The Online
ANGLE ORTHODONTIST
An International Journal of Orthodontics and Dentofacial Orthopedics
78th Anniversary 1930–2008

TABLE OF CONTENTS

The Angle Orthodontist: **Vol. 72, No. 2, pp. 175–179.**

Orthodontically Induced Inflammatory Root Resorption. Part I: The Basic Science Aspects

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ABSTRACT

Orthodontically induced inflammatory root resorption (OIIRR) or, as it is better known, root resorption, is an unavoidable pathologic consequence of orthodontic tooth movement. It is a certain adverse effect of an otherwise predictable force application. Although it is rarely serious, it is a devastating event when it is radiographically recognized. Orthodontics is probably the only dental specialty that actually uses the inflammatory process as a means of solving functional and esthetic problems. Force application initiates a sequential cellular process. We know exactly how and when it is evoked, but we are unable to predict its actual overall outcome. The extent of this inflammatory process depends on many factors such as the virulence or aggressiveness of the different resorbing cells, as well as the vulnerability and sensitivity of the tissues involved. Individual variation and susceptibility, which are related to this process, remain beyond our understanding. We are therefore unable to predict the incidence and extent of OIIRR after force application. This contemporary review is divided into two parts. In Part I, we discuss the basic sciences aspects of OIIRR as a continuation of our previously published work. In Part II, we present the clinical aspects of this subject.

KEY WORDS: Root resorption, Orthodontic treatment, Review.

Accepted: October 2001. Submitted: May 2001

INTRODUCTION Return to TOC

Orthodontically induced inflammatory root resorption (OIIRR) or, as it is better known, root resorption, is an unavoidable pathologic consequence of orthodontic tooth movement. This contemporary review of OIIRR is divided into two parts. In this part, we discuss the basic sciences aspects of OIIRR in a continuation of our previously published work.[1] In part II, we present the clinical aspects of this subject.

THE ROOT RESORPTION PROCESS Return to TOC

The publications of Wehrbein et al[2,3] made substantial contributions to the research concerning OIIRR in humans. These authors discussed different grades of root resorption in detail, mainly in terms of the close proximity of the root to the cortical nonmetaplastic bones, as well as other pathologic phenomena such as dehiscence and fenestrations. These publications highlighted the risk and perhaps the iatrogenic effect of orthodontic treatment. However, it is important to note that the aforementioned studies were based solely on one human nonexperimental study.

Orthodontic force applications induce a local process that includes all of the characteristics of inflammation[4]: rubor (redness), calor (heat), tumor (swelling), dolor (pain), and, to a small extent, functio laesa (inhibited function). This inflammation, which is essential to tooth movement, is actually the fundamental component behind the root resorption process.[5]

Therefore, in light of the full extent of the histologic process, orthodontic force–induced root resorption should be more accurately termed *orthodontically induced inflammatory root resorption* (OIIRR).

There are three degrees of severity of OIIRR:

1. Cemental or surface resorption with remodeling. In this process, only the outer cemental layers are resorbed, and they are later fully regenerated or remodeled. This process resembles trabecular bone remodeling.[6]

2. Dentinal resorption with repair (deep resorption). In this process, the cementum and the outer layers of the dentin are resorbed and usually repaired with cementum material. The final shape of the root after this resorption and formation process may or may not be identical to the original form.

3. Circumferential apical root resorption. In this process, full resorption of the hard tissue components of the root apex occurs, and root shortening is evident. Different degrees of apical root shortening are, of course, possible. When the root loses apical material beneath the cementum, no regeneration is possible. External surface repair usually occurs in the cemental layer. Over time, sharp edges may be gradually leveled. Ankylosis is not a common sequel of OIIRR.

The cellular process

The studies in mice and rats conducted by Brudvik and Rygh[7–12] confirmed that OIIRR is a part of the hyaline zone elimination process. The first cells to be involved in this necrotic tissue removal are cells that are negative for tartrate resistance acid phosphatase (TRAP) and that have no ruffled borders. These are Macrophage-like cells, which are most probably activated by signals coming from the sterile necrotic tissue, the result of the orthodontic force application. Macrophages are scavenger cells from the hematopoietic lineage, and their role is to eliminate necrotic tissues. As described by Brudvik and Rygh,[7] the initial elimination process takes place at the periphery of the hyaline zone, where blood supply to the periodontal ligament exists or is even increased.[5] During removal of the hyaline zone, the nearby outer surface of the root, which consists of the cementoblast layer covering the cementoid, can be damaged,[13] thus exposing the underlying highly dense mineralized cementum. It is possible that the orthodontic pressure itself directly damages the outer root surface layers in such a way that there is a need for their removal as well. The root surface under the main hyaline zone is resorbed only several days later when the repair process in the periphery is already taking place. These finding have been confirmed by studies of human premolars that were moved buccally before their extraction.[14–23]

The resorption process continues until no hyaline tissue is present and/or the force level decreases. Resorption lacunae expand the root surfaces involved and thereby indirectly decrease the pressure exerted through force application. Thus, decompression allows the process to reverse and the cementum to be repaired.[11,12]

The role and the kinetics of the osteoclasts in tooth movement and root resorption during different reactivation schedules have been studied in rats.[24–28] In all but one of the activation and reactivation schedules, the amount of root surface involved in the resorption reached a plateau of 50% of the root surface on the compression side.[24] The extent of root resorption was increased only when force reactivation was performed at the peak presence of osteoclast count in the involved region (day 4). In all other reactivation schedules, tooth movement was improved with no risk to root surfaces. Osteoclast recruitment after activation and reactivation do not exhibit the same pattern. There are unexplained different refractory periods of 10, seven, four, and one days after reactivation and after one, four, seven, and 10 days, respectively. Since the refractory period is 10 days following the first activation, it is only a matter of simple mathematics that if reactivation is after one day, the refractory period is 10 days, and if reactivation is after four days, the refractory period is 7 days, etc. The immediate increase in osteoclasts count after one day indicates that in rats, when force is applied to the teeth, progenitor cells in the osteoclast lineage are ready in the periodontal ligament or its immediate vicinity, waiting to be fully expressed.

Brudvik and Rygh[7] found that in addition to the mononucleated macrophage-like cells, multinucleated TRAP-positive giant cells without ruffled borders are involved in the hyaline tissue removal. These cells may be osteoclasts or odontoclasts that did not come to full expression earlier (ie, preosteoclasts) and that become involved in the necrotic tissue elimination. On introduction of a new mechanical stimulus, they differentiate into fully developed osteoclasts or odontoclasts in a matter of hours.[6,29,30] (TRAP-positive cells with ruffled borders and a clear zone are pathognomonic to the clast lineage.)

Most studies agree that osteoclasts and odontoclasts are comparable cells. Lasfargues and Saffar[31] believe that the odontoclast, unlike the osteoclast, is prostaglandin independent because indomethacin administration to rats during physiologic tooth movement decreases bone resorption but enhances root resorption. Others have not confirmed the results of this study.

Root resistance to resorption

All new reports in the literature recognize the overall protective function of the root's outer layers, the cementoblasts, and the outer uncalcified cementum (the precementum or cementoid).[12,32,33] These layers might contain noncollagenic materials, eg, the cells themselves, that possess potent anticollagenase properties. This is in agreement with the findings of previous reports.

A clinical study revealed that exposure of the roots to two sequential orthodontic treatment procedures, one administered during adolescence and the other administered later during adulthood, actually decreased the extent of OIIRR.[34] Accordingly, two questions might be raised: (1) what sort of protective effect was provided to the roots by the first treatment? and (2) could the remodeled cementum contribute some additional protective effect in the outer layers? These questions have yet to be answered.

The repair process

Morphologically, the repair process of the resorbed lacunae is described as beginning from the periphery,[35] the bottom,[17] or all directions.[13] It begins about two weeks after force removal, with the placement of acellular cementum succeeded by cellular cementum. This process is evident in 38% and 82% of human premolar lacunae after two and five weeks, respectively.[17]

In bone, osteoclasts undergoing apoptosis leave at the bottom of the lacuna a protein layer that is composed partially of osteoponin and bone sialoprotein. This layer is recognized later as the cemental line with which the osteoblasts meet on bone formation. According to Bosshardt and Schroeder,[36] the odontoclasts leave the root lacunar surfaces exposed with no sediment at the base of the crater. Recently, however, it has been reported that a specific cementum attachment protein (CAP) has been identified in human cementum.[37] This protein has the ability to bind to mineralized root surfaces with high affinity. Its role in cementogenesis and cementoblast recruitment is still under investigation.[37,38] Individual variations characterize the repair process as evident in OIIRR.[17,39]

Effects of pharmacologic agents on OIIRR

The effects of L-thyroxine on root resorption are still controversial. Administration of very low doses of this hormone to rats during 10 days of tooth movement decreased the amount of root resorption by about 50% relative to that in a control group.[40] Although the conclusions of this study were far from definitive, the investigators prescribed 0.5 g of thyroid to three so-called OIIRR high-risk patients.[41] They reported that treatment with the hormone produced no new root resorption, no worsening of the existing resorption, and no adverse effects. The definition of *high-risk* patients that was used in the study was partly challenged by Owman-Moll and Kurol.[17] Christiansen's[42] comment on this study did not clarify the mechanism by which the hormone acts. It is assumed that the hormone either increases the resistance of the cementum and dentin to clastic activity or increases the rate of alveolar bone resorption (high levels of alkaline phosphatase were found). Thus, the hormone enhances tooth movement as it indirectly reduces OIIRR.

Furthermore, no substantial differences were found in the response of monocytes to L-thyroxine and thyrocalcitonin in a study that measured the amount of interleukin-β and tumor necrosis factor in two groups that had completed orthodontic treatment, one with severe root resorption and the other with no root resorption.[43] Shirazi et al[44] demonstrated that in rats, increasing doses of L-thyroxine decreased the extent of root resorption while increasing the amount of tooth movement.

Few studies have been published in the last decade about the role of prostaglandins in OIIRR. Two studies confirmed the known role of this major cytokine.[45,46]

Bisphosphonates, potent inhibitors of bone resorption, causes a significant dose-dependent inhibition of root resorption in rats after force application.[47–49] On the other hand, others have reported increased resorption with bisphosphonate treatment.[50,51] According to Alatli et al,[50,51] injections of 1-hydroxyethylidene-1-bisphosphonate (HEBP) in rats induces a cementum surface alteration, inhibits formation of acellular extrinsic fiber cementum (AEFC), and delays formation of cellular mixed-fiber cementum. Hence, HEBP increases the vulnerability of the root surface to resorption during orthodontic tooth movement.[50,51]

Reports in the literature do not agree on the origin of AEFC. Is it a part of the cementum itself, laid down by cementoblasts,[50–52] or a part of the Sharpey fibers[53] that is subsequently incorporated into the acellular cementum layer?

Administration of corticosteroids in doses of 15 mg/kg to rats during orthodontic treatment increases root resorption,[53] whereas low doses of one mg/kg decrease root resorption.[54]

Alcohol consumption in adults during orthodontic treatment tends to increase root resorption through vitamin D hydroxylation in the

liver.[39]

General considerations

The causal relationship between force application and root resorption has never been fully answered. Is it a local mechanism that actually expands the root surface area, thereby decreasing the level of actual pressure? Is it an essential mechanism that maintains the width or subsistence area of the periodontal membrane and its different components? Or is it a side effect of the inflammatory process? Is it a reaction to loss of integrity of the cementum layer (including cracks)? Or is it a part of the normal sequence of root adaptation over years?

The last question has been addressed in several publications. Bishara et al,[55] performed an extensive radiographic survey and found no systematic root shortening between early and mid adulthood. Harris et al,[56] however, reported that resorption was present in about 10% of teeth that had not been orthodontically treated, and 1%–2% demonstrated severe resorption, mostly in the upper incisors. Recent publications include an anecdotal description of idiopathic root resorption[57] and two cases of long-term stability with severely resorbed roots after orthodontic treatment.[58,59]

Looking ahead

The previous view that the immune system is linked to OIIRR has been abandoned in the last 10 years. What about genetic causation? Thus far, we have found that only one group has used polymerase chain reaction analysis to analyze two mRNA-encoded collagenolytic enzymes, matrix metalloproteinase-1 (MMP-1) and cathepsin K, in root resorbing tissue.[60] This research offers a new direction for root resorption research, which might be cultivated extensively in the years ahead.

SUMMARY Return to TOC

In this part of the review, we have continued our previously published work.[1] We have reviewed publications covering the progress in our understanding of the cellular process of root resorption and the effects of pharmacologic agents on this process. Unfortunately, the ultimate predictive factors that might prevent OIIRR remain unknown. Still, several clinical measures may be considered, and these measures are presented and discussed in Part II of this publication.

REFERENCES Return to TOC

1. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: Part 1. Literature review. *Am J Orthod Dentofac Orthop.* 1993; 103:62–66. [PubMed Citation]

2. Wehrbein H, Fuhrmann RA, Diedrich PR. Human histologic tissue response after long-term orthodontic tooth movement. *Am Orthod Dentofac Orthop.* 1995; 107:360–371. [PubMed Citation]

3. Wehrbein H, Bauer W, Diedrich PR. Gingival invagination area after space closure: a histologic study. *Am J Orthod Dentofac Orthop.* 1995; 108:593–598. [PubMed Citation]

4. *Stedman's Medical Dictionary.* 24th ed. Baltimore, Md: Williams & Wilkins Publisher; 1982.

5. Bosshardt DD, Masseredjian V, Nanci A. Root resorption and tissue repair in orthodontically treated human premolars. In: Davidovitch Z, Mah J, eds. *Biological Mechanisms of Tooth Eruption, Resorption and Replacement by Implants.* Boston, Mass: Harvard Society for the Advancement of Orthodontics; 1998:425–437.

6. Roberts WE, Turley PK, Brezniak N, Fiedler PJ. Bone physiology and metabolism. *California Dent Assoc J.* 1987;15:54–61.

7. Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the periodontal ligament. *Eur J Orthod.* 1993; 15:249–263. [PubMed Citation]

8. Brudvik P, Rygh P. Non-clast cells start orthodontic root resorption in the periphery of hyalinized zones. *Eur J Orthod.* 1993; 15:467–480. [PubMed Citation]

9. Brudvik P, Rygh P. Root resorption beneath the main hyalinized zone. *Eur J Orthod.* 1994; 16:249–263. [PubMed Citation]

10. Brudvik P, Rygh P. Multi-nucleated cells remove the main hyalinized tissue and start resorption of adjacent root surfaces. *Eur J Orthod.* 1994; 16:265–273. [PubMed Citation]

11. Brudvik P, Rygh P. Transition and determinants of orthodontic root resorption-repair sequence. *Eur J Orthod.* 1995; 17:177–188. [PubMed Citation]

12. Brudvik P, Rygh P. The repair of orthodontic root resorption: an ultrastructural study. *Eur J Orthod.* 1995; 17:189–198. [PubMed Citation]

13. Hellsing E, Hammarstrom L. The hyaline zone and associated root surface changes in experimental orthodontics in rats: a light and scanning electron microscope study. *Eur J Orthod.* 1996; 18:11–18. [PubMed Citation]

14. Owman-Moll P. Orthodontic tooth movement and root resorption with special reference to force magnitude and duration. A clinical and histological investigation in adolescents. Review. *Swed Dent J.* 1995; 105: (suppl). 1–45.

15. Owman-Moll P, Kurol J, Lundgren D. Continuous versus interrupted continuous orthodontic force related to early tooth movement and root resorption. *Angle Orthod.* 1995; 65:395–401. [PubMed Citation]

16. Owman-Moll P, Kurol J, Lundgren D. Repair of orthodontically induced root resorption in adolescents. *Angle Orthod.* 1995; 65:403–408. [PubMed Citation]

17. Owman-Moll P, Kurol J. Root Resorption pattern during orthodontic tooth movement in adolescents. In: Davidovitch Z, Mah J, eds. *Biological Mechanisms of Tooth Eruption, Resorption and Replacement by Implants.* Boston, Mass: Harvard Society for the Advancement of Orthodontics; 1998:415–424.

18. Kurol J, Franke P, Lundgren D, Owman-Moll P. Force magnitude applied by orthodontists. An inter- and intra-individual study. *Eur J Orthod.* 1996; 18:69–75. [PubMed Citation]

19. Owman-Moll P, Kurol J, Lundgren D. Effects of a doubled orthodontic force magnitude on tooth movement and root resorptions. An inter-individual study in adolescents. *Eur J Orthod.* 1996; 18:141–150. [PubMed Citation]

20. Owman-Moll P, Kurol J, Lundgren D. The effects of a four-fold increased orthodontic force magnitude on tooth movement and root resorptions. An intra-individual study in adolescents. *Eur J Orthod.* 1996; 18:287–294. [PubMed Citation]

21. Kurol J, Owman-Moll P, Lundgren D. Time-related root resorption after application of a controlled continuous orthodontic force. *Am J Orthod Dentofac Orthop.* 1996; 110:303–310. [PubMed Citation]

22. Kurol J, Owman-Moll P. Hyalinization and root resorption during early orthodontic tooth movement in adolescents. *Angle Orthod.* 1998; 68:161–165. [PubMed Citation]

23. Owman-Moll P, Kurol J. The early reparative process of orthodontically induced root resorption in adolescents—location and type of tissue. *Eur J Orthod.* 1998; 20:727–732. [PubMed Citation]

24. Hughes B, King GJ. Effect of orthodontic appliance reactivation during the period of peak expansion in the osteoclast population. *Anat Rec.* 1998; 251:80–86. [PubMed Citation]

25. Zhou D, Hughes B, King GJ. Histomorphometric and biochemical study of osteoclasts at orthodontic compression sites in the rat during indomethacin inhibition. *Arch Oral Biol.* 1997; 42:717–726. [PubMed Citation]

26. King GJ. Effect of timing of orthodontic appliance reactivation on osteoclast and root resorption. In: Davidovitch Z, Mah J, eds. *Biological Mechanisms of Tooth Eruption, Resorption and Replacement by Implants.* Boston, Mass: Harvard Society for the Advancement of Orthodontics; 1998:451458.

27. King GJ, Archer L, Zhou D. Later orthodontic appliance reactivation stimulates immediate appearance of osteoclasts and linear tooth movement. *Am J Orthod Dentofac Orthop.* 1998; 114:692–697. [PubMed Citation]

28. Gu G, Lemery SA, King GJ. Effect of appliance reactivation after decay of initial activation on osteoclasts, tooth movement, and root resorption. *Angle Orthod.* 1999; 69:515–522. [PubMed Citation]

29. Lindskog S, Blomlof L, Hammarstrom L. Dentin resorption in replanted monkey incisors. Morphology of dentinoclast spreading in vivo. *J Clin Periodontol.* 1988; 15:365–370. [PubMed Citation]

30. Sismanidou C, Hilliges M, Lindskog S. Healing of the root surface-associated periodontium: an immunohistochemical study of orthodontic root resorption in man. *Eur J Orthod.* 1996; 18:435–444. [PubMed Citation]

31. Lasfargues JJ, Saffar JL. Inhibition of prostanoid synthesis depresses alveolar bone resorption but enhances root resorption in the rat. *Anat Rec.* 1993; 237:458–465. [PubMed Citation]

32. Roberts WE. Bone physiology, metabolism, and biomechanics in orthodontic practice. In: Graber TM, Vanarsdall RL, eds. *Orthodontics: Current Principles and Techniques.* St Louis, Mo: Mosby; 2000:231–234.

33. Emslie RD. Some considerations on the role of cementum in periodontal disease. *J Clin Periodontol.* 1978; 5:1–12. [PubMed Citation]

34. Mirabella AD, Artun J. Prevalence and severity of apical root resorption of maxillary anterior teeth in adult orthodontic patients. *Eur J Orthod.* 1995; 17:93–99. [PubMed Citation]

35. Rygh P. Orthodontic root resorption studied by electron microscopy. *Angle Orthod.* 1977; 47:1–16. [PubMed Citation]

36. Bosshardt DD, Schroeder HE. How repair cementum becomes attached to the resorbed roots of human permanent teeth. *Acta Anat (Basel).* 1994; 150:253–266. [PubMed Citation]

37. BarKana I, Narayanan AS, Grosskop A, Savion N, Pitaru S. Cementum attachment protein enriches putative cementoblastic populations on root surfaces in vitro. *J Dent Res.* 2000; 79:1482–1488. [PubMed Citation]

38. Metzger Z, Weinstock B, Dotan M, Narayanan AS, Pitaru S. Differential chemotactic effect of cementum attachment protein on periodontal cells. *J Periodont Res.* 1998; 33:126–129. [PubMed Citation]

39. Ghafari JG. Emerging paradigms in orthodontics—an essay. *Am J Orthod Dentofac Orthop.* 1997; 111:573–580. [PubMed Citation]

40. Loberg EL, Engstrom C. Thyroid administration to reduce root resorption. *Angle Orthod.* 1994; 64:395–399. [PubMed Citation]

41. Poumpros E, Loberg E, Engstrom C. Thyroid function and root resorption. *Angle Orthod.* 1994; 64:389–393. [PubMed Citation]

42. Christiansen RL. Thyroxine administration and it effect on root resorption [commentary]. *Angle Orthod.* 1994; 64:399–400.

43. Rossi M, Whitcomb S, Lindemann R. Interleukin-1 beta and tumor necrosis factor-alpha production by human monocytes cultured with L-thyroxine and thyrocalcitonin: relation to severe root shortening. *Am J Orthod Dentofac Orthop.* 1996; 110:399–404. [PubMed Citation]

44. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. *J Clin Pediatr Dent.* 1999; 23:259–264. [PubMed Citation]

45. Leiker BJ, Nanda RS, Currier GF, Howes RI, Sinha PK. The effects of exogenous prostaglandins on orthodontic tooth movement in rats. *Am J Orthod Dentofac Orthop.* 1995; 108:380–388. [PubMed Citation]

46. Boekenoogen DI, Sinha PK, Nanda RS, Ghosh J, Currier GF, Howes RI. The effects of exogenous prostaglandin E2 on root resorption in rats. *Am J Orthod Dentofac Orthop.* 1996; 109:277–286. [PubMed Citation]

47. Igarashi K, Adachi H, Mitani H, Shinoda H. Inhibitory effect of the topical administration of a bisphosphonate (risedronate) on root resorption incident to orthodontic tooth movement in rats. *J Dent Res.* 1996; 75:1644–1649. [PubMed Citation]

48. Adachi H, Igarashi K, Mitani H, Shinoda H. Effects of topical administration of a bisphosphonate (risedronate) on orthodontic tooth movements in rats. *J Dent Res.* 1994; 73:1478–1486. [PubMed Citation]

49. Igarashi K, Mitani H, Adachi H, Shinoda H. Anchorage and retentive effects of a bisphosphonate (AHBuBP) on tooth movements in rats. *Am J Orthod Dentofac Orthop.* 1994; 106:279–289. [PubMed Citation]

50. Alatli I, Hellsing E, Hammarstrom L. Orthodontically induced root resorption in rat molars after 1-hydroxyethylidene-1,1-bisphosphonate injection. *Acta Odontol Scand.* 1996; 54:102–108. [PubMed Citation]

51. Alatli I, Hammarstrom L. Root surface defects in rat molar induced by 1-hydroxyethylidene-1,1-bisphosphonate. *Acta Odontol Scand.* 1996; 54:59–65. [PubMed Citation]

52. Strocchi R, Raspanti M, Ruggeri A, Franchi M, De Pasquale V, Stringa L, Ruggeri A. Intertwined Sharpey fibers in human acellular cementum. *Ital J Anat Embryol.* 1999; 104:175–183.

53. Ashcraft MB, Southard KA, Tolley EA. The effect of corticosteroid-induced osteoporosis on orthodontic tooth movement. *Am J Orthod Dentofac Orthop.* 1992; 102:310–319. [PubMed Citation]

54. Ong CK, Walsh LJ, Harbrow D, Taverne AA, Symons AL. Orthodontic tooth movement in the prednisolone-treated rat. *Angle Orthod.* 2000; 70:118–125. [PubMed Citation]

55. Bishara SE, Vonwald L, Jakobsen JR. Changes in root length from early to mid-adulthood: resorption or apposition?. *Am J Orthod Dentofac Orthop.* 1999; 115:563–568. [PubMed Citation]

56. Harris EF, Robinson QC, Woods MA. An analysis of causes of apical root resorption in patients not treated orthodontically. *Quintessence Int.* 1993; 24:417–428. [PubMed Citation]

57. Rivera EM, Walton RE. Extensive idiopathic apical root resorption. A case report. *Oral Surg Oral Med Oral Pathol.* 1994; 78:673–677. [PubMed Citation]

58. Parker WS. Root resorption—long-term outcome. *Am J Orthod Dentofac Orthop.* 1997; 112:119–123. [PubMed Citation]

59. Desai HM. Root resorption: another long-term outcome. *Am J Orthod Dentofac Orthop.* 1999; 116:184–186. [PubMed Citation]

60. Domon S, Shimokawa H, Matsumoto Y, Yamaguchi S, Soma K. In situ hybridization for matrix metalloproteinase-1 and cathepsin K in rat root-resorbing tissue induced by tooth movement. *Arch Oral Biol.* 1999; 44:907–915. [PubMed Citation]

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