



BIOLOGICAL AND PHYSICAL APPROACHES TO ACCELERATE TOOTH MOVEMENT

Orthopaedics

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KEYWORDS

INTRODUCTION-

Attempts to modify bone growth, both in terms of amount and direction have been made since antiquity. Initially orthodontists generally limited their mission to simply move the teeth, allowing the alveolar bone to remodel naturally, ignoring the alveolar topography. There are two basic ways to reduce the treatment duration, first approach is by making the treatment mechanics more efficient and another approach involves interventions to increase the velocity of orthodontic tooth movement by enhancing the bone remodeling. This intervention can be classified into three categories-use of certain biochemical; mechanical or physical stimulation of the alveolar bone which includes the use of cyclic vibration, magnets or direct electric current and surgical interventions to accelerate tooth movement.

CLASSIFICATION

1) BIOLOGICAL-

- cytokine.
- prostaglandins.
- parathyroid hormone.
- dihydroxy vitamin D₃(1,25-OH)₂D₃
- relaxin.

2) PHYSICAL STIMULATION

- resonance vibration.
- low level laser therapy (LLLT).
- direct electric current.
- pulsed electromagnetic field.
- static magnetic field.
- low intensity pulsed ultrasound.

3) SURGICALLY FACILITATED ORTHODONTIC TREATMENT (SFOT)-

- Corticotomy facilitated.
- periodontally accelerated osteogenic orthodontics (PAOO)

: WILCKODONTICS

- selective alveolar decortication (SAD).
- Osteotomy facilitated (DISTRACTION OSTEOGENESIS)-
 - Dental distraction (DD)
 - Interdental distraction (ID)/Periodontal distraction (PD).
- Dentoalveolar distraction (DAD)-
 - Transport dentoalveolar distraction.

PROSTAGLANDINS (PGE₂)-

Prostaglandins act by regulating the synthesis of cyclic AMP in many tissues. This allows prostaglandins to affect a wide range of cellular and tissue functions. According to Raisz et al, PGE act by increasing the number of osteoclasts, thereby stimulating bone resorption. Among the prostaglandins that had been found to affect bone metabolism (E₁, E₂, A₁ and F₂ -α), PGE₂ stimulated osteoblastic cell differentiation and new bone formation, coupling bone resorption in vitro.

CYTOKINES

High concentration of cytokines such as interleukins IL-1, IL-2, IL-3, IL-6, IL-8, and tumor necrosis factor alpha (TNF) were found to play a major role in bone remodeling; moreover, interleukin-1 (IL-1) stimulates osteoclast function through its receptor on osteoclasts¹. It was found that mechanical stress due to orthodontic treatment increased the production of prostaglandin PGE and IL-1β in the periodontal ligament. Other cytokines which are also involved in the acceleration of tooth movement are RANKL, which is a membrane-bound protein on the osteoblasts that bind to the RANK on the osteoclasts and causes osteoclastogenesis^{2,3,4}. On the other hand, osteoprotegerin (OPG) competes with RANKL in binding to osteoclast to inhibit osteoclastogenesis. The process of bone remodeling is a balance between (RANKL-RANK) system and OPG compound^{5,6}. In the study it was found that juvenile teeth move faster than adults, which is due to the lower amount of RANKL/OPG ratio in the gingival crevicular fluid (GCF) in adult patients measured by the enzyme-linked immunosorbent assay method.

PARATHYROID HORMONE:

PTH is produced by the parathyroid glands to regulate serum calcium concentration. PTH affects osteoblasts cellular metabolic activity gene transcriptional activity, and multiple protease secretion. Its effects on osteoclasts occur through the production of RANKL.

PTH has shown to accelerate orthodontic tooth movement in rats, which was studied by continuous infusion of PTH (1 to 10 μg/100g of body weight/day) which made the molars move 2 to 3 fold faster mesially by orthodontic coil spring⁹. Studies have shown that locally injected PTH induces local bone resorption and hence it is more advantageous to give PTH locally rather than systemically. It was confirmed that a slow-release local application of PTH was very efficient when a daily injection of PTH was dissolved in gel medium caused 1.6-fold faster acceleration of teeth compared to daily injection of PTH dissolved in saline which did not cause any acceleration¹¹.

EFFECT OF Vit. D₃

Vitamin D₃ has also attracted the attention of some scientist to its role in the acceleration of tooth movement, 1,25-dihydroxycholecalciferol is a hormonal form of vitamin D and plays an important role in bone formation.

Orthodontic tooth movement is brought about by alveolar bone and periodontal ligament remodeling. The two principal types of cells—osteoblasts and osteoclasts—maintain this balance between bone resorption and deposition during remodeling of the alveolar bone. Vitamin D (calcitriol) in its active form—1,25-dihydroxycholecalciferol—is one of the known potent stimulators of osteoclastic activity. Collins et al demonstrated that intraligamentary injections of 1,25-dihydroxycholecalciferol resulted in an increase in the number of osteoclasts and a 60% increase in tooth movement during canine retraction with light forces in cats. The receptors for vitamin D have been demonstrated not only in osteoclasts but are present in osteoblasts, also.

RELAXIN-

Relaxin has been known for decades as a pregnancy hormone. It is

released just before child birth to loosen the pubic symphysis. The role of relaxin is known in the remodeling of soft tissue rather than remodeling of bone. It has been shown that it increases collagen in the tension site and decreases it in compression site during orthodontic movement 14,15 Also, the administration of human relaxin may accelerate the early stages of orthodontic tooth movement in rat experiments 18

The remodeling of PDL by relaxin might reduce the rate of relapse after orthodontic treatment as suggested by others 15. However, the mechanism of how relaxin accelerates tooth movement is not yet fully understood.

RESONANCE VIBRATION

Nishimura et al in 2008, used a Niti expansion spring on the first molar of wistar rats, and applied a vibration of 60Hz/1m/S². In the sectioned samples, they showed increased RANKL expression in the fibroblasts and osteoclasts of the periodontal ligament of the rats that received the vibration. 13 Lieu et al in 2009 conducted a study on thirty mice, they used an omega shaped NiTi expander to deliver a force of 20g on the first molar. Mechanical vibration (4Hz for 20 min/day) was applied to the occlusal surface of the first molar. This regimen was repeated seven times every three days, upon micro CT examination of the jaws of the killed mice, it showed 40% more tooth movement. 15

Recently a product by the name Accedent has arrived, which makes use of this technology. This device consists of an activation which is the active part of the appliance that delivers the vibration implies with a USB interface through which it can be connected to a computer to review the patient usage of the appliance, a mouthpiece that contacts the teeth, it is a portable device that can be charged similar to any other electronic device and has to be worn for 20 minutes a day. Various case studies using this device have shown the treatment times to be reduced by 30-40%.

LOW LEVEL LASER THERAPY-

Photobiomodulation or low level laser therapy (LLLT) is one of the most promising approaches today. It has been found that laser light stimulates the proliferation of osteoclast and fibroblasts and thereby affects bone remodeling and accelerates tooth movement. The mechanism involved in the acceleration of tooth movement is by the production of ATP and activation of cytochrome C, that LLLT enhanced the velocity of tooth movement via RANK/RANKL and the macrophage colony-stimulating factor and its receptor expression. In 2004, Cruz et al. showed that the irradiated canines were retracted at a rate 34% greater than the control canines over 60 days.

Laser wavelength of 800nm and output power of 0.25mw have indicated significant stimulation of bone metabolism, rapid ossification, and also acceleration of tooth movement to 1.5 fold in rat experiments. Lately in a clinical trial study, the laser wavelength they have used in a continuous wave mode at 800nm, with an output of 0.25 mw and exposure of 10s was found to accelerate tooth movement at 1.3 fold higher than the control. More experiments are needed to differentiate the optimum energy, wavelength and the optimum duration for usage.

LOW INTENSITY PULSED ULTRASOUND-

Ultrasound is a sound wave having frequency above the limit of human ear perception, which can be transmitted into biological tissues. In a very recent study involving rat model, LIPUS accelerated orthodontic tooth movement by 45% and promoted alveolar bone remodeling by stimulating the HGF/Runx2/BMP-2.

PULSED ELECTROMAGNETIC FIELD-

The effect of electric current in bone was examined by Fukada and Yasuda. They hypothesized that electricity is produced when bone is stressed. This phenomenon, termed piezoelectricity, results from tension and compression in bone. The electric currents generated within the alveolar bone by orthodontic forces are thought to provide the signal for the directionality of the response (that is either resorption or deposition occurring during the remodeling process), whereas the generalized enhancement of the cellular activity is a function of the magnetic field strength. It has been suggested that PEMF affects the activity of intracellular cyclic AMP and cyclic GMP by causing a change in membrane permeability, allowing increased flow of calcium, sodium and potassium ions across the cell membrane.

A study on the effects of PEMF showed that tooth movement was

accelerated as a result of increase in quantity of active cells without changing the cell structure.

DIRECT ELECTRIC CURRENT-

This technique was tested only on animals by applying direct current to the anode at the pressure sites and cathode at the tension sites (by 7 V), thus, generating local responses and acceleration of bone remodeling 20. Their studies were more successful because electrodes were placed as close as possible to the moving tooth. The bulkiness of the devices and the source of electricity made it difficult to be tested clinically.

Further development of the direct electric device and the biocatalytic fuel cells is needed to be done so that these can be tested clinically.

REFERENCES

1. Davidovitch Z et al. Neurotransmitters, cytokines, and the control of alveolar bone remodeling in orthodontics. *Dent Clin North Am.* 1988; 32(3):411-35.
2. Udagawa N et al. Osteoblasts/stromal cells stimulate osteoclast activation through expression of osteoclast differentiation factor/RANKL but not macrophage colony-stimulating factor: receptor activator of NF-kappa B ligand. *Bone.* 1999; 25(5):517-23.
3. Drugarin DDM et al. RANKL/RANK/OPG molecular complex control factors in bone remodeling. *TMJ.* 2003; 53:296-302.
4. Kim SJ et al. Effects of low-intensity laser therapy on periodontal tissue remodeling during relapse and retention of orthodontically moved teeth. *Lasers Med Sci.* 2013; 28(1):325-33.
5. Simonet WS et al. Osteoprotegerin: a novel secreted protein involved in the regulation of bone density. *Cell.* 1997; 89(2):309-19.
6. Oshiro T et al. Osteoclast induction in periodontal tissue during experimental movement of incisors in osteoprotegerin-deficient mice. *Anat Rec.* 2002; 266(4):218-25.
7. Kanzaki H, et al. Local RANKL gene transfer to the periodontal tissue accelerates orthodontic tooth movement. *Gene Ther.* 2006; 13(8):678-85.
8. Soma S et al. Effects of continuous infusion of PTH on experimental tooth movement in rats. *J Bone Miner Res.* 1999; 14(4):546-54.
9. Soma S et al. Local and chronic application of PTH accelerates tooth movement in rats. *J Dent Res.* 2000; 79(9):1717-24.
10. Gurbax Singh1 et al 2ISSN 2637-7764 November 14, 2017 Modern Research in Dentistry.
11. Kale S et al. Comparison of the effects of 1,25-dihydroxycholecalciferol and prostaglandin E2 on orthodontic tooth movement. *Am J Orthod Dentofacial Orthop.* 2004; 125:607-614.
12. Collins MK et al 1988 The local use of vitamin D to increase the rate of orthodontic tooth movement. *Am J Orthod Dentofacial Orthop.* 94(4): 278-284.
13. Kale S et al. Comparison of the effects of 1,25-dihydroxycholecalciferol and prostaglandin E2 on orthodontic tooth movement. *Am J Orthod Dentofacial Orthop.* 2004; 125(5):607-14.
14. Nicozisis JL et al. Relaxin affects the dentofacial sutural tissues. *Clin Orthod Res.* 2000; 3(4):192-201.
15. Han GL et al. Expression of cathepsin K and IL-6 mRNA in root-resorbing tissue during tooth movement in rats. *Zhonghua Kou Qiang Yi Xue Za Zhi.* 2004; 39(4):320-3.
16. Bumann A et al. Collagen synthesis from human PDL cells following orthodontic tooth movement. *Eur J Orthod.* 1997; 19(1):29-37.
17. Liu ZJ et al. Does human relaxin accelerate orthodontic tooth movement in rats? *Ann N Y Acad Sci.* 2005; 1041:388-94.
18. Madan MS et al. Effects of human relaxin on orthodontic tooth movement and periodontal ligaments in rats. *Am J Orthod Dentofacial Orthop.* 2007; 131(1):8.e1-10.
19. Masella RS, Meister M. Current concepts in the biology of orthodontic tooth movement. *Am J Orthod Dentofacial Orthop.* 2006; 129(4):458-68.
20. Davidovitch Z et al. Electric currents, bone remodeling, and orthodontic tooth movement. *Am J Orthod.* 1980; 77(1):33-47.