Review Article

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Raise the Pace of Tooth Movement – Accelerated Orthodontics

Ritu Garg[1]* Kratika Mishra[2] Amit Bhardwaj[3]

[1]Professor and Head, Department of Orthodontics and Dentofacial Orthopaedics, Geetanjali Dental and Research Institute, Udaipur, India.
[2]Assistant Professor, Department of Orthodontics and Dentofacial Orthopaedics, Modern Dental College and Research Centre, Indore, India.
[3]Professor and Head, Department of Orthodontics and Dentofacial Orthopaedics, Modern Dental College and Research Centre, Indore, India.

ABSTRACT

Background: One of the main concerns in orthodontics is prolonged treatment duration. Long time-consuming orthodontic treatment may lead many patients, especially adults, to either avoid treatment or to seek shorter alternative solutions with compromised results. Therefore, the search for treatment modalities that decrease treatment time without compromising the treatment outcome is an active area of research in orthodontics today. In an ideal scenario where both the treatment rendered by clinicians and patient's cooperation are flawless, biology would be the only factor that dictates the rate of tooth movement in response to orthodontic forces. This review encapsulates the current knowledge on the biological principles and molecular mechanisms that govern tooth movement, and the plausible points of intervention along the molecular pathways where ability to stimulate target cells to achieve faster tooth movement.

Keywords: Accelerated Tooth Movement, Cortectomy, Micro-osteoperforations, Piezosurgery.

INTRODUCTION

In today's scenario, the prior question of people is how long the duration of an orthodontic treatment? The major concern is lengthy orthodontic treatment prompts many patients especially adults, to either avoid or search for an alternative solution with compromised results. Prolonged treatment time is a perplexing challenge of clinical orthodontics that has been not solved completely. If this challenge is overruled than there will be improved quality of orthodontic care to individuals, they will be highly motivated towards the orthodontic treatment and not only the aesthetic but functional as well as harmonized facial appearance can be achieved in less duration of time without compromised results.

Orthodontic appliances are not intentionally built to activate or inhibit specific remodeling pathways and specific cells. Rather, they are built to generate biomechanical force systems that produce the desired tooth and jaw movements needed to establish an ideal occlusion— regardless of the cellular mediators of the response.[1] Should we be designing orthodontic appliances to target specific remodeling pathways to move teeth and jaws into an ideal occlusion faster [2] To achieve quicker results its important to understand the the variables that control treatment duration to overcome the treatment time challenge. In general, these variables can be grouped under three categories:

Practitioner -dependent factors include-accurate diagnosis and treatment planning, sound mechanotherapy, appropriate appliance design, an delivery of treatment in timely fashion.

Patient-dependent factors includes attending scheduled appointments, compliance with practitioners' instructions, good oral hygiene, and maintaining the integrity of appliances.

Biological factors are tightly regulated by different molecular and cellular pathways and vary in magnitude for each individual.

In an ideal scenario where both the treatment rendered by clinicians and patient's cooperation are flawless, biology would be the only factor that

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***Correspondence Dr. Ritu Garg.**
Department of Orthodontics and Dentofacial Orthopaedics, Geetanjali Dental and Research Institute, Udaipur, India.
Email: gargritu14@gmail.com

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dictates the rate of tooth movement in response to orthodontic forces. First we will discuss about the biological principles and molecular mechanisms that govern tooth movement, and the plausible points of intervention along the molecular pathways where we are able to stimulate target cells to achieve faster tooth movement.

Based on these biological principles we can categorize the existing proposed techniques into two types:

- indirect
- direct

The indirect techniques aim to activate target cells that control the rate of tooth movement by increasing the release of upstream cytokines, while the direct techniques utilize various stimulants to directly activate the target cells. The indirect techniques range from minimally invasive techniques such as microosteoperforations (MOPs) to more invasive approaches such as piezocision and the significantly aggressive corticotomy.

The direct techniques include vibration, laser and ultrasound.

Biological principles of accelerated tooth movement

Orthodontic tooth movement is possible because the complex anatomy of the teeth, their alveolar housing and the soft tissues covering and coursing through the mineralized tissues are capable of remodeling. Without this remarkable normal biological phenomenon, the practice – indeed, the very concept – of orthodontics would not be possible.

The biology of tooth movement is not a new field of inquiry. What is new is that we are now designing innovative appliances and treatments that optimize skeletal and dental target cell responses that produce controlled, safe accelerated tooth movement.

By identifying and harnessing reactions of the target cells we can develop two different approaches to accelerate the rate of tooth movement:

- directly stimulate the target cells by artificial, physical, or chemical means to increase their numbers and their activity, or
- Indirectly stimulate the body to recruit and activate more target cells.

In either scenario, identifying the target cells and understanding how they are activated is crucial.

Bone cells and their role in biology of tooth movement

Bone is a surprisingly dynamic tissue that remodels in response to mechanical force. The cells that perform this remarkable response are distributed throughout the bone and each is specialized to perform specific functions needed to detect force (both its magnitude and direction), recruit cells that resorb bone at specific sites and activate cells to deposit new bone matrix and promote mineralization that will withstand mechanical force.

Osteoblasts-Osteoblasts are mononuclear cells found along the surface of bones. They are derived from mesenchymal stem cells in the bone marrow and synthesize collagenous and non-collagenous proteins that form the organic bone matrix called osteoid. Inactive osteoblasts that cover bone surfaces, particularly in the adult skeleton, are called bone lining cells. These cells are **quiescent** until growth factors or other anabolic stimuli induce them to proliferate and differentiate into cuboidal osteoblasts. While osteoblasts play an important role in maintaining the integrity of alveolar bone during tooth movement, they are not the cells that control the rate of tooth movement.

Osteocyte-The osteocyte is a mature osteoblast embedded in lacunae within the bone matrix. Although immobile, osteocytes possess exquisitely fine processes, which traverse the mineralized matrix in tunnels called canaliculi, to make contact with other osteocytes, as well as with osteoblasts residing on the bone surface. Given their preponderance in bone, and their intricate three-dimensional network, osteocytes are key **mechanosensors**. Loading of bone results in strain, or deformation, in the matrix, including the lacuanae and canaliculi. This deformation evokes osteocytic responses via fluid shear stress (produced by increased fluid flow in the lacuno-canalicular system) or electrical strain potential.

Osteocytes orchestrate the overall remodeling response by secreting key factors, such as prostaglandins, nitric oxide, insulin-like growth factors (IGFs), which activate osteoblasts and osteoclasts and the bone remodeling system. Under the influence of orthodontic forces, osteocytes play a critical role in detecting force and activating osteoclast-osteoblast coupling, but they are not the cells that regulate the rate of tooth movement.

Osteoclast-it is the osteoclast that determines the rate of bone resorption and, therefore, the rate of tooth movement. These cells are the major bone resorbing cells. They are specialized monocyte/macrophage family members that differentiate from haematopoietic stem cells in the bone marrow. Mature osteoclasts are giant multinucleated cells that form from the fusion of monocytic precursors. Terminal differentiation in this lineage is characterized by the acquisition of mature phenotypic markers, such as the calcitonin receptor and tartrate-resistant acid phosphatase (TRAP), and the appearance of an astounding ruffled border rich in proton pumps that acidify the bone surface to which the cells are attached, resulting in resorption pits. When viewed physiologically, normal healthy bone remodeling is a tightly choreographed sequence of cellular activity. Mechanical force distorts osteocytes housed in lacunae and canaliculi, often producing micro-fractures, which are cleared out by osteoclasts. Osteoblasts follow to fill in the newly excavated site. Some of those osteoblasts become embedded in the new bone to form new osteocytes to replace those lost at the remodeling site. Thus, healthy strong bone that can withstand mechanical force applications is formed due to signaling between osteocytes, osteoclasts and osteoblasts.

Biological approach-

Role of Chemokines

Chemokines are small proteins released by local cells that can attract other cells into the area. The release of chemokines in response to orthodontic forces facilitates expression of adhesion molecules in blood vessels and stimulates further recruitment of inflammatory and precursor cells from the microvasculature into the extravascular space. One of the chemokines that is released during tooth movement is monocyte chemoatractant protein-1 (MCP-1 or CCL2) [2], which plays an important role in recruiting monocytes. These cells leave the bloodstream and enter the surrounding tissue to become tissue macrophages or osteoclasts. Similarly, the release of CCL3 [3] and CCL5 (RANTES) [4] during orthodontic tooth movement leads to osteoclast recruitment and activation. Within the first few hours of orthodontic treatment there is further release of a broader spectrum of inflammatory mediators.

Role of cytokines

Cytokines are released during orthodontic treatment. These extracellular proteins play an important role in regulating the inflammatory process. Many cytokines are pro-inflammatory. They amplify or maintain the inflammatory response and activation of bone resorption. Importantly, other cytokines are anti-inflammatory and prevent an unrestrained inflammatory response. The main pro-inflammatory cytokines that are released during orthodontic tooth movement are IL-1α,IL-1β,TNF-α and IL-6 [5]. These cytokines are produced by inflammatory cells such as macrophages, and by local cells such as osteoblasts, fibroblasts and endothelial cells.

Prostaglandins and Neuropeptides-

Another series of inflammatory mediators that are released during orthodontic tooth movement are prostaglandins (PGs) and neuropeptides. PGs are derived from the metabolism of arachidonic acid and can mediate virtually every step of inflammation such as vasodilation, increase vascular permeability, and adhesion of inflammatory cells. During orthodontic tooth movement, these mediators can be directly produced by local cells or by inflammatory cells in response to mechanical stimulation, or indirectly by cytokines. For example, TNF-α is a potent stimulator of PGE2 formation [6]. Prostaglandins act locally at the site of generation and then decay spontaneously or are enzymatically destroyed [7, 8]. Similar to PGs, neuropeptides can participate in many stages of the inflammatory response to orthodontic forces. Neuropeptides are small proteins, such as substance P, that transmit pain signals, regulate vessel tone and modulate vascular permeability [9].The importance of all these

inflammatory makers can be appreciated in the role that they play in osteoclastogenesis.

Osteoclastogenesis

As previously discussed, osteoclasts are multinucleated giant cells that resorb bone and are derived from hematopoietic stem cells of the monocyte-macrophage lineage. After recruitment to the compression sites, osteoclast precursors begin to differentiate into osteoclasts . Cytokines are important mediators of this process. For example, TNF-α and IL-1 bind to their receptors, TNFRII [10] and IL-1R [11], respectively, and directly stimulate osteoclast formation from precursor cells and osteoclast activation .

Additionally, IL-1 and IL-6 [12] can indirectly stimulate local cells or inflammatory cells to express M-CSF (macrophage colony-stimulating factor) and RANKL (receptor activator of nuclear factor κ B ligand). These ligands, through cell-to cell interactions bind to their respective receptors, c-Fms and RANK, which are both expressed on the surface of osteoclast precursors .

Other inflammatory mediators that enhance osteoclast formation through enhancing RANKL expression by stromal cells are PGs, especially PGE2 [13]. As mentioned before, PGs can be produced by local cells directly in response to orthodontic forces or indirectly as down-stream of cytokines such as TNF α It should be emphasized that local cells normally try to down regulate osteoclastogenesis by producing a RANKL decoy receptor, osteoprotegerin (OPG), [14]. Therefore, OPG levels in compression sites should decrease to enable tooth movement.

Stimulation of cytokines to increase the rate of tooth movement

As previously discussed, orthodontic force induces an aseptic inflammatory response [15], during which many cytokines and chemokines are activated and play a significant role in osteoclastogenesis. The importance of these molecules in controlling the rate of tooth movement can be appreciated through the dramatic results obtained from studies that block their effects.

For example, injections of IL-1 receptor antagonist (IL-1Ra) or TNF-α receptor antagonist (sTNF-α-RI) result in a 50% reduction in the rate of tooth movement [16].

Similarly, tooth movement in TNF type II receptor-deficient mice is reduced compared to wild type mice [17]. Animals that are deficient in CC chemokine receptor 2 (CCR2), which is a receptor for CCL2, or animals that are deficient in CCL3, demonstrate a significant reduction in orthodontic tooth movement and number of osteoclasts [2]. Likewise, non-steroidal anti-inflammatory drugs (NSAIDs) reduce the rate of tooth movement by inhibiting PG synthesis [18]. Inhibiting other derivatives of arachidonic acid, such as leukotrienes, also significantly decreases the rate of tooth movement [19].

Systemic application of misoprostol, a PGE1 analog, to rats undergoing tooth movement for 2 weeks, significantly increases the rate of tooth movement [20]. Similarly, local injection of other arachidonic acid derivatives, such as thromboxane and prostacyclin [21], increases the rate of tooth movement.

Effect of vitamin D3 on tooth movement:

Vitamin D3 has also attracted the attention of scientists 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 calcium homeostasis with calcitonin and parathyroid hormone (PTH). Another set of investigators [13] conducted an experiment where they have injected vitamin D metabolite on the PDL of cats for several weeks; it was found that vitamin D had accelerated tooth movement at 60% more than the control group due to the increase of osteoclasts on the pressure site as detected histological. A comparison between local injection of vitamin D and PGEs on two different groups of rats was also done. It was found that there is no significant difference in acceleration between the two groups. However, the number of osteoclasts on the pressure side which was injected by vitamin D was greater than on the PGE2 side. This indicates that vitamin D may be more effective in bone turnover.

PTH effect on tooth movement: PTH has shown to accelerate orthodontic tooth movement in rats, which was studied by continuous infusion of PTH (1

to 10µg/100g of body weight/day) implantation in the dorsocervical region which made the molars move 2 to 3 fold faster mesially by orthodontic coil spring [14] Some 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 also confirmed that a slowrelease 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 acceleration.

The effects of low intensity pulsed ultrasound on the rate of orthodontic tooth movement

LIPUS (30–100 mW/cm2) is a **form of mechanical energy** that is transmitted through living tissues as acoustic pressure waves, resulting in biochemical changes at the cellular and molecular levels.

These biochemical changes may have several therapeutic benefits, one of which includes an increase in rate of soft and hard tissue healing.

Since LIPUS can improve the rate of bone healing after trauma, a number of studies have tried to understand its biostimulatory effects, especially in regard to the osteoblastic and osteoclastic responses.It has been reported that in response to LIPUS, there is upregulation of osteogenic markers, such as IL8, bFGF, VEGF, TGF-β, alkaline phosphatase , and non-collagenous bone proteins, as well as concomitant down regulation of the osteoclastic markers

LIPUS can be used in orthodontics to reduce the risk of root resorption, increase the rate of tooth movement, or modify mandibular growth. In addition, LIPUS promotes the proliferation of cells in PDL and alveolar bone, which improves the quality of the periodontium and reduces the possibility of relapse after orthodontic treatment.

Regional acceleratory phenomenon

This phenomenon is introduced by Frost in 1983.

Traumatic or surgical wounding usually results in intense but localized modeling and remodeling responses. After an osteotomy or placement of an endosseous implant, callus formation and resorption of necrotic osseous margins are modeling processes; however, internal replacement of the devitalized cortical bone surrounding these sites is a remodeling activity. In addition, a gradient of localized remodeling disseminates through the bone adjacent to any invasive bone procedure. This process, called *regional acceleratory phenomenon*, is an important aspect of postoperative healing. Orthodontists can take advantage of the intense postoperative modeling and remodeling activity

(1) to position a maxilla orthopedically with headgear, occlusal biteplates, or cervical support within a few weeks after a LeFort osteotomy and

(2) to finish orthodontic alignment of the dentition rapidly after orthognathic surgery.

Micro-osteoperforations: minimally invasive accelerated tooth movement

THE novel method to accelerate tooth movement is based on the natural inflammatory response of the body to physical trauma.

A study done by Alikhani et aL hypothesized that controlled micro-trauma in the form of micro-osteoperforations (MOPs; which maintain the integrity and architecture of hard and soft tissue) **will amplify the expression of inflammatory markers that are normally expressed during orthodontic treatment** and that this amplified response will **accelerate both bone resorption and tooth movement.**

MOPs significantly stimulated expression of inflammatory markers and significantly increased the number of osteoclasts and bone resorption,

Advantages-

-Less time in fixed appliances reduces the risk for external apical root resorption

and demineralization/caries ;

-patient burn-out is less likely

This procedure is minimally invasive and flapless, allowing orthodontists to deliver care in their offices.

Pain and external apical root resorption

Two main concerns about MOPs are pain and root resorption. MOPs are done under infiltration of local anesthetic.

1. **PAIN-**Patients that receive MOPs did not demonstrate additional pain or discomfort when compared with patients that receive only orthodontic treatment and do not require additional pain medications or additional care other than regular oral hygiene.

2.**External apical root resorption (EARR)** is not increased following MOPs treatment. One main reason for EARR is high stress that produces a cell free zone when a tooth is pushed towards dense bone. In these areas, osteoclasts are recruited from the surrounding PDL and endosteal surfaces. The prolonged presence of osteoclasts, rather than the number of osteoclasts, causes EARR.

While MOPs significantly increased the number of osteoclasts, these osteoclasts are on the adjacent endosteal bone surface not in the PDL (data not shown). Moreover, since MOPs decreases the density of the adjacent alveolar bone, the cell free zone is smaller and cleared faster, which would prevent prolonged osteoclast activity adjacent to tooth roots.

Thus, **EARR risk decreases significantly in MOPs treatment, even during tooth movement over long distances.**

Clinical applications

MOPs can easily be incorporated into our orthodontic mechanics. Application of MOPs during leveling and aligning stages should be postponed until adequate space has been created. While MOPs can increase the number of osteoclasts, it will not change the side effects of the biomechanics plan and therefore similar to classic mechanics, the teeth without adequate space will not be able to engage in the main archwire.

MOPs can facilitate one of the most difficult movements to accomplish in orthodontics, root movement. By activating osteoclasts and decreasing the bone density, application of similar bodily movement mechanics can produce faster tooth movement and less stress on anchor teeth, since movement occurs in less time.

For these reasons, MOPs are an excellent adjunct technique during protraction/retraction of a single tooth or group of teeth.

MOPs between the roots of teeth decreases the bone density while the bone density around anchor teeth remains unchanged.

This procedure is especially useful when a tooth is moved into an edentulous space where alveolar bone is dense with a narrow ridge.

MOPs can significantly decrease the bone density and allow faster and safer tooth movement while enhancing alveolar bone remodeling in that area.

MOPs should also be considered during segmental intrusion, during which there is a possibility of root resorption due to high stress area around the root apex.

While keeping the force light, MOPs application around the apex prevents the prolonged cell-free zone that can cause root resorption. Clinicians should take into consideration that since the increase in cytokine activity decreases after two months of MOPs application, repeating the procedure every other month is recommended.

And if TADs are being used to increase anchorage, application of MOPs adjacent to the location of the TADs should be avoided since decreased bone density around the TADs will likely decrease their stability.

PIEZOCISION ASSISTED ORTHODONTICS: PAST, PRESENT AND FUTURE

It has evolved from being initially a minimally invasive surgical alternative to conventional corticotomies to a more sophisticated “intellectual” approach to comprehensive orthodontic and multidisciplinary care.

A minimally invasive surgical procedure aimed to accelerate orthodontic tooth movement. A tool given to the orthodontist to control anchorage. A useful complement to treatment with clear aligners. A way to modify/strengthen a patient’s thin biotype.

Piezocision™ can be used in a generalized localized or sequential manner.

The Technique

Piezocision™ is performed one week after the placement of orthodontic appliances (fixed or removable). -A small vertical incision is made buccally and interproximally.This mid-level incision, between the roots of the teeth will allow for the insertion of the piezoelectric knife.

Piezocision™ has a localized and selective effect on the teeth, only the teeth or arch(es) to be moved need to be operated upon. The areas not surgerized have a higher anchorage value, since they are not affected by the demineralization process following Piezocision™ and can be used as such in the global treatment plan.

The tip of the Piezotome (Satelec, Acteongroup, Merignac France) is inserted in the gingival openings previously made and a 3 mm deep piezoelectrical corticotomy is done. The decortication has to pass the cortical layer and reach the medullary bone to get the full effect of the regional acceleratory phenomenon (RAP).

In the areas with thin or little gingiva (recessions) or with thin or no cortical buccal bone (dehiscences, fenestrations), hard and/or soft tissue grafts can be added via a tunneling procedure. The patient is seen every one or two weeks after surgery by the orthodontist in order to change aligners or activate wires and take advantage of the temporary demineralization phase created by Piezocision™. This results in faster tooth movement and early completion of treatment.

How Does It Work?

When the bone is injured, a very dynamic healing process occurs at the site of the bone injury. This process is called the regional acceleratory phenomenon (RAP), which is proportional to the extent of the surgical insult, There is a localized surge in osteoclastic and osteoblastic activities which results, in the early phases, in a decrease in bone density with an increased bone turnover.

This transient osteoporotic condition facilitates tooth movement.

Piezocision and the use of the piezotome at specific vibration frequency settings appeared to induce a more extensive and diffuse demineralization followed by increased remineralization effect on the bone then the bur.

This could be due to the additive effect of the osteocytes response to micro-vibrations created by the ultrasonic handpiece at specific settings.

Once the bone has demineralized following bur corticotomy, there is a three- to four-month window of opportunity to move teeth rapidly through the demineralized bone matrix before the alveolar bone remineralizes.

The effect of Piezocision™ on the length of this window of opportunity are being investigated. Our clinical experience indicates that this RAP could last up to 6 months. This is best observed in our daily practice by patients treated with Piezocision™-assisted Invisalign where the patient changes the aligners every 5 days instead of every two weeks. After the 5th or 6th month of treatment, the tooth movement appears to slow down.

Reduction in Treatment Time and More…

Piezocision™ can now be defined as another tool for creating differential anchorage. Since it has been shown that the density of the bone around the Piezocision™ cut is less, the anchorage values of the teeth at the decortication site would be different.

Piezocision™ can be done selectively around the teeth that are going to be moved and the anchorage values of these teeth can be decreased. Therefore the need for additional anchorage devices can be eliminated by designing the alveolar decortication according to the desired tooth movements.

Piezocision™ can be repeated more than once in the same area(s) to re-activate the RAP[22] (after 5-6 months) and keep the area demineralized (depending on the difficulty of the movements being performed and the morphology of the patient's bone) .

Periodontal Biotype Modifications

The orthodontic treatment of teeth beyond the limits of the labial alveolar plate can lead to dehiscence formation and predispose the patient to recession.

Hard and/or soft tissue grafting can prevent such disastrous outcomes or correct an existing recession. This is done via a tunneling procedure without the need of a conventional flap elevation.

Clinical Applications

Piezocision™ can be used in a generalized, localized or sequential manner.

• Generalized: If the correction of the malocclusion requires moving all of the teeth in both maxilla and mandible at the same time.

• Localized: If the malocclusion affects only one part of the dentition or one arch (i.e. An anterior crowding case with a perfect posterior occlusion, single tooth extrusions intrusions, etc.).

• Sequential: If the correction of the malocclusion requires a "staged" approach, where selected areas or segment of the arch are being demineralized at different times during orthodontic treatment to help achieve specific results

Indications :

- Class I malocclusions with moderate to severe crowding (extraction and non -extraction),
- selected Class II malocclusions (end-on),
- selected Class III malocclusions (dental),
- correction of deep bite,
- correction of open bite,
- distalization of molars,
- palatal version of root apices,
- rapid adult orthodontic treatment,
- orthodontic treatment with clear aligners,
- rapid intrusion and extrusion of teeth,
- prevention or simultaneous correction of osseous and mucogingival defects that may occur during or after orthodontic treatment, multidisciplinary treatments.

Contra-Indications:

- Medically compromised patients,
- patients taking drugs modifying normal bone physiology (i.e. biphosphonates),
- any bone pathology, ankylosed teeth, non-compliant patients,
- patient and/or operator having a pacemaker or any other active implant

Future directions

Piezocision™ may offer a substitute for complete corticotomies in less severe cases requiring surgically-assisted rapid palatal expansion.

It helps in distalization of molars, intrusion of molars, correction of open bites, may provide a mechanism to treat cases of alveolar insufficiency and certain class III malocclusions can successfully be treated in this "unconventional" manner.

Periodontally accelerated osteogenic orthodontics therapy

Alveolar decortication with augmentation bone grafting technique combined with orthodontics is called **periodontally accelerated osteogenic orthodontics or PAOO** and was first described by **Wilcko et al**.

- This surgical technique has been sufficiently described and the main features include full thickness periosteal flap reflection followed by intentionally scaring or scoring the alveolar cortical bone, both labial and lingual, in the area of desired tooth movement .

-Bone grafting is placed at the corticotomy sites and is typically comprised of a mixture of demineralized freeze dried bone allograft (DFDBA) and bovine bone.

-The surgical flap is sutured, and the patient is seen every 2 weeks after the surgery for orthodontic adjustments. It is recommended that the surgical procedure be performed within 1 week of fixed orthodontic appliance placement and that the appliance be activated at the time of surgery.

PAOO accelerates orthodontic tooth movement and malocclusions are resolved 3–4 times faster; treatment times for complex cases average 6 months compared to 18–27 months.

Orthodontic tooth movement limits

Proffit's "envelope of discrepancy," describing limits of orthodontic treatment in the context of producing normal, stable occlusion, has been cited by most authors exploring orthodontic treatment limits . These limits when applied to the non-growing individual vary by the tooth movement that would be needed if other restraints, such as soft tissue limits, do not apply. According to Proffit's opinion, teeth can be moved further in some directions than others ; the range described is 7mm (maxillary incisor retraction) in the anteroposterior spatial plane to 2 mm (maxillary incisor protraction and some incisor vertical dimension changes).

While limits of tooth movement are a matter of clinical experience and opinion and are not based in scientific fact ,in general, **PAOO treatment limits for incisors exceed the envelope of discrepancy described by Proffit for adult , nongrowing patients by 2–3 times in all dimension except retraction.**

Corticotomy surgery is recommended within 1week of fixed orthodontic appliance placement ; alignment of incisors is rapid after surgery,but the time it takes to align incisors encroaches upon post corticotomy healing.

Retraction movement is usually constrained because RAP has dissipated and healing has advanced ahead of space closure precluding accelerated retraction. The combination of corticotomy and dental distraction is recommended to facilitate retraction tooth movement.

-Corticotomy surgery increases the soft and hard tissue turnover, augmentation bone grafting expands the bony alveolus and both processes expand hard and soft tissue limits and enable teeth to be moved a greater distance.

-The augmentation bone grafting of PAOO increases the alveolus size and expands the soft tissue envelope gradually with much less constraint from the craniofacial muscles.

The accelerated tooth movement process involves a demineralization of alveolar bone adjacent to the corticotomy bone matrix transport to a new position secondary to orthodontic biomechanical forces, and remineralization of the alveolus after active tooth movement has been completed.

It is surmised that the remarkable stability of PAOO treatment outcomes is the consequence of increased tissue turnover and increased thickness of cortical bone in conjunction with low constraint from preexisting soft tissues and muscles

Large changes in the alveolus housing the dentition are very amenable to PAOO technique. Hence, **PAOO is applicable to the most moderate to severe malocclusion conditions because soft tissue constraints are reduced and the limits of the alveolar housing are increased.**

Moderate to severe malocclusions amenable to PAOO therapy are problems requiring alveolar extrusion or intrusion, transverse alveolar expansion (orthodontists rarely constrict), and alveolar protrusion.

PAOO cannot extend the limits of alveolar retraction because this tooth movement in severe malocclusion is typically defined by the skeletal position of the jaw in the anteroposterior dimension.

For conditions of microganthia or macrognathia accompanied by excess or deficient overjet, only orthognathic surgery is applicable because PAOO does not have the capacity to move jaws in the anteroposterior spatial plane.

The benefits of PAOO therapy include the following:

(1) increase limits of tooth movement created by augmentation bone grafting
(2) enhanced stability of orthodontic treatment outcomes,[23,24]
(3) rapid orthodontics for severe malocclusion averaging 6–8 months of active treatment time,[25] and
(4) increased robustness of the periodontium.

CYCLIC LOADING (VIBRATION)

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 include low-energy laser irradiation,[26] magnetic fields,[27] as well as

pharmacological interventions with the injection of prostaglandin E_2 [27] and vitamin D.[28] However, adverse events, such as local pain and severe root resorption,[29] were associated with these treatments.

Low level mechanical oscillatory signals (vibrations) have been shown to increase the rate of remodeling in mechanical loaded long bones, which is currently used in the prevention of osteoporosis based on an increase in bone metabolism and decrease in bone loss in postmenopausal women.

• **Nishimura et al in 2008**, used a Ni-Ti expansion spring on the 1st molar of Wistar rats, and applied a vibration of 60 Hz, 1 m/s2. They stated that the rats that received the vibration showed increased orthodontic tooth movement.[30]

• **Liu et al in 2009** conducted a study on thirty mice, in which they used an omega shaped Ni-Ti expander to deliver a force of 20 g on the 1st molar. Mechanical vibration (4 Hz for 20 min/day) was applied perpendicular to the occlusal surface of the first molar. This regimen was repeated seven times, every 3 days. Upon micro-CT examination of the jaws of the killed mice, it showed that the mice that received vibration showed 40% more tooth movement.[31]

Acceledent which makes use of this technology. This device consists of an activator, which is the active part of the appliance that delivers the vibration impulses 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 up to 30-40%.

A new orthodontic company "OrthoAccel" founded in 2007 brought his brand generation of dental vibrator named "AcceleDent" into the market in 2009.

Kau et al conducted a clinical trial in which 14 orthodontic patients were recruited and instructed to use the device for 20 minutes daily for a period of 6 consecutive months.

As a result, it was found that the total rate of movement for the mandibular crowding was 2.1 mm per month and for the maxillary arch was 3.0 mm per month, which apparently is faster than the traditional finding as of about 1.0 mm per month.

The patient compliance was 67% with good patient perception. It was thus concluded that the **AcceleDent device is a useful adjunct to orthodontic treatment. If used appropriately, it can accelerate routine orthodontic tooth movement.**

According to the manufacturer, AcceleDent is a simple, removable dental device that patients need to use between the teeth for twenty minutes daily. This device can be used with any type of appliance, such as fixed braces and/or clear aligners. If proven efficacious, we may face a revolution in the orthodontic arena

Photobiostimulation

Einstein first described the manipulation of light in 1917 with the concept of laser ("Light Amplification by Stimulated Emission of Radiation").

The ability of light to interact with tissues is based on the wavelength being used and the incident photons in the beam of light. Of interest to us are the non-thermal effects, termed collectively as *photobiostimulation,* produced by the application of relatively low levels of irradiation from monochromatic light to tissues.

DEFINITION-Used interchangeably with terms such as biostimulation, photostimulation, and biophotomodulation, photobiostimulation refers to the alterations in chemical, physical, and metabolic processes and any associated downstream effects when energy is absorbed by a target tissue with little or no temperature change.

Photobiostimulation has been widely used in medicine and dentistry to reduce pain and inflammation, accelerate wound healing and hair growth, prevent cell death and tissue damage, improve blood circulation, and during orthodontic tooth movement (OTM).

Photobiostimulation Modalities

Photobiostimulation may be therapeutically applied in the form of lasers (lowlevel/intensity/energy laser therapy or LLLT) and light emitting diodes (LEDs). Both types of applications utilize a near-infrared wavelength of approximately 600-1000 nm, with a range of 730-850 nm being viewed as most appropriate for photobiostimulatory effects. The relatively narrow wavelength range allows for greater absorption by target tissues.

Cellular and Molecular Mechanisms of Photobiostimulation-

- Increased cellular metabolism has been proposed to be the major photobiostimulatoryeffect of infrared light.
- Photobiostimulation has been proposed to stimulate mitochondrial cytochrome c oxidase, the primary photo-acceptor in the wavelength range of 600-950 nm, resulting in increased cell metabolism (via ATP, cAMP and Ca2+).
- Another proposed mechanism is the **"singlet-oxygen hypothesis"**, where molecules like porphyrins and some flavoproteins can become activated and interact with oxygen to create reactive singlet oxygen that removes energy deficits and increases overall cell metabolism.
- A complementing hypothesis is the **"redox properties alteration hypothesis"** where photobiostimulation changes the overall cell redox potential towards that of greater oxidation, thereby encouraging cells with a lower resting intracellular state towards an up-regulation of transcription factors.

Finally, it has been proposed that **photobiostimulation can directly augment RNA and DNA synthesis and replication, protein synthesis and overall cell metabolism by increases in Na+/K+ pump activity and intracellular Ca2+.**

CONCLUSION

Orthodontics is not a single stage mechanical approach where a clinician could merely use one arch wire throughout the treatment and count on acceleration techniques to shorten the time needed to establish an ideal,stable occlusion. . Orthodontic treatment consists of many stages , and at each stage we need to target some teeth without moving the others. Some movements are prerequisites for others; for example one should not engage a blocked tooth to the main arch wire until enough space is created.

Application of acceleration technique around a blocked tooth does not change the forces and movements required to produce a desired type of tooth movement and therefore should not be applied until the tooth from a mechanical standpoint is ready to be moved.

Since each stage of treatment has its specific target teeth, acceleration techniques should be simple, inexpensive and comfortable enough to be repeated as often as needed throughout the course of treatment.

CONFLICTS OF INTEREST

The authors declare they have no potential conflict of interests regarding this article.

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