

Name:

Nwadiuto Ekeh

Group:

10 A-5

Basic Science Question:

How does tooth movement and bone remodelling occur?

Report:

In orthodontics, the movement of teeth in human dentition is a result of alveolar bone and periodontal ligament remodeling surrounding the roots of teeth. Bone remodeling is the dynamic, cyclic, coupled formation and destruction of bone with minor reversible injury to the periodontium [1,2]. The alveolar bone is thus able to physiologically adapt to applied mechanical strains. The theory of pressure-tension of bone remodeling suggest that healthy bone mass is maintained throughout the lifespan, providing the best, functional occlusion of dentition [2,3,4]. Orthodontic tooth movement is thus a type of biological response to applied force.

Tooth movement occurs in three generalized stages: the initial, lag and postlag phases [5]. The initial phase occurs immediately after the application of force (i.e. from 24-48 hours) and is characterized by the rapid displacement that reflects the viscoelastic properties of the tooth-supporting structure in the bony socket. The lag phase occurs for 20-30 days and involves minimal to no movement due to the hyalinization of compressed periodontal ligament that allows the removal of the dead tissue by various cells. The postlag phase is seen after 40 days and is characterized by gradual or sudden movement of the bone in the linear direction, along with continued development and removal of necrotic tissue.

In bone remodeling, the coordinated effects of mechanical stimuli and bone cells in the oral and maxillofacial tissues facilitates constant bone surface renewal [2,3]. With force loading, teeth in the periodontal ligament space can shift, leading to the release of biologically active molecules such as chemokines, neurotransmitters, growth factors, and arachidonic acid metabolites that initiate aseptic inflammatory cascades and create unique microenvironments at different sites of the bone. The culminating result is that the periodontal ligament compresses at certain location in the periodontium and stretches, or experiences tension, in other areas. Localized hypoxia, lowered blood flow, and a marked spike in osteoclasts occurs on the compression side while hyperoxia, increased blood flow, and an increase in osteoblastic activity occurs on the tension side. Tooth movement is a result of begins once dead tissues is removed by osteoclasts and osteoblasts have laid down new osteoid in the bone [1].

Osteoclasts are large cells with multiple nuclei and are formed by fusion of hematopoietic stem cells, specifically monocytes and/or macrophages[1,5]. Osteoclasts decalcify the bone matrix by hydrochloric acid and proteolysis. Osteoblasts are mesenchymal-derived, single-nucleus cells that function in the (re-)mineralization of bone matrix [1]. Osteocytes are derived from osteoblasts that become entrapped in the bone matrix during bone apposition, residing in specialized areas of deposited minerals such as hydroxyapatite, calcium carbonate and calcium phosphate that form a lacuna. They use dendritic processes and gap junction to connect to surface osteoblasts, to communicate with adjacent osteocytes using canaliculi and to mediate the bone remodeling process by sensing physical stimuli [1,2]. They are thus considered mechanosensors that couple strain from tooth loading to the process of

bone remodeling [2].

References:

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