The hard tissues (dentin, cementum, and enamel) of permanent teeth do not normally undergo resorption. When resorption of permanent teeth is observed clinically, the resorption is usually the result of trauma; chronic inflammation of the pulp, periodontal tissues, or both; or induced pressure in the periodontal ligament associated with orthodontic tooth movement, tumors, or tooth eruption.

Resorption on the external root surface usually accompanies simultaneous reactions within the alveolar bone; the process of tooth resorption is considered to be similar to that of bone resorption. Resorption of alveolar bone occurs as a result of local inflammation and as part of life-long remodeling of the jaws. The resorption is primarily by osteoclasts, but other cells, such as macrophages, monocytes, and osteocytes, have been reported to have bone-resorbing ability.

Osteoclasts are large, multinucleated cells that originate from blood-borne leukocytes from the bone marrow; the precursor cell is from the monocyte cell line. Osteoclasts locate near the bone surface or in bay-like erosions called Howship’s lacunae at the bone surface. Toward the bone surface, they have ruffled borders delineated by clear zones. Osteoclasts resorb bone by releasing demineralizing agents and degrading enzymes into Howship’s lacunae under the ruffled border, then ingest the bone degradation products by phagocytosis. Osteoclasts also have an important role in the inflammatory response to infection, as seen in resorption of necrotic bone in osteomyelitis and root and bone resorption in inflammatory resorption of teeth with pulp necrosis, infection, or both.

Resorption of the teeth may occur as a result of inflammatory conditions, mechanical stimulation, or neoplastic processes. The resorptive process of the dental tissues is similar to that of bone, but with some notable differences. The dentin-resorbing cells (dentinoclasts) have fewer nuclei and are smaller than the osteoclasts. Dentinoclasts have very small or no clear zones in contrast to the well-developed clear zones of actively resorbing osteoclasts. This has been attributed to the difference in composition of the dental tissues when compared with bone.

PHYSIOLOGIC MECHANISMS INHIBITING TOOTH RESORPTION

On the external root surface, the teeth are surrounded by the periodontal ligament (PDL), which is a specialized connective tissue that attaches the tooth to the surrounding bone and acts as a barrier between the alveolar bone and cementum. It is believed that cells of the PDL are responsible not only for osteogenesis and osteoclastosis, but also for fibrogenesis and fibroclasis in the ligament itself, as well as for cementogenesis and cementoclastosis on the root surface. In addition, it is theorized that, by interactions between bone cells and soft connective tissue cells of the ligament, territorial boundaries are established, which result in maintenance functions of the periodontal ligament.
of the PDL space. Furthermore, it is speculated that the cellular boundaries may be influenced to expand, shrink, or disappear by other external stimuli, such as inflammation, mechanical stimulation, or trauma.\(^8,14\)

Some studies\(^15,16\) have demonstrated that a low-molecular-weight proteolytic activity inhibitor, the anti-invasion factor, is present in cartilage, blood vessel walls, and teeth. When extracts containing this inhibitor were placed in vitro with bone cells, osteoclasts lost their ruffled borders, as well as their attachment to bone, and bone resorption was inhibited. It was also shown that when newly extracted teeth with remnants of the PDL were placed in physiologic saline solution, a protease inhibitor similar to the anti-invasion factor was released.\(^16\) In another study,\(^17\) explants of vital PDL inhibited invasion of cells associated with bone, whereas the absence of viable PDL cells led to invasion by bone cells. Hence, it may be inferred that anti-invasion factors in the periodontal tissues play a role in maintaining integrity of the tooth root.

Cementum has a composition similar to bone but is less readily resorbed than bone.\(^12,16\) The cervical two thirds of the root is covered by acellular cementum. Cellular cementum is mainly found toward the apical third of the root and contains cementocytes enclosed in lacunae similar to osteocytic lacunae.\(^12,18\) Beneath the cementum, adjacent to root dentin, is a structureless zone that seals the peripheral end of the dentinal tubules; this is the hyaline layer of Hopewell-Smith,\(^19\) also termed the intermediate cementum.\(^20\) The intermediate cementum is hypercalcified\(^21\) in relation to adjacent dentin and cementum. It is critical for preventing development of inflammatory resorption in replanted teeth with pulpal pathosis, possibly by forming a barrier against egress of noxious agents from the dentinal tubules to the PDL.\(^8,22\)

**EXTERNAL RESORPTION ASSOCIATED WITH TRAUMATIC INJURIES**

**Surface resorption**

If there is injury or irritation to the dentin, cementum, or PDL, clastic (resorbing) cells will be attracted to the affected areas of the root surface, and resorption will occur as part of the normal scavenging function of the cells.\(^12,23\) Cellular scavenging (phagocytosis) of tissue debris and invading microbes is an integral aspect of the inflammatory response to tissue injury. Phagocytosis is carried out by elements of the mononuclear phagocyte system, consisting of neutrophils and mononuclear phagocytes; osteoclasts are also described as participants in the debridement of hard tissue wounds.\(^8\) Mononuclear phagocytes arise as monocytes in the bone marrow. They enter the blood as monocytes and become macrophages (tissue histiocytes) in the connective tissue. Macrophages migrate and respond to the site of injury by macrophage chemotactic factors derived from bone and tissue breakdown products. Macrophages

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**Fig 1.** Right central incisor was avulsed and replanted at age 10. Radiograph (10 years after injury) and extracted tooth depict massive inflammatory resorption with concomitant alveolar bone destruction.
possess large numbers of heterogeneous dense cytoplasmic granules that contain acid hydrolases, capable of digesting tissue debris and foreign matter. These phagocytes (resorbing cells) enter inflammatory foci with the express purpose of ingesting and disposing of unwanted particulate material, such as bacteria, broken-down cells, and tissue debris. However, it has been shown that resorbing cells require continuous stimulation for phagocytosis. This type of scavenging involved in initial wound debridement does not appear to be sufficient to sustain the resorption for more than 2 to 3 weeks. In the absence of additional stimulation of the cells, repair with cementum-like tissue usually occurs on both the external root surface and the root canal wall. Some studies have demonstrated that when there are only minor inflammatory changes, new PDL and cementum begin to form on the external root surface as early as 1 week after the injury. This type of limited resorption occurs with any injury to the PDL or cementum and is referred to as surface or transient resorption. The patient does not usually experience discomfort, and, on radiographic examination, the roots will likely appear normal because the resorptive defect is very small.

**Inflammatory resorption**

In more severe cases of trauma, or in cases in which the resultant inflammatory response is more intense, the damage to the cementum progresses to involve resorption of the intermediate cementum layer that caps the ends of the dentinal tubules. The dentinal tubules are then open and communicate with inflammatory resorptive cells (macrophages and osteoclasts) within the PDL and alveolar bone. If the pulp is necrotic and infected, bacterial
by-products escape by way of the dentinal tubules and become the stimulus for ongoing phagocytosis, causing inflammation and further root and bone resorption. If allowed to progress, the resorptive process may destroy the root and adjacent alveolar bone (Fig 1). This type of resorption may be effectively arrested by early removal of the necrotic, infected pulp, followed by suitable endodontic treatment.\(^{28,29}\) Some studies\(^{30,31}\) have shown that an age or maturation factor may be relevant because inflammatory resorption is more frequent in replanted immature and young mature teeth than in older mature teeth. It should also be noted that, for cases in which the pulp remains vital and uninflamed (without a leukocyte zone), resorption leading to repair will occur irrespective of the depth of the resorption cavity.\(^{22}\)

When inflammatory resorption is detected, calcium hydroxide (Ca(OH)\(_2\)) is often considered the intra-canial dressing of choice (Fig 2). Because of high pH and antibacterial properties, the use of Ca(OH)\(_2\) has been suggested to promote healing.\(^{23,28,29}\) Recommendations vary considerably as to the length of treatment time with Ca(OH)\(_2\) before obturation. A study\(^{32}\) examined the incidence of inflammatory resorption in avulsed teeth with a 25-minute extraoral drying time and with pulps removed within 4 weeks; there was no therapeutic benefit from an interim treatment with Ca(OH)\(_2\) when compared with immediate obturation with gutta-percha and sealer. However, other studies\(^{29,33}\) recommend a 1- or 2-week course of Ca(OH)\(_2\), followed by obturation with gutta-percha and sealer for cases in which an avulsed tooth is replanted within 1 hour and the pulp removed within 2 to 3 weeks. Furthermore, it is suggested that longer treatment with Ca(OH)\(_2\) is beneficial when there has been a delay in pulp removal or when inflammatory resorption is detected.\(^{29}\)

### Replacement resorption

In luxation injuries with loss of viability of PDL cells or in cases of tooth avulsion in which extensive damage to the ligament occurred because of drying or inappropriate storage, healing occurs without an intervening periodontal attachment. Depending on the extent of injury, cell lysis will occur on the root surface where the PDL has become necrotic. If the tissue damage is mild (with less than 20% of the root surface involved), the resorption may be transient and may repair itself with cells from adjacent healthy PDL.\(^{34,35}\) However, in the more typical situation involving teeth that have been out of the mouth long enough for cells on the root surface to desiccate and die, the condition is progressive and will result in eventual tooth loss.\(^{23}\) Here, instead of connective tissue cells participating in PDL repair, cells of the alveolar bone replace the periodontal attachment and continue to resorb the root, gradually replacing it with alveolar bone; thus the term replacement resorption is used. The root dentin is fused to the alveolar bone (ankylosis). It has been theorized\(^{12}\) that the protective cells (cementoblasts) of the root surface are replaced by an osteoblastic type of cell that responds to the normal factors involved in bone remodeling. Another explanation is that the root becomes part of the skeletal system and undergoes the same process of remodeling.

Clinically, ankylosis is recognized by a metallic sound on percussion, and the radiograph depicts absence of the lamina dura or PDL space, with the root and bone blending into a moth-eaten appearance (Fig 3). There is no effective treatment for dentoalveolar ankylosis. Ankyloic teeth may remain in function for several years; the speed of resorption seems to be dependent on the metabolism of the patient.\(^{23}\) A retrospective study\(^{31}\) concluded that in younger patients (8-16 years), a replanted tooth with necrotic PDL survived for 3 to 7 years, whereas in older patients (17-39 years) ankylosed teeth functioned for decades or for life. Because adjacent teeth continue to erupt, ankylosed teeth often display infraocclusion or submergence of the crown in the dentition in boys before age 16 and in girls before age 14.\(^{36}\) If detected, a technique for root submergence of the ankylosed root that allows for continued, normal devel-
Development of the alveolus for later prosthetic replacement of the tooth has been proposed. 37

EXTERNAL RESORPTION FROM PULP NECROSIS AND PERIRADICULAR PATHOSIS

Apical inflammation and resorption may result from necrosis and infection of the pulp. Resorption is enhanced by substances released from inflammatory cells in the surrounding tissues, such as osteoclast activating factor, macrophage chemotactic factor, and prostaglandins.12,23 This type of external resorption probably affects all teeth undergoing periradicular apical periodontitis; some studies have demonstrated that it is related to the presence of bacteria in the canal.38,39 In long-standing cases, there may be large areas of periapical bone resorption, but without much apically resorbed root dentin. This is in contrast to the quickly developing inflammatory resorption seen in traumatic injuries in which the dentinal tubules are patent and open to an infected pulp. This difference in development of external apical resorption from pulp necrosis may be attributed to the insulating capacity of the intermediate cementum, which remains unaffected when pulpal infection and necrosis are not caused by trauma. Proper debridement and obturation will arrest this type of inflammatory resorption and lead to both cemental and osseous repair.

EXTERNAL RESORPTION FROM PRESSURE IN THE PDL

Apical external resorption, resulting in shortening of the root, is a complication of orthodontic tooth movement. The etiology is not well understood. Histologically, the repaired resorption cavities have been described as very similar to surface resorption.40 Pressure resorption of permanent teeth is also seen with slow-growing tumors such as cysts, giant cell tumors, osteosclerosis, and other fibro-osseous lesions. Rapidly growing tumors tend to be more destructive to the bone than to roots.9 During eruption of permanent teeth, pressure resorption of maxillary laterals by the canines and mandibular second molars by adjacent third molars may occur. This type of resorption is usually arrested when the stimulus for resorbing cells is discontinued.9,23

INTERNAL ROOT RESORPTION

Internally, from the pulpal side, dentin is lined by the odontoblasts and predentin. The odontoblasts have no resorbing ability and, in combination with the unmineralized predentin, appear to form a barrier against dentin resorption.23,41 Internal resorption is preceded by chronic pulpal inflammation, a disappearance of the odontoblasts and predentin, and a pulpal invasion of macrophage-like resorbing cells.9,41 A study in which internal resorption was experimentally produced in monkeys’ incisors concluded that the lesions may be transient or progressive; the progressive type was stimulated by ongoing inflammation from infection.41

Internal resorption appears radiographically as a uniform, round to oval radiolucent enlargement of the canal (Fig 4). In most cases it is asymptomatic and

Fig 4. Internal resorption occurring in different areas.
detected on radiographic screening. Frequently, it is observed in the cervical region but may occur in all areas of the root canal system. If coronal, the tooth may demonstrate a pinkish hue because of the prolific capillaries in the pulpal inflammatory (granulomatous) tissue resorbing the coronal dentin and enamel.\textsuperscript{42}

When internal resorption is clinically detected, pulpectomy is needed to arrest the resorption. When resorption has progressed to involve an external communication with the PDL space, this complication is often so serious that the tooth cannot be maintained. In some situations, complex surgical procedures may be used to gain access for repair.\textsuperscript{42}

**CERVICAL RESORPTION**

Cervical resorption is a progressive external inflammatory resorption that appears to follow injury to the cervical attachment apparatus. The injury may be just below the epithelial attachment or more apical on the root surface where damage to the PDL or cementum has occurred.\textsuperscript{23,43} Cervical resorption may result from orthodontic movement, as a late complication of traumatic injuries, or after internal bleaching, periodontal treatment, or other orthognatic and dentoalveolar surgery.\textsuperscript{43,44}

In many instances, the history is obscure and thus is referred to as “idiopathic.” Except for bleaching-induced cervical resorption, cervical resorption may occur with an apparently healthy pulp; the resorptive process is neither stimulated nor sustained by necrotic pulp tissue.\textsuperscript{44}

With internal bleaching, the suggested stimulus for resorption is damage to the cervical attachment apparatus by chemical stimuli (30\% hydrogen peroxide) penetrating patent dentinal tubules.\textsuperscript{45,46}

The pathogenesis of cervical resorption has been described as beginning from a small area of damage to root cementum. The resorbing cells penetrate and spread the resorption into the root dentin. The stimulus for resorbing cells may be bacterial contamination in the gingival sulcus and along the affected root surface.\textsuperscript{23,43} Because of the presumed protective qualities of the predentin,\textsuperscript{23,41} the spreading of resorption occurs around the pulp canal, leaving intact a thin shelf of dentin over the pulp, which remains viable. If the resorption infiltrates the crown, the cervical coronal

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*Fig 5. Cervical resorption. Integrity of outline of root canal space (arrow) is superimposed over resorption lacuna. Tooth received root canal treatment; the resorptive defect was repaired with amalgam. Recall (lower right), 1 year after treatment.*
enamel may appear pink in color from the well-vascularized resorbing tissue. This may be confused with the previously described “pink tooth” of internal resorption. Cervical resorption has been designated extra-canial invasive resorption, invasive cervical resorption, or internal-external resorption because of this pattern of spreading within the root. Debridement and restoration of the resorption cavity is the recommended treatment (Fig 5). In many cases, root canal treatment will also be necessary when the resorption lacuna has progressed close to, or occasionally into, the pulp.

REFERENCES

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