Accelerated orthodontics

Nausheer Ahmed[1], Nishat Sidiqha[2*], Shraddha Suryavanshi[3]

[1]Professor, [2,3]Post graduate Student, [1-3]Dept. of Orthodontics and Dentofacial Orthopedics, Government dental college and research institute, Bangalore, Karnataka, India

Corresponding Author: Nishat Sidiqha
Email: drnsidiqha@gmail.com

Abstract
Orthodontics has developed over years to meet the demands of the clinician and the patients. One of the primary concerns of the patients seeking orthodontic treatment is the duration of the treatment. In general, the duration required for fixed orthodontic treatment is about 2-3 years. There is increased demand for methods which accelerates the orthodontic tooth movement (OTM) with least possible side effects. Several methods have been reported to accelerate orthodontic tooth movement which include surgical methods, pharmacological methods, and mechanical stimulation. The aim of this article is to enumerate, and discuss the various methods of accelerating orthodontic tooth movement.

Keywords: Regional accelerate phenomenon**,** Corticotomy, Piezocision, Pharmacological agents, Low level laser therapy.

Introduction
Orthodontics has developed over years to meet the demands of the clinician and the patients. Continuous modifications in the arch wires and bracket systems has led to increased efficiency of the orthodontic treatment. Currently, the number of patients both young and adult seeking orthodontic treatment has increased. One of the primary concerns of the patient seeking orthodontic treatment is the duration of the treatment. In general, the duration of fixed orthodontic treatment is about 2-3 years. Prolonged duration of treatment poses risk of caries, external root resorption, gingival recession and decreased patient compliance.[1] Thus, there has been increased search for techniques that accelerates the orthodontic tooth movement (OTM).

Several methods have been reported to accelerate OTM, such as corticotomy, low level laser therapy (LLLT), pezocision, mechanical vibration,[2] and pharmacological agents (prostaglandins, vitamin D3, relaxin etc.).[3] To understand the mechanism of these methods, it is necessary to understand the basics of orthodontic tooth movement, the factors which initiate, modulate and inhibit the tooth movement. When orthodontic force is applied on the teeth it causes alteration of the blood flow, which in turn causes a change in the microenvironment around the PDL due to the release of various inflammatory mediators like cytokines, arachidonic acid metabolites, colony stimulating factors (CSF), growth factors, and neurotransmitters.[4,5] This brings about the remodelling of the bone. The purpose of this article is to review the various methods of accelerated OTM and their clinical applicability.

Different methods of accelerating orthodontic tooth movement
1. Drugs/ pharmacological methods.
2. Surgical method.
3. Physical or mechanical stimulation method.

Drugs
Prostaglandins (PGs)
Prostaglandins are a group of chemical messengers which belongs to a family of hormones called eicosanoids.[6] The three major classes of eicosanoids are prostaglandins, thromboxanes and leukotrienes. All eicosanoids are derived from arachidonic acid. PGs are paracrine hormones, i.e. they act only on cells near the point of hormone synthesis. Prostaglandins regulate the synthesis of cyclic AMP and in turn affect a wide range of cellular and tissue functions. They also sensitize the nerve ending to pain.

Synthesis of PGs in the body
During inflammation arachidonic acid liberated from the membrane phospholipid is converted to the cyclic endoperoxides prostaglandin G_2 (PGG_2) and PGH_2 by the cyclo-oxygenase (cox) enzymes present in the 'microsomal' fraction of most mammalian cells. The unstable endoperoxides can degrade nonenzymatically to the primary prostaglandins PGD_2, PGE_2 and $PGF_{2\alpha}$[7,13]

Role of PGs in orthodontic tooth movement
PGE2 is by far the most extensively tested agent for its capacity to modulate the tooth movement.[3] PGE2 increases the bone resorption by directly increasing the number of osteoclast cells and their bone resorptive activity during orthodontic tooth movement[4]. Experiments on rats by Yamasaki, showed that local administration of exogenous PGE2 increased the number of osteoclasts and accelerated orthodontic tooth movement.[8] Yamasaki et al. also studied the effect of prostaglandins on the rate of orthodontic tooth movement in Macaca fuscata monkeys and examined the possible side effects on gingival tissues. The results of experiments showed that the local administration of PGE1 or PGE2 in gingiva distal to canine to be retracted, caused double the rate of tooth movement compared to the opposite, control side with no side effects on the gingiva[9] except for a slight pain reaction consistent with tooth movement.[10]

Human studies revealed that rate of OTM increased by two times by local injection of prostaglandins.[10,11] PGs cause a significant increase in the cyclic AMP and intracellular calcium. These intracellular second messengers are important modulators of osteoclasts and bone resorption. It has also been demonstrated that repeated mucosal injections of 50 micro gram of PGE_1, produced marked changes in alveolar bone morphology such as increased resorption, extensive loss of bone matrix, fibrous replacement, and increased vascularity.[10]

Currently, the use of PGs is limited due to the need for repeated injection, severe pain associated with injection, and the possible risk of root resorption[3].

When PGE2 was administered in the presence of calcium gluconate, the calcium ions stabilized the root resorption while significantly increasing OTM.[12] Misoprostol, a synthetic PGE1 analogue demonstrated enhanced tooth movement without significant increase in the amount of root resorption in doses as low as 10–25 mg/kg, twice daily.[13,14]

Vitamin D3

1,25 dihydroxycholecalciferol (1,25[OH]2D3) or calcitriol is the most active hormonal form of vitamin D which is involved in calcium homeostasis. It regulates the calcium and phosphate serum levels, by promoting their intestinal absorption and reabsorption in the kidneys. Furthermore, it promotes bone deposition and inhibits PTH release.[15] Based on these mechanisms, one would expect that vitamin D3 should decrease the rate of tooth movement. To the contrary, local injection of vitamin D3 increased the rate of tooth movement by increasing the expression of RANKL by local cells which in turn activated the osteoclasts.[14]

Intraligamentous injection of 1,25D in dimethylsulfoxide (DMSO) in cats at weekly interval demonstrated 60% further tooth movement than matched control teeth.[16] Microscopically, increased numbers of mononuclear osteoclasts recruitment and activation, resulting in greater amounts of alveolar bone resorption on the pressure side was observed, with no obvious clinical, microscopic, or biochemical side effects[15].

A comparative evaluation of vitamin D3 and PGs on OTM by Kale et al showed that both significantly increase the amount of tooth movement when compared to controls, with increased number of osteoblasts on the external surface of the alveolar bone in vitamin D3 group in comparison to prostaglandin. Thus, it was concluded that 1,25 DHCC facilitated tooth movement through bone remodeling[17] and it also enhances the reestablishment of supporting tissue, which would improve post treatment stability.[18]

With the increasing prevalence of vitamin D deficiency worldwide, it may be possible to use vitamin D metabolites to enhance the rate of OTM. Further studies are needed to determine the safety of vitamin D in orthodontic treatment.

Parathyroid Hormone (PTH)

PTH is secreted by the parathyroid glands. PTH causes an increase blood calcium level by stimulating calcium absorption from intestine, by reducing the calcium excretion by kidneys, and release of calcium from bones. PTH has shown to influence OTM by binding to PTH 1 receptors on osteoblasts, leading to expression of insulin-like growth factor-1 (IFG-1), which in turn promotes osteoblastogenesis, increases osteoblast survival, expression of RANKL, and osteoclast activation.[19] The role of PTH on the rate of tooth movement is dose-dependent. Continuous elevation of PTH leads to bone resorption, while intermittent short elevations of the hormone level can be anabolic for the bone[14]

Li. et al investigated the effect of PTH injection on experimental tooth movement in rats and found that Intermittent exposure to PTH seems to increase bone formation, while continuous and long-term exposure (longer than 1–2 years) enhances bone resorption. Intermittent exposure to PTH enhances both osteoblast and subsequently osteoclast activity which facilitates in bone remodelling/turnover.[20] Soma's experiment on rats demonstrated that, continuous subcutaneous infusion of PTH (10 μg PTH /100 g of body weight/day), accelerated the mesial movement of upper first molar. PTH infusion accelerated OTM by producing a 2 to 3-fold increase in the number of osteoclasts and enhancing bone resorption only in the compressed periodontium (pressure side) without undesired bone loss in other areas of alveolar bone[21]. It also caused rapid removal of necrotic tissue by stimulating bone cells to secrete proteolytic enzymes. Root resorption is usually initiated beneath the area of necrotic tissue in the compressed periodontal membrane. Thus, PTH may reduce the incidence of unfavourable root resorption by rapid removal of necrotic tissue from PDL during orthodontic therapy. Systemic administration of PTH may possibility enhance undesired bone resorption in weight-bearing bones such as vertebrae. Therefore, it is more appropriate to give PTH locally into the circumferential tissue of the tooth to be moved, rather than to give it systemically in orthodontic therapy.[21]

Relaxin

Relaxin is an ovarian hormone which belongs to the insulin superfamily. It helps in widening of the pubic ligaments in females during delivery. Presence of this hormone has also been demonstrated in cranial suture and PDL.

Relaxin stimulates bone cell activity and connective tissue turnover. It is mainly known for remodeling of soft tissue rather than bone.[14]

Madan, Lui et al evaluated the effect of relaxin on OTM and PDL in rats which showed increased collagen at tension site and decreased collagen at pressure site. It was concluded that the human relaxin may not accelerate orthodontic tooth movement in rats; it only reduces the level of PDL organization, reduce the mechanical strength of PDL, and increase mobility of the tooth at early stages of orthodontic treatment.[22,23] Results of a randomized clinical trial which was performed on humans using recombinant human relaxin showed that there was no significant difference between the relaxin and the placebo control group regarding the acceleration of tooth movement and

relapse[24]. Thus, Further studies are needed to understand the exact mechanism of action of relaxin.

Surgical Methods

In 1931, Bichlmayr introduced a surgical technique for rapid correction of severe maxillary protrusion where in, wedges of bone were removed to reduce the volume of bone through which the roots of the maxillary anterior teeth would need to be retracted.[25] All the surgical method of accelerating OTM is based on concept of "Regional Acceleratory Phenomenon" (RAP**).** Frost in 1983, described the (RAP) as a process in which tissue forms faster than the normal regional regeneration in the presence of local trauma (local response to a noxious stimulus). Thus, healing occur 2–10 times faster than normal physiologic healing[5].

Surgical irritation of bone increases levels of cytokine and chemokines around the tooth which play an important role in the recruitment of osteoclast precursor cells through RANK/RANKL pathway and also in the differentiation of precursors cells into mature osteoclasts. Thus, accelerate OTM.[26-28]

The various surgical methods available are

Corticotomy and Osteotomy

Heinrich Kole was first to describe corticotomy‐facilitated orthodontics in 1959 who is considered pioneer in corticotomy.[26] Osteotomy is a procedure where in a segment of the bone is cut involving the cortical and medullary bone. Then it is separated and moved as a unit.[29] In corticotomy only the cortical bone is cut and perforated but not the medullary bone. Kole attributed the accelerated tooth movement by selective corticotomy to moving "blocks of bone".[26] Wilcko et al combined corticotomy‐facilitated orthodontic technique with alveolar augmentation using particulate bone graft. This procedure was named as the accelerated osteogenic orthodontics (AOO) technique, and more recently, the periodontally AOO (PAOO).[27] Vertical cuts are placed between the and horizontal cuts 2-3 away from the apices in order to preserve the nerve bundles using micromotor under irrigation. This can be followed by placement of a graft material, wherever required, to augment thickness of bone.[30] The aim of corticotomy is to induce local temporary osteopenia to accelerate orthodontic tooth movement.

Corticision

'Corticision' refers to 'cortical bone incision'. Park et al in 2006 introduced this minimally invasive periodontal procedure to accelerate tooth movement without the need for flap elevation.[31] Armamentarium required involves: reinforced scalpel (No. 15T), ordinary scalpel holder and a surgical mallet.

The procedure is performed under local anesthesia. The scalpel is inserted gradually into the bone by tapping the scalpel holder with the surgical mallet, at an angle of 45–60° to the long axis of the tooth. The vertical cuts are placed 5 mm away from the papillary gingiva to avoid crestal bone loss which leads to development of the 'black triangle' and also to avoid damage to adjacent root. The depth of alveolar penetration is about 10 mm and the length of the cuts is about two-third of the root length. The effects of Corticision reaches its peak at 2 months and drops at 3 months, after which the patients are advised to visit every other week to keep the Corticision gap open, thus preventing the transformation woven bone into mature lamellar bone. post-operatively dizziness due to the malleting and inability to place bone graft during the procedure are the limiting factors.[31]

Piezocision

Piezocision is a less invasive approach than the conventional corticotomies. To reduce the morbidity associated with conventional corticotomy, Dibart et al in 2009, introduced a flapless method of corticotomy, using piezosurgery.[32]

It involves use of a vertical incision in the soft tissue mesial and distal to the tooth to be moved and the use of a piezoelectric knife to create linear incisions in the bone along the soft tissue openings created by a scalpel. The surgery is performed 1 week after placement of orthodontic appliance, under local anaesthesia. Using a No.15 scalpel gingival vertical incisions are made only bucally, and 2mm below the interdental papilla, as far as possible, in the attached gingiva. These incisions need to be deep enough so as to pass through the periosteum, and contact the cortical bone. Next, using a Piezotome (BS1 insert) which is an ultrasonic instrument, corticotomy cuts to a depth of 3 mm is made through the previously made incisions. In the areas requiring bone augmentation, submucosal tunnelling is performed using an elevator, to create sufficient space to accept a graft material. In the anterior mandibular region, due to narrow interdental bone and reduced width of the teeth, incisions between central and lateral incisor can be omitted.[32,33] No suturing is required, except for the areas, where the graft material needs to be stabilized. Patient is placed on an antibiotic, mouthwash regimen.[33] As incisions and corticotomy cuts are "blindly" done, there may be risk of root damage. To reduce this risk, Jorge et al in 2013, suggested a method, called Minimally Invasive Rapid Orthodontic procedure (MIRO) by using metal wire as a guide for placement of the incisions[34].

Micro-Osteoperforations (MOP)

Micro-Osteoperforations is a micro invasive technique which was developed to further reduce the invasive nature of surgical irritation of bone. It is performed using device called Propel, was introduced by Propel Orthodontics. It works by activating the cytokine cascade which in turn, results in a marked increase in osteoclast activity and in turn the rate of tooth movement. This process is called as Alveocentsis.[34] Alveocentesis, literally translates to puncturing bone[35]

Propel device is available as ready-to-use sterile disposable device with an adjustable depth dial which can be positioned to 0 mm, 3 mm, 5 mm, and 7 mm of tip depth, depending on the area of operation.[34] MOPs are placed in attached gingiva or upto 1mm apical to mucogingival

junction. To achieve increased recruitment of osteoclasts (catabolic effect), deep perforations of 5-7 mm are required, whereas if increased recruitment of osteoblasts is required (Anabolic effect) then shallow perforations of 1 mm, spread over a large area is required. To enhance the rate of tooth movement, reactivation of the orthodontic appliance is done after 2 weeks.

Mani Alikhani er al studied the effect of MOPs on humans which showed significant increase in levels of cytokines and chemokines which recruit osteoclast precursors and increase their differentiation into osteoclast. The rate of canine retraction was 2.3 times faster than control.[35] MOP causes 50-60% faster tooth movement than traditional orthodontics,[34] which leads to shorter treatment time.

Few of the Advantages of MOP involve: Less chair-side time, Less postoperative discomfort, zero recovery time and the patients can return to their normal daily routine immediately, specific teeth or quadrants can be targeted rather than applying to the whole dentition that may lead to anchorage issues, less chances of decalcifications and root blunting with shortened orthodontic treatment time,[33] can be used in conjunction with TADs, Invisalign, SureSmile, and conventional braces.[34]

Physical/Mechanical stimulation method

Cyclic forces (vibrations)

Cyclic forces or mechanical vibration has recently been emphasized in orthodontics as a non-invasive approach of accelerating tooth movement.[37]

light alternating forces were applied on the teeth via portable acyclic device to produce impulses of 20-30 Hz for 20 minutes each day. when vibrations are combined with orthodontic force, it increased the rate of tooth movement by upregulating the levels of inflammatory mediators, chemokines (CCL2), cytokines (IL-1b, and TNF-a) and RANKL in human periodontal ligament cells in vitro. It also stimulated differentiation of osteoclasts from hematopoietic cells by increasing the blood flow. Thus, stimulating the remodeling activity which brought about tooth movement at the rate of 2-3 mm/month and the treatment times could be reduced by up to 30-40%.[38]

Nishimura et al through his experimental studies, found that vibration may enhance orthodontic tooth movement via direct effects on the cell membrane, changes in ion transport, activation of stretch-activated channels, changes in the attachments between skeletal bones and extracellular matrix, or modification of intracellular signals that regulate gene expression to promote bone remodelling.[39] Randomized controlled trials showed that when vibrations were combined with compressive force, it increased the rate of tooth movement in canine distalization; however, it did not increase the rate of crowding correction.[37]one of the latest oral vibrating devices is the Acceledent system introduced in 2009. It has been approved by FDA, and Clinically Tested by SoftPulse Technology. It applies low magnitude cyclic forces by having the patient bite on the rubber interdental bite surface.[40]

Kau et al conducted a clinical trial, where in the patients were asked to wear the device for 20 minutes daily for a period of 6 consecutive months. Results of the study showed that total rate of tooth movement was 2.1 mm per month in the mandibular arch and for the maxillary arch it was 3.0 mm per month, which apparently is faster than the traditional finding of about 1.0 mm per month.[41] Mao and co-workers found cyclical forces between 1 Hz and 8 Hz, with forces ranging from 0.3N to 5N, increased bone remodelling. It was Concluded that even though mechanical vibration has shown to accelerate orthodontic tooth movement, further research is needed to clearly identify the optimal vibration protocol for desired results. Moreover, patient compliance and cost-effectiveness also needs to be considered.[37]

Light Accelerated Orthodontics (OrthoPulse)

OrthoPulse, was originally developed by Biolux Research company to accelerate bone regeneration after extractions and healing for implant placement. The OrthoPulse system involves wearing a device that fits over the patient's head, similar to a telephone headset. Light is delivered from an LED source extraorally to the site requiring treatment for extractions and implants.

The Biolux Company saw benefit for this technology in accelerating orthodontic movement and developed an intraoral version of the device. The product relies on the use of near-infrared light emitting diode (LED) therapy to accomplish accelerated bone remodeling. LED modifies cellular biology by exposure to light in red to near infrared range (600- 1000nm). This causes Cytochrome C oxidase which is present in the cell mitochondria to absorb light photons and convert it into chemical energy (ATP). The design is similar to a mouthguard, working with both traditional and clear aligner orthodontic therapy.[42, 43]

Low level laser therapy

Low Level Laser Therapy (LLLT) also known as Photobiomodulation Therapy (PBMT) is one the promising approach to accelerate tooth movement. It Utilizes low power lasers in the range of 600-1100nm. It enhances the velocity of tooth movement via stimulating the RANK/RANKL system, angiogenesis, proliferation of osteoclast, osteoblast, and fibroblasts, the macrophage colony-stimulating factor and receptor expression which are involved in the bone remodelling.[44]

LLLT caused an increase of fibronectin and type I collagen levels. Fibronectin not only facilitates in the turnover of connective tissue but also up-regulates RANKL which leads to osteoclastic differentiation during tooth movement. Thus, facilitating in bone and PDL turnover.[44,45] Yamaguchi et al. showed that low level laser therapy can increase Macrophage-colony stimulating factor (M-CSF) which stimulates proliferation and differentiation of osteoclastic progenitors on the compressed side leading to accelerated tooth movement.[46]

LLLT effectively controls pain caused by the application of the first arch wire by reducing the expression

of PGE2, tumor necrosis factor, plasminogen activator, and COX-2.[47] In human trials, canines were retracted at a rate 34% greater on the laser irradiated side than the control canines over a period of 60 days.[48] laser also has biostimulatory effect on bone tissue, which may be a useful tool for bone regeneration in mid palatal suture[49].

In-spite of the various positive findings, there are a lot of contradictory results related to the LLLT. Therefore, more evidence is needed to determine the optimum energy, wavelength, and the duration of usage for LLLT.

Conclusion

With the increasing number of adult patients seeking orthodontic treatment the demand for newer, less invasive, and safer methods to accelerate orthodontic tooth movement has also increased. Researchers have yet to seek a single most ideal technique for the patient. The invasive nature of the conventional surgical methods of accelerating the tooth movement has made them less desirable by the patients. Today newer methods such as Microsteoperforation, LLLT, vibrations show promising results in accelerating OTM with least discomfort. The new devices like propel and acceledent used in conjunction with orthodontic treatment offers an efficient method to reduce treatment time. Yet, more research is necessary to substantiate claims, enhance technology and techniques in the field of orthodontics.

Conflict of Interest: None.

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