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Feature Article

Root Resorption

Luxation Injuries and External Root Resorption -- Etiology, Treatment, and Prognosis

Martin Trope, DMD

abstract

When a tooth sustains a luxation injury, attachment damage of varying degrees will occur. In addition, necrosis of the pulp might result, thereby making the pulp space susceptible to infection. These circumstances can lead to root resorption. Treatment for root resorption includes preventing it by avoiding causes of root surface injury, minimizing initial inflammation, and reversing resorption.

When a tooth sustains a luxation injury, attachment damage of varying degrees will occur. In addition, necrosis of the pulp might result, thereby making the pulp space susceptible to infection.

Unlike root resorption in primary teeth, which occurs predictably when the permanent tooth erupts, resorption in permanent teeth will occur only under pathologic conditions. This fact appears to be due to the anti-resorptive properties of the precementum covering of the root that protects it in the presence of inflammation (**Figure 1**). However, if a luxation injury removes or alters the precementum, the inflammatory response will include root resorption with multinucleated clastic cells in addition to the "usual" bone resorption.

Thus, for root resorption to occur, two circumstances must exist: a change must have occurred to the protective attachment layer (precementum externally) of the root and an inflammatory process must be present adjacent to the damaged root surface.

Treatment strategies for root resorption include preventing it by recognizing the causes of the root surface injury and avoiding them; minimizing initial inflammation and thus the resorptive consequences by manipulating the inflammatory response; and reversing resorption by eliminating the inflammatory stimulator.

External Root Resorption Due to the Injury Alone

The luxation injury is the cause of the attachment damage. The byproducts of this mechanical damage stimulate the inflammatory response. The type of tissue that will cover the damaged root surface depends upon the extent of the initial damage.

Localized Injury -- Cemental Healing

When the injury is localized (e.g., after concussion or subluxation), mechanical damage to the cementum occurs, which results in a local inflammatory response and a localized area of root resorption. If no further inflammatory stimulus is present, periodontal healing and root surface repair will occur within 14 days[1] (**Figure 2**). The resorption is localized to the area of mechanical damage, and treatment is not required since it is free of symptoms and not even visible radiographically in most cases. However, in a minority of cases, small radiolucencies can be seen on the root surface if the radiograph is taken at a specific angle. It is important not to misinterpret these cases as progressive in nature. Failure to isolate an inflammatory stimulus (e.g., a necrotic infected pulp space) is a clue that no treatment should be performed. A "wait and see" attitude should be taken to allow spontaneous healing to take place.

Diffuse Injury -- Osseous Replacement

When the traumatic injury is severe (e.g., intrusive luxation or avulsion with extended dry time), involving diffuse damage on more than 20 percent of the root surface, an abnormal attachment can occur after healing.[2] The initial reaction as always is inflammation in response to the severe mechanical damage of the traumatic and concomitant injuries to the root surface. After the initial inflammatory response, the result is a diffuse area of root surface devoid of cementum. Cells in the vicinity of the denuded root compete to repopulate it, and often cells that are precursors of bone -- rather than slower-moving periodontal ligament cells -- will move across from the socket wall and populate the damaged root. Bone comes into contact with the root without an intermediate attachment apparatus. This phenomenon is termed dentoalveolar ankylosis.[3] Bone resorbs and forms physiologically throughout life. Thus, root is resorbed by osteoclasts; but, in the reforming stage, bone is laid down instead of dentin, thus the root is slowly replaced with bone. This process is termed osseous replacement (**Figure 3**). The initial inflammation to remove the mechanical debris of the traumatic injury is a pathologic response that in the author• s opinion may be reversed. However, the ankylosis and osseous replacement that follows cannot be reversed and can be considered a physiologic process since bone resorbs and reforms throughout life. In these traumatic cases, however, the resorptive phase includes the root.

Treatment

Because osseous replacement is not localized to a particular area and occurs peri-radicularly, local treatment is impossible.

Treatment strategies are directed at avoiding or minimizing the initial inflammatory response. More specifically, these strategies comprise preventing the initial injury; minimizing additional damage after the initial injury; minimizing the initial inflammatory response; possibly stimulating cemental (rather than bone) healing; and slowing down the osseous replacement when inevitable.

Preventing the Initial Injury

Nothing can be done about the initial damage due to the primary traumatic injury. However, there is evidence that preventive measures can limit these injuries. In athletics, the mouthguard is a proven protective device against traumatic damage to the teeth.[4] Education should be used to increase mouthguard use by athletes of all levels.

Minimizing Additional Damage After Injury

Steps must be taken to minimize additional avoidable damage to the periodontal ligament after the initial injury has taken place.

The tooth should be repositioned into its original location as soon as possible and as atraumatically as possible.[5] If splinting is necessary, it should be performed with a functional (nonrigid) splint for seven to 10 days[6] (**Figure 4**).

In the case of avulsion, replanting the tooth as soon as possible is the preferable treatment. If the tooth cannot be replanted immediately, the extraoral dry time must be minimized by placing the tooth in an appropriate storage medium.[7] Milk is a practical storage medium.[8] It is found near most accident sites, is relatively free of bacteria, and has an osmolarity that is not excessively harmful to the periodontal ligament cells. Sterile saline or saliva (vestibule of the mouth) are practical alternatives.[8,9] Water is not an acceptable medium to store avulsed teeth.[8] Newer specialized media such as Hank• s Balanced Salt Solution, which is a common culture medium, or ViaSpan, a liver transplant medium, might be able to sustain the viability of periodontal ligament cells for considerably longer periods than milk. Therefore, they might become practical for severe injuries where life-threatening situations do not allow immediate replantation.[9]

Limiting Initial Inflammatory Response

An additional approach to be considered is to pharmacologically manipulate the inflammatory response to minimize destruction and facilitate repair of the damaged root surface by new cementum and periodontal ligament.

Tetracycline has been widely used in the treatment of periodontal disease because of its sustained antimicrobial effects. Tetracycline has been shown to possess anti-resorptive as well as its antimicrobial properties.[10] Specifically, it has a direct inhibitory effect resorbing osteoclast cells and collagenase.[10] Thus, if these drugs were found to be as effective as the penicillin drugs in limiting bacterial contamination after a traumatic injury and they possessed an anti-resorptive property in addition, they might replace penicillin as the systemic antibiotic of choice after these injuries.

The effectiveness of systemic tetracycline in an "infection" model in dogs has been tested, and the results were similar to those of penicillin.[10] In addition, when used in an osseous remodeling model, tetracycline was found to result in significantly more healing than penicillin, which in fact was not different from the control group.[11] The authors therefore recommend replacing systemic penicillin with tetracycline after a traumatic injury.

While tetracycline affects osteoclasts at the site of resorption, drugs are available that affect the recruitment of osteoclasts to the site of injury.[12] Thus the combination of the two types of drugs might have a synergistic effect on the inhibition of root resorption. Glucocorticoids have been widely used to reduce the deleterious effects of inflammatory responses. More specifically, they have been shown to reduce osteoclastic bone resorption by affecting cell recruitment by a plethora of mechanisms. Therefore, they could also potentially be useful in manipulating the initial inflammatory response after attachment damage. Thus, repair by cementoblasts rather than bone-derived cells would be encouraged. The effect of local and systemic dexamethasone in a dog model that has been shown to produce a peak of inflammatory root resorption has been tested. Topical dexamethasone was found to be useful, while

systemic usage was not.[12] Other drugs such as bisphosphonates that appear to work locally and systemic NSAIDs are being tested and might offer beneficial results.

Stimulating Cemental Healing

If it were possible to stimulate periodontal cell growth, it might be possible to limit the number of osseous cells able to attach to the denuded root surface and thus decrease osseous replacement and slow down the loss of the tooth. Fibroblast-like cells in culture secrete factors that have been shown to have significant activity in tissue repair processes. It was hypothesized that the supernatant of cultured gingival fibroblasts, if used as a storage medium, might contain a number of biologically active factors that might promote periodontal ligament regeneration and repair. This "conditioned medium" was tested as a storage medium in dogs• teeth for extended periods. The results were extremely promising, as the "conditioned medium" appeared superior to Hanks• Balanced Salt Solution and Viaspan after 96-hour storage.[13] However, when tested as a periodontal-ligament-stimulating medium on roots with various extended dry periods, the results were disappointing in that Viaspan was the only medium with a beneficial result.[14] Therefore, work needs to continue to promote periodontal ligament stimulation after a traumatic injury. Specialized media such as Emdogain might prove beneficial in the future.

Slowing Down Osseous Replacement

When osseous replacement is thought to be unavoidable, such as in avulsion with extensive extraoral dry times or in a serious intrusive luxation injury, the root should be prepared to be as resistant to resorption as possible in an attempt to slow the process. Teeth should be soaked in citric acid for five minutes to remove all remaining periodontal ligament cells, soaked in 2 percent stannous fluoride for five minutes, and replanted.[15] If the tooth has been dry for more than 60 minutes and no consideration is given to preserving the periodontal ligament, the endodontics could be performed extraorally. In the case of a tooth with a closed apex, there is no advantage to performing this additional step at the emergency visit. However, in a tooth with an open apex, the endodontic treatment, if performed after replantation, involves a long-term apexification procedure. In these cases, completing the root canal treatment extraorally, where a seal in the blunderbus apex is easier to achieve, may be advantageous. When endodontic treatment is performed extraorally, it must be performed aseptically with the utmost care to achieve a root canal system that is free of bacteria.

External Root Resorption With Luxation Injury and Inflammatory Response

Pulp Space Infection

This usually occurs after a fairly serious injury resulting in areas of cemental covering of the root being damaged and its protective quality lost. If the pulp is necrotic and infected (usually from the same injury), the bacterial toxins can pass through the dentinal tubules and stimulate an inflammatory response in the corresponding periodontal ligament.[16] The result is resorption of the root and bone. The periodontal infiltrate consists of granulation tissue with lymphocytes, plasma cells, and polymorphonuclear leukocytes. Multinucleated giant cells resorb the denuded root surface and this continues until the stimulus (pulp-space bacteria) is removed[17] (**Figure 5A**). Radiographically, the resorption is observed as progressive radiolucent areas of the root and adjacent bone (**Figure 5B**). Root canal disinfection removes the stimulus to the periradicular inflammation so the resorption will stop.[1] In most cases, a new attachment will form; but if a large area of root is affected, osseous replacement can result by the mechanism already described.

Treatment

The practitioner• s attention to pulp space infection should ideally be seven to 10 days after the injury.[19,20] Again, treatment principles include prevention of pulp space infection or elimination of bacteria if they are present in the pulp space.

Prevention of Pulp Space Infection

*** Maintain the vitality of the pulp --** If the pulp stays vital, the canal will be free of bacteria. In severe injuries where vitality has been lost, it is possible under some circumstances to promote revascularization of the pulp. Revascularization is possible in young teeth with incompletely formed apices if replaced in their original position within 60 minutes[18] (**Figure 6**). If the tooth has been avulsed, soaking it in doxycycline for five minutes before replantation has been shown to double the revascularization rate.[18]

*** Prevent root canal infection by root canal treatment at seven to 10 days --** In teeth with closed apices, revascularization cannot occur. These teeth should be endodontically treated within seven to 10 days of the injury before the ischemically necrosed pulp becomes infected.[19,20] In this early stage, chemomechanical preparation and a short-term (at least seven days) intracanal dressing with a creamy mix of calcium hydroxide can sufficiently disinfect the root canal system[19] (**Figure 7**). Notwithstanding, in a compliant patient, the calcium hydroxide can be applied for a long term (up to six months) to ensure periodontal health prior to root canal filling with gutta-percha[20] (**Figure 8**).

Eliminate Pulp Space Infection

When root canal treatment is initiated later than 10 days after the accident or if active resorption is observed, the preferred antibacterial protocol consists of chemomechanical preparation followed by long-term dressing with densely packed calcium hydroxide.[20] Calcium hydroxide can affect an alkaline pH in the surrounding dentinal tubules, kill bacteria, and neutralize endotoxin, a potent inflammatory stimulator. Long-term application of calcium hydroxide is particularly beneficial in serious dental injuries with extensive areas of cemental damage that allow intracanal bacteria to interact with the attachment apparatus.

Sulcular Infection

This progressive root resorption of inflammatory origin occurs immediately below the epithelial attachment of the tooth, usually but not exclusively at the cervical area of the tooth. Its exact pathogenesis is not fully understood.[17] However, since its histological appearance and progressive nature is identical to other forms of progressive inflammatory root resorption, it appears logical that the pathogenesis would be the same (i.e., an unprotected or altered root surface attracting resorbing cells and an inflammatory response maintained by infection). Causes of the root damage immediately below the epithelial attachment of the root include orthodontic tooth movement, trauma, nonvital bleaching, and other less definable causes. The pulp plays no role in cervical root resorption and is mostly normal in these cases.[17] Because the source of stimulation (infection) is not the pulp, it has been postulated that it is the bacteria in the sulcus of the tooth that stimulate and sustain an inflammatory response in the periodontium at the attachment level of the root.[17]

Resorption of this type is asymptomatic and usually detected only through routine radiographs.

Occasionally symptoms of pulpitis will develop if the resorption is extensive. When the resorption is long-standing, the granulation tissue can be seen undermining the enamel of the crown, resulting in a pinkish appearance. This "pink spot" has traditionally been used to describe the pathognomonic clinical picture of internal root resorption, resulting in many cervical root resorption cases being misdiagnosed and treated as internal root resorption (**Figure 9**).

The radiographic appearance of subepithelial inflammatory root resorption can be quite variable. If the resorptive process occurs mesially or distally on the root surface, it is common to see a small radiolucent opening into the root. The radiolucency expands coronally and apically in the dentin and reaches, but usually does not perforate, the root canal (**Figure 10A**). If the resorptive process is buccal or palatal-lingual, the radiographic picture depends upon the extent to which the resorptive process has spread in the dentin. It can be seen as a radiolucency at the attachment level or have spread a considerable way coronally or apically with a mottled appearance (**Figure 10B**). Because the pulp is not involved, its outline can usually be distinguished through the resorptive defect (**Figure 10B**).

Treatment

Unlike the treatment of external root resorption where the focus is on eliminating the cause of the inflammatory stimulus, i.e., pulpal bacteria, the long-term removal of sulcular bacteria is not possible. The treatment strategy for this type of resorption is to remove the resorptive tissue from the root (and surrounding bone) and change the root surface to be less susceptible to further resorption. Treatment is much more difficult than in pulp space root resorption, and therefore the results are less predictable. For a review of treatment strategies see Trope M, Subattachment inflammatory root resorption: Treatment strategies. *J Pract Periodont Aesthet Dent* 10(8): 1005-10, 1998.

Author

Martin Trope, DMD, is the chair and J.B. Freedland Professor of Endodontics at the University of North Carolina at Chapel Hill School of Dentistry.

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To request a printed copy of this article, please contact: Martin Trope, DMD, Department of Endodontics, School of Dentistry, UNC Chapel Hill, Chapel Hill, NC 27599-7450 or at Martin_Trope@dentistry.unc.edu.

Figures

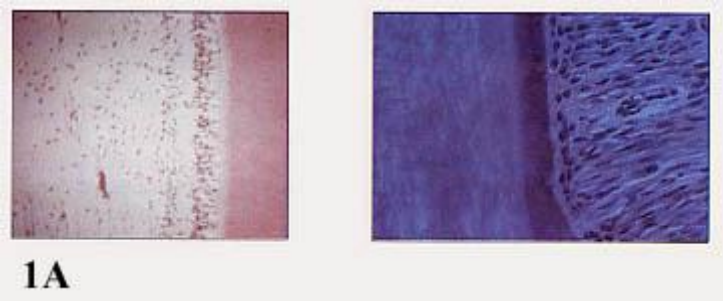

Figure 1. The histologic appearance of periodontal ligament and adjacent dentin. The precementum has anti-resorptive properties.

Figure 2. The histologic appearance of a localized area of root resorption that has healed with new cementum and periodontal ligament. The initial damage was caused by a mild localized luxation injury.

Figure 3A. The histologic appearance of active osseous replacement. Bone attaches directly to the root without an intermediate periodontal ligament. Areas of active root resorption are seen in the bone and root.

Figure 3B. Radiographically, a mottled appearance of bone replacing root is seen.

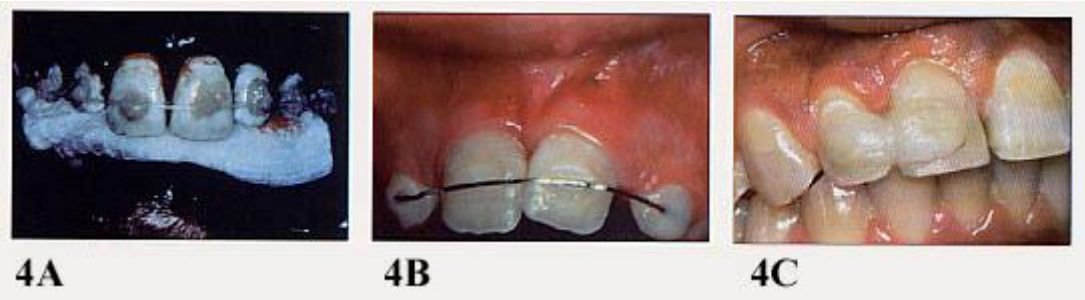

Figure 4A. Physiologic splints can be made in a variety of ways. In this example, nylon fish line is bonded to multiple teeth with resin.

Figure 4B. A physiologic splint made of dead soft wire bonded with resin between permanent and primary teeth.

Figure 4C. A physiologic splint of resin between two teeth.

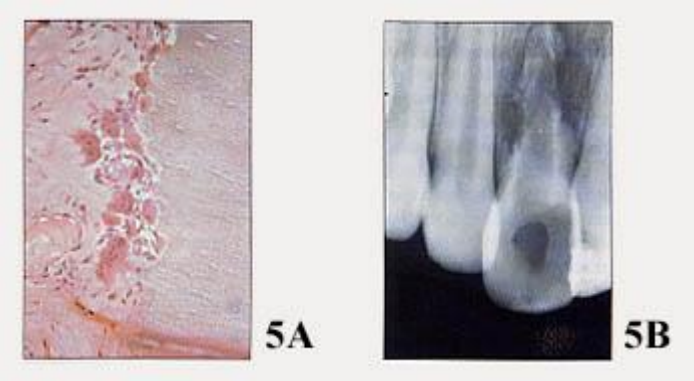

Figure 5A. External inflammatory root resorption of pulpal origin. The histologic appearance shows multinucleated giant cells resorbing the root surface.

Figure 5B. The radiographic appearance shows radiolucencies in the root and adjacent bone.

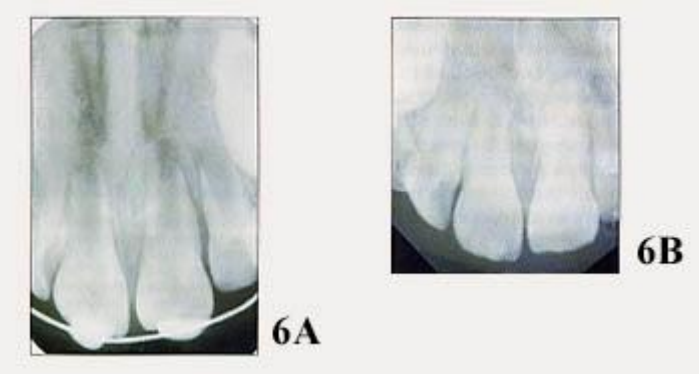

Figure 6A. Immature maxillary central incisors that have revascularized after replantation. A radiograph made at the time of replantation and splinting (courtesy Dr. Joe Camp).

Figure 6B. A radiograph made at 18 months showing continued development of the teeth (courtesy Dr. Joe Camp).

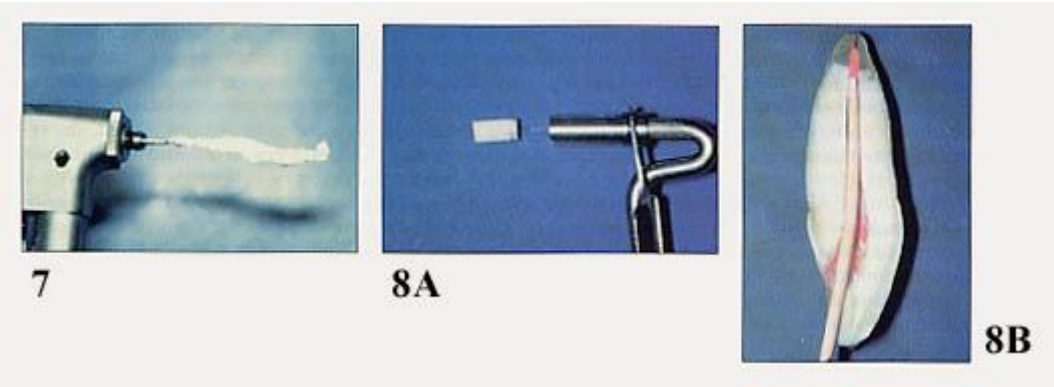

Figure 7. A creamy mix of calcium hydroxide on a lentulo spiral. Calcium hydroxide powder mixed with sterile water to this consistency and placed into the canal with a lentulo spiral is the most effective way to use this medicament for its antibacterial action.

Figure 8A. A powdery mix of calcium hydroxide placed into the canal orifice with an amalgam carrier

Figure 8B. Calcium hydroxide packed incrementally to fill the canal in an apical-coronal direction.

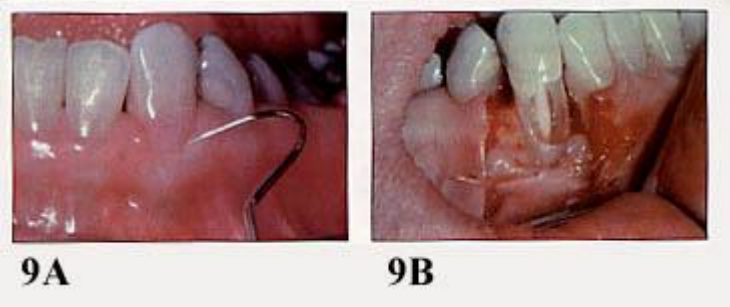

Figure 9A. A "Pink spot" of external inflammatory root resorption. The granulomatous tissue has spread coronally and undermined the enamel resulting in the pink color in the crown (courtesy Dr. Hank Rankow).

Figure 9B. Careful removal of the granulomatous tissue shows the canal to be almost entirely encircled by the resorptive defect but not penetrated (courtesy Dr. Hank Rankow).

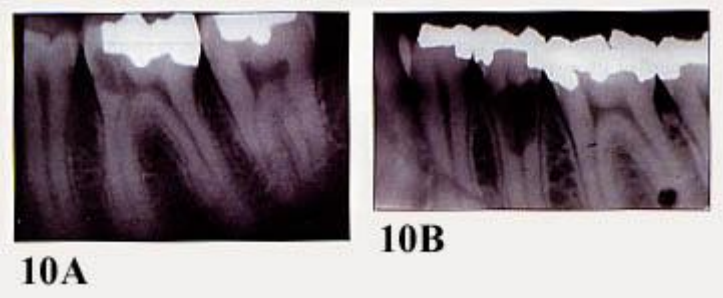

Figure 10A. Variable radiographic appearance of subepithelial inflammatory root resorption. The resorptive defect on the mesial of the first molar shows a small opening into the root, expansion apically and coronally reaching but not penetrating the pulp canal. Note the adjacent bone resorption.

Figure 10B. The resorption is buccal or lingual on both mandibular premolar teeth. The appearance of the resorption extends coronally and apically. Note that the outline of the canal can be distinguished through the resorptive defect.