Review Article

Vibrations in orthodontics: Is it the future?

ABSTRACT

Ever since the advent of orthodontic therapy, time required for orthodontic treatment has always been under the scanner. Various studies have been done solely for the purpose of decreasing the treatment time. Few methods were invasive while others are not successful enough in accelerating the treatment time. One of the latest methods to accelerate orthodontic treatment is the use of high-frequency small-magnitude vibrations at specific locations. Various animal studies have already been carried out to enhance the methods used to increase orthodontic tooth movement (OTM) and in turn decrease the treatment time. Electronic databases of PubMed library were searched from 1998 to 2018. Ten clinical studies were evaluated after considering the inclusion and exclusion criteria. It was concluded that high-frequency low-magnitude vibrations can increase OTM by activation of receptor activator of nuclear factor-kappa B/receptor activator of nuclear factor-kappa B ligand pathway and stimulating the periodontal tissues.

Keywords: Orthodontic tooth movement, osteoprotegerin, receptor activator of nuclear factor-kappa B/receptor activator of nuclear factor-kappa B ligand, vibrations

INTRODUCTION

In the present era, there is an increased tendency for researchers and orthodontists to focus on accelerating methods for tooth movement due to the huge demand seen among adults in seeking shorter orthodontic treatment time. It has been seen that long orthodontic treatment time poses several disadvantages such as higher predisposition to caries and gingival recession. Further, it has been reported that the total treatment duration also proves to be highly correlated with root resorption.[1]

This increases the demand to find the best method to accelerate tooth movement with the least possible disadvantages. However, it is still very challenging to reduce the duration of orthodontic treatments. A number of attempts have been made to create different approaches both preclinically and clinically in order to achieve quicker results. Those attempts can broadly be categorized into biological, physical, biomechanical, and surgical approaches.[1]

Tooth movement involves both remodeling and modeling of bone through coordinated action of osteoclasts and osteoblasts in response to mechanical loading.[2] Physiologically, the rate of tooth movement reflects the rates of bone turnover and remodeling. Earlier approaches that have been used in an attempt to accelerate tooth movement have included low-energy laser irradiation, magnetic fields, and pharmacological interventions with the injection of prostaglandin E2 (PGE2) and Vitamin D. However, adverse events, such as local pain and severe root resorption, were associated with these treatments. Corticotomy-facilitated orthodontics has limited clinical use due to the morbidity of the surgery, cost, and insufficient clinical evidence.[3]

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Thus, accelerating orthodontic tooth movement (OTM) and shortening the total treatment duration has been a primary goal of the orthodontists, and it can prevent detrimental effects of longer treatment time and increase patient satisfaction.[4] Low-level mechanical oscillatory signals (vibrations) have shown to increase the rate of remodeling in mechanically loaded long bones, which is currently used in osteoporosis in menopausal women. There are evidence from animal studies using cranial suture model and long bone periosteum, suggesting that dynamic loading improves bone formation and increases OTM compared to a static force.[3]

While there is an emerging body of evidence that vibration enhances OTM in animals, the effect of analogous level vibrations on tooth movement in patients had not been widely investigated.[4] Studies have shown that whole-body vibration (30, 45, and 90 Hz) may have an anabolic response on bone mass and architecture.[5] Miles *et al*.,[6] in their randomized controlled trial, showed that application of 111 Hz of vibrational frequency for 20 min/day did not speed tooth movement when compared with the controls. Kalajzic *et al*.[7] showed the inhibitory effect of cyclical forces (30 Hz and 40 g of force applied with an electromechanical actuator) on OTM in rats.

Recently, low-frequency magnetic vibration has gained interest in accelerating OTM by increasing alveolar bone turnover. The osteocytes are thought to orchestrate "mechanotransduction" by reacting to different forms of mechanical loading through biologic signals. The role of osteocytes in bone remodeling and modeling has been well documented.[8,9] It has been shown that osteocytes are the major source of sclerostin (product of SOST (sclerostin) gene), and they antagonize the canonical Wnt signaling pathway, thus exhibiting an inhibitory effect on bone formation.[10,11] Matsumoto *et al*.[12] demonstrated the role of osteocytes in resorption modeling during OTM (mechanotransduction) using osteocyte-ablated mice.

MATERIALS AND METHODS

A MEDLINE search was conducted to identify clinical articles published between the years 1998 and 2018 on vibrations used in orthodontics using the keywords, such as "periodontal tissue activation" and "vibration," "accelerating tooth movement" and "vibration," "low magnitude high frequency vibration" and "periodontal tissue," and "mechanical vibrations" and "orthodontic tooth movement." In addition, a manual search of PubMed journals from the years 1998 to 2018 was also conducted. Articles which were related to other methods of accelerating OTM apart from mechanical vibrations such as low-energy laser and corticotomy were excluded. No restrictions were placed with regard to study designs such as case–control, cohort, pilot, and randomized clinical trials.

Studies included for the present study fulfilled the following inclusion criteria:

1. Clinical study
2. OTM with the use of low-magnitude high-frequency mechanical vibrations
3. The magnitude and frequency of vibrations employed specified with the parameters followed
4. The outcome of the therapy
5. The associated advantages and limitations specified.

Accordingly, ten clinical articles were reviewed under the headings of biology of tooth movement, effect of receptor activator of nuclear factor-kappa B/receptor activator of nuclear factor-kappa B ligand (RANK/RANKL) in OTM, and effect of vibrations in OTM. The analysis outshines the key aspects of vibrations in relation to orthodontic therapy which will help in determining the use and indication of mechanical vibrations.

BIOLOGY OF ORTHODONTIC TOOTH MOVEMENT

Orthodontic mechanotherapy is aimed at tooth movement by remodeling and adaptive changes in paradental tissues. To affect this outcome, only small amounts of force, i.e. 20–150 g/tooth, are usually required. However, craniofacial orthopedics is aimed at delivering higher magnitudes of mechanical forces, i.e., more than 300 g, in an attempt to modify the form of craniofacial bones.[13]

The early phase of OTM always involves an acute inflammatory response, characterized by periodontal vasodilatation and migration of leukocytes out of the capillaries. These migratory cells produce various cytokines, the local biochemical signal molecules, that interact directly or indirectly with the native paradental cells.[13]

The acute inflammatory process that takes place in the initial phase of OTM is predominantly exudative, in which plasma and leukocytes leave the capillaries in areas of paradental strain. One or 2 days later, the acute phase of inflammation subsides and is replaced by a chronic process that is mainly proliferative, involving fibroblasts, endothelial cells, osteoblasts, and alveolar bone marrow cells. During this period, leukocytes continue to migrate into the strained paradental tissues and modulate the remodeling process.

Chronic inflammation prevails until the next clinical appointment, when the orthodontist activates the

Figure 1: Propel VPro5 used for applying high-frequency vibrations during orthodontic treatment

Figure 2: Acceledent commonly used device for vibration delivery

tooth-moving appliance, thereby starting another period of acute inflammation, superimposing it on the ongoing chronic inflammation.[13]

EFFECT OF RECEPTOR ACTIVATOR OF NUCLEAR FACTOR-KAPPA B/RECEPTOR ACTIVATOR OF NUCLEAR FACTOR-KAPPA B LIGAND IN ORTHODONTIC TOOTH MOVEMENT

Cytokines are recognized as extracellular signaling proteins that act on nearby target cells in low concentrations. Various cytokines that were found to affect bone metabolism, and thereby OTM, include interleukin-1 (IL-1), IL-2, IL-3, IL-6, IL-8, tumor necrosis factor (TNF)-alpha, gamma-interferon, and osteoclast differentiation factor.

The role of cytokines of the RANKL/RANK/osteoprotegerin (OPG) system in inducing bone remodeling was demonstrated by Drugarin *et al*.[14] The TNF-related ligand RANKL and its two receptors, RANK and OPG, have been shown to be involved in the remodeling process. RANKL is a downstream regulator of osteoclast formation and activation, through which osteoresorptive effect is produced by many hormones and cytokines. In the bone system, RANKL is expressed on osteoblast cell and exerts its effect by binding the RANK receptor on osteoclast cells.[13]

OPG is a decoy receptor produced by osteoblastic cells, which competes with RANK for RANKL binding. Bone remodeling is controlled by a balance between RANK-RANKL binding and OPG production.

Kanzaki *et al*.[15] reported recently that OPG gene transfer to periodontal tissues inhibited RANKL-mediated osteoclastogenesis and inhibited experimental tooth movement in rat.

EFFECT OF VIBRATION ON ORTHODONTIC TOOTH MOVEMENT

Till date, various methods have been investigated to accelerate tooth movement, such as physical approaches with low-energy laser irradiation[16], use of magnetic fields,[17] and pharmacological approaches with the injection of PGE2[18] and 1, 25-dihydroxycholecalciferol or 1, 25-(OH) D[19] during tooth movement. However, many side effects, such as local pain, severe root resorption,[20] and drug-induced side effects,[21] have been reported. The initial response of cells to mechanical stress *in vitro* appears within 30 min.[22]

The loading of resonance vibration that is equal to the natural frequency of the first molar and its periodontal tissue stimulates the periodontal tissue more effectively.[23] Hence, it was thought that the application of resonance vibration during OTM would affect the acceleration of tooth movement by increasing the activity of the cells in the periodontal ligament (PDL). Nishimura[15] *et al*. have demonstrated the activation of the RANK-RANKL signaling pathway in response to the loading of resonance vibration. It has been reported that signaling molecules, such as c-fos (a proto-oncogene, activation leads to overexpression of cyclin D1, A, and E in osteoblasts),[22] MAPK,[24] and nitric oxide,[25] are increased in the PDL immediately after mechanical stimulation.

Loading a vibrational force for 1.5 h/day over 3 weeks was reported to give about 1.3–1.4 times greater tooth movement than loading a static force.[26]

They concluded that the application of resonance vibration might accelerate OTM through enhanced RANKL expression in the PDL with no additional damage to periodontal tissues, such as root resorption.

The speed of tooth movement was influenced mainly by bone resorption, with osteoclasts induced on the alveolar bone surface on the pressure side.

In a study done, it has been reported that RANKL is an essential factor for osteoclast formation, function, and survival.[27]

Kartsogiannis *et al*.[28] reported that the levels of RANKL mRNA and protein appear to correlate with resorptive capability, whereby osteoclasts on actively resorbed surfaces display high-level RANKL expression. Increased RANKL expression in PDL fibroblasts and osteoclasts might induce and activate osteoclasts. Consequently, alveolar bone remodeling could be enhanced.

Resonance vibration can be applied as a mechanical stress on PDL cells. Ultrasonic vibration is a form of vibrational stimulation that is similar to resonance vibration. It has been reported that ultrasonic vibration accelerates tooth movement.[29] However, ultrasonic vibration of teeth might prove to be hazardous, such as thermal damage to the dental pulp.[30]

Alikhani *et al*.[31] reported that micro-osteoperforation increased the tooth movement by 2.3 fold,measured during the period of initial 28 days of canine retraction into a first bicuspid extraction space. Their results are consistent with studies using other invasive procedures, such as corticotomy[32] and similar surgical interventions. A recent systematic review and meta-analysis (which did not include vibration) revealed some evidence for effectiveness of low laser therapy and corticotomy and only a weak or no evidence for the effectiveness of interseptal bone reduction, photobiomodulation, and pulsed electromagnetic fields.[33]

Pavlin *et al*.[3] in their study concluded that the application of cyclic loading (vibration) of 0.25 N (25 g) at the frequency of 30 Hz, as an adjunct to treatment with a fixed orthodontic appliance, significantly increases the rate of OTM.

Vibrational loading stimulates bone remodeling, but the biological mechanism underlying this effect is not much understood. Mechanical loading initiates signaling pathways in bone, and osteocytes were identified as mechanoresponsive cells during OTM, in which signals can be triggered by fluid shear stress, bone microfractures, or bone bending, all of which occur during vibrations. Early responses in osteocytes are followed by differentiation of osteoblasts and stimulation of other bone genes.

CONCLUSION

In relation to various articles reviewed, it can be deduced that the effect of vibration on OTM could prove to be boon in the context of accelerating tooth movement and reducing the treatment time. This review has shown many evidence with regard to increase in activation of cells in the PDL by the use of vibration in orthodontic therapy with the use of devices like Propel VPro5 [Figure 1] or Acceledent [Figure 2]. It can be concluded that compared to invasive methods to accelerate OTM such as corticotomy or microperforations, using mechanical vibrations could prove to be a much safer and comfortable alternative. However, future studies should address the question whether cyclic loading, as an adjunct to orthodontic stress, activates known or new signaling pathways underlying the faster tooth movement.

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Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Nimeri G, Kau CH, Abou-Kheir NS, Corona R. Acceleration of tooth movement during orthodontic treatment – A frontier in orthodontics. Prog Orthod 2013;14:42.
2. Hadjidakis DJ, Androulakis II. Bone remodeling. Ann N Y Acad Sci 2006;1092:385-96.
3. Pavlin D, Anthony R, Raj V, Gakunga PT. Cyclic loading (vibration) accelerates tooth movement in orthodontic patients: A double-blind, randomized controlled trial. Semin Orthod 2015;21:187-94.
4. Yadav S, Dobie T, Assefnia A, Gupta H, Kalajzic Z, Nanda R. Effect of low-frequency mechanical vibration on orthodontic tooth movement. Am J Orthod Dentofacial Orthop 2015;148:440-9.
5. Rubin C, Turner AS, Müller R, Mittra E, McLeod K, Lin W, *et al.* Quantity and quality of trabecular bone in the femur are enhanced by a strongly anabolic, noninvasive mechanical intervention. J Bone Miner Res 2002;17:349-57.
6. Miles P, Smith H, Weyant R, Rinchuse DJ. The effects of a vibrational appliance on tooth movement and patient discomfort: A prospective randomised clinical trial. Aust Orthod J 2012;28:213-8.
7. Kalajzic Z, Peluso EB, Utreja A, Dyment N, Nihara J, Xu M, *et al.* Effect of cyclical forces on the periodontal ligament and alveolar bone remodeling during orthodontic tooth movement. Angle Orthod 2014;84:297-303.
8. Nishiyama Y, Matsumoto T, Lee JW, Saitou T, Imamura T, Moriyama K, *et al.* Changes in the spatial distribution of sclerostin in the osteocytic lacuno-canalicular system in alveolar bone due to orthodontic forces, as detected on multimodal confocal fluorescence imaging analyses. Arch Oral Biol 2015;60:45-54.
9. Bonewald LF. The amazing osteocyte. J Bone Miner Res 2011;26:229-38.
10. van Bezooijen RL, ten Dijke P, Papapoulos SE, Löwik CW. SOST/sclerostin, an osteocyte-derived negative regulator of bone formation. Cytokine Growth Factor Rev 2005;16:319-27.
11. van Bezooijen RL, Roelen BA, Visser A, van der Wee-Pals L, de Wilt E, Karperien M, *et al.* Sclerostin is an osteocyte-expressed negative regulator of bone formation, but not a classical BMP antagonist. J Exp

Med 2004;199:805-14.
12. Matsumoto T, Iimura T, Ogura K, Moriyama K, Yamaguchi A. The role of osteocytes in bone resorption during orthodontic tooth movement. J Dent Res 2013;92:340-5.
13. Krishnan V, Davidovitch Z. Cellular, molecular, and tissue-level reactions to orthodontic force. Am J Orthod Dentofacial Orthop 2006;129:469.e1-32.
14. Drugarin D, Drugarin M, Negru S, Cioace R. RANK-RANKL/OPG molecular complex – Control factors in bone remodeling. TMJ 2003;53:296-302.
15. Kanzaki H, Chiba M, Takahashi I, Haruyama N, Nishimura M, Mitani H. Local OPG gene transfer to periodontal tissue inhibits orthodontic tooth movement. J Dent Res 2004;83:920-5.
16. Kawasaki K, Shimizu N. Effects of low-energy laser irradiation on bone remodeling during experimental tooth movement in rats. Lasers Surg Med 2000;26:282-91.
17. Tengku BS, Joseph BK, Harbrow D, Taverne AA, Symons AL. Effect of a static magnetic field on orthodontic tooth movement in the rat. Eur J Orthod 2000;22:475-87.
18. Yamasaki K, Miura F, Suda T. Prostaglandin as a mediator of bone resorption induced by experimental tooth movement in rats. J Dent Res 1980;59:1635-42.
19. Collins MK, Sinclair PM. The local use of Vitamin D to increase the rate of orthodontic tooth movement. Am J Orthod Dentofacial Orthop 1988;94:278-84.
20. Brudvik P, Rygh P. Root resorption after local injection of prostaglandin E2 during experimental tooth movement. Eur J Orthod 1991;13:255-63.
21. Ochiai H. Histological investigations on the effect of prostaglandin E2 (PGE2) applied to experimental tooth movement. Nihon Kyosei Shika Gakkai Zasshi 1987;46:500-16.
22. Yamaguchi N, Chiba M, Mitani H. The induction of c-fos mRNA expression by mechanical stress in human periodontal ligament cells. Arch Oral Biol 2002;47:465-71.
23. Emata T. The mechanical response of the periodontal structure in the maxillary lateral incisor of the *Macaca fuscata yakui*, loading by a vibrating force. J Oral Biol Sci 1979;21:571-85.
24. Matsuda N, Morita N, Matsuda K, Watanabe M. Proliferation and differentiation of human osteoblastic cells associated with differential activation of MAP kinases in response to epidermal growth factor, hypoxia, and mechanical stress *in vitro*. Biochem Biophys Res Commun 1998;249:350-4.
25. Kikuiri T, Hasegawa T, Yoshimura Y, Shirakawa T, Oguchi H. Cyclic tension force activates nitric oxide production in cultured human periodontal ligament cells. J Periodontol 2000;71:533-9.
26. Shimizu Y. A study of the movement of the lateral incisor of the *Macaca fuscata* loaded by a vibrating force. Nippon Kyosei Shika Gakkai Zasshi 1986;45:56-72.
27. Boyle WJ, Simonet WS, Lacey DL. Osteoclast differentiation and activation. Nature 2003;423:337-42.
28. Kartsogiannis V, Zhou H, Horwood NJ, Thomas RJ, Hards DK, Quinn JM, *et al.* Localization of RANKL (receptor activator of NF kappa B ligand) mRNA and protein in skeletal and extraskeletal tissues. Bone 1999;25:525-34.
29. Ohmae M, Saito S, Morohashi T, Qu H, Seki K, Kurabayashi H, *et al*. Biomechanical acceleration of experimental tooth movement by ultrasonic vibration *in vivo* – Part 1. Homo-directional application of ultrasonication to orthodontic force. Orthod Waves 2001;60:201-12.
30. Trenter SC, Walmsley AD. Ultrasonic dental scaler: Associated hazards. J Clin Periodontol 2003;30:95-101.
31. Alikhani M, Raptis M, Zoldan B, Sangsuwon C, Lee YB, Alyami B, *et al.* Effect of micro-osteoperforations on the rate of tooth movement. Am J Orthod Dentofacial Orthop 2013;144:639-48.
32. Chung KR, Oh MY, Ko SJ. Corticotomy-assisted orthodontics. J Clin Orthod 2001;35:331-9.
33. Gkantidis N, Mistakidis I, Kouskoura T, Pandis N. Effectiveness of non-conventional methods for accelerated orthodontic tooth movement: A systematic review and meta-analysis. J Dent 2014;42:1300-19.