# **Quality Resource Guide**

## **Tooth Root Resorption**

## **Author Acknowledgements**

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## **Educational Objectives**

Following this unit of instruction, the practitioner should be able to:

- 1. Discuss the mechanism by which tooth roots resist resorption.
- 2. Discuss the mechanism by which clastic cells may inhabit the inner and outer root dentin surface.
- 3. Discuss the causative agents and pathophysiology of root resorptions.
- 4. Cite the distinguishing clinical and radiographic characteristics of internal and external tooth root resorption.
- 5. Discuss the various types of external resorption and give treatment options and prognoses for each.
- 6. Discuss the use of cone beam computed tomography in the diagnosis and treatment planning of tooth root resorption.

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## Introduction

Although there are times when we welcome tooth root resorption, such as in the shedding of deciduous teeth, pathologic root resorption of permanent teeth is unwanted, and can result in premature tooth loss (**Figure 1**). This is a very frustrating condition to both dentist and patient. In many cases the dentist has limited treatment options to offer and the patient experiences a condition that is not the result of their actions or neglect. This guide will give an overview of the various types of tooth root resorption, describe suspected resorptive mechanisms, and discuss diagnosis, management and associated prognoses of teeth with root resorption.

#### **Etiology and Pathogenesis**

The tooth root is a combination of mineralized tissues (dentin and cementum), surrounded by another mineralized tissue (bone) that is in a constant state of resorption and apposition. Approximately 25% of trabecular bone is replaced in the adult skeleton every year. Given this relationship, one must ask:

- Why doesn't tooth root resorption happen more often?"
- How does the tooth root resist resorption by adjacent alveolar osteoclasts?
- Is the thin connective tissue layer of the periodontal ligament enough to protect the tooth root from resorption?

Figure 1



Representative examples of internal (panel A) and external (panel B) root resorption.

We know that even with mild trauma, multinucleated clastic cells can readily migrate through the periodontal ligament to the root surface, but they do not always cause progressive external resorption. Likewise, such cells can also

#### Table 1 - Two Types of Resorption: Internal and External

	Internal Resorption (non-perforating)	External Resorption			
Conventional Radiopgraphic Findings	<ul> <li>Radiolucency with the following features:</li> <li>Well-defined border</li> <li>Pulp canal space merges into lesion</li> <li>Lesion does not appear to move in angled radiographs</li> </ul>	<ul> <li>Radiolucency with the following features:</li> <li>Irregular borders</li> <li>Pulp canal space can be seen through lesion</li> <li>Lesion shifts in angled radiographs</li> </ul>			
Clinical       Pulp         Findings       • Pulp is vital at onset of process; may eventually become necro         Periodontium		<ul> <li>Pulp</li> <li>Pulp is typically vital and normally responsive (except in resorption due to apical periodontitis)</li> <li>Periodontium</li> </ul>			
	Periodontal attachment unaffected by process	May be a probing defect to the level of the resorptive lesion			
	<ul> <li>Crown</li> <li>May have 'pink tooth' appearance if resorption undermines enamel</li> </ul>	<ul> <li>Crown</li> <li>May have 'pink tooth' appearance if resorption undermines enamel</li> </ul>			
Treatment	<ul> <li>Non-surgical root canal therapy</li> <li>Chemical debridement of resorptive defect <ul> <li>Sodium hypochlorite irrigant</li> <li>Calcium hydroxide intracanal medicament for at least 1 week</li> </ul> </li> <li>Obturation with softened gutta percha</li> </ul>	Surface <ul> <li>No treatment necessary</li> </ul>			
		<ul> <li>Inflammatory</li> <li>Endodontic therapy with calcium hydroxide as intracanal medicament until resorptive process is halted; followed by obturation</li> </ul>			
		<ul> <li>Replacement</li> <li>No treatment: advise patient that the tooth will eventually be lost; plan on replacement</li> </ul>			
		<ul> <li>Extra canal Invasive</li> <li>Combined surgical/restorative approach to debride and restore lesion</li> <li>Endodontic therapy if pulp will likely be exposed during treatment</li> </ul>			

be recruited to the dental pulp in response to inflammation caused by trauma or caries, but we don't typically see subsequent internal resorption. What are the resistant mechanisms? It turns out that clastic cells recognize the substrate to which they attach via cell surface receptors called integrins. Integrins are ubiquitous transmembrane proteins that regulate myriad cell-cell and cellextracellular matrix interactions throughout the body. The specific integrins responsible for the attachment of the cells that resorb tooth root structure rely on a unique amino acid sequence for proper function. If the substrate does not have that sequence, the cells cannot attach. Both unmineralized precementum on the external root surface and unmineralized predentin on the internal root surface, lack the necessary attachment sequences. Therefore, these surfaces offer protection against root resorption. Damage to, or malformation of, the precementum or predentin, are a prerequisite for external or internal resorption, respectively. In some cases the damage to the precementum or predentin is mild, and there is no persistent inflammatory stimulus. In those instances, the resulting resorption will be self-limiting, and repair may occur. In other cases, greater damage may occur and/or the presence of bacteria may perpetuate the resorptive process, and a significant amount of root dentin may be lost.

### Types of Tooth Root Resorption

Tooth root resorption can be broadly classified as internal or external, depending upon its point of inception. While the clinical and radiographic presentations and treatment modalities differ greatly between the two types of resorption (**Table 1**), their pathogenesis is thought to be similar, as described above.

#### **Internal Resorption**

Etiology & Conventional Radiographic Features Of the two general types of resorption, internal is by far easier to manage; however, correct and early diagnosis is the key. In most cases, a chronic (persistent) subclinical pulpal inflammation results in the recruitment of clastic cells into the pulp, and as mentioned, there is thought to be an exposed area of dentin that allows cell attachment. The destruction of the root is thus asymptomatic, and internal resorption is largely diagnosed radiographically. Since the resorption expands outward from the pulp canal space, radiographic features of internal resorption typically include a radiolucency with a smooth, well-defined border, and the inability to distinguish root canal anatomy through the lesion (**Figure 2**).

#### **Clinical Presentation**

Clinical signs and symptoms are usually absent, however, if the lesion extends into the tooth crown, it may be visible through the translucent enamel, resulting in the appearance of a 'pink tooth'. This is not a frequent finding with internal resorption, however, since the pulp coronal to the defect is often partially necrotic. Pulp vitality tests may reveal a responsive pulp (either to thermal or electrical stimuli); or, the pulp may have succumbed to the chronic inflammatory stimulus and may be nonresponsive at the time of the examination. The pulp must have been vital during the initiation and active phase of internal resorption, however, as the cells causing the damage require a blood supply via the vital pulp.

#### **Treatment Options**

Treatment of internal resorption involves nonsurgical root canal therapy to eliminate any remaining blood supply to the resorptive tissues, and to remove all remnants of organic tissues that may become a source of nutrients to support bacterial growth. Because the internal defect presents many undercut irregularities of the canal walls, thorough cleansing cannot be achieved mechanically (i.e., solely with root canal instruments). Sodium hypochlorite is advocated as a tissue solvent during irrigation. The placement of calcium hydroxide paste into the defect for a least one week will facilitate the action of sodium hypochlorite at subsequent visits. Most calcium hydroxide preparations contain a radiopacifying agent (such as barium sulfate); therefore, it is helpful to obtain a radiograph after placement of the medicament. When the defect is seen radiographically to be filled with calcium hydroxide, the clinician can be relatively sure that the area has been sufficiently debrided and is ready for obturation. Since a continuously tapering funnel shape cannot be obtained, it is imperative to obturate the defect using thermoplasticized gutta percha that is both vertically and laterally compacted (**Figure 3**).

#### Prognosis

The prognosis for internal resorption is generally good, if the resorptive defect can be thoroughly cleaned, disinfected and obturated, and as long as the resorption has not perforated to the outer root surface and into periodontal tissues.

#### Figure 2



Typical radiographic features of internal resorption. Note the absence of evidence of the root canal system in the resorptive defect, and the smooth, regular outline of the lesion (panel A). Even with an altered horizontal angle, the lesion appears centered (panel B).

#### Figure 3



Internal resorption defect in which obturation was attempted with a single cone technique (panel A); the same defect obturated with thermoplasticized gutta percha (panel B).

#### **External Resorption**

#### Etiology and Clinical and Conventional Radiographic Presentation

External resorption is a much more difficult condition to diagnose and manage. It has been described by a variety of names, which may describe the location, etiology, or pathogenesis of the lesion. External resorption may be caused by physical damage to the external protective layer of precementum; by trauma, infection, or both. In certain cases, the etiology is of unknown origin.

Mild physical damage due to trauma (i.e., concussion or subluxation), may result in recruitment of clastic cells to the area with resultant isolated foci of resorption. However, without persistent irritation, these resorptive lacunae may be repaired with new cementum as healing takes place. This condition is termed surface resorption, and requires no treatment. In fact, it is only discovered on histologic examination of teeth extracted for other reasons. Pressure from ectopically erupting teeth or excessive orthodontic forces may also cause external resorption that is self-limiting; once the pressure stimulus is removed (the ectopically erupting tooth is extracted, or orthodontic forces are discontinued), the resorptive process halts.

Infection alone may damage the external root surface and allow initiation of the resorptive process. This is commonly seen in teeth with necrotic, infected pulps with resultant apical periodontitis. Not only have clastic cells resorbed bone to the point where radiolucency can be seen apically, but the root surface can be resorbed in the area as well. This is usually seen as a shortened root adjacent to a periapical bony lesion (Figure 4A). While the root length cannot be regained, nonsurgical root canal therapy with adequate debridement and disinfection will allow healing of the bone, and halt the root resorptive process. A normal periodontal ligament space will be reestablished (Figure 4B). Because resorption of the root apex often results in the loss of the natural apical constriction, the clinician must take care not to over-extend the obturation material. Interim radiographs can help with length control.

When damage occurs to the cementum layer and bacterial infection is present, the external resorption that ensues is called **inflammatory root resorption**, and often has devastating effects. A typical case scenario involving inflammatory resorption would be a mature avulsed tooth that is replanted, but does not receive root canal therapy within 7-10 days. The clastic cells that inhabit the root surface as part of the normal wound healing process are stimulated by bacteria and their by products that arrive on the scene by way of dentinal tubules extending from the necrotic pulp (**Figure 5**).

If an avulsed tooth is not replanted within minutes, and is transported in a manner that does not allow survival of periodontal ligament cells on the root surface, the entire root surface becomes devoid of vital cementum. When dentin is in apposition to alveolar bone without a protective layer of cementum (ankylosis), a different type of resorption takes place. In effect, the root dentin is resorbed by alveolar osteoclasts, and is replaced by bone produced by resident osteoblasts. Accordingly, this type of external root resorption is called replacement resorption (**Figure 6**).

A final category of external root resorption originates beneath the junctional epithelium of the periodontium, and has been called invasive cervical resorption, or extra canal invasive resorption. This condition is particularly confusing, as it often extends in a coronal direction, undermining enamel, and results in what is commonly called the 'pink tooth' (Figure 7). The cause of the damage to the precementum layer is often obscure; the patient may not recall an episode of trauma, and there may be no other obvious predisposing factors, such as excessive orthodontic forces, periodontal therapy or tooth bleaching. Extra canal invasive resorption is often asymptomatic, and typically discovered only during routine radiographic examinations. The lesion is usually seen as a diffuse radiolucency with ill-defined borders, and because the defect is external to the pulp canal space, it is seen to be superimposed on normal root canal anatomy. That is, the pulp canal space can be seen through

#### Figure 4



Root-end resorption caused by chronic inflammation in the periapical tissues resulting from a necrotic, infected dental pulp (panel A). Following adequate root canal therapy, the resorptive process is halted, and the bony lesion has healed (panel B) (17 month recall).

#### Figure 5



Inflammatory root resorption. The root dentin is very quickly consumed by clastic cells that are stimulated by bacteria and their byproducts. This tooth was avulsed and quickly replanted, however, endodontic therapy was delayed for 4 weeks following the injury.

#### Figure 6



Replacement resorption. Note the replacement of the root structure by trabecular bone. the resorptive lesion (**Figure 8**). The pulp is an innocent bystander, not a causative agent, therefore pulp vitality testing often reveals a normally responsive pulp.

#### **Treatment Options and Prognosis**

Treatment of external resorption depends on the cause. Surface resorption resulting from mild trauma is self-limiting; it requires no treatment and has an excellent prognosis. Resorption caused by pressure alone is treated by removing the source of the pressure; the resorptive process then stops. The prognosis depends on the amount of tooth root that has been resorbed.

Inflammatory resorption is caused by bacterial infection in an area of damaged root surface. In these cases, it is critical that bacteria be eradicated by physical and chemical means in order to allow the resorptive process to stop. This is done by performing non-surgical root canal therapy with adequate irrigation and extended application of calcium hydroxide as an intracanal medicament. For root end resorption, 1-2 weeks of intracanal calcium hydroxide should be sufficient. For inflammatory resorption secondary to avulsion, the treatment must be initiated guickly, and the calcium hydroxide is left in place until the process is seen to be under control by radiographic examination. This may take several weeks or months. The prognosis depends upon quick recognition of the problem and proper treatment. As shown in Figure 5, this type of resorption can cause a significant amount of damage in a very short period of time.

Replacement resorption caused by a completely devitalized root surface following avulsion with extended time out of the mouth is not amenable to any known treatment. The patient is advised of the condition, and it is monitored until the tooth has lost sufficient root structure to warrant extraction and prosthetic replacement.

Treatment of extra canal invasive resorption requires a combined restorative-periodontal approach. The lesion must be exposed in order to curette the granulomatous resorptive tissues in the defect. This often requires reflection of a full thickness flap in the area. Because the resorptive process often extends with fingerlike projections into multiple areas (thus, the irregular radiographic appearance), complete physical removal is difficult. These areas can be chemically cauterized by the application of an escharotic agent into the exposed crypt. Great care must be taken to not allow this agent to touch any other surfaces, as it will cause immediate necrosis. The defect must then be restored with a surgical establishment of normal biologic width. If the lesion is not diagnosed in its early stages, achieving these goals may become esthetically impractical in the anterior region, or impossible in the posterior region due to furcation exposure. Successful management depends on early radiographic recognition!

### Concluding Remarks and Future Directions

Regardless of the type of tooth root resorption, early diagnosis is the key to a successful outcome. Diagnosis relies on careful radiographic interpretation. Too often, when an obvious resorptive lesion is discovered, the subtle, early signs can then be observed in historical radiographs, after the clinician knows where to look. The clinician must carefully assess all radiographs for these early signs.

#### Figure 9





A. Conventional periapical radiograph showing a resorptive defect at the coronal aspect of the distal root that displays characteristics of both internal (well-defined borders) and external (superimposed pulp chamber and canal space) resorption. B. Cone beam computed tomograph. This representative slice shows the resorptive defect in mesio-distal, bucco-lingual and axial views. In the axial view, the white arrow points out the extent of the lesion at its greatest diameter, indicating that this is a totally internal defect without perforation to the periodontium.

#### Figure 7



A. Extra canal invasive resorption that has extended under the enamel of the tooth, resulting in a pink coloration. B. Radiograph of the same tooth, displaying the typical features of extra canal invasive resorption.

#### Figure 8



Typical radiographic features of extra canal invasive resorption. Note the irregular outline, and the ability to distinguish the normal canal space anatomy through the defect. The introduction of cone beam computed tomography (CBCT) to oral and maxillofacial radiography gives dental practitioners a distinct advantage in the diagnosis and treatment planning of all types of tooth root resorption. Three¬ dimensional imaging of the root allows the clinician to accurately distinguish between internal and external resorption processes. The periapical radiograph in Figure 9, panel A shows an obvious resorptive defect on the coronal aspect of the distal root. It has characteristics of both internal and external resorption in that the borders appear well defined (internal), but also allows the image of the pulp chamber to show through (external). Panel B is a representative slice of a CBCT scan through the level of the defect. In the axial view, the white arrow indicates the extent of the lesion at its greatest dimension, clearly showing a completely internal process that should respond to endodontic treatment with an excellent prognosis.

Treatment planning the management of external root resorption is also greatly aided by CBCT. **Figure 10** panel A shows a conventional radiograph of a lower canine tooth with extra canal invasive resorption. Panel B gives information regarding the full extent of the defect. It is obvious that surgical repair has a hopeless prognosis. The appropriate treatment plan is extraction.

A greater understanding of the biologic process of root resorption will also help the clinician determine if treatment is indicated, and what the proper treatment entails. Many resorptive lesions can be successfully treated if caught early. This is especially true for internal resorption that has not reached the external root surface. It is also true for extra canal invasive resorption that has not involved the periodontal attachment apparatus. In the future, it may be possible to deliver drugs that inhibit clastic activity via the dentinal tubules to the external root surface, obviating the need for a combined surgical restorative procedure.

#### Figure 10





A. Conventional periapical radiographic image of a lower canine tooth displaying an extra canal invasive resorptive defect in the cervical region of the root. The full buccolingual extent of the lesion cannot be determined. B. A cone beam computed tomograph of the same tooth. The axial view (white arrow) indicates that the resorptive defect extends from the buccal aspect of the root through the mesial proximal region to the lingual area. This is not surgically accessible, and thus the tooth is recommended for extraction with implant replacement.

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- 1. Although tooth roots are surrounded by a continually resorbing hard tissue (alveolar bone), external root resorption typically does not occur because:
  - a. There are no clastic cells in the vicinity of the tooth root.
  - b. Tooth root dentin is too unlike alveolar bone.
  - c. Unmineralized precementum does not allow attachment of clastic cells.
  - d. Cells that resorb bone cannot resorb dentin.
- 2. Internal, non-perforating root resorption is best treated by:
  - a. Pulp removal and obturation with gutta percha.
  - b Observation to determine if the process is arrested.
  - c. Extraction.
  - d. Pulp removal followed by the placement of an anti-clastic agent.
- 3. Root canal therapy can be expected to halt internal resorption. Root canal therapy will have no effect on inflammatory resorption.
  - a. Both statements are true.
  - b. Both statements are false.
  - c. The first statement is true, the second is false.
  - d. The first statement is false, the second is true.

#### 4. Ankylosis is often associated with:

- a. surface resorption.
- b. inflammatory resorption.
- c. replacement resorption.

## 5. Endodontic obturation of a tooth with apical root resorption may pose a unique problem with:

- a. Length control.
- b. Three-dimensional filing of the root canal space.
- c. The ability to obturate lateral canals.
- d. Achieving adequate density of the fill.

- 6. The most rapid root resorption is associated with:
  - a. surface resorption.
  - b. inflammatory resorption.
  - c. replacement resorption.
- Internal resorption starts in the pulp and causes an alteration in the canal shape as seen in the radiograph. External resorption starts in the periodontium and causes no alteration in the root canal shape as seen in the radiograph.
  - a. Both statements are true.
  - b. Both statements are false.
  - c. The first statement is true, the second is false.
  - d. The first statement is false, the second is true.
- 8. Since both internal and external resorption are typically asymptomatic, clinical diagnosis relies heavily on radiographs. Any radiographically visible defect in the root of a tooth that is not caused by caries or fracture should be suspected of being the result of a resorptive process.
  - a. Both statements are true.
  - b. Both statements are false.
  - c. The first statement is true, the second is false.
  - d. The first statement is false, the second is true.
- 9. Predentin has a protective effect against internal resorption. Precementum has no protective effect against external resorption.
  - a. Both statements are true.
  - b. Both statements are false.
  - c. The first statement is true, the second is false.
  - d. The first statement is false, the second is true.
- 10. Surface resorption can result from minor injury to the root surface. surface resorption is repaired by new cementum formation and does not require treatment.
  - a. Both statements are true.
  - b. Both statements are false.
  - c. The first statement is true, the second is false.
  - d. The first statement is false, the second is true.

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