Tooth resorption
Rita F. Ne, DDS*/David E. Witherspoon, BDSc, BEcon MS*/James L. Gutmann, DDS*

Tooth resorption is a common sequela following injuries to or irritation of the periodontal ligament and/or tooth pulp. The course of tooth resorption involves an elaborate interaction among inflammatory cells, resorbing cells, and hard tissue structures. The key cells involved in resorption are of the clastic type, which include osteoblasts and odontoclasts. Types of tooth resorption include internal resorption and external resorption. There are two types of internal resorption: root canal (internal) replacement resorption and internal inflammatory resorption. External resorption can be classified into four categories by its clinical and histologic manifestations: external surface resorption, external inflammatory root resorption, replacement resorption, and ankylosis. External inflammatory root resorption can be further categorized into cervical resorption with or without a vital pulp (invasive cervical root resorption) and external apical root resorption. Other variations of resorption include combined internal and external resorption and transient apical breakdown. (Quintessence Int 1999;30:9-25)

Key words: external resorption, inflammatory resorption, internal resorption, odontoclast, osteoclast, periodontal ligament, pulp, replacement resorption

KEY CELLS AND FACTORS INVOLVED IN THE MECHANISMS OF RESORPTION

Monocytes and macrophages
Monocytes and macrophages, along with osteoclasts, play an important role in bone and tooth resorption. They are found in tissue sections adjacent to bone-resorbing surfaces of rheumatoid arthritis, periodontal disease, periradicular granulomas and cysts, and in metastatic bone tumors. These cells play a critical role in the development and healing of all wounds. Initially, monocytes are recruited to the site of injury or irritation by the release of many pro-inflammatory cytokines. Subsequently, they differentiate into macrophages, whose major role is wound debridement. The migration and recruitment of macrophages into bone are regulated by macrophage chemotactic factors that are derived from bone and tissue breakdown products and are controlled by increased intracellular levels of adenosine 3',5'-cyclic phosphate (cAMP) and calcium. Although macrophages have a structure similar to that of osteoclasts and, like osteoclasts, can also become multinucleated giant cells, macrophages lack a ruffled border that is attached to hard tissue substrates during resorption and do not create lacunae on the dentinal surface.

Osteoclasts
Osteoclasts are bone-resorbing cells derived from hemopoietic cells of the monocyte-macrophage lineage.
Osteoclasts have a life span of approximately 2 weeks. The osteoclast is a very active cell; it has the ability to move between resorbing sites and is highly vacuolated with numerous mitochondria, indicating a high metabolic rate. It is a multinucleated giant cell (20 to 30 nuclei), formed from the fusion of mononuclear precursor cells that arrive at the site of resorption via the bloodstream. These precursor cells proliferate and differentiate into osteoclasts through a complex cell-cell interaction with osteoblastic stromal cells.

These cells are characterized by specialized membrane structures, clear zones, and ruffled borders. The size of the clear zone is indicative of the cell's resorbing activity and, in combination with the ruffled border, is responsible for the process of resorption.³ Osteoclasts are found in tiny depressions, pits, or irregular grooves, termed Howship lacunae, in cementum, dentin, and bone.³⁻¹⁰ (Fig 1).

Resorption mechanism. The unique structural arrangement of the osteoclasts to hard tissues allows the cell to establish a microenvironment between the ruffled border and the bone in which resorption takes place.³ The resorptive process itself can be described as being bimodal, involving the degradation of the inorganic crystal structure of hydroxyapatite and the organic structure of collagen, principally type I.⁹ The activated osteoclasts produce an acidic pH (3.0 to 4.5) in their microenvironment. At pH 5.0 or lower, the solubility of hydroxyapatite increases dramatically, and resorption of hard tissue can occur. This acidic environment is primarily achieved through the action of a highly active polarized proton pump contained within the ruffled border.

The enzyme carbonic anhydrase II (CA II), which is specific to osteoclasts, also plays a critical role in establishing a subosteoclastic acidic pH. The CA II catalyzes the intracellular conversion of CO₂ to H₂CO₃, which provides a readily available source of H⁺ ions to be pumped into the subosteoclastic region. The critical nature of this enzyme to the resorptive process is highlighted by osteoporosis, a disease characterized by a failure to resorb bone and a congenital deficiency in CA II.

The degradation of the organic matrix is accomplished by three groups of proteinase enzymes: collagenases and matrix metalloproteinases (MMP), which act at neutral or just below neutral pH (7.4),³⁻¹⁰ and the cysteine proteinase family, which act at an acidic pH.¹⁹ The cysteine proteinases appear to work closer to the ruffled border, where the pH is more acidic, while the collagenases appear to be active at the resorbing bone surface, where the pH is closer to neutral because of the buffering capacity of the dissolving bone salts. Cysteine proteinases are secreted directly by the osteoclast into the clear zone via the ruffled border. While the source of the collagenases remains in question, two potential origins exist—the osteoclast itself or the surrounding osteoblasts.¹⁵⁻²⁰ Recent evidence suggests that it may be a combination of both, with the osteoclast responsible for the secretion of MMP 9 in particular.²¹

Systemic regulatory factors. The major systemic regulatory factors of osteoclastic function include parathyroid hormone (PTH), 1,25-dihydroxyvitamin D₃ (1,25(OH)₂D₃), and calcitonin (CT).²⁰ Increases in the circulating concentration of PTH promote bone resorption by increasing both the number of cells present and the rate of activity among individual clastic cells. The effect of PTH appears to be multifactorial: (1) stimulation of osteoblasts using a receptor-mediated, cAMP-dependent pathway to increase the production of neutral proteases and to decrease the amount of protease inhibitor and matrix deposition; (2) direct action on the osteoclast to increase CA II activity by cAMP-mediated phosphorylation of the enzyme; and (3) promotion of the fusion of marrow cells, leading to the formation of multinucleated giant cell of osteoclastic phenotype.

The major effect of 1,25-dihydroxyvitamin D₃ is to increase the resorbing activity of osteoclasts already present, without increasing osteoclastic numbers. The exact mechanism of action, however, remains unclear. Calcitonin inhibits resorption by inhibiting cytoplasmic motility and producing cell retraction. This effect occurs at minimal concentration of CT and is highly sensitive, prolonged, and modulated through a specific receptor unique to the osteoclast.²²⁻²⁵ The inhibitory action of calcitonin has also been demonstrated on cells actively resorbing tooth structure.²²⁻²⁵
Local regulatory factors. Osteoclasts are subject to extensive regulatory mechanisms that are facilitated to a large degree by osteoblasts mediating the effects of osteotropic hormones and local mediators on these cells. The presence of leukocyte antigens on the osteoclast cell membrane favors the derivation of osteoclasts and their precursors from the multipotential stem cells that produce peripheral blood leukocytes.3,26 Several local factors are key to osteoclastic formation, regulation, and activation, including macrophage colony-stimulating factor (M-CSF), interleukin 1 (IL-1), interleukin 6 (IL-6), interleukin 11 (IL-11), and tumor necrosis factor-alpha (TNF-α). Generally, these cytokines stimulate development of osteoclasts when tested individually. However, in most cases, they require cell-cell recognition among osteoblast cells, osteoclast progenitors, and osteoclasts for formation and activation. They are secreted from inflammatory cells (macrophages and polymorphonuclear neutrophil leukocytes) and osteoblasts as a result of stimulation by bacteria, tissue breakdown products, and the cytokines themselves.

Macrophage colony-stimulating factor is probably the most important soluble factor. It appears to be necessary not only for proliferation of osteoclast progenitor but also for the subsequent differentiation into mature osteoclasts and survival.

Interleukin 6 acts on osteoblastic stromal cells to induce osteoclast differentiation factor, which recognizes osteoclastic progenitors and prepares them to differentiate into mature osteoclasts. It has been found that IL-6 induces osteoclast-like cell formation in human marrow cultures and stimulates osteoclastic bone resorption in vivo. Serum levels of IL-6 are increased in several metabolic bone diseases.27 Interleukin 1 was one of the first bone-resorbing cytokines to be identified. It is active at all stages in osteoclast formation, differentiation, and activation indirectly through the osteoblast. It increases resorption locally by stimulating the production and release of prostaglandin E2 (PGE2) and acting directly on the osteoclast. However, its action appears to be enhanced in the presence of IL-6.

The major effect of TNF-α on hard tissues is to stimulate osteoclastic activity. Like IL-1, PGE2, and IL-6 mediate the effect of TNF-α.

Arachidonic acid metabolites, particularly prostaglandin, are associated with diseases in which marked bone resorption is found, such as periodontal and periodontitis, hypercalcemia of malignancy, and rheumatoid arthritis. Release of PGE2 locally by chronic inflammatory cells simulates the formation of osteoclasts by enhancing fusion of osteoclastic precursors and increasing the resorbing activity of existing cells adjacent to mineralized tissue.28

Bacteria play an important role in many destructive bone diseases. Likewise, bacteria are integral to the process of tooth resorption. Two possibilities exist for the mechanism of bacteria-induced resorption: (1) bacteria produce acids and proteases that destroy the bone matrix components, and (2) bacteria stimulate the production of osteolytic factor, which promotes osteoclastic activity. In the case of tooth resorption, the overriding mechanism is likely to be induction of osteocytic factors because of the effect of endotoxin (lipopolysaccharides). These substances represent the outer surface of the gram-negative bacteria. Lipopolysaccharides stimulate a number of molecular biologic events, including lysosomal enzyme release, collagenase release from macrophages, and osteoclastic secretion of osteolytic factors IL-1, IL-6, M-CSF, and PGE2. Together, these events result in the proliferation of osteoclasts and enhanced bone resorption. However, with large amounts of endotoxin, osteoclastic responses decrease, suggesting a toxic reaction.39

**Odontoclasts**

It is unknown whether osteoclasts and tooth-resorbing cells (dentinoclasts, odontoclasts, and cementoclasts) are the same cell, but a number of similarities do exist. Odontoclasts are smaller, have a ruffled border, contain fewer nuclei than osteoclasts, and have smaller or no clear zone.3,13 Both cells have similar enzymatic properties and intense tartrate-resistant acid phosphatase activity. Their resorption patterns also appear to be the same mononuclear odontoclasts (4% of odontoclasts) that participate in tooth resorption. However, the majority of odontoclasts (94%) that form lacunae on the dentin are multinucleated, having 10 or fewer nuclei. Oligonuclear odontoclasts (cells with fewer than five nuclei) resorb more dentin per nucleus than do cells with a higher number of nuclei.16

**TYPES OF TOOTH RESORPTION**

Tooth resorption is classified based on the site, nature, and pattern of the process.31 It is generally differentiated into internal and external resorption. Occasionally, combinations of both internal and external resorption can be found on the same tooth.

**Internal resorption**

Internal resorption is rare in permanent teeth.2,6,31 In luxation injuries, it has been recorded as a sequel in approximately 2% of recalled patients. However, it has also been reported in nontraumatized teeth. Typically asymptomatic, it is usually discovered during a routine
Internal inflammatory resorption and internal replacement resorption: (1) necrotic pulp; (2) vital pulp; (3) vital pulp; (4) internal inflammatory resorption; (5) internal replacement resorption.

Radiographic evaluation and is often misdiagnosed as external resorption. There are two types of internal resorption: root canal replacement resorption and internal inflammatory resorption (Fig 2).

**Root canal replacement resorption** (metaplastic resorption). Etiology. This disease process appears to result from a low-grade irritation of pulpal tissue, such as chronic irreversible pulpitis or partial necrosis, that is usually localized to a small area of the root canal system. Root canal replacement resorption involves resorption of the dentin and a subsequent deposition of hard tissue that resembles bone or cementum, but not dentin. This type of resorption takes place when a chronic inflammatory process occurs juxtaposed to a region in which the odontoblastic layer and predentin are absent or damaged, which can occur as a result of trauma or application of extreme heat to the tooth.

**Clinical evaluation.** This phenomenon is typically asymptomatic, and the affected teeth may respond within normal limits to thermal or electric pulp testing. The condition can become painful if the process perforates the root or crown of the tooth.

**Radiographic evaluation.** Internal replacement resorption generally appears as enlargement of canal space, including discontinuity of the normal canal space. This space is then engorged with a less radiodense material, giving the appearance of partial canal obliteration (Figs 3 and 4).

**Histologic evaluation.** There is a gradual enlargement of the pulp space because of continuous formation of bone or osteodentin at the expense of dentin. The normal pulpal tissue is replaced by a cancellous type of hard tissue. Variations. Internal tunneling resorption is usually caused by luxation injuries. Evidence of resorption may be found on a coronal fragment of root fracture in which a tunneling resorption process occurs next to the root canal. Resorptive processes may be arrested, or complete pulp canal obliteration may take place.

**Treatment.** Once internal resorption is diagnosed, treatment with nonsurgical root canal therapy is necessary. It is critical that the pulp and granulation tissue with odontoclasts be removed to arrest the process.

**Internal inflammatory resorption.** Etiology. This type of resorption involves a progressive loss of root substance without deposition of hard tissue in the resorption cavity. It frequently results from chronic inflammation of the pulp. The progression of inflammatory resorption is dependent on the interaction between vital pulp tissue and necrotic tissue. Chronic irritation of pulpal tissues occurs when bacteria and their components enter root canals via dentinal tubules that are exposed by mechanical damage. Bacteria can also enter the canals at areas of dilaceration or cracks in the cervical area of the root.
Most commonly, internal resorption is found in the cervical region; however, it can occur in all areas of the root canal system. Areas of internal root resorption usually seem shallow.\(^\text{13}\) Infrequently, in root-filled teeth, bacteria present in the root canal may communicate with the periodontum via an accessory canal, from which soft tissue may proliferate into the root canal and resorb contaminated dentin.\(^\text{2}\)

Clinical evaluation. As in root canal replacement resorption, internal inflammatory resorption is generally asymptomatic and is usually identified on routine radiographs. The process of resorption is active only if part of the pulp remains vital; therefore, pulp testing can be positive. However, usually the coronal pulp is necrotic while the apical pulp is vital, resulting in a nonresponsive test.\(^\text{33}\) Pain may be present if perforation of the crown or root occurs.\(^\text{17}\) If resorption takes place in the coronal portion of the tooth, the tooth may exhibit a pinkish or reddish hue because of the presence of numerous capillaries in the pulpal granulation tissue undermining the coronal enamel.\(^\text{15}\)

Internal inflammatory resorption can be transient or progressive. The transient type of resorption occurs frequently in traumatized teeth or in teeth that have undergone orthodontic or periodontal treatment. In case of progressive internal inflammatory resorption, the dentinal tubules have a special and fortuitous course. These tubules must open to an area of the root canal where the tissue is necrotic and infected, so that microorganisms may enter the tubules and lead to an area of canal with vital pulpal tissue. The resorptive area and the root canal apical to this area contain vital tissue that is necessary for the resorption to continue.\(^\text{34}\)

Radiographic evaluation. Internal inflammatory resorption appears as a circumscribed, oval enlargement (radiolucency) continuous with the root canal wall, usually in the coronal or radicular portion of the tooth.\(^\text{2,13}\) Labially or lingually located external root surface resorption may have a similar appearance; therefore, additional radiographs, taken from mesial and distal angles, are recommended to locate the area in question\(^\text{31}\) (Figs 5 and 6).

Histologic evaluation. Normal pulpal tissue is present. This transforms into granulomatous tissue with giant cells that resorb the predentin of the root canal. A necrotic zone containing bacteria is usually found coronal to resorbing tissue.\(^\text{2,33,36}\)

Treatment. Nonsurgical root canal therapy is recommended, unless the resorption cavity is in the vicinity of the apical foramen and suspected of being related to pulpal revascularization.\(^\text{31}\) Failure to treat internal inflammatory resorption can lead to its eventual extension to the periodontal ligament, via a crown or root perforation.\(^\text{15}\) In these cases, a periodontal procedure, such as crown lengthening or root extrusion, may be implemented to gain access for repair.
External resorption of teeth

External resorption can be classified into four categories by the clinical and histologic manifestations: external surface resorption, external inflammatory root resorption, ankylosis, and replacement resorption (Figs 7 and 8). In addition to these four categories, external resorptions are often described according to their location on the tooth: cervix, body, or apex of the tooth. As a general rule, external resorption can be differentiated from internal resorption by its radiographic presentation. The resorptive radiolucencies are superimposed over the root canal system.

External surface resorption. External surface resorption is a transient phenomenon in which the root surface undergoes spontaneous destruction and repair. It is found in all teeth, in varying degrees, and is likely to be a normal physiologic response. It is the least destructive form of external root resorption and is a self-limiting process; hence, it requires no treatment.

Etiology. External surface resorption occurs as a response to indirect physical injury, caused by physiologic function, to localized areas of periodontal ligament or cementum on root surface. In cases of trauma, it occurs because of direct mechanical contact of the root surface and alveolar bone proper. It can be considered as part of the repair process of physically damaged calcified tissue by recruitment of cells from adjacent normal tissue.

Clinical evaluation. No significant signs of external surface resorption are detectable on the supragingival portion of the tooth.

Radiographic evaluation. External surface resorption is usually not visible on radiographs because of its small size. However, when visible, it appears as small excavations on the root surface with normal lamina dura and periodontal space. These excavations can be found on the lateral surface of the root or at the apex, resulting in the appearance of shorter roots.

Histologic evaluation. External surface resorption occurs as small, superficial lacunae in the cementum and the outermost layer of dentin, which is simultaneously being repaired with new cementum. While there is generally no significant inflammatory reaction in the adjacent periodontal ligament, it can occur in areas of localized necrosis of the periodontal ligament and where cementoblasts are damaged.

Treatment. No treatment is indicated.

External inflammatory root resorption (EIRR). This represents the most common type of external root resorption. It is best described as a bowl-shaped resorptive defect that penetrates dentin.

Etiology. Several etiologic factors have been found to cause external inflammatory root resorption. In general, injury to or irritation of the periodontium from trauma, periodontal infection, or orthodontic treatment initiates an inflammatory response within the periodontal ligament and leads to resorption. External inflammatory
Figs 6a and 6b Internal inflammatory resorption. (Left) Radiographic appearance. (Right) Diagrammatic description.

Fig 7 External inflammatory root resorption at the cervical and apical areas of the tooth when the pulp is vital: (1) vital pulp; (2) external inflammatory root resorption at the cervical area; (3) external inflammatory root resorption at the apical area.

Fig 8 External inflammatory root resorption at the cervical areas of the tooth with external replacement resorption and ankylosis when the pulp is necrotic: (1) necrotic pulp; (2) external inflammatory root resorption at the cervical area; (3) external replacement resorption; (4) ankylosis.
root resorption can occur on any part of the root; however, it is frequently subcategorized according to that part of the tooth to which it has occurred.

The most common cause of external inflammatory root resorption is trauma, particularly in cases where the injury results in pulpal necrosis and damage to the root surface, leaving dentinal tubules exposed. This creates a communication between the internal and external surfaces of the root. Bacteria, bacterial by-products, and tissue breakdown products from within the root canal system stimulate inflammation in the adjacent periodontal tissue and lead to aggressive progressive inflammatory resorption of the root. Pressure has also been hypothesized as a possible etiologic agent of external inflammatory root resorption, as evidenced by resorption that transpires in orthodontic tooth movement, tooth eruption, bone lesions, tumors, cysts, impacted teeth, and occlusal trauma. In these cases, resorption tends to cease when the source of pressure is removed from the root surface

1. **Cervical.** External inflammatory root resorption (invasive cervical resorption) can occur following injury to the epithelial cervical attachment apparatus and to the area of the root surface just below the attachment apparatus. Clastic cells then colonize the damaged area and begin resorbing the tooth. If left untreated, resorption will eventually penetrate the root canal. The injuries can be differentiated into physical and chemical injuries. Physical injury, which occurs to nonendodontically and endodontically treated teeth, typically includes all forms of tooth trauma, surgical procedures, orthodontic treatment, bruxism, and periodontal root planing and scaling. Chemical injury can occur from agents used within the root canal system, such as internal bleaching solutions. When 30% hydrogen peroxide is used for internal bleaching, gingival or periodontal tissues can become irritated as the hydrogen peroxide leaches through cervical dentinal tubules

2. **Apical.** There are three variants of external inflammatory root resorption. They are related to (1) traumatic injury, particularly intrusive luxation; (2) periradicular periodontitis; and (3) orthodontic treatment in which the pulp remains vital (external apical root resorption). Intense and progressive inflammation confined to the apex causes sufficient pressure to overcome the "resistance" of the cemental layer to resorption. In traumatic injuries, especially intrusive luxation, microbial stimuli from the infected root canal cause inflammatory root resorption at the apex. The specific causes of external root resorption during orthodontic treatment are not well understood, but excessive forces, notably intrusive or tipping in nature, are commonly implicated. It is hypothesized that the excessive forces initiate inflammation either as a result of stimulation of a phagocytic process by tissue breakdown products or through some form of neurogenic inflammation.
Figs 10a and 10b  External inflammatory root resorption at the cervical and apical parts of the root, related to trauma. (Left) Radiographic appearance. (Above) Diagrammatic description.

Figs 11a and 11b  External inflammatory root resorption at the apex, related to periodontal periodontitis. Note the widened periodontal ligament and loss of adjacent lamina dura and tooth structure. (Left) Radiographic appearance. (Right) Diagrammatic description.
Clinical evaluation. External inflammatory root resorption is considered a major resorptive condition without symptoms. This type of resorption is found to progress more rapidly and frequently in immature teeth because of wide tubules and thin dentinal walls. The most common location for external inflammatory resorption is the cervical aspect of the tooth, where the progression of resorption is inward and lateral but leaves the canal intact. The resorption can begin 2 to 12 weeks after injury. It progresses rapidly, especially after tooth replantation in patients between the ages of 6 and 10 years.

The process of EIRR can be transient or progressive. The transient type occurs frequently in traumatized teeth and in teeth that have undergone orthodontic and periodontal treatment. For resorption to continue, resorbing cells require continuous stimulation. However, stimulation by resorptive cells on denuded dentin or cementum surface appears to be sustained for no more than 2 to 3 weeks; therefore, the transient type of resorption may arrest spontaneously.

The progressive type of external inflammatory resorption occurs when the source of inflammation is not removed, causing an ongoing resorptive process. Extensive resorption can ultimately lead to an increase in mobility of the tooth. Coronal, as resorption advances further inward at the cervical level, the crown of the tooth becomes undermined. Granulomatous tissue grows into the resorptive defect, resulting in pink discoloration of the crown.

Radiographic evaluation. With EIRR, the periodontal ligament space becomes widened and there is a loss of adjacent lamina dura and tooth structure (Figs 10, 11, and 13), resulting in ragged, poorly defined mesial and distal borders. There may also be a gradual increase in radiopacity toward the root canal space. External resorption in the cervical aspect of the tooth is visible clinically and radiographically as a sin-
External inflammatory root resorption at the apex, related to orthodontic treatment. (Left) Radiographic appearance. (Right) Diagrammatic description.

Histologic evaluation. Histologically, EIRR is represented by a bowl-shaped resorption area into cementum and dentin with inflammation of adjacent periodontal tissue and presence of infected or necrotic pulp in the root canal. In the periodontium, granulomatous tissue is present with lymphocytes, plasma cells, and polymorphonuclear leukocytes. The adjacent resorbing surface of the root has many Howship lacunae, which occasionally contain osteoclasts.

Variations. In undermining EIRR, a small opening on the external surface of the root and a resorptive cavity within the body of the root are present. The cavity contains granulomatous tissue, with active odontoclastic cells, and inflammatory cells (Figs 15a and 15b). Late external inflammatory root resorption occurs years after injury and is usually located at the cementoenamel junction.
Treatment. Treatment of external inflammatory root resorption is dependent on the etiology:

1. Resorption as a result of orthodontic treatment: In cases where orthodontic treatment is the sole etiologic agent for the resorptive process, removal of the pressure of orthodontic movement will arrest the resorption.

2. Cervically located resorption in which the pulp is vital and treatment of the cervical resorption is unlikely to cause pulpal injury: If pulpal injury is unlikely during the repair of EIRR, baseline thermal and electrical pulp tests should be recorded and the defect restored.

3. Cervically located resorption in which the pulp is vital and treatment of the cervical resorption is likely to cause pulpal injury: Nonsurgical root canal therapy is performed, and the external resorbed defect is restored.

4. Cervically located resorption in which the pulp is nonvital: Nonsurgical root canal therapy is performed, and the external resorbed defect is restored.

5. Resorption as a result of pulpal necrosis and periodontal injury: Nonsurgical root canal therapy is performed, and the external resorbed defect is restored, when indicated.

In the event that the pulp is necrotic or likely to undergo irreversible injury during the treatment of external cervical resorption, nonsurgical root canal therapy with the use of calcium hydroxide as an interim medicament is recommended. In some instances, endodontic and periodontal therapy may be necessary to eliminate resorption-causing bacteria and inflammation. Furthermore, all teeth with external inflammatory root resorption should be evaluated radiographically on a regular basis.

Posttreatment complications. The major concern after treatment of external inflammatory root resorption is the high rate of recurrence. This may be caused by cells in the adjacent periodontal ligament undergoing a metaplastic process and again becoming resorptive. Other posttreatment complications include cervical root fracture with severe cervical resorption and periodontal defects resulting from flap reflection during excavation and restorations of resorption defects. Alternatively, as inflammatory resorption becomes arrested, ankylosis can occur.

Ankylosis. Etiology. Ankylosis is primarily associated with a history of luxation injuries, especially avulsion. It occurs after extensive necrosis of the periodontal ligament with formation of bone on the denuded area of the root surface. Ankylosis is a union of tooth and bone, with no intervening connective tissue, following external inflammatory resorption. Studies of mechanical removal of all or part of the periodontal ligament prior to tooth replantation have demonstrated that this procedure consistently leads to dentoalveolar ankylosis.

Ankylosis can be progressive or transient. The likelihood of progressive ankylosis increases dramatically
when greater than 20% of the root surface is damaged. In replanted teeth, physical and biochemical alterations to the periodontal ligament may influence the phenotypic expression of the progenitor cells and account for the development of ankylosis and replacement resorption.

Ankylosis is not a disease process. It occurs as a "mistake" because cells involved in the remodeling of bone are not able to distinguish among root cementum, dentin, and bone. The tooth root is thus incorporated into the normal remodeling process of the alveolus and its gradual replacement by bone. The rate of progression of ankylosis is directly related to the initial damage to the root surface and the age of the patient. The progression of ankylosis is very rapid in young individuals.

Clinical evaluation. The tooth will be immobile once it is ankylosed; therefore, percussion of the teeth is an important diagnostic tool. When a tooth is ankylosed, there is a high pitch or metallic tone that will be very different from that elicited from adjacent uninjured teeth. In some cases, a change in tone on percussion may be evident before radiographic changes become apparent.

However, in some cases, ankylosis is transitory and may disappear within a year of injury as the tooth returns to normal percussion tone. Studies show that loss of mobility occurs when more than 10% of the root surface is ankylosed. Ankylosis can usually be diagnosed 4 to 6 weeks after replantation. High-pitched percussion tone occurs when 20% of the root surface is ankylosed. Other clinical signs that may be present include infraocclusion, incomplete alveolar process development in a young patient, and lack of a normal mesial drift.

Radiographic evaluation. It is difficult to identify ankylosis on radiographs because of overlapping structures and bone marrow spaces. However, a complete disappearance of the periodontal space and an uneven root surface contour is common (Figs 16 and 17).

Histologic evaluation. There is a continuous replacement of root substance with bone, resulting in a union between bone and root substance without intervening connective tissue. Ankylosis occurs when the external surface of the replanted tooth becomes populated by osteoblasts and osteoclasts that are continuous with the endosteal spaces of the adjacent bone marrow. The endosteal spaces of alveolar bone contain a reservoir of progenitor cells that are capable of migrating into the periodontal ligament to become osteoblasts or cementoblasts.

Treatment. There is no treatment for ankylosis at present. Ultimately the crown of the tooth will fracture off at the gingival crest as ankylosis progresses, resulting in a complete replacement of the root by bone.

Potential complications. Ankylosis in young patients can complicate normal bone growth and development, resulting in problems such as malocclusion or supereruption of opposing teeth. Careful consideration is necessary if extraction is the treatment of choice for ankylosed incisors because this may significantly reduce alveolar bone height that will be difficult to restore.

External replacement resorption. Etiology. This breakdown process is related to the repair processes of a traumatically injured pulp and/or periodontium of luxated mature teeth. This process is invariably followed by surface resorption and/or obliteration of the pulp canal. The injured periradicular tissue generally returns to normal following repair, which usually takes place 1 year after trauma.

Etiology. This breakdown process is related to the type of injury and stage of root development.
Typically, it is caused by moderate injuries to the pulp, such as subluxation, extrusion, and lateral luxation, or a moderate combined injury to the periodontal ligament and the pulp in mature teeth. It is rare after a slight injury and absent after a severe injury. Other possible causes of TAB are infections, orthodontic treatment, and occlusal insult to the periodontium.\cite{49}

**Clinical evaluation.** The tooth will often respond within normal limits to pulp tests. Clinically, in some instances, the tooth may undergo a color change and/or have varying results from electrical pulp testing. However, following repair, clinical and radiographic findings will return to normal\cite{49} (Figs 18 and 19).

**Radiographic evaluation.** Transient apical breakdown can only be found in teeth with fully formed roots and closed or half-closed apices. A transient localized change in the size of the apical periodontal ligament space, ranging from 2-times normal width to a semicircular radiolucency, combined with a blunting of the apex from surface resorption, can be observed. Also, pulp canal obliteration may be seen during TAB or following TAB\cite{48,49} (Figs 18a and 18b).

**Histologic evaluation.** No human or animal studies have examined the histologic healing events of transient apical breakdown.\cite{48,49}

**Treatment.** No treatment is recommended.
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Figs 18a and 18b  Transient apical breakdown at the initial visit. Results of the sensitivity test were within normal limits. (Left) Radiographic appearance. (Right) Diagrammatic description.

Figs 19a and 19b  Transient apical breakdown at the 1-year follow-up. Results of the sensitivity test were within normal limits. (Left) Radiographic appearance. (Right) Diagrammatic description.

**Combined internal and external resorption**

Internal and external resorptions can occur simultaneously on the same tooth. This can be detected at various stages of progression and may appear as separate or joined defects. As the resorptions advance inward from the external surface and outward from the internal surface, the defects will eventually communicate (Figs 16, 17, 20, and 21).
Figs 20a and 20b Combined internal and external resorption. (Left) Radiographic appearance. (Right) Diagrammatic description.

Figs 21a and 21b Combined internal and external resorption. (Left) Radiographic appearance. (Right) Diagrammatic description.

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