Editorial

Defining and framing orthodontitis: A new term in orthodontics

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It is well known that orthodontic force application induces aseptic local inflammation due to necrosis in the periodontal ligament (PDL) and that there is no tooth movement without this inflammation process. It is also well known that the inflammation process is important for both the bone as well as the cementum remodeling processes.[1–5] If we consider these well-known phenomena together, we can name the process related to orthodontic tooth movement, as well as the Inflammatory Root Resorption Concurrent with Orthodontics (IRRCWO),[6] as ''Orthodontitis''. Orthodontitis, then, is the inflammation behind tooth movement, where the prefix is our profession and the suffix ''itis''[7] is used in medical terminology to describe inflammation of an organ or a tissue. The combination, Orthodontitis, is therefore a term we would like to introduce along with a classification system to describe the inflammation resulting from orthodontic force application.

Orthodontitis is an aseptic local inflammation in the PDL induced by orthodontic forces. Orthodontitis can be divided into 2 groups: Instrumental Orthodontitis (IO) and Instrumental-Detrimental Orthodontitis (IDO):

Instrumental Orthodontitis (IO): IO initiates controlled bone modeling,[8] as well as bone and cemental remodeling (reversible changes).[8,9] IO enables tooth movement to occur due to frontal and undermining alveolar bone resorption and apposition on the pressure and tension sides, respectively.[10] The roots next to IO areas also undergo surface resorption,[11] mainly by cemental remodeling. This biological process ceases when orthodontic forces are removed. The periodontal ligament that surrounds the roots is fully regenerated. IO symptoms include mild to moderate tooth mobility and/or sensitivity, and pain during the first days following force application. IO signs include mild to moderate tooth mobility and radiographic PDL widening. Signs and symptoms disappear following orthodontic force cessation.

The mechanism behind the process is that orthodontic force enables normal blood flow, but induces local electrical current and pH changes as well as release of different biological materials from the damaged cells (e.g. cytokines and prostaglandins). These events trigger local inflammatory activity in the area surrounding the roots and is limited to the PDL, alveolar bone and cementum. The inflammation in the pressure area induces mainly a bone modeling process by resorbing the alveolar bone while the inflammation in the tension area induces bone modeling by apposition; new bone is being laid down on the affected surfaces. Surface cemental remodeling is induced in both areas as well.

The inflammation mechanism is genetically controlled. It is activated regularly during our lifetime and it remains behind the normal remodeling/modeling process.

Regarding treatment, analgesics are sometimes prescribed during orthodontic treatment. No further action is needed.

Instrumental and Detrimental Orthodontitis Grade 1 (IDO1): IDO1 is similar to IO. However, the inflammation in IDO1, for unknown reasons, changes its character on the cemental side (the effect on the bone on the pressure and tension sides is similar to that of IO) and the remodeling process becomes a modeling process; the resorption process goes beyond the cementum into the dentin. IDO1 causes minor to moderate root shortening[12] as well as scattered lacunae on other root surfaces. Both of these are irreversible changes. The results are usually diagnosed using X-rays during, close to the end, or following orthodontic treatment. The symptoms and treatment are similar to IO. After orthodontic treatment is completed, there are only radiographic signs (root shortening or peripheral surface resorption) but no symptoms.

Instrumental and Detrimental Orthodontitis Grade 2 (IDO2): IDO2 is very similar to IDO1. However, in this case, the inflammation results in severe root shortening. The symptoms are tooth mobility and sensitivity during or following orthodontic treatment. The signs include tooth mobility/sensitivity and severe

root shortening[12] as viewed on X-rays. The consequences of IDO2 require treatment. The treatment for IDO2 depends on the time that it is discovered. If IDO2 is diagnosed after debonding, we suggest that fixed retention be used to splint the affected teeth together with unaffected teeth. In rare situations, fused crowns can be a good treatment solution. Extractions and implant replacements should be considered only in extremely rare cases, if ever.

The mechanism for both IDO1 and IDO2 is similar to that described for IO. However, the level of the resorptive activity on the root surface is different, and it is probably individually genetically determined.[13–16] The remodeling process is being disturbed in the transition between the resorption and the reversal stages.[8,9] The coupling between resorption and apposition disappears or is delayed and, therefore, resorption continues into the dentin.

Furthermore, we suggest that IDO should be regarded as a self-defense mechanism of the body to an extreme local condition (similar to the body's reaction or behavior in hypovolemic shock)[17] which occurs in the PDL following force application. By shortening the roots, the body decreases the current or future moments developed in the apical areas of the teeth due to the orthodontic force being applied at the crown. In addition, we suggest that the irreversible lacunar resorption along the roots' surfaces is a mechanism that may defend against the loss of teeth with resorbed short roots by increasing their surface areas, thereby keeping the damaged teeth in a stable condition. It has been demonstrated that those teeth can remain in the mouth for many years.[18,19]

We believe that the profession should adopt this new terminology and use it to explain the actual process underlying tooth movement and IRRCWO during consultation with the parents/patients/guardians. If patients and parents can understand this process better, the number of lawsuits against orthodontists in this matter would decrease tremendously. IRRCWO can be considered one of the body's self-defense mechanisms. Even today, it has not been determined conclusively the level of force and/or the duration of force application at which this reaction will be expressed.

REFERENCES

1. Bletsa A, Berggreen E, Brudvik P. Interleukin-1alpha and tumor necrosis factor-alpha expression during the early phases of orthodontic tooth movement in rats. *Eur J Oral Sci*. 2006;114:423–429.
2. Garlet TP, Coelho U, Silva JS, Garlet GP. Cytokine expression pattern in compression and tension sides of the periodontal ligament during orthodontic tooth movement in humans. *Eur J Oral Sci*. 2007;115:355–362.
3. Tzannetou S, Efstratiadis S, Nicolay O, Grbic J, Lamster I. Comparison of levels of inflammatory mediators IL-1beta and betaG in gingival crevicular fluid from molars, premolars, and incisors during rapid palatal expansion. *Am J Orthod Dentofacial Orthop*. 2008;133:699–707.
4. Surlin P, Rauten AM, Silosi I, Foia L. Pentraxin-3 levels in gingival crevicular fluid during orthodontic tooth movement in young and adult patients. *Angle Orthod*. 2012;82: 833–838.
5. Kim SJ, Park KH, Park YG, Lee SW, Kang YG. Compressive stress induced the up-regulation of M-CSF, RANKL, TNF-α expression and the down-regulation of OPG expression in PDL cells via the integrin-FAK pathway. *Arch Oral Biol*. 2013;58:707–716.
6. Brezniak N, Wasserstein A. Orthodontically induced inflammatory root resorption. Part 1: The basic science aspects. *Angle Orthod*. 2002;72:175–179.
7. http://www.merriam-webster.com/medical/itis. Accessed November 15, 2013.
8. Roberts WE. *Orthodontics Current Principles and Techniques*. [5th ed.] Vanarsdall RL Jr, Vig KWJ, Graber LW. St Louis: Mosby; 2012:386–453.
9. Viecilli RF, Katona TR, Chen J, Hartsfield JK Jr, Roberts WE. Orthodontic mechanotransduction and the role of the P2X7 receptor. *Am J Orthod Dentofacial Orthop*. 2009;135: 694.e1–e16.
10. 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.
11. Andreasen JO. Review of root resorption systems and models. Etiology of root resorption and the homeostatic mechanisms of the periodontal ligament. In: Davidovitch Z, ed. *Biological Mechanisms of Tooth Eruption and Root Resorption*. Birmingham, Alabama: EBSCO Media; 1988: 9–22.
12. Levander E, Malmgren O. Evaluation of the risk of root resorption during orthodontic treatment: a study of upper incisors. *Eur J Orthod*. 1988;10:30–38.
13. Al-Qawasami RA, Hartsfield JK Jr, Everette ET, Flury L, Liu L, Foroud TM, Marci Jv, Roberts WE. Genetic predisposition to external apical root resorption. *Am J Orthod Dentofacial Orthop*. 2003;123:242–252.
14. Low E, Zoellner H, Kharbanda OP, Darendeliler MA. Expression of mRNA for osteoprotegerin and receptor activator of nuclear factor kappa beta ligand (RANKL) during root resorption induced by the application of heavy orthodontic forces on rat molars. *Am J Orthod Dentofacial Orthop*. 2005;128:497–503.
15. Bastos Lages EM, Drummond AF, Pretti H, et al. Association of functional gene polymorphism IL-1beta in patients with external apical root resorption. *Am J Orthod Dentofacial Orthop*. 2009;136:542–546.
16. Iglesias-Linares A, Yañez-Vico R, Ballesta-Mudarra S, et al. Postorthodontic external root resorption is associated with IL1 receptor antagonist gene variations. *Oral Dis*. 2012;18: 198–205.
17. Rhee P. Shock, electrolytes and fluid. In: *Sabiston Textbook of Surgery*. 19th ed. Philadelphia: Elsevier; 2012:66–119.
18. Becker A, Chaushu S. Long-term follow-up of severely resorbed maxillary incisors after resolution of an etiologically associated impacted canine. *Am J Orthod Dentofacial Orthop*. 2005;127:650–654.
19. Marques LS, Chaves KC, Rey AC, Pereira LJ, Ruellas AC. Severe root resorption and orthodontic treatment: clinical implications after 25 years of follow-up. *Am J Orthod Dentofacial Orthop*. 2011;139(4 Suppl):S166–S169. doi: 10.1016/j.ajodo.2009.05.032.