and granulomas develop in exposed pulps of rats kept under ordinary laboratory conditions.

The species of bacteria recovered from inflamed or infected pulps are many and varied. Although lactobacilli (acidogenic organisms) are commonly found in carious dentin, they are seldom recovered from the pulp because of their low degree of invasiveness. Microorganisms need not be present in the pulp to produce inflammation: the by-products of bacteria in the dentin may be sufficiently irritating to cause an inflammatory reaction.

The bacteria most often recovered from infected vital pulps are *streptococci* and *staphylococci*, but many other microorganisms including anaerobes have also been isolated. With the introduction of improved methods of molecular identification of pathogens, many new organisms have been identified by researchers. Species that have been found significantly include *Porphyromonas gingivalis*, *Porphyromonas endodontalis*, *Fusobacterium nucleatum*, and others. Polymerase chain reaction (PCR) methods show higher prevalence of

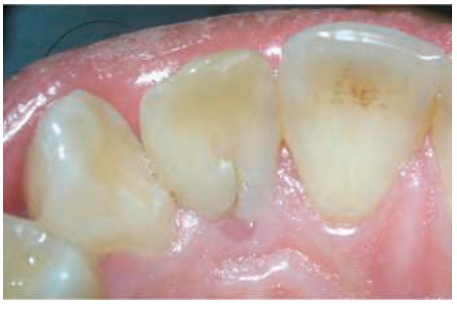


Figure 4.9 Developmental palatogingival groove in maxillary lateral incisor acts as a pathway for carious invasion of the pulp space.

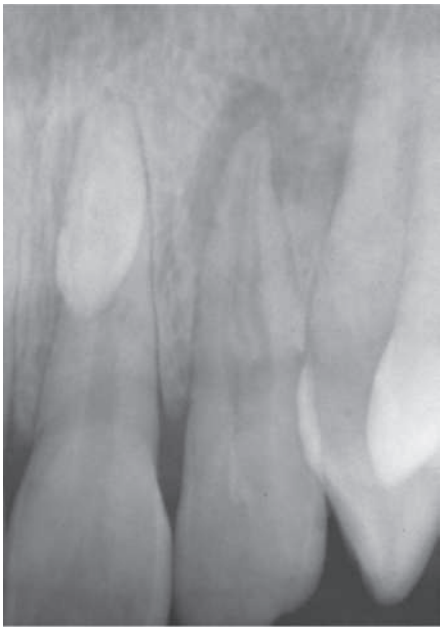


Figure 4.10 Radiographic evidence of pulpal involvement along with periradicular changes.

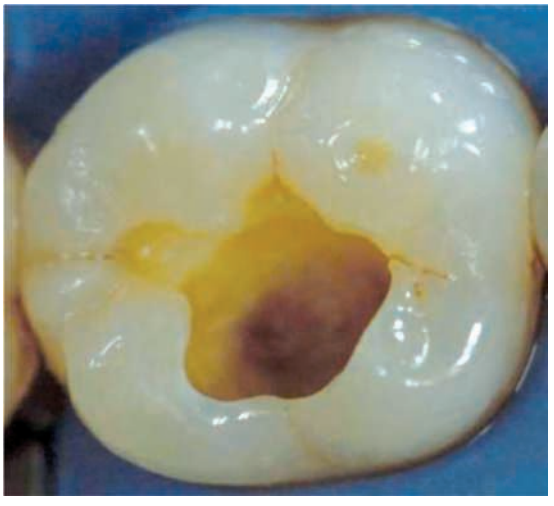


Figure 4.11 Mandibular molar with clinical evidence of carious pulpal exposure.

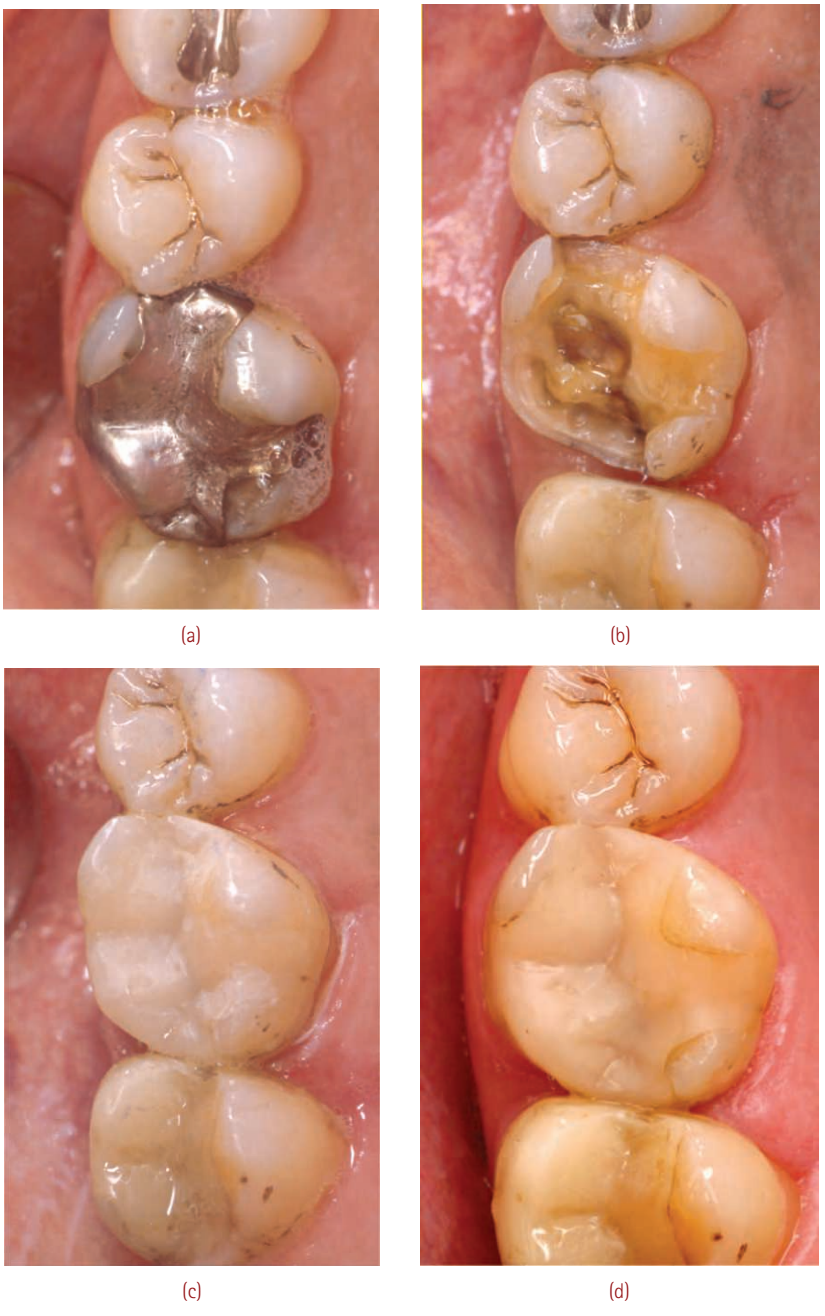


Figure 4.13 Upper molar with clinically good amalgam restoration: (a) Tooth was symptomatic for biting forces and cold application. (b) After amalgam removal. (c) After restoration with direct composite and total etch technique. (d) Symptomless, vital tooth after 5 years. *(Courtesy: Niek Opdam, Netherlands.)*

Cause

The most common cause of irreversible pulpitis is bacterial involvement of the pulp through caries, although any clinical factor, chemical, thermal, or mechanical, already mentioned as a cause of pulp disease, may also cause pulpitis. As previously stated, reversible pulpitis may deteriorate into irreversible pulpitis.

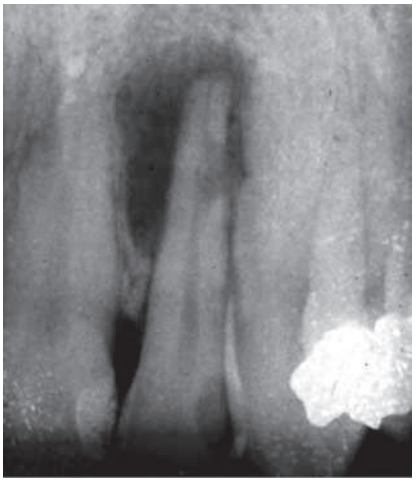
Symptoms

- In the early stages of irreversible pulpitis, a paroxysm of pain may be caused by the following: sudden temperature changes, particularly cold; sweet or acid foodstuffs; and pressure from packing food into a cavity or suction exerted by the tongue or cheek.
- Symptomatic irreversible pulpitis exhibits pain usually caused by a hot or cold stimulus, or pain that occurs

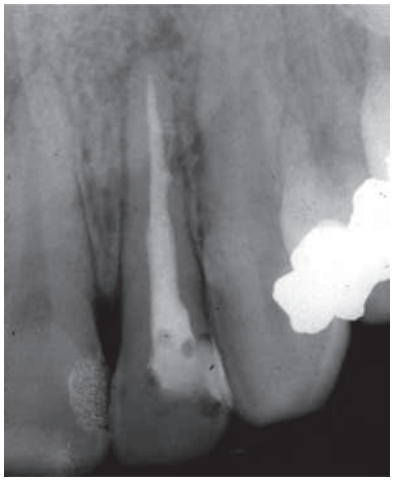
angles may help to resolve the question. When bone adjacent to the area of resorption is involved and the resorbed area is externally concave and when the root canal is intact, as seen in the radiograph, external resorption is present.

Treatment

Internal resorption ceases when the pulp is removed or becomes necrotic. Root canal therapy is the treatment of choice (**Fig. 5.24**). The treatment of external resorption varies with the etiologic factor. If external resorption is caused by extension of pulpal disease into the supporting tissues, root canal therapy will usually stop the resorptive process. External resorption produced by excessive forces from orthodontic appliances can be stopped by reducing those forces. In cases of external cervical root resorption, intervention in the form of surgical exposure of the defect and restoration with a suitable restorative material is the treatment of choice before the resorptive defect invades the pulp space (**Figs 5.25** and **5.26**).

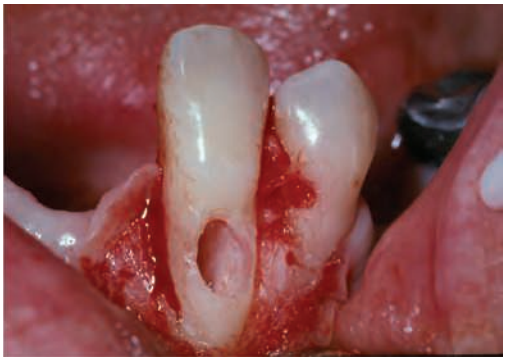


(a)

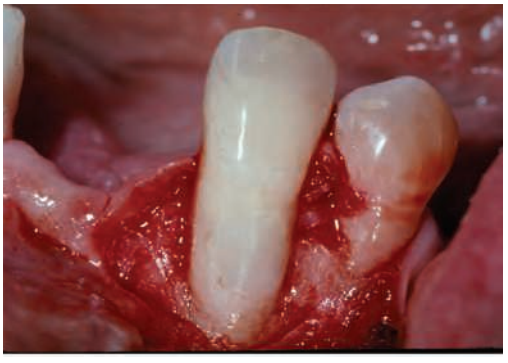


(b)

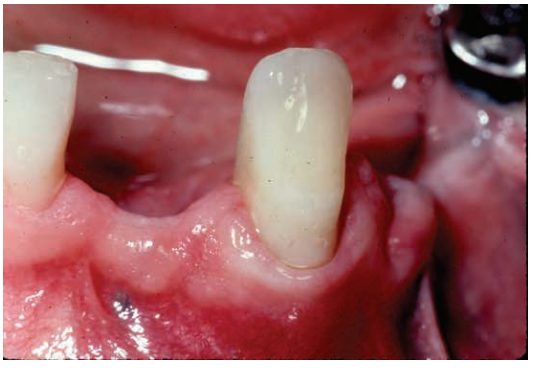
Figure 5.24 (a) Maxillary lateral incisor: pulp is necrotic and there is a perforating internal resorptive defect in the apical one-third of the root. (b) Re-examination 2 years later after root canal treatment shows healing of the resorptive defect. (*Courtesy: James L. Gutmann, USA.*)



(a)



(b)



(c)

Figure 5.25 External inflammatory cervical resorption: (a) Surgical isolation and preparation of an external cervical resorptive defect. (b) Placement of a bonded compomer. (c) One-month healing of the tissue around the restored defect. (*Courtesy: James L. Gutmann, USA.*)

Prognosis

The prognosis of a tooth with external resorption is *guarded*. If the etiologic factor is known and it is removed, the resorptive process will stop, but it may leave a weak tooth unable to sustain functional forces. In some cases, regardless of treatment, the tooth is lost. The differences between external and internal resorption are listed in **Table 5.2**.

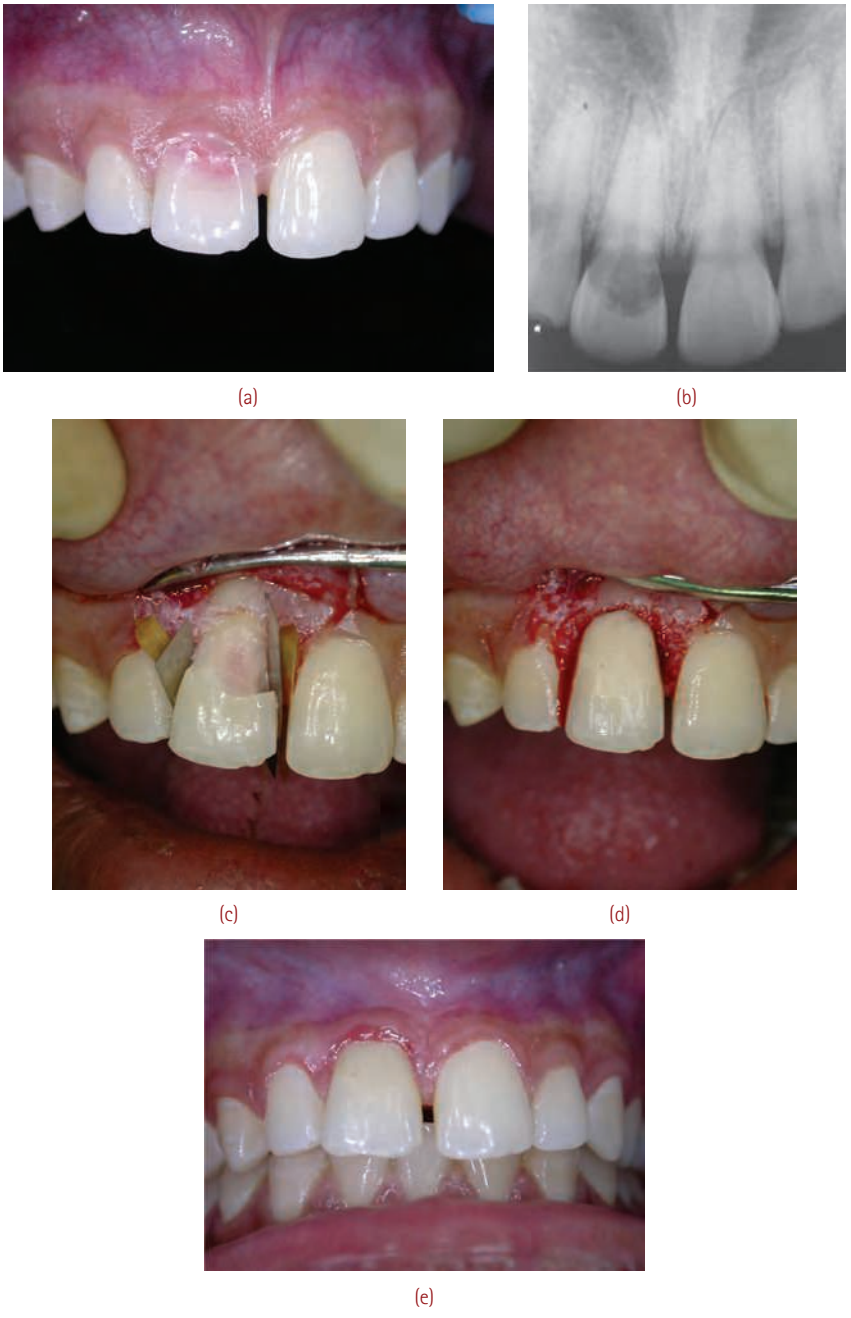


Figure 5.26 External inflammatory cervical root resorption: (a) Maxillary central incisor with enlarged marginal gingiva and break in the facial cervical enamel margin. (b) Characteristic cervical radiolucency seen. (c) Wedges and matrix strip shown before placement of restorative material. (d) Labial view showing glass ionomer restoration of the defect. (e) Labial view at 2 months after composite resin placement over glass ionomer. (*Courtesy: Hemalatha Hiremath, India.*)

V. DISEASES OF THE PERIRADICULAR TISSUES OF NONENDODONTIC ORIGIN

Periradicular lesions not only arise as extensions of pulpal diseases but may also originate in the remnants of odontogenic epithelium. Such lesions may be manifestations of systemic diseases, such as multiple neurofibromatosis, or they may have other causes, such as periodontal diseases.

Some of these periradicular lesions, radiographically and clinically, resemble the sequelae of pulpal diseases in the periradicular area and should be differentiated from them to avoid errors in treatment.

Lesions of nonendodontic origin with vital pulps include:

- Periapical cemental dysplasia or cementoma
- Cementoblastoma (**Fig. 5.27**)

- *Arginine* is being incorporated in a number of recently developed toothpastes (Colgate Sensitive Pro-Relief, Colgate-Palmolive Company, New York). The combination of arginine with calcium carbonate and phosphate results in precipitate formation on the open dentinal tubules. In addition to toothpastes, it is available in several forms such as:
  - Arginine in-office paste (8%)
  - Arginine mouthwash (0.8%)
- *Calcium sodium phosphosilicate (CSPS): NovaMin* is the trade name for a CSPS bioactive glass. It acts by occlusion of dentinal tubules by precipitating calcium and phosphate. Calcium and phosphate ions react with oral fluids to form hydroxycarbonate apatite layer that is similar to natural tooth structure. It is available in the form of toothpaste (5% CSPS) and prophylaxis paste (15% CSPS), e.g., Sensodyne Repair and Protect.

C. Dentin Adhesives

The use of dentin adhesives to treat hypersensitive dentinal surfaces has gained popularity. Reductions in sensitivity can result from the formation of resin tags and a hybrid layer when a dentin adhesive is used.

OBJECTIVE SYMPTOMS

Objective symptoms are determined by tests and observations performed by the clinician. These tests are provided in **Box 6.6**.

Although it may not be necessary to perform all these tests at any one time, a combination of corroborating tests is desirable to ensure a correct diagnosis. One should not rely on the results of any single test.

I. Visual and Tactile Inspection

The simplest clinical test is visual examination. Too often, it is done casually during examination, and as a result, much essential information is lost inadvertently. A thorough visual and tactile examination of hard and soft tissue relies on checking the "three Cs": ***color***, ***contour***, *and* ***consistency***.

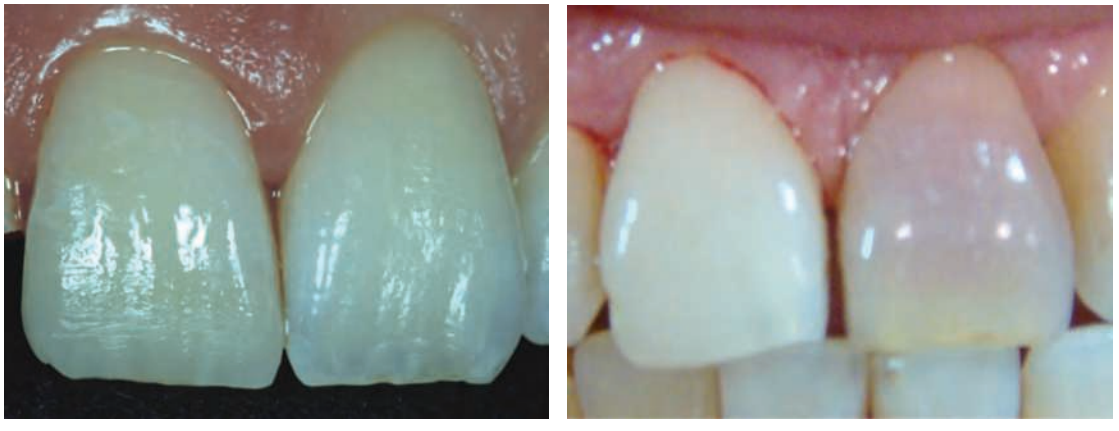
Box 6.6

Diagnostic Methods in Endodontics

1. Visual and tactile inspection
   (a) Hard tissues
   (b) Soft tissues
      (i) Gingiva
      (ii) Periodontium
2. Percussion
3. Palpation
4. Mobility and depressibility
5. Bite test
6. Radiography
   (a) Intraoral periapical radiographs
   (b) Bitewing radiographs
   (c) Digital radiography
   (d) Cone beam computed tomography (CBCT)
7. Assessment of pulp vitality
   (a) Neural sensibility tests
      (i) Thermal tests
      (ii) Heat testing
      (iii) Cold testing
      (iv) Electric pulp test (EPT)
      (v) Anesthetic test
      (vi) Test cavity
   (b) Pulp vascularity tests
      (i) Pulse oximetry
      (ii) Laser Doppler flowmetry
      (iii) Recent technologies
         - Dual-wavelength spectrophotometry
         - Thermography
         - Crown surface temperature
         - Transmitted light photoplethysmography

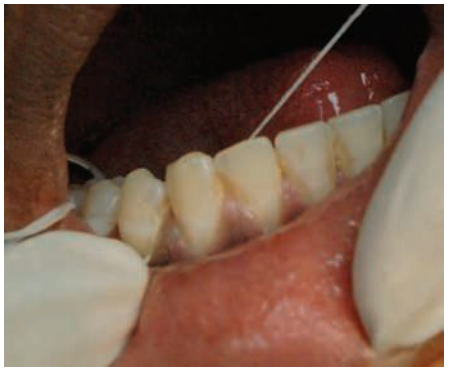
A. Hard Tissues

Teeth should be visually examined using the "three Cs." A normal-appearing crown has a life-like translucency and sparkle (**Fig. 6.4a**) that is missing in pulpless teeth (**Fig. 6.4b**). Teeth that are discolored, opaque, and less life-like in appearance should be carefully evaluated because the pulp may already be inflamed, degenerated, or necrotic. Not

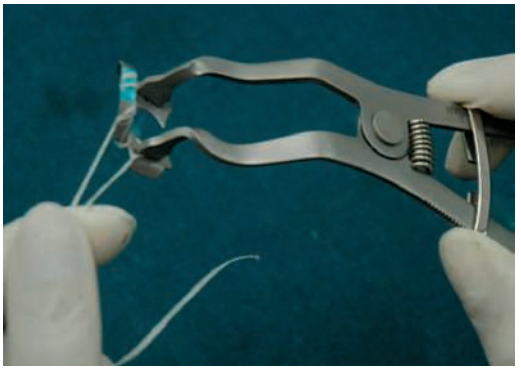


(a) (b)

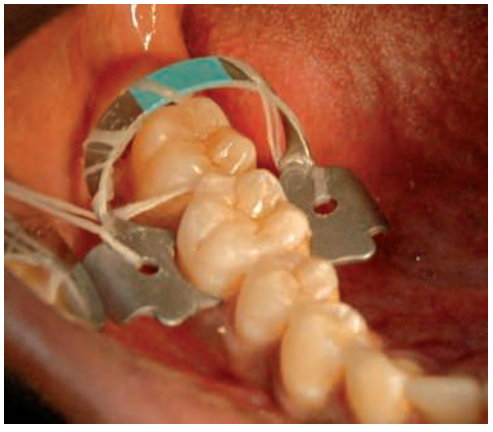
Figure 6.4 (a) Translucency and appearance of a normal vital tooth. (b) Loss of translucency and change of color in a nonvital tooth.



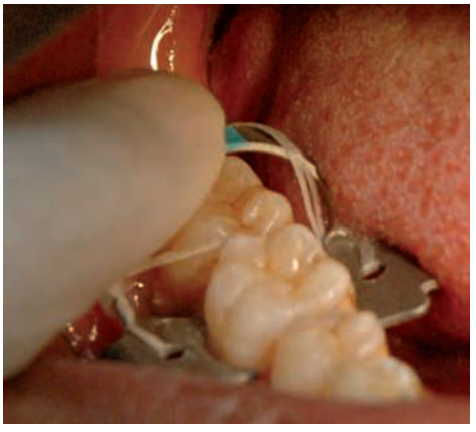
(a)



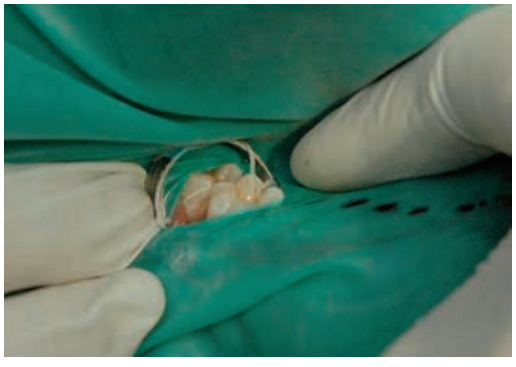
(b)



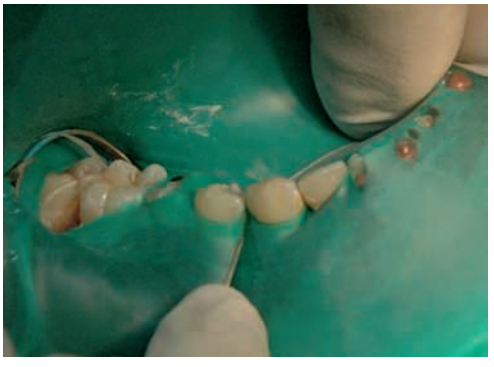
(c)



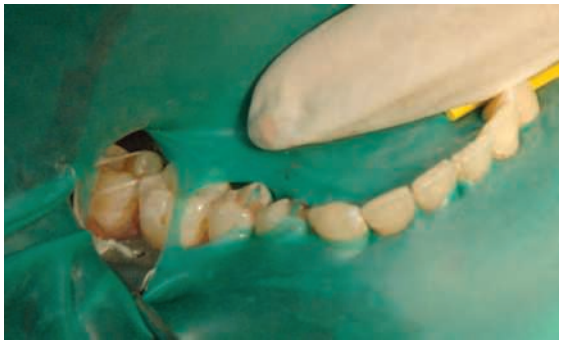
(d)



(e)



(f)



(g)

Figure 10.12 (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) The stability of the dam is checked with the index finger on the bow of the clamp. The clamp should not rock. (e) The dam is first passed over the bow of the clamp. (f) The dam is stretched over the teeth and passed below the contact area with the help of dental floss. (g) After securing the dam on the anterior anchor, the dam is carefully passed around the clamp. *(continued)*

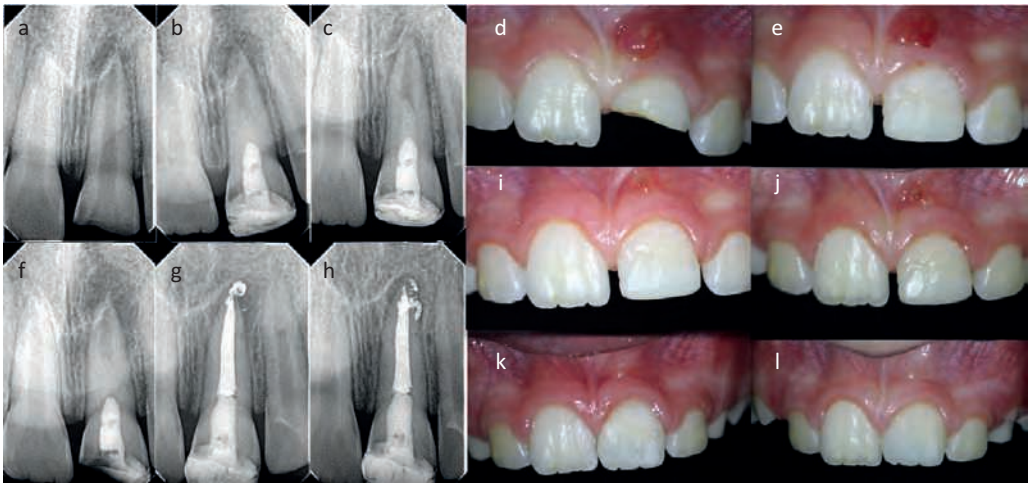


Figure 12.5 (a) Preoperative periapical radiograph of maxillary central incisor showing periapical lesion growing in the mesial side of the periapex and incomplete apical closure. (b) Postoperative periapical radiograph after single-step regenerative endodontic procedures. (c) Six-month follow-up radiograph demonstrating partial healing of the periapical lesion and signs of apical closure and dentinal wall thickening. (d) Preoperative clinical image showing a buccal swelling and fractured crown. (e) Postoperative clinical image after single-visit regenerative procedure and composite resin restoration. (f) Thirteen-month follow-up radiograph showing recurrence of the periapical disease, fracture of the coronal restoration, and convergent apical dentinal walls. (g) Postoperative radiograph after conventional retreatment procedures revealing a lateral canal and slight material extrusion. (h) Three years after the initial treatment follow-up radiograph suggesting almost complete healing of the periapical lesion. (i) Six-month clinical image demonstrating resolution of the initial buccal swelling and no discoloration. (j) Twelve-month clinical image demonstrating the reappearance of the sinus tract. (k) Postoperative clinical image after conventional retreatment procedures and composite resin restoration repair. (l) Three-year clinical image after the initial regenerative effort showing healthy soft tissues and no discoloration. (*Courtesy: Antonis Chaniotis, Greece.*)

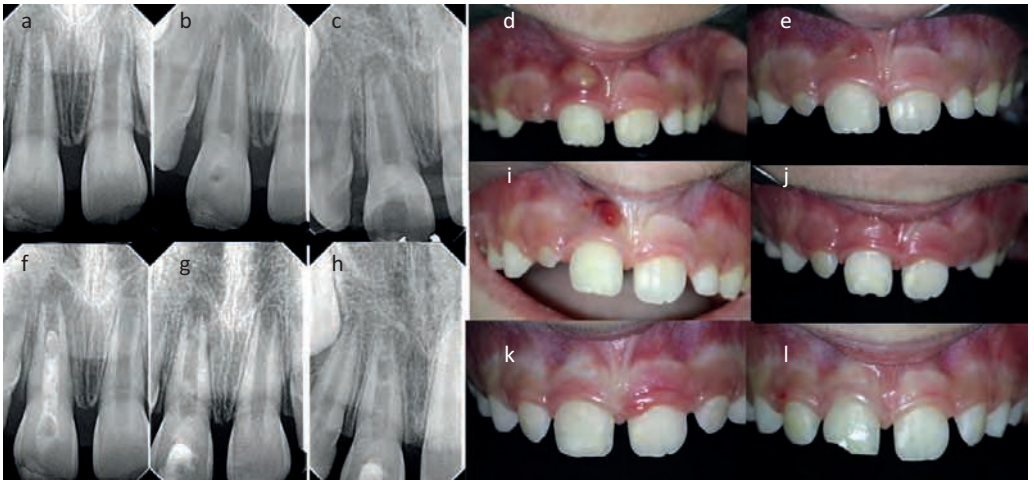


Figure 12.6 (a) Preoperative periapical radiograph of maxillary central incisor showing thin dentinal walls and periapical lesion. (b) Postoperative radiograph after single-step regenerative procedures with Biodentine coronal plug. (c) Six-month follow-up periapical radiograph showing unaltered dentinal walls and existence of periapical lesion. (d) Preoperative buccal clinical view showing buccal swelling. (e) Three-month buccal clinical view showing initial resolution of the buccal swelling. (f) Six-month follow-up radiograph after retreatment procedures and calcium hydroxide placement showing periapical lesion and unaltered walls. (g) One-year follow-up radiograph after removal of calcium hydroxide and repetition of the regenerative endodontic procedure showing unaltered walls and partial healing of the periapical lesion. (h) Three-year follow-up radiograph revealing healing of the periapical lesion, dentinal wall thickening, apical closure, and a hard tissue bridge at the middle third of the canal. (i) Six-month buccal clinical view demonstrating the reappearance of the sinus tract. (j) Twelve-month buccal clinical view showing resolution of the sinus tract after calcium hydroxide placement. (k) Twelve-month buccal clinical view after repetition of the regenerative endodontic procedure and composite resin restoration. (l) Three-year follow-up buccal clinical view after the repetition of the regenerative endodontic procedure. (*Courtesy: Antonis Chaniotis, Greece.*)

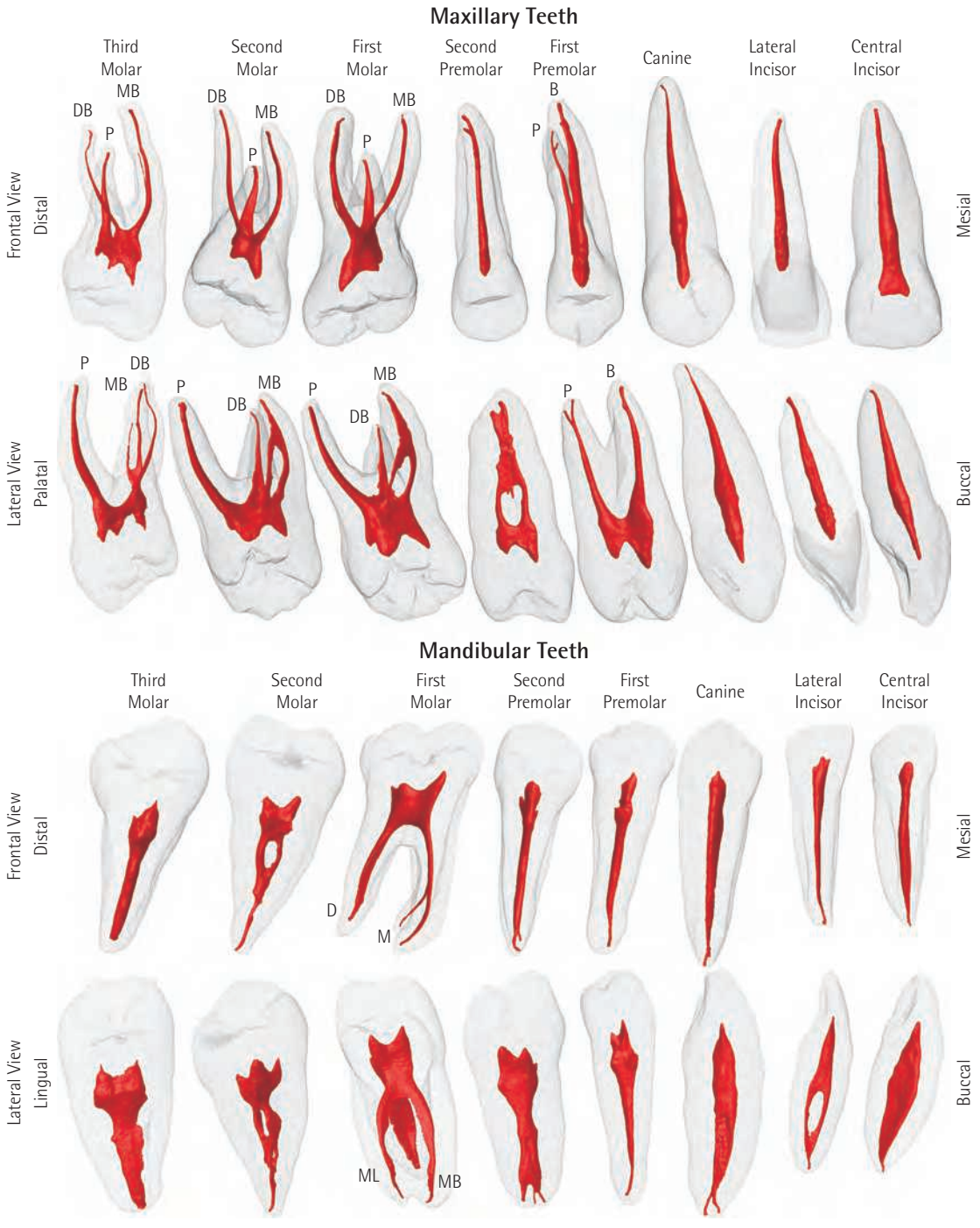
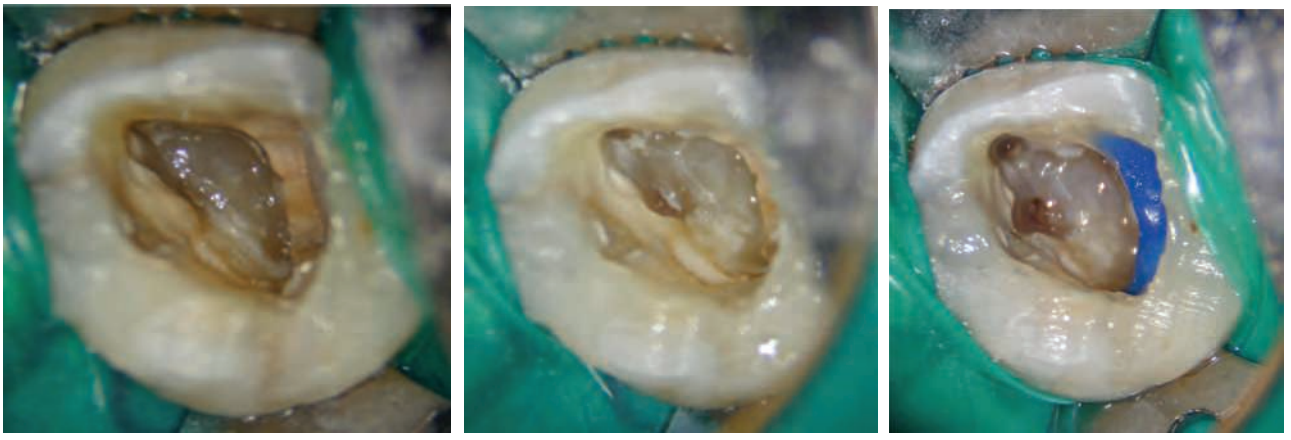


Figure 13.8 Micro-computed tomographic three-dimensional models of the most common root canal configurations in all groups of teeth. In most of the teeth, the common root canal morphology is the presence of one canal per root with the exception of the mandibular incisors, the maxillary premolars, the mesiobuccal root of the maxillary first molar, and the mesial root of mandibular molars, which have two root canals. B, buccal; D, distal; DB, distobuccal; M, mesial; MB, mesiobuccal; P, palatal; ML, mesiolingual. (*Courtesy: Marco Versiani, Pecora, and Sousa-Neto, Brazil.*)

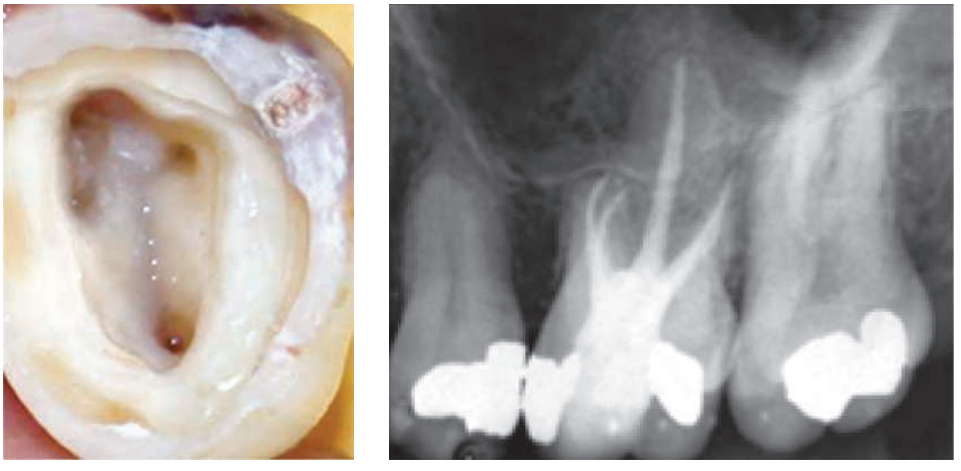
Figure 13.9 Diamond burs with rounded cutting ends. (*Courtesy: Dentsply Sirona.*)

Figure 13.10 Fissure carbide burs with non-end cutting safety tips. (*Courtesy: Dentsply Sirona.*)



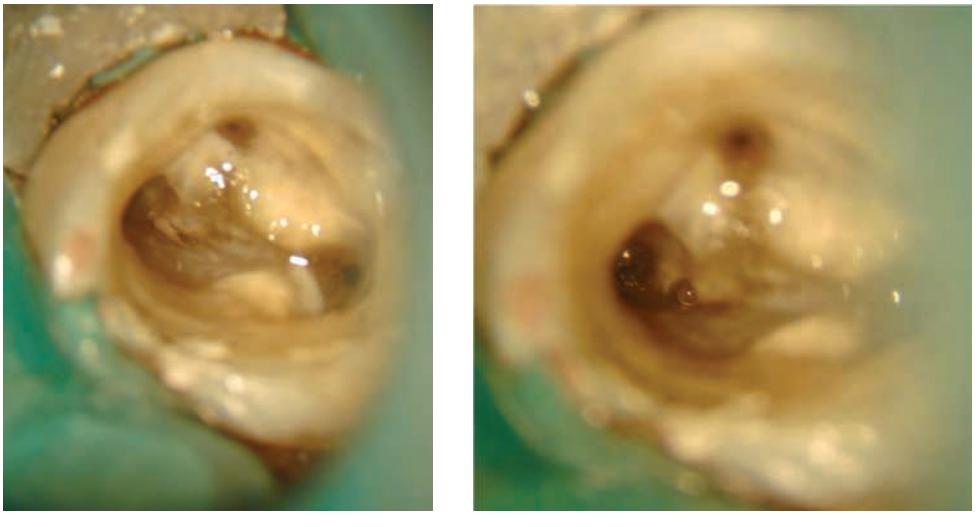
(i) (ii) (iii)

(a)



(i) (ii)

(b)



(i) (ii)

(c)

Figure 13.29 (a) MB-2 canal on the developmental line that connects MB-1 and palatal canal. (b) MB-2 canal mesial to the developmental line that connects MB-1 and palatal canal. (c) MB-2 appears as a groove on the palatal wall of the MB-1 canal. *(continued)*

Table 13.7 Clinically Significant Features During Access Preparation of Mandibular Teeth (*continued*)

	Average Tooth Length	Shape of Pulp Chamber	Roots and Root Canals	Clinical Significance	Shape of Access Preparation	Most Common Anomalies
Mandibular second molar	• 19–21 mm (Average 20 mm)	• Similar, but smaller than mandibular first molar	• Majority have two roots (71%), but with one root (27%) and three roots (2%) also seen	• C-shaped canal significantly higher • C-shaped canal system classified into: merging, symmetrical, asymmetrical	• Similar to mandibular first molar	• C-shaped canal significantly higher
Mandibular third molar	• 17.5–19.5 mm (Average 18 mm)	• Resembles mandibular first and second molar • C-shaped root canal orifices	• Usually two roots and two canals; occasionally, one root and one canal or three roots and three canals may be present	• Apex of root in close proximity to mandibular canal	• Similar to mandibular first and second molar	• Frequently has complex anatomic structure

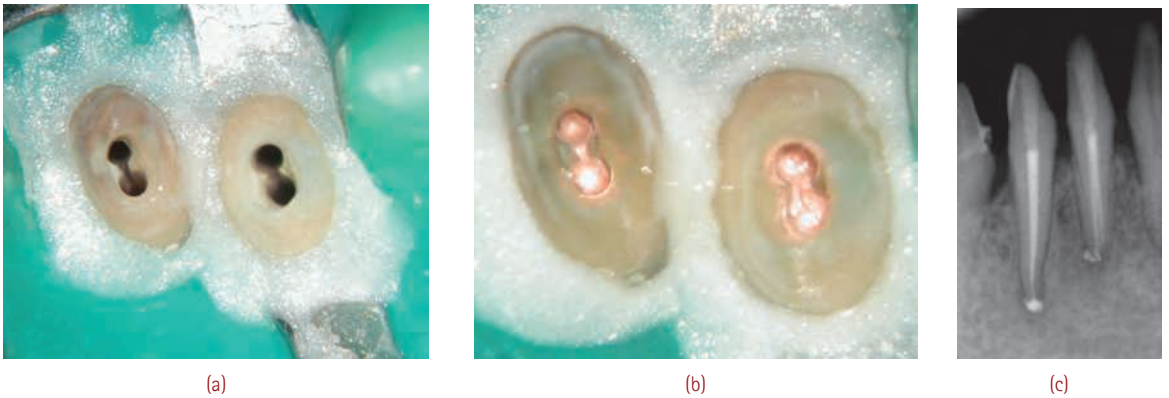
Table 13.8 Root Canals and Apical Foramina in Mandibular Incisors

Investigators	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
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

Clinical Note

- The second canal is normally located lingual to the primary canal (**Fig. 13.36**).
- Care should be taken during access opening in order to avoid buccal perforations.

Access Opening: The access opening of the mandibular central incisor is made in a similar manner as for the maxillary anterior teeth, with the variations that its smaller size demands. The shape of the access opening of the mandibular incisor is long and oval, with its greatest dimension oriented incisogingivally. Proper access enables one to explore the



(a) (b) (c)

Figure 13.36 Endodontic management of mandibular incisors with two canals. (*Courtesy: Siju Jacob, India.*)

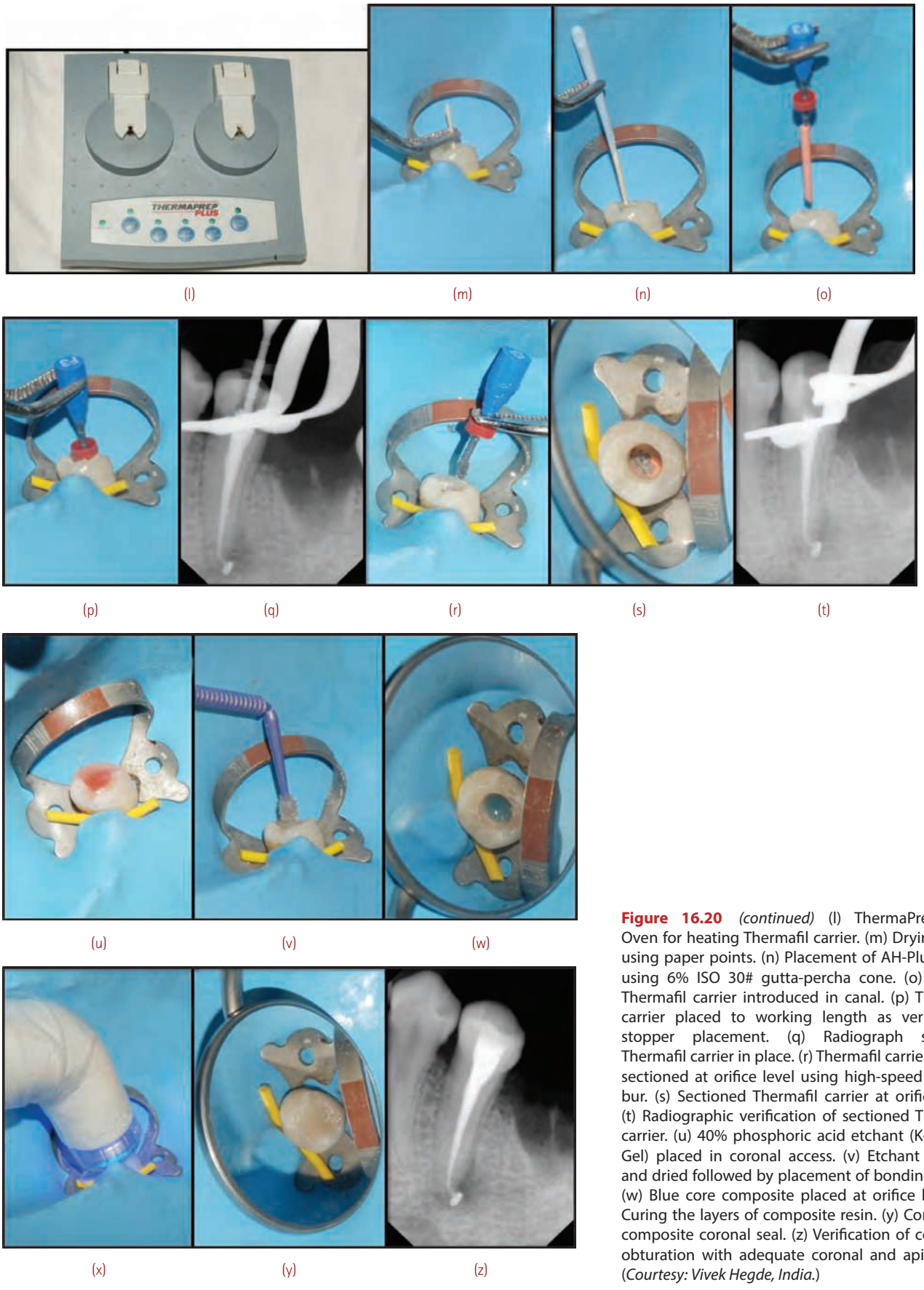
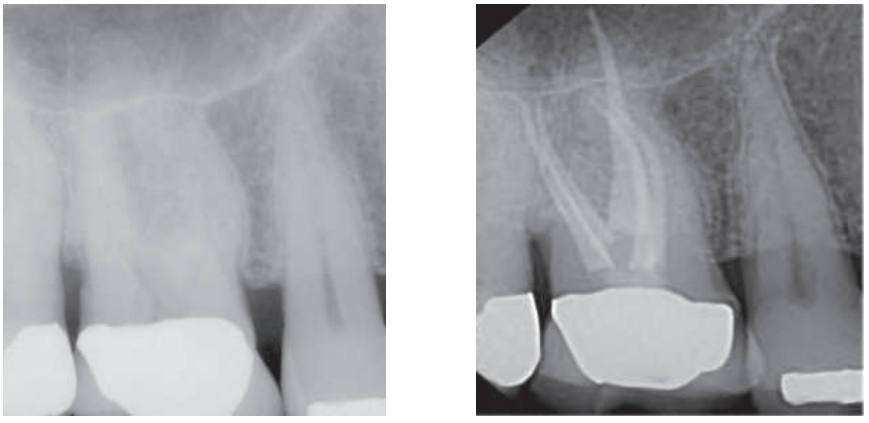
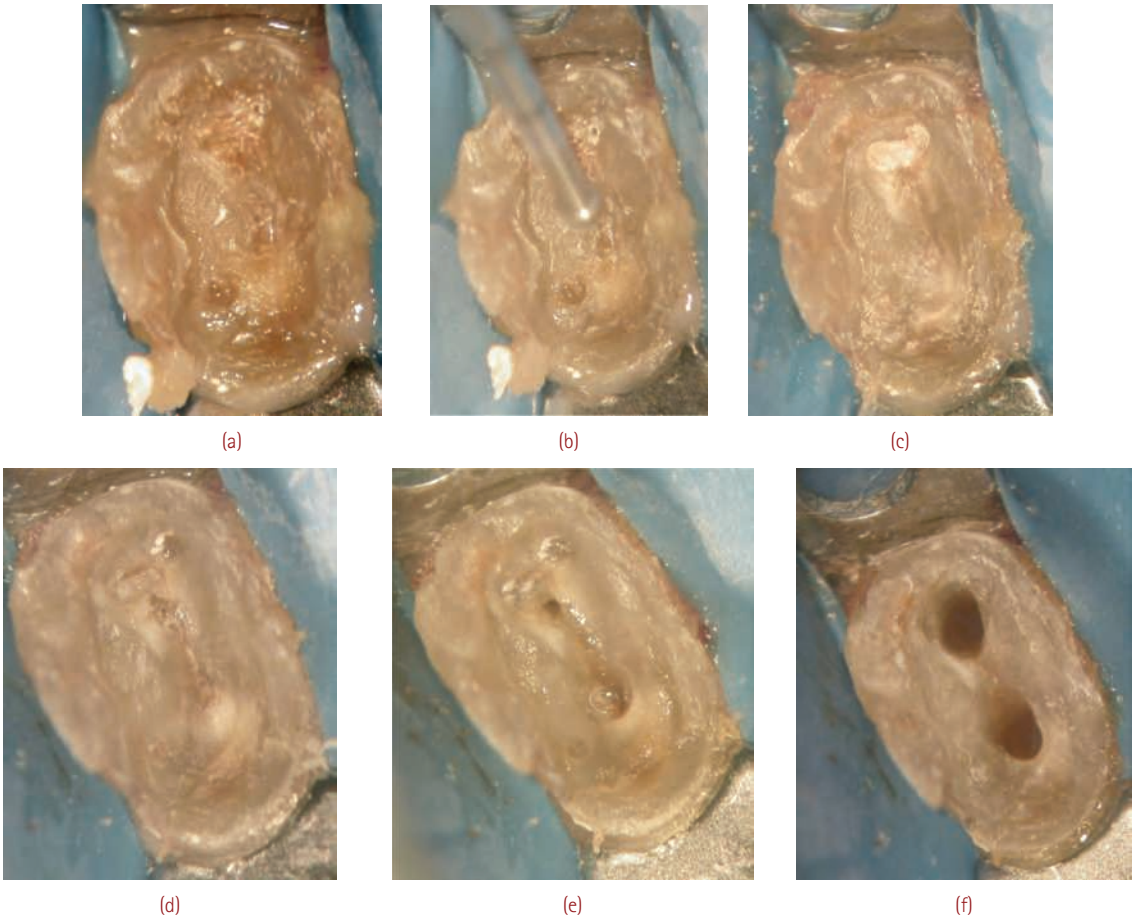


Figure 16.20 *(continued)* (l) ThermaPrep Plus Oven for heating Thermafil carrier. (m) Drying canal using paper points. (n) Placement of AH-Plus sealer using 6% ISO 30# gutta-percha cone. (o) Heated Thermafil carrier introduced in canal. (p) Thermafil carrier placed to working length as verified by stopper placement. (q) Radiograph showing Thermafil carrier in place. (r) Thermafil carrier handle sectioned at orifice level using high-speed carbide bur. (s) Sectioned Thermafil carrier at orifice level. (t) Radiographic verification of sectioned Thermafil 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. (*Courtesy: Vivek Hegde, India.*)



(a) (b)

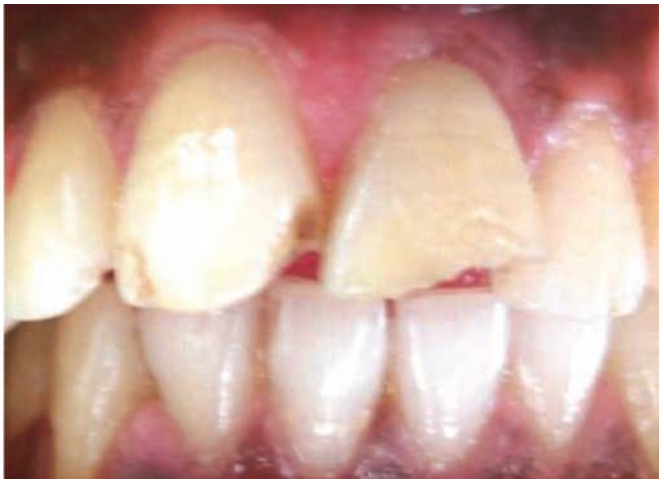
Figure 17.6 (a) Preoperative radiograph of the maxillary first molar indicative of the possibility of extra canals. (b) Postoperative radiograph showing evidence of five canals with two canals each in the mesiobuccal and distobuccal roots. (*Courtesy: Julian Webber, England.*)



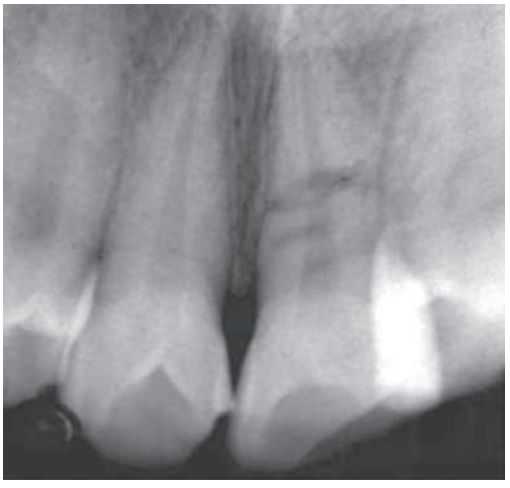
(a) (b) (c)

(d) (e) (f)

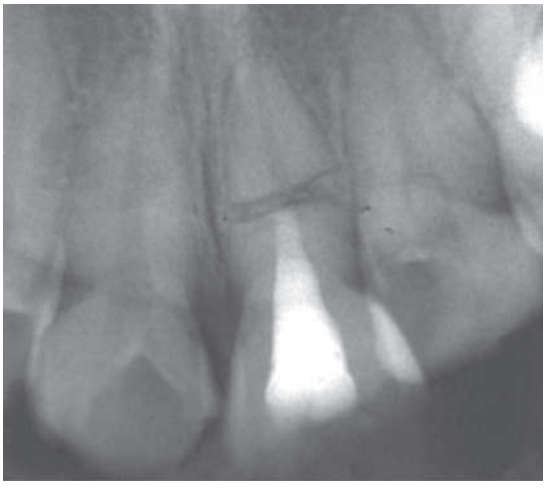
Figure 17.7 (a) Maxillary premolar with a calcified pulp chamber. (b) Troughing of the floor of the chamber with a Munce access refinement bur. (c) Canal orifices are becoming evident on deeper troughing. (d) Buccal and palatal canal orifices located. (e and f) Canals negotiated followed by shaping and cleaning. (*Courtesy: Siju Jacob, India.*)



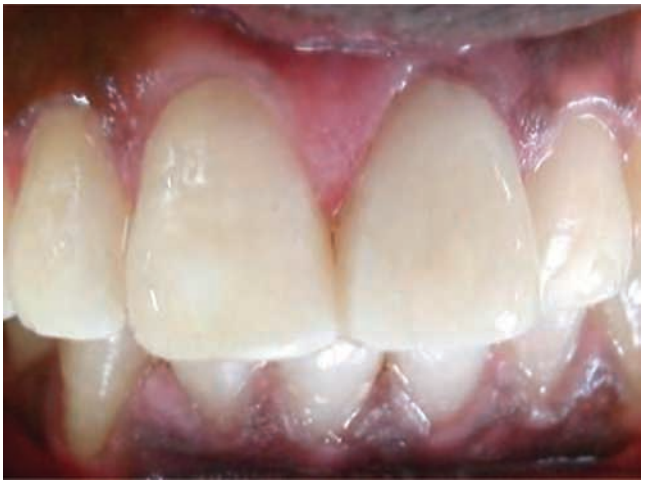
(a)



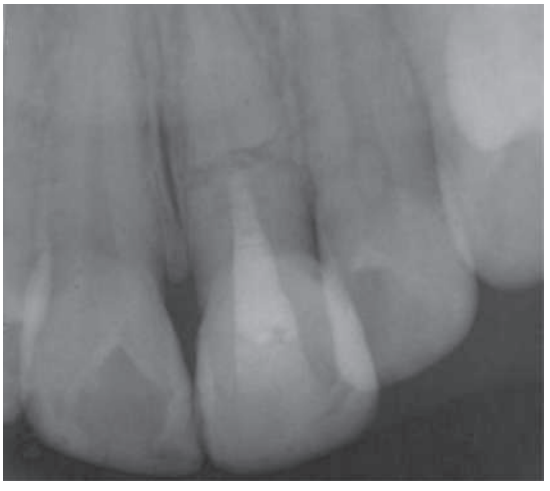
(b)



(c)



(d)



(e)

Figure 19.9 (a) Incisal third fracture of upper central incisors. (b) Radiograph revealing no pulpal involvement in left upper central incisor. But note the midroot fracture in the same tooth. (c) The coronal segment was endodontically treated and obturated with an apical matrix of mineral trioxide aggregate (MTA) and thermoplasticized gutta-percha. (d) Esthetic rehabilitation of the fractured tooth structure with direct composite resin. (e) Two-year follow-up radiograph showing healing of the interfragment tissues. Note the normal radiographic picture around the apical segment.

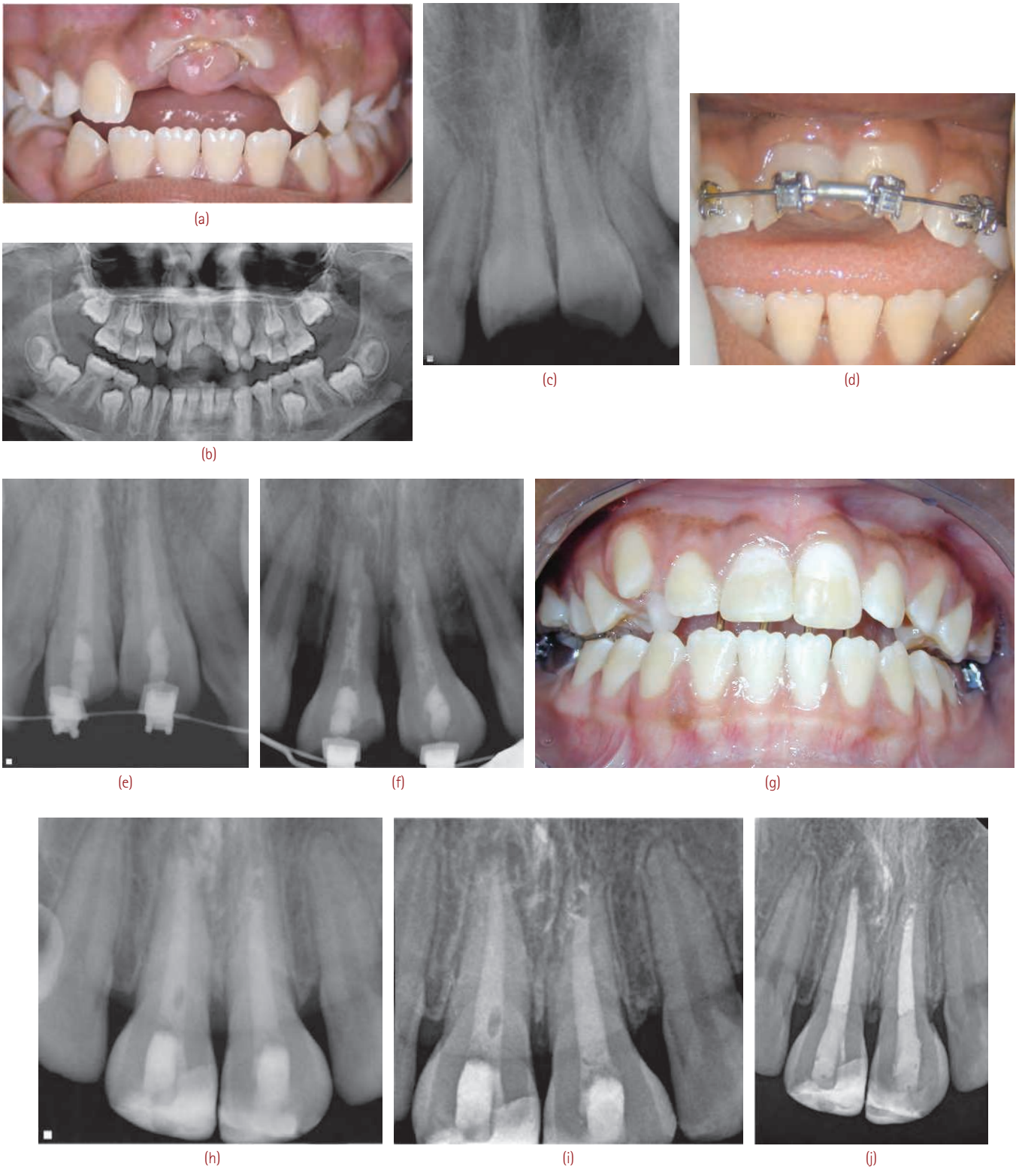


Figure 19.11 (a) Day 1 view: intrusive luxation of the maxillary central incisors in an 8-year-old boy due to trauma. (b) Day 1: orthopantomogram (OPG) view of the intrusion injury. (c) Two-month view: intraoral radiographic view of the intrusion. Nonvital response in both teeth. (d) Three-month view: 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-1-month view: orthodontic treatment discontinued. (h) One-year-and-1-month view: intracanal calcium hydroxide dressing changed. (i) One-year-and-7-month view: intracanal calcium hydroxide dressing changed. (j) One-year-and-10-month view: obturation completed. (*Courtesy: Sashi Nallapati, Jamaica.*)

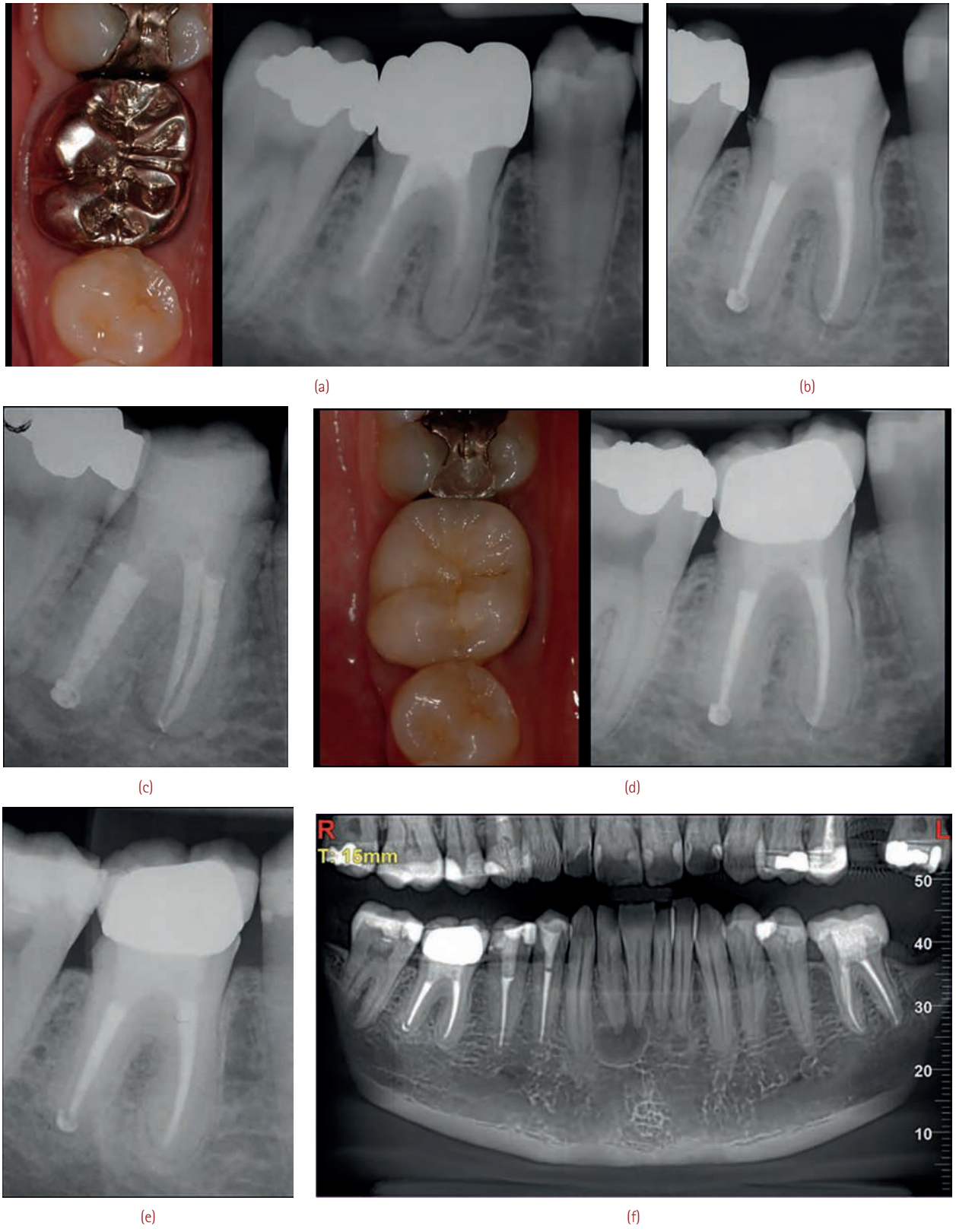


Figure 21.1 (a) Root canal–treated lower mandibular molar with periapical lesions on mesial and distal roots. The root canal filling is poor and the apex of the distal root looks slightly resorbed (year 2006). (b) Shaping completed and intracanal calcium hydroxide closed dressing given for 4 weeks. (c) Postoperative radiograph after obturation of the root canal system (warm vertical compaction—gutta-percha and ZOE sealer) and build-up with composite. (d) One-year follow-up (year 2007). (e) Twelve-year follow-up (year 2018). (f) Fourteen-year follow-up (year 2020)—CBCT done for the presence of a big lesion at level of mandibular incisors *(Case Courtesy: Filippo Cardinali.)*

Figure 21.4 WAM Key. (*Courtesy: WAM.*)

OBSTRUCTION FROM PREVIOUS OBTURATING MATERIALS

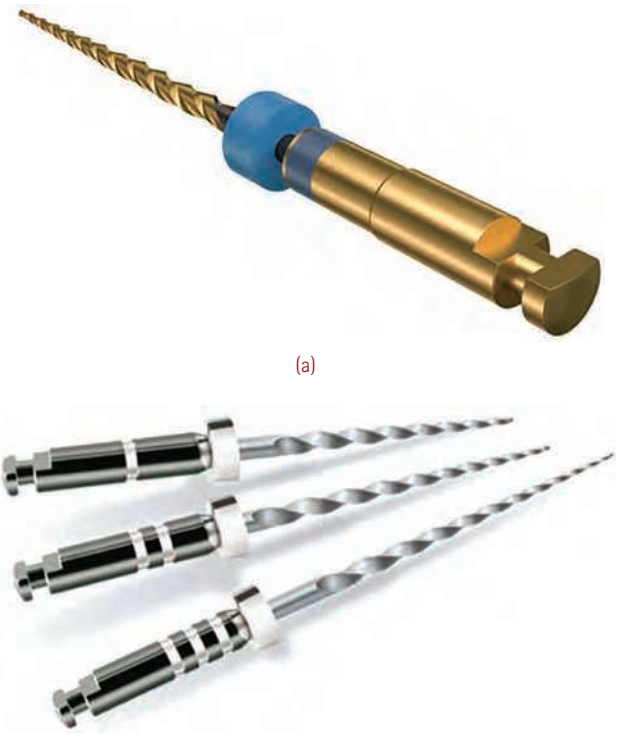
When retreatment of a previously endodontically treated tooth becomes necessary, the filling material must be removed or bypassed; otherwise, salvaging the tooth from extraction may require an endodontic surgical procedure. Because most teeth to be retreated are sealed with gutta-percha, and in some cases silver points, the following sections discuss the removal of these materials from root canals.

REMOVAL OF GUTTA-PERCHA

Gutta-percha and sealer can be removed by the application of the following:

i. Rotary instrumentation
ii. Solvents
iii. Heat carrier instruments
iv. Ultrasonics
v. Combination of the above

- One of the most common methods for removing gutta-percha obturation is by using rotary instrumentation. This can be achieved with normal nickel titanium shaping instruments; however, there are specific rotary retreatment files that are also available, for example, HyFlex Remover (Coltene) and ProTaper Universal retreatment files (Dentsply Sirona) (**Fig. 21.5**).
- One of the other methods for removing gutta-percha from the orifice and middle root canal is to use a solvent, which softens the gutta-percha and permits its removal through sequential instrumentation.
- Chloroform was the most popular solvent because of its efficiency. However, the use of chloroform is no longer recommended because of its reported toxicity and potential carcinogenicity.
- Some of the popular commercially available solvents used are Endosolv E and Endosolv R (Septodont) (**Fig. 21.6**). The Endosolv E is used for dissolving obturation materials based on zinc oxide and eugenol while Endosolv R is recommended for resin-based pastes and sealers.
- Other gutta-percha solvents that are also available include xylene, eucalyptol, and halothane.



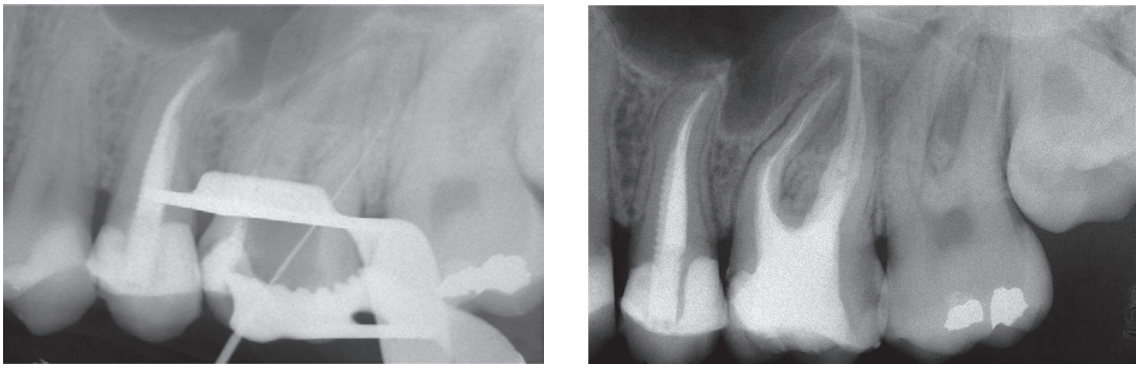
(a)

(b)

Figure 21.5 (a) HyFlex Remover. (*Courtesy: Coltene.*) (b) ProTaper Universal Retreatment files. (*Courtesy: Dentsply Sirona.*)

Figure 21.6 Endosolv E and Endosolv R solvents. (*Courtesy: Septodont.*)

- Solvents should be employed cautiously and should be administered a few drops at a time. The operator must use caution because these solutions frequently escape from the syringe needle without any apparent pressure on the plunger and there is no control over the depth of action of these solvents; hence, they are not recommended for apical third gutta-percha removal (**Fig. 21.7**).
- Gutta-percha can also be removed from the pulp chamber by using a heat carrier and searing the exposed gutta-percha. However, this technique is slower and would not allow complete removal of the obturating material from

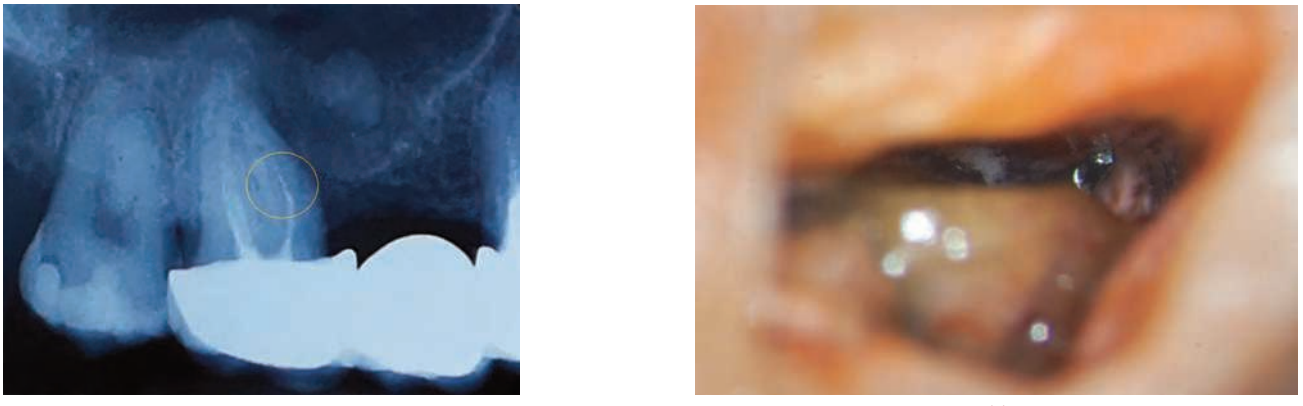


(g) (h)

Figure 21.16 *(continued)* (g) The radiographic image confirms the removal of the fragment. (h) Postoperative radiograph. (*Courtesy: Arnaldo Castellucci, Italy.*)

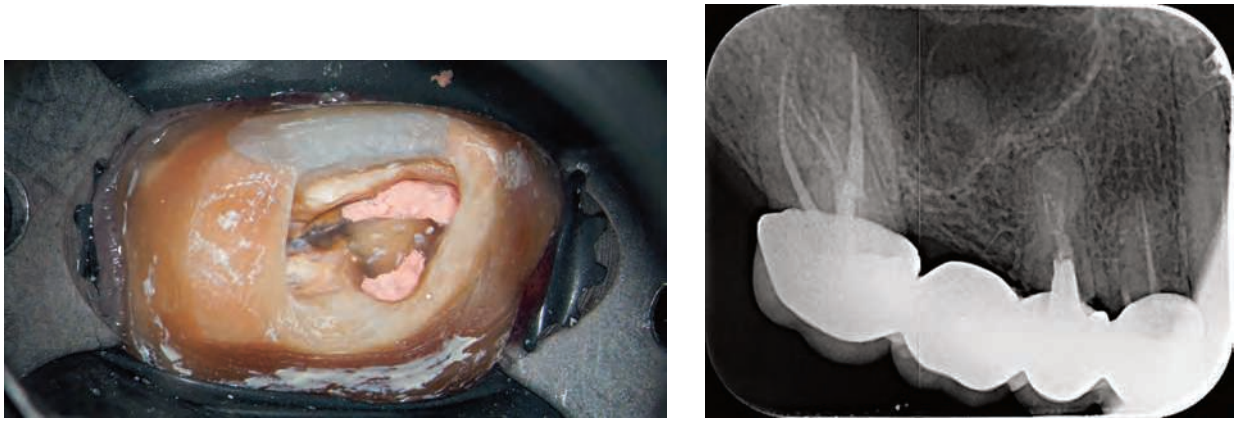
(a) (b)

Figure 21.17 (a) Endo Chuck for ultrasonic files. (*Courtesy: EMS.*) (b) U files. (*Courtesy: MANI.*)



(a) (b)

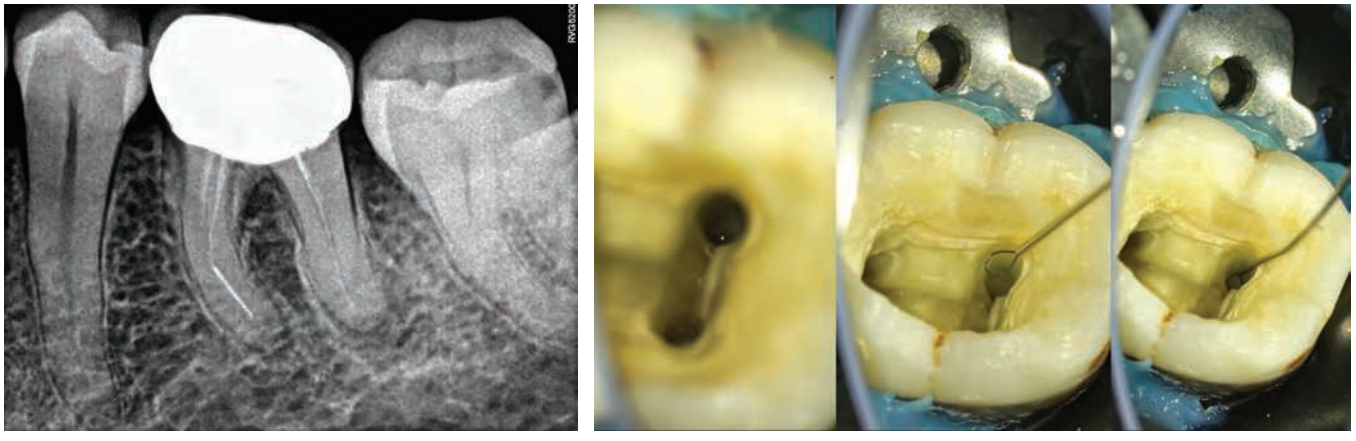
Figure 21.18 (a) Chronic alveolar abscess in a root canal–treated maxillary molar with radiographic evidence of a separated instrument in the mesial canal. (b) Separated instrument visualized under the microscope. *(continued)*



(f) (g)

Figure 21.18 *(continued)* (f) Postobturation view. (g) Follow-up X-ray revealing resolution of disease. (*Case Courtesy: Sergiu Nicola, Romania.*)

Figure 21.19 BTR pen. (*Courtesy : Cerkamed.*)



(a) (b)

Figure 21.20 (a) Preoperative x-ray showing separated instrument beyond the level of curvature in the apical third of the mesiobuccal canal of a lower first molar (b) Use of a BTR pen loop to engage the coronal third of the fragment after creating a channel path. *(continued)*

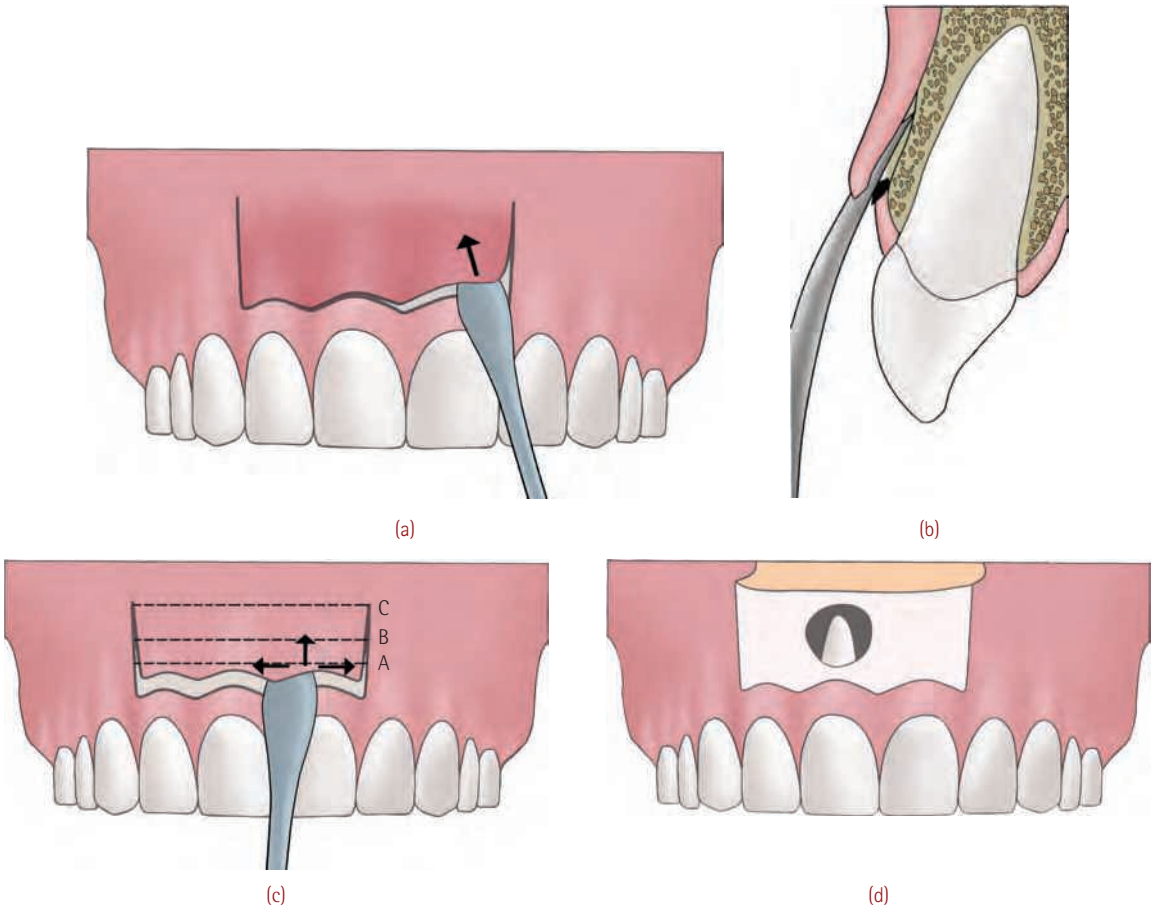


Figure 22.12 (a and b) Raising a Luebke-Ochsenbein flap: the elevator edge, with its concave surface facing the bone, cleaves the periosteum from the bone apically and laterally until the bone above the lesion is exposed. (c) The periosteal elevator should be moved laterally and apically without losing contact with the bone. (d) The extent of the flap should be sufficient enough to expose bone above, below, and around the lesion.

FLAP ELEVATION

Even though several types of elevators are available, the Molt curette no. 2–4 is suitable for both elevation and curetting with minimum trauma. One has to gently use 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. 22.12**).

FLAP RETRACTION

Flap retraction is required for proper visibility and access to the surgical area. Several types of retractors are available and are designed to have wider and thinner working ends than standard retractors.

HARD-TISSUE CONSIDERATIONS

OSTEOTOMY

Osteotomy involves the removal of cortical plate to expose the root end in microendodontic surgical procedures. Once the flap has been elevated and placed in retracted position, the surgical area is taken into control.

Technique

- Hemostasis is the primary issue at this stage of surgery.
- In most of the clinical cases, there is a breach in the cortical plate and this can be located around the root apex by gently probing with a DG16 explorer, and if the breach is located, the explorer will sink and this could be the starting point for an efficient osteotomy. However, in most cystic pathosis, the cortical plate is thinned out due to the growth of the cyst and has an eggshell crackling appearance. In these situations, the cortical plate can be peeled off leaving the cystic lining which balloons out once the cortical plate is removed.
- If the cortical plate is intact, obtain a periapical radiograph after marking the approximate root-end portion of the cortical plate with a small piece of sterile GP stick. This will indicate the approximate location of the apex and the apical pathosis.

which results in better adaptation of filling materials, fewer microfractures, and less leakage. These retrotips are either stainless steel, diamond-coated, or made of zirconium nitride.

Advantages of Ultrasonic Retrotips in Endodontic Surgery

- Size in osteotomy preparation is reduced to less than 5 mm. The ideal diameter needed is only 4 mm, thereby allowing a 3-mm ultrasonic tip to move freely within the bone crypt
- Reduced size of osteotomy leads to faster and better healing of the surgical wound
- More precise and efficient retropreparation than when compared with burs
- Reduced risk of lingual perforation of the root-end cavity preparation
- Access to the root end is easier and more predictable in terms of the final preparation design
- It is an efficient method to manage the isthmus area between the root canals within one root
- It allows cleaner and deeper root-end cavities which helps in improving the prognosis of the procedure

Retro/root-end preparations are usually 3 mm deep with ultrasonic tips. Final inspection with a retromirror should show no gutta-percha remaining in the 3-mm depth of preparation (**Figs 22.18–22.21**).

PREPARING THE ROOT END FOR A ROOT-END RESTORATION

The area is dried and isolated after irrigation with normal saline or distilled water. The root canal seen through the cut end of the root is located.

Root-End Filling Materials

- Tricalcium silicate cement
- MTA
- Intermediate restorative material (IRM)
- SuperEBA
- Glass ionomer cement
- Diaket
- Composite resins and resin ionomer hybrids

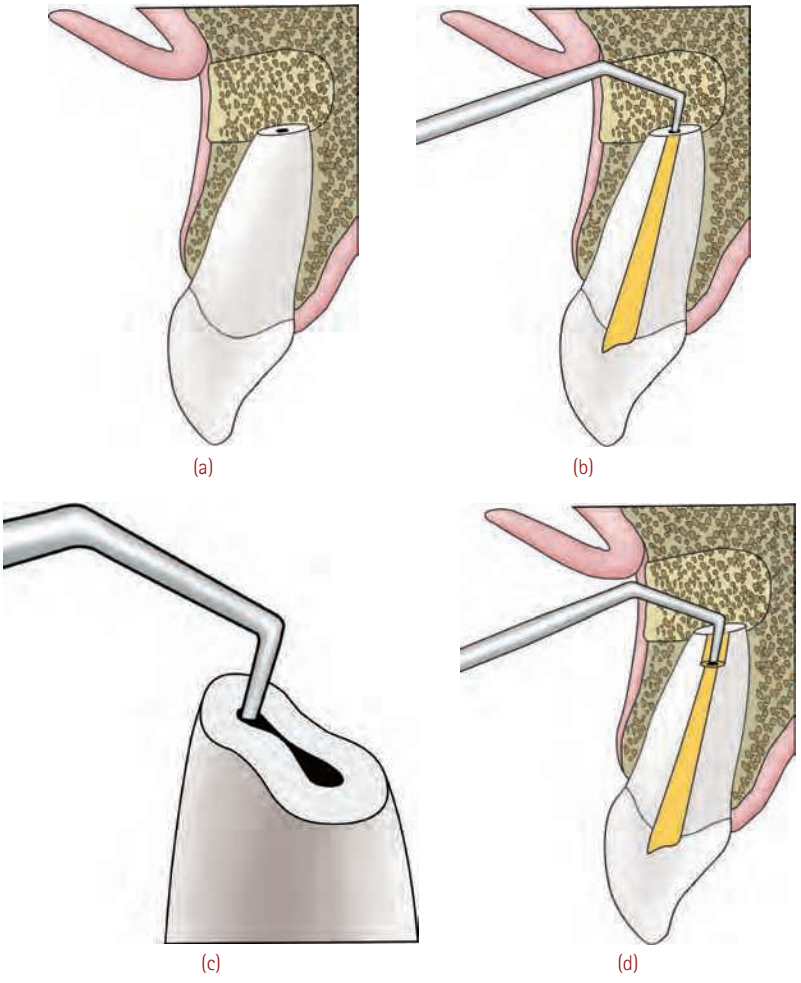
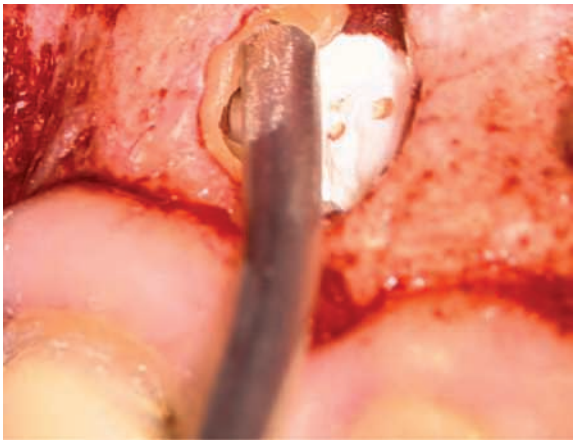
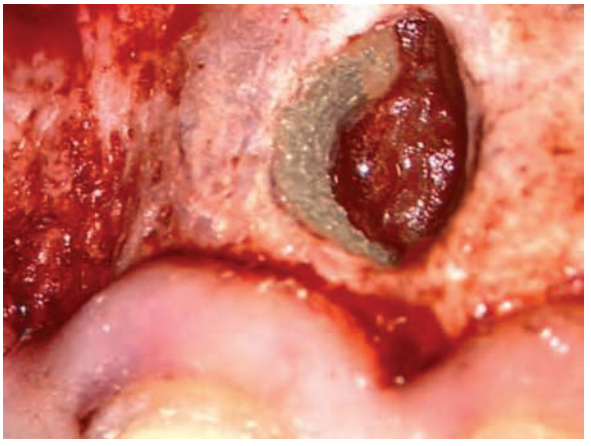


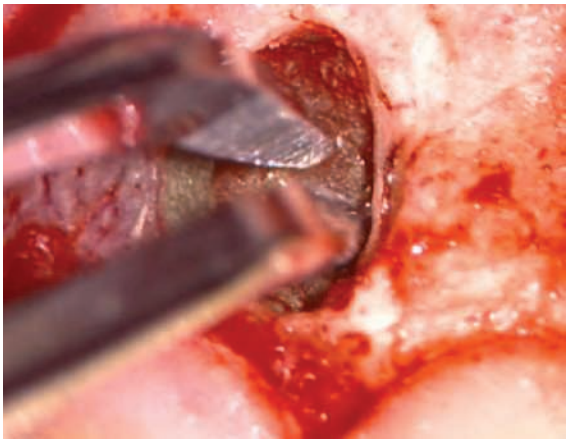
Figure 22.18 (a) Schematic representation of root-end resection. (b) Ultrasonic retrotip being employed to begin the retropreparation. (c) Isthmus region is best negotiated with an ultrasonic retrotip. (d) A depth of 3 mm is the depth of an ideal retropreparation.



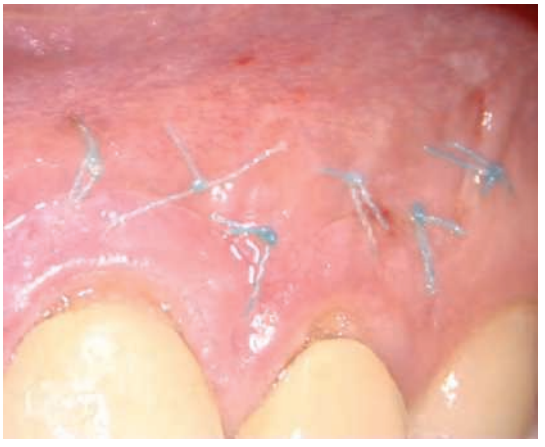
(e)



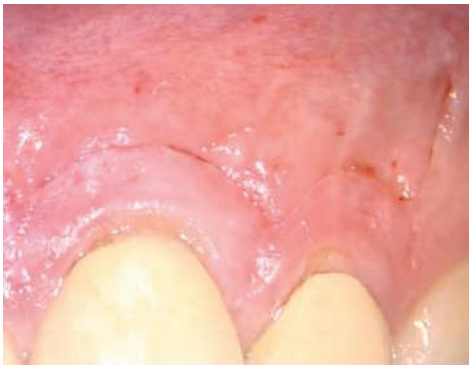
(f)



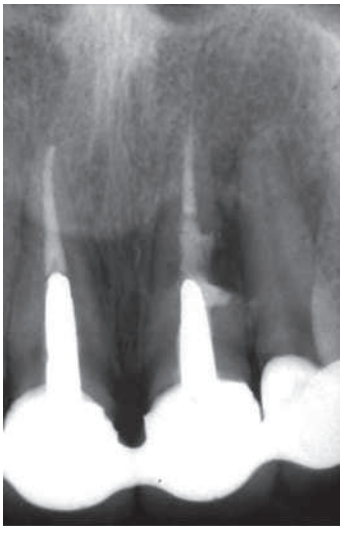
(g)



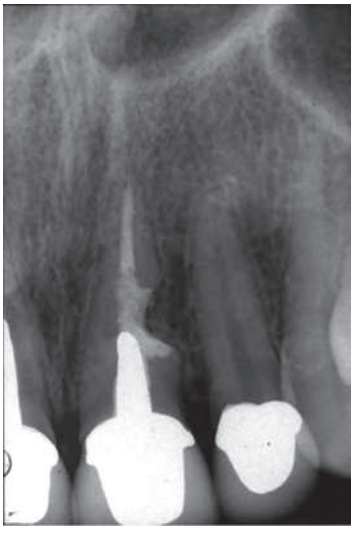
(h)



(i)



(j)



(k)

Figure 22.27 *(continued)* (e) The Dovgan carrier is positioning gray MTA in the defect. A few pieces of CollaCote have been positioned on the bottom of the bony defect to collect the debris of MTA. (f) The resorption has been filled with gray MTA. (g) The cotton pliers are used to remove the CollaCote. (h) Postoperative sutures placed. (i) Removal of the suture after 24 hours. (j) Postoperative radiograph. (k) Two-year recall. Radiograph showing excellent healing. (*Courtesy: Arnaldo Castelluci, Italy.*)