Seltzer and Bender's Dental Pulp

SECOND EDITION

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Cover image illustrating the innervation of normal human dental pulp using antibodies for neurons (N52, green; PGP, blue) and the receptor TRPA1 (red). Image courtesy of Michael A. Henry, DDS, PhD.

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Preface

Welcome to the second edition of *Seltzer and Bender's Dental Pulp*. Like the first edition, this book focuses on the dental pulp and its interaction with other tissues during health and disease, with each chapter providing the latest information on the biologic principles and the basis for clinical treatment procedures. As such, the book is ideally suited for practicing dentists as well as residents and dental students. This newly revised second edition includes entirely new topics (eg, regenerative endodontics) as well as greatly expanded reviews on dental implications of biofilms, immune interactions, pain mechanisms, the interactions between restorative dental procedures and pulpal health, and neuroanatomy, among other topics. We welcome many new and returning authors to this edition who have shared their incredible expertise with you, our reader.

The central theme of this book—a fundamental theme of dentistry in our opinion—is the critical role that pulp tissue plays in dental health. Both local (eg, caries, periodontitis) and systemic (AIDS, hyperparathyroidism) disease can contribute to pulpal pathosis. In turn, pulpal pathosis can contribute to both local (eg, root resorption, periodontitis) and systemic (eg, referred pain) conditions. The astute clinician needs this information to provide accurate diagnoses and effective treatment. Accordingly, we have focused on the biology of dental pulp and its interaction with other tissues during health and disease in order to provide comprehensive, biologically based clinical recommendations for practicing dentists.

We have been gratified by the support and encouragement generated from the first edition of this text, and we were thrilled that both I. B. Bender and Sam Seltzer lived to enjoy its publication. We have now lost many of the pioneering giants of endodontics and pulp biology. Their early contributions laid the foundation for generations of dentists to deliver biologically based dental care. In this age of gene arrays, signal transduction pathways, novel restorative materials, and computerized data retrieval, it is difficult to appreciate the magnitude of their contributions based entirely upon intellectual rigor and using relatively simple tools. To their memories, we dedicate this second edition.

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Chapter 18

Aging and the Pulp

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Advances in living standards, including medical and dental care, have contributed to increased life spans and a growing proportion of elderly people in the population. The elderly often need more medical and dental services compared to the average citizen, including root canal treatment.[1] Results of a survey[1] of Diplomates of the American Board of Endodontists indicated that Diplomates examine patients covering a wide spectrum of ages but most fall into the age range of 45 to 64 years. Respondents indicated that about 26% of their patients are at least 65 years old. A substantial majority (59%) of respondents (n = 334) indicated that the number of patients aged 65 years or older is increasing in their practices.

The increased need for endodontic treatment among older individuals is due partly to naturally occurring anatomical and physiologic senescent changes that are associated with the aging process and partly to diseases that occur more commonly in older adults[2] (see chapter 20). Oral health is important because oral diseases affect more than the mouth.[3] Normal aging processes in healthy individuals often have few adverse effects in the oral cavity, but tooth loss, caries, periodontal diseases, and pulpal and periradicular diseases will have deleterious consequences.[4]

The increased need for dental services for older individuals is also a reflection of the greater retention of teeth into old age.[5] Utilization of dental care services increases with increasing age; clinical findings suggest that individuals 65 years old or older have more caries than young children,[6,7] although the caries attacks cervical rather than occlusal surfaces. Further, the number of teeth with carious or restored root surfaces increases the longer a person lives; more than half the retained teeth in individuals 75 years of age or older are affected.[8] The changes occurring in the dental pulp of elderly patients may explain the increase in requests for endodontic treatment.[9] This chapter reviews age-dependent and age-independent processes in the dental pulp and evaluates their impact on the quality of oral health care in the elderly patient.

Process of Aging

A discussion of aging in a particular tissue or area of the body is based on biologic theories of replicative senescence, together with the roles of oxidative stress and telomeres on the aging process. *Organismal senescence* is the aging of whole organisms. The term *aging* has commonly been equated with *senescence* such that the terms can be used interchangeably. The role of telomeres, structures found at the ends of chromosomes in the cells of

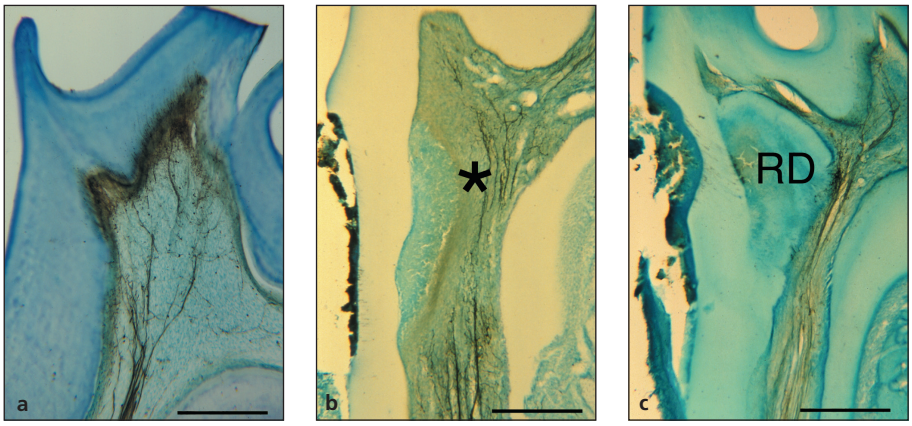

Fig 7-8 Different patterns of nerve fibers detected by immunocytochemistry for CGRP in rat molars. *(a)* Pattern in normal molars (bar = 0.2 mm). (Modified from Kimberly and Byers[56] with permission.) *(b)* Pattern 4 days after a large abscess *(asterisk)* is induced near a cervical dentinal cavity (bar = 0.2 mm). (Modified from Taylor and Byers[54] with permission.) *(c)* Pattern after 3 weeks of healing and reparative dentin (RD) formation at an injury site that was similar to that in *(b)* (bar = 0.2 mm). (Modified from Taylor and Byers[54] with permission.)

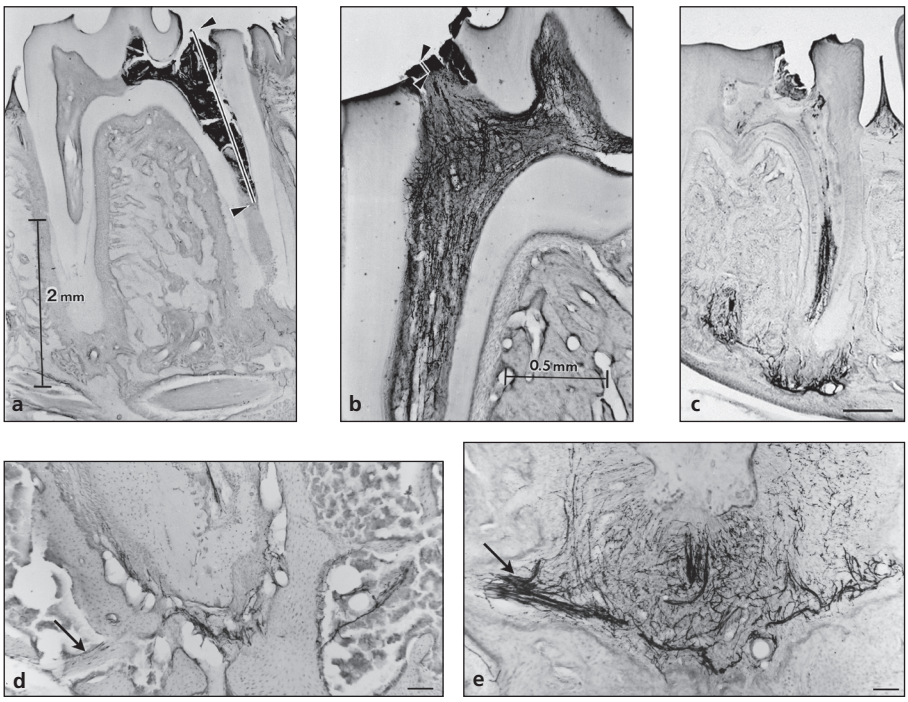

Fig 7-9 *(a)* Rat molar denervated several days before a small pulpal exposure. By 6 days later, the pulpal damage and necrosis are severe *(arrowheads and bar)*. (Reprinted from Byers and Taylor[59] with permission.) *(b)* Innervated contralateral tooth with many sprouting nerve fibers. Compared with the tooth in *(a)*, it has only a small loss of pulp *(arrowheads and bar)* after a small pulpal exposure. The sprouting nerve fibers show immunoreactivity for CGRP. (Reprinted from Byers and Taylor[59] with permission.) *(c)* Rat molar root with nerves and vital pulp retained. At 14 days after a pulpal exposure, there is already a periapical lesion with CGRP-immunoreactive sprouting fibers (bar = 0.5 mm). (Reprinted from Khayat et al[60] with permission.) *(d)* Normal periapical region of a rat molar immunoreacted for CGRP. The region shows normal, sparse innervation of the periodontal ligament. The *arrow* shows the adjoining periapical nerve (bar = 0.1 mm). (Reprinted from Kimberly and Byers[56] with permission.) *(e)* Periapical changes and sprouting nerve fibers appear 3 to 5 weeks following establishment of irreversible pulpitis subsequent to pulpal exposure lesions. Compared to that observed in *(d)*, the nerve fiber immunoreactivity for CGRP was also enhanced in the adjoining periapical nerve *(arrow)* (bar = 0.1 mm). (Reprinted from Kimberly and Byers[56] with permission.)

Distant plasticity in the trigeminal nerve, ganglion, and central endings

The discussion so far has focused on dental sensory reactions in the terminal branches within the tooth or nearby tissues. These neurons also have extensive changes in their alveolar branches[56] (see Fig 7-9), at their cell bodies and satellite cells in the trigeminal ganglion[4,45] (see Fig 7-7), at their sensory endings in the brainstem, and in the neurons within the central nervous system. Many of the responses at the ganglion are similar to those shown for spinal nerves responding to tissue inflammation, including altered expression of neurotrophin receptors, neuropeptides, and voltage-gated ion channels by the neurons and increased expression of injury proteins by the satellite cells. Those changes can have profound

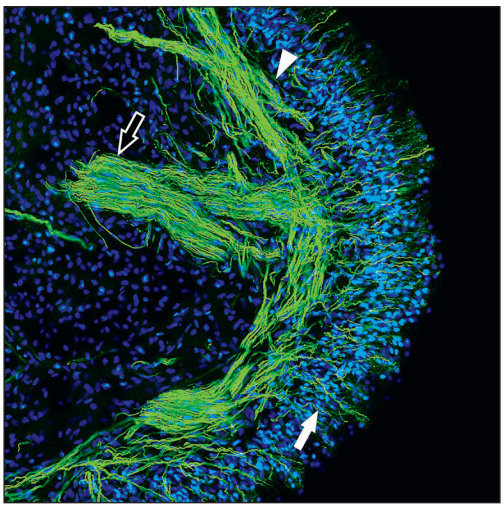

Fig 7-10 Confocal micrograph to demonstrate the overall nerve innervation pattern within the coronal region of a normal molar dental pulp. Nerve fibers are identified with both neurofilament 200-kD and GAP-43 immunoreactivities *(green)*, while nuclei are identified with TO-PRO immunoreactivity *(blue)*. Axon bundles are located within the midcoronal region *(black arrow)*, which leads to the many axons within the subodontoblastic plexus *(arrowhead)*. Some of the fibers within the subodontoblastic plexus enter and traverse the odontoblastic layer *(white arrow)*. The nuclei of the odontoblasts are more numerous and larger than are the nuclei of other cellular profiles elsewhere in the pulp.

effects on central pain pathways. For example, tooth injuries can cause persistent expression of the c-Fos transcription factor by central neurons, which may indicate altered central pain pathway functions.[62,63] Atypical chronic dental pain and referred pain both involve long-term shifts in central processing of peripheral inputs. Chapters 8 and 9 provide further discussion of tooth pain and the extraordinary functional and cytochemical plasticity of peripheral and central neurons responding to the input of orofacial sensory neurons.

Delayed neural reactions

Both the sensory and the sympathetic fibers can have important reactions that are not launched until days or weeks after tooth injury. For example, the alveolar nerves that carry dental axons can greatly change their neuropeptide content by several weeks after a pulpal exposure in rats[56] (see Figs 7-9d and 7-9e). The sympathetic innervation initially was not found to sprout during the early stages of neuropulpal reactions to pulpal exposure, but, by several weeks later, it too has focal responses directed toward the lesion.[9] The late sympathetic reactions have a major effect on immune cell invasion of the injured pulp and may even alter the quality of tooth pain. Thus, while the initial sensory sprouting reactions are important, subsequent reactions in those fibers, in the sympathetic neurons, and at central neural pathways must also be appreciated for their roles in tooth pain.[64–66]

Human teeth

The results of studies performed in animals have provided important information regarding the neuroanatomical responses in the diseased or damaged dental pulp. Certainly the advantage of these studies is that responses can be evaluated at different time points following a standardized insult. Another distinct advantage is the ability to evaluate the broad effect of these injuries within the entire trigeminal neuroaxis. Even given these advantages, some limitations exist in animal studies, and most notable is the relationship of these neuroanatomical responses to pain and especially pain in humans. In this regard, knowledge gained in animal studies must be applied to the study of the human dental pulp, where pain levels and response to stimuli can be documented prior to extraction.

The human dental pulp is richly innervated—a common source of pain—and so its use is well-suited for such studies. Also, the routine extraction of both normal third molars and diseased teeth provides an abundant supply of specimens for study. Together, the results from human and basic animal studies can further the understanding of possible correlations between neuroanatomical responses and pain mechanisms in an attempt to more fully understand pulpal pain and its important relationship to the practice of endodontics. In general, the innervation of human dental pulp (Fig 7-10) is similar to that seen in experimental animals, and these similarities strengthen the use of animals as a model for understanding response to injury in the human dental pulp.

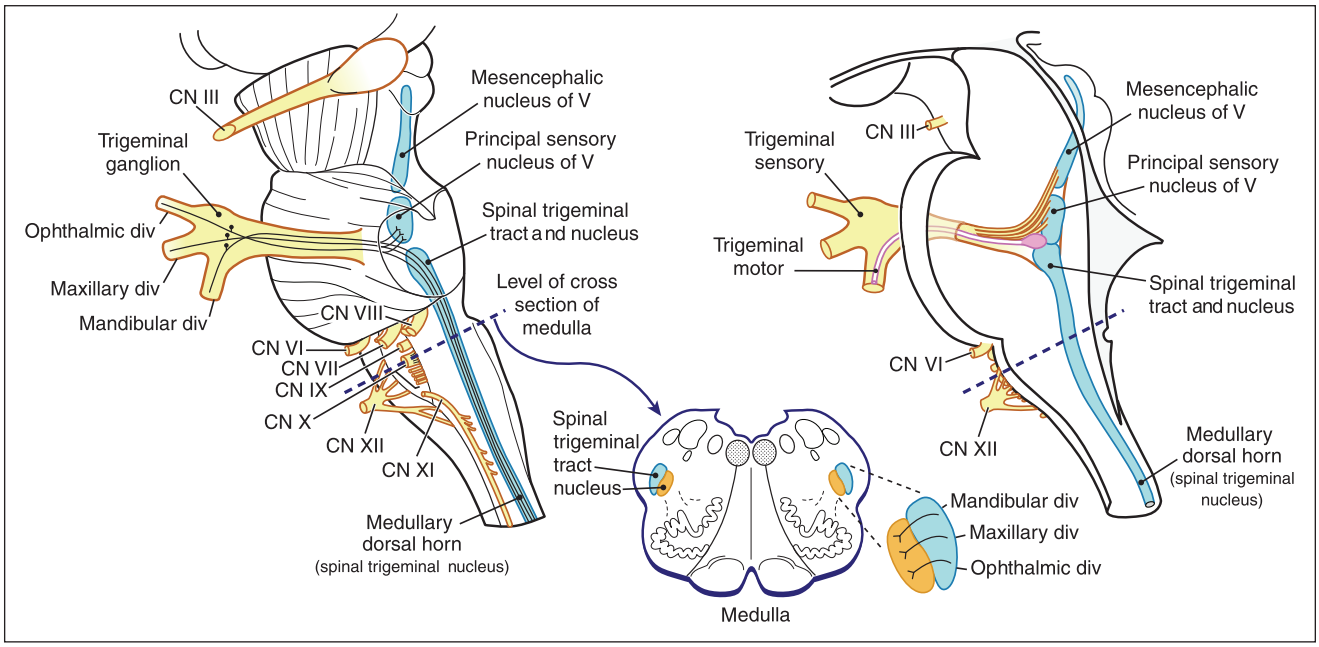

Fig 8-1 General steps in pain transmission in the orofacial region. Detection of noxious injury occurs via primary afferent nociceptors that travel in one of the three divisions (div) of the trigeminal nerve (ophthalmic, maxillary, and mandibular). (In certain chronic pain conditions, detection also may occur by other afferent fibers such as the Aβ fibers.) Processing occurs primarily in the medullary dorsal horn. Nociceptive signaling may be increased by central mechanisms of hyperalgesia or allodynia. Nociceptive signaling may also be reduced by endogenous analgesic systems. The output from the medullary dorsal horn is conveyed predominantly along the trigeminothalamic tract. Perception occurs primarily in the cerebral cortex. Other sensory nerves are also responsible for additional craniofacial signaling (eg, cranial nerves [CN] VII, IX, and X as well as afferent fibers from the cervical spinal cord).

fying processes are active at the time of the clinical evaluation and may add to the challenges associated with the diagnostic task. This chapter reviews those peripheral and central pain mechanisms that should be considered when the clinician evaluates the symptomatic patient.

Pathways Responsible for Detection, Processing, and Perception of Dental Pain

Odontogenic pain is usually the result of a noxious physical stimulus or the release of inflammatory mediators that stimulate receptors located on the terminal endings of nociceptive (pain-detecting) afferent C and Aδ nerve fibers[12–16] (see chapter 7). Physical stimuli, via their effect on dentinal fluid flow, can activate the nociceptors that innervate dentinal tubules, leading to the perception of dentinal pain[16] (Fig 8-2). Inflammatory mediators, via activation of their respective receptors, can sensitize or depolarize the nociceptors that innervate pulp tissue. These topics are discussed in detail later in the chapter and elsewhere.[17] Experimental studies have shown that activation of nerves within the dental pulp by these physiologic (eg, thermal, mechanical, or chemical) stimuli results in a pure sensation of pain, although other studies using certain electrical stimuli can elicit a "prepain" sensation.[18]

The activation of the peripheral nociceptor produces a generator potential; if great enough, this depolarization will trigger a nerve impulse (action potential). The action potential is propagated along a peripheral trigeminal nerve to the primary afferent neuronal cell body located in the trigeminal ganglion and then into the central nervous system along the central process of this same neuron[14,19–21] (Fig 8-3).

The central process of the primary afferent cell body enters the brainstem at the level of the pons by way of the trigeminal root entry zone and then enters the trigeminal tract. The trigeminal tract carries the primary afferent fiber to the trigeminal sensory nucleus located in the pons and medulla, where it then terminates. The most rostral portion of the trigeminal sensory nucleus is the main sensory nucleus, while the caudal portion is represented by

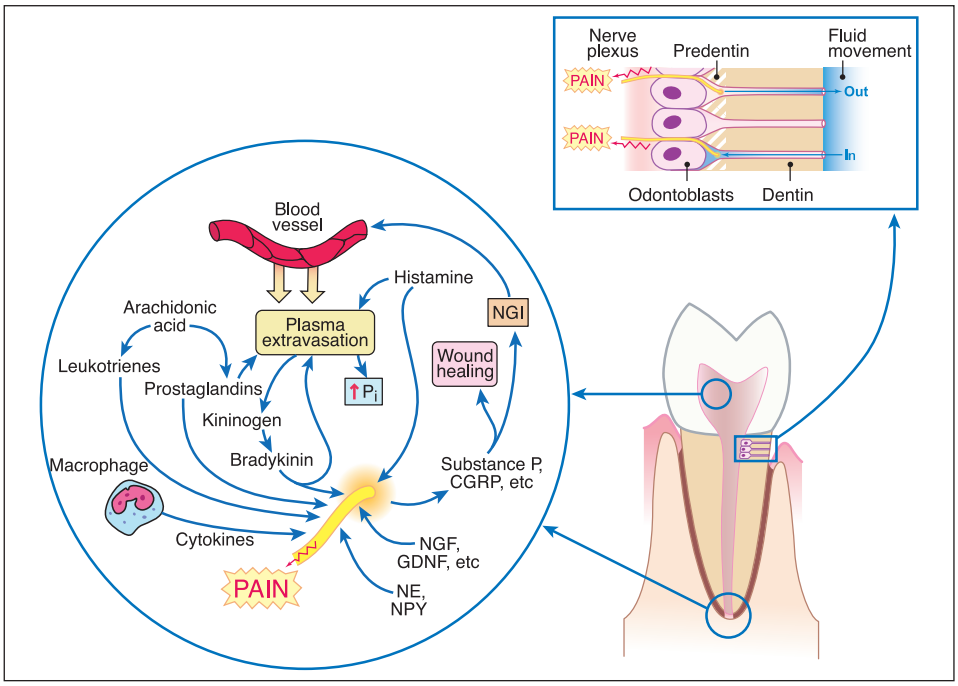

Fig 8-2 Two mechanisms for the peripheral stimulation of nociceptive nerve fibers in tooth pulp. *Acute dentinal pain:* According to the hydrodynamic theory, stimuli that cause fluid movement in exposed dentinal tubules result in the stimulation of nociceptive nerve fibers. *Pain with inflammation:* Inflammation is associated with the synthesis or release of mediators, including prostaglandins, bradykinin, substance P, and histamine (as well as other mediators not shown). The interrelationships of these inflammatory mediators form a positive feedback loop, allowing inflammation to persist far beyond cessation of the dental procedure. P_i, intrapulpal pressure; NGI, neurogenic inflammation; CGRP, calcitonin gene–related peptide; NGF, nerve growth factor; GDNF, glial cell line–derived neurotrophic factor; NPY, neuropeptide Y; NE, norepinephrine.

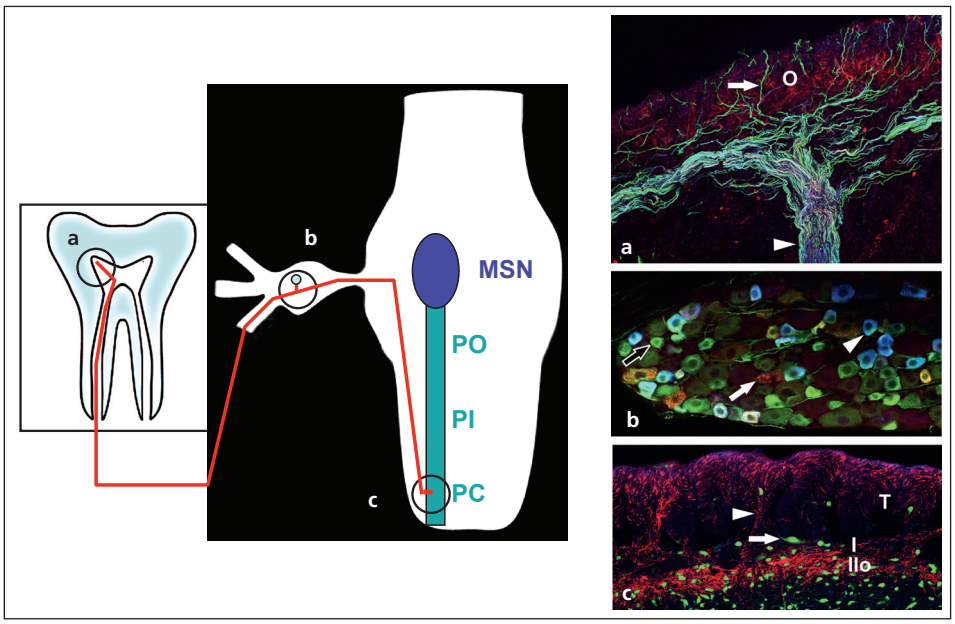

Fig 8-3 Pathway and confocal micrographs of neuroanatomical structures responsible for the transmission of pulpal nociceptive stimuli within the trigeminal system. Peripheral nociceptive nerve fibers terminate as free nerve endings within the dental pulp *(a)* and arise from primary afferent cell bodies within the trigeminal ganglion *(b)*. The central processes of these primary afferent cell bodies pass into the brainstem and enter the trigeminal tract. These fibers exit the tract to terminate within the trigeminal sensory nucleus *(c)*, composed of the main sensory nucleus (MSN) and the spinal trigeminal nucleus. The trigeminal nucleus consists of pars oralis (PO), pars interpolaris (PI), and pars caudalis (PC). *(a)* Pulpal nerve fibers are seen within the pulp horn of a human specimen and are stained with antibodies against N52 *(green)*, PGP9.5 *(blue)*, and TRPA1 *(red)*. A nerve fiber bundle *(arrowhead)* gives rise to an extensive arbor within the subodontoblastic plexus and with some fibers that enter and traverse *(arrow)* the odontoblastic layer (O). *(b)* Neuronal cell bodies are seen within the rat trigeminal ganglion and are stained with antibodies against peripherin *(green; black arrow)*, TRPV1 *(blue; arrowhead)* and CGRP *(red; white arrow)*. Larger cell bodies lack staining, while the smaller cell bodies are stained individually or multiply with these antibodies used to identify nociceptors. *(c)* Intrinsic neuronal cell bodies are stained with NeuN *(green; arrow),* and the central processes of CGRP-containing primary afferent fibers *(red)* are seen within a transverse section of the rat brainstem at the level of caudalis. The CGRP-containing primary afferent fibers are located in the trigeminal tract (T). Some of these fibers exit the tract *(arrowhead)* to enter and terminate especially within the superficial laminae I and II outer (o) zones of caudalis, where they form synapses with processes of intrinsic and descending neurons.

the spinal trigeminal nucleus. The spinal trigeminal nucleus is further subdivided into the following subnuclei: pars oralis (most rostral), pars interpolaris, and pars caudalis (most caudal).[22,23]

Animal studies have shown that primary afferent neurons that innervate dental pulp terminate in all of the different subnuclei located within the ipsilateral trigeminal sensory nucleus, including prominent projections to caudalis.[24] The projection to cau-

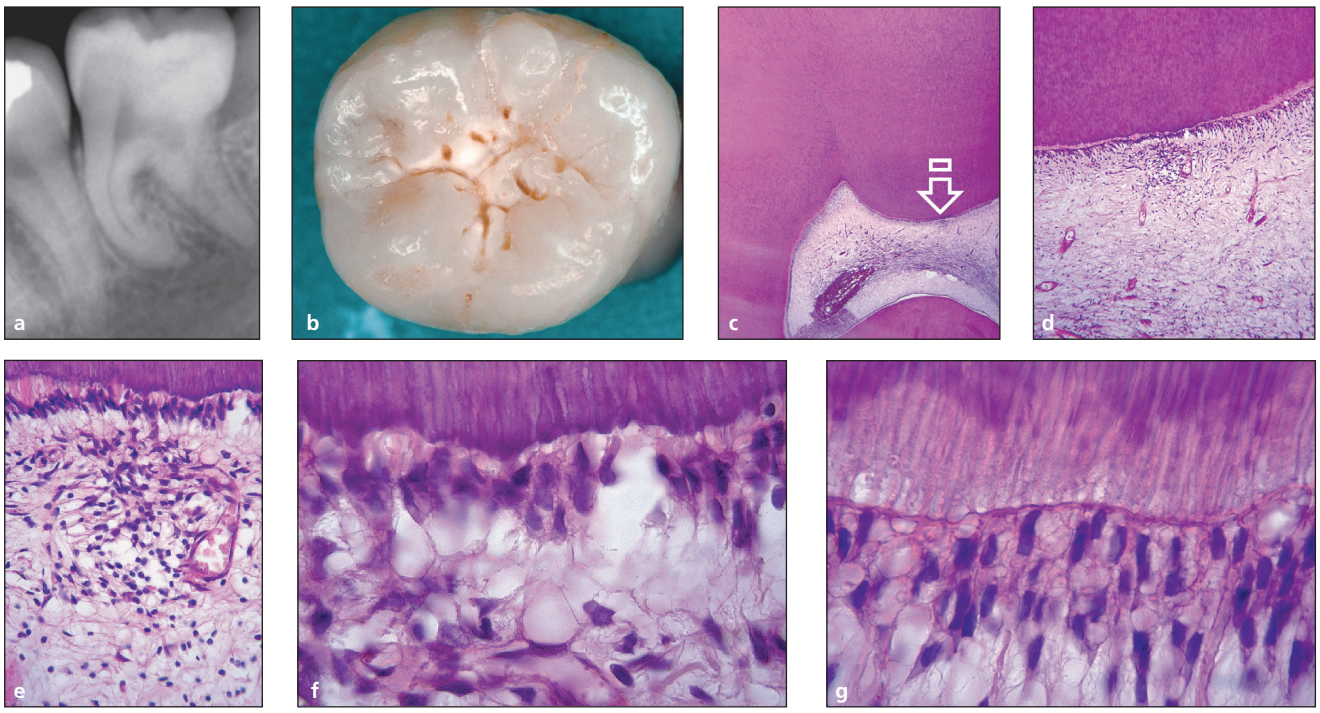

Fig 14-8 *(a)* Mandibular third molar of a 23-year-old woman. The radiograph does not show a caries lesion, but the tooth was extracted after repeated episodes of pericoronitis. *(b)* Fissure discoloration is present on the occlusal surface. The tooth was processed for light microscopy. Sections were cut on a mesiodistal plane. *(c)* Overview of the pulp chamber and the entire dentin thickness (hematoxylin-eosin [H&E] stain; original magnification ×25). *(d and e)* Progressive magnifications of the region indicated by the arrow in *(c)*. Moderate accumulation of chronic inflammatory cells in a localized area of the subodontoblastic space (H&E stain; original magnification ×100 and ×400, respectively). *(f)* Disruption of the odontoblastic layer in that region (H&E stain; original magnification ×1,000). *(g)* Normal odontoblastic layer at a very short distance from the affected region (H&E stain; original magnification ×1,000). (Modified from Ricucci[54] with permission.)

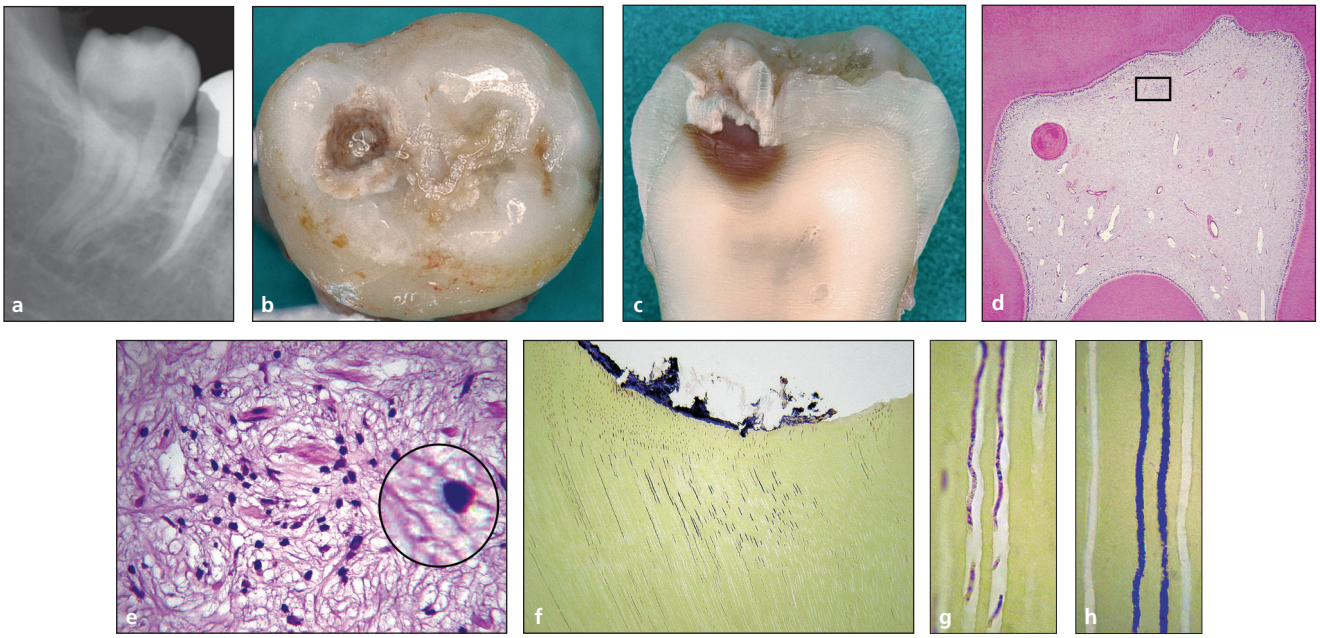

Fig 14-9 *(a)* Occlusal caries is present in this mandibular third molar. The pulp responded normally to sensitivity tests. The tooth was extracted. *(b)* Occlusal view of the extracted tooth. *(c)* The extent of dentinal caries became evident after a surface of the tooth was ground. *(d)* Despite caries penetration to the midcoronal dentin, most of the sections of the pulp exhibited normal histology. A pulp stone could be identified in the distal part of the pulp chamber (H&E stain; original magnification ×25). *(e)* Magnification of the rectangular area in *(d)*. Sparse accumulation of lymphocytes (H&E stain; original magnification ×400). *(f to h)* Dentinal tubules directly beneath the caries lesion were colonized by bacteria (Taylor's modified Brown & Brenn stain; original magnification ×100, ×1,000, and ×1,000, respectively). (Modified from Ricucci[54] with permission.)

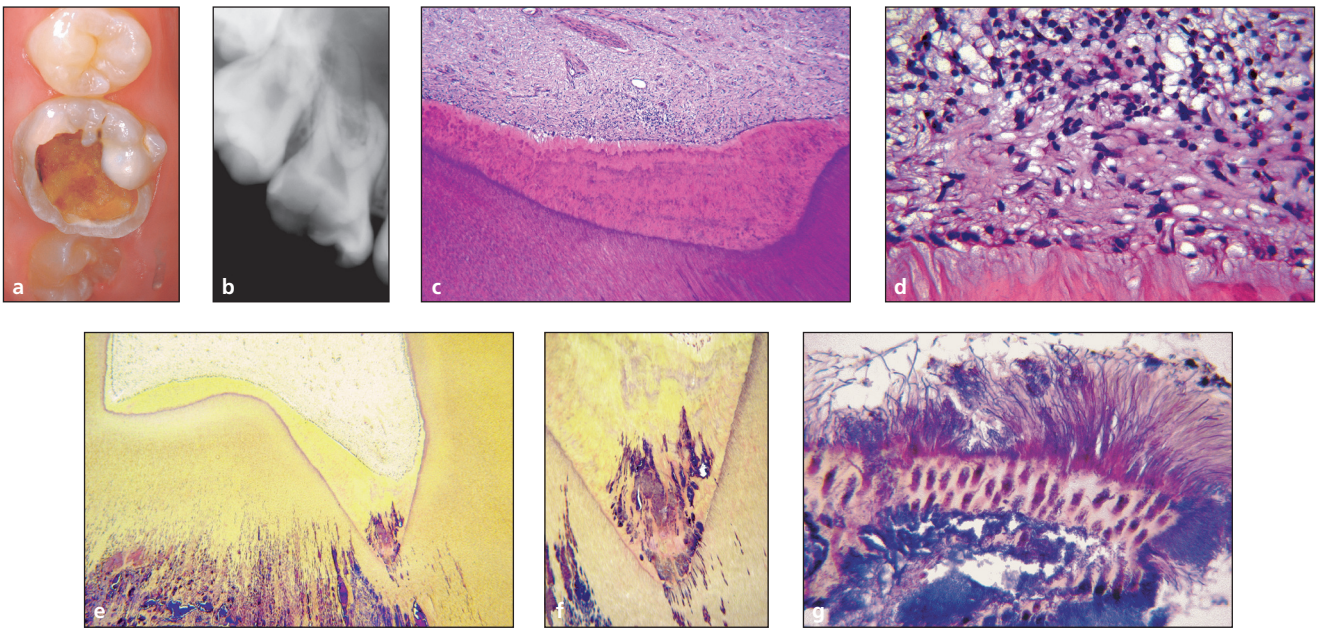

Fig 14-10 *(a)* Maxillary first molar of an 11-year-old girl whose crown has been destroyed by extensive caries. The tooth was painful to cold and sweet stimuli, but there was no spontaneous pain. Sensitivity tests (hot, cold, and electric) elicited exaggerated responses. The young patient and her parents requested extraction. *(b)* The radiograph confirms the severity of the destruction but does not show any periapical changes. *(c)* A considerable amount of reactionary dentin is present on the pulpal side beneath the pulp chamber roof (H&E stain; original magnification ×50). *(d)* The reactionary dentin is bordered by an incomplete layer of flattened odontoblasts. Beneath the odontoblastic layer, a high concentration of chronic inflammatory cells can be seen (H&E stain; original magnification ×400). *(e)* Overview of the caries-infected dentin, the reactionary dentin, and the pulp. In the mesial pulp horn, the reactionary dentin is colonized by bacteria (Taylor's modified Brown & Brenn stain; original magnification ×25). *(f)* Higher magnification of the mesial pulp horn in *(e)* (Taylor's modified Brown & Brenn stain; original magnification ×100). *(g)* Dentin chip at the external surface of the caries lesion. The dentinal tubules are completely filled with bacteria. This spicule provides attachment to a biofilm that is composed primarily of filamentous bacteria (Taylor's modified Brown & Brenn stain; original magnification ×1,000). (Modified from Ricucci[54] with permission.)

tions may become acute and uncontrolled as bacteria approach and penetrate the pulp (Fig 14-11).

Although inflammation may be regarded as a defense response to injury, severe reactions can result initially as localized microabscesses. Further ingress of bacteria into the pulp produces clinically identifiable abscesses (Fig 14-12) that eventually result in pulpal necrosis and development of periradicular lesions[70] (Fig 14-13). The end result of adaptive immunity is an exaggerated inflammatory response intended to eliminate the infection. However, if the source of caries infection is not eliminated, immune inflammation in pulpitis eventually leads to irreversible destruction of the pulp.

Repair responses

The pulpodentin complex reacts to stimuli from the bacterial biofilm with dentinal sclerosis and tertiary dentin formation.[71] Dentinal sclerosis has been discussed in previous sections. Unlike primary and secondary dentinogenesis, tertiary dentinogenesis is restricted to the vicinity of the dentin that is directly affected by the caries process. It is not unusual to see partial obliteration of the dental pulp by tertiary dentin in slowly progressing caries lesions that have not undergone restorative treatment.

Tertiary dentinogenesis has been redefined in relation to the nature of the injury (see chapter 2). The term *reactionary dentinogenesis* has been

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