

Root resorption – diagnosis, classification and treatment choices based on stimulation factors

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Abstract – Etiology of different types of root resorption requires two phases: mechanical or chemical injury to the protective tissues and stimulation by infection or pressure. Injury can be similar in various types of root resorption. The selection of proper treatment is related to the stimulation factors. Intrapulpal infection is the stimulation factor in internal root resorption and external periradicular inflammatory root resorption. Adequate root canal treatment controls intrapulpal bacteria and arrests the resorption process. In cervical root resorption, infection originates from the periodontal sulcus and stimulates the pathological process. As adequate infection control in the sulcus is unlikely, removal of granulation tissue from the resorption lacuna and sealing are necessary for repair. Removal of the stimulation factor, i.e. pressure, is the treatment of choice in root resorption related to pressure during orthodontic treatment, or an impacted tooth or tumor. In ankylotic root resorption, there is no known stimulation factor; thus, no predictable treatment can be suggested. Therefore, various types of root resorptions can be classified according to the stimulation factors: pulpal infection resorption, periodontal infection resorption, orthodontic pressure resorption, impacted tooth or tumor pressure resorption, and ankylotic resorption.

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Root resorption is a dental complication that can lead to tooth extraction. There are many classifications and terms for different types of root resorption. For example, 'apical replacement resorption' has been used for apical root resorption following orthodontic treatment (1). The same pathological process has been included under the category of 'inflammatory root resorption' (2). In the classical classification of root resorption following traumatic injuries (3), replacement and inflammatory resorption are related to completely different etiologies and treatment protocols. As this phenomenon creates confusion, it is essential to develop a new clinical-related classification which will improve communication among clinicians, teachers, and researchers. This new classification should be simple to learn and teach, and

should include the most common types of root resorption.

The etiology of root resorption requires two phases: injury and stimulation (2, 4). Injury is related to non-mineralized tissues covering the external surface of the root, the precementum, or internal surface of the root canal, the predentin. The injury is similar to several types of root resorption and may be mechanical following dental trauma, surgical procedures, and excessive pressure of an impacted tooth or tumor. It may also occur, following chemical irritation, during bleaching procedures using hydrogen peroxide 30% or other irritating agents (5). Denuded mineralized tissue is colonized by multinucleated cells, which initiate the resorption process. However, without further stimulation of the resorption cells, the process will

end spontaneously. Repair with cementum-like tissue will occur within 2–3 weeks if the damaged surface does not cover a large surface area. If the damaged root surface is large, bone cells will be able to attach to the root before the cementum-producing cells; ankylosis is the result of this process. Continuation of the active resorption process is dependent on a common stimulation factor of the osteoclastic cells, either infection or pressure. Its origin is different for each type of root resorption. Therefore, the various types of root resorption should be identified according to the stimulation factors. When these stimulating factors are identified, it will be possible to reverse the process by removing the etiological factor. The purpose of this article is to suggest a clinical-related classification of root resorption that will assist clinicians in diagnosis and treatment of this pathological process.

Pulpal infection root resorption

The most common stimulation factor for root resorption is pulpal infection (Fig. 1). Following injury to the precementum or predentin, infected dentinal tubules (Fig. 2) may stimulate the inflammatory process with osteoclastic activity in the periradicular tissues or in pulpal tissues, consequently initiating external or internal root resorption. Clinically, teeth are usually not symptomatic in the early period of the process, and resorption may be seen at this stage only in radiographs. However, as the process progresses, the teeth may become symptomatic and periradicular abscesses may develop with increasing tooth mobility.

Radiographically, radiolucency is observed in the external root surface of the dentin and adjacent bone, or in the internal root canal dentinal walls (Fig. 3).

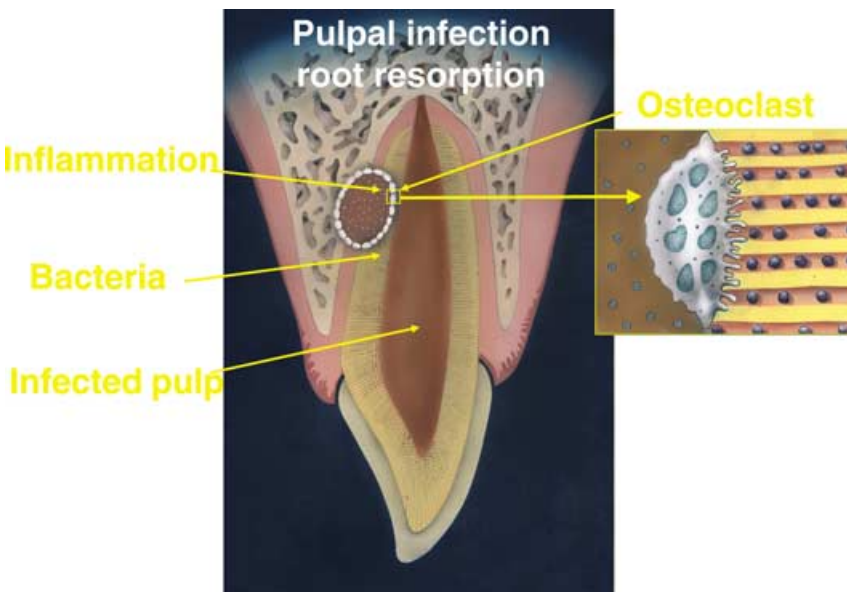


Fig. 2. Histological appearance of pulpal infection root resorption. (A) Necrotic infected root canal space, (B) infected dentinal tubules, and (C) external root resorption adjacent to the infected tubules.

Fig. 3. Radiograph showing external pulpal infection root resorption. Radiolucency is observed along the external root surface of the dentin and adjacent bone.

Fig. 1. Graphical illustration of pulpal infection root resorption. Root canal and dentinal tubules are necrotic and infected, and inflammatory response with osteoclastic activity is taking place in the dentin and the bone. Enlargement of osteoclast attached to dentin on the right demonstrates the stimulation factor of bacteria in the dentinal tubules.

Treatment: the resorbing cells in internal resorption are pulpal in origin. Therefore, a pulpectomy will remove the granulation tissue and blood supply of these cells. For this reason, a pulpectomy alone is a predictable treatment form in this type of resorption. For external resorption, it is critical to control the pulpal bacteria that act as a stimulant for the resorptive process. Bacterial stimulation removed from the dentinal tubules can predictably arrest this type of root resorption (6). Calcium hydroxide (CH) for 6–24 months is the intracanal medicament of choice for treatment of external pulpal infection (7). Its strong antibacterial effect and low solubility create a long-term effect in the root canal, and remove the stimulation factor from the main canal. CH also increases the pH of dentin (8.0–10.0), and therefore inhibits the activity of osteoclastic acid hydrolases in the periodontal tissues and activates alkaline phosphatases (8). However, the pH of the medium surrounding roots containing CH for 10 days with patent dentinal tubules does not change significantly (9, 10). The low solubility of CH and buffering effect in dentinal tubules do not allow permeability of hydroxyl ions through dentinal tubules. The antibacterial effect of CH with no additives in the dentinal tubules is significantly weaker than electrophoretically activated CH or CH with additives, such as IKI or copper (11, 12). The new intracanal medicament, Activ Point (Activ point, Roeko, Langenau, Germany), which contains chlorhexidine 5%, has shown significantly stronger antibacterial effect in dentinal tubules to a depth of 500 μm compared to CH or irrigation with chlorhexidine alone (13) (Fig. 4). These recent data should be considered in future protocols of treatment of pulpal infection root resorption.

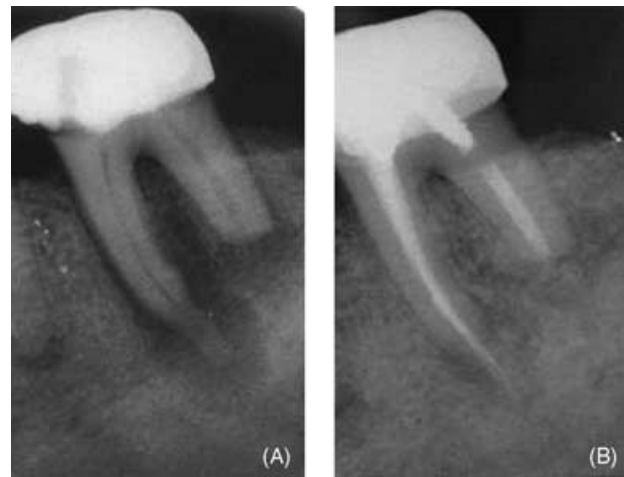


Fig. 4. Radiograph showing (A) external apical pulpal infection root resorption treated for 1 week with chlorhexidine slow-release device (Activpoint®) and (B) 6-month follow-up after obturation of the root canals.

Periodontal infection root resorption

Infrequently, external root resorption may occur after injury of the precementum, apical to the epithelial attachment, followed by bacterial stimulation originating from the periodontal sulcus (Fig. 5). Injury may be caused by dental trauma, and chemical irritation may be caused by bleaching agents, e.g. hydrogen peroxide 30%, orthodontic treatment, or periodontal procedures. Bacteria from the periodontal sulcus may penetrate patent dentinal tubules, coronal to the epithelial attachment, and exit apical to the epithelial attachment without penetrating the pulpal space (2). Consequently, the damaged area of the root surface is colonized by hard tissue resorbing cells, which penetrate into dentin through a small denuded

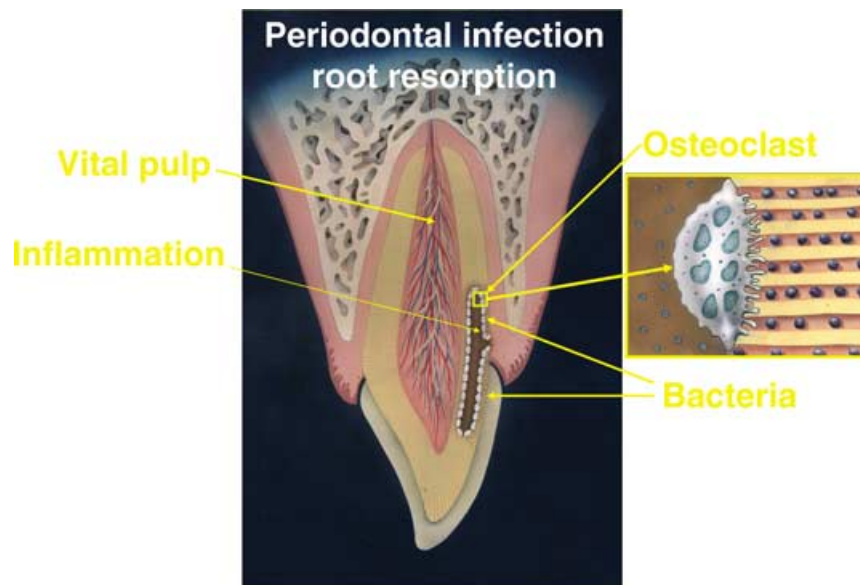


Fig. 5. Graphical illustration of periodontal infection root resorption. Pulp is vital and healthy, but the dentinal tubules adjacent to coronal cemental trauma are infected and inflammatory response with osteoclastic activity is taking place in dentin. Enlargement of osteoclast attached to dentin on the right demonstrates the stimulation factor of bacteria in the dentinal tubules. The bacteria originate from the periodontal tissues and not from the pulp space.



Fig. 6. Clinical view of pink discoloration of periodontal infection root resorption in central incisor. Granulation tissue has spread coronally and undermined the enamel, resulting in the 'pink tooth'.

area, causing the resorption inside the root to spread. At the first stage, the resorptive process does not penetrate the pulp space because of the protective layer of predentin (14), but rather spreads around the root in an irregular fashion. With time, the process may penetrate into the root canal. Additionally, periodontal infection resorption will include the alveolar bone adjacent to the resorption lacuna in the tooth. If the resorptive process reaches a supragingival area of the crown, the vascularized granulation tissue of the resorption lacuna may be visible through the enamel showing a pink discoloration at the crown (Fig. 6).

Radiographically, periodontal infection resorption can be seen as a single resorption lacuna in the dentin usually at the crestal bone level, expanding to the coronal and apical direction (Fig. 7). With progression of



Fig. 7. Radiographic view demonstrates typical periodontal infection root resorption lacuna (A) and predentin (B).

the process, radiolucency may be observed at the alveolar bone adjacent to the resorption lacuna of the dentin.

Treatment: as predictable and long-term disinfection of the periodontal sulcus is not possible, the most effective therapy is to expose the resorption lacuna orthodontically or surgically, and to remove the granulation tissue. The resorptive defect should be shaped as a cavity with retentive areas and restored with composite resins or amalgam according to esthetic demands (Fig. 8). Endodontic treatment is necessary only when there is perforation to the root canal. Where perforation is strongly suspected or ascertained, root canal treatment may be performed prior to the surgical exposure of the resorption lacuna. If the resorption lacuna is with minute external entrance openings, it can be cleaned out and obturated from the root canal (15) instead of adopting a surgical approach. Follow-up examinations are especially important to determine that the resorptive process has been arrested.

Orthodontic pressure root resorption

One complication of orthodontic treatment is the apical root resorption, with injury originating from the pressure applied to the roots during tooth movement (Fig. 9). Continuous pressure stimulates the resorbing cells in the apical third of the roots, a possibility of significant shortening of the root. Teeth are asymptomatic and the pulp is usually vital unless the pressure of the operative procedure is high, which disturbs the apical blood supply.

Radiographically, orthodontic pressure resorption is located in the apical third of the root (Fig. 10), and no signs of radiolucency can be observed in the bone or the root.

Treatment: removal of the source of the pressure results in cessation of the resorption, therefore no root canal treatment or operative procedure is needed.

Impacted tooth or tumor pressure root resorption

Pressure root resorption can be observed during eruption of permanent dentition, especially of maxillary canines (affecting lateral incisors) and mandibular third molars (affecting mandibular second molars) (Fig. 11). Tumors and osteosclerosis impinging on the root of the tooth could also be an etiological factor for pressure resorption, which includes both the injury and the stimulation phases. Stimulation is related to the pathological process that activates the resorptive cells. Tumors that produce root resorption are most frequently those in which growth and expansion are not rapid, such as cysts, ameloblastomas, giant cell tumors, and fiber-osseous lesions (2). This type of root resorption is asymptomatic with vital pulp through-



Fig. 8. Treatment of periodontal infection root resorption. (A) Surgical exposure of the resorptive lacuna, (B) removal of granulation tissue without exposing the dental pulp, (C) shaping the defect as a cavity with retentive areas, and (D) restoration with composite resin.

out the process unless the impacted tooth or tumor is located near the apical foramen, disturbing the blood supply to the pulp.

Radiographically, the resorption area is located adjacent to the stimulation factor, the impacted tooth, or the tumor (Fig. 12). There are no radiolucent areas as no infection is involved in the process. The site is filled with the stimulation factor, the impacted tooth, or the tumor.

Treatment: As the stimulation factor is related to bodily objects in the bone, surgery is necessary to remove the pressure and arrest the process. In this case, prognosis is no good.

Ankylotic root resorption

In severe traumatic injuries (intrusive luxation or avulsion with extended dry time), injury to the root surface may be so large that healing with cementum is not possible, and the bone may come into contact with the root surface without an intermediate attachment apparatus (Fig. 13). This phenomenon is termed as 'dento-alveolar ankylosis' (Fig. 14). Normally, the bone is resorbed and formed physiologically in a remodeling process without any specific stimulation with organic tissues protecting the dentin. Osteoclasts are in direct contact with the mineralized dentin in

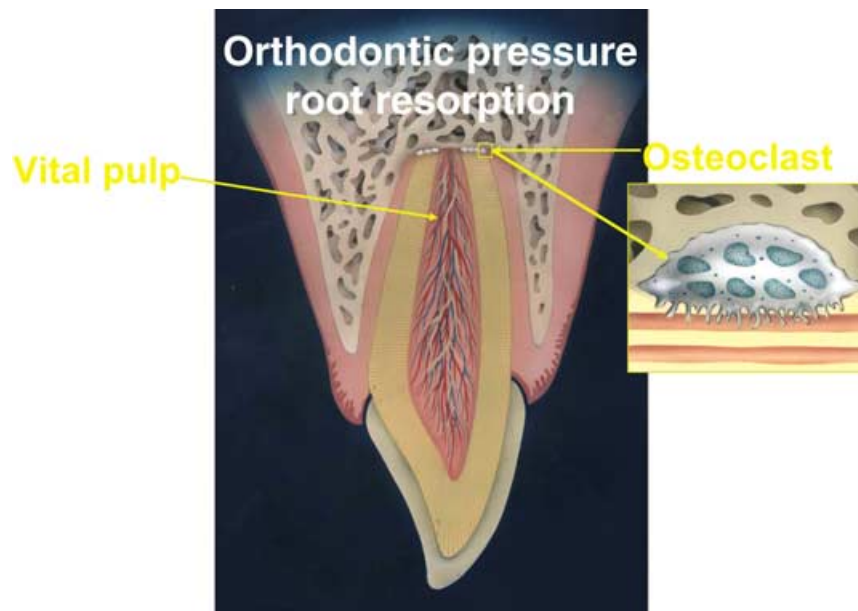


Fig. 9. Graphical illustration of orthodontic pressure root resorption. Pulp is vital and stimulation to the osteoclastic activity in the apex is related to extensive pressure during orthodontic treatment. Enlargement of an osteoclast attached to the dentin on the right demonstrates intact dentinal tubules with no bacteria.



Fig. 10. Radiographic view demonstrates typical orthodontic pressure root resorption (arrows) at the apex of three roots. No radiolucency is observed at the resorption site.

the exposed root surface after severe trauma to the root surface. Therefore, resorption can occur without any further stimulation and the bone is laid down instead of the dentin. The process may be reversed if less than 20% of the root surface is involved. Because there is no stimulation factor and the process proceeds as a result of the direct bone attachment to dentin, the term 'ankylotic resorption' is adequate. Clinically, ankylotic teeth lack the physiological mobility of normal teeth. This is one diagnostic sign for ankylotic

resorption. In addition, these teeth usually have a special metallic percussion sound and if the process continues, they are in infra-occlusion.

Radiographically, resorption lacunae are filled with bone, and the periodontal ligament space is missing (Fig. 15). No radiolucent areas are observed, and at some stage, the whole root may be replaced by bone.



Fig. 12. Radiographic view demonstrates impacted tooth pressure root resorption (arrow) of lateral incisor by an impacted canine. No radiolucency is observed at the resorption site.

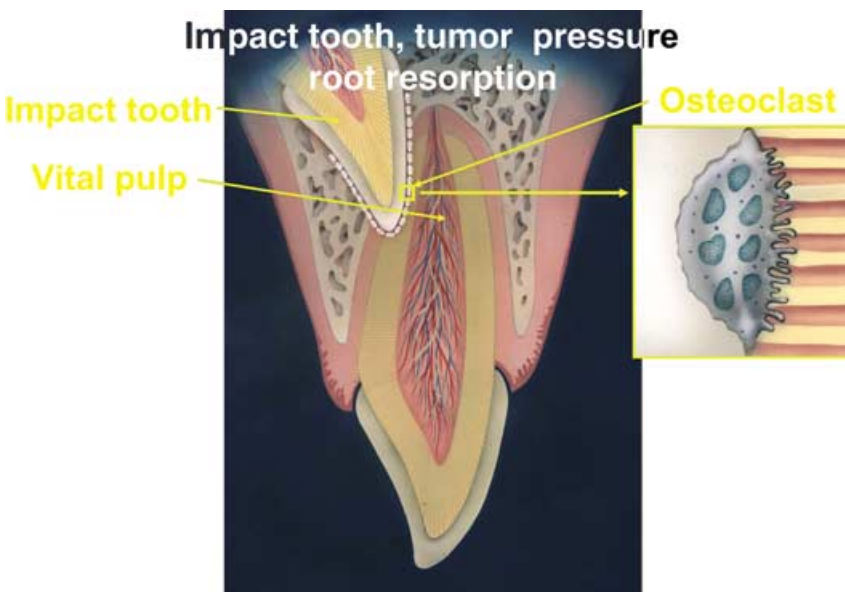


Fig. 11. Graphical illustration of impacted tooth or tumor pressure root resorption. Pulp is vital and stimulation to the osteoclastic activity is related to extensive pressure by the impacted tooth. Enlargement of an osteoclast attached to the dentin on the right demonstrates intact dentinal tubules with no bacteria.

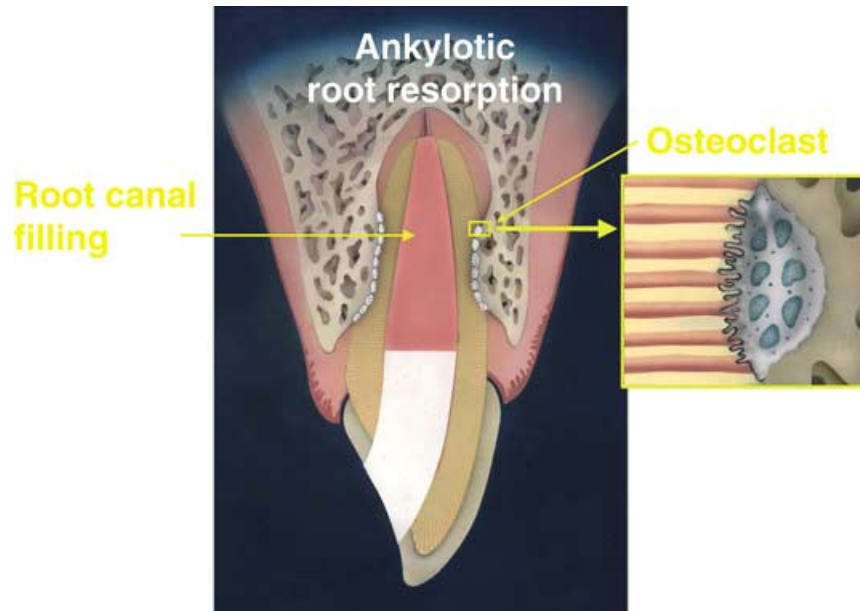


Fig. 13. Graphical illustration of ankylotic root resorption. The root canal is obturated to prevent pulpal infection root resorption and as a result of ankylosis of bone to dentin, 'physiologic' osteoclastic activity takes place with no stimulation.

Treatment: As there is no stimulation to remove, there is no predictable treatment available at present. The rate of tooth resorption varies and cannot be controlled by the patient or by the operator. Prevention by minimizing periodontal ligament damage immediately following an injury is the only treatment. The best approach is immediate replantation or placing the tooth in milk or other suitable solution to prevent dehydration of the periodontal cells (16). Functional splint is recommended for placement for 7–10 days, and root canal treatment to prevent pulpal infection root resorption (7,17). A recent experimental

approach is to arrest the initial inflammatory response to minimize or slow down the production of clastic and osteogenic cells, and to allow greater time to cementoblasts to repopulate damaged and denuded root surfaces (18). Alternately, when it is apparent that the condition of the root is such that ankylotic resorption is inevitable, certain measures (e.g. soaking the tooth in fluoride gel) can be used to slow the ankylotic resorption and allow time for planning of permanent replacement.

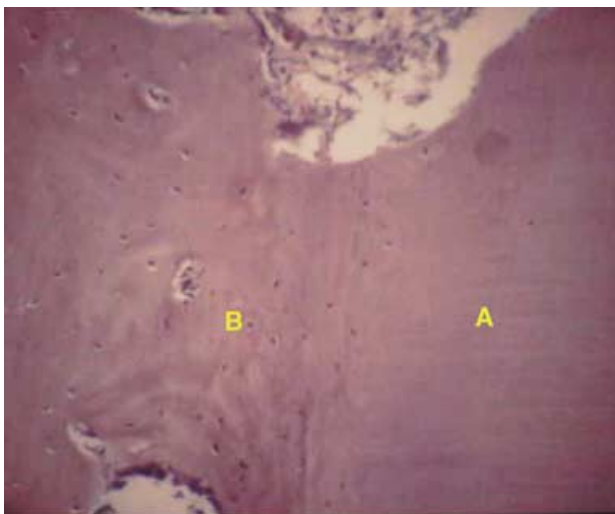


Fig. 14. Histological appearance of ankylotic root resorption. Dentin (A) is in direct contact with bone (B), and no periodontal ligament is observed.



Fig. 15. Radiographic view demonstrates ankylotic root resorption (arrows) of central incisor. No radiolucency is observed at the resorption site and bone fills the resorptive lacuna.

Summary

Identification of the stimulation factor of root resorption is useful in order to render proper treatment by removing the etiological factor. This paper presented four stimulation factors of four different types of root resorption that may assist the clinician in diagnosis and treatment of this pathological process.

References

1. Bender IB, Byers MR, Mori K. Periapical replacement resorption of permanent, vital, endodontically treated incisors after orthodontic movement: report of two cases. *J Endod* 1997;23(12):768–73.
2. Tronstad L. Root resorption – etiology, terminology and clinical manifestations. *Endod Dent Traumatol* 1988;4: 241–52.
3. Andreasen JO, Hjørtting-Hansen E. Replantation of teeth. Part I. Radiographic and clinical study of 110 human teeth replanted after accidental loss. *Acta Odontol Scand* 1966; 24:263–86.
4. Trope M. Root resorption of dental and traumatic origin: classification based on etiology. *Pract Periodont Aesthet Dent* 1998;10:515–22.
5. Friedman S, Rotstein I, Libfeld H, Stabbola A, Heling I. Incidence of external root resorption and esthetic results in 58 bleached pulpless teeth. *Endod Dent Traumatol* 1988;4: 23–6.
6. Trope M. Clinical management of the avulsed tooth. *Dent Clin North Am* 1995;39:93–112.
7. American Association of Endodontics Recommended Guidelines. Treatment of the avulsed permanent tooth. *Dent Clin North Am* 1995;39:221–5.
8. Tronstad L, Andreasen JO, Hasselgren G, Kristerson L, Riis I. pH changes in dental tissues after root canal filling with calcium hydroxide. *J Endod* 1981;7:17–21.
9. Fuss Z, Szajkis S, Tagger M. Tubular permeability to calcium hydroxide and to bleaching agents. *J Endod* 1989;15: 362–4.
10. Fuss Z, Rafaeloff R, Tagger M, Szajkis S. Intracanal pH changes of calcium hydroxide pastes exposed to carbon dioxide *in vitro*. *J Endod* 1996;22:362–4.
11. Fuss Z, Lin S, Mizrahi A, Weiss EI. Effect of electrophoretically-activated calcium hydroxide on bacterial viability in dentinal tubules *in vitro*. *Int Endod J* 2002;35:82 (Abstract).
12. Fuss Z, Mizrahi A, Lin S, Cherniak O, Weiss EI. A laboratory study of the effect of calcium hydroxide mixed with iodine or electrophoretically-activated copper on bacterial viability in dentinal tubules. *Int Endod J* 2002;35:522–6.
13. Lin S, Zukerman O, Weiss EI, Mazor Y, Fuss Z. Antibacterial efficacy of a new chlorhexidine slow-release device to disinfect dentinal tubules. *J Endod* 2003, in press.
14. Wedenberg C. Evidence for a dentin-derived inhibitor of macrophage spreading. *Scand J Dent Res* 1987;95(5):381–8.
15. Frank AL. External–internal progressive resorption and its nonsurgical correction. *J Endod* 1981;7:473–6.
16. Blomlof L, Lindskog S, Andersson L, Hedstrom KG, Hammarstrom L. Storage of experimentally avulsed teeth in milk prior to replantation. *J Dent Res* 1983;62:912–6.
17. Fuss Z. Successful self-replantation of avulsed tooth with 42-year follow-up. *Endod Dent Traumatol* 1985;1:120–2.
18. Trope M. Clinical management of the avulsed tooth: present strategies and future directions. *Endod Dent Traumatol* 2002;18:1–11.