Root resorption--etiology, terminology and clinical manifestation

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The purpose of this study are (1) to review the literature on root resorption (2) to bring the various resorptin phenomena into a general framework and to try and explain their occurrence in the light of recent knowledge about the origin and function of cells which resorb tooth. (3) to arrive at a predictable method for correctly differentiating internal and external root resorption. (4) introduce several treatment techniques.

Tooth and bone resorption as a result of osteoclast activity can be seen as the expression of three main functions of these cells (1). a maintenance function, where osteoclasts, together with osteoblasts, during continous activity maintain or change the structure of the jaws. Unless ankylosis occurs, the root is normally protected against the activity of osteoclasts and osteoblasts in their maintenance function.

The osteoclasts also have an important role in the defence against infection. Thus, inflammatory resorption of teeth due to infection in the root canal and dentinal tubules, as wee as resorption of infected necrotic bone in osteomyelitis, can both be regarded as very appropriate means of eliminating infected calcified tissues.

Another function of the osteoclast appears to be its participation in the repair of the traumatized tooth and bone. This function is typically exemplified by the resorption processes of tooth and bone after orthodontic procedures and the trancient breakdown of the apical and marginal periodontium after trauma.

The mineralized tissues of the permanent teeth are not normally resorbed. the resistance of the dental tissues to resorption seems to be an effect of 1) periodontal membrane 2) the epithelial net (Remnants of the epithelial root sheath surround the roots like a net). 3) cementoblasts in combination with non-mineralized cementoid. 4) the intermediate cementum. 5) odontoblasts in combination with the underlying predentin(2).

If the predentin or precementum becomes mineralized

or in case of precementum is mechanically damaged or scraped off, multinucleated cells will colonize the mineralized or denuded surfaces and resorption will ensue. This type of resorption may be referred as inflammatory root resorption. Replacement resorption is seen in ankylosed teeth that have become incorporated in bone. It is not a result of a disease process, but occured as a mistake because the cells involved in he remoldeling of bone are able to distinguished between the dental tissues and bone (3). It appears practical to keep the terms inflammatory resorption and replacement resorption. Whereas progressive inflammatory resorption today can be treated with a high rate of success, replacement resorption still remains beyond our clinical competence.

Transient inflammatory resorption

As mentioned above, mineralized or denuded areas of the root surface will attract hard tissue-resorbing cells that will colonize the damaged area of root. However, resorbing cells require continuous stimulation during phagocytosis(4), and stimulation by a denuded dentin or cementum surface appears not to be sufficient to sustain the resoptive process for more than 2-3 weeks. A phagocytic colonization of denuded areas of the root, therefore, will be trancient without additional stimulation of cells, and repaired with formation of a cementum-like tissue will occur both in the root canal and on the root surface. Transient root resorption occurs frequently in traumatized teeth and in teeth that have undergone orthodontic and periodontal treatment, but is also seen in other teeth, apparently as a result of wear and tear. This type of resorption is without clinical importance and the resorption defects are usually too small to even be detected radiographically.

Progressive inflammatory resorption

Root resorption initiated by a mineralized and denuded area of root surface may be prolonged by

(1) **mechanical stimulation** of macrophages and osteoclasts to sustain progressive root resorption. For instance, the sharp edges of the root at the fracture line have been selectively resorbed to give the fragments a rounded unirritating shape.

(2) **pressure resorption** in permanent dentition which may be seen during tooth eruption, especially of maxillary canines (root resorption of lateral incisor) and mandibular third molar (resorption of second molar), and in patients with the results of the expansion of a cyst or neoplasm such as ameloblastomas, radicular, dentigerous, and nasopalatine duct cysts etc.

There are two possible reasons to explain the root resorption(5). First, there is evidence that vital cyst tissue in culture releases a potent boneresorbing factor that is predominantly a mixture of prostaglanding E2 and prostaglanding E3, the source of this resorbing factor is thought to be the capsule and its leukocyte content. Another possible reason for the dentigerous cyst's potential for root resorption may be based on the fact that this cyst is derived from the dental follicle of a tooth which is prevented from erupting.. A resemblance between tooth eruption and the evolution of this cyst has been noted. Lysosomes were demonstrated in enamel epithelium, which produces the esterases and phosphotases apparantly essential for the dissolution of the bone above the eruption tooth. Similarly, the erupting tooth's follicle is observed to resorb rapidly the roots of primary teeth. It seems possible, therefor, that the capability of dentigerous cysts for resorbing the roots of adjacent teeth may be related in the origin of these cysts from dental follicles. Moreover, and impacted tooth without any follicular enlargement may be result in root resorption.

It is not reasonable to explain variations in root resorptive potential by differences in intracystic pressure. It has been shown that the ntigerous and

radicular cysts have very similar intracystic pressures and that there was a much lower intracystic pressure in ameloblastomas.

Pressure resorption is also commonly seen during orthodontic movement of teeth and digital sucking (thumb and/or finger)[6], usually in the form of apical resorption and shortening of the roots. Pressure resorption may be quite destructive if diagnosed late. However, the resorption process will be arrested when the stimulation of the resorbing cells stops.

(3) **infection of the dentin and the root canal.** Root resorption sustained by infection is mostly an endodontic complication, but is seen as a sequela to periodontal and orthodontic treatment as well. An infectious inflammation is accompanied by production and release of the normal stimulators of hard tissue resorption such as macrophage-chemotactic factor, osteoclast-activating factor, and prostaglandings. In addition, bacterial products and compoments such as endotoxins will be present[3]. A resorptive process sustained by infection can,, therefore, progress quite rapidly, and within months may cause completely destruction of the root.

(4) **systemic diseases** such as hyperparathyroidism, monostotic Paget's disease, Gaucher's disease, renal osteodystrophy, herpes zoster etc. were noted.

Internal resorption

Internal resorption has been associated with a longstanding, chronic inflammatioon in the pulp. The resorptive activity is sustained by infection of necrotic pulp tissue in the root canal coronally to the area where the resorption take place. Inflammatory cause by bacterial invasion has been shown to be accompanied by the productin and release of potent stimulators of hard tissue resorption such as osteoclast activating factor, prostaglandins, macrophage-chemotactic factor and bacterial lipopolysaccharides[7]. This stimulation may necessary for the progression of the internal resorption as the macrophages have been shown to require continuous stimulation during phagocytosis. It may concluded the speeding of macrophage-like resorbing cells was related both to the abscence of a predentin layer and bacterial status of pulp. Hense, trauma (denuded of odontoblast and predentin) and infection may be regarded as two important etiological factor in internal resorption. Thus, in order for internal resorption become progressive. The dentinal tubules have to have a special and fortuitous course. They have to be open to an area of the root canal where the tissue is necrosis and infected so that microorganisms may enter the tubules, and then lead to an area of the canal with the vital pulp tissue. This is a rather unlikely occurrence and probably explains why progressive internal resorption is a rarity in permanent teeth[3].

The resorptive area and the root canal apical to this area will contain vital tissue. In some instances the entire pulp will be necrotic. The resorptive process will then have stopped since vital tissue is obviously needed for the resorption to go on. However, in most instances it will be due to an external resorptive process perforating to the root canal. The type of external resorption most often misdiagnosed as the internal resorption is a cervical resorption.

External resorption

From a practical and clinical point of view, external root resorption may be divided into 3 categrories: 1) progressive external inflammatory resorption; 2) cervical resorption; and 3) replacement resorption.

Progressive external inflammatory resorption

This type of resorption is mainly an orthodontic or endodontic problem. Progressive root resorption caused by orthodontic tooth movement is related to tissue pressure. Thus when the orthodontic forces are removed, the resorption should stop.

Practically all teeth with apical periodontits will exhibit apical resorption. The pulp tissue is necrosis, the root canal is infected, and periapical granuloma has developed leading to resorption of dental tissues and bone. Clinically, this condition is rarely of concern and when the root canal is instrumented, disinfected and sealed, the resorption will stop.

However, in teeth that have suffered displacement injuries, external root resorption may become extensive. Extrusion or intrusion of teeth as well as the subsequent repositioning procedures inevitably will cause damage to the Root, resulting in denuded areas on the root surface, which will be chemotactic to hard tissue-resorbing cells. Root resorption will then ensue[3].

In addition, the displacement of the teeth leads to a disruption of the blood vessels at the apical foramina and to ischemic pulp necrosis. Microorganisms will reach the root canal throught enamel-dentin cracks and exposed dentinal tubules, and an infection will established within a peroid of 2-3 weeks. Bacterial products from the infected root canal will then reach the resorptive lacunae on the root surface through the dentinal tubules and sustain the resorption of the root.

In luxated teeth, the root resorption is initiated by mechanical traum, resulting in removal of cementoblast, precementum and sometimes cementum in areas of root surfaces. The resorptive process is then maintained by microbial stimuli from the infected root canal. Intense inflammatory reaction was founded in the periodontal membrane with the present of lymphocytes, plasma cells and polymophpnuclear leukocytes in granulation tissue. There are also observed in the same are a vivid proliferation of capillaries. In relation to this inflammation the root surface showed intensive resorption with numerous Howship's lacunae, which occasionally contain osteoclasts.

The inflammatory reaction was apparantly caused by toxic products from necrotic pulp tissue. After a few weeks the condition can be recognized radiogra phically as periradicular radiolucent areas usually encompassing areas of the root and adjacent alveolar bone. If allowed to progress, the resorptive process will cause eventual destruction of the root if the inflammatin is not eradicated. Any adequate endodontic treatment method will have an effect on the resorptive process. Furthermore, the use of antibiotics in combination with a corticosteroid paste such as Ledermix, triamcinolone, dexamethasone or indomethacine etc.[8,9] eliminitated the inflammatory reaction in the periodontal membrane in traumatically injuried teeth. Because this form of therapy results in no adverse local or systemic effects and effective in reducing postoperative pain, it use in the treatment and prevention of inflammatory root resorption is advocate. (Ledermix Paste consists of a broad-range antibiotic, demethylchlortetracycline, and a corticosteroid triamcinolone[8].

However, at present long term root canal treatment with calcium hydroxide is the method of choice. The high PH(12.5) Having a bactericidal and necrotizing effect seemed to be sufficient to explain the therapeutic effect of caicium hydroxide on root resorption.

In addition, introduction of calcium hydroxide into the root canal could have an active influence on the local enviroment of the resorption areas through the dentinal tubules. Calcium hydroxide may necrotize the cells of resorption lacunae or at least will neutralize the lactic acid from macrophages and osteoclast, thus preventing a dissolution of the mineral component of the root[10,11]. Moreover, an alkaline PH at the resorption sites will be unfavorable for the collagenase and acid hydrolase activity of the resorption cells and might also activate a calcium-dependent ATP-ase, which has been associatedwith hard tissue formation[12]. The antibacterial effect of calcium hydroxide and its ability to denature proteins in the root canal, rendering them less toxic or nontoxic, may be of importance.

The calcium hydroxide treatment is discountinued when a continuous periodontal ligament space is observed radiographically along the root, usually within 6-12 months. The root canal is then permanently obturated. A success rate of 96% has been reported for the treatment of the progressive external inflammatory resorption with this method[13].

Cervical resorption

Cervical resorption is seen clinically and radiographically as a single resorption lacuna in the cervical area of the tooth. It appears to follow injury to the cervical attachment apparatus, most importantly to an area of cervical root surface (precementum) below the epithelial attachment. However, necessary stimulation of the resorbing activity for longer periods of time may be provided by bacterial products via the tubules of the cervical dentin, not from the root canal, but apparantly from the gingival sulcus and the surface of tooth. If the local injury lead to necrosis of the periodontal ligament tissue in the area, cervical resorption may take the form of ankylosis and replacement resorption.

The process appears to originate in the cemental surface of the cervical area and may extend with long finger-like projections deeply into dentin[14]. At first the resorptive process will not penetrate to the pulp because of the protective qualities of the predentin[2], but rather spread around the root canal in an irregular fashion. In general, the resorptive process does not affect the predentin or dentin immediatly adjacent to the pulp[15]. It is often characterizedby a secondary deposition of mineralized tissue which presents a mottled radiographic apperance[16]. Because of this pattern of spreading inside this the root, cevical resorption is also referred to as external-internal[17] or invasive resorption. With time the resorptive process usually will penetrate the root canal. In addition, cervical resorption will include the alveolar bone adjacent to the resorption lacuna in the tooth[3].

Althought the resorption may be extensive and superimposed radiographically over the root canal, close examination of the radidograph will show that the outline integrity of the root canal and pulp chamber remain intact[18]. The integrity of the pulp space and its separation from the resorptive process are confirmed by examining extracted teeth involved with this type of resorption. In spite of the often extensive resorptive destrution of detine. Pulp responses are positive, indicating pulp vitality. Since the condition tends to asymptomatic, it is usually observed on a radiograph.

Fluctuance or a fistula may develop after the external origin becomes supraosseous caused by periodontal breakdown[17]. If the resorptive process reaches a supra gingival area of crown, the well vascularized granulation tissue of the resorption lacuna may be visible throught the enamel and the patient will present with a so-call pink spot or pink tooth[3].

In most instances cervical resorption is seen as a late complication to traumatic injuries of the teeth. It may also occured after orthodontic movement, orthognathic and other dento-alveolar surgery[19], congenital defects in the development of cementum, periodontal inflammation, and bleaching processes in root-filled teeth[20]. In many instances the history is obscure.

It is not a frequently occuring complication following tooth movement. Cervical resorption is sustained by infection and will, therefore, not stop when the orthodontic forces are removed.

External cervical resorption may be further classified as intraosseous or supraosseous, depending on the location of the origin and entry of the resorptive process[18].

Suggested treatment of the intraosseous external cervical resorption: The treatment procedure is based upon the necessity to debride the resorptive defect and to close the site of its entry. To accomplish this, the first step is to extirpate the pulp and fill the root canal. Then, throught the coronal access opening, the resorptive defect is debrid-

ed with the use of long shank round burs, after which the entire defect and access opening is filled with a restorative material. The debridement of the resorptive defect tends to be random and blind since the site of the resorptive origin is difficult, if not impossive, to determine preoperatively. Since the entry site is intraosseously, there is no periodontal breakdown to assist in locating a potential site for surgical intervention and the extent of the destruction cannot determined radiographically. Along with the weakening of the tooth due to loss the tooth struture, all of these factors combine to offer a guarded prognosis for the intraosseous external cervical resorption. Hovever, it may be preferable to attemp to treat, in spite of the poor diagnosis, than to merely ''watch'' the destrutive resorption progress to the inevitable finality--extraction[17].

Suggestion treatment of the supraosseous external cervical resorption: Treatment for supraosseous invasive resorption has generally been to extirpate the pulp and fill the root canal, followed by burring out the defect and placing a restoration from an external approach. The prognosis for this clinical technique has been favorable due to the relative ease in locating the resorption externally. If the external aspect of a defect can be located, it can be enlarged and carefuly excavated without exposing the pulp, this circumventing the routine inclusion of root canal treatment in the procedure. If a perforation to the canal is ascertained or strongly suspected during the examination of the patient, the root canal treatment should performed prior to a surgical exposure of the resorption lacuna[18].

However, if this method is used, follow-up examinations are espscially important to make certain that the resorptive process in fact has been arrested.

Dento-alveolar ankylosis and replacement resorption

Ankylosis represents a fusion of the alveolar bone with the root surface and cn be demonstrated histologically 2 weeks after replantation. The pathogenesis of replacement resorption is manifested in two ways-- either permanent (progressive) replacement resorption which gradually resorbs the entire root, or transient replacement resorption in which a once-established ankylosis later disappears. The progressive form is always elicited when the entire periodontal ligament is removed before replantation is possibly related to minor areas of damage on the root surface.

Clinically, the condition is most often seen as a complication to luxation injuries, expecially in avulsed teeth that have been out of the mouth long enough for the cells on the root surface to dry out and die. If less than 20% of the root surface is involved, reversal of ankylosis may occur[21,22]. If not, ankylosed teeth are incorporatedin the alveolar bone and will become part of the normal remodeling process of bone. The osteoclasts will resorb the dental tissues just as readily as they resorb the bone, and the osteoblasts tha are not capabe of forming dentin or cementum subsequently will replace the resorbed areas of the root with bone. Furthermore, masticatory stimulation during the healing period will maximize the area of functional PDM and minimize dentoalveolar ankylosis. Antibiotics should be administered as early as possible and endodontic treatment should be performed within the first weeks after replantation to prevent inflammatory resorption[23].

Clinically, dento-alveolar ankylosis is recognized because of a lack of mobility of ankylosed teeth[22]. These teeth will also have a special metallic percussion sound, and after sometime they will be in infraocclusion. Radiographically, dento-alveolar ankylosis may be recognized by the abscence of a periodontal ligament space. Also, replacement resorption with ingrowth of bone into the dental tissues will give the tooth a characteristic motheaten appearance. There is no treatment for this condition at present, and ultimately the crown of the tooth will break off at the gingival crest and fall out.

The speed with which the root is replaced by bone varies, and is dependent, among other factors, on the metabolic rate of the patient. In most instances, it takes years, sometimes decades before the root is fully resorbed. The rate of replacement resorption was also shown to be age related. Even though replacement resorption is present,a tooth in an old individual can remain functional for a long period, possible throughout life. Furthermore, residual infection and associated periodontal inflammation may be moderating factors in the progression of replacement resorption[23].

Summary of radiographic appearances of various lesions

Lesion	Radiographic appearance
External resorption	Apex shortened, flattened, or square
	Foramen is at apex, opening can be seen
	Walls of canal converge apically
	Margins of lesion are ragged and irregular
	Lesion may be superimposed over canal
	Canal can be followed all the way to apex, unaltered
Internal resorption	Canal or chamber shows enlarged area
	Lesion can be symmetrical or eccentic
	Margins of lesion are sharp, smooth, and clearly defined
	Canal not present in area of lesion
	Size and location can vary considerably
Early pulp death	Foramen wide open
(incomplete formation)	Walls does converge apically bu diverge
	Apex blunt and square
	Root length short
Dental caries	Usually closer to crown of tooth
	Less sharp and more poorly defined lesion
	Progresses from outward to inward
	Margin of lesion not abrupt[24]

Conclusion

As can be seen from the above, root resoption is a multidisciplinary problem in dentistry. However, with our present understanding of etiology, pathogenesis and clinical manifestations of the resorptive processes, our therapeutic measures can be more focus and effective than before. Only the replacement resorption occurring in teeth with dento-alveolar ankylosis remains beyond our therpeutic competence. This article presents here provides practitioner with a guide to identify predictably the particular entity present, which enables him to establish a diagnosis and prognosis more readily, and to institute appropriate treatment.

BIBLIOGRAPHY

1,J.O. Andreasen. external root resorption: its implication in dental traumatology, paedodontics, periodontics, orthodontics and endodontics. International Endodontic Journal. 1985, 18, 109-118.

2,Lars Hammarstrom & Sven Lindskog. General morphological aspects of resorption of teeth and alveolar bone. Internatinal Endodontic Journal. 1985, 18, 93-108.

3,Tronstad L. Root resorption- etiology, terminology and clinical manifestations. Endod·Dent Traumatol 1988; 4: 241-252.

4,Shaw DR, Griffin FM Jr, Phagocytosis requires repeated triggering of macrophage phagocytic receptors during particle ingestion. Nature 1981; 289: 401-11

5,P, Struthers and M. Shear Root resorption by amelobiastomas and cysts of the jaws. Int.J.Oral Surg. 1976:5:128-132.

6,Irving Rubel. Atypical root resorption of maxillary primary central incisors due to digital sucking: a report of 82 cases. J. of dentistry for children. 1986; May-June: 201-204.

7,Cecilia Wedenberg and Sven lindskog. Experimental internal resorption in monkey teeth. Endod Dent Traumatol 1985; 1: 221-227.

8,Pierce A, Heithersay G, Lindskog S. Evidence for direct inhibition of dentinoclasts by a corticosteroid/antibiotic endodontic paste. Endod Dent Traumatol 1988; 4: 44-45.

9,Angela Pierce, Sven Lindskog & Stockholm. The effect of an antibiotic/corticosteroid paste on inflammatory root resorption in vivo. Oral Surg. August 1987; 216-220.

10,Tronstad, J.O. Andreasen G. Hasselgren, L. Kristerson & I. Riis. PH changes in dental tissues after root canal filling with calcium hydroxide. J. of Endodontics Vol 7, No1, Jan 1981.

11,Hammarstrom LE, Blomlof LB, Feiglin B, Lindskog SF. Effect of calcium hydroxide treatmant on periodontal repair and root resorption. Endod Dent Traumatol 1986; 2: 184-189.

12,Magnusson, B.C., and Linde, A. Alkaline phosphatase, 5-nucleotidase and ATP-ase activity in the molar region of the mouse. Histochemisty 42: 221-232, 1974.

13,Cvek M. Endodontic treatment of traumatized teeth. In: Andreasen JO. ed. Traumatic injuries of the teeth. 2nd ed. Copenhagen: Munksgaard, 1981; 321-84.

14,Alexander J. Moule & Robyn P. Thomas Cervical external root resorptin following trauma- a case report. International Endodontic Journal 1985 18, 277-281.

15,Pindborg, J.J. (1970) Pathology of the dental hard tissues, p. 337.

16,Makkes, P.C. & Thoden Van Velzen, S.K. (1975) Cervical external root resorption. J. of dentistry, 3, 217-222.

17,Alfred L. Frank, External-internal progressive resorption and its nonsurgical correction. J. of endodontics Vol 7, No 10, Oct.1981.

18,Alfred L. Frank & Leif K. Bankland, Nonendodontic therapy for supraosseous extracanal invasive resorption. J. of endodontics Vol 13, No.7 July, 1987.

19,GernerNW, Hurlen B, Bergland O, Semb G, Beyer-Olsen EMS. External root resorption in patients with secondary bone-grafting of alveolar clefts. Endod Dent Traumatol 1986; 2: 263-266.

20,Willian W.Y. Goon, Stephen Cohen & Ronald F. Borer, External cervical root resorption following bleaching. J. of Endodontices, Vol. 12, No. 9, September 1986.

21,Andreasen JO, Kristersson L. The effect of limited drying or removal of the periodontal ligament. Periodontal healing after replantation of mature permanent incisors in monkeys. Acta Odontol Scand. 1981; 39: 1-13.

22,Andreasen JO. Periodontal healing after replantation of traumatically avlued human teeth. Assessment by mobility testing and radiography. Acta Odontol Scand. 1975; 33:325-35.

23,Andersson-L. Dentoalveolar ankylosis and associated root resorption in replanted teeth. Experimental and clinical studies in monkeys and man. Swed-Dent-J-Suppl. 1988.56.P 1-75.

24,Arnold H. Garter, Thomas Mack, Richard G. somerlott & Lawrence C. Walsh. Differential diagnosis of internal and external root resorption. J. of Endodontics Vol.2 No.11, Nov. 1976.