Accelerated Tooth Movement- A review

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Abstract

Nowadays, there is an increased tendency for researches that focus on accelerating methods for tooth movement. This is due to the huge demand for adults for a shorter orthodontic treatment time. Unfortunately, long orthodontic treatment time poses several disadvantages like higher predisposition to caries, gingival recession, and root resorption. This increases the demand to find the best method to increase tooth movement with the least possible disadvantages.

Keywords: *accelerated tooth movement, corticotomy, piezoelectric currents, Cytokine Expression.*

Introduction

Time has been recognized as the 4th dimension in orthodontics. It has immense importance not only in orthodontic treatment planning but also has become one of the major contributing factors in patient compliance.Hence an orthodontist should not be responsible to provide esthetics but also be able to complete his treatment plan within a time limit.

This has given rise to the concept of accelerated tooth movement. The ideology behind accelerated tooth movement was to increase tooth movement thus reducing the overall treatment time.

Tooth movement is caused due to the application of a mechanical force that leads to remodeling changes in theparadental tissues. This movement occurs due to the cellular responses in the PDL and alveolar bone.[1,2] Studies at cellular, molecular, and tissue-levelon the biologic mechanism of toothmovement suggest that a mechanical force is just one of the stimulus for inducing tooth movement.[3] The use of pharmaceutical, electromagnetic, laser and surgicalstimuli in combination with the mechanical force foraccelerating orthodontic tooth movement has attractedthe interestof many orthodontists.

Many such techniques are described below as methods for increased tooth movement.

Corticotomy assisted tooth movement

Extensiveinjury to the cortical plate boneis known as corticotomies. This procedure is currently being used to accelerate orthodontic tooth movement in private practice.

The corticotomy accelerates tooth movement by inducing a response in the alveolar bone by demineralizingthe bone around the dental roots. Once the bone has demineralized, there isa three- to four-month window of opportunity where the teeth can be moved rapidly throughthe demineralized bone matrix before theremineralisation of the alveolar bone commences. Frost in 1989[4,5] first described thisbone response is called “regional acceleratory phenomenon” or RAP. It was initially coined to describe rare cases of fracture healing.

In this method, tooth movement involves surgical incisionsthrough the alveolar bone. To determine whetherteeth move by distraction osteogenesis or

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byregional accelerated phenomenon (RAP). An osteotomy frees a bony segment to be distracted with tooth-bornedistractors or aligned with orthodontic wires and springs.

Wilcko et al in 2001[6] stated that selective buccal and lingual decortication ofalveolar bone has been used to accelerate orthodontic tooth movement.Bogoch et al. in 1993[7]; Schilling et al.in 1998[8] postulated that osteoclastand osteoblast cell populations shift in number, resulting in an osteopeniceffect. Iino et al. in 2007[9]observed that corticotomy-assistedtooth movement is associated with a lack of hyalinization and early tartrateresistant alkaline phosphatase staining.

Lee et al in 2008 [10] hypothesized that a distraction site under orthodontic tension can be created which will be similar to the long bone by both corticotomies andosteotomies in alveolar bone.The osteotomies producedchanges resembling a distal distraction site, while thecorticotomies produced a regional loss of bone supportingthe dental roots, typical of regional accelerated phenomenon.

The cause for differences in bone response tocorticotomy vs. osteotomy probably lie in the enhancedmobility of the osteotomized segment and the fracture-like healingvs. the post-corticotomy healing produced by the openings intothe marrow vascular spaces. These openings expose the surgicalsite to an enhanced healing potential along with maintaining theinvolved segment in a stable state.

The use of RAP in dentistrycan result from local trauma. Several studies suggested that use of as corticotomy used inperiodontal surgery by Nyman et al., 1978, [11]orthodontic forceby Verna et al., 1999,[12] implant in Roberts, 1988,[13] and infection andsystemic change in bone due to menopause and PTH treatmentby Frost, 1989.[4,5] In the rat model, a local RAP response wasassociated with increased systemic inflammation markers (Schilling et al., 19989.[8] The RAP response was divided into aninitial phase of maximally stimulated bone formation, wherewoven or fibrous bone was produced to span a cortical gap.

This bone eventually remodeles into lamellar bone. The initialphase is proceeded by period of predominant resorption. The medullary bone disappeares and there is a decrease in the osteoblastnumber in the medullary bone. Resorptive activity,as measured by a 22% decrease in ^{45}Ca retention. This occurrsduring the first week and peaked by day 21.

Ilizarov in 1988[14]described osteogenesisas an orthopedic technique forlengthening limbs Currently, there are many craniofacialapplications, including some that use interdental incisions to createdistraction sites as mentioned by Liou et al., 2000; Kisniscu et al., 2002; Iseri et al., 2005;Yen et al., 2005 andSukurica et al., 2007.[15-19] Ilizarov,[14] developed the tensionstressprinciples of distraction osteogenesis and believed that a corticotomy wasthe ideal method for creating a distraction site, because thebone marrow is intact during distraction procedures. While important forlimb lengthening, it is unclear whether the alveolar bone responds to corticalincisions in the same manner as a long bone.

Considering that it is impossible to control theforce magnitude clinically not to produce hyalinization,in the view of a biologic approach rather than a mechanicalcontrol, the lag phase of tooth movement canbe shortened by stimulating the removal of hyalinizedtissue. This can be attributed to the RAP because itstimulates cell-mediated responses around the tooth,thereby providing a favorable microenvironment for tissueremodeling. Various researchers have focused oncontrolling the microenvironment of the alveolar boneby using the RAP in an attempt to reduce tissue resistance.[6] The transient osteoporotic condition involvedincreased release of calcium, decreased bonedensity, and increased bone turnover, all of whichwould facilitate tooth movement.[20] This mechanismbased on the RAP differed from the classical conceptsof tooth movement such as the pressure-tension theory, [21] bone-bending theory,[22] mechanostat theory,[23] and bony block movement in corticotomy.[24]

It is important that the cortical bone itself is not abarrier or resistance to orthodontic tooth movement.Garg[25] emphasized that the RAP is primarily a phenomenonobserved in the cortical bone. This new conceptof cortical remodeling enabled the continuous advancementof supplemental surgical procedures involvingminimal and conservative interventions. Germecet al [26] revealed that single-sided partialcorticotomy in the mandible appeared to be sufficientto stimulate rapid tooth movement.

Electric currents, bone remodeling, and orthodontic tooth movement:

The activation of bone cells in mechanically stressed bone is carried out by piezoelectric currents.This "piezoelectric" phenomenon was observed in both in vitro and in vivo.[27]

Exogenous electric currents have been employed and have resulted in successful attempts to initiate osteogenesis in intact bones[28,29]and to enhance bone apposition in healing of uncomplicated [30,31], or nonunion[32], fractures.[33]

In spite of evidences, little is known about the mechanism whichproduces the tissue changes seen after the application of electricity. Lavine et al in 1974[34] reported that cells in bone fracture sites treated by constant D.C. currents presentedan increased number of membrane-bound vesicles, morphologic transition of rough endoplasmicreticulum to ribosome-lined cisternae, and vacuolization of the mitochondria.

Rodan, Bourret, and Norton in 1978 [35] reported that the application of oscillating electric fields to check chondrocytes in vitro resulted in enhancement of the incorporation of ^{3}H-thymidineinto DNA. They suggested that Na^{+} and Ca^{2+} fluxes were responsible for this effect.

Brighton and Friedenberg in 1974 [36] observed alterations in oxygen consumption and tissue pHnear the cathode.

Davidovitch. Z., Korostoff, E.Shanfeld, J., Montgomery, P., and Finkelson[37-39] investigated the role of cyclic nucleotides in the mechanism of cell activation byexternal electric currents in vivo was investigated earlier. Cyclic nucleotideswere selected as the main target of that investigation because of their implication asintracellular 'second messengers' in the action of specific bone cell activators, such asparathyroid hormone and calcitonin,[40-42], on their target cells. By using immune-histochemicaltechniques, we discovered that external electric currents increased boneand PDL cyclic nucleotide contents.[43] a step leading toward heightened enzymatic phosphorylationreactions, synthetic and secretory activities, and an enhanced rate of tissueremodeling. Earlierthe involvement of adenosine 3'.5'-monophosphate(cyclic AMP. CAMP) in the periodontal tissue response to orthodontic treatment andconcluded that mechanical forces might not be the most efficient means to activate PDLand alveolar bone cells.[44,45] That conclusion, coupled with the recent observation that electriccurrent can activate a large number of cells in a small, well-delineated area,[43] led tohypothesize that the application of electric currents to periodontal tissues during orthodontictreatment will potentiate the effect of the mechanical forces and lead to anenhanced rate of cell activation, tissue remodeling, and tooth movement.

Cytokine Expression and Accelerated Tooth Movement:

An aseptic inflammatory response is induced by orthodontic forces.Krishnan and Davidovitch, 2006;[46] Meikle, 2006[47] studied the early stages of tooth movement, there is an increase in vascular permeability and cellular infiltration of leukocytes. Ren and Vissink, 2008;[48] Krishnan and Davidovitch, 2009[49] suggested that migrated immune cells along with native cells such as fibroblasts and osteoblasts produce inflammatory cytokines that include lymphocyte and monocyte-derived factors, colony-stimulating factors, growth factors, and chemotactic factors.

Alhashimi et al., 2000; Garlet et al., 2007; Ren et al., 2007).[50-52] studied the high concentrations of inflammatory cytokines such as interleukin-1 (IL-1), IL-2, IL-3, IL-6, IL-8, tumor necrosis factor-á (TNFá), interferon-ã (IFNã), andosteoclast differentiation factor that were foundin the gingival crevicular fluid surrounding moving teethand found the osteoclast differentiation factor in the gingival crevicular fluid surrounding moving teeth.

The role of cytokines during tooth movement is not clear. Saito et al., in199[53] suggested that cytokines and other inflammatory markers, such as prostaglandin E2, Davidovitch et al.,[54] 1988; Garlet et al., 2007[51] suggested that this may activate bone remodeling characterized by bone resorption in the compression region and bone deposition in the tension region of the periodontal ligament. Studies by Frost, 1983,[55] 1989;[4,5] Shih and Norrdin, 1985;[56]Yaffe et al., 1994[57] positively agreed that demonstrated that bone injury which causes cytokine release leads to an accelerated bone turnover and a decrease in regional bone density. This may by one possible mechanism through which inflammatory cytokines may affect bone remodeling is through recruitment of osteoclast precursors from the circulation, their maturation and activation.

Glantschnig et al., 2003; Seidenberg and An, 2004; Yao et al., 2008, Uematsu et al., 1996; Basaran et al., 2006[58-

[62] suggested that the cytokines that promote osteoclast formation and activation, such as IL-1, IL-6, and TNFá, have also been found in crevicular fluid during orthodontic tooth movement.

The effect of cytokine expression on bone remodeling is important, since the rate of tooth movement correlates with the efficiency of bone remodeling in the alveolar process. Studies by Yoshimatsu et al., 2006[63] of knockout mice deficient for TNFá receptors showed a slower rate of tooth movement in response to orthodontic forces. Previous reports by Arias and Marquez-Orozco, 2006[64] have shown that anti-inflammatory medication can decrease the rate of tooth movement.

It is important to remember that inflammation is known as 'a two-sided sword', and while it can work as a benefit by accelerating bone remodeling and tooth movement, if uncontrolled, it may also have a destructive effect on the periodontium and tooth structure. Root resorption in response to osteoperforation is undergoing investigation.

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