ONLINE ONLY

Cellular, molecular, and tissue-level reactions to orthodontic force

Vinod Krishnan[a] and Ze'ev Davidovitch[b]
Tamilnadu, India, and Cleveland, Ohio

Remodeling changes in paradental tissues are considered essential in effecting orthodontic tooth movement. The force-induced tissue strain produces local alterations in vascularity, as well as cellular and extracellular matrix reorganization, leading to the synthesis and release of various neurotransmitters, cytokines, growth factors, colony-stimulating factors, and metabolites of arachidonic acid. Recent research in the biological basis of tooth movement has provided detailed insight into molecular, cellular, and tissue-level reactions to orthodontic forces. Although many studies have been reported in the orthodontic and related scientific literature, a concise convergence of all data is still lacking. Such an amalgamation of the rapidly accumulating scientific information should help orthodontic clinicians and educators understand the biological processes that underlie the phenomenon of tooth movement with mechanics (removable, fixed, or functional appliances). This review aims to achieve this goal and is organized to include all major findings from the beginning of research in the biology of tooth movement. It highlights recent developments in cellular, molecular, tissue, and genetic reactions in response to orthodontic force application. It reviews briefly the processes of bone, periodontal ligament, and gingival remodeling in response to orthodontic force. This review also provides insight into the biological background of various deleterious effects of orthodontic forces. (Am J Orthod Dentofacial Orthop 2006;129:469e.1-460e.32)

Tooth movement by orthodontic force application is characterized by remodeling changes in dental and paradental tissues, including dental pulp, periodontal ligament (PDL), alveolar bone, and gingiva. These tissues, when exposed to varying degrees of magnitude, frequency, and duration of mechanical loading, express extensive macroscopic and microscopic changes. Orthodontic tooth movement differs markedly from physiological dental drift or tooth eruption. The former is uniquely characterized by the abrupt creation of compression and tension regions in the PDL.[1] Physiological tooth movement is a slow process that occurs mainly in the buccal direction into cancellous bone or because of growth into cortical bone. In contrast, orthodontic tooth movement can occur rapidly or slowly, depending on the physical characteristics of the applied force, and the size and biological response of the PDL.[2] These force-induced strains alter the PDL's vascularity and blood flow, resulting in local synthesis and release of various key molecules, such as neurotransmitters, cytokines, growth factors, colony-stimulating factors, and arachidonic acid metabolites. These molecules can evoke many cellular responses by various cell types in and around teeth, providing a favorable microenvironment for tissue deposition or resorption.[3,4]

Studies in the early 20th century attempted mainly to analyze the histological changes in paradental tissues after tooth movement. Those studies showed extensive cellular activities in the mechanically stressed PDL involving fibroblasts, endothelial cells, osteoblasts, osteocytes, and endosteal cells.[5] Apart from this finding, it was discovered that mechanical stresses alter the structural properties of tissues at the cellular, molecular, and genetic levels. Current literature has much data on molecular- and genetic-level cellular responses to orthodontic force. The rapid reactions at the initial stage of mechanotherapy and slower adaptive changes later are well explained in the literature. The following discussion on cellular, molecular, and tissue reactions is intended to provide basic information about histological and chemical changes of orthodontic tooth movement. It tries to update the readers with recent developments in cellular, molecular, tissue, and genetic reactions in response to orthodontic force application along with a brief description on the processes of bone, PDL, and gingival remodeling in response to orthodontic force. This review also provides insight into the

[a]Assistant professor, Department of Orthodontics, Rajas Dental College, Tirunelveli District, Tamilnadu, India.
[b]Clinical professor, Department of Orthodontics, Case Western Reserve University, Cleveland, Ohio.
Reprint requests to: Dr Vinod Krishnan, Gourivilasam, Kudappanakunnu PO, Trivandrum, Kerala State 695043, India; e-mail, vikrishnan@yahoo.com.
Submitted, May 2005; revised and accepted, October 2005.
0889-5406/$32.00

doi:10.1016/j.ajodo.2005.10.007

biological background of various deleterious effects of orthodontic forces.

ORTHODONTIC VERSUS ORTHOPEDIC FORCE

Orthodontic force has been defined as "force applied to teeth for the purpose of effecting tooth movement, generally having a magnitude lower than an orthopedic force," whereas orthopedic force is defined as "force of higher magnitude in relation to an orthodontic force, when delivered via teeth for 12 to 16 hours a day, is supposed to produce a skeletal effect on the maxillofacial complex."[6] These definitions show that there is no clear distinction between orthodontic and orthopedic forces, even in terms of magnitude; furthermore, many widely variable arbitrary suggestions about the characteristics of orthodontic forces abound in the literature.

Orthodontic mechanotherapy is mainly aimed at tooth movement by remodeling and adaptive changes in paradental tissues. To effect this outcome, only small amounts of force—20 to 150 g per tooth—might be required. But craniofacial orthopedics is aimed at delivering higher magnitudes of mechanical forces—more than 300 g—in attempts to modify the form of craniofacial bones. The appliances, called craniofacial orthopedic devices, deliver macro-scale mechanical forces, which produce micro-structural sutural bone strain and induce cellular growth response in sutures.[7]

OPTIMAL ORTHODONTIC FORCE

Orthodontic tooth movement is mediated by coupling bone resorption and deposition in compressed and stretched sides of the PDL, respectively. Orthodontic forces, by virtue of altering the blood flow and localized electrochemical environment, upset the homeostatic environment of the PDL space. This abrupt alteration initiates biochemical and cellular events that reshape the bony contour of the alveolus.[8] It is assumed that an optimal orthodontic force moves teeth efficiently into their desired position, without causing discomfort or tissue damage to the patient. Primarily, an optimal force is based on proper mechanical principles, which enable the orthodontist to move teeth without traumatizing dental or paradental tissues, and without moving dental roots redundantly (round-tripping), or into danger zones (compact plates of alveolar bone). Traditionally, orthodontic forces have been categorized as "light" or "heavy," and it was assumed that light forces are gentler and therefore more physiologic than heavy forces. However, Burstone[9] reported that orthodontic forces are never distributed equally throughout the PDL, and Storey[10] observed that some trauma is always associated with applied orthodontic forces, even light ones. Moreover, it is impossible, with the available instrumentation, to measure precisely the amount of force applied to roots or parts thereof under any mode of treatment. Consequently, at present, it can be stated that, to engender adequate biological response in the periodontium, light forces are preferable, because of their ability to evoke frontal resorption of bone. Unlike light forces, heavy forces often cause necrosis (hyalinization) of the PDL and undermining bone resorption,[11] and have been implicated in root resorption.

The concept of optimal orthodontic force is changing along with the specialty. The classic definition of optimal force by Schwarz[12] in 1932 was "the force leading to a change in tissue pressure that approximated the capillary vessels' blood pressure, thus preventing their occlusion in the compressed periodontal ligament." According to Schwarz, forces below optimum produce no reaction, whereas forces above that level lead to tissue necrosis, thus preventing frontal resorption of the alveolar bone. Oppenheim[13] and Reitan,[11] who recommended applying light forces for tooth movement, demonstrated cell-free compressed areas in the PDL. Storey and Smith[14] also reported the same finding in 1952. They studied distal movement of canines in orthodontic patients and suggested that there is an optimum range of pressure (150-200 g) on the tooth-bone interface that produces a maximum rate of tooth movement. Pressure below this range produced no tooth movement. When the force was increased above optimum, the rate of tooth movement was decreased and finally approached zero within a week.

The current concept of optimum force views it as an extrinsic mechanical stimulus that evokes a cellular response that aims to restore equilibrium by remodeling periodontal supporting tissues. So the mechanical input that leads to the maximum rate of tooth movement with minimal irreversible damage to root, PDL, and alveolar bone is considered to be optimal. This concept means that there is a force of certain magnitude and temporal characteristics (continuous v intermitted, constant v declining) capable of producing a maximal rate of tooth movement, without tissue damage, and with maximum patient comfort.[15,16] According to this concept, the optimal force might differ for each tooth and for each patient. Clinically, the relationship between orthodontic force magnitude and rate of tooth movement during active treatment is now considered to be a practical tool in identifying optimal forces on an individual basis.

THEORIES OF ORTHODONTIC MECHANISMS

Orthodontic tooth movement has been defined as the result of a biologic response to interference in the