ACCELERATED ORTHODONTIC TOOTH MOVEMENT

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Index:

INTRODUCTION

Orthodontic treatment involves the movement of teeth through alveolar bone using forces that results in a biological reaction within the dentoalveolar tissues. Hence, orthodontics is characterised as bone manipulation therapy and the biomechanical manipulation of bone is the physiologic basis of orthodontic and dento-facial orthopaedics. The remodelling of bone following injury or stimulus involves a complex array of interwoven processes and these ultimately determine the rate of tooth movement. Hence, there is a limitation to the rapidity at which orthodontic treatment can be completed without adverse effects. Limitations of traditional orthodontic techniques and the length of requisite treatment times often result in difficulties for providers and create barriers to patient willingness to accept orthodontic care. Several adjunctive treatment modalities have been examined in order to accelerate orthodontic tooth movement.

Most attempts can broadly be categorised into biological, physical, biomechanical, and surgical approaches. Experiments have been done using various molecules such as PGE, vitamin D3,cytokines exogenously to enhance tooth movement both in animal experiments and humans. Another approach in accelerating tooth movement is by using device-assisted therapy. This technique includes direct electric currents, pulsed electromagnetic field, static magnetic field, resonance vibration and low-level laser.

Furthermore, clinically most effective technique used for adult patients, where duration of orthodontic treatment is critical in selected group of patients, are surgical approaches. Several surgical approaches have been tried in order to accelerate tooth movement such as corticotomy, selective alveolar decortication, osteotomy, accelerated osteogenic orthodontic tooth movement(AOOO),periodontally accelerated osteogenic orthodontic tooth movement(PAOO) Piezocision technique.

A reduction in active treatment time would logically mean that teeth would need to move faster. It is well established that tooth movement rate is, in part, a function of alveolar bone density(Verna *et al.*, 2000), and that tooth movement is accelerated under conditions of low bone density. Bone densities change regularly as bone renews itself. Roberts *et al.* (2004) contrasted the process of bone renewal for cortical and trabecular bone; cortical bone requires an activation that initiates resorption (cutting cones) followed by formation (filling cones) in a couple sequence; that is, remodelling, otherwise known as secondary osteon formation. Trabecular bone is thin, which sustains a simpler process; that is, modelling, wherein stimulus activation can result in either apposition or resorption. The milieu of orthodontic tooth movement is trabecular bone modelling with the exception of the thin cortical lamina dura surrounding each tooth root.

Orthodontist understand that tooth movement results from alveolar bone resorption and formation. Application of a biomechanical force results in a shift in cell population dynamics within the periodontal ligament (PDL) till sufficient osteoclasts and osteoblasts have accumulated within the PDL.

Tooth movement after initial force application is limited to the width of the PDL space; this 3–5-week “lag” phase dissipates as PDL cell populations supportive of tooth movement have accumulated and hyalinization has diminished (Von Böhl and Kuijpers-Jagtman, 2009).

Orthodontic researchers understand “bone turnover” as a phrase describing the dynamics of a living osseous tissue that by nature is compensatory and adaptive. The research methods for the study of bone turnover include histomorphometry; that is, quantitative analysis of the physical size and shape (form) of bone. “Bone turnover” is a histomorphometric expression that includes anabolic (apposition) and catabolic (resorption) bone changes that occur and an appraisal of the varying degrees and relative amounts of mineral salts, namely calcium etc.

Alveolar demineralization or decreased alveolar bone density leads to increased tooth movement rate (Verna *et al.*, 2000). Reduction of the availability of calcium metabolite (i.e., calcium manipulation either pharmacologically or through diet), results in greater tooth movement rate and scope (Midgett *et al.*, 1981; Goldie and King, 1984; Engström *et al.*, 1988;Verna and Melsen, 2003).

Additionally , extended treatment duration can be associated with increased risks of root resorption, periodontal disease, caries and most crucial , change or loss of patient motivation.(Mizrahi, 2010; Krishnan et al 2007).Improvements in treatment efficiency can help to alleviate these challenges.

This literature will be divided into 3 subsections. It will begin with some of the basic principles of orthodontic tooth movement at the cellular, molecular, device-assisted and clinical levels.

BIOLOGICAL APPROACH TO ACCELERATE TOOTH MOVEMENNT

Biology of orthodontic tooth movement

Orthodontic tooth movement is a mechanically mediated inflammatory process. The application of orthodontic forces, which can be considered to be static and therapeutic, results in a response within the alveolar process. The mechanical distortion of cell membranes results in activation of phospholipase A2, making arachidonic acid available for the action of cyclooxygenase and lipoxygenase enzymes, such as prostaglandin E and prostacyclin (PGI2). The prostaglandins bind to cell membrane receptors, resulting in the stimulation of secondary messengers, which leads to a cell response. This control process affects cells along the bone surface, as well as osteocytes that are situated in a rigid matrix. Osteocytes are mechanosensory cells that are able to translate mechanical strain into biochemical signals that regulate bone modelling and remodelling. These cells are ideally positioned to detect changes in mechanical stresses and to relay signals to surface lining osteoblasts, which progress to bone formation and bone resorption. Prostaglandin EP2 receptors are involved because signals are transmitted across gap junctions between osteocyte processes. This activates the cAMP-protein kinase pathway, which has been implicated in tooth movement. Other secondary messengers, such as inositol phosphate and intracellular calcium, are also involved and their activation will evoke a nuclear response, which will either result in production of factors involved in osteoclast recruitment and activation, or bone forming growth factors. Thus, the stimulation of osteocytes supports osteoclast formation and activation. Osteoblasts have receptors for many of the hormones and growth factors that stimulate bone modelling and remodelling. In contrast, the osteoclast is comparatively insensitive to these signals. Osteoclasts are more responsive to inhibitory signals, such as calcitonin and prostaglandin, which inhibit them from resorbing calcified matrix. Osteoblasts are responsible for the recruitment and activation of osteoclasts when they are stimulated by various hormones. Osteoblasts activate osteoclasts through the OPG/RANK/RANKL regulatory system.

Orthodontic tooth movement involves two interrelated processes: deflection or bending of alveolar bone and remodelling of periodontal tissues which is a factor that distinguishes the remodelling process from typical bone responses. The role of the PDL, which is considered to be an extension of the periosteum, is essential because the PDL is intimately connected to the bundle bone of the alveolus and the cementum of the root. It could be considered that the tension within the fibres of the PDL are transmitted to the adjacent structures, but it has been found that the PDL behaves as a viscoelastic gel that flows and bounces and hence forces are not transferred (38). Additionally, when the PDL is disrupted, orthodontic tooth movement still occurs (39). Another differing factor is that during orthodontics, the forces applied to the teeth are intermittent, rather than constant. This is due to the role of the occlusion, which causes a ‘jiggling’ effect as teeth come into occlusal contact.

During tooth movement, should the orthodontic force be too great, the pressure in the PDL becomes too high, resulting in hyalinization and indirect resorption of boneat a distance from the PDL. The indirect resorption is considered to be an ‘undermining resorption’ starting from the adjacent bone marrow and occurs in the absence of formative activity on the tension side of the tooth. This is because only a minor displacement of the tooth occurs. The periodontal ligament in the region is compressed and hyalinization due to excessive compression and local tissue necrosis may occur.

This hyalinised or necrotic tissue is removed by phagocytic cells when the undermining resorption reaches the PDL. At this point, the tooth begins its displacement and loosens due to the widened PDL. Once tooth movement commences, bone apposition occurs at the tension side, followed by either renewed hyalinization or a continuation of the tooth movement through direct resorption of the alveolar wall. With no compensatory apposition of bone, there is a net loss of bone and the tooth may be moved outside the alveolar process without bone coverage. The determining factor for the bone response to tooth movement is the stress/strain distribution in the PDL, which is modified by the magnitude of force, bone area, and type of tooth movement. The amount of force and the stress/strain distribution determines the biological reaction and, using Frost’s mechanostat theory, it was considered that the direct resorption of bone could be due to lowering of the normal strain from the functioning periodontal ligament, resulting in increased remodelling space. Physiological loads would balance the resorption and formation, producing new lamellar bone. Excessive strains would result in a negative balance as repair would not be able to keep up with the occurrence of microfractures and may produce woven bone. This would result from excessive loads that would be considered to be traumatic.

Frost described the presence of woven bone as a response seen when the stimulus exceeded a certain value, below which lamellar bone was formed. He also determined that 1500-3000 µm is a typical minimum effective strain for lamellar bone to start modelling, and that if the strain is below 100-300 µm, remodelling is activated as a result of inactivity.

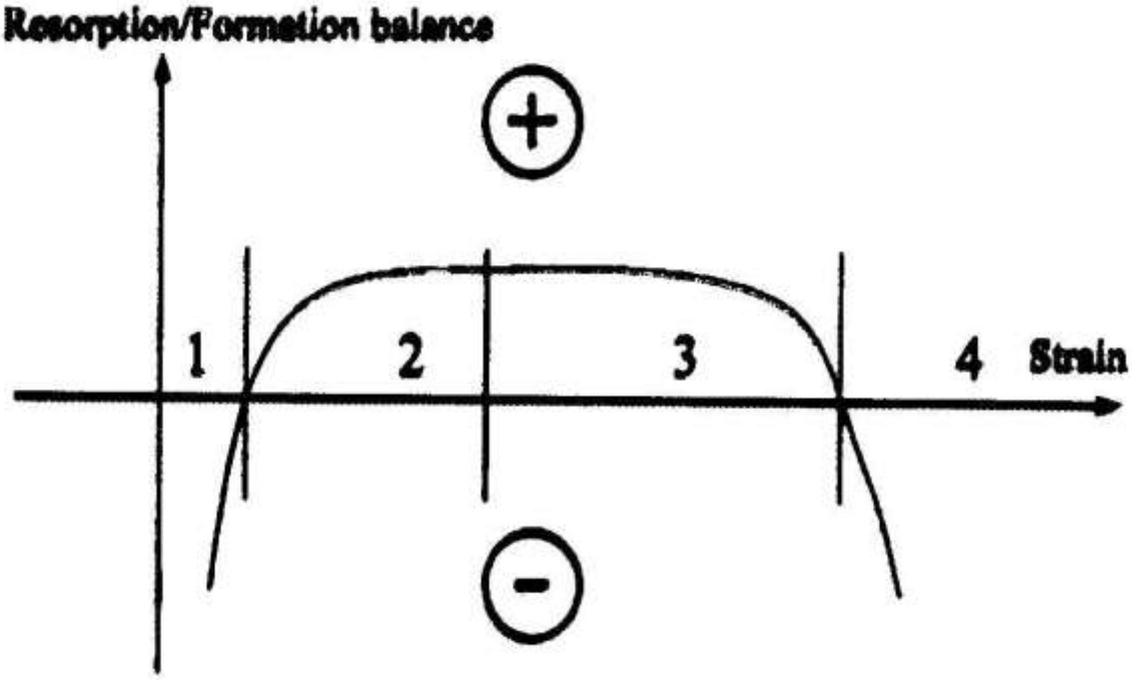


Figure 1: Graphical illustration of bone biological reaction to variation in strain values. In the case of low strain values, remodelling is turned on and a negative balance will be the result. With higher strain values, modelling is turned on and formation of lamellar bone occurs. Further increase in strain results in the formation of woven bone. Severe overloading results in negative balance due to the repair process related to the microfractures occurring at the strain level.(From Melsen).

Melsen in 2001 proposed a new paradigm for tissue reactions to orthodontic tooth movement. This aimed at overcoming the conflicting beliefs between groups:
orthodontists generated resorption by applying pressure whilst orthopaedic surgeons caused bone apposition through loading. The main argument for consistency between the two conflicting views was based upon a study by Epker and Frost, which showed that the stretching of the PDL resulted in a ‘bending of the bone’ in the tension zone and hence that the apposition of the alveolar wall could be considered to be a reaction to bending. In the monkey model, Melsen found that strain values in the direction of displacement (or the ‘compression’ zone) were below the minimum effective strain. This was due to compression of the PDL and thus would cause underload remodelling, accounting for the direct resorption on the ‘compression’ side.

On the ‘tension’ side, the PDL fibres were stretched, generating a strain level corresponding to modelling, thus causing new bone formation. Additionally, woven bone was seen ahead of the alveolus in the direction of the displacement.

Melsen also found that the PDL fibres were stretched and that formation activity was found along the major part of the alveolus, leading to the delivery of strain values corresponding to modelling. As it was an intrusive movement, the apical fibres were not stretched and hence the apical bone resorption was thus interpreted as remodelling.

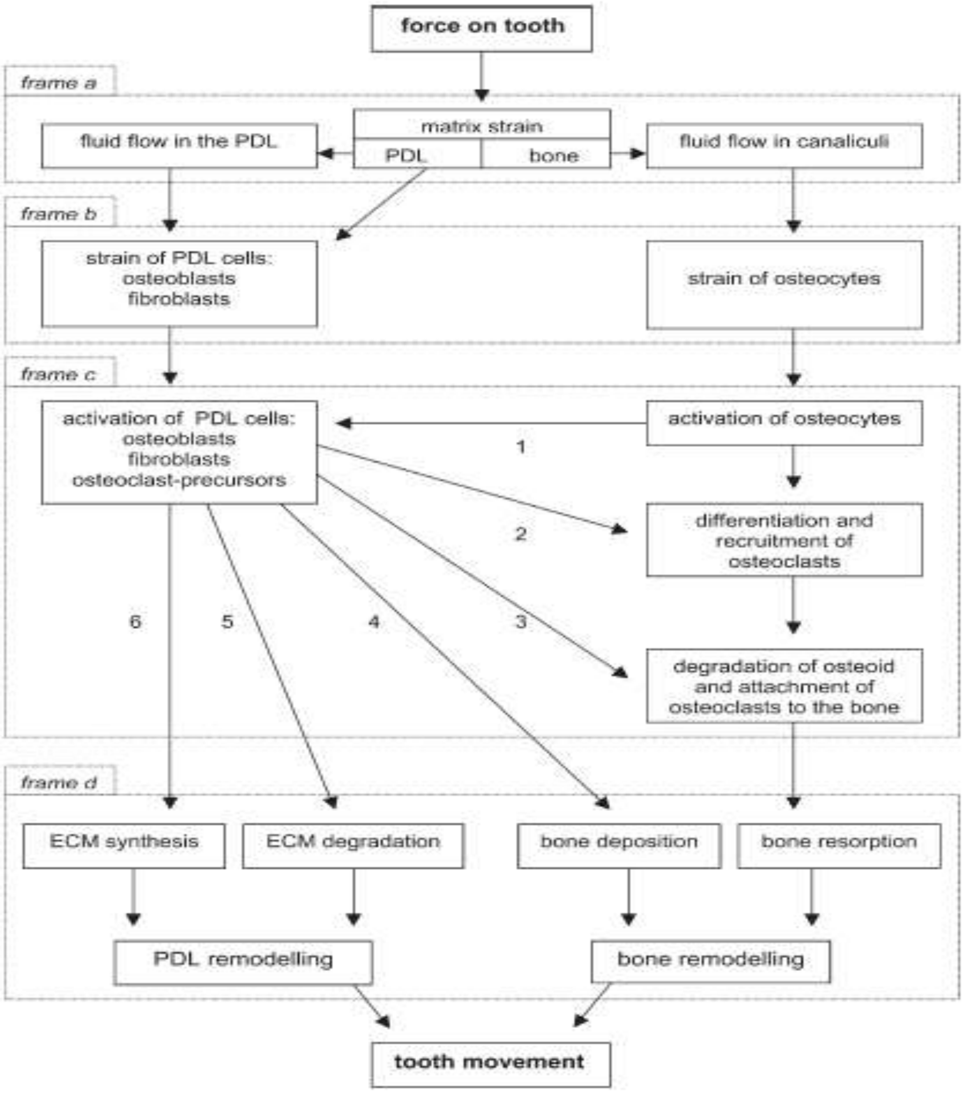


Figure 2: A theoretical model of tooth movement. The model describes four different stages in the induction of tooth movement. Frame (a) represents matrix strain and fluid low, (b) cell strain, (c) cell activation and differentiation, and (d) remodelling of the periodontal ligament (PDL) and bone. From Henneman et al.

Henneman et al. presented a theoretical model that involves four stages in the induction of tooth movement (Figure 2). Immediately after the application of a force, the tooth is able to shift a small distance within its socket. This results in a negative strain (compressive deformation) within the PDL on the future resorption side of the root, relaxing the collagen fibres. Conversely, there is positive strain (tensional deformation) within the PDL on the future apposition side of the root, with stretching of collagen fibres connecting the tooth to bone.

This is depicted in the following picture:

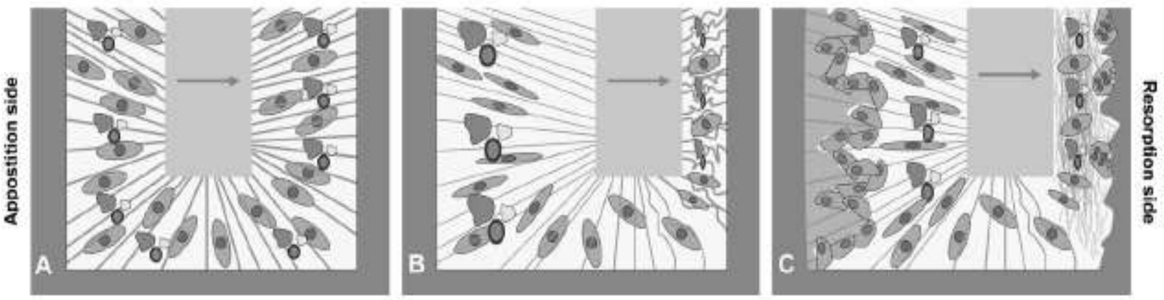


Figure: Schematic drawing of a tooth, the periodontal ligament with cells, and alveolar bone. (a) An external force is applied (arrow). (b) At the apposition side, fibres are stretched. Compression of fibres takes place at the resorption side. (c) After prolonged application of force, bone formation by osteoblasts can be found at the apposition side and osteoclasts resorb the bone at the resorption side. From Henneman et al.

The application of an external force on the tooth causes fluid flow in the PDL and in the canaliculi, leading to shear stress on osteocytes. This can result in apoptosis of osteocytes, subsequently leading to the attraction of osteoclasts. Additionally, microcracks occur in bone as a result of material fatigue, which may provide further stimulus to apoptosis and attraction. Verna et al. found that there were increased numbers of microcracks present at the resorption side during the initial phase of orthodontic tooth movement in pigs. These cracks reflect areas of initial damage of the bone that will be remodelled at later stages. Following the stages of cell deformation and activation, osteocytes produce specific cytokines (such as prostaglandins and TNF-alpha) that activate osteoclast precursors in the PDL side, while nitric oxide inhibits osteoclast activity at the apposition side. The remodelling process is induced through the complex regulatory network of fibroblasts, osteoblasts, and osteocytes, resulting in tooth movement. At the resorption face, osteoclast precursors migrate to the bone surface and differentiate into osteoclasts. Activated osteoclasts dissolve the inorganic and organic matrix, creating space for tooth movement and removing the attachment of the principal fibres of the PDL to bone. At the apposition side, the PDL fibres are stretched, stimulating osteoblasts to produce new extracellular matrix and to mineralise the osteoid.

The new bone is thickened, trapping osteocytes and PDL, forming new Sharpey’s fibres. The cycle continues until the removal of the orthodontic force. Thus, the rate of tooth movement is dependent upon the rate of bone turnover and, as normal bone remodelling would proceed at too slow a rate to allow efficient tooth movement, a phenomenon to increase the rate of remodelling is an essential factorin orthodontics. Its absence would make it impossible to perform tooth movement, significantly increasing overall treatment times beyond the lifespan of the patient.

Regional Acceleratory Phenomenon

The regional acceleratory phenomenon (RAP) was first described by Frost and is defined as ‘a complex reaction of mammalian tissues to diverse noxious stimuli’.RAP is a phenomenon that affects the skeletal and soft tissues in an anatomical area.

Both the stimulated area and surrounding tissues are affected. RAP is characterised by an acceleration of ongoing normal vital processes and may be considered to be a protective mechanism that evolved to potentiate tissue healing and fortify local tissue.

immune reactions. The response in the bone may be considered to be exaggerated or intensified. This is recognisable when the RAP is hindered, because deficient healing, reduced resistance to infection, and mechanical abuse may occur.

Causes

RAP is initiated when a regional noxious stimulus of sufficient magnitude affects the tissues. Frost observed that the size of the affected region and the intensity of its response varied directly with the magnitude of the stimulus, though there was individual variation in the degree of the response. The noxious stimulus can greatly vary in nature and can include any perturbation of bone, including traumatic injuries, fractures, osseous surgery, vascular surgery, crushing injuries, thermal trauma, infections, and most non-infectious, inflammatory joint processes, including rheumatoid arthritis. RAP can also occur by extracting a tooth, raising a periodontal flap, or placing a dental implant.

Nature

Once RAP has been evoked, normal regional hard- and soft-tissue vital processes accelerate above normal response levels. Collectively, these accelerated processes represent the RAP and they include: growth of connective tissue structures, remodelling of connective tissues, skin epithelialisation, soft tissue and bone healing, perfusion, and cellular turnover and metabolism. RAP does not seem to provide new processes but increases the rapidity of healing through all the post fracture stages, including granulation, modelling, and remodelling. This results in healing occurring two to ten times more rapidly than otherwise, which means that additional remodelling cycles of resorption followed by formation are activated. The affected region, as a result of the acceleration of these processes, exhibits inflammatory changes, including erythema and oedema, subsequently increasing temperature and bone turnover. Hot regions found in acute and chronic osseous conditions, including osteomyelitis, healing fractures, joint inflammation, and bone metastases, can be attributed to the uptake of bone-seeking isotopic agents. Frost suggested that the appearance of the cardinal signs of inflammation represented an early recognised manifestation of a stereotyped, more general phenomenon. In a situation involving the fracture of bone, as was being examined by Frost and Lee, the RAP response is divided into phases, with an initial phase of maximally stimulated bone formation in which woven or fibrous bone is produced to span a cortical gap. This new bone is eventually remodelled into lamellar bone. This process is followed by a period of predominant resorption, in which medullary bone disappears and the number of osteoblasts decreases. This decreased regional bone density due to increased modelling space may also lead to regional tissue plasticity. The increased intracortical bone remodelling produces tunnelling within the cortex that can be seen on clinical radiographs. It is postulated that osteoclast and osteoblast cell populations shift in number, resulting in an osteopenic effect. As well as variation due to different causes of RAP, the response will depend upon the anatomy, competence and autonomic innervations of the regional blood supply, regional sensory innervations, and mechanical loading, as well as local biochemical and biological factors known to be associated with injury, repair, metastasis, and inflammation. Frost proposed several mechanisms for RAP, such as a decrease in osteoblast cell number, cell proliferation responses, neovascularization, and local and systemic mediators.

Anatomical distribution

The RAP involves the anatomical region where its stimulus arose, such the periodontium surrounding a tooth during orthodontic tooth movement. The distribution of the RAP reflects the regional vascular anatomy and innervations, allowing transition of the phenomenon from involved to uninvolved regions. This is a gradual change, rather than an immediate or rapid shift. Additionally, it has been observed that with severe stimuli, RAP can occur in contralateral regions of the body.

Duration

Frost estimated the total duration required for the remodelling – activation, resorption, and formation (ARF) – to be 12 weeks. The duration of the RAP depends upon the severity of the stimuli, although in healthy humans, a single traumatic stimulus, such as a gunshot wound, will result in clinical evidence of RAP of approximately four months duration in bone. RAP begins within a few days of the fracture, typically peaks at one to two months, and may take six to more than 24 months to subside. The duration in soft tissues is shorter. With more severe trauma, such as acute paralysis or severe thermal burn, the RAP can last from six months to over two years. Prolonged stimulation, such as that resulting from rheumatoid arthritis, osteomyelitis, Paget’s disease, or osteoid osteoma, can produce a persistent RAP without limit to its duration.

Clinical application of Regional Acceleratory Phenomenon

In normal conditions, less than 5% of the adult human tibial compacta is remodelled annually. Should a fracture occur, and if no other phenomenon modifies the healing response, less than 5% of the tibial fracture interface would bridge within the first year, and complete bridging would require over 20 years of healing time. However, due to trauma as a result of the fracture and also the reparative surgery, the local bone turnover is accelerated ten- to fifty-fold above normal for more than a year. This allows the union to occur within six months and is accompanied by a concurrent acceleration in the healing process of the soft tissues. Conversely, should the RAP fail to promote fracture callus formation sufficiently, a fracture non-union will result (less than 3% of all fractures). Thus, it is suggested that normal fracture healing may routinely require an accompanying RAP and its absence may result in a delayed union. Additionally, according to ‘Wolff’s law’, living bone can modify its internal architecture in response to an alteration in applied mechanical loads. This ensures that the bone architecture is optimally prepared mechanically to support altered loads. Extrapolation of this concept suggests that mechanical usage of a bone can influence its architecture and that the reported bone reactions are typical RAP manifestations. The rate of remodelling, when elevated as a result of the regional acceleratory phenomenon, has been shown to increase the rapidity of tooth movement. Verna et al. investigated the influence of bone metabolism on the rate and the type of orthodontic tooth movement in the rat model. In comparing groups with high, low, and control rates of bone turnover, it was found that the bone turnover significantly affected the rate of tooth movement. As a result, one area of significant interest is increasing the rate of bone turnover through the utilisation of RAP associated with orthognathic surgery. The advent of rigid fixation allows orthodontists to take advantage of the surgically induced RAP to achieve extensive orthodontic tooth movement postoperatively.

Melsen investigated the relationship between the strain levels and biological tissue reaction in monkeys by using closed coil springs to achieve tooth translation. By comparing this with unloaded control teeth, they observed a relative extension of resorption from 3-5% in the control group to 7-13% of the total cancellous surfaces surrounding the loaded teeth. There was also an increase in extension of appositional surfaces from 15-20% in the controls to 35-49% around the loaded teeth. Relative to control teeth, the density in the direction of tooth movement was increased by a factor of 2 to 3. The alveolar wall in the direction of the tooth movement was completely resorbed, while woven bone formation was seen in the alveolar bone ahead of the direction of tooth movement. The extension of affected region and the intensity of the response varied directly with the magnitude and nature of the stimulus. Verna et al. used a rat model to examine the regional effects of orthodontic tooth movement. An orthodontic force was applied to mesialise the maxillary first molar, but bony changes were visible histologically around all the teeth in the region. Verna considered this force to be perceived as a noxious stimulus against which the surrounding bone developed a defensive mechanism. It was concluded that not only the alveolar bone surrounding the alveolar socket of the tooth was affected by the mechanical perturbation, but also the bone that surrounded the adjacent teeth. Verna also argued that because there was new bone formation on the periosteal side at an early stage, followed by further bone formation at a later stage, that the first periosteal response is non-specific whilst the later response is the result of a regional acceleratory phenomenon.

Biological molecules used to accelerate tooth movement

Orthodontics has been developing greatly in achieving the desired results both clinically and technically. This is especially so by using new technologies, like stimulation software that can assist in treatment planning and translational products. In addition, continuous modification of wires and brackets as a result of the biomechanical efficiencies in orthodontics has greatly improved. However, these biomechanical systems may have reached their limit and there is a need to develop new methods to accelerate teeth movement. Today, 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, but still there are a lot of uncertainties and unanswered questions towards most of these techniques. Most attempts can broadly be categorized into biological, physical, biomechanical, and surgical approaches. Before going into details of these attempts, we need to understand the basics of orthodontic tooth movements and the factors that initiate inhibition and delayed tooth movement. Orthodontic tooth movement occurs in the presence of a mechanical stimuli sequenced by remodelling of the alveolar bone and periodontal ligament (PDL). Bone remodelling is a process of both bone resorption on the pressure site and bone formation on the tension site. Orthodontic tooth movement can be controlled by the size of the applied force and the biological responses from the PDL. The force applied on the teeth will cause changes in the microenvironment around the PDL due to alterations of blood flow, leading to the secretion of different inflammatory mediators such as cytokines, growth factors, neurotransmitters, colony-stimulating factors, and arachidonic acid metabolites. As a result of these secretions, remodelling of the bone occurs.

Other Methods of accelerating tooth movement:

There are three phases of tooth movement: the initial phase, which is characterized by rapid movement after the application of force; followed by a lag period, where little or no movement, and the last phase, where gradual or sudden increase of movement occurs.

The early phase of tooth movement involves acute inflammatory responses characterized by leucocytes migrating out of blood capillaries and producing cytokines, which stimulates the excretion of prostaglandins and growth factors. The acute phase is followed by the chronic phase that involves the proliferation of fibroblast, endothelial cells, osteoblasts, and alveolar bone marrow cells remodelling process.

Experiments have been done using these molecules exogenously to enhance tooth movement both in animal experiments and humans. Example of these molecules are prostaglandin E (PGE), cytokines that include lymphocytes and monocytes-derived factors, receptor activator of nuclear factor kappa B ligand (RANKL), and macrophage colony-stimulating factor (MCSF).

Effect of cytokines on tooth movement

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 remodelling; moreover, interleukin-1 (IL-1) stimulates osteoclast function through its receptor on osteoclasts. It was also found that mechanical stress due to orthodontic treatment increased the production of prostaglandin PGE and IL-1 beta in the periodontal ligaments. These experiments were done on cats where one canine was tipped distally by 80 gm of force from hours to days, then immunohistochemistry and microphotometry experiments where done to measure the intensity of PGE and IL-1 beta which was found to be highest on the tension.

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 osteoclast genesis. On the other hand, osteoprotegerin (OPG) competes with RANKL in binding to osteoclast to inhibit osteoclast genesis. The process of bone remodelling is a balance between (RANKL-RANK) system and OPG compound. In relation to this, using biological molecules in the acceleration of tooth movement has been shown in two unique experiments in which it was demonstrated that the transfer of RANKL gene to the periodontal tissue induced prolonged gene expression for the enhancement of osteoclast genesis and acceleration of tooth movements in rats. On the other hand, the transfer of OPG gene inhibited orthodontic tooth movements. In another 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. Also a correlation was found among RANK, OPG, and root resorption during orthodontic teeth movement, and patients with root resorption produced a large amount of RANKL in the compressed site.

Prostaglandin effect on tooth movement

Prostaglandins (PGs) are inflammatory mediator and a paracrine hormone that acts on nearby cells; it stimulates bone resorption by increasing directly the number of osteoclasts. In vivo and in vitro experiments were conducted to show clearly the relation between PGs, applied forces, and the acceleration of tooth movement. Yamasaki was among the first to investigate the effect of local administration of prostaglandin on rats and monkeys. In addition, experiments done in have shown that injections of exogenous PGE2 over an extended period of time caused acceleration of tooth movements in rats. Furthermore, the acceleration rate was not affected by single or multiple injections or between different concentrations of the injected PGE2. However, root resorption was very clearly related to the different concentrations and number of injections given. It has also been shown that the administration of PGE2 in the presence of calcium stabilizes root resorption while accelerating tooth movement. Furthermore, chemically produced PGE2 has been studied in human trials with split-mouth experiments in the first premolar extraction cases. In these experiments the rate of distal retraction of canines was 1.6-fold faster than the control side.

Possible modifications in orthodontic tooth movement (OTM) and root resorption as a result of local injections of prostaglandin E2 (PGE2) alone and with calcium gluconate (Ca) was studied by *Massoud Seifi et all.* The findings show the importance of calcium ions working in association with PGE2 in stabilizing root resorption while significantly increasing OTM.

The mean OTM in the PGE2 and PGE2+ Ca groups was significantly higher than in the control group, although the PGE2 + Ca group demonstrated a nonsignificant decrease in OTM, in comparison with the PGE2 group. The rise in root resorption was significant in the PGE2 group compared with the normal group, which was not surprising in view of the destructive effect of PGE2 in cysts in the oral region. No significant differences were found for root resorption in the PGE2 + Ca group compared with either the normal or control group. Goldie and King (1984) reported a reduction in root resorption for calcium deficient rats; however, Bielaczyc and Golebiewska (1997) demonstrated a rise in root resorption with a diet low in calcium and deficient in vitamin D.

The tendency towards a reduction of resorption in the PGE2 + Ca group may be a result of the transient hypoparathyroidism and diminished resorptive activity subsequent to injection of the calcium compound.

No information is available regarding injection of calcium compounds during OTM. Goldie and King (1984) found that systemic calcium deficiency increased OTM. Midgett *et al.* (1981) demonstrated significantly decreased bone density and increased bone remodelling in animals with hyperparathyroidism, indicating that the reduction in bone density seems to facilitate tooth movement within bone. It can be inferred from the above that the hypoparathyroidism caused by calcium injection in the present study should have inhibited bone remodelling and resisted tooth movement whereas this was not the case. This can be explained by the dominant role of PGE2 with a dose of 1 mg/ml, although a minor insignificant drop was observed in OTM.

Effect of Vitamin D3 on tooth movement

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 calcium homeostasis with calcitonin and parathyroid hormone (PTH).

Another set of investigators has made 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 increasement of osteoclasts on the pressure site as detected histologically. A comparison between local injection of vitamin D and PGEs on two different groups of rats was also investigated. It was found that there is no significant difference in acceleration between the two groups. However, the number of osteoblasts 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 bone turnover.

Parathyroid Hormone effect on tooth movement

PTH has been shown to accelerate orthodontic tooth movement on rats, which was studied by continuous infusion of PTH (1 to 10 μg/100 g of body weight/day) implantation in the dorsocervical region, and the molars were moved 2- to 3-fold faster mesially by orthodontic coil spring. Some studies have shown that locally injected PTH induces local bone resorption, and it is more advantageous to give PTH locally rather than systemically. The development of a slow-release application that keeps the local concentration of PTH for a long time was very efficient as shown later in where the daily injection of PTH dissolved in gel medium allowed a slow release which caused 1.6-fold faster acceleration of teeth compared to daily injection of PTH dissolved in saline solution which did not cause any acceleration.

Soma , *Matsumoto S*, *Higuchi Y*, *Takano-Yamamoto T*, *Yamashita K*, *Kurisu K*, *Iwamoto M*.(2000)reported that whereas systemic continuous infusion of parathyroid hormone (PTH) accelerated orthodontic tooth movement, systemic but intermittent injection of PTH did not increase the rate of tooth movement. Analysis of these data suggested that continuous administration of PTH could be applicable for orthodontic therapy. Histological examination revealed active osteoclastic bone resorption and a widened periodontal space on the compression side of the periodontal tissue in the PTH-MC-injected rats. These results suggest that local injection of PTH in a slow-release formulation is applicable to orthodontic therapy.

Local Injection of Biomodulators

Orthodontic forces create areas of tension and compression in the PDL, which affects remodelling of the periodontium. Following mechanical stress, changes to vascularity and blood flow within the PDL are induced by signalling molecules. The signalling cascade initiates with arachadonic acid metabolites (eicosanoids), neurotransmitters, (substance P and calcitonin gene-related peptide) and second messengers, such as cyclic AMP, phosphoinositol phosphate and diacyl-glycerol.2 These molecules trigger the release of cytokines, growth factors and colony stimulating factors, which affect biological mediators such as RANKL, OPG, MMPs and TIMPs.13 Recent research advances have suggested that these biological modulators, which enhance or inhibit recruitment, differentiation or activation of osteoclasts, could be used to provide new adjunctive approaches to orthodontic treatment. In other words, local injections of biomodulators could be used to accelerate OTM, reduce root resorption, enhance anchorage and improve stability of orthodontic results. Numerous reports have described the pharmacological acceleration of OTM through activation of osteoclasts. A previous study reported that vitamin D3 activated osteoclasts and accelerated OTM.

Local administration of prostaglandins (PGs), osteocalcin, or PTH also induced OTM. However, because these drugs are rapidly flushed by blood flow, daily systemic administration or daily local injection are needed. In addition, frequent injections of this substances in local regions may evoke fear in patients and cause problems in medical treatment. The undesired movement of anchor teeth and the relapse of previously moved teeth are major clinical problems in Orthodontics. Recent research advances suggest that biological modulators which inhibit osteoclasts could be used to address these problems and provide new adjunctive approaches to orthodontic treatment. Several inhibitors have been examined, including bisphosphonates and osteoprotegerin (OPG), and their efficiency in preventing tooth movement has been proved in animal models. Moreover, advances in understanding cytokine-mediated development and progression of rheumatoid arthritis have led to efforts to neutralize these cytokines by using antibody or soluble receptor techniques. Soluble receptors are able to bind their ligands with specificity and affinity, and effectively neutralize cytokine activity. It has been shown in animal models that systemic application of soluble receptors to IL-1 (sIL-RII) or TNF-α (sTNF-α-RI) leads to reduction or even prevention of root resorption. The concentration of these soluble receptors in the local microenvironment of the target periodontium was also sufficient to interfere in the remodeling processes induced in the periodontal tissues, reducing the number of osteoclasts and, consequently, the amount of OTM.Nevertheless, routine clinical use of these biomodulators in orthodontics still requires further investigations, to determine the correct dosage, frequency of administration and, especially, the possible local and systemic side effects of its long term use.

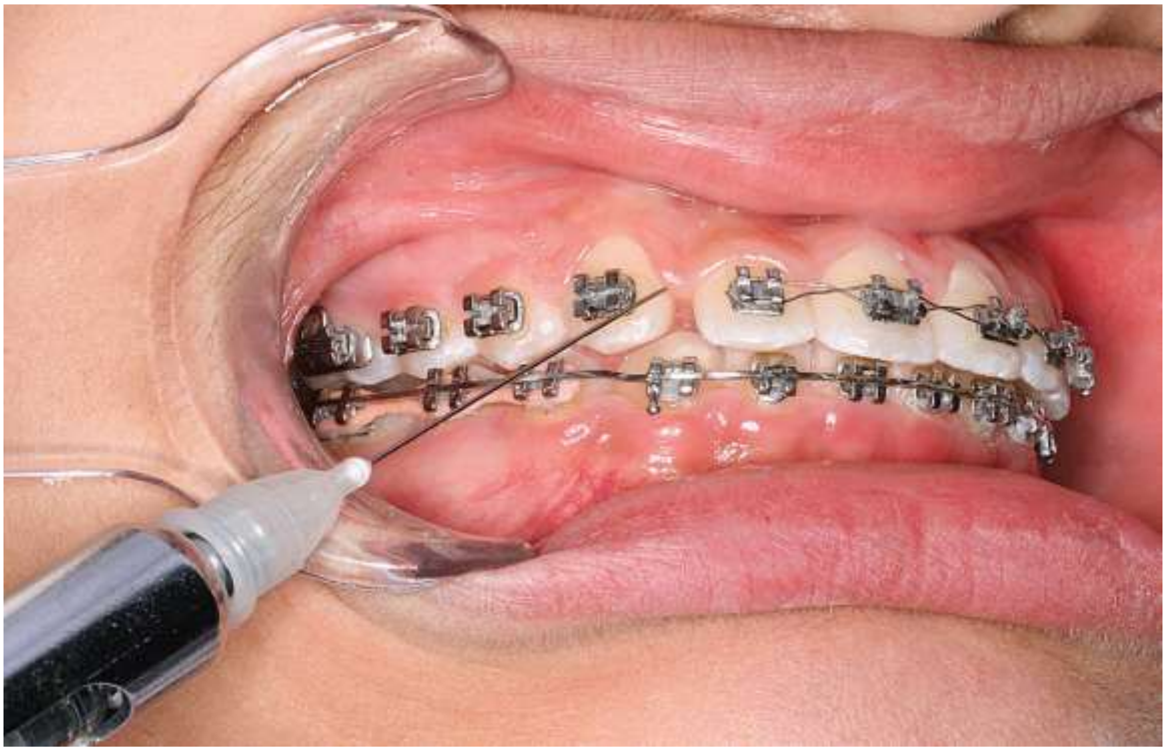


Figure - Injection of biomodulators. Injection of inflammatory mediators in the periodontium

Gene Therapy

The original premise behind gene therapy (GT) in the 90s was the believe that if a defective gene resulting in a specific disease could be replaced by a healthy gene, then the disease could be cured.However, the potential role of GT as a clinical tool has expanded and it is no longer limited to replacement of defective genes, but rather has become a tool for producing individual proteins to specific tissues and cells. Although all cells contain the genes for all proteins, cells derived from a particular tissue express only a limited selection of these proteins. With GT, it is possible to deliver a gene to a given cell, which allows the inserted gene product to be expressed constitutively.Modern technology has allowed the manufacture of these proteins (human recombinant proteins) for therapeutic use. However, their life spam is short after injection into the human body. As GT provides the gene for protein production rather than just replacing degradable protein, it achieves higher and more constant levels of protein expression. For this reason, it has became an effective method used to deliver these proteins to specific tissues. Once protein and location of protein delivery have been chosen, the next step is to choose the vector to deliver the protein. The objective is to get the DNA that encodes the specific protein into the target cell and force it to express the desired protein. The most common delivery vector is by means of a virus, a process also known as "transduction." Nonviral vectors are also used, in which case the process is referred to as "transfection". It is carried out by means of several methods, including liposome and gene gun. The easiest way to implement local GT is by injecting the vector into a specific tissue. The vector may be delivered systemically to all cells in the body (as in treatment of metastatic diseases) or locally to the target tissue, only (as desired in Orthodontics). Direct GT has been effectively used in knee and ankle joints, skeletal muscle, bone and ligaments. Nevertheless, in indirect GT, target cells are harvested from the patient and then reinserted. It is advantageous for being able to accurately select a particular cell as the protein delivery vehicle. The indirect method has been effectively used to target articular cartilage, spine and human metacarpophalangeal joints.Numerous reports have described the pharmacological acceleration of OTM through activation of osteoclasts. However, due to their rapid flush out by blood circulation, daily systemic administration or daily local injection is needed. Local gene transfer has two advantages. First, it maintains local effective concentration and prolonged protein expression, regardless of blood circulation. Second, protein expression occurs at a local site, thereby avoiding systemic effects.

A previous animal study demonstrated that transfer of RANKL gene to periodontal tissue activated osteoclast genesis and accelerated OTM without producing any systemic effects. When comparing corticotomy surgery and RANKL gene transfer to periodontal tissue as two methods that might substantially reduce orthodontic treatment time, RANKL GT demonstrated higher efficacy than standard surgical methods. Local GT has also been used to inhibit OTM, which might be, in the near future, an important tool to enforce the anchorage unit or increase stability of orthodontic results. Local OPG gene transfer significantly inhibited RANKL-mediated osteoclast genesis in the periodontium caused by experimental tooth movement.66 Moreover, local OPG gene transfer might be a biologic method employed to prevent or inhibit relapse after orthodontic treatment.67 Other local or systemic pharmacological agents, such as bisphosphonates and simvastatin, also decrease the extent of initial relapse, but they are rapidly distributed by blood circulation and, for this reason, require daily systemic administration. Local OPG gene transfer is also clinically relevant for enhancing external root resorption (ERR) repair during retention. However, the precise biological mechanism behind this finding has not yet been fully elucidated and further studies are required to assess the role of RANK ⁄ RANKL ⁄ OPG axis in ERR repair.

In short, GT is a pioneering new therapeutic modality based on complex biological systems occurring at the leading edge of biomedical knowledge. It offers an alternative method to deliver proteins to a given target tissue, which, in turn, can enhance or inhibit osteoclast recruitment and lead to a more or less OTM. Nonetheless, further research is needed to determine the safety and efficacy of these techniques.

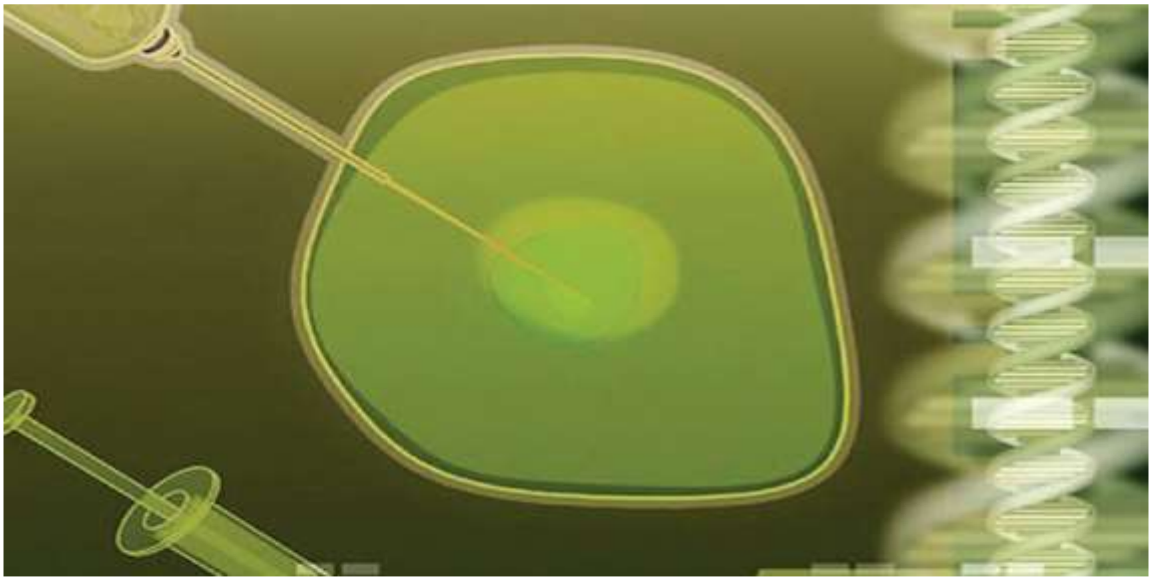


Figure- Gene Therapy. Delivering a gene to a given cell allows the inserted gene product to be expressed constitutively.

RELAXIN EFFECT ON TOOTH MOVEMENT

Relaxin effect has also been investigated. Relaxin is a hormone that helps during childbirth by widening of the pubic ligaments in females and is suggested to be present in cranial suture and PDL. The role of relaxin is known in the remodelling of soft tissue rather than remodelling of bone. It has been shown that it increases collagen in the tension site and decreases it in compression site during orthodontic movement. Also, the administration of human relaxin may accelerate the early stages of orthodontic tooth movement in rat experiments. However, another study showed that human relaxin does not accelerate orthodontic tooth movement in rats but can reduce the level of PDL organization and mechanical strength of PDL and increase tooth mobility. In these experiments in vitro studies were also performed to test the PDL mechanical strength and tooth mobility using tissue from additional 20 rats that had previously received the same relaxin treatment for several days. The remodelling of PDL by relaxin might reduce the rate of relapse after orthodontic treatment as suggested by others. Recently, randomized clinical trials on humans were done by weekly injections of 50 μg of relaxin or placebo control for 8 weeks. Tooth movement was measured weekly on polyvinyl siloxane impressions which were scanned digitally. There was no significant difference between the relaxin and the placebo control group regarding the acceleration and relapse. However, the mechanism of how relaxin accelerates tooth movement is not yet fully understood.

Device-assisted treatment

Another approach in accelerating tooth movement is by using device-assisted therapy. This technique includes direct electric currents, pulsed electromagnetic field, static magnetic field, resonance vibration, and low level laser which was mostly investigated and gave the most promising results.

The concept of using physical approaches came from the idea that applying orthodontic forces causes bone bending (bone bending theory) and bioelectrical potential develops. The concave site will be negatively charged attracting osteoblasts and the convex site will be positively charged attracting osteoclasts as detected by Zengo in his measurements on dog alveolar bone. The bioelectrical potential is created when there is application of discontinuous forces, which leads to the idea of trying cyclic forces and vibrations. It has been found that applying vibrations for different duration per day accelerated tooth movements between 15% and 30% in animal experiments.

Laser Therapy

The term "laser" originated as an acronym for "light amplification by stimulated emission of radiation". It is a device that emits light through a process of optical amplification based on the stimulated emission of electromagnetic radiation. Lasers differ from other light sources by their coherence which allows them to be focused to a limited spot, to stay narrow over long distances or to have a very narrow spectrum (emitting a single color of light). In medicine, lasers have many important applications: bloodless surgery, laser healing, surgical treatment, kidney stone treatment, eye treatment and many others. The laser technique has also been widely applied in Dentistry; in orthodontic treatment, it has proved to have many benefits. They can be used to perform gingivectomy, frenectomy, surgical exposure of tooth (with less bleeding and swelling, improved precision, reduced pain and improved healing), enamel etching, bonding, bracket debonding, pain control, treatment of traumatic ulcers in the oral mucosa and to accelerate tooth movement.

Lasers can be classified as low and high-intensity lasers of which main differences are their potency and mechanism of action. High-intensity lasers, such as the CO_2 laser, Nd laser: Yttrium aluminum garnet (Nd:YAG), argon laser, Er:YAG laser, and the excimer laser act by increasing the temperature, showing a destructive potential, and are usually usedin surgical procedures. Meanwhile, the low-intensity laser (also known as soft laser, cold laser or laser therapy) does not have a destructive potential. Its photobiomodulation mechanism of action penetrates tissues and stimulates cellular metabolism, bone remodelling and tooth movement which is of greatest interest in Orthodontics. Different low-energy laser modalities have been used in different doses and in various treatment protocols, including helium-neon (632.8 nm wavelength) and semiconductor lasers (emitting light in the range of 780–950 nm), gallium-aluminum-arsenide (GaAlAs) (805 ± 25 nm wavelength) and gallium-arsenide (904 nm wavelength).GaAlAs diode laser has been repeatedly used in the past years and has proved to have higher depth of tissue penetration in comparison to other modalities, therefore, providing the clinicians with a suitable penetrative instrument with great efficiency in orthodontic treatment. The exact mechanism of laser–cell interaction is still to be investigated. The stimulation of photoreceptors in the mitochondrial respiratory chain, changes in cellular ATP levels and cell membrane stabilization have been discussed. It is generally accepted that laser effects on cells are wavelength and dose-dependent. The existence of a "window of specificity" at certain wavelengths and energy dosages has been postulated. Molecular absorption of laser light is a prerequisite for any cellular effect.

Figure - Laser irradiation. Application of LLLT in areas of intended tooth movement.

A previous study demonstrated that low-level laser therapy (LLLT) stimulates cellular proliferation and differentiation of osteoblast lineage nodule-forming cells, especially in committed precursors, resulting in an increase in the number of differentiated osteoblastic cells as well as in bone formation. Meanwhile, another study found that low energy laser irradiation stimulated the amount of tooth movement and formation of osteoclasts on the side of pressure during experimental tooth movement *in vivo*. As bone remodelling is a physiological process that involves osteoclastic bone resorption and osteoblastic bone formation, those findings are not surprising. Furthermore, recent studies showed that low-energy laser irradiation accelerated orthodontic movement of human teeth. However, the effect of LLLT on tooth movement is reportedly controversial, as different stimulatory, inhibitory and irrelevant effects have been shown in the literature. A previous study reported that low-energy laser irradiation significantly inhibited the production of prostaglandin E (PGE2), and that interleukin (IL-1β) was increased by mechanical stress *in vitro*. If low-energy laser irradiation functions to inhibit these pro-inflammatory cytokines, OTM might be slow. Another LLLT study demonstrated low stimulatory or inhibitory effect on the rate of orthodontic tooth movement. Conversely, other studies reported that IL-1, RANKL, M-CSF, MMP-9, cathepsin K, and α(v)β3 integrin were stimulated via their respective pathways during the differentiation of bone cells, and the amount of tooth movement was increased by low-energy laser irradiation. Moreover, an *in vitro* study showed that the gene expression of RANK in osteoclast precursor cells increased when cells were irradiated with low-energy laser. On the basis of the findings of this review, it is possible to assert that LLLT speed up tooth movement via RANK ⁄ RANKL expression.

Although further studies are necessary to evaluate the effects of different irradiation dosages, the prolonged use of laser irradiation on tooth movement or bone remodelling, or both, and the introduction of laser therapy at an early stage of tooth movement in orthodontic treatment seem feasible and may be of great therapeutic benefit to abbreviate treatment time.

Phototherapy

There is increasing application for phototherapy in areas of wound healing, tissue repairand regeneration and reductions in dental sensitivity and post-surgical and post-orthodontic adjustment pain (Dosi-Mehta and Bhad-Patil, 2012; Barolet and Boucher, 2010; de Paula Eduardo et al., 2010; Xiaoting et al., 2010; Torammano et al., 2009; Trelles and Allones, 2006; Weiss et al., 2005; Meguro et al., 2002). Phototherapeutic applications are reliant upon the bio stimulatory effects of phototherapy. The term “bio stimulation” was first introduced in the1960’s to describe the “photochemical interactions” of low intensity lasers with tissue (Niemz2007; Desmet et al., 2006; Sommer et al., 2001) and has since also been referred to as photo stimulation, photo modulation and photo bio stimulation.

Phototherapy is hypothesized to produce bio stimulatory effects from increased blood circulation (Cruz et al., 2004) and pro-inflammatory mediators such as IL-1b (Saito and Shimizu, 1997) and PGE1 (Yamasaki et al., 1984) increased ATP availability and cell metabolism via the removal of electron deficits by the appearance of singlet oxygen or by direct stimulation of the electron carriers (cytochrome c oxidase) in the electron transport chain (Bashardoust Tajali et al., 2010; Conlan et al., 2006; Desmet et al., 2006; Karu et al., 2005; Eells et al., 2004) and/or increased Na+/K+ pump activity and intracellular Ca2+ causing augmentation of protein synthesis as well as DNA and RNA replication to accelerate cell metabolism (Bashardoust Tajali et al., 2010; Coombe et al., 2001). It is hypothesized that the biostimulatory effect of phototherapy is governed by the Ardnt-Schultz law (Figure 1.1), where a low-moderate level of energy is required to achieve cell activation but excess energy results in cell retardation (Kim et al., 2009; Sommer et al., 2001).

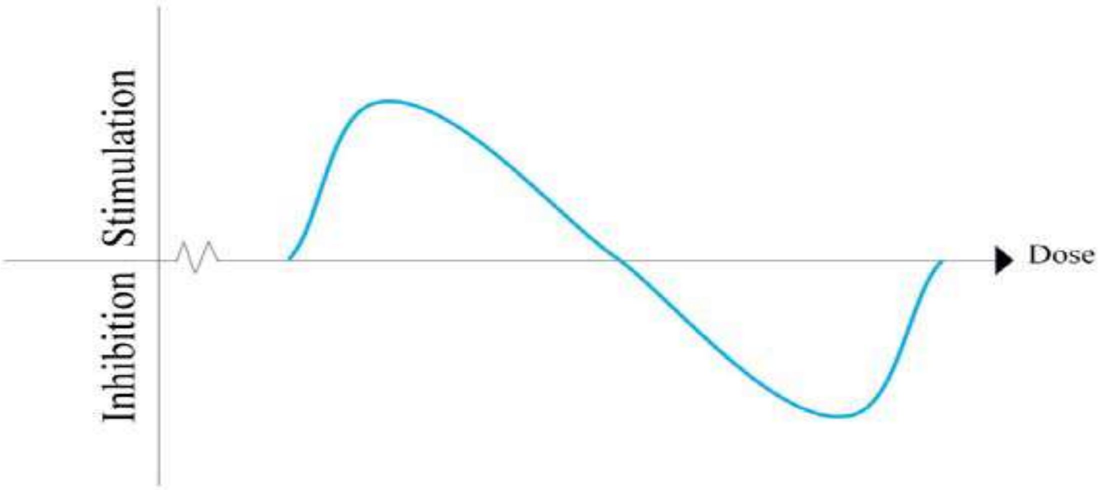


Figure 1.1 The Ardnt-Schultz effect describes the observation of a stimulatory effect in response to a low-moderate dose of an intervention whereas an inhibitory response is seen when higher doses of an intervention/drug are used.

Furthermore, “due to the cooperative behaviour of photo stimulated cells, it is important to irradiate the application field simultaneously to avoid adverse effects with respect to the intended aims...creating a homogenously distributed mean energy density with the necessary local light intensity, as required for activation” (Sommer et al., 2001). In other words, to observe bio stimulation, phototherapy should be delivered to the entire field of tissue uniformly. Thus, two conceptual methods of delivering phototherapy include lasers (eg. LLLT) and LEDs (Vladimirov et al., 2004; Karu, 2003; Sommer et al., 2001).

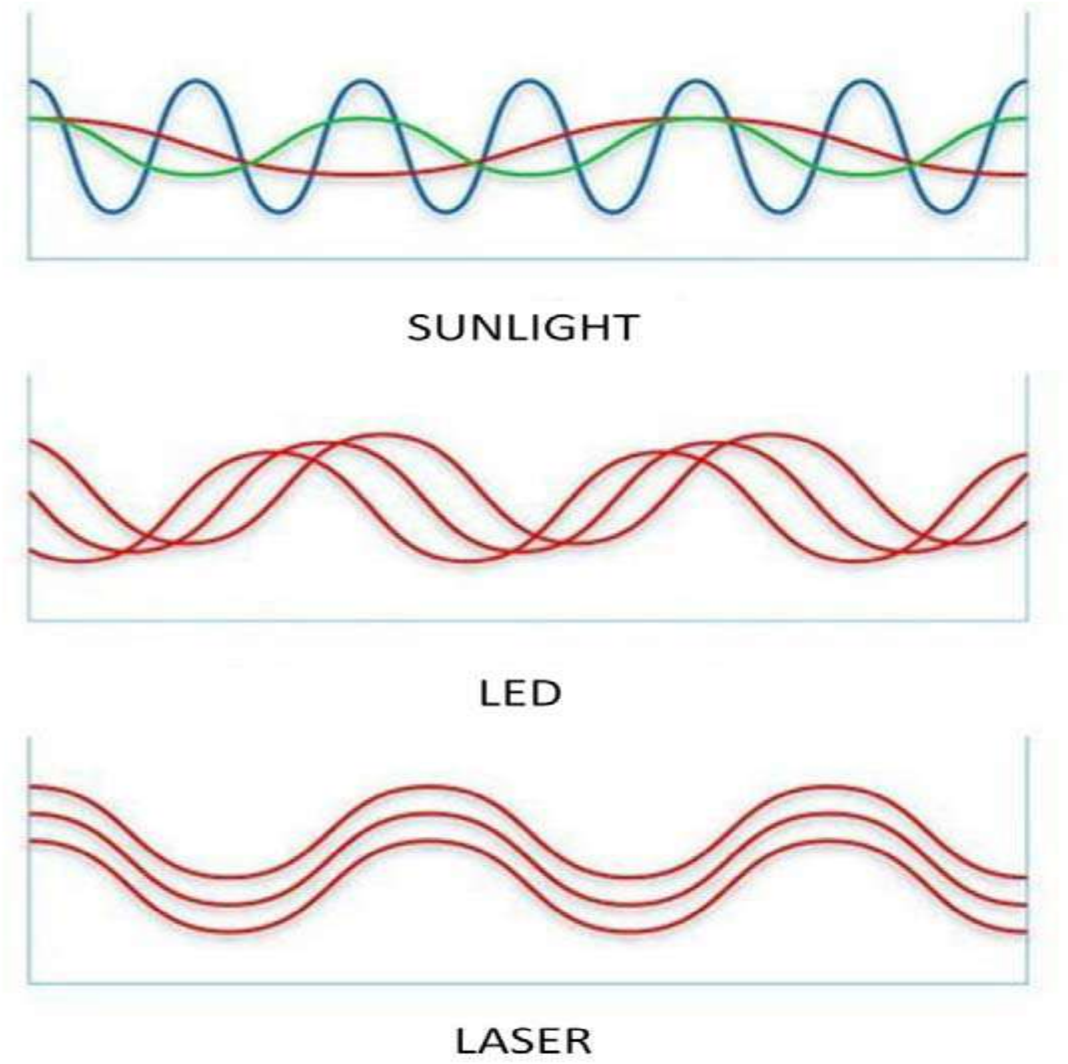


Figure 1.2 The comparison of sunlight, LED and lasers. Sunlight is composed of light of various wavelengths, and amplitudes. LED light is composed of light that has a narrow wavelength and amplitude, with low spatial coherence. Laser light displays spatial and temporal coherence of light with a narrow wavelengths and amplitude.

In general, when electromagnetic radiation (i.e. laser or LED) is applied to tissues, it is reflected, transmitted, scattered and/or absorbed. This can result in different effects, e.g., photochemical (photonically induced chemical reactions), photothermal (alterations to chemical bonds resulting in heat, ablation and/or coagulation) and photomechanical or photoionizing (cell damage resulting from destruction of cell membranes, proteins and/or DNA) effects (Graber et al., 2012; Norton et al., 2008).The term "laser" (Light Amplification by Stimulated Emission of Radiation) was first proposed by Gordon Gould in 1957 and describes a form of electromagnetic radiation with a very narrow wavelength and focus. In comparison to sunlight, laser light is coherent in nature, with a fixed relationship between the electric field values at different locations (spatial coherence) or at different times (temporal coherence). It is generated in a resonator and emitted continuously or in a pulsed manner, such that they are propagated over long lengths without divergence (Paschotta, 2008). Lasers are classified according to their ability to produce tissue damage. The classification of a laser is dependent upon the characteristics of power, wavelength, exposure and cross-sectional area of the laser beam. In accordance with the International Electrotechnical Commission, the safety of lasers is expressed in terms of *maximum permissible exposure*, describing the highest power that tissues can be exposed to without damage. The safety ratings range from 1 which is safe under all conditions to the maximum 4 which is always hazardous to view and has devastating damage to the eye and skin and with an ability to ignite material. Additionally, within the first and second safety ratings is a sub-category denoted as "M", referring to the power not exceeding a certain limit as measured through a 7mm aperture at a distance of 10cm from the source (Graber et al., 2012). Typically, lasers used in phototherapy are in the 1M category (denoting a low intensity and hence the term LLLT). Although lasers can be set to emit different wavelengths, an infrared (IR) wavelength (approximately 600-1000nm) in the range of approximately 730-850nm is viewed as the most appropriate to promote bio stimulation as well as increases in OTM (Yoshida et al., 2009; Kocoklu-Altan et al., 2009; Desmet et al., 2006; Karu et al, 2005; Eells et al., 2004; Schieke et al., 2003; Stolik et al., 2000).

1.1 Light Emitting Diodes - An Alternative to Low Level Laser Therapy

In contrast to lasers (Figure 1.2 and Table 1.1), LED light is generated via electroluminescence (light emission triggered by electric influences, eg. electron beam) to produce a light with low spatial coherence; being emitted in all directions with low focus ability and a wider wavelength range. LEDs benefit from low energy consumption and low heat emission. As with lasers, the wavelength of the emitted light is dependent on the material that is used to generate the light emission, with a combination of Gallium-Arsenide-Aluminum (GaAsAl) producing IR-LED in the 680-860nm range. These characteristics of LEDs help explain why their typical uses include: LED displays, ambient lighting, signal lights, cellular phone displays, TV screens and streetlights. (Paschotta, 2008) The form of the light energy (i.e., LLLT or LED) does not appear to be a prerequisite for biostimulatory effects given that “under physiological conditions, the absorption of low intensity light by biological systems is of purely non-coherent (i.e., photobiological) nature” (Karu, 2003). Since lasers are coherent in nature, targeting a region of tissue would necessitate the combination of high powered lasers with optical lenses. However, this adaptation can be very expensive. Lasers also have limitations in wavelength capabilities and beam width, the potential for significant heat production which can burn tissue, the potential for eye damage due to the narrow focus of the beam and a greater cost when compared to LED devices (Casalechi et al., 2009, Eells et al., 2004). Furthermore, lasers require the clinician to deliver the exposure to the patient on a regular basis, rather than the patient themselves. Given the potentially limitingcharacteristics of lasers in terms of regional tissue application, LEDs stand to be a promising alternative for biostimulation (Vladimirov et al., 2004; Sommer et al., 2001) with the potential to be more effective and practical as a medium to uniformly deliver energy to an entire field of tissue and to influencing OTM.

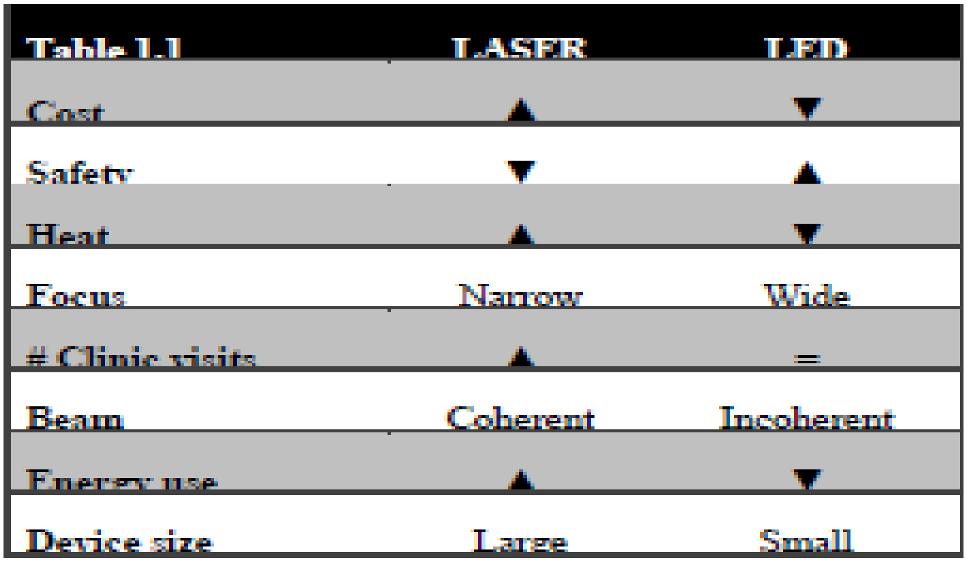


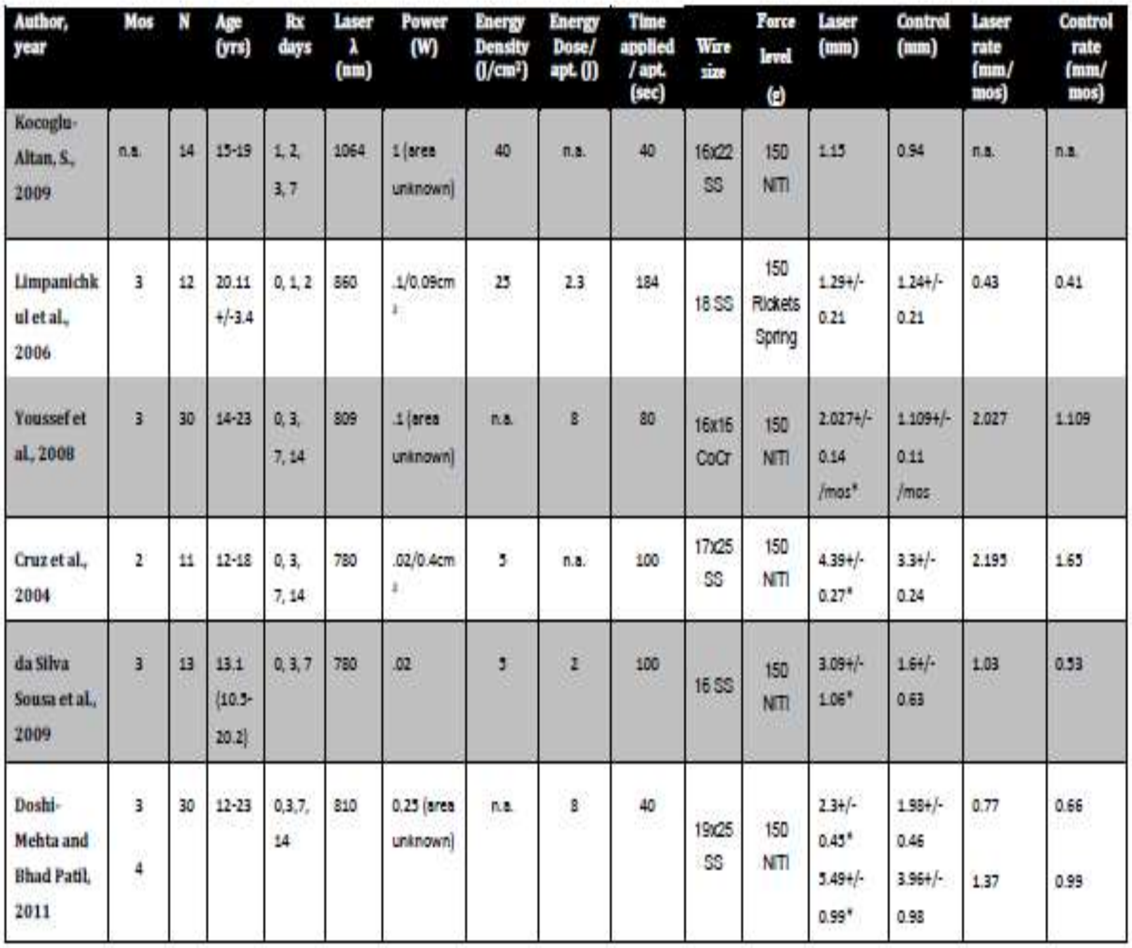
Table 1.1	LASER	LED
Cost	▲	▼
Safety	▼	▲
Heat	▲	▼
Focus	Narrow	Wide
# Clinic visits	▲	=
Beam	Coherent	Incoherent
Energy use	▲	▼
Device size	Large	Small

Table 1.1 A Comparison of the Characteristics of Lasers and Light Emitting Diodes

Regardless of the mechanism behind bio stimulation, the downstream effects of phototherapy have been observed as increased cellular proliferation, collagen and procollagen synthesis, the release of growth factors from cells, enhanced fibroblast and osteoblast activity, collagen and bone formation, calcium and phosphate incorporation, nerve stabilization, ATP production and reductions in pH, MMP-8, collagenase activity, IL-1, TNF and interferon when lasers are used in treatment (Abi-Ramia et al., 2010; Youssef et al., 2008; Desmet et al., 2006; Sommer et al., 2001). These findings have prompted the use of lasers alone or in combination with traditional therapeutic approaches in periodontics, oral surgery, endodontics, and restorative procedures, and more recently, orthodontics (Walsh, 2003). Clinical benefits that have been described with the use of LLLT include the reduction of post-surgical and post-orthodontic adjustment pain and gingival health, improvements in post surgical healing times, antimicrobial effects and the ability to reduce dentinal sensitivity(Bicakci et al., 2012; Doshi-Mehta and Bhad-Patil, 2012; Esper et al., 2011; Bashardoust Tajali et al., 2010; de Paula Eduardo et al., 2010; Xiaoting et al., 2010; Torammano et al., 2009; Fujiyama et al., 2008; Youssef et al., 2008; Turhani et al., 2006; Meguro et al., 2002; Harazaki et al., 1998; Luger et al., 1998; Saito and Shimizu, 1997; Lim et al., 1995). However, despite the recent recognition of phototherapy to produce bio stimulatory effects, more clinical studies to discern the mechanism of action are needed (de Paula Eduardo et al., 2010).

1.2 The Role of Low-Level Laser Therapy in Orthodontic Tooth Movement

In an attempt to elucidate the mechanisms behind bio stimulation, i*n vitro* experiments have shown that LLLT increases the number of osteoclasts (Aihara et al., 2006), fibroblasts (Vinck et al., 2003) and the rate of proliferation and DNA synthesis of clonal osteoblastic cells (Yamada, 1991) with such changes associated with increased pulp vascularity (Abi-Ramia et al., 2006), protease activity (Yamaguchi et al., 2010) and connective tissue (Kim et al., 2010) and bone turnover (Habib et al., 2010; Yoshida et al., 2009). Furthermore, LLLT has been found to result in increases in RANK expression (Fujita et al., 2008; Aihara et al., 2006), RANKL (Fujitaet al., 2008), TRAP (tartrate-resistant acid phosphatase - indicative of osteoclatsic activity) (Kawasaki et al., 2000), M-CSF (macrophage colony stimulating factor) and csf-1 (colony stimulating factor-1) (Yamaguchi et al., 2007); implicating major involvement in signalling pathways of bone metabolism and OTM. Evidence of such implications have been shown in animal experiments where LLLT enhanced bone fracture healing (Bashardoust et al., 2010; Hadjiargyrou et al., 1998; Luger et al., 1998) and the rate of OTM (Habib et al., 2012; Goulart et al., 2006). These and other studies of similar nature have underwritten the potential for LLLT to influence OTM in humans. Although the effects of LLLT on OTM in humans are limited and mixed results, several studies (Table 1.2) have shown the effect of LLLT on OTM during canine retraction using asplit-mouth design. There have been 2 reports citing no significant effect of LLLT on OTM. The lack of effect was cited as a result an incorrect phototherapy dose leading to reduced levels of arachidonic acid and PGE2, with subsequent reductions in osteoclastic activity and enhancement of osteoblastic activity. Despite no notable benefits on OTM, these studies demonstrated that there were no iatrogenic effects resulting from LLLT exposure (Kocoglu-Altan et al., 2009 Limpanichkul et al., 2006). Conversely, several studies have demonstrated positive effects of LLLT on OTM (Gencet al., 2012; Doshi-Mehta and Bhad-Patil, 2011; da Silva Sousa et al.,2009; Youssef et al., 2008; Cruz et al., 2004). The majority of these studies concluded that the application of LLLT increased the amount of OTM by approximately 15-95% during canine retraction in a split mouth design over 1-4 months (Table 1.2). They also described no differences in root resorption or crestal bone loss associated with LLLT during OTM (Doshi-Mehta and Bhad- Patil, 2011; da Silva Sousa et al., 2009; Youssef et al., 2008; Cruz et al., 2004). Together, these studies illustrated that although the dose dependent effect of LLLT has not yet been determined, LLLT can affect bone metabolism and OTM at the cellular, animal and human levels without any harmful effects.



Author, year	Mos	N	Age (yrs)	Rx days	Laser λ (nm)	Power (W)	Energy Density (J/cm²)	Energy Dose/ apt. (J)	Time applied / apt. (sec)	Wire size	Force level (g)	Laser (mm)	Control (mm)	Laser rate (mm/ mos)	Control rate (mm/ mos)
Kocoglu-Altan, S., 2009	n.a.	14	15-19	1, 2, 3, 7	1064	1 (area unknown)	40	n.a.	40	16x22 SS	150 NiTi	1.15	0.94	n.a.	n.a.
Limpanichkul et al., 2006	3	12	20.11 +/-3.4	0, 1, 2	860	.1/0.09cm^2	25	2.3	184	18 SS	150 Rickets Spring	1.29+/-0.21	1.24+/-0.21	0.43	0.41
Youssef et al., 2008	3	30	14-23	0, 3, 7, 14	809	.1 (area unknown)	n.a.	8	80	16x16 CoCr	150 NiTi	2.027+/-0.14 /mos*	1.109+/-0.11 /mos	2.027	1.109
Cruz et al., 2004	2	11	12-18	0, 3, 7, 14	780	.02/0.4cm^2	5	n.a.	100	17x25 SS	150 NiTi	4.39+/-0.27*	3.3+/-0.24	2.195	1.65
da Silva Sousa et al., 2009	3	13	13.1 (10.5-20.2)	0, 3, 7	780	.02	5	2	100	16 SS	150 NiTi	3.09+/-1.06*	1.6+/-0.63	1.03	0.53
Doshi-Mehta and Bhad Patil, 2011	3 4	30	12-23	0,3,7, 14	810	0.25 (area unknown)	n.a.	8	40	19x25 SS	150 NiTi	2.3+/-0.45* 5.49+/-0.99*	1.98+/-0.46 3.96+/-0.98	0.77 1.37	0.66 0.99

TABLE 1.2 The effects of LLLT on OTM during canine retraction with a split mouth design. (*denotes $p<0.05$, when comparing lasers to controls)

1.3 Light Emitting Diodes – Cell, Molecular, Tissue and Metabolic Effects with a Potential Role in Orthodontic Tooth Movement

The reports on the effects of LED phototherapy on tissue and OTM are more limited than that of LLLT (lasers). At the cellular level, it has been demonstrated that peak absorption measurements of cellular monolayers occur in the range of 730-850nm (Karu et al., 2005). Furthermore, LED phototherapy has been shown to increase mesenchymal stem cell and fibroblast proliferation (Li et al., 2010; Vinck et al., 2003), DNA replication, cell adhesion (Karu et al., 2005), cell growth, proliferation rates and mitochondrial activity, while rescuing mitochondrial dysfunction (Holder et al., 2012). These findings are consistent with animal studies showing enhanced wound healing and tissue regeneration (Rosa et al., 2013; Tada et al.,2009; Casalechi et al., 2009; Al-Watban et al., 2006; Whelan et al., 2003), decreased inflammation (Fonseca et al., 2013), increased mini-implant stability (Uysal et al., 2010) and increased periodontal tissue vascularity and repair during OTM (Fonseca et al., 2013). At the clinical level, the safety and benefits of LED phototherapy have been demonstrated in other health fields than dentistry as reduced healing times and post-surgical post erythema, increased medicament absorption (Barolet and Boucher, 2010), accelerated reepithelialisation and decreased symptoms (burning, peeling, redness, swelling, peeling) (Trelles and Allones, 2006; Weiss et al., 2005). Although very limited, the dental benefits have been reported as positive effects on socket preservation (Brawn et al., 2007) using LED phototherapy 10 minutes per day for 21 days (600-650nm, 12J/cm2, 3.6cm2) and reduced pain associated with OTM when applied for 70 seconds (640nm, 4J/cm2, 0.1W) at a given orthodontic appointment (Esper et al., 2011). Despite the similarities in the effects of LED phototherapy to LLLT, there have been no publications on the effects of LED phototherapy on OTM in humans to date. Given the safety of LEDs as compared to lasers (Casalechi et al., 2009, Eells et al., 2004) and the fundamental similarities at the biological level, this study investigates the role of LED phototherapy on OTM with the intention of establishing a foundation for IR- LED phototherapy mediated increases in OTM rates. This study will also offer opportunities to understand the cellular and molecular mechanisms that underlie OTM and possibly tissue repair/regeneration and in more generalterms, connective tissue metabolism.

Vibration

Tooth movement is closely related to response to applied orthodontic forces that cause remodelling of periodontal tissues, especially the alveolar bone. Bone is a highly specialized form of connective tissue and consists of a cortical bone that overlies the softer inner structure named cancellous or trabecular bone. Its formation and regeneration involve chemotaxis, cell proliferation, differentiation and synthesis of extracellular matrix; a result of interaction established amongst biochemical, biomechanical, cellular and hormonal signals. Low-intensity pulsed ultrasound (LIPUS) stimulation is a clinically established, widely used and FDA (Food and Drug Administration) approved intervention for accelerating bone growth during healing of fractures, non-unions and other osseous defects. Therapeutic ultrasound is also widely used, especially in sports medicine and myofunctional therapy, to decrease joint stiffness, reduce pain and muscle spasms, and improve muscle mobility. The frequency and intensity of ultrasound used not only for imaging the human brain (7.5-20 MHz), but also for operative procedures (1 to 3 W/cm2) are much higher than that used for LIPUS which generally uses frequencies varying between 0.5 – 1.5 MHz frequency pulses (with a pulse width of 200 μs) and intensity output of 30 mW/cm2 (which is the output signal of devices approved for clinical use), 5-20 minutes per day.LIPUS is a form of physical energy that can be delivered into living tissues as acoustic intensity waves. *In vivo*41 and *in vitro*studies have shown the direct effect of LIPUS on bone cells.

Figure- Low-intensity pulsed ultrasound. LIPUS stimulation used to accelerate OTM (Acceledent, Ortho Accel Technologies, Huston, USA).

Although the mechanism by which LIPUS increases the rate of fracture healing is unclear, it is known that the mechanical strains received by cells are translated into biochemical events. LIPUS, in essence a wave of alternating pressure, is translated into an extracellular mechanical force at the cell membrane where it is transduced into intracellular electrical and/or biochemical signals. Previous studies indicate that LIPUS accelerates the differentiation pathway of mesenchymal stem cells in the osteogenic lineage via activated phosphorylation of MAPK (mitogen-activated protein kinase) pathways, up-regulation of cyclo-oxygenase-2 (COX-2), prostaglandin E2 (PGE2), altering the OPG/RANKL ratio in the microenvironment and stimulating the production of bone morphogenetic proteins.

As bone, the PDL is also a dynamic tissue which is constantly being remodelled to adapt to mechanical loading. Therefore, it is expected that an appropriate level of mechanical stress be able to induce an anabolic response of the periodontium. The PDL is both the medium of force transfer and the means by which alveolar bone remodels itself in response to applied forces. Moreover, PDL cells (PDLCs) play an important role not only in the maintenance of the periodontium, but also in promoting periodontal regeneration during and after the OTM. They are a heterogeneous cell population, including cells at different stages of differentiation and lineage commitment. Mechanical vibration can affect osteogenesis by increasing the commitment of PDLSCs to the osteogenic lineage. A previous study has shown that the protein levels of RUNX2 and OSX (transcription factors that play a role in the differentiation and activation of osteoblasts) were both prominently enhanced under ultrasound stimulation. It has also been shown that LIPUS stimulation accelerates OTM by increasing osteoclast number and activity, probably by enhancing the expression of RANKL on the pressure sites. These same studies have hypothesized that resonance vibration might prevent blood flow obstruction and hyalinization at the compression sites. Furthermore, LIPUS minimizes orthodontically induced tooth root resorption by enhancing dentine and cementum deposition, thereby forming a preventive layer against root resorption. In short, LIPUS has many clinical advantages, including the fact that it is a biological stimulus, easy to use and non-invasive, in addition to being widely used in clinical works.

Cyclical force device effect on tooth movement

They have also used this concept by using the cyclical force device with patients and achieved 2 to 3 mm/month of tooth movement. The vibration rate was 20 to 30 Hz and used for 20 min/day. Further results needed to be investigated to clearly identify the range of Hertz that can be used in these experiments to get the maximum desired results.

Effect of photo biomodulation on orthodontic tooth movement

Zhifeng Liu, Juan Xu, Lingling E, and Dongsheng Wang examined the effect of low-intensity pulsed ultrasound (LIPUS) on orthodontically induced root resorption in rats.

His study showed results revealing smooth root surfaces and few root resorption pits in the negative control group (Figure 2). The positive control group displayed widespread root resorption lacunae. These resorption lacunae had clear-cut yet irregular rims (Figure 2B). Root surfaces in the LIPUS-treated groups were coarser than those in the negative control group, and small isolated resorption pits were seen on the root surfaces (Figure 2C,D). Under high-power lens, a large amount of new cementum was evidently found in both LIPUS-treated groups (Figure 3; Table 1). The total number and area of the resorption lacunae in the LIPUS-treated groups were smaller than those in the positive control group.

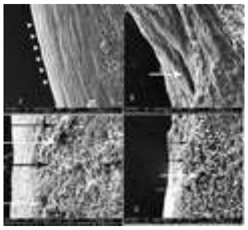


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Figure 2SEM images of control and LIPUS-treated rats. (**A**) Negative control group showing smooth root surfaces (Δ) and few root resorption pits. (**B**) Positive control group showing widespread resorption lacunae with clear-cut yet irregular rims (⇨). (**C**) 100 MW/cm^2 LIPUS-treated group and (**D**) 150 MW/cm^2 LIPUS-treated group showing small, isolated resorption pits (⟶) and new cementum (⟶) on the root surfaces.

Histologic Observations

Staining with hematoxylin-eosin (HE) indicated that root resorption mainly occurred at the root furcation and in the vicinity of the stress-side apex. The LIPUS-treated groups showed less resorption than the positive control group.

Root resorption was less active in the LIPUS-treated groups.100 MW/cm^2 LIPUS was more effective than 150 MW/cm^2 LIPUS, consistent with previous reports that the stimulatory effect of LIPUS is dose-dependent.

Treatment with LIPUS increased OPG and decreased RANKL expression in the experimental groups, thereby reducing the number and activity of osteoclasts and naturally reducing root resorption. By treating cement oblasts with high-intensity (150 MW/cm^2) or low-intensity (30 MW/cm^2) ultrasonic radiation, Dalla-Bon previously found that LIPUS increases OPG expression but does not affect RANKL expression. There are at least two potential reasons for this observed difference in results. First, LIPUS can influence stem cells in the periodontal membrane and can induce interactions among cells. For example, exposure of human umbilical cord–derived mesenchymal stem cells to LIPUS increases the mesenchymal stem cell yield by promoting release and enhancing proliferation. Second, the mechanical stimulus elicits the strong release of inflammatory factors, such as tumor necrosis factor-α (TNF-α), interleukin-β (IL-β), and prostaglandin E2 (PGE2), from the weakened periodontal ligament. These inflammatory factors can stimulate RANKL expression. LIPUS can reduce the levels of these inflammatory factors, TNF-α and IL-β, thereby reducing RANKL expression and osteoclast differentiation. Prolonged treatment with LIPUS previously was shown to promote bone and incisor growth and repair as well as the rate of tooth eruption during mandibular distraction. Exposure of cement oblasts to LIPUS affects the mRNA expression of alkaline phosphatase and increases cellular calcium levels, which regulate the mineralization process. However, LIPUS had no effect on cell proliferation in this previous study. Recently, LIPUS was shown to stimulate MSC (mesenchymal stem cells) differentiation along cartilage and osteogenic lineages. These findings suggest that therapeutic ultrasound may be advantageous for stem cell and tissue engineering applications and has great potential in hard tissue repair and regeneration. The different phylogenic status between humans and lower animals such as mice, rats, and rabbits makes it difficult to extrapolate results from animal models to the clinic. The growth pattern of cementum in lower animals involves continuous eruption, with cementum being formed throughout their lifetime. However, the study of root resorption in an adequate sample size of higher animals (eg, monkeys) is prohibitively expensive. As a result, the rat model for root resorption remains widely used.

Therapeutic ultrasound can be used to stimulate the expression of bone proteins (osteonectin, osteopontin, and bone sialoprotein) in a dose-dependent manner. The frequency and intensity of ultrasound used for imaging the human brain (7.5–20 MHz) are much higher than those used for LIPUS (1.5 MHz); therefore, the latter is much safer. Ultrasound also is being used to diagnose early stages of cancer. Thus, ultrasound is considered to be noncarcinogenic and has no known deleterious effects. These findings may help promote the development of convenient, noninvasive devices to prevent tooth resorption and improve human health and quality of life.LIPUS has a reparative effect on orthodontic root resorption in rats by modulating the OPG/RANKL ratio and osteoclast differentiation. Therefore, LIPUS may serve as a potential treatment for orthodontically induced root resorption.

PHOTOBIOMODULATION IN ORTHODONTIC TOOTH MOVEMENT

Light-accelerated orthodontics (LAO) is a technique within the scope of photo biomodulation or low-level light therapy (LLLT). The terms photo biomodulation and LAO can be interchangeably used to define the specific wavelength range, intensity, and light penetration and to differentiate from other methods utilizing light for treatment elsewhere in dentistry. LAO shows promise in producing a non-invasive stimulation of the dentoalveolar complex with a potential impact on ATP production by mitochondrial cells. The assumption is that an increase in ATP at a localized site will induce cells to undergo a remodelling process due to an elevated metabolic activity. Cytochrome oxidase c mediates ATP production. It is upregulated twofold by infrared light. During the tooth movement phase, higher ATP availability helps cells 'turnover' more efficiently leading to an increased remodelling process and accelerated tooth movement. LAO may also be functioning through an increased vascular activity, which would also contribute to the rapid turnover of the bone and is amenable to light. A number of clinical case series have suggested an enhanced impact by LAO, increased velocity of canine movement and decreased pain, and a significantly higher acceleration of retraction of treated canines. However, there are also some studies that show questionable efficacy. There have been, however, no large-scale human studies correlating the use of an LAO device, which delivers low-level light therapy to the alveolus, and the rate of orthodontic tooth movement. The aim of this study was, therefore, to determine if a LAO device reduces treatment time in the alignment phase in a specific dental malocclusion by comparing the results to a similarly matched cohort of individuals. The null hypothesis of the study was that there was no difference in treatment effects as a result of photo biomodulation.

Direct electric current effect on tooth movement

Another approach is to use 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 remodelling as shown by group of investigators. Their studies were more successful than the previous attempts 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. Several attempts were made to develop biocatalytic fuel cells to generate electricity intraorally by the use of enzymes and glucose as fuel. Further development of the direct electric device and the biocatalytic fuel cells is needed to be done so that these can be tested clinically.

Low-level laser therapy

Photo biomodulation or low level laser therapy (LLLT) is one of the most promising approaches today. Laser has a biostimulatory effect on bone regeneration, which has been shown in the midpalatalsuture during rapid palatal expansion, and also stimulates bone regeneration after bone fractures and extraction site. It has been found that laser light stimulates the proliferation of osteoclast, osteoblast, and fibroblasts, and thereby affects bone remodelling 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 low-energy laser irradiation enhanced the velocity of tooth movement via RANK/RANKL and the macrophage colony-stimulating factor and its receptor expression. Animal experiments have shown that low-level laser can accelerate tooth movement. Furthermore, clinical trial attempts were made in which different intensities of laser were used and different results were obtained. Low-level laser therapy can be a very useful technique for acceleration of tooth movement since it increases bone remodelling without side effects to the periodontium. Laser wavelength of 800 nm and output power of 0.25 mW 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 10 swas found to accelerate tooth movement at 1.3-fold higher than the control. In another study done by Kau on 90 subjects (73 test subjects and 17 controls), there was 1.12-mm change per week in the test subjects versus 0.49 mm in the control group. Having said this, there are a lot of contradictory results related to the LLLT. Therefore, more experiments are needed to differentiate the optimum energy, wavelength, and the optimum duration for usage.

Introduction to surgical approach

The use of corticotomy to correct malocclusion was first described in 1892 by L.C. Brian and G. Cunningham in 1893.The former proposed making linear corticotomies surrounding the teeth as a means of mobilizing teeth for immediate movement and presented some cases at the American Dental Society of Europe. The latter proposed the idea that immediate correction of irregular teeth is possible at the Dental Conference in Chicago. In 1931, Bichlmayr applied corticotomy-ostectomy for patients older than 16 years to correct maxillary protrusion after extraction of first premolars with palatal osteotomies and removal of alveolar bone distal to the canines using removable orthodontic appliances. In 1959, Heinrich Kole introduced a surgical procedure involving the reflection of full-thickness flaps followed by removal of the interdental alveolar cortical bone, leaving the medullary bone intact with a through-and-through subapical osteotomy. He believed that this procedure allowed for blocks of bone to move rather than the individual teeth, minimizing root resorption and retention time.

Later in 1978, Generson treated open-bite malocclusion using selective alveolar decortication in conjunction with orthodontics and eliminated the subapical osteotomy. In 1990, Gantes used a surgical technique that involved circumscribing corticotomies buccally and lingually around the six maxillary anterior teeth including buccal and lingual corticotomies over the first premolar extraction socket. In 1991, Suya reported treating 395 adult Japanese patients by means of a refinement of the above mentioned methods which substituted the subapical horizontal osteotomy by horizontal corticotomy and termed it Corticotomy-Facilitated Orthodontics (CFO). Suya believed that teeth are handles by which the bands of less dense medullary bone are moved block by block and CFO allows for moving blocks of bone rather than only individual teeth. In 2001, Wilcko and Wilcko patented and trademarked their technique as “Periodontally Accelerated Osteogenic Orthodontics” procedure. Upon raising labial and lingual full thickness flaps, interdental decortication is performed slightly into the medullary bone using a surgical bur. Flaps are sutured following application of demineralized freeze-dried bone (DFDBA) and bovine bone infused with Clindamycin phosphate solution. Orthodontic tooth movement is initiated during the week prior to the surgery and orthodontic appliances are activated every 2 weeks. The authors attributed the enhanced tooth movement to a regional acceleratory phenomenon (RAP). More specifically, a redirection of this normal physiologic bone response to insult is exploited to mobilize and accelerate tooth movement. In 2009, Lee, Chung, and Kim introduced “speedy surgical orthodontics” in order to treat maxillary protrusion in adults using a peri segmental corticotomy, a C-palatal miniplate, and a C-palatal retractor. It differs from the techniques described above in that it involves moving a corticotomized bone block of 6 maxillary anterior teeth instead blocks of a single tooth.

Historical perspective

- The use of corticotomy to correct malocclusion was first described in 1892 by **L.C. Brian** and **G. Cunningham** in 1893. The former proposed making linear corticotomies surrounding the teeth as a means of mobilizing teeth for immediate movement and presented some cases at the American Dental Society of Europe. The latter proposed the idea that immediate correction of irregular teeth is possible at the Dental Conference in Chicago.
- 1921, **Cohn-Stock** removed the palatal bone near the maxillary teeth to facilitate retrusion of single or multiple teeth
- Later, **Skogsborg** (1926) divided the interdental bone, with a procedure he called “septotomy,” and a decade later Ascher (1947) published a similar procedure, claiming that it reduced treatment duration by 20–25%.
- In 1931, **Bichlmayr** applied corticotomy-ostectomy for patients older than 16 years to correct maxillary protrusion.
- This was employed with canine retraction and first bicuspid extraction and “excoriating” cortical plates of the palatal and crestal alveolus, and cortices of the extraction sites.
- Important to note is that Bichlmayr excised significant amounts of medullary bone with his procedure.
- He redefined orthognathic surgery by reclassifying it into two categories: “major” (total or segmental maxillary and mandibular correction) or “minor” (interdental osteotomy or corticotomy) and was the first to described the corticotomy procedure to close diastemata.
- Bichlmayr’s extensive wedge-shaped bone resection was more extensive than the punctate. The latter seem more discrete and somewhat sophisticated, but the fundamentals of induced osteopenia and recalcification in retention are the same. If protracted decalcification is

desired or if the degree of tooth movement is onerous then Bichlmayr's extensive decortication is appropriate.

- **Kole** derived his work from many previous German publications, particularly that of Bichlmayr.
- When Köle popularized corticotomy in the English literature he also promoted the so-called "bony block" hypothesis. In his series of articles in 1959 postulated that the corticotomy extended into the medullary bone created bony blocks that had tooth as handles for force application.
- He relied on the reduction of cortical resistance and tried to preserve the vascular supply from the trabecular bone to the teeth. Some years later this vascular issue was the focus of criticism by **Bell and Levy**
- Köle's observations led him to surmise that the roots of the teeth were not moving through the bone, but rather the bone was moving *with* the roots of the teeth.
- The procedure was thought to eliminate the PDL mediated mechanism of tooth movement
- This independent movement was thought possible due to the connection of only the medullary bone. Kole believed in movement of the bone block in its entirety and demonstrated a decrease in root resorption, without observing any other sequelae.

Criticism to Kole's technique

- Reichenbach was very wise in concern for alveolar blood supply, because the aggressive surgery described certainly poses risks.
- Bell and levy (1972) Their histological study showed the risk of this type of procedure (full mucoperiosteal detachment plus deep cutting of medullar bone) to the vascularity of a dental pulp and surrounding medullar bone. They demonstrated distinct avascular zones that progressively recovered 3 weeks after surgery, except for the central incisor.

Duker modification

- Duker, 1975 used Kole's basic technique on beagle dogs to investigate how rapid tooth movement with corticotomy affects the vitality of the teeth and the marginal periodontium. The health of the periodontium were preserved by avoiding the marginal crest bone during corticotomy cuts. It was concluded that neither the pulp nor the periodontium was damaged following orthodontic tooth movement after corticotomy surgery.

- The results helped to substantiate the belief regarding the health of crestal bone in relation to the corticotomy cuts. Design of the subsequent techniques has taken this into consideration; the interdental cuts are always left at least 2 mm short of the alveolar crestal bone level.
- Generson (1978) Generson*et al.* (1978) applied corticotomy to the treatment of apertognathia
- The authors' concern for the possibility of compromising the blood supply to the teeth was settled when the authors make a point that "the bony cuts were made only through the cortex."

Bony block confrontation

- Rynearson (1988) tested Köle's hypothesis that, under orthodontic force, teeth in a corticotomy-treated segment move as a tooth– bone unit.
- Utilizing implanted radiopaque pins and bone labeling, he found no evidence of movement of the cortical plates and concluded that the corticotomy procedure, albeit efficacious, did not effect a mechanical movement of a tooth–bone unit, but rather elicited a facilitation of normal physiologic tooth movement metabolism.
- The fallacy of "bony block" movement was finally beginning to be challenged, at least for space-closing procedures. However, the exact cell- and molecular-level mechanism responsible for the facilitated tooth movement was still an enigma.
- Seven years later Yaffe*et al.* (1994) added a very important clue to unraveling that mystery by reporting a robust RAP response in the jaw bone of rats to a simple mucoperiosteal flap reflection. They documented not only massive decalcification of the alveolar bone, but also a widening of the periodontal ligament space.
- The demineralized zone was observed as early as 10 days, and the alveolar bone returned to control levels 120 days after surgery. The authors also suggested that RAP might be responsible for tooth mobility and bony dehiscence formation where the bone was thin
- Suya (1991) reported corticotomy-assisted orthodontic treatment of 395 adult Japanese patients.

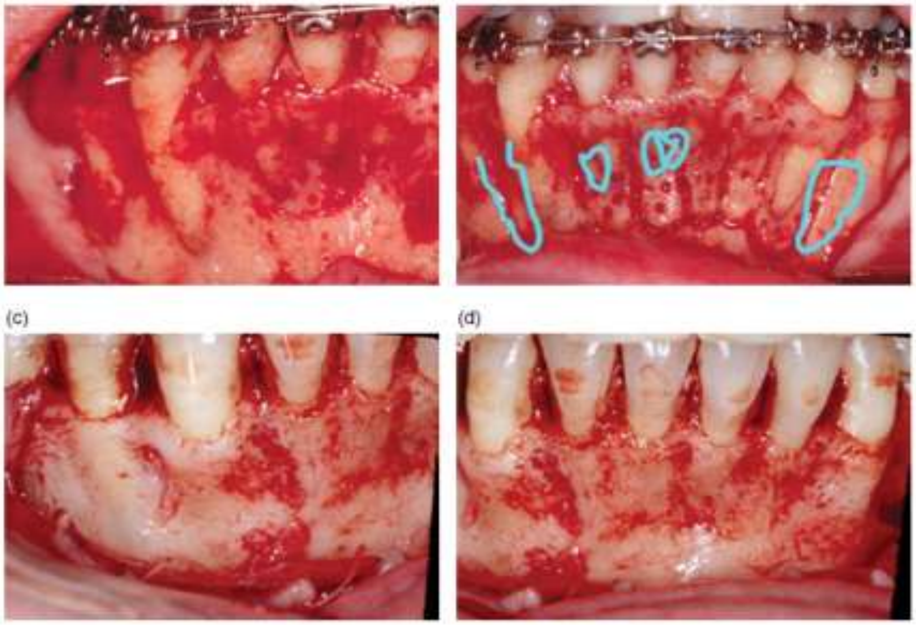
- Suya's technique differed from Kole's with the substitution of a subapical horizontal corticotomy cuts in place of the horizontal osteotomy cut beyond the apices of the teeth.
- Fixed orthodontic appliances were used. Some cases were completed in 6 months, other cases were completed in less than 12 months.
- Suya contrasted his technique with conventional orthodontics in being less painful, producing less root resorption, and exhibiting less relapse. Outstanding results and extreme patient satisfaction with corticotomy procedures were reported.Completing tooth movement in 3–4 months were recommended, after which time the edges of the blocks of bone would begin to fuse together.
- Suya (1991) stimulated significant academic interest in Asia (Chung *et al.*, 2001; Hwang and Lee, 2001; Kim and Tae, 2003) for nearly two decades.
- Chung *et al.* (2001) in Asia also reported a decortication assisted orthodontic method.
- Hwang and Lee (2001) introduced a technique for intrusion of overerupted molars, using a combination of decortication and magnets.
- Kim and Tae (2003) moved teeth facilitated by decortication, referring again to the phenomenon as "distraction osteogenesis," and citing it as a "new paradigm in orthodontics." They removed part of the cortical bone, which resulted in "a speedy rate" compared with "conventional" OTM.
- Park et al. (2006) and Kim et al. (2009) reported an interesting technique that is often contrasted with flap reflection methods. They used a method of surgical incision called "corticision," wherein a reinforced scalpel is used as a thin chisel to separate the interproximal cortices trans-mucosally, without a surgical flap reflection. This transmucosal incisional manipulation, similar in effect to TMP of alveolar bone, minimizes morbidity but may fail to recruit significant RAP, which occurs simply with mucoperiosteal flap reflection.

Surgically driven accelerated orthodontics

AOO/PAOO

[*Accelerated Osteogenic Orthodontic Tooth Movement/Periodontally Accelerated Osteogenic Orthodontic Tooth Movement]*

Described by Wilcko and co-workers It is a combination of a selective decortications facilitated orthodontic technique and alveolar augmentation. This method claims to have several advantages. These include a reduced treatment time, enhanced expansion, differential tooth movement, increased traction of impacted teeth and, finally, more post-orthodontic stability. With this technique, one is no longer at the mercy of the preexisting alveolar volume, and teeth can be moved 2 to 3 times further in 1/3rd to 1/4th the time required for traditional orthodontic therapy.



In PAOO technique, cortical bone is scarred surgically on both labial and lingual sides of the teeth to be moved followed by grafting. The patient is seen every 2 weeks, and the rapid tooth movement produced after PAOO is substantially different than periodontal ligament cell-mediated tooth movement. Recent evidence suggests a localized osteoporosis state, as a part of RAP may be responsible for the rapid tooth movement after PAOO. Shih and Norrdin demonstrated that when intraoral cortical bone was injured by corticotomy, RAP accelerated the normal regional healing processes by transient bursts of hard- and soft-tissue remodeling. Goldie and King induced an osteoporosis state by depleting calcium intake in lactating rats and found an increase in the orthodontic tooth movement. Most recently, Wilcko et al. showed radiographic evidence of an osteoporosis state in an alveolar bone treated with corticotomy, a characteristic seen in RAP. Sebaoun et al concluded that selective alveolar decortication induced increased turnover of alveolar spongiosa, and the activity was localized; dramatic escalation of demineralization-remineralization dynamics is the likely biologic mechanism underlying rapid tooth movement following selective alveolar decortication.

Additionally, the researchers found comparable tooth movement acceleration with small, round cortical perforations and with corticotomy cuts in a split-mouth design. This finding further supported that RAP is responsible for rapid orthodontic tooth movement.

Case Selection

PAOO can be used to accelerate tooth movement in most of the cases requiring orthodontic treatment. It has been shown to be particularly effective in treating moderate to severe crowding, in Class II malocclusions requiring expansion or extractions, and mild Class III malocclusions. PAOO can be used in both maxillary and mandibular arches. However, the decision regarding the site of PAOO can be made based on clinical judgement. For example, maxillary expansion generally requires more time than correction of mild mandibular anterior crowding. So a case with a narrow maxilla and mild anterior crowding may benefit with PAOO in the maxilla and traditional orthodontic therapy in the mandibular arch. On the other hand, a case of bimaxillary dentoalveolar protrusion requiring extractions in both the arches can be treated with PAOO to hasten the result in both the arches. Having both arches corrected in a similar time frame is ideal.

Surgical technique

The surgical technique for PAOO consists of 5 steps viz. raising of flap, decortication, particulate grafting, closure and orthodontic force application.

Flap Design

A proper flap design is essential for the success of any surgical procedure. In PAOO also the flap should provide proper access to the alveolar bone wherein corticotomies are to be performed. Preservation of the gingival form is also important for proper esthetic appearance. The basic flap design is a combination of a full thickness flap in the most coronal aspect of the flap with a split-thickness dissection performed in the apical portions. The flap should be extended beyond the corticotomy sites mesially and distally so that vertical releasing incisions are not required. For esthetic purposes the papilla between the maxillary central incisors should be preserved on the labial and palatal aspects. Access to the labial alveolar bone in this area is achieved by “tunneling” from the distal aspect.

Interseptal alveolar surgery

Interseptal alveolar surgery or distraction osteogenesis is divided into distraction of PDL or distraction of the dentoalveolar bone; example of both is the rapid canine distraction. The concept of distraction osteogenesis came from the early studies of limb lengthening. Also from surgical treatments of craniofacial skeletal dysplasia, this concept was later adapted in relation to the rapid tooth movement. In the rapid canine distraction of PDL, the interseptal bone distal to the canine is undermined surgically at the same time of extraction of the first premolars, thus, this will reduce the resistance on the pressure site. In this concept the compact bone is replaced by the woven bone, and tooth movement is easier and quicker due to reduced resistance of the bone. It was found that these rapid movements are during the initial phases of tooth movement especially in the first week as show in . In this technique the interseptal bone is undermined 1 to 1.5 mm in thickness distal to the canine after the extraction of the first premolar, and the socket is deepened by a round bur to the length of the canine. The retraction of the canine is done by the activation of an intraoral device directly after the surgery. It has been shown that it took 3 weeks to achieve 6 to 7 mm of full retraction of the canine to the socket of the extracted first premolars. Rapid canine distraction of the dentoalveolar bone is done by the same principle of the distraction of PDL, with the addition of more dissection and osteotomies performed at the vestibule as shown in following figure. In all the studies done, both techniques accelerated tooth movement with no evidence of significant root resorption, ankylosis, and root fracture. However, there were contradictory results regarding of the electrical vitality test of the retracted canines. Liou reported 9 out of 26 teeth showed positive vitality, while Sukurica reported that 7 out of 20 showed positive vitality after the sixth month of retraction. So, there are still some uncertainties regarding this technique.

Piezocision technique

One of the latest techniques in accelerating tooth movement is the Piezocision technique. Dibart was among the first to apply the Piezocision technique which starts with primary incision placed on the buccal gingiva followed by incisions by Piezo surgical knife to the buccal cortex. Piezocision technique did not cause any periodontal damage as reported by Hassan. Another benefit of this technique is that it can be used with Invisalign, which leads to a better aesthetic appearance and less treatment time as reported by Keser. Piezocision is a promising tooth acceleration technique because of its various advantages on the periodontal, aesthetic, and orthodontic aspects.

Piezotome-corticision Procedure [Efficiency of Piezotome-Corticision Assisted Orthodontics in Alleviating Mandibular Anterior Crowding - A Randomized Controlled Clinical trial Rana Mehr] Subjects underwent the piezotome-corticision procedure at the University of Connecticut Orthodontic Clinic. This procedure was performed by one of the authors (KA) according to the technique explained by Dibart et al. Panoramic radiographs were utilized to assess the long axes of the teeth and root proximity prior to the procedure. Local anaesthetic was administered using 2% Lidocaine with 1:100,000 Epinephrine. The depth of gingival tissue was determined by bone sounding using a Williams periodontal probe. A #15C Bard-Parker scalpel was used to make three incisions through the gingiva, 4mm below the interdental papilla to preserve the coronal attached gingiva. These three vertical incisions were made interproximally between mandibular canines and lateral incisors, and central incisors on the labial aspect of the mandible through the gingiva and the underlying bone. The incisions were 4mm in length. After the incisions were made, the gingiva was slightly elevated laterally to visualize the bone and roots. A piezo surgery knife (BS1 insert, Satelec Acteon Group), which is an ultrasonic microsaw, was used to create the cortical alveolar incisions to a depth of 1mm within the cortical bone. In a study by Farnsworth et al. using cone beam computed tomography, the cortical bone thickness between mandibular lateral incisor and canine in an axial slice taken from the thinnest portion of the cortical bone was measured. The vertical level of the measurement was established 4mm apical to the crest of the alveolar bone by using a coronal slice. The mean cortical thickness was reported to be 1.2 mm in adults ranging 20 to 45 years old. The depth of the cortical incision was limited to 1mm for a safety margin in these severely crowded cases, by ensuring that the BS1 insert penetration does not exceed the measured depth of the gingiva plus 1mm of cortical incision. Postoperatively, subjects were advised to rinse with chlorhexidine mouthwash twice a day for one week and take acetaminophen as needed. All experimental subjects were contacted the day after the procedure to ensure no complications with surgery and were followed up one week post-surgery to assess for signs of infections.

Decortication

Decortication refers to the removal of the cortical portion of the alveolar bone. However, it should be just enough to initiate the RAP response and should not create movable bone segments. After flap elevation, decortications of bone adjacent to the malpositioned teeth is performed by using low-speed round burs under local anesthesia. In the PAOO procedure, decortication is performed

at clinical sites without entering the cancellous bone, avoiding risk of damage to underlying structures, such as the maxillary sinus and the mandibular canal. The corticotomies may also be achieved with a piezoelectric knife. The corticotomies are placed on both the labial and lingual(palatal) aspects of the alveolar bone.

Particulate Grafting

The materials most commonly used for grafting after decortication are deproteinized bovine bone, autogenous bone, decalcified freeze-dried bone allograft, or a combination thereof. Grafting is done in most areas that have undergone corticotomies. The volume of the graft material used is dictated by the direction and amount of tooth movement predicted, the pretreatment thickness of the alveolar bone, and the need for labial support by the alveolar bone. A typical volume used is 0.25 to 0.5 ml of graft material per tooth.

Closure Techniques

The flap should be closed using non resorbable interrupted sutures without creating excessive tension. No packing is required. The sutures are usually left in place for 1 to 2 weeks.

Timing of Orthodontic Treatment

The placement of orthodontic brackets and activation of the arch wires are typically done the week before the surgical aspect of PAOO is performed. However, if complex mucogingival procedures are combined with the PAOO surgery, the lack of fixed orthodontic appliances may enable easier flap manipulation and suturing. After flap repositioning, an immediate heavy orthodontic force can be applied to the teeth and in all cases initiation of orthodontic force should not be delayed more than 2 weeks after surgery. A longer delay will fail to take full advantage of the limited time period that the RAP is occurring. The orthodontist has a limited amount of time to accomplish accelerated tooth movement. This period is usually 4 to 6 months, after which finishing movements occur with a normal speed. Given this limited “window” of rapid movement, the orthodontist will need to advance arch wire sizes rapidly, initially engaging the largest arch wire possible.

Indications and Clinical Applications

Several clinical applications for PAOO have been reported. Corticotomy was used to facilitate orthodontic tooth movement and to overcome some shortcomings of conventional orthodontic treatment, such as the long-required duration, limited envelope of tooth movement and difficulty of producing movements in certain directions. These applications include the following:

a. Resolve Crowding and Shorten Treatment Time
b. Accelerate Canine Retraction after Premolar Extraction
c. Enhance Post-Orthodontic Stability
d. Facilitate Eruption of Impacted Teeth
e. Facilitate Slow Orthodontic Expansion
f. Molar Intrusion and Open Bite Correction
g. Manipulation of Anchorage

Contraindications and limitations

Patients with active periodontal disease or gingival recession are not good candidates for PAOO. In addition, PAOO should not be considered as an alternative for surgically assisted palatal expansion in the treatment of severe posterior cross-bite. PAOO also should not be used in cases where bimaxillary protrusion is accompanied with a gummy smile, which might benefit more from segmental osteotomy.

Complications and Side Effects

Although PAOO may be considered a less-invasive procedure than osteotomy-assisted orthodontics or surgically assisted rapid expansion, there have still been several reports regarding adverse effects to the periodontium after corticotomy, ranging from no problems to slight interdental bone loss and loss of attached gingiva, to periodontal defects observed in some cases with short interdental distance. Subcutaneous hematomas of the face and the neck have been reported after intensive corticotomies. In addition, some post-operative swelling and pain is expected for several days. No effect on the vitality of the pulps of the teeth in the area of corticotomy was reported. Long-term research on pulpal vitality after rapid movement has not been evaluated in the literature. In an animal study, Liou et al. demonstrated normal pulp vitality after rapid tooth movement at a rate of 1.2 mm per week. However, pulp vitality deserves additional investigation. It is generally accepted that some root resorption is expected with any orthodontic tooth movement. An association between increased root resorption and duration of the applied force was reported. The reduced treatment duration of PAOO may reduce the risk of root resorption. Ren et al. reported rapid tooth movement after corticotomy in beagles without any associated root resorption or irreversible pulp injury. Moon et al. reported safe and sufficient maxillary molar intrusion (3.0 mm intrusion in two months) using corticotomy combined with a skeletal anchorage system with no root resorption. Long-term effect of PAOO on root resorption requires further study.

Corticotomy

A corticotomy is defined as a surgical procedure in which the cortical bone is cut, perforated, or mechanically altered without involvement of the medullary bone. In laboratory studies, when a surgical incision was made into the head of the tibia in rabbits, new bone, including trabecular bone, formed around the incision area as a result of increased bone turnover. Furthermore, orthodontists have long noted increased rates of tooth movement following orthognathic surgical procedures, though this effect is usually attributed to a postoperative acceleration of bone remodelling. A consequence of this observation is that maxillary corticotomy is now a routine procedure for surgically assisted rapid palatal expansion. However, alveolar corticotomy to enhance the rate of tooth movement has developed more slowly, largely because of concerns about periodontal outcomes. The most widely known benefit of the modern corticotomy procedure is faster tooth movement, with some authors claiming it to be three to four times quicker than traditional orthodontic movement. Shorter treatment time may provide greater motivation for patients. More importantly, these procedures can modify the dentoalveolar complex so that the teeth, alveolar bone, and skeletal components can be appropriately addressed for maximising ideal functional and aesthetic relationships. In addition, these techniques could reduce root resorption and provide a more stable result than traditional cell-mediated tooth movement alone. The shortened treatment time reduces the risk of periodontal inflammation, dental caries, and decalcification. The correction of numerous interdisciplinary dental-facial problems will result in arches that are perceived to be more aesthetically appealing in modern society. It was believed that the rapid tooth movement after corticotomy surgery was due to the movement of small outlined blocks of bone with the teeth as handles. The resistance of the cortical layer of bone was presumably eliminated with the circumscribing corticotomy cuts. The only resistance to the tooth movement would thus be provided by the less dense medullary bone. It was thought that this could overcome the slow PDL-mediated process of traditional orthodontics because the tooth–PDL complex was being moved with the block of bone and not through the bone. Corticotomy was often a highly morbid, hospital-based procedure requiring surgical cuts to be made entirely through the buccal and lingual alveolar process periapically.

The old 19th and 20th century version of the corticotomy procedure risked the devitalisation of teeth as well as alveolar necrosis. Moreover, some surgeons made such deep bony cuts interproximally that the orthodontist considered it to be regional orthognathic surgery. Since this unrefined technique lacked an evidence base and had a high morbidity, it justifiably enjoyed rather little popular support. Iino et al. found that the insult of circumscribing corticotomy cuts alone does not elicit an osseous response that is sustainable enough to permit tooth movement through a large thickness of bone in the mesiodistal orientation of the alveolus. Thus, they suggested that bone thinning be accomplished with an ostectomy through the entire thickness of the alveolus to include the labial and lingual cortical plates and interspersed medullary bone. In 2001, Wilcko et al. reported a revised corticotomy-facilitated technique that included periodontal alveolar augmentation, called accelerated osteogenic orthodontics (AOO) or periodontally AOO technique (PAOO). This technique demonstrated acceleration of treatment in two cases, reducing the usual overall treatment time by two-thirds AOO involves a combination of “bone activation” (selective alveolar decortication, ostectomies, and bone thinning with no osseous mobilisation), alveolar augmentation using particulate bone grafting material, and orthodontic treatment. Facial and lingual surgical flaps are elevated and the cortical bone adjacent to the teeth to be moved is scored with a surgical bur penetrating barely into medullary bone. AOO technique employs a bone graft over the bleeding cortical bed, but the graft is not essential to induce alveolar osteopenia. The principal objective of the AOO surgery is the creation of a relatively thin layer of bone (approximately 1.5 mm) over the root prominence in the direction of the intended tooth movement.

The design of the corticotomy cuts and perforations is not important but only needs to perforate the cortical layer of bone and extend into the superficial aspect of the medullary bone.

The corticotomy surgery acts as a noxious insult to the area, causing the induction of the alveolar structures into a more pliable condition favouring rapid tooth movement. There is a substantial increase in alveolar demineralisation resulting in a transient and reversible condition (osteopenia). Calcium is released from alveolar bone, resulting in a decrease in bone mass (mineral content or density) but no change in bone volume. Longitudinal tunnelling takes place in cortical bone, while both surface resorption and osteocyticosteolysis converts as much as 50% of local trabecular bone to osteoid in six weeks. The osteopenia enables rapid orthodontic tooth movement because teeth are supported by and moved through trabecular bone. Bogoch found a fivefold increase in bone turnover in a long bone adjacent to a corticotomy surgery site. The rapid tooth movement associated with corticotomy-facilitated orthodontics is more likely the result of a demineralisation/remineralisation process consistent with the initial phase of the regional acceleratory phenomenon, namely an increase in cortical bone porosity and a dramatic increase of trabecular bone surface turnover due to increased osteoclastic activity. As long as tooth movement continues, the RAP is prolonged. When RAP dissipates, the osteopenia also disappears. When orthodontic tooth movement is completed and retainers are delivered, an environment is created that fosters alveolar remineralisation. Recently, Binderman and co-worker suggested that the major stimulus for the alveolar bone remodelling that enabled periodontally accelerated osteogenic orthodontics was not RAP. Instead, the stimulation was attributed to the detachment of the bulk of dentogingival and interdental fibres from the coronal part of the root surfaces, which the authors considered to be sufficient to stimulate alveolar bone resorption and to lead to widening of the periodontal ligament space. This would allow accelerated osteogenic orthodontic movement of teeth.

Additionally, the fiberotomy would transiently disrupt the positional physical memory of the dentition, allowing accelerated tooth movement and reducing relapse. The basis of the argument by Binderman and co-workers is that the episode of osteoclastic alveolar bone and soft tissue remodelling is attained through the elevation of a full-thickness mucoperiosteal flap alone, without surgical wounding of the cortical bone. Additionally, in the rat model, alveolar bone resorption has been shown to occur when full-thickness flap surgery is performed by a coronal approach (sulcular incision), whereas an apical surgical approach, without disruption of the gingival attachment to the root surface, does not result in significant alveolar bone remodelling. Binderman and co-workers concluded that mucoperiosteal flap surgery could be separated into two procedures: (1) surgical detachment of dentogingival and interdental fibres, which produces a strong signal for osteoclastic bone resorption on the inner aspect of the PDL facing the tooth, and (2) separation of mucoperiosteum from bone and corticotomy, which produces a burst of regional bone remodelling that is consistent with RAP. It should also be noted, however, that at the control sites in the experiment, where only surgical incisions were performed without elevation of the periosteum, nonalveolar bone resorption was observed. Additionally, although less alveolar bone height loss was found at apical approach sites, which was expected because the gingival margin was not involved, evidence of bone remodelling was present histologically on the surface of the alveolar bone. Thus, surgical incisions, which are comparable to a fiberotomy procedure, did not result in alveolar bone resorption and elevation of the mucoperiosteum (coronally and apically) results in alveolar bone changes, though possibly only on the surface of the alveolar bone.

Selective alveolar decortication is conventionally performed on buccal and lingual/palatal bone plates using rear-vented high-speed, rotary surgical instrumentation and carbide burs under copious saline irrigation. Adequate irrigation is essential to minimize intraoperative thermal bone damage and optimize postoperative bone healing. Any fine surgical bur of choice can be used to perform thorough decortication all over the area. Recently, piezoelectric instrumentation has also been used to perform decortication and demonstrated to be effective by some clinicians. However, at the time of publication of this material, the short and long-term efficacy in causing comparable RAP to conventional surgery and the predictability of clinical outcomes is unknown and hasnot been widely studied or reported. Decortication refers to surgical violation of the cortex and purposeful entry into medullary bone. The specific design or type of decortications (e.g., holes, lines, grooves, any geometrical figure) is not relevant. Entry into medullary bone is a prerequisite, and this is verified by evidence of bleeding in the decortications sites. Care should be taken to stop 2–3 mm short of the alveolar crest, as violation of this area can cause complications with soft tissue esthetics and loss of periodontal support. The clinician must also respect other vital anatomic structures, such as maxillary sinus, inferior alveolar nerve canal, and dental roots, which could be damaged by overzealous instrumentation. Care should be taken not to injure the anterior loop of the inferior alveolar nerve that could extend several millimeters (6 mm loop) mesial to the mental foramen and is usually positioned just beneath the buccal cortical plate in this region. Additional cortical perforation can be made at selected, safe areas to increase blood supply to the graft material. The decortications performed is interdentally in vertical fashion paralleling the roots. Many a time, owing to dental crowding, roots have very close proximity to each other, and thus extreme caution must be exercised in order to avoid iatrogenic damage.

Pre-operative considerations

Medical and surgical history should be obtained and considerations for surgery are similar to routine indications and clearance for intraoral dentoalveolar procedures. A panoramic radiograph is recommended to evaluate the maxillary sinus, nasal cavities, and other skeletal and dental structures from a general perspective .Full-mouth periapical X-rays are recommended to evaluate root proximity and other structures, such as periodontal health and status, lamina dura, and so on. Cone beam computed tomography scans are becoming widely popular and have the benefit of a precise evaluation of the thickness of buccal and lingual cortical plates and their intimate relationship to the roots of teeth, besides giving all the information that plain films would give. Orthodontic appliances are placed approximately1–2 weeks week prior to the surgery. Standard brackets, arch wires, and normal orthodontic force level can be used. Surgery is performed in an office setting with or without sedation, depending on patient and doctor preference. The surgical armamentarium required is similar to any intraoral, minor dentoalveolar surgical, or implant surgery procedure (hand instruments, rotary instrumentation ,piezoelectric module, bone graft materials, sutures, etc.). Local anesthetic with vasoconstrictor should be infiltrated buccally/labially and palatally at least 7 min prior to incision to maintain optimal hemostasis. Use of appropriate antibiotics with adequate oral flora coverage (oral amoxicillin or clindamycin) and oral chlorhexidine rinse is recommended prior to surgery. These are usually started approximately1 h prior to the procedure and continued for 1–2 weeks post-operatively.

Incision design

A no. 15 Bard–Parker surgical blade is used on a suitable scalpel handle and held at a slight angle to the teeth. For four quadrant cases, a smooth continuous stroke is made in the gingival sulcus from first molar to first molar crossing the midline in one arch. A vertical releasing incision can then be made behind the first molar. Vertical incisions are not recommended in the anterior region because of esthetic reasons. Although some cases can be performed with only sulcular incisions, vertical releases ensure increased access and ease of flap reflection, and may especially be indicated for those practitioners who are less experienced with surgical procedures. The authors also recommend that, in the anterior midline region, the incision be designed in a manner that avoids incising the triangular papilla on the labial mucosa between the central incisors (papilla-sparing incision) (Figure 3.1a).The above-mentioned incision design is indicated for conventional surgery; many other modifications have been proposed including vertical separate incisions for piezoelectric instrumentation (Figure 3.1b).

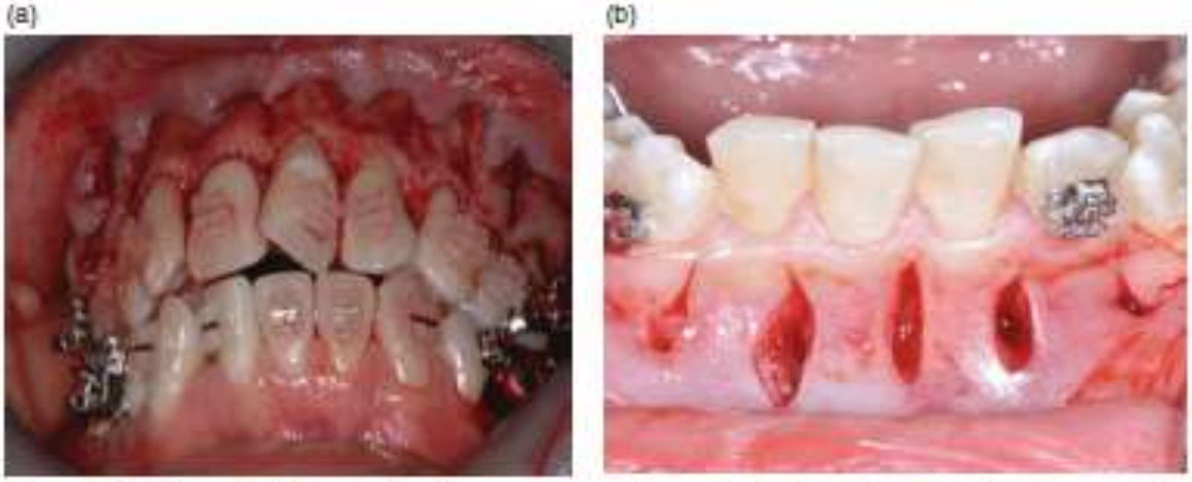


Figure 3.1 (a) A sulcular incision has been made around the necks of the individual teeth in the areas to be decorticated. The flap has been reflected apically to demonstrate bone exposure. (b) Modified incisions have been proposed by some authors, especially using piezoelectrical instrumentation. This photograph depicts five vertical incisions in the areas where piezoelectric instrumentation would be used to decorticate the bone in the interdental regions.

Flap reflection and exposure

The reflection of a full-thickness mucoperisoteal flap is carried out next. The sharp end of a no. 9 Molt’s periosteal elevator or Woodson is slipped underneath the papilla in the area of the incision and is turned laterally to reflect the papilla away from the underlying bone(Figure 3.2). This technique is then utilized for the remainder of the flap, extending laterally. Care should be exercised not to damage any of the neurovascular bundles exiting the bone and stay subperiosteal so as not to disturb deeper muscle attachments. After this initial reflection of the free edge of the flap, the broad end of the periosteal is often then used to reflect the entire mucoperiosteal flap, and this exposes the alveolar housing and bone plates (Figure 3.2). This technique assures an atraumatic, hemostatic reflection of the mucoperiosteal flap. Once the flap has been reflected, a Seldin , Minnesota, or similar retractor should be used subperiosteally to hold the flap to its reflected position and maintain adequate exposure and protection for soft tissues. Clinicians should be careful not to force the retractor into the soft tissue flap, but instead place tissue retractors firmly against the bone.

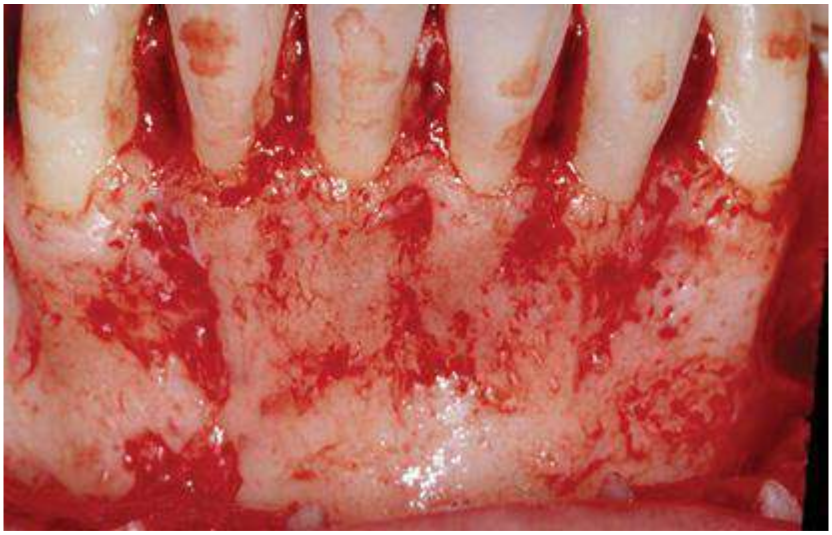


Figure 3.2 A full-thickness mucoperiosteal flap has been reflected exposing the anterior mandible, which is the area to be decorticated. Care should be taking while reflecting this flap, as many areas may have bony dehiscence and root exposures and overzealous instrumentation may cause complications such as root resorption. Flaps should be retracted with standard instrumentation, such as Seldin, Minnesota, or other retractors.

Bone Grafting

The use of bone grafting is controversial. Some practitioners use it universally on all cases, whereas others do not recommend its use in any case. Still others recommend using a bone graft to augment the alveolus if there are areas of dehiscence of roots and also if the native bony cortical plates are thin (<2 mm thickness on a cone beam computed tomography scan).We recommend the use of a particulate bone graft in all cases as we feel it promotes bone healing and, in our experience, improves the clinical and radiographic appearance of the soft and hard tissues of the periodontium. If grafting is elected, any US Food and Drug Administration-approved, good-quality bone grafting material sourced from a reputable tissue bank can perhaps be used. We recommend use of either human freeze-dried bone or bovine allografts and have found both to be successful and comparable in clinical outcomes. Grafting material is placed over the exposed decorticated area directly without any membrane use (Figure 3.4). Approximately 0.5–1 cm3of particulate graft material is required for each tooth-bearing segment that is decorticated. Care Should be taken not to a place an excessive amount of bone graft volume as it may interfere with proper flap repositioning and suturing.

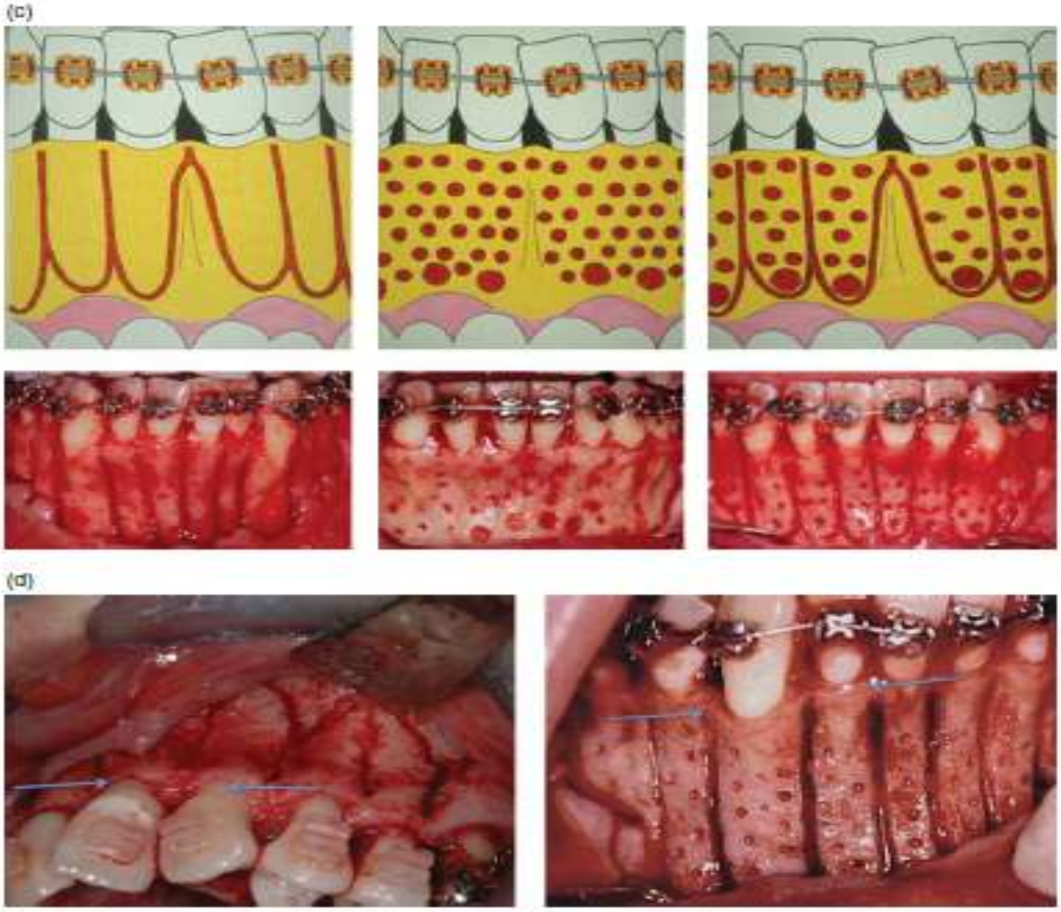


Figure 3.3 (Continued)

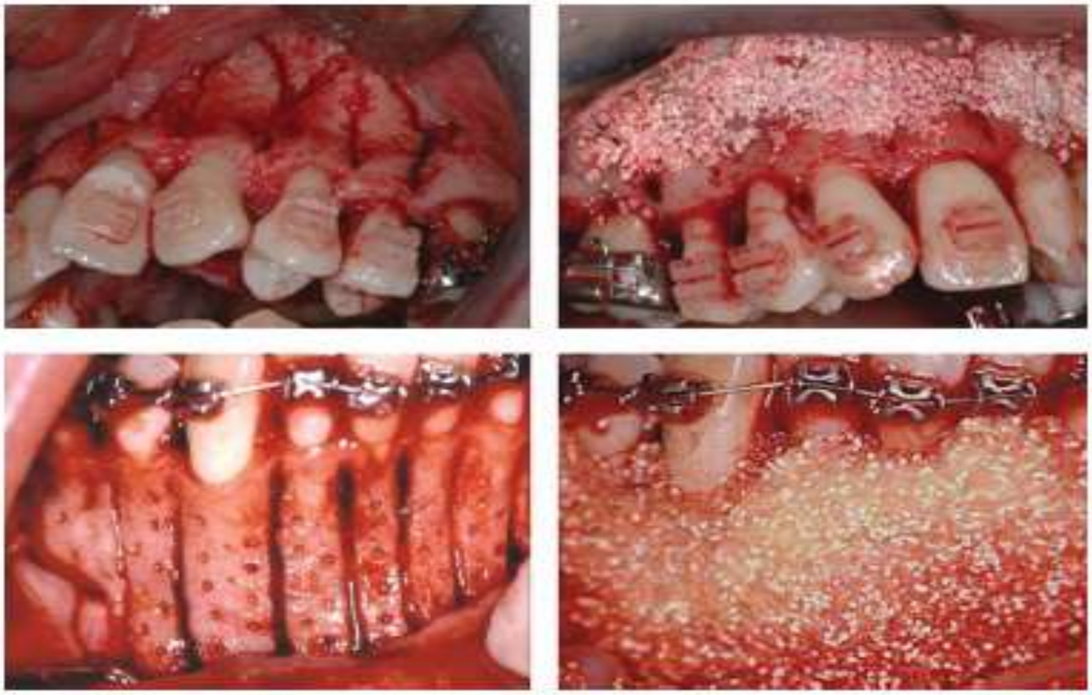


Figure 3.4 When indicated, bone grafting of the alveolus is performed with particulate bone grafts. Some clinicians prefer soaking the graft with antibiotic solution, but this is not mandatory. The maxillary quadrant has been bone grafted with bovine bone graft and the mandibular quadrant has been grafted with demineralized human freeze-dried bone. Approximately 0.5–1 cm^3 of particular bone graft is required for each tooth-bearing segment. Care should be taken not to overpack, as this could compromise a tension-free closure of the soft tissues overlying the decortication and grafted areas.

Closure

Once the above surgical steps have been completed, the flap is then returned to its original position and reapproximated with resorbable sutures (3-0 or 4-0 vicryl or chromic gut are commonly used, although any other suture material is also acceptable). The suture type(interrupted, mattress, continuous) is not criticalin our opinion; instead, what is critical is that the closure be tension-free. To achieve this, periosteal scoring under the flap may be required in some cases if closure is found to be difficult. When passing the needle through the tissue, the needle should enter the surface of the mucosa at a right angle, to make the smallest possible hole in the mucosal flap. If the needle passes through the tissue obliquely, the suture will tear through the surface layers of the flap when the suture knot is tied, which results in greater injury to the soft tissue. It is also important to ensure that adequate amount of tissue is taken when passing the needle through the flap, to prevent the needle or suture from pulling through the soft tissue flap. Once the sutures are passed through the mobile flap and immobile lingual tissue, they are tied with an instrument tie. Care must be taken not to excessively tighten the knots to avoid ischemia of flap margins. A reliable clinical indicator offlap ischemia is blanching of wound margins, which if it occurs should be dealt with immediately by re-performing the suture of the area.

Post-surgical considerations

Standard instructions that are used for minor oral surgical procedures are recommended. Oral antibiotics (7–10 days) and antimicrobial mouthwash (oral chlorhexidine for 1–3 weeks) should be routinely prescribed for all patients. Moderate-strength analgesics are generally required for the first 3–7 days, and then over-the-counter pain medications can be used. Rapid orthodontic treatment is then initiated within a few days (range 3–14 days) of surgery and continued until correction of malocclusion. It is imperative to remember that the goal is to complete as much orthodontic treatment as rapidly as possible while RAP peaks and exists. Thus, patients should be evaluated by their orthodontist weekly after surgery, and patients undergo standard orthodontic retention treatment after treatment.

Indications

1. Decreasing the duration of orthodontic treatment in patients who are undergoing conventional, nonsurgical orthodontic therapy (treatment of dental malocclusions with orthodontics alone)
2. Expanding the alveolar basis, therefore reducing the need for premolar extractions and strengthens the periodontium, lowering the risk for periodontal damage during and after treatment .
3. Selectively altering the differential anchorage among groups of teeth, hastening and facilitating the movement of teeth that have to be moved and diminishing the counter effect in the teeth that should not be moved.
4. As a tool in multidisciplinary treatment, including managing of partial edentulism in adult and growing patients .
5. Modifying the lower third of the face.
6. Adjunctive measure in facilitating treatment of impacted teeth.

7. Decreasing the duration of pre-operative orthodontic treatment in patients undergoing conventional, combined surgical–orthodontic therapy (treatment of skeletal malocclusions with orthognathic surgery).
8. Alternative to orthognathic surgery for combined surgical–orthodontic management of select dentoskeletal malocclusions.
9. Salvage technique for the management of post-orthognathic, occlusion-related complications.
10. Management of clinically refractory orthodontic dental conditions.

Contraindications

These are similar to those for any minor oral surgery or periodontal surgery procedures, especially when related to conditions affecting systemic health and illness (cardiac, endocrine, musculoskeletal, etc.). Additionally, SAD may be contraindicated in certain local disease states, such as active periodontitis or systemic conditions (e.g., uncontrolled osteoporosis). It may also have an increased complication rate in patients who have a history of use of certain medications (e.g., nonsteroidal anti-inflammatory drugs, immunosuppressive medications, steroids, bisphosphonates) and radiation therapy to the maxillofacial region.

Advantages

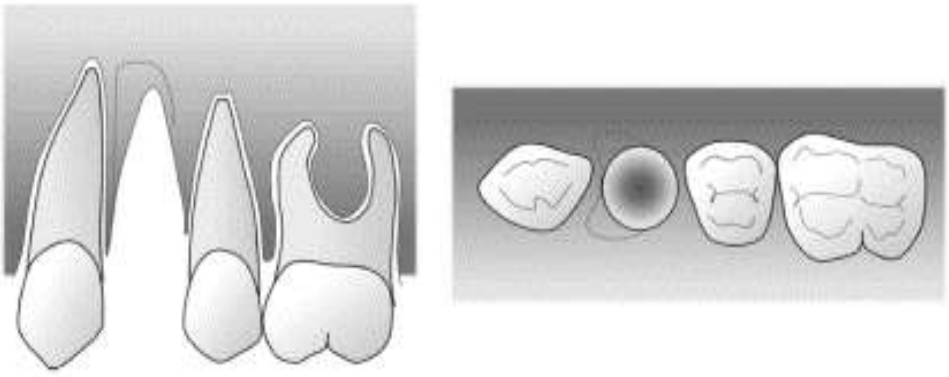
1. Minimally invasive surgery:
    i. Decreased post-operative discomfort (compared with orthognatic surgery such as surgically assisted rapid palatal expansion (SARPE)).
    ii. Minimal complications.
2. Eliminates the need for dental extractions in many patients.
3. Improved post-surgical outcomes:
    i. Less root resorption during active orthodontic movement due to decreased resistance of cortical bone.
    ii. Improved quantity and quality of periodontium; more bone support due to the addition of bone graft.
4. Decreased duration of treatment:
    i. Orthodontic treatment;
    ii. Total treatment time;

  iii. Decrease of length-related side effects of orthodontics due to plaque accumulation, such as decay and periodontal disease.

5. Ability to perform surgery in an office setting:
   i. Improved efficiency.
   ii. Decreased costs.
   iii. No requirement for hospitalization.

Recent studies

Makki et al studied the outcome stability of patients treated with corticotomy and grafting, concluded that more stable results were yielded after 10 years.(AO, accepted 2014, unpublished)Chidchanok Leethanakul et al from Thailand evaluated interseptal bone reduction combined with the use of a conventional orthodontic fixed appliance to accelerate canine retraction. RAP induced peaked at 1 to 2 months and lasted for about 6 months after the completion of the surgical procedure.



Case report for molar protraction by use of corticotomy and TAD was published by Flavio Uribe et al. in 2013Dogan Dolanmaz et al (2010) reported Orthodontic Treatment of an Ankylosed Maxillary Central Incisor through Osteogenic Distraction. Osteotomy cuts were placed and force for distraction was applied through orthodontic mechanics instead of bulky intraoral distractors.

CONCLUSION

Recent animal and clinical studies have helped us to understand the biology of tooth movement with alveolar decortication, and its effect on the teeth and bone. The journey of accelerated tooth movement that started in the 1890s has been continuing with constant and deliberative speed in the development of new techniques. We are now understanding the underlying mechanisms and developing minimally invasive techniques to accelerate the treatment to satisfy both the patients' and the dental professionals' expectations. This achieves the best possible treatment, building on the clinical art of the past, to achieve outcomes that dentistry can be proud of and patients can safely count on, in a timely manner. That is a laudable goal for any doctor. We must collectively achieve it as a rational, scientific enterprise, unfettered by specious and unlettered criticism, progressively yet deliberatively. Unfortunately, dentistry is surfeit with intemperate, ill-conceived criticism of SAD variants. This is to be expected with innovation and is not all bad. The specious claims areas common as the clinical variations of SAD and PAOO. Yet the science, the abiding and prolific epistemological tool of ultimate truth, is undeniable and, like the proverbial “genie out of the bottle,” it tells us that SAD procedures, from crude corticotomy to PSCT, are here to stay.

Alas, so will the controversy, that inevitable cacophony of dialectical noise which gives birth to scientific innovation. Still, we live in a post-industrial, post-modern age that challenges the Enlightenment precepts of reason and scientific pre-eminence. So the clinicians employing enlightened perceptions must be the arbiters of truth as a fiduciary. He or she is charged with the directing of a final act on the stage of this manifest destiny – for better or worse. The ultimate challenge is pernicious social forces which, manifest as inviolable artistic interpretations, can deconstruct the edifice of orthodontic science claiming it as irrelevant. This accounts for the disregard for scientific foundations that interfere with commercial exploitation of the specialty. Yet the scientific truths explicated by selective alveolar decortication and the nascent science of oral tissue engineering are difficult to exploit commercially. So the specialty can lie secure in the bosom of enlightened science and the absolute mathematical imperatives of the logical positivist. Whether one perspective – artistic, commercial, or scientific – will prevail over the others is an existential choice that each individual doctor and patient must make. Preferably, their choices will be concordant.

Carpe Diem!

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