# 1 Pulp and Periradicular Pathways, Pathosis, and Closure

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## PULP AND PERIRADICULAR PATHWAYS

The root canal system and the periodontium communicate through natural and artificial (iatrogenic) pathways. The pulp tissue is encased in the root canal system, is surrounded by dentin, and communicates with the periodontium through the apical foramen and occasionally, small channels known as accessory or lateral canals. Iatrogenic pathways of communication between the root canal system and the periodontium are created during accidental procedures such as perforations during root canal treatment. In addition, removal of enamel and dentin by decay or by traumatic injuries as well as removal of cementum during periodontal treatment may result in communication between the root canal system, its dental pulp, and the periodontium.

## NATURAL PATHWAYS

The natural pathways of communication between the root canal system and the periodontium include the apical foramen, lateral canals, and dentinal tubules.

### Apical foramen

The apical openings of roots are the main pathways between the root canal system, its contents, and the periradicular tissues (cementum, periodontal ligament, and alveolar bone). The apical foramen is initially very large (Fig. 1.1). As tooth eruption and its formation continue, the root canal space is narrowed by apposition of dentin and the apical foramen is modified by cementum

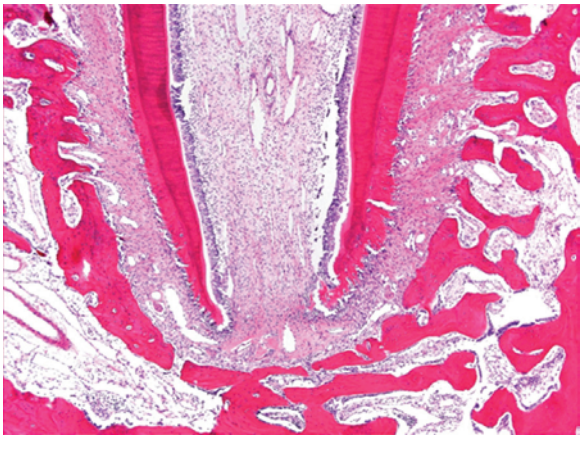

**Fig. 1.1** Newly erupted teeth have large root canals with wide-open apices.

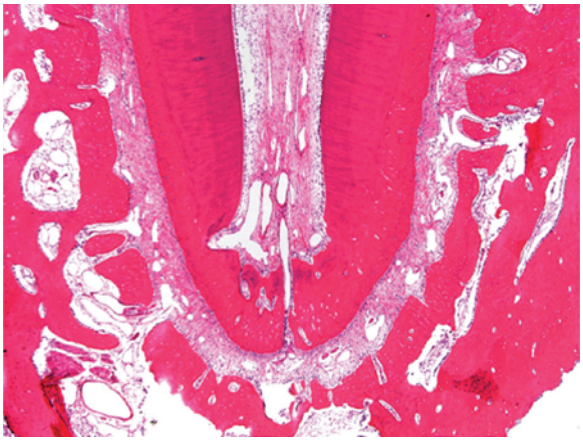

**Fig. 1.2** As a tooth erupts, its root canal space is narrowed by apposition of dentin and its apical foramen is modified by cementum apposition.

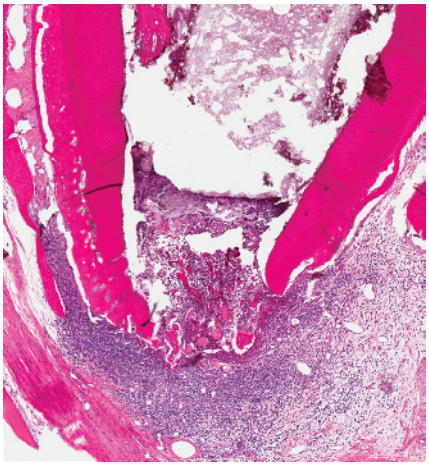

**Fig. 1.3** Egress of irritants through the apical foramen into periapical tissues initiates a periapical lesion and destruction of periradicular tissues.

apposition (Fig. 1.2). Continuous passive eruption of the teeth and mesial drifting cause apposition of new layers of cementum at the root apices. As the tooth matures, the apical foramen is reduced in size. Single-rooted teeth usually have a single foramen. However, multi-rooted teeth often contain multiple foramina at each apex (Green 1956, 1960).

Egress of irritants from pathologically involved necrotic pulps via the apical foramen into periapical tissues initiates and perpetuates an inflammatory response and its consequences, such as destruction of apical periodontal ligament and resorption of bone, cementum, and even dentin (Fig. 1.3).

## Lateral canals

When the epithelial root sheath breaks down before the root dentin is formed, or the blood vessels that run between the dental papilla and dental sac persist, a direct contact may be established between the periodontal ligament and the dental pulp. This channel of communication is called a lateral or accessory canal. In general, lateral canals occur more frequently in posterior teeth than in anterior teeth and more frequently in the apical portions of roots than in their coronal segments (Hess 1925; Green 1955; Seltzer *et al*., 1963) (Fig. 1.4). The incidence of lateral canals in the furcation of multi-rooted teeth is reported to be as low as 2 to 3% and as high as 76.8% (Burch & Hulen 1974; De Deus 1975; Vertucci & Anthony 1986). Despite these variations, there is no doubt that a patent lateral canal can contain and carry toxic substances from the root canal system into the periodontium and induce periradicular inflammation.

## Dentinal tubules

The dentinal tubules extend from the pulp to the dentinoenamel and cementodentinal junctions. The diameters of these tubules are approximately 2.5 µm near the pulp and about 1 µm at the dentinoenamel and cementodentinal junctions (Garberoglio & Brännström 1976). Although an actual count of the dentinal tubules has not been performed, their numbers are high, with approximately 15,000 dentinal tubules present in a square millimeter of dentin near the cementoenamel junction (Harrington 1979). The dentinal tubules contain tissue fluid,

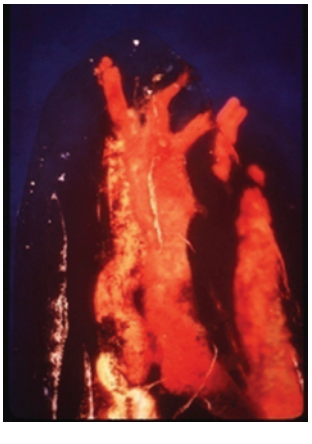

**Fig. 1.4** Presence of multiple lateral canals at the end of the mesiobuccal root of a maxillary first molar. Courtesy of Dr. John West.

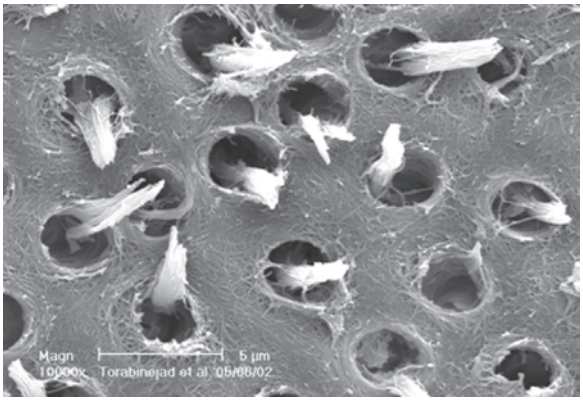


**Fig. 1.5** An SEM picture of dentinal tubules containing odontoblastic processes.

odontoblastic processes, and nerve fibers (Fig. 1.5). As the tooth ages or experiences irritation, these tubules tend to reduce in diameter or calcify, thus reducing patency. A continuous layer of cementum on the root surface is an effective barrier for the penetration of bacteria and their byproducts into the root canal system. Congenital absence of cementum, caries, or removal of the cementum during periodontal treatment or vigorous tooth-brushing may result in the opening of numerous patent small channels of communication between the pulp and the periodontium. Theoretically, these tubules could carry the toxic metabolites produced during pulpal or periodontal disease in both directions.

## PATHOLOGICAL AND IATROGENIC PATHWAYS

The pathological and iatrogenic pathways of communication between the root canal system and the oral cavity as well as the root canal system and the periodontium include carious pulp exposure, root perforation during access preparation, cleaning and shaping, post preparation and vertical fracture during obturation.

### Dental caries

Carious dentin and enamel contain numerous species of bacteria such as *Streptococcus mutans*, lactobacilli, and actinomyces (McKay 1976). The presence of these microbes elicits toxins that penetrate through the dentinal tubules into the pulp. Studies have shown that even small lesions in the enamel are capable

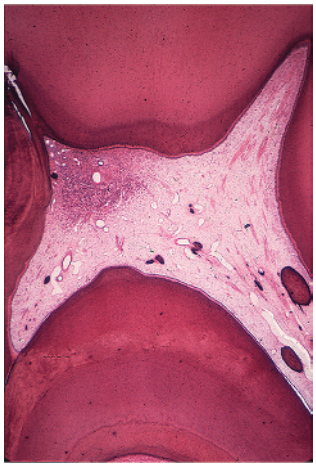

**Fig. 1.6** Presence of severe inflammatory response at the site of carious exposure in a human molar.

of attracting inflammatory cells into the pulp tissue (Brännström & Lind 1965; Baume 1970). As a result of the presence of microorganisms and their byproducts in dentin, the pulp tissue is infiltrated locally (at the base of tubules involved in the caries) by chronic inflammatory cells such as macrophages, lymphocytes, and plasma cells. As the decay progresses toward the pulp, the inflammatory process markedly changes in intensity and character (Fig. 1.6). Upon exposure, the pulp is infiltrated predominantly by polymorphonuclear (PMN) leukocytes to form an area of liquefaction necrosis at the site of pulp exposure (Lin & Langeland 1981). Bacteria can colonize in the area of liquefaction necrosis and persist. Pulpal tissue may stay inflamed for long periods before undergoing eventual necrosis, while in other instances the pulp may die quickly. Virulence of bacteria, host resistance, amount of circulation, and most importantly, the amount of drainage play a major role in this process.

### *Role of microorganisms*

As a consequence of pulp exposure to the oral cavity, the root canal system acquires the ability to harbor bacteria and their byproducts. Because of its location, general lack of collateral circulation, and its low compliance (Van Hassel 1971; Heyeraas 1989), the pulp does not have the ability to defend itself against the invading bacteria. Sooner or later the bacterial infection will spread throughout the root canal system and the bacteria and/or bacterial byproducts will diffuse from the root canal into the periradicular tissues with resultant development of a periradicular lesion.

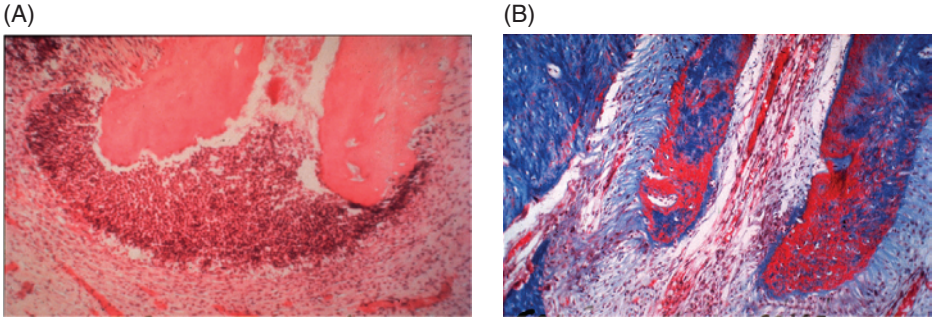


**Fig. 1.7** (A) Presence of a periapical lesion in the molar of a rat exposed to bacteria present in its normal micro flora. (B) Absence of pulp and periapical pathosis in a molar of a germ-free rat exposed to its oral cavity. Source: Kakehashi 1965. Reproduced with permission of Elsevier.

To show the importance of bacteria in pathogenesis of pulp and periradicular diseases, Kakehashi *et al*. (1966) exposed the dental pulps of conventional and germ-free rats to their oral flora. Pulpal and periapical lesions were developed in conventional rats. In contrast, they were absent in germ-free rats. (Fig. 1.7). Möller and associates (1981) sealed sterile and contaminated dental pulps in the root canals of monkeys. After 6–7 months, their clinical, radiographic, and histological examinations showed absence of any pathologic changes in the periapical tissues in teeth that had been sealed with sterile amputated pulps. In contrast, teeth sealed with infected pulps had developed inflammatory reactions in their periapical tissues. These studies show the importance of microorganisms in the pathogenesis of pulpal and periapical lesions.

## Root perforations

Roots may be perforated during access preparation, cleaning and shaping, or post space preparation.

### *Root perforations during access preparation*

Lateral surface or furcation perforations can occur during access preparations (Fig. 1.8). Lack of attention to the degree of axial inclination of a tooth in relation to its adjacent teeth and failure to parallel the bur with the long axis of a tooth can result in gouging or perforation (see Chapter 7).

Searching for the pulp chamber or the orifices of the root canals through an under-prepared access cavity can also result in accidents. Failing to recognize

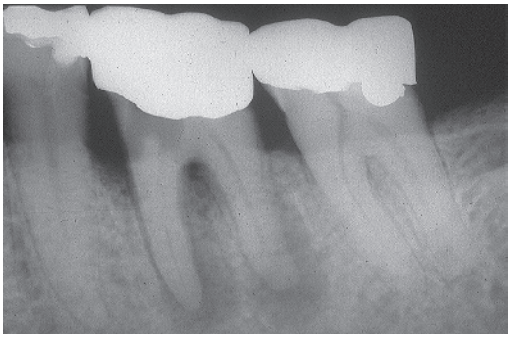

**Fig. 1.8** Searching for orifices of the root canals through an under-prepared access cavity can result in lateral root perforation.

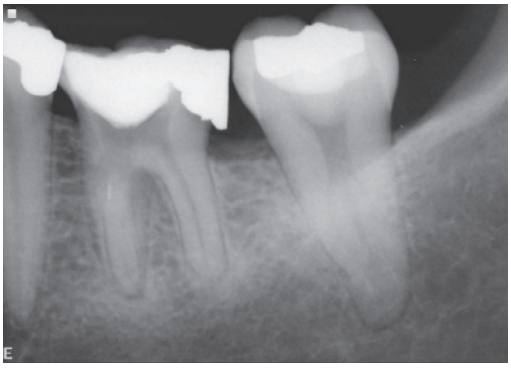

**Fig. 1.9** Failure to recognize the depth of bur penetration during an access preparation can cause gouging or furcation perforation.

when the bur passes through a small or flattened calcified pulp chamber in multi-rooted teeth may cause gouging or perforation in the furcation (Fig. 1.9). Furcation perforations can also occur during post space preparation.

### *Root perforations during cleaning and shaping*

Roots may be perforated at different levels during cleaning and shaping. The level of root perforation is critical, that is, whether the perforation is apical, mid-root, or cervical. The level directly affects treatment and prognosis. The further the perforation is from the crestal bone, the better its prognosis. Apical perforations may occur either directly through the apical foramen or through the body of the root itself. Instrumentation of the root canal beyond its anatomic apical foramen results in perforation of the apical foramen. Incorrect working length or inability to maintain proper working length results in apical root perforation.

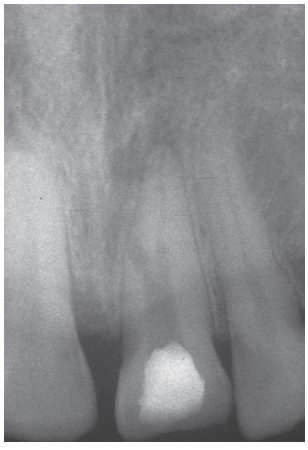

**Fig. 1.10** Misdirected pressure and forcing a file during cleaning and shaping can result in a lateral-root perforation.

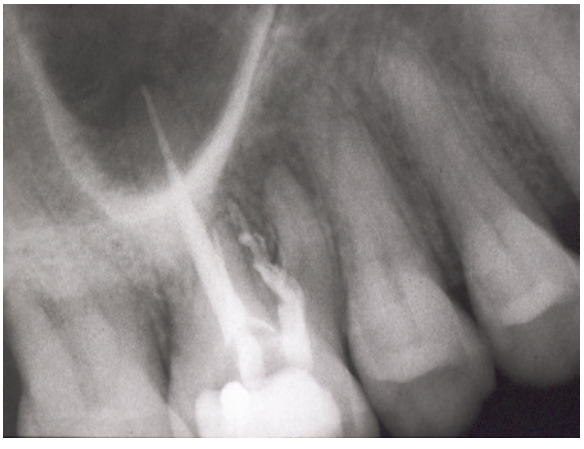

**Fig. 1.11** Coronal root perforations can occur by over enlarging canals by files, Gates–Glidden drills, or Peeso reamers. Courtesy of Dr. George Bogen.

Penetration of the last file beyond the radiographic apex is evidence of such a procedural accident. Lateral root perforations are usually a result of the inability of an operator to maintain the curvature of a canal during negotiation of a root canal or after ledge formation. Negotiation of ledged canals is not always possible; misdirected pressure and the forcing of a file may result in the formation of a new canal and eventually a lateral root perforation (Fig. 1.10). Coronal root perforations occur as a result of misdirected burs when the operator is attempting to locate canal orifices. They are also produced by over-enlarging canals by files, Gates-Glidden drills, or Peeso reamers (Fig. 1.11).

### *Root perforations during post space preparations*

Root perforations can occur during post space preparations if the post space is too large or misdirected in the root. Ideally, a post space is a conservative enlargement of the prepared canal space with an optimal length for retention and adequate remaining root canal filling to provide adequate apical seal. The post should be parallel with long access of the root. Its width should not exceed a third of the width of the root and its length should not be more than two-thirds of the working length (Fig. 1.12). Preferably, the preparation should be performed primarily with hand instruments.

### *Vertical fracture*

Although other factors such as post placement and restoration may be co-factors, the principal etiologic factor is associated with root canal treatment procedures (Gher *et al*. 1987). Apparently this results from an overzealous application of condensation forces to obturate an underprepared or over-prepared canal with subsequent vertical root fracture (Holcomb *et al*. 1987). The best means for prevention of vertical root fractures is appropriate canal preparation as well as use of balanced pressure during obturation.

Radiographically, a frank root fracture (Fig. 1.13) or lack of sharp demarcation between an irregular and poorly condensed filling material and the dentinal walls also indicates presence of a vertical root fracture. Long-standing vertical

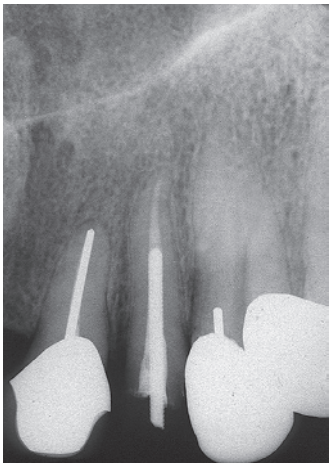

**Fig. 1.12** An ideal post should be parallel with the long access of the root, its width should not exceed a third of the width of the root and its length should not be more than two thirds of the working length.

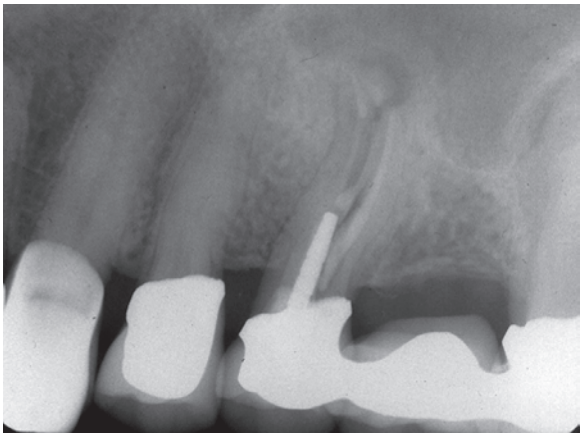

**Fig. 1.13** A frank vertical root fracture is usually associated with a narrow periodontal pocket and/or sinus tract stoma and a lateral radiolucency extending to the apical portion of the root.

root fractures often are associated with a narrow periodontal pocket and/or sinus tract stoma and a lateral radiolucency extending to the apical portion of the vertical fracture.

## PERIRADICULAR PATHOSIS

In contrast to pulp tissue, periradicular tissues (periodontal ligament and bone) have an almost unlimited source of undifferentiated cells that can participate in the process of inflammation as well as repair. In addition, the periradicular tissues have rich collateral blood supply and lymph drainage. These characteristics enable the periradicular tissues to combat the destructive factors related to the irritants from the root canal system.

### Inflammatory process of periradicular lesions

Depending on the severity of irritation, its duration, and host response, periradicular pathosis of pulpal or iatrogenic origin can range from slight inflammation to extensive tissue destruction. Injury to periradicular tissues usually results in cellular damage and the release of nonspecific as well as specific immunologic mediators of inflammatory reactions (Torabinejad *et al*. 1985) (Fig. 1.14). Physical or chemical injury to the periradicular tissues during root canal therapy can cause a release of vasoactive amines such as histamine, activation of the Hageman factor, activation of the kinin system, the clotting cascade, the

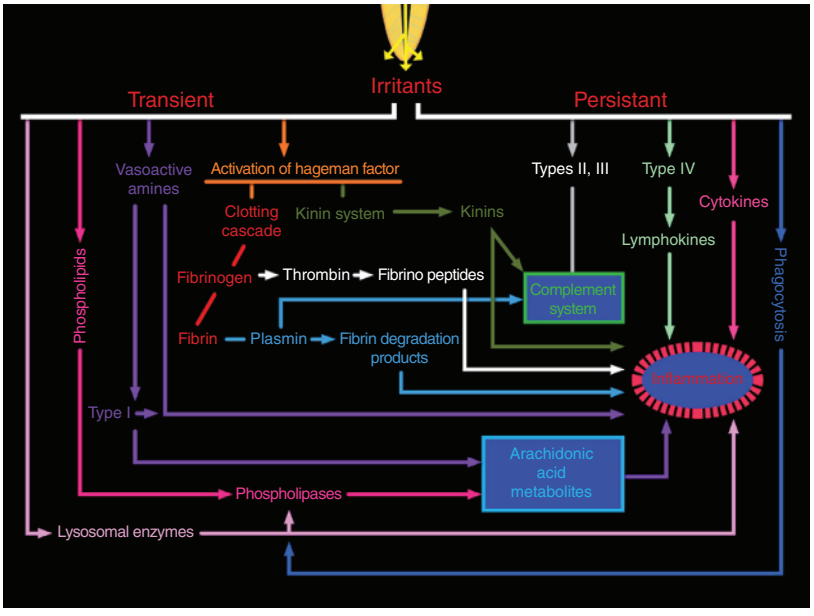


**Fig. 1.14** Egress of irritants from an infected root canal into periapical tissues can result in activation of nonspecific as well as specific immunologic mediators of inflammatory reactions.

fibrinolytic system, and the complement system with its release of C3 complement fragments in human periradicular lesions (Pulver *et al*. 1978). Release of these factors can contribute to the inflammatory process in the periradicular tissues and cause inflammation, swelling, pain, and tissue destruction. Inhibition of formation of periapical lesions by systemic administration of indomethacin in cats shows the importance of another group of nonspecific mediators of inflammation (the arachidonic acid metabolites) in the pathogenesis of periradicular lesions (Torabinejad *et al*. 1979).

In addition to the mediators of nonspecific inflammatory reactions, immunologic reactions can also participate in the formation and perpetuation of periapical lesions (Fig. 1.14). Presence of various immunologic *factors* (i.e., antigens, immunoglobulin E (IgE), mast cells in pathologically involved dental pulps and periapical lesions) indicates that a type I immunologic reaction can occur in periapical tissues. Various *classes* of immunoglobulins and different types of immunocompetent cells, such as PMN leukocytes, macrophages, B and T cells, C3 complement fragments, and immune complexes, have been found in human periapical lesions (Torabinejad & Kettering 1985). Presence of these components in periapical lesions indicates that types II, III, and IV immunologic reactions can also participate in the genesis of these lesions.

## MATERIALS TO SEAL THE PATHWAYS TO THE ROOT CANAL SYSTEM AND THE PERIODONTIUM

Numerous materials have been suggested to seal off the communication between the root canal system and external surfaces of the tooth. They include: gutta-percha, amalgam, polycarboxylate cements, zinc phosphate cements, zinc oxide eugenol paste, IRM cement, EBA cement, Cavit, glass ionomers, composite resins, and other materials such as gold foil and leaf, silver points, cyanoacrylates, polyHEMA and hydron, Diaket root canal sealer, titanium screws, and Teflon. For years, existing materials did not possess the "ideal" characteristics of a repair material, and therefore an experimental material, mineral trioxide aggregate (MTA), was developed in 1993.

In a series of tests, Torabinejad and associates investigated *in vitro* dye leakage with and without blood contamination, *in vitro* bacterial leakage, scanning electron microscope (SEM) examination of replicas for marginal adaptation, setting time, compressive strength, solubility, cytotoxicity, implantation in bone, and a usage test in animals (Torabinejad *et al*. 1993; Higa *et al*. 1994; Pitt Ford *et al*. 1995; Torabinejad *et al*. 1995a, b, c, d, e, f, g; Tang *et al*. 2001). Existing materials, such as amalgam, Intermediate Restorative material (IRM), or SuperEBA (*O*-ethoxybenzoic acid) were used for comparison. The sealing ability of MTA was superior to that of amalgam and SuperEBA in both dye, bacterial, and endotoxin leakage methods and was not adversely affected by blood contamination (Torabinejad *et al*. 1993, 1995a; Higa *et al*. 1994; Tang *et al*. 2001). The marginal adaptation of MTA was better than that of amalgam, IRM, and SuperEBA (Torabinejad *et al*. 1995 g). The setting time of MTA was found to be less than three hours, which is much longer than that of amalgam and IRM. Compressive strength and solubility of MTA were similar to that of IRM and SuperEBA, respectively (Torabinejad *et al*. 1995c). It also has some antibacterial effects on some of the bacterial species in the oral cavity (Torabinejad *et al*. 1995e).

The cytotoxicity of MTA was investigated by two methods, agar overlay and radiochromium release. MTA was ranked less cytotoxic than IRM and SuperEBA, but more cytotoxic than amalgam in the agar overlay method. It was found to be less cytotoxic than amalgam, IRM, and SuperEBA when the radiochromium release method was used (Torabinejad *et al*. 1995f). With implantation of materials in guinea pig mandibles and tibias, MTA was more biocompatible than other test materials (Torabinejad *et al*. 1995d). Root-end fillings or furcation perforations repaired with MTA or amalgam placed in the teeth of dogs and root-end filling in monkeys were examined histologically (Pitt Ford *et al*. 1995; Torabinejad *et al*. 1995b, 1997). There was less inflammation around the root ends filled with MTA, with the evidence of healing in the surrounding tissues. In

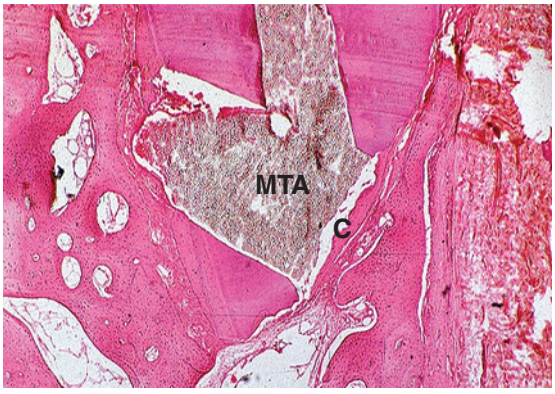


**Fig. 1.15** Cementum (C) formation was found on the surface MTA when it was used as a root-end filling in dogs.

addition, with the longer-term teeth filled with MTA new cementum was found on the surface of the material when used as a root-end filling or furcation perforation material; this was not the case with amalgam (Fig. 1.15). Based on these studies, it appears that MTA is an alternative material to be used for root-end fillings.

Since its introduction, numerous studies have been published regarding various aspects of this material. Parirokh and Torabinejad (2010a) conducted a search (electronically and manually) of the literature regarding the chemical and physical properties and antibacterial activity of MTA from November 1993 to September 2009. Their search showed that there are many published reports regarding the properties of MTA and a material composed of calcium, silica, and bismuth. It has a long setting time, high pH, and low compressive strength. It possesses some antibacterial and antifungal properties depending on its powder-to-liquid ratio. Based on their search, they concluded that MTA is a bioactive material that influences its surrounding environment. In the second part of their review, Torabinejad and Parirokh (2010) conducted a comprehensive literature search using electronic and manual methods for the sealing ability and biocompatibility of MTA from November 1993 to September 2009. Their review showed the presence of numerous studies regarding these properties of MTA. Based on the available evidence, they concluded that MTA seals well and is a biocompatible material. In the third part of their literature review Parirokh and Torabinejad (2010b) conducted a comprehensive review of the literature regarding the clinical applications of MTA in experimental animals and humans, as well as its drawbacks and mechanism of action from November 1993 to September 2009. Their search of the literature shows that MTA is a promising material for root-end filling, perforation repair, vital pulp therapy, and apical barrier formation for teeth with necrotic pulps and open apices. Furthermore, they reported that MTA has some known drawbacks such as a long setting time, high cost, and potential for discoloration. Regarding its mode of action, it appears that hydroxyapatite crystals form over MTA when it comes in

contact with tissue synthetic fluid. This can act as a nidus for the formation of calcified structures, following the use of this material in endodontic treatments.

Based on the available information, they concluded that MTA is the material of choice for sealing the pathways of communication between the root canal system and its external surfaces.

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