International Journal of Dentistry Research 2019; 4(1): 30-37

International Journal of Dentistry Research

Review Article

ISSN: 2581-3218
IJDR 2019; 4(1): 30-37
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Procedures to accelerate orthodontic treatment: Review of techniques and biological bases

Nelson José Rossi[1], Rosa Carrieri Rossi[1], Nelson José Carrieri Rossi[1]

***1** R Tijuco Preto 1694, Brasil; Brazilian Society of Orthodontics and Dentofacial Orthopedics-SBOOM*

Abstract

To study the most common means to accelerate orthodontic treatment it was done a search in Pubmed, LILACs and Bireme databases with the uniterms: corrective orthodontics, inflammatory mediators, tooth movement, lymphokines and a manual search at Scholar Google with the terms: corticotomy, periodontal accelerated osteogenic orthodontics, piezocison, piezopuncture and alveolar microperforations. All the techniques are based on the variation of chemical mediators that stimulate colonies of ostoclasts and osteoblasts that take place in the bone metabolism. It was shown that surgical maneuvers are the most common procedures, following by phyiscal estimuli and there is great diversity between the techniques and evaluation methods so we have to do a narrative review based on chronologic appearance in literature. It was concluded that of the procedures to accelerate dental movement in orthodontic treatment, corticotomy, alveolar microperforations and accelerated periodontal osteogenic orthodontics are those that appear to be more indicated in clinical applications when considered the benefits over conventional treatment, although research with larger groups and controlled conditions is necessary to fully validate these techniques.

Keywords: Corrective orthodontics, Mediators of inflammation, Tooth movement, Lymphokines.

INTRODUCTION

Orthodontic movement is a complex process involving metabolic changes in the gingiva, periodontal ligament, alveolar bone and cementum. Due to the diversity in the histophysiology of these tissues, it is not yet possible to fully clarify the interaction between the physical and biological phenomena involved.

Recently there was an increase in the search for treatments of shorter duration, which led the researchers to study the hypothesis of accelerating the mechanisms of bone resorption and apposition and the variation of chemical mediators capable of stimulating the formation of colonies of osteoclasts and osteoblasts. This paper aims to describe the current state of knowledge and the clinical evidence available at the time.

MATERIAL AND METHODS

In the literature the terms: Corrective orthodontics, mediators of inflammation, tooth movement, lymphokines were searched in Pubmed, LILACs and Bireme databases. In addition, the terms corticotomy, periodontal accelerated osteogenic orthodontics, piezocison, piezopuncture and alveolar microperforations were searched in Scholar Google to access articles with the names of the most common procedures to accelerate orthodontic treatment.

it was found that due to the great disparity between the techniques presented in the studies and the diversity of evaluation methods in the researches, we could not do a systematic review on the subject, so we made a narrative review detailing the main procedures, their conceptual bases and results obtained so far.

Literature review

The alveolar bone is constantly renewed by functional demands, especially for being covered by periosteum and endosteum, which are osteogenic vascular membranes present throughout life that allow a system of growth by apposition. It has two dense cortical plates separated by a trabecular bone[1].

The periosteum has a cellular and fibrous layer where the muscles or fibers of the periodontal ligament are inserted, while the endosteum is formed by a single layer of cells because it is not subjected to traction.

***Corresponding author:**
Dr. Nelson José Rossi
R Tijuco Preto 1694, Brasil; Brazilian Society of Orthodontics and Dentofacial Orthopedics-SBOOM
Email:
nelsonjrossi@gmail.com

During the formation of new bone the osteoblasts produce collagen fibers and extracellular matrix that undergoes calcification forming osteoid, which later becomes bone tissue[2]. This osteoid can not be attacked by osteoclasts, a condition that will be important in the course of this discussion.

During the formative phase, osteoblasts are recruited and become osteocytes, blood vessels are also involved and form the nutritional channels of the bone formed. New collagen fibers are synthesized and form the adhesion system of the periosteum with the periodontal ligament. This initially formed fibrous bone, when it reaches a certain thickness, is organized according to the functional demand in lamellar bone[1].

The periodontal ligament is a highly cellular and vascular connective tissue formed along with cementum from the follicle surrounding the dental germ and having collagen fibers that attach to both the periosteum of the alveolar bone and the cementum.

Mechanical and biochemical factors may cause the mechanisms of attraction and activation of osteoblasts and osteoclasts as well as the rhythm of remodeling of supporting structures to be altered and influence orthodontic movement[3]. A brief explanation about them will be done before entering the acceleration of the dental movement.

a) Bone response to loading: Frost[4] proposed the mechanostatic theory, in which the normal bone undergoes a deformation according to the load exerted on it, expressed in microstrains (µs, deformation per unit of length X 10 -6). The application of an orthodontic force would be transmitted to the membrane of the cells involved in the remodeling (mecanoinduction) and would activate the mechanism of proliferation and synthesis of peptides that will be described next. Also the hypoxia produced in the pressure areas of the periodontal ligament could modify the metabolism of mesenchymal cells, initiating the inflammatory process[5].

b) Dental movement involves the participation of cells present in the inflammatory process, such as T cells, macrophages and fibroblasts, as well as osteoblasts and osteoclasts. All of them are able to synthesize proteins such as those that will form collagen fibrils and the intercellular substance[5].

In addition, prostaglandins present in the inflammatory process of the periodontal ligament induce an increase in the number and capacity of cells that reabsorb the bone[5,6]. Low molecular weight proteins (cytokines) measure the intensity of remodeling depending on the strength and biological response of the individual[7].

After resorption, the new bone is remodeled by the differentiation of osteoblasts that are stimulated by osteoprotegerin that is synthesized by the osteoclasts themselves, as if they signal the need to finish the resorptive part and begin remodeling[8,9].

Although new substances have been discovered in this process and the functions are not fully understood, the following table 1 shows a simplified description of the function of each in the orthodontic movement.

Table 1: Mechanism of action of thr molecules involved in orthodontic movement

Molecule involved in the process	Mechanism of action	Function in orthodontic movement
Prostaglandins 1 and 2 (PGE_1) e (PGE_2)	Activation of osteoclasts in the initial phase of the process	Reabsorption of the alveolar process in the pressure areas[10,11]
Interleukynes 1, 6 e 8 (IL-1), (IL-6), (IL-8)	Leukocyte attraction, osteoblasts and osteoclasts	Leukocytes attracted produce peptides that modulate the process and the other cells reabsorb and form bone Kindle, Sari (IL-6), (IL-6) [12,13]
Vascular Endotelial Growth Factor (VEGF)	Stimulus to angiogenesis Recruitment of mononuclear precursors of osteoclasts in the bone marrow Differentiation of osteoclast precursor cells	Maintenance of osteoclasts Increased expression in cells subjected to hypoxia Local injection of VEGF stimulates osteoclastogenesis and increases the speed of dental movement[14]
Tumor-alpha necrosis factor (TNFα-)	Stimulating macrophage colony	Accelerates bone resorption[15,]
Receptor activator of nuclear factor-K (RANK)	Osteoclast differentiation factor, is of the family of TNFs	It stimulates the osteoclastic action when activated by RANKL[16,17]
RANK Binding Factor (RANKL)	Differentiation of pre osteoclasts and mature osteoclasts	Stimulates osteoclastic action acting as the membrane ligand for RANK[18,19]
Osteoprotegerin (OPG)	modulates the action of RANKL Inhibiting the binding of RANK and RANKL	Decreases osteoclast action[20]
Bone morphogenetic proteins (BMP-2), (BMP-4), (BMP-6)	From the family of growth factors Beta	Accelerate bone regeneration in fractures[21,22]

In summary, the molecular bases of orthodontic movement can be partially explained by the following:

a) The application of orthodontic forces causes a one-hour release of IL-1β, TNF-α and prostaglandins mediators peaking within 24 hours and causing initial orthodontic movement.

b) Significant decrease in mediators favorable to bone formation as OPG occurs immediately after the application of orthodontic force and is a key factor for initial movement.

c) The level of cytokines decreases after reaching the peak of 24 hours, but increases gradually each time the force is applied again by the activation of the apparatus. During most of the process, RANK / RANKL / OPG levels determine the recruitment and intensity of action of osteoblasts and osteoclasts.

d-The difference in the level of cytokines in remodeling and the speed of movement is higher in young than in adults, signaling that remodeling is faster in younger individuals.

Based on the metabolic principles of tooth movement, several ways of accelerating orthodontic movement were proposed. Kole[23] was the first to describe a technique in which the alveolar cortical was surgically weakened, suggesting that less resistance would lead to faster movement.

Currently, this concept has been reviewed in light of the knowledge of molecular biology and new techniques have been developed. It will not be discussed those that only serve for extraction cases, such as periodontal distraction.

1) Corticotomy: It is a surgical process in which the vestibular and palatine gums are detached below the periosteum and the exposed alveolar cortical bone is segmented between the teeth, down to the root apexes. Next the flap is closed and after two to three weeks of the surgical act the orthodontic forces are applied[24,25]. Figure 1

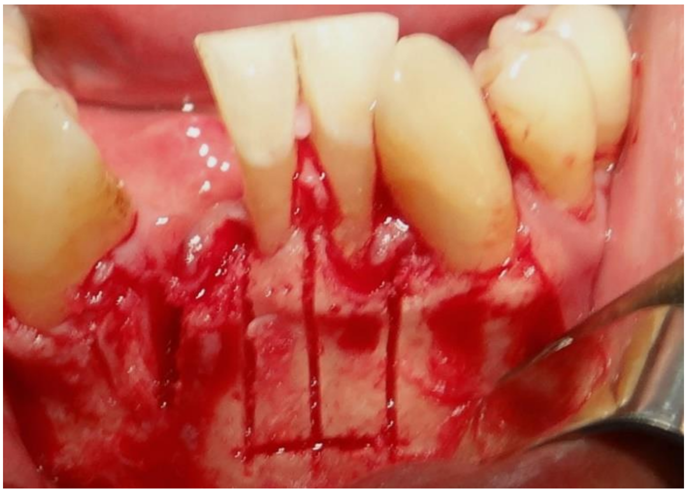

Figure 1: Grooves in the vestibular cortical bone in corticotomy

A process called Frost's "regional accelerating phenomenon" (RAP) causes an increase in bone metabolism and accelerates its remodeling. Recently, a study has revised the subject under the light of the practical application of corticotomy and the biological mechanisms involved[26].

The initial response occurs with the immediate constriction of blood vessels to reduce bleeding, followed by hematoma and blood clot within a few hours. Within seven days of the injury, an aggregate of cells from adjacent tissues is then formed, consisting of fibroblasts, intercellular substance and other support cells, interspersed with small blood vessels[27].

Many of the fibroblasts present in the site were differentiated from inactive pericytes, cells that surround the small vessels in the areas surrounding the injury. These pericytes are activated by VEGF and in addition to initiating angiogenesis in the affected area, they become osteoblasts, helping to form the matrix for the primary callus[28].

The formation of this granulation tissue takes about 2 weeks. A few days later, periosteum cells near the lesion become chondroblasts form hyaline cartilage. Periosteum cells distal to the lesion site induced by cytokines become osteoblasts and begin to form bone.

These processes result in a mass of hyaline cartilage and bone tissue, called "bone callus", which is subsequently replaced by lamellar bone. The duration of time between bone callus formation and mineralization is 1 to 4 months, depending on the extent of the lesion[29].

The accelerating phenomenon would be related to the decrease of the bone volume, the decrease of the mineral amount of the bone and the greater release of biological mediators such as RANKL and others involved in the regulation of osteoblasts and osteoclast function. There is less time in the hyalinization phase of the periodontal ligament, in this way the latent phase of remodeling of the same decreases in the same proportion[30].

Two recent systematic review on the clinical effects of corticotomy[31] showed that the procedure can actually shorten orthodontic treatment time with few deleterious effects, although the number of studies and the lack of prospective studies do not provide robust scientific evidence for its broad application.

Another important variable in the practice of corticotomy is the ideal depth of the sulcus in relation to the alveolar bone[32]. Histologically and by means of microtomographies, the results of furrows of different depths were evaluated in dogs. They concluded that the higher the insult to bone, the greater the amount of immature bone and the lower its density. These phenomena were present the longest distance in the deepest cuts.

The decortification associated with the cuts does not seem to influence the metabolic effect on the alveolar bone. In animals subject only to cuts and subject to cuts and decortification, they found only statistically significant differences in the amount of movement and in the radiographic characteristics.

The accelerating phenomenon peaks from one to four months and can last from six to twenty four months, thus being very variable. The efficiency of corticotomy should be considered in treatments whose duration can exceed by several times this term.

2) Periodontal accelerated osteogenic orthodontics: The possibility of taking advantage of the accelerating phenomenon to accelerate dental movement and the presence of important mediators in the increase of bone remodeling to aid in the incorporation of osteoinductive material led to the appearance of the so-called periodontal accelerated osteogenic orthodontics (PAOO).

Figure 2: lyophilized bone added to the exposed area to penetrate into the grooves and holes

Figure 3: Final result after sevem month orthodontic treatment

It is a variant of the corticotomy in which, in addition to the vertical cuts, perforations of 3,00 mm can be made or until there is exposure of the medullary bone in the vestibular cortical of the alveoli of the involved teeth[33]. Some type of osteoinductive material, such as lyophilized or autogenous bone, is added to the exposed area and penetrates into the grooves and holes[34]. After a period of two to three weeks, the orthodontic movement is initiated, taking advantage of the favorable remodeling phenomena to incorporate the graft. Figures 2 and 3

The probable mechanism involved in the agility of graft repair and incorporation is related to the application of orthodontic force during the bone repair process after corticotomy[35].

Accoding to Perren[36], depending on the stress on the bone in recomposition, the process of intramembranous ossification, endochondral, the formation of a granulation tissue or even the absence of union can occur. This sequence of possibilities would be directly related to force.

If te fracture is relatively consolidated, the mechanism is intramembranous. If there is a low intensity tension, a cartilaginous matrix and consequent endochondral ossification will be formed, with the temporary callus that would have the function of stabilizing the stumps.

Chondrocytes are able to survive in a higher loading environment than the osteoblasts until the fracture stabilizes and is re-colonized by the latter. If the traction is even greater, primary repair will occur by fibroblasts, which survive another level of force, and the fracture may or may not consolidate depending on the continuity of the force.

In the best scenario, the fibrous callus is replaced by chondrocytes and subsequently colonized by osteocytes. In case of excessive force, only one fibrous callus would be created incapable of consolidating the fracture.

Thus, tooth movement in the repair area of the corticotomy would be responsible for generating forces maintaining a mechanical environment that promotes fracture ossification and consolidation.

There is a smaller reduction of the callus and the presence of more neoformed bone in the cases in which the graft was used as a framework.[37,38]. Regarding the material used in the graft, there is a difference in the requirements for tooth movement and in those used in periodontics and implantology[39,40,41,,].

In addition to stability and biodegration, the graft used in tooth movement must have two attributes: Do not prevent the displacement of the roots and not be rigid to the point of causing damage to the cement of the teeth moved towards them.

The autogenous graft, especially that of bone blocks removed from the symphysis, is considered the best when it comes to implant insertion in the recipient region, but the large portions of dead bone remaining in the repair area may constitute a barrier to movement of the roots.

To the present, the best combination appears to be the deproteinized bovine bone mixture (Bio-Oss; Geiistlich Sons, Wolhusen, Switzerland) and demineralized bone matrix (Orthoblast II; IsoTis, Irvine, USA) mixed with Ahn saline. Although demineralized bovine bone is not superior to other means when used alone Turhani, associated with a material with morphogenetic proteins presents the best result for the orthodontic surgical modality.

It was mentioned at the beginning of this text[2] that osteoclasts can not be attacked by osteoclasts, and since repair under load seems to induce an increase in formation phenomena at the expense of resorption, the applied graft would tend to be incorporated by the newly formed bone.

The latency period of two to three weeks is necessary for initial consolidation and the application of the low intensity and long duration load induced by the orthodontic appliance acts as a accelerating phenomenon will be present.

In an in vitro study[42] with two co-cultures, one of periodontal ligament cells and human osteoblast-like cells (MG63) and another of myofibroblasts and MG 63 cells, both subjected to traction, has been shown to increase the synthesis of collagen and the production of osteoclacin in myofibroblasts submitted to loading. Since myofibrobes are present in the periodontal ligament, this would be another favorable phenomenon for the remodeling of the alveolar bone under the action of orthodontic movement.

Similarly, the combination of cultures of endothelial progenitor cells and mesenchymal stem cells mixed with a β-tricalcium phosphate framework was used to promote the vertical increase of bone in rats by Zigdon-Giladi[43], leading to the hypothesis that cell therapy trunk may be a promising pathway for the study of the incorporation of osteoprogenic materials in conjunction with the orthodontic movement.

Apparently this modality of orthodontic-surgical treatment can fulfill both proposals, that is, the speed of movement is increased and some material osteoid is added to the original bone, being indicated in cases where the concern with the precariousness of alveolar bone may influence the treatment[44].

3) Corticocison: Corticocision is a procedure in which a reinforced scalpel is used to make not only a vertical incision in the interdental region, but also penetrates the cortical bone. In this way, the regional acceleratory phenomenon would be induced in a manner similar to corticotomy in a less invasive way, as was shown in a study done in cats[45].

The surgical technique consists of infiltrative anesthesia followed by incision with a blade number 15, which has the necessary stiffness to allow penetration into the vestibular cortical. The inclination of 900 should be maintained relative to the alveolus and the extent of the incision ranges from below the interdental papilla to 1mm above the mucogingival junction of each dentition[46].

As the cervical incision is extended the blade is forced into the medullary bone and must be withdrawn without pivoting to prevent injury to the lips. The original technique recommends the incision also by palatine or lingual, in this case, even greater care should be taken regarding the tongue.

4) Piezocision: Piezocision is a variation of the corticotomy or orthodontic periodontal accelerated, except for the absence of detachment of the gingival flap to access the cortical bone and the way to section it[47].

While it is possible to perform alveolectomy with surgical drills in the above variants, piezoelectric requires the use of an ultrasonic instrument. Ultrasonic osteotomy is based on the principle that the application of energy at its tips generates an electric potential in the mineral structure of the bone, which, alternately, causes expansion and contraction of the bone, resulting in its separation and consequent fission.

For this reason, the piezoelectric scalpel can not separate soft tissues such as mucosa and nerve tissue, hence its wide application in oral surgery, periodontics and implantodontics.

The procedure is done under local anesthesia. After the vertical incision which runs from 3.00 mm below the interdental papillae to above the groove bottom in the proximal teeth involved, is carried out the separation of edges and insertion of an ultrasonic blade which allows the depth corticotomy 3, 00 mm, reaching the medullary bone[47].

In case of opting for corticotomy alone, the suture may be optional after a period of observation in which the bleeding is assured. One variant of this procedure is the tunneling by detachment of the mucosa between two vertical incisions, allowing the access of another ultrasonic tip to promote perforations or other grooves in the cortical.

In this case it is possible the insertion of osteoinductive material, as in accelerated periodontal osteogenesis orthodontics, and the edges of the cutaneous incisions must be approached by means of suture.

A recent systematic review[31] has shown that, although there is no retrospective or large sample studies, there appears to be increased in orthodontic movement speed in patients undergoing piezocison.

4) Piezopuncture: The piezopuncture is a variant in which, instead of performing the alveolar cortical sections are made only 3.0 mm hole with a rounded tip of the ultrasonic instrument. The tip should be kept perpendicular to the gum for 5 seconds under irrigation with saline solution.

The punctures should be preceded by perforations to allow the access of the ultrasonic tip directly to the alveolar cortical, to the mesial and distal portions of the involved teeth.

Comparing two groups of dogs subject to piezopuncture and control, it was found[48] that the mesial movement of a tooth was 3.26 times faster in the maxilla and 2.45 times in the jaw. In addition, histological observation by fluorescence showed that the affixing of new bone after six weeks was 2.55 times greater in the animals submitted to piezopuncture than in the control groups.

There are no more studies on this modality of treatment, probably because alveolar microperforations that do not require the concurrence of the ultrasonic instrument began to show similar effects with more simplicity.

5) Alveolar Microperforations: Recently the process called "microperforations" began to be adopted, based on the principle that the simple perforation of the vestibular alveolar bone through the mucosa can present the same benefits of previous ones with lower surgical risk.

Studies in animals[9] at demonstrated that the release of inflammatory markers increases significantly with the microperforations and the time of the dental movement can be halved.

To date, only one instrument developed in the United States of America has been developed for micro-perforations (Propel) and is a device that features a stainless steel drill bit with a diameter of 1,5 mm and a length that can be varied between zero and 7, 0 mm.

According to the manufacturer, different depths should be used to allow perforation of the alveolar cortical. At the back of the device you can select the length of the active tip to the desired size.

The active tip should be forced against the moving gingiva clockwise until a light comes on, indicating that the depth has been reached. Apparently the intensity of bone aggression is directly proportional to the release of cytokines in corticotomies[32], which leads to the assumption that the proximity of the perforation would need to be variable depending on the thickness of alveolar bone that surrounds each tooth.

Contrastingly, in a study done in humans[49] only the length between 2.0 and 3.0 mm was systematically used with positive results, which included an increase in velocity in orthodontic movement two to three times in relation to control and significant release of cytokines in the crevicular groove of the teeth near the perforations.

This study did not demonstrate deleterious effects in the application of the procedure, despite the small number of participants.

Apparently, although alveolar perforations may not reach depth to the point of causing metabolic changes in the bone closer to the periodontal ligament, Swapp et al. Al[50] showed by means of microtomographies and the analysis of the speed of movement in dogs that appear microfractures in the alveolar cortices that can be responsible for the weakening of the boards and consequent increase in the velocity of the vestibular-lingual movement, without affecting the horizontal movements of the roots.

8) Discision: A novel method to accelerate tooth movement has been introduced to replace corticotomies[51]. After local anesthesia, incisions with a Depth of 3,0mm are performed at the interrradicular regions by a disc saw. A clinical trial comparing discision, piezocision and a control group has shown that both surgical maneuvers are comparable in terms of shortening treatment time[52]. Figure 4.

Figure 4: Variation of corticotomy made with a surgical disc (discision)

7) Some drug-induced adjuvants and physical stimuli were introduced to improve the effects of surgery[53]. introduced platelet-rich plasma submucosal injection (PRP), a technique developed to accelerate orthodontic tooth movement, simulating the effects of bone insult without surgery and loss of alveolar bone that accelerated mandibular or maxillary alignment 1, 7 times faster on average[52].

Physical stimuli to accelerate orthodontic movement have been introduced recently, but Xiao et al.[54] in a published meta-analysis showed that evidence regarding the efficacy of photobiomodulation, pulsed electromagnetic field, interseptal bone reduction, two vibrational devices and electric current were of very low quality. Relaxin injections and extracorporeal shock waves were reported as having no impact on the velocity of the orthodontic movement according to evidence of low and very low quality, respectively.

It was shown that there is no advantage neither in the time nor in reducing pain from using vibrational devices during orthodontic treatment[55] as well as in the retraction of canines and the level of RANKL and OPG[56].

DISCUSSION

Apparently, all surgical processes that have the purpose of accelerating the speed of tooth movement present some degree of scientific evidence. The application, however, needs to be well planned so that the benefits of this acceleration are actually greater than the risks, since conventional orthodontic treatment is enshrined.

The main criteria of evaluation would be the morbidity of the procedure, the costs, the ease in the clinical management and its effectiveness. Based on this principle, alveolar micro-perforations could be considered easy to apply and little chance of error, since the system is very similar to that used in the installation of temporary anchoring devices, before insertion of the miniscrews.

On the other hand, the lack of clinical studies that can prove the efficiency in any type of orthodontic treatment does not allow it to be considered as essential in clinical practice.

In the same way, the corticocision, piezocision and piezopuncture showed in the experimental studies that can produce the liberation of biological mediators present in the regional phenomenon with little invasiveness. Especially the last two, when processed with ultrasonic tips, do not predispose to the risk of perforating the soft tissues and attacking the periodontal ligament.

However, the costs of the procedure increase significantly and the low number of clinical trials can not prove that the benefits of these treatment alternatives outweigh the conservative treatment in a definitive way.

Corticotomy is the more studied surgical procedure to reduce the time of orthodontic treatment. Although there is controversy regarding its efficiency, mainly due to the disparity between the duration of the regional accelerating phenomenon and the time elapsed in the orthodontic treatment, there is strong evidence that the bone metabolism is influenced by the technique.

Taking into account that the initial stages of treatment (alignment and canine retraction) demand the greatest number of directions of root movement and consume more time, something that would allow a transient acceleration would be convenient.

Even so, it is the procedure with greater morbidity, since it requires an important surgical phase with detachment of the gingiva and extensive penetrations in the alveolar cortical. It is necessary to take into account that the patient seeking orthodontic treatment and the healthy periodontal structures of support may prefer a treatment that is more time consuming than to run the risk of infection, postoperative pain and discomforts of the surgery.

Discision can be an easier alternative to corticotomy and do not requires special equipement to be developed, but there is just one clinical trial with few patientes to validate ths procedure.

Accelerated periodontal osteogenic orthodontics equals corticotomy in terms of risks and discomforts. However, because of the possibility of facilitating the incorporation of graft into the alveolar bone, it may be an alternative in cases of patients with little thickness in the vestibular cortex where the teeth have to be protruded or labioverted.

It also presents a good number of clinical cases in which it was applied partially to facilitate the movement of a tooth or a group of teeth and in patients with sequelae of bone loss due to periodontal disease.

The incorporation of the graft associated to the increase of the calcified matrix in the alveolar process of the area submitted to the procedure may even be considered advantageous in the case of future loss of the teeth involved, providing a greater calcified bed for the insertion of prosthetic implants.

Thus, in cases of doubtful prognosis, the application of this mechanism could be indicated as a maneuver to give greater initial stability in the rehabilitation with implants if the survival of the moved teeth is not significant.

Observing the similarity of some processes and the efficiency of the techniques, it is possible to suggest that at least two of them could be of more frequent use in the clinic. The orthodontic accelerated periodontal osteogenesis seems a good alternative in some cases of difficult conduction in which the shortage of alveolar bone is limiting, since besides the reduction in the time of the treatment would have the benefit of the incorporation of calcified matrix as periodontal support.

More research is need on new materials that would meet the requirements of graft incorporation under conditions of root movement, such as optimized biodegradation, osteoconductive property and the use of nanoparticles to avoid deleterious effects on tooth cement in the path required.

Application of tissue engineering in bone regeneration is the future of accelerated periodontal osteogenic orthodontics, since the production of cells with specific capacity for remodeling of the dental support tissues would be the ideal situation to replace the methods currently in use.

The alveolar microperforations, despite the little evidence demonstrated, are not compicated due to the ease of application, the low morbidity and surgical risk, when compared to the other means to promote the release of biological mediators favorable to the bone metabolism.

So far the evidence does not seem to clarify the true reason why alveolar perforations accelerate orthodontic movement, whether this procedure serves for movements in all directions that orthodontic treatment requires, the ideal depth of the holes and the need for repetition of them the duration of the accelerating phenomenon.

Since patients, especially adults, demonstrate an interest in shorter treatments, the adoption of microperforations can become an efficient means to shorten the time suggesting a major advance in the specialty. It would also be useful to study other means of performing the perforations beyond the exclusive technique of a manufacturer, which is what we have at the moment.

Apparently the study of alveolar perforations offers a field to be studied due the great interest to the specialty. The physical and drug maneuvers to assist the surgical procedures still lack further studies to validate them.

The use of surgical maneuvers to accelerate orthodontic treatment seems to be in the initial phase of the learning curve and more research with larger groups and under more controlled conditions is necessary to be able to state that the conventional treatment should be systematically modified.

CONCLUSION

Of the means to accelerate dental movement in orthodontic treatment, corticotomy, alveolar microperforations and accelerated periodontal osteogenic orthodontics are those that appear to be more indicated in clinical applications when considered the benefits over conventional treatment.

Research with larger groups and controlled conditions is necessary so that adunctive procedures in orthodontic treatment can be widely diffused in the daily clinic, although there is a signaling for its application in selected cases.

Conflicto f Interest

None.

Source of Support

Nil.

REFERENCES

1. Thilander B. Reações teciduais em ortodontia. In: Ortodontia, princípios e técnicas atuais/ [editores Lee W. Graber, Robert L. Vanarsdall Jr., Katherine W.L. Vig] ; [tradução Adriana do Socorro Lima Figueiredo...et al.]. –Rio de Janeiro: Elsevier, 2012. p. 247-286.
2. Enlow, Donald H. Noções básicas sobre crescimento facial- Segunda Edição/ Donald H. Enlow, Mark G. Hans; [tradução Terezinha Oppido, revisão científica Silvia Fuerte Bakor].-São Paulo: Santos, 2012 532p.
3. Rossi NJ, Rossi RC, Rossi NJC. Ortodontia e Ortopedia Maxilar - Crescimento, Ortodontia Interceptativa e ortopedia maxilar – vol1 São Paulo: Editora All Print, 2008.308p.
4. Frost HM. Skeletal structural adaptations to mechanical usage (SATMU).Redefining Wolff's law: the remodeling problem. Ant Rec 1990;226:414.
5. Chacko SM, Ahmed S, Selvendiran K, Kuppusamy ML, Khan M,Kuppusamy P. Hypoxic preconditioning induces the expression of prosurvival and proangiogenic markers in mesenchymal stem cells. Am J Physiol Cell Physiol 2010;299:C1562-70.
6. Krishnan V, Davidovitch Z . Cellular,molecular and tissue-level reactions to orthodontic force. Am J Orthod Dentofacial Orthop, 2006, 129:469.e1-469.e32.
7. Krishnan V, Davidovitch Z . On a path to unfolding the biological mechanisms of orthodontic tooth movement. J Dent Res.2009, 88:597-608.
8. Garlet TP, Coelho U, Silva JS, Garlet GP .Cytokine expression pattern in compression and tension sides of the periodontal ligament during orthodontic tooth movement in humans.2007,Eur J Oral Sci 115:355-362.
9. Teixeira CC, Khoo E, Tran J, Chartres I, Liu Y, Thant LM, Khabensky I, Gart LP,Cisneros G, Alikhani M. Cytokine expression and accelerated tooth movement. J Dent Res. 2010; 89(10):1135–41.
10. Kanzaki H, Chiba M, Shimizu Y, Mitani H. Periodontal ligament cells under mechanical stress induce osteoclastogenesis by receptor activator of nuclear factor kappaB ligand up-regulation via prostaglandin E2 synthesis. J Bone Miner Res. 2002; 17:210–20.
11. Celebia AA, Demirerb S, Catalbasc B, Arikand S. Effect of ovarian activity on orthodontic tooth movement and gingival crevicular fluid levels of interleukin-1β and prostaglandin E2 in cats. Angle Orthod. 2013; 83:70–5.
12. Hamamcı N, Acun Kaya F, Uysal E, Yokuş B. Identification of interleukin 2,6, and 8 levels around miniscrews during orthodontic tooth movement.Eur J Orthod. 2012; 34(3):357–61.
13. Sari E, Uçar C. Interleukin 1beta levels around microscrew implants during orthodontic tooth movement. Angle Orthod. 2007; 77(6):1073–78.
14. Holmes, Katherine; Roberts, Owain Ll; Thomas, Angharad M.; Cross, Michael J. "Vascular endothelial growth factor receptor-2: Structure, function, intracellular signalling and therapeutic inhibition". Cellular Signalling, 2007 19 (10): 2003–12.
15. Kindle L, Rothe L, Kriss M. Human microvascular endothelial cell activationby IL-1 and TNF-alpha stimulates the adhesion and transendothelial migration of circulating human CD14 monocytes that develop with RANKL into functional osteoclasts. J Bone Miner Res. 2006; 21:193–206.
16. Lowney JJ, Norton LA, Shafer DM, Rossomando EF. Orthodontic forces increase tumor necrosis factor alpha in the human gingival sulcus. Am J Orthod Dentofacial Orthop. 1995; 108(5):519–24.
17. Theoleyre S, Wittrant Y, Tat SK, Fortun Y, Redini F, Heymann D. The molecular triad OPG/RANK/RANKL: involvement in the orchestration of pathophysiological bone remodeling. Cytokine Growth Factor Rev. 2004;15(6):457–75.
18. Nakao K, Goto T, Gunjigake KK, Konoo T, Kobayashi S, Yamaguchi K. Intermittent force induces high RANKL expression in human periodontal ligament cells. J Dent Res. 2007; 86:623–28.
19. Nakao K, Goto T, Gunjigake KK, Konoo T, Kobayashi S, Yamaguchi K. Intermittent force induces high RANKL expression in human periodontal ligament cells. J Dent Res. 2007; 86:623–28.
20. Oshiro T, Shiotani A, Shibasaki Y, Sasaki T. Osteoclast induction in periodontal tissue during experimental movement of incisors in osteoprotegerin-deficient mice. Anat Rec. 2002;
21. Huang H, Ray C. Williams RC, Kyrkanides S. Accelerated orthodontic tooth movement:Molecular mechanismsAm J Orthod Dentofacial Orthop 2014;146:620-32
22. Kapoor P, Kharbanda OP, Monga N, Miglani R, Kapila S.Effect of orthodontic forces on cytokine and receptor levels in gingival crevicular fluid: a systematic review Progress in Orthodontics 2014, 15:65.
23. Köle H. Surgical operations of the alveolar ridge to correct occlusal abnormalities. Oral Surg Oral Med Oral Pathol 1959;12:515-529.
24. Long H, Pyakurel U,Wang Y, Liao L, Zhou Y, Lai W. Interventions for accelerating orthodontic tooth movement A systematic review. Angle Orthod Vol 83, No 1, 2013.
25. Ferguson DJ, Wilcko WM, Wilcko MT. Selective alveolar decorticationfor rapid surgical-orthodontic resolution of skeletal malocclusion treatment. In: Bell WE, Guerrero C (eds): Distraction osteogenesis of the facial skeleton. Hamilton, BC, Decker, 2006:199-203.
26. Frost, H.M. The regional acceleratory phenomenon: a review. Henry Ford Hosp Med J. 1983; 31: 3–9
27. Buschang P,Campbell PM, Ruso S.Accelerating Tooth Movement With Corticotomies: Is It Possible and Desirable? Semin Orthod 2012;18:286-294.
28. Chang, H.N., Garetto, L.P., Katona, T.R., Potter, R.H., Lee, C.H., and Roberts, W.E. Angiogenesis and osteogenesis in an orthopedically expanded suture. Am J Orthod Dentofacial Orthop. 1997; 111: 382–390
29. Baloul S, Gerstenfeld LC, Morgan EF, Carvalho RS, Van Dyke TE, Kantarci A. Mechanism of action and morphologic changes in the alveolar bone in response to selective alveolar decortication–facilitated tooth movement. Am J Orthod Dentofac Orthop. 2011, 139(4)S, p. S83–S101.
30. Iino S, Sakoda S, Ito G, Nishimori T, Ikeda T, Miyawaki S. Acceleration of orthodontic tooth movement by alveolar corticotomy in the dog. A J Orthod Dentofac Orthop. 2007, 131(4), p.448.e1–448.e8.
31. Hoogeveen EJ, Jansma J, Ren Y.Surgically facilitated orthodontic treatment: A systematic review. Am J Orthod Dentofac Orthop,2014, 145, (4), S51–S64
32. McBride MD, Campbell PM, Opperman LA, DehcouwPC, Buschang PH. How does the amount of surgical insult affect bone around moving teeth? Am J Orthod Dentofacial Orthop. 2014; 145S: S92–9
33. Wilcko, M.T., Wilcko, W.M., Pulver, J.J., Bissada, N.F., and Bouquot, J.E. Accelerated osteogenic orthodontics technique: a 1-stage surgically facilitated rapid orthodontic technique with alveolar augmentation. J Oral Maxillofac Surg. 2009; 67: 2149–2159
34. Murphy, K.G., Wilcko, M.T., Wilcko, W.M., and Ferguson, D.J. Periodontal accelerated osteogenic orthodontics: a description of the surgical technique. J Oral Maxillofac Surg. 2009; 67: 2160–2166
35. Prabhu MN, Sabarinathan J. Periodontically accelerated osteogenic orthodontics - a review. American Journal of Biomedical Research,2013 1 (4), pp 132-133.
36. Perren SM. Physical and biological aspects of the fracture healing with special reference to internal fixation. Cil Orthop 1979;138:175-96.
37. Mellonig, J.T. Human histologic evaluation of a bovine-derived bone xenograft in the treatment of periodontal osseous defects. Int J Periodontics Restorative Dent. 2000; 20: 19–29
38. Carmagnola, D., Berglundh, T., and Lindhe, J. The effect of a fibrin glue on the integration of Bio-Oss with bone tissue. An experimental study in labrador dogs. J Clin Periodontol. 2002; 29: 377–383.
39. Chiu GSC,Chang CHN,Roberts WE. Bimaxillary protrusion with an atrophic alveolar defect: Orthodontics, autogenous chin-block graft, soft tissue augmentation, and an implant. 2015;147:97-113.
40. Ahn YH, Ohe JY, Lee SH, Park YG, KIM SJ.Timing of force application affects the rate of tooth movement into surgical alveolar defects with grafts in beagles. 2014; 145: 486-95.
41. Turhani, D., Weissenbock, M., Watzinger, E., Yerit, K., Cvikl, B., Ewers, R. et al. In vitro study of adherent mandibular osteoblast-like cells on carrier materials. Int J Oral Maxillofac Surg. 2005; 34: 543–550.
42. Xu H, Han X, Meng Y, Gao L, Guo Y, Jing Y, Bai D. Favorable effect of myofibroblasts on collagen synthesis and osteocalcin production in the periodontal ligament. 2014, Am J Ortohod Dentofac Orthop; 145:469-79.
43. Zigdon-Giladi H, Bick T, Lewinson D, Machtei E E. Co-transplantation of endothelial progenitor cells and mesenchymal stem cells promote neovascularization and bone regeneration. Clin Implant Dentistry Rel Research. 2015, 17 (2): 353-9.

44. Rossi NJ, Rossi RC, Rossi NJC, Rossi Jr R, Yamashita HK, Voudouris JC. Ortodontia osteogênica periodontal acelerada associada aos sistemas autoligáveis – relato de caso. Orthod. Sci. Pract. 2014; 7(28):
45. Kim, S.J., Park, Y.G., and Kang, S.G. Effects of corticision on paradental remodeling in orthodontic tooth movement. Angle Orthod. 2009; 79: 284–291
46. Mimura H. Protraction of mandibular second and third molars assisted by partial corticison and miniscrew anchorage. Am J Orthod Dentofac Orthop, 2013; 144:278-89.
47. Dibart, S., Sebaoun, J.D., and Surmenian, J. Piezocision: a minimally invasive, periodontally accelerated orthodontic tooth movement procedure. Compend Contin Educ Dent. 2009; 30: 342–344 (346, 348-50)
48. Kim YS, Kim SJ,Yoon HJ, Lee PJ, Moon W, Park YG. Effect of piezopuncture on tooth movement and bone remodeling in dogs.Am J Orthod. Dentofac Orthop. 2013; 144:23-31.
49. Alikhani M, Raptis M, Zoldan B, Sangsuwon C, Lee YB, Alyami B, Corpodian C, Barrera LM, Alansari S, Khoo E, Teixeira C. Effect of micro-osteoperforations on the rate of tooth movement. Am J Orthod Dentofacial Orthop. 2013; 144(5):639–48.
50. Swapp A, Campbell PM, Robert Spears R, Buschang PH. Flapless cortical bone damage has no effect on medullary bone mesial to teeth being moved. Am J Orthod Dentofacial Orthop 2015;147:547-58.
51. Buyuk SK ,Yavuz MC, Genc E, Sunar O. A new method to accelerate orthodontic tooth movement.Saudi Med J.2018;39(2):203-208.
52. Yavuz MC, Sunar O, Buyuki SK, Kantarci A.Comparison of piezocision and discision methods in orthodontic treatment.Progress in Orthod. 2018;19-44.
53. Liou EJ. The development of submucosal injection of platelet rich plasma for accelerating orthodontic tooth movement and preserving pressure side alveolar bone. APOS Trends Orthod 2016;6:5-11
54. Xiao YJ, Li HY X. Li, Zhao Z.Effectiveness of adjunctive interventions for accelerating orthodontic tooth movement: a systematic review of systematic reviews. https://doi.org/10.1111/joor.12509
55. Effects of vibrational devices on orthodontic tooth movement: A systematic review. Aljabaa A, Almoammar K, Aldrees A, Huang G.Am J Orthod Dentofacial Orthop 2018;154:768-79.
56. Siriphan N, Leethanaku, C,Thongudomporn U. Effects of two frequencies of vibration on the maxillary canine distalization rate and RANKL and OPG secretion: a randomized controlled trial. doi: 10.1111/ocr.12301.