The role of biology in the orthodontic practice (Part 2)

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The goal of this article is to enhance the biological awareness of the orthodontic practitioner in order to minimise or avoid tissue damage during orthodontic treatment. In this second part, the aetiology of orthodontic root resorption will be examined, as well as the identification of high-risk subjects and treatment of this condition.

Ketcham first reported on severe root resorption associated with orthodontic treatment in 1927. Subsequent reports attributed orthodontic root resorption to hormonal influences, nutritional status, genetic predisposition, dental root morphology, and the treatment-related factors, such as force magnitude, duration and direction. A comparison of the rates of tooth movement and the degree of root resorption between human premolars that had been moved buccally for 12 weeks by either a stainless steel arch with exerted dissipating forces, or by a superelastic wire that applied constant forces, revealed that the latter wires had moved the teeth faster, but at a cost of significantly greater amounts of root resorption (Weiland 2005).

The most susceptible teeth for orthodontic root resorption are the maxillary incisors, probably because these teeth are usually moved the longest distances during the course of treatment. In a number of current orthodontic techniques, much of this incisor root movement is redundant. As discussed in more detail below, maxillary incisor root resorption is frequently associated with uncontrolled tipping of the incisors, where the crown moves palatally, while the root apex moves labially, into contact with and even through the palatal plate of compact alveolar bone. This movement is then followed by torquing of the incisor roots, in order to return them to proper angulation and position. In many such cases, about 90% of the root movement is unwarranted.

Although root resorption can occur on any surface of the root, it is most frequently observed radiographically in the apical region, where both cementum and dentine have been removed irreversibly. Severe root resorption, which accounts for the loss of at least 25% of the root length, occurs in 1 to 3% of the orthodontic patient population. Despite this excessive loss, the affected teeth’s longevity is usually not jeopardised, provided the resorptive process is halted when orthodontic treatment ceases, and when periodontal health is good, with maintenance of the height of the alveolar crest.

Cells participating in the remodelling of dental and periodontal tissues during tooth movement are derived from the native cell population of these tissues, from the circulation (platelets and leukocytes), and from alveolar bone marrow cavities. These cavities are connected with the periodontal ligament (PDL) by intra-bony channels that provide passageways for marrow cells with osteogenic potential (McCollough et al. 1987). In human patients, peri-apical radiographs have revealed that the largest marrow cavities are situated around the apical region of the root, thus potentially providing a plethora of marrow-derived osteoclasts to the PDL, in that area, increasing the probability of root resorption, in comparison with the coronal section of the root.

Since incisors, particularly maxillary, are the teeth most susceptible to root resorption, obtaining peri-apical radiographs of these teeth every six months is recommended, at least once a year. This routine may assist the clinician in identifying individuals who display early signs of resorption, and in modifying their treatment plan to minimise the risk of severe resorption. On rare occasions, treatment must be stopped altogether, at least for a few months, to give the tissues time to rest.

A comparison between patients with a Class II Division I malocclusion, with an overjet smaller than 7 mm, who had been subjected to either a one- or a two-phase orthodontic treatment with fixed appliances revealed that the proportion of maxillary incisors with moderate to severe resorption was slightly higher in the one-phase treatment group (Brin et al. 2001). There was only a slight increase in frequency of root resorption in teeth with irregular root morphology. However, significant associations were found to exist between root resorption, the magnitude of overjet reduction, and the duration of treatment.

The question arises of whether all orthodontic techniques are responsible for causing equal frequencies and extents of root resorption. The comparison of peri-apical radiographs by a number of research teams has revealed differences in this regard between certain methods of tooth movement. In one study, patients treated with...
of the prerequisites for avoiding orthodontic root resorption is the use of rectangular wires with a diameter smaller than that of the bracket, thereby applying light torque forces to the teeth.

With regard to the incidence of orthodontic root resorption, it might be interesting to know which teeth are more susceptible, those with fully formed roots or those with immature, short roots. Measurements of maxillary incisors’ root length before and after treatment for correction of a Class II/1 malocclusion (Marvagani et al. 2002) revealed that the immature teeth continued to grow and elongate during treatment, while the mature teeth were shortened by resorption.

A similar question to the one whether there is a specific gene or combination of genes whose activation will precipitate orthodontic root resorption has been asked regarding the identity of genes responsible for the development of orofacial clefts (Perucchini 2002). Here, there is a complex mode of inheritance with the possible involvement of two to 29 genes. Transgenic mice lacking transforming growth factor (TGF-β3) and cleft lip from the city of Columbia. No mutations were identified in the coding region of TGF-β3. However, a polymorphic variant was found in the upstream regulatory that may alter the gene’s function.

A similar search was performed in families whose members had orthodontic root resorption, with 15% of maxillary incisor root resorption apparently dependent on this association. Furthermore, individuals homozygous for the IL-1β allele B were found to have a 5.6-fold increased risk of resorption of less than 2 mm compared with those not homozygous for this gene.

It is concluded that orthodontic root resorption may be caused by a number of contributing elements acting separately or in combination. Most prominent factors are faulty mechanics, systemic diseases, and modifications in specific genes. The main tool at the disposal of the orthodontist to avoid causing root resorption is the exclusion of unwarranted tooth movements during the entire course of orthodontic treatment. Patients treated in this way require relatively short treatment completion times. The use of appliances that generate forces that do not injure the PDL also reduces the risk of root resorption. Hence, these conclusions imply that avoidance of root resorption is primarily in the hands of the orthodontist.

The biological nature of an optimal orthodontic force

The discussion earlier in this article has revealed that tissue remodelling that facilitates orthodontic tooth movement is performed by various cell types. Some of these cells are local, such as fibroblasts and bone surface lining cells; other cells are migratory, like macrophages and lymphocytes, but evidently play a crucial role in modulating the effect of mechanical forces on para-dental cells. Thus, an optimal orthodontic force is capable of evoking an inflammatory response in para-dental tissues, leading to remodelling of these tissues and tooth movement in a desirable direction.

In an effort to provide a rationale for the use of magnets in orthodontics, in 1998 Blechman proposed that static magnets generate electromagnetic fields that stimulate bone formation in PDL tension sites, thereby reducing tooth mobility, pain and discomfort. He stated that in routine orthodontic treatment, bone formation lags behind resorption, causing widening of the periodontal space and increased tooth mobility. Re-examination of histological sections of cat jaws after seven and 14 days of combined orthodontic force/electric stimulation (Davidovich et al. 1990) supported Blechman’s proposition that exogenous electric signals increase the amount of new bone formation in PDL tension sites. These observations suggested that an optimal orthodontic force is one accompanied by an additional signal, such as an electric current, which accelerates the rate of alveolar bone formation.

Experiments with avian long bones in vivo, in search of the features of an optimal force for evoking osteogenic reactions, Lanyon and Rubin (1984) observed that the most efficient force was dynamic (intermittent) rather than static (continuous).

A short duration of between five and ten minutes a day was adequate to stimulate potent periosteal and endosteal osteogenic reactions. The force magnitude was found to be of importance, defined as optimal being in the range of 2,000 to 4,000 microstrains. However, this magnitude could be much lower, provided the frequency of force application was increased. The target cells in these experiments are those responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in the osteoblasts, and the molecules responsible for bone’s “strain memory” and involved in

...inflammation is an integral part of the tissue response to orthodontic forces.
The most significant situation associated with root resorption in the maxillary arch was the approximation of incisor apices against the lingual or labial plates of bone. In such cases, the likelihood of severe root resorption increased by 20 times. Moreover, the degree of resorption correlated with the overall length of treatment time and the duration of full engagement of rectangular wires in the brackets. In a study of dental radiographs of 42 patients treated by the Begg technique (Goldson & Henrikson 1976), all subjects developed root resorption at the completion of treatment. Furthermore, a laminographic study of patients treated by this method revealed that the resorptive damage to the maxillary incisors is associated with uncontrolled tipping and subsequent round tripping of the teeth (Ten Hoeve & Mulier 1976). A rare opportunity to examine histologically the maxilla of a patient in the midst of treatment with a Straight Wire appliance was granted to three orthodontists (Wherbein et al. 1995). The jaw was obtained from a 19-year-old patient who had been killed in a traffic accident. At the time of death, she had completed 19 months of treatment, which included movement of molar roots into the vestibular and palatine cortical plates of bone. These authors report finding widespread dehiscences and fenestrations of the alveolar bone cortical plates, as well as extensive lateral, buccal and palatine root resorption. These destructive changes were particularly pronounced in teeth that had been subjected to uncontrolled tipping, and less severe in teeth moved by translation. These histological findings could not have been diagnosed by macroscopic inspection of the specimen.

Radiographic cephalometry and computed tomography were utilized in studying the maxillary and mandibular alveolar bone plates following incisor retraction (by controlled tipping) in patients with hi-maxillary protraction (Sarikaya et al. 2002). A comparison of pre- and post-retraction records revealed that in both jaws there had been significant reductions in the width of the lingual bone as a result of treatment, with some patients demonstrating dehiscences that were not visible macroscopically or cephalometrically. It was concluded that forcing dental roots against cortical bone may cause adverse sequelae. Concerned about the extent of tissue damage encountered by his patients who had been treated with the Edgewise and Begg appliances, De Angelis (2005) concluded that this damage, particularly root resorption, is associated with the overall length of treatment time and treatment with rectangular wires that fully engage the Edgewise bracket slot, amongst other factors. The jiggling of teeth in the sagittal, vertical and transversal planes, as well as the round tripping or uncontrolled tipping of teeth was also identified as contributing to this condition. In addition, root resorption can be caused by bringing dental roots into contact with the cortical plates of alveolar bone.

Conclusion
It may be concluded that an optimal orthodontic force is one that is applied with full attention to the anatomical constraints and peculiarities of every individual patient. Therefore, orthodontic treatment plans must focus on the desired changes in dental root position, rather than on adherence to some “universal” system of mechanotherapy as a solve-all approach. Issues such as force magnitude, duration and direction must be considered individually for each patient, with the clear understanding that anatomical constraints should not be violated or ignored during the correction of a malocclusion. When potentially damaging movements of dental roots are avoided, orthodontic forces may be considered biologically and clinically optimal. From this point of view, the amalgamated technique appears presently as the closest to being defined as an optimal mechanotherapeutic system biologically, as well as clinically. A complete list of references is available from the publisher.