Tooth Resorption: The “Black Hole” of Dentistry

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Effective Date: 12/01/15 Expiration Date: 12/01/18

About the Author

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Disclosure: Dr. Germain reports no disclosures.

The video arcade game Pac-Man was developed in Japan in 1980 and released in the United States one year later. This iconic slice of American pop culture has transcended generations, and fans still are entertained as Mr. P gobbles up dots while avoiding his enemies Blinky, Pinky, Inky, and Clyde. When I played this game for the first time, my “dentocentric” mind immediately associated the animated effects of Pac-Man with the process of resorption, giving me an uncanny visual for this destructive disease process.

In its broadest sense, tooth resorption has been historically divided into the following 2 categories: internal resorption, caused by cells of the dental pulp; and, external resorption, caused by cells of the periodontal ligament (PDL). It is ubiquitous in nature and not limited to the human dentition.

Tooth resorption represents one of the most common diseases seen in domestic cats (Figure 1), and is theorized to be caused by feline herpes virus. Resorption is also seen in elephant tusks, monkeys’ teeth, and ferrets (Figure 2), and in many other animals. Figure 3 reveals a resorptive defect on my golden retriever’s molar. (I am happy to report that she has been flossing more regularly since it was discovered.)

Like a black hole in space, the “black holes” created by tooth resorption are mysterious, seemingly appearing from nowhere and without a reason. I will be the first to admit that I find the diagnosis, treatment protocols, and management of tooth resorption confusing. Perhaps this is because, unlike other dental pathology, it is not a specific disease. Resorption of a tooth is a sign of an underlying pathologic condition caused by a variety of disease processes. There are several types of resorptive lesions, with the best treatment approach being determined by the etiology and radiographic morphology of the lesion. Once the type of resorption is determined, the case can be evaluated and treated accordingly. While it might be expedient to recommend extraction of teeth with resorptive lesions, it is frequently not necessary and it may not be the correct treatment for your patient.

The literature cites many classification systems for root resorption. Andreasen’s contribution, based on many years of studying the sequelae of trauma cases where it is a common ramification, is widely accepted. However, Andreasen’s body of work does not include the more invasive types of root resorption that cannot be explained by avulsion or luxation injuries. Heithersay and Lindskog et al have taken the work of Andreasen and expanded it to include the following types of lesions, subdividing them into the following 3 categories: (1) trauma-induced resorption, (2) infection-induced resorption, and (3) hyperplastic invasive resorption.

TRAUMA-INDUCED RESORPTION (NONINFECTIVE)

In addition to the mechanical disruption of the PDL caused by
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avulsion and luxation injuries, this category encompasses any resorption created by external forces, including thermal, chemical, surgical, biomechanical, neoplastic, and pressure from tooth eruption. These resorptions generally present with several different clinical profiles.

**Surface Resorption**
If a tooth has sustained very minor trauma and the PDL does not undergo necrosis, small, cup-shaped indentations may be detected on radiographic examination (Figure 4). These lesions involve the cementum and a small amount of the underlying dentin. This process is self-limiting due to the continued ability of Hertwig's epithelial root sheath to remodel the hard tissues. No treatment is recommended.

**Transient Apical Resorption**
Often seen in undeveloped luxated teeth, the radiographic appearance is characterized by an internal defect within the apical portion of the canal space. Andreasen\(^4\) described the frequent self-limiting nature of this process due to the regenerative potential associated with vascular ingress into this area. He noted\(^4\) that if revascularization occurred, it would aid healing of the traumatized pulp that in turn would allow hard tooth structure to develop normally. In addition, associated color change in the coronal tooth structure from intracoronal hemorrhage would likely be transient.

If, however, the injury were to result in pulp necrosis, the tissues would lose their spontaneous vascular healing ability (Figure 5). At this point, the clinician would need to intervene with treatments intended to promote apexogenesis, including procedures to induce revascularization even in instances where there is a nonvital pulp.\(^9\) Resulting root development makes endodontic treatment less complicated and protracted, with the tooth being more resistant to fracture. The intent is to keep the tooth at least long enough to maintain the site for predictable and aesthetic implant therapy, once the patient matures.

**Pressure Resorption**
Resorption of primary teeth is regulated by the stellate reticulum and the dental follicle of the
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underlying permanent tooth via the secretions of cytokines. Tooth exfoliation is part of the normal remodeling process of the dentition (Figure 6). However, if pressure resorption occurs as a result of an impacted wisdom tooth (Figure 7), prompt removal of the source is recommended, and the affected tooth should be evaluated for treatment. Other undesirable types of pressure resorption can occur as well. For example, neoplastic diseases can develop in the jaw that create significantly more morbidity than tooth loss. Figure 8 shows radiographic resorption caused by the pressure of an osteosarcoma lesion located between the roots of the tooth No. 30. It is important to be aware that this is possible, albeit not a common occurrence.

Orthodontic Resorption

A form of external apical root resorption is sometimes seen as an undesirable result of orthodontic treatment (Figure 11). The resulting blunted and shortened roots are thought to be multifactorial in etiology related to genetics, systemic factors, root morphology, bone density, and other patient specific factors. In addition, the type, direction, duration, and amount of force placed on the teeth induce risk related to treatment itself.

While this process is not reversible, the root structure is replaced with bone and will usually have little impact on the long-term function and health of the teeth, unless the result is an extremely poor crown-to-root ratio (Figure 12). If pulp vitality tests are normal, no treatment is required.

Surgical Trauma

It is not an uncommon finding to see resorptive changes in normal tooth anatomy near the placement of ligature wires and other fixtures required for stabilization of the bone. Maxillofacial procedures often require “rearrangement” of portions of the mandible and or maxilla along with segments of the dentition (Figure 9). If the pulp tests nonvital, root canal therapy (RCT) is recommended (Figure 10).

Dento-Alveolar Ankylosis/Replacement Resorption (RR)

RR occurs as a result of luxation, avulsion, and intrusion injuries to teeth. On a cellular level, the osteoclasts are in direct contact with the exposed root surface due to necrosis of the PDL. In the absence of the PDL, fusion of the tooth and the bone occurs as the bone replaces the dentin. It has been suggested by Tronstad that rather than being the result of a disease process, RR occurs as a...
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“mistake” whereby the cells involved in bone remodeling are not able to distinguish among root cementum, dentin, and bone.

A thorough patient history will usually reveal trauma that at times occurred many years prior. For example, it is not uncommon for an adult to recall an old high school football injury, or an elbow to the mouth when playing basketball on a playground. Upon clinical exam, the tooth will frequently appear darker and less translucent than the surrounding unaffected teeth and many times this discoloration is the patient’s chief complaint and only sign that there is a problem.

Due to its asymptomatic proclivity, RR is often first detected by chance with routine radiographs. The PDL will not be detectable on a radiographic image and the canal may have an amorphous appearance (Figure 13). Since there is a direct bone-to-tooth contact, these teeth lack the physiologic mobility of normal teeth with a healthy ligament.

There is no treatment available for RR. RCT will not arrest the disease process, since the etiology is not pulpal in nature. In cases where the ankylosis is severe, and the patient is not old enough for implant replacement therapy, decoronation and root submergence are recommended to encourage axial and vertical alveolar bone growth. In adults, if the tooth is functional and in the correct position, treatment can be delayed as long as the bone is not adversely affected.

If post-traumatic injury cases are handled quickly and with care to preserve the PDL by keeping it hydrated, damage can be minimized, but usually not prevented. Splinting for 7 to 10 days is recommended in cases where there is excessive mobility of the tooth; however, long-term fixation is not recommended. While RCT is not effective in arresting the affects of RR, it is recommended after trauma to prevent infection and other types of resorption from occurring.

INFECTION-INDUCED RESORPTION

Inflammation from infectious by-products of dental disease can cause resorption of both the teeth and the alveolar attachment apparatus. Treatment involves removing the source of the infection. The most widely known cause is infective endodontic pathosis; however, it can also manifest in combination with trauma-induced resorption, thus complicating the diagnosis,
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**Internal Inflammatory ( Infective) Root Resorption (IIRR)**
IIRR is a rare clinical finding accounting for its poorly understood etiology. Either trauma or caries with bacterial invasion into the pulp are cited as predisposing factors. Haapasalo and Endal\(^\text{12}\) proposed that damage to the odontoblastic cells covering mineralized dentin inside the root canal must occur, which in turn allows vital pulpal cells with resorbing potential to have access to the tooth structure.\(^\text{12}\)

Clinically, IIRR will most likely present as asymptomatic, although pulpitis can occur due to progressive inflammation of the vital pulp tissue. It typically appears on a radiograph as a round or oval, symmetric, radiolucent widening of the intracanal space. It is important to note that the original shape of the canal can no longer be discerned (Figure 14a), which distinguishes IIRR from external resorption.

Since IIRR needs vital pulp tissue and bacterial contamination to occur, completed cleaning, shaping, and 3-D obturation of the root canal system will arrest the disease process (Figure 14b). If perforation has occurred, the defect needs to be repaired as well.

**External Inflammatory Infective Root Resorption (EIR)**
Peri-radicular periodontitis caused by lesions of endodontic origin will frequently exhibit some peri-radicular resorption. The tooth will test nonvital, and RCT becomes necessary to arrest the disease process, since the diseased pulp tissue becomes the primary source of resorption.

EIR will occur primarily on the bone side of the attachment apparatus while the root remains resistant. If, however, trauma occurs and the cementum at the primary portals of exit is damaged, inflammatory mediators can spread from the infected pulp space through the dentinal tubules into the PDL that will cause resorption of both the root and the bone.\(^\text{13}\)

If left untreated, this disease process will continue to destroy both the tooth and the bone. Since the cause of EIR is toxins from necrotic pulp tissue, the process can be arrested if the portals of exit can be sealed with RCT and the bone will heal (Figure 15). The root of the tooth will most likely not regenerate to its original anatomy.

**HYPERPLASTIC INVASIVE RESORPTION (HIR)**
The insidious nature of HIR presents the clinician with multifactorial challenges. However, unlike RR, it can be arrested and treated with long-term success if the damage to the tooth structure is accessible and has not progressed beyond the point of repair. While this category can be subdivided, the most common type is invasive external cervical resorption (ICR). Since many of these cases are prematurely diagnosed with a hopeless prognosis, the focus in this section will be to present pertinent clinical information regarding the successful diagnosis, treatment, and management of these lesions.

HIR occurs when the soft tissue of the PDL becomes resorptive and aggressively attacks the hard tissue of the tooth. While it can occur anywhere on the root surface, it frequently affects the tissues at the epithelial junction, destroying the cervical area of the tooth. The coronal tooth structure often presents with a pinkish hue indicative of the granulomatous tissue ingrowth from the PDL that has filled the resorptive defect. These lesions are usually asymptomatic.

When examining radiographs, ICR can be mistaken for a carious lesion (Figure 16a). However, with ICR, the outline of a canal system is distinguishable within the larger radiolucency (Figure 16b). Histologic examination of ICR shows highly vascular fibrous tissue with destructive multinucleated cells along the surface of the dentin. It is unclear as to whether it is...
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caused solely by inflammation but infective microorganisms are seldom found.4-6

In order to arrest and prevent recurrence of ICR, the resorptive granulomatous tissue needs to be completely removed. A favorable outcome can be expected with proper restoration and maintenance of the tooth if the resorptive defect can be accessed and repaired.7,15 Figure 17 is an illustration showing the difference between an area of inaccessible root resorption versus accessible root resorption. Communication within the sulcus allows access to the resorptive granulation tissue.

Heithersay15 divided ICR lesions into 4 categories based on the location and severity of the cervical defect. Class 1 (CL1) denotes a small invasive resorptive lesion near the cervical area with shallow penetration into the dentin. CL2 denotes a well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into the radicular dentin. CL3 denotes a deeper invasion of dentin by resorbing tissue, not only involving the coronal dentin but also extending into the coronal third of the root. CL4 denotes a large invasive resorptive lesion that has extended beyond the coronal third of the root.

Many Class 1, 2, and 3 lesions can be treated successfully. Figure 18 shows a radiographic example of what appears to be a CL1 lesion; however, it is not accessible. Figure 19 appears to be an accessible CL2 lesion and Figure 20 shows a large but accessible CL3 lesion. Heithersay recommends extraction of Class 4 lesions due to their poor treatment prognosis.7 Figure 21 is an example of an untreatable CL4 lesion. Confirmation of the extent of these lesions can be verified by the use of CBCT imaging so that a 3-D assessment of the defect can be made.

The primary treatment and prevention of recurrence of ICR is dependent on the total removal of the resorptive granulation tissue. While the tissue can be removed mechanically, Heithersay15 suggests using a 90% solution of aqueous trichloracetic acid, being careful to isolate the area completely. If there is pulp involvement, RCT is done and the defect is filled with a glass ionomer, bioceramic material, hydrophilic bisphenol A-glycidyl methacrylate (Bis-GMA), or other appropriate restorative material.

CASE REPORT 1
A 35-year-old white female presented with the chief complaint of a “hole in the back” of her tooth. The radiographic exam revealed a large resorptive defect in the coronal third of the root extending into the coronal tooth structure (Figure 22a). Clinical exam revealed a pink hue on the lingual surface of tooth No. 11 (Figure 22b).

The tooth was isolated with a rubber dam and the resorptive granulation tissue was removed mechanically with a curette and a slow-speed round bur (Figure 22c). Once the tissue was removed, a pulp exposure was evident, and RCT was performed (Figure 22d). A palatal envelope flap was made and the bone was slightly recontoured so that a margin could be identified for restoration. The defect was repaired with a dual-cure hydrophilic Bis-GMA (Geristore [DenMat]). Designed for subgingival lesions and restorations involving soft tissue, Geristore restorative material adheres well to dentin and cementum and also releases fluoride. The patient was referred to her general dentist for a bonded composite overlay on top of the supragingival crown surface (Figure 22e). Figure 22f shows a 3-year follow up.

CASE REPORT 2
A 61-year-old white male (my husband) was unaware of the ICR on tooth No. 18 until it was picked up on a routine dental exam (Figure 23a). The canals were cleaned and shaped. An attempt was made to repair the distal resorptive lesion. Due to the inclination of the ramus of the mandible in that area as well as a thick retromolar pad, all attempts to gain adequate access were unsuccessful. Alternatively, RCT was completed on the mesial root and a distal hemisection was done. Ultimately, this was challenging as well due to the fusion of the mesial and distal roots. Figure 23b shows that a portion of the distal root remains attached and submerged.

The decision not to restore the tooth with a full-coverage crown was made until long-term prognosis could be better predicted. Function was normal with slight occlusal reduction, and there was no danger of bone loss that might compromise future implant replacement. Figure 23c shows a 2-year recall. The patient did not want a crown, preferring to leave it as it was. He is currently asymptomatic, and tissue has grown over the distal root tip. Periodontal probing is normal.

IN SUMMARY
The diagnosis, management, and treatment protocols for tooth resorption present the clinician with a unique challenge. If the diagnosis is an invasive progressive type of external resorption, options for treatment are available for many of these lesions. Protocol requires access to the area, complete removal of the resorptive granulation tissue, and appropriate restoration.

Because diagnosis of resorption is a daunting task, there might be a tendency to put off treating these lesions. Patients are often told that we will “watch it” to see if it progresses. However, the best chance for successful treatment is to intervene early. It is also important to understand what the limits are for treating resorptive lesions so that the patient will receive optimal care.◆
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References


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1. There are several types of resorptive lesions, with the best treatment approach being determined by the etiology and radiographic morphology of the lesion.
   a. True  b. False

2. If a tooth has sustained very minor trauma and the periodontal ligament does not undergo necrosis, small cup-shaped indentations may be detected on radiographic examination.
   a. True  b. False

3. Resorption of primary teeth is regulated by the stellate reticulum and the dental follicle of the underlying permanent tooth via the secretions of cytokines.
   a. True  b. False

4. External apical root resorption is never seen as an undesirable result of orthodontic treatment.
   a. True  b. False

5. Inflammation from infectious by-products of dental disease can cause resorption of both the teeth and the alveolar attachment apparatus. Treatment involves removing the source of the infection.
   a. True  b. False

6. When examining radiographs, invasive external cervical resorption (ICR) can be mistaken for a carious lesion.
   a. True  b. False

7. The primary treatment and prevention of recurrence of ICR is not dependent on the total removal of the resorptive granulation tissue.
   a. True  b. False

8. Because diagnosis of resorption is a daunting task, there might be a tendency to put off treating these lesions.
   a. True  b. False
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ANSWER FORM: VOLUME 34 NO. 12 PAGE 78
Please check the correct box for each question below.

1. ☐ a. True.  ☐ b. False
2. ☐ a. True.  ☐ b. False
3. ☐ a. True.  ☐ b. False
4. ☐ a. True.  ☐ b. False
5. ☐ a. True.  ☐ b. False
6. ☐ a. True.  ☐ b. False
7. ☐ a. True.  ☐ b. False
8. ☐ a. True.  ☐ b. False

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