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LIGHT AT THE END OF THE TUNNEL

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ABSTRACT

Accelerating orthodontic tooth movement can significantly reduce treatment duration and risks of side effects. The rate of orthodontic tooth movement is chiefly determined by the remodeling of tissues surrounding the roots; this in turn is under the control of molecular mechanisms regulating cellular behaviors in the alveolar bone and periodontal ligament. This review summarizes the current knowledge on the molecular mechanisms underlying accelerated orthodontic tooth movement, and the clinical and experimental methods that accelerate orthodontic tooth movement with possible molecular mechanisms. The review also shows directions for future studies to develop more clinically applicable methods to accelerate orthodontic tooth movement.

KEYWORDS: Tooth movement Non pharmacological techniques, Photobiomodulation.

INTRODUCTION

Orthodontic movement of teeth under mechanical force depends on the remodeling of tissues surrounding the roots. Accelerating orthodontic tooth movement has long been desired for its multiple potential benefits, including shorter treatment duration, reduced side effects (such as oral-hygiene related problems, root resorption, and open gingival embrasure spaces), enhanced envelope of tooth movement,[1-5] differential tooth movement, and improved posttreatment stability.[6] Attempts to accelerate tooth movement can be dated back to the 1890s, almost contemporary with Angle's groundbreaking work in modern orthodontics.

For the next half century, the intervention to accelerate tooth movement involved osteotomy, the surgical procedure that completely cuts both the cortex and the medulla of the alveolar bone.

The rationale for performing osteotomy was to reduce mechanical resistance during tooth movement. In the 1950s, introduced corticotomy, the perforation of the cortex of the bone alone without intrusion into the medulla, to replace osteotomy except in the subapical region, to reduce invasiveness.

Since it was less destructive, corticotomy completely replaced osteotomy as the preferred surgical method to accelerate tooth movement. Despite the evolution of clinical methods, the scientific explanation of accelerated tooth movement was still believed to be reduced mechanical resistance after osteotomy or corticotomy, enabling the teeth to be moved en bloc with the tissues surrounding them.[7-8]

This view was challenged by Wilcko (including a periodontist and an orthodontist) circa 2000. They described the demineralization and remineralization process of the alveolar bone after corticotomy that resembled the regional acceleratory phenomenon (RAP), indicating increased bone remodeling activity.

Bone Modeling, Remodeling, And Orthodontic Tooth Movement: Fig.1

Bone modeling is the uncoupled process of activation-resorption (catabolic) or activation-formation (anabolic) on bone surfaces, resulting in changes of the shape, size, or position of the bone Even though bone remodeling renews the internal content of the bone without changing the size or shape of the bone under physiologic conditions, it also affects the rate of orthodontic tooth movement. Both bone modeling and remodeling are controlled by the cellular activities of osteoclasts, osteoblasts, and osteocytes.[9-13]

Apparently, osteoclasts carry out resorption, whereas osteoblasts carry out bone formation during bone modeling. The resorption-formation sequence of the

bone remodeling process is performed by basic multicellular units, which are organized osteoclasts and osteoblasts. Both biochemical and mechanical factors regulate the rates of bone modeling and remodeling.

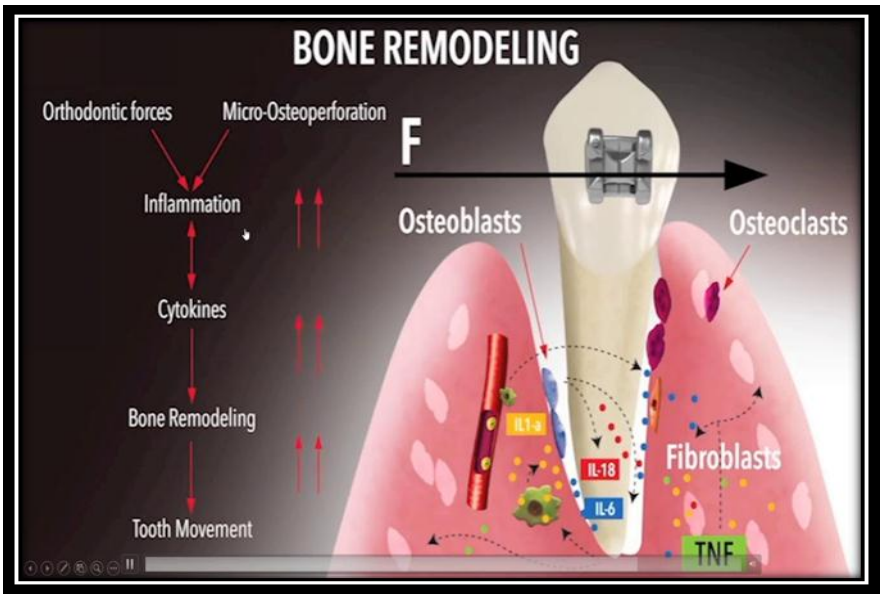

Fig. 1: Bone Remodeling.

Osteoclast Formation and Bone Resorption

The rate-limiting step in orthodontic tooth movement is considered to be bone resorption at the leading (compression) side. Histologic studies show that the formation of osteoclasts is induced at the compression side during orthodontic tooth movement.[14]

Alveolar corticotomy and nonsurgical interventions that accelerate tooth movement significantly increase the numbers and functions of osteoclasts. The formation of osteoclasts depends on the effects of stromal and osteoblastic cell-derived factors on osteoclast precursors. One of these factors is receptor activator of nuclear factor kappa B ligand (RANKL), which binds to its receptor, RANK, on the surface of developing osteoclastic cells.

The RANKL/RANK binding is crucial for the differentiation, function, and survival of osteoclasts.[15-17] On the other hand, osteoprotegerin (OPG), another osteoblastic cell-derived factor, interrupts the RANKL/RANK binding as a decoy receptor of RANKL, inhibiting osteoclastogenesis. Therefore, the RANKL/OPGratio expressed by osteoblastic cells and the RANK expression by osteoclast precursor cells largely determine the formation of functional osteoclasts and the activation of the initial step of bone remodeling. RANKL level in the gingival crevicular fluid becomes significantly higher than in the contralateral control side after 24 hours of continuous compressive force in adolescent patients.

Osteoblast Formation and Bone Apposition

Osteoblast proliferation, differentiation, survival, and function are regulated by a number of extracellular factors including growth factors, cytokines, and hormones, as well as by interactions with osteoclastic cells. Transforming growth factor (TGF)-b1 is a secreted protein that enhances bone formation by chemotactic effects on osteoblastic cells, promoting osteoblast proliferationand differentiation at early stages while inhibiting osteoclast formation by reducing RANKL and increasing OPG expression.[18-20]

The combination of orthodontic tooth movement and alveolar corticotomy is not simply the addition of 2 separate procedures but, rather, a more sophisticated synergy on bone cell.

Role of Osteocytes

Osteocytes are terminally differentiated osteoblasts that are embedded in the bone matrix during bone formation. They are stellate cells that form a functional network with other osteocytes, bone surface cells, bone marrow cells, and endothelial cells via long cytoplasmic extensions, or dendrites.[18]

This network of osteocytes occupies the lacunae-canaliculi system within the bone matrix, where the cell bodies and dendrites reside, respectively. Cell-to-cell signaling and material exchanges take place through the gaps between the dendrites of osteocytes and the interstitial fluid-filled lacunae-canaliculi system. Mechanical loading on the bone can cause strain in the bone structure, resulting in interstitial fluid flow in the lacunae-canaliculi system, which generates shear stress on the surface of osteocytes, activating mechanoreceptors on the cytoplasmic membrane of osteocytes and triggering intracellular signaling pathways, most notably the canonical Wnt pathway and the protein kinase. A pathway These signaling pathways lead to changes in the production level of biochemical

factors that are crucial for osteoclasts, osteoblasts, and bone remodeling.[19]

Osteocytes express M-CSF, RANKL, and OPG and regulate osteoclast formation and function. The expression of these factors by osteocytes is affected by microdamage in bone and mechanical loading during orthodontic tooth movement. Microdamage in bone causes osteocyte apoptosis, and the apoptotic bodiescontain RANKL to induce osteoclast formation. Osteocyte damage induced in cell cultures also resulted in increased production of M-CSF and RANKL, increasing osteoclast formation.[19]

Osteocyte apoptosis has been shown to be acutely induced by orthodontic tooth movement in mice, before osteoclast formation. RANKL expression was increased in osteocytes located close to the resorption lacunae. Targeted ablation of osteocytes significantly reduced osteoclast formation and bone resorption during orthodontic tooth movement.

Clinical and Experimental Methods To Accelerate Orthodontic Tooth Movement: (Fig.2)

Direct injury to the alveolar or basal bones of the maxilla and mandible accelerates orthodontic tooth movement by inducing RAP as a wound-healing process, which is the basis for clinical procedures such as corticotomy-assisted orthodontics, piezocision-aided orthodontics, and surgery-first orthodontics. The wound-healing process after trauma is similar, if not identical, as reviewed above. Nonsurgical methods, such as various physical and pharmacologic approaches, also enhance bone remodeling and facilitate tooth movement, and have been shown to be effective in animal and human experiments.[19-20]

The intentional use of alveolar surgery to speed up tooth movement began with the employment of osteotomy, the complete cutting of cortical and medulla of the bone, in the 1890s. Corticotomy, a surgical procedure to perforate the cortical bone without going into the medulla, was used in combination with osteotomy to accelerate tooth movement starting in the 1950s. Later, it was found that corticotomy alone was also effective in achieving faster tooth movement with much less tissue destruction and lower risks to periodontal tissues and dental pulp.

Bone grafts are embedded in tunnels connecting the vertical cuts. According to case reports in the literature, it appears that piezocision is similarly effective in accelerating tooth movement and augmenting periodontal tissues with much less trauma. Surgery-first orthodontics is a strategy to significantly shorten treatment duration for patients who need orthognathic surgery to correct a severe dentofacial deformity.[20]

A2 increase the speed of orthodontic tooth movement. Local submucosal injection of PGE1 in human patients was also successful in accelerating tooth movement by 1.6-fold. Alternatively, orthodontic tooth movement is impaired by nonsteroidal anti-inflammatory drugs, the compounds that inhibit the COX-1 and COX-2 enzymes that catalyze the ratelimiting step of prostaglandin formation. One concern of using PGs clinically is the pain reaction from patients, since PGs are potent pain inducers. Another concern is increased root resorption concomitant with accelerated tooth movement, as indicated by several independent studies.

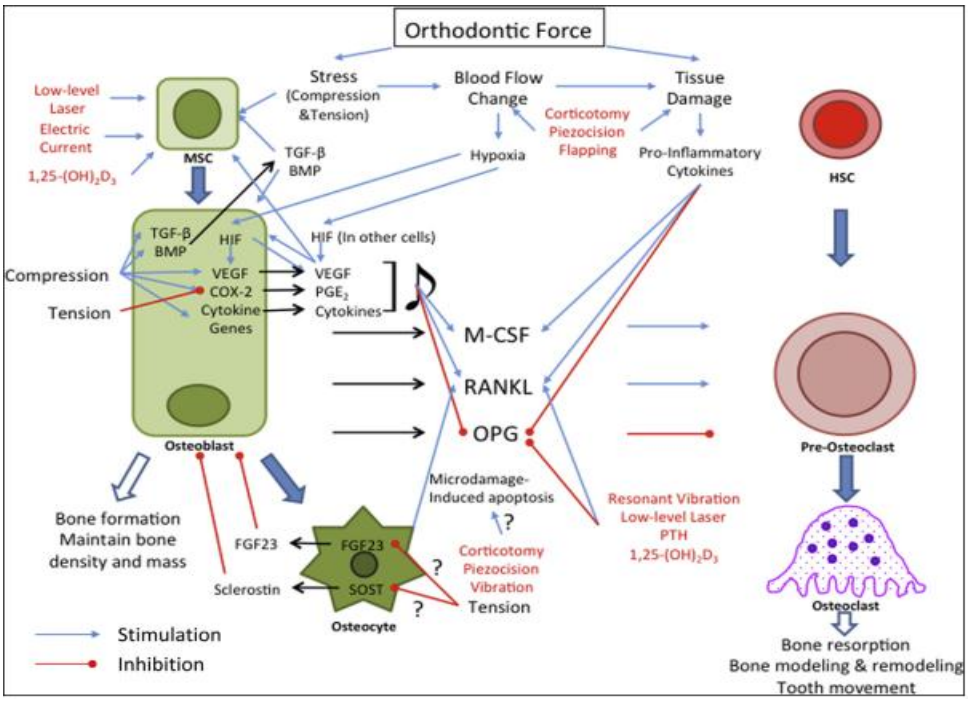

Figure 2: Orthodontic Forces.

CONCLUSION

The rate of orthodontic tooth movement depends onthe modeling and remodeling of the alveolar process while adapting to the new biomechanical environment. The rate of alveolar modeling and remodeling is determined by the level of activity of bone cells (osteoclasts, osteoblasts, and osteocytes), which are under the control of mechanical and biochemical factors, most notably PGs and cytokine.

Osteoclast activation is crucial for elevated bone modeling and remodeling required for accelerated tooth movement. Osteoblastic cell-derived cytokines M-CSF and the RANKL/OPG ratio determine osteoclast formation and function. Methods that accelerate orthodontic tooth movement stimulate M-CSF and increase the RANKL/OPG ratio directly or indirectly through changes in blood flow and hypoxia, and tissue damage, promoting the production of cytokines including VEGF, TNF-a, interferon-b, ILs, matrix metalloproteinases, and others. Osteoblasts are important in maintaining normal bone density and mass in the alveolar process. Some methods that accelerate tooth movement also induce enhanced osteoblast function by stimulating mesenchymal stem cells to differentiate into osteoblasts through cytokines including TGF-b, BMPs, VEGF, and others. Osteocytes, the most abundant bone cells, may also mediate the effects of methods that accelerate tooth movement by inducing osteoclast formation through apoptosis. The role of osteocytes is still not clear.

New knowledge in this field will empower us to revolutionize orthodontic therapy and its practice in the future.

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