The role of biology in the orthodontic practice

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Orthodontic tooth movement results from forces applied to teeth that evoke cellular responses in the teeth and their surrounding tissues, including the periodontal ligament (PDL), alveolar bone and gingiva. It is advantageous for the orthodontist to know the details of the biological events that unfold during tooth movement because some of these details may differ from one person to another, owing to variables such as gender, age, psychological status, nutritional habits and drug consumption.

The purpose of this article is to emphasise that orthodontics is a field of endeavour in which mechanics and biology are integrated, and to stress the reality that tooth movement is conducted in individual human beings, each with a unique and intricate physiological system. Biological variations may be the foundation of the differences that are frequently observed in the outcomes of orthodontic treatment between patients with similar malocclusions treated identically.

Principles of orthodontic biomechanics are usually taught with the help of a typodont, consisting of artificial teeth embedded in wax. This set-up ignores entirely the biological aspect of tooth movement. In the clinical setting, living patients are encountered, and mechanical forces mobilise their teeth. These movements result from the development of strains in dental and para-dental tissues, followed by modelling and remodelling of these tissues.

In some patients, systemic conditions may exist, causing complications such as root resorption, dehiscences and fenestrations of the alveolar bone. Hence, clinical orthodontics must be viewed as a specialty staunchly entrenched in biology, all the way to the molecular level. As a clinical profession, it must be based on a commanding knowledge of mechanisms, biology, physiology, and pathology. 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. It will demonstrate that this objective may be achieved by closely focusing on the nature of root movements, and avoiding the dogmatic following of "prescription" methods that promise "automatic" correction of all malocclusions.

Tissue remodelling and orthodontic tooth movement
The actual rate of tooth movement may depend on the rate of bone turnover. The latter was modified pharmacologically in rats undergoing maxillary molar mesial movement, by inducing either hypocalcaemia or hyperthyroidism (Verna et al., 2000). In rats with high bone turnover, the rate of tooth movement was increased, whereas it was reduced in animals with a low turnover. Although all teeth had been moved in the same direction (controlled by tooth tipping), the location of the centre of rotation differed, depending on the nature of the force applied. Examination of histological sections from the jaws of these rats (Verna et al., 2005) showed that root resorption had occurred in both groups, as well as in the control group, but that it was more pronounced in the low bone turnover group. However, bone metabolism normally demonstrates measurable diurnal fluctuations that may affect the rate of tooth movement. Rats that were exposed to light for 24 or 12 hours per day for 21 days, and were subjected to orthodontic force only during the light period, presented doubling of the rate of tooth movement and bone remodelling, as compared with animals that received the force during the 12 hours of darkness (Miyoshi et al., 2001).

The realisation that tissue remodelling in orthodontics is mediated by a variety of cells, including fibroblasts, root and bone surface lining cells, endothelial, epithelial, and nerve cells, as well as different leukocytes, prompted clinical investigators to apply physical and chemical agents, concomitant with orthodontic forces in order to augment the effect of the mechanical forces. In this vein, Tweedle (1965) used local application of heat to para-dental tissues surrounding orthodontically treated teeth in dogs, Davidovitch et al. (1980) used minute electric currents, and Blechman (1988) advocated the use of static magnetic fields.

Davidovitch et al. placed the electrodes much closer to the cat’s ca- nine, resulting in a significant enhancement of movement. Blechman hypothesised that magnets generate mechanical forces, as well as magnetic fields, and that this combined effect surgically causes the teeth to move faster. However, an experiment in rats (Tenuku et al., 2000) revealed that magnets do not speed up the mesial movement of maxillary molars, and actually increase root resorption in the early phases of treatment.

Utilisation of chemical agents in attempts to increase the pace of tissue remodelling and tooth movement has been tested in various laboratories and clinics. Ya- masaki et al. (1984) injected prostaglandin (PG) E1 into the gingiva of moving teeth in human subjects, resulting in rapid movement. Systemic application of misoprostol, a PGE1 analogue, to rats undergoing tooth movement for two weeks increased the rate of movement significantly with the help of mechanical forces, and was most effective when applied for brief periods rather than continuously. This assumption was found to be correct in an experiment in rats by Gibson, King, and Keeling (1992). In that experiment, maxillary molars were subjected to mesially moving forces for one hour, one day, or 14 days. Treatment with one hour of force application continued to move mesially for 14 days, and achieved 35% of the movement reached by the teeth that had been subjected to orthodontic forces continuously for 14 days.

The age factor
The effect of age on the tissue response to orthodontic force has occupied the minds of orthodont-
tists since Hunter, in the 18th century, and probably earlier. Hunter observed that orthodontic treatment increases gingival erythema in children. Studying histological sections of human teeth and their surrounding tissues, Brittan concluded that the PDL is less cellular in adults than in children. Therefore, he recommended, when treating adults, to subject their teeth to light forces initially, in order to stimulate cellular proliferation, then to increase the force magnitude, in order to stimulate these cells to remodel the para-dental tissues. This observation implies that, in essence, the nature of the biological response to orthodontic forces is similar in young and adult subjects. This hypothesis was confirmed by Shamp et al. (2005). These investigators moved molars bilingually in young (15-week-old) and old (60-week-old) rats, then studied their compensatory alveolar bone apposition under the lingual periosteum. They reported that in both age groups there had been vigorous compensatory alveolar bone growth. Thus, alveolar bone is successfully maintained, even in aged rats. Age also refers to the duration of healing of a post-operative regenerate following distraction osteogenesis (Nakamoto et al., 2002). In an experiment on 15-month-old beagles, mandibular premolars were moved into a two-week or a 12-week regeneration period. The former consisted of immature, fibrous, and poorly mineralised bone, while the latter was composed of mature, well-organised and mineralised bone. Tooth movement was significantly faster in the "young", immature regenerate, but this movement was accompanied by extensive root resorption that extended from the cemento-enamel junction to the root apex.

The effects of pre-existing medical conditions and the development of complications

It is estimated that 10 to 15% of all children under the age of 16 are affected by chronic, long-term medical problems. These problems may affect the outcome of orthodontic treatment (Burden et al., 2001). Common medical problems in this age group include infective endocarditis, bleeding disorders, leukaemia, diabetes, cystic fibrosis, juvenile rheumatoid arthritis and renal failure. An even higher percentage of adult patients may be affected by a variety of medical problems that involve one or more of the tissue systems. These conditions, and the medications used to treat them may have profound effects on the response of dental and para-dental cells to mechanical loading.

Endocarditis is a life-threatening infection that requires primary prevention in the form of administration of antimicrobial agents prior to certain orthodontic procedures. The orthodontist must weigh the risk of endocarditis against the risk of an adverse reaction to the prescribed antibiotic therapy. Fortunately, most orthodontic procedures do not cause bacteremia. Lucas et al. (2002) obtained blood samples from children 50 seconds after taking dental impressions, separator placement, band placement and insertion of an adjusted arch wire. Significant bacteremia was found only after separator placement.

Orthodontic braces, fixed and removable, can accumulate bacterial plaque that may be harmful to soft and hard tissues. This problem has been addressed by adding antimicrobial agents to bracket bonding materials, elastic bands and crown coating varnishes. The addition of benzalkonium chloride to a composite resin added antimicrobial properties to the compound without altering its mechanical properties (Ohbman et al., 2002). Likewise, coating teeth in orthodontic patients with a sustained-release chlorhexidine varnish decreases Streptococcus mutans levels in the patients’ saliva (Beyth et al., 2003).

Children treated for childhood cancers with both radiation and chemotherapy often exhibit disturbances in dental development, such as tooth agenesis, teeth with short roots or with no roots altogether. A retrospective analysis of treatment outcome in ten orthodontic patients with such a background revealed that five had been treated with lighter forces than usual, one displayed root resorption, and four achieved unsatisfactory results (Dahlolf et al., 2001).

The development of inflammation in dental and para-dental tissues during the course of orthodontic treatment implies that circulating plasma and leukocytes migrate out of capillaries, and interact with native cells. The blood plasma may contain endogenous hormones produced by endocrine glands, as well as a variety of molecules derived from consumed drugs and nutrients. Some of these molecules may interact with para-dental target cells, augmenting or inhibiting the effects of mechanical forces on these cells.
One of the main complications of such interactions is the development of root resorption.

Diabetes mellitus affects 3 to 4% of the population, and is characterised by hyperglycaemia caused by the body’s deficient management of insulin. There are two main types of diabetes: type 1 and type 2. In type 1, there is a total deficiency in insulin secretion, while in type 2 there is a combination of resistance to insulin action and insufficient compensatory insulin secretion. Diagnosis and monitoring of diabetes is based on blood glucose concentration or glycosylated haemoglobin concentration.

Periodontitis, acute or chronic, may be present before the onset of orthodontic treatment, or occur during the course of treatment owing to the accumulation of a bacterial plaque around the braces. Peripheral blood monocytes obtained from individuals with chronic periodontitis synthesised large amounts of pro-inflammatory cytokines when incubated in vitro with bacterial lipopolysaccharides. If such primed monocytes find their way into strained parodontal tissues, their increased production levels of cytokines may increase the risk of root resorption. An indicator of such an increased risk may be the concentration of cytokines in the gingival crevicular fluid. Previous studies reported on increased levels of cytokines, such as tumour necrosis factor-α and interleukin-6 in the gingival crevicular fluid of orthodontically treated teeth in humans (Kim and Park, 2000). The origin of these cytokines is most likely PDL cells.

Allergies and asthma are conditions involving periodic productions of large amounts of pro-inflammatory cytokines in the airway mucosa and the skin. Primed leukocytes derived from these tissues may travel through the circulatory system into the extraosseous space of the tissues surrounding orthodontically treated teeth. Consequently, patients with a history of allergies or asthma appear to be at a high risk of developing excessive root resorption during the course of orthodontic treatment (Davidovitch et al., 1999). Hence, it is postulated that any inflammatory condition, such as gastes–enteritis, arthritis and thyroiditis, may increase the risk of orthodontic root resorption.

Allergy manifestations in orthodontics are infrequent, although the frequency of allergic diseases in the industrialised world is rising. The WHO reports that 15% of the population has had or will have an allergic disease. Allergic reactions to orthodontic materials can develop during treatment, manifesting as urticaria, angioedema, stomatitis or cheilitis (Beaudovin et al., 1999). Hence, it is postulated that any inflammatory condition, such as gastes–enteritis, arthritis and thyroiditis, may increase the risk of orthodontic root resorption.

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