# HISTOLOGY OF THE ORTHODONTIC MOVEMENT

**MARIA PITTNER**[1*], RICARDO CESAR GOBBI DE **OLIVEIRA**[2]

**1.** Undergraduate student in Dentistry by UNINGÁ; **2.** PhD in Orthodontics, Faculty of Dentistry of Bauru - USP, Professor of Dentistry, UNINGÁ.

* Quintino Bocaiúva Street, 1154, Ap. 34, Zone 07, Maringa, Parana, Brazil. ZIP CODE: 87020-160, mariapittner@hotmail.com

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## ABSTRACT

**The orthodontic movement occurs by reorganization of periodontal tissue in response to application of a mechanical force, and various biochemical substances are synthesized and released for initiation and maintenance. Bone cells recognize the application of the mechanical load generating coordinated bone remodeling in bone resorption which is adjacent to areas of compression of the periodontal ligament and bone formation adjacent to areas voltage of the periodontal ligament, with subsequent adaptive changes ligament. Therefore, cells and tissues using sensitivity and transduction in response to application of mechanical force, with subsequent movement of the tooth.**

**KEYWORDS:** Bone remodeling, fibroblasts, osteoclasts, neoformation.

## 1. INTRODUCTION

To better understand orthodontic movement, we need to understand the tissue phenomena that occur at each stage of tooth movement. Thus minimizing the risks of root resorption, optimizing the treatment and comfort of the patient.

Two processes are fundamental for orthodontic tooth movement to occur, and one of them is reabsorbed in the areas of pressure, while in the areas of tension a new bone is formed. This is possible because bone is a dynamic tissue that allows a constant remodeling, thus, a process that requires interactions between different cell types, regulated by biochemical factors or mechanical factors[1].

Pressure-tension theory explains the process of bone remodeling, where this process is slower on the pressure side, than on the tension side of the fibers belonging to the periodontal ligament. On the pressure side, it occurs to the compression of the tissues, periodontal, generating a process of degeneration, evolving to necrosis with aseptic characteristic, exhibiting hyaline appearance[2]. The collagen fibers constituting the periodontal ligament suffer a disaggregation, accompanied by hemodynamic changes, increased vascularization and, consequently, migration of inflammatory cells to the site, mainly neutrophils. Concomitantly, on the tension side, where the fibers of the periodontal ligament are stretched, the number of fibroblasts and osteoblasts increases, resulting in the production of collagen and bone matrix, promoting bone neoformation of the wall adjacent to the tractioned tooth[3,4,5].

It was observed that the repair of the tissue by the orthodontic movement occurs after the reduction of the force applied[6], requiring the use of intermittent forces, which requires gradual reduction and constant reactivation is required. However, with the development of super-elastic nickel-titanium (NiTi) yarns, it was possible to apply continuous forces during the period of orthodontic movement, analyzing the possible responses of the periodontal tissue of sustentation against mechanical stimuli.

In this context of the histology of the orthodontic movement, it is necessary to understand what precedes this, that is, how these tissues are formed and how they behave, as we activate them. The fact that there is no resorption in cement occurs due to the absence of receptors on the cementoblasts membrane for mediators of bone resorption, since the mechanism of orthodontic movement can be explained by molecular actions[7].

The inflammation is responsible for the orthodontic movement, and the orthodontic force is the cause of the trauma, which is characterized biologically by the formation of free protein in the body. Proteins are bound together, and when a protein becomes free, it becomes unstable, and undergoes degradation by enzymes. The proteins that are free generate a series of events fundamental for orthodontic movement, which will be discussed later[2].

Thus, as the orthodontic movement occurs through the restructuring of the periodontal tissues in response to the application of a mechanical force, and several chemical substances are synthesized and released for their initiation and maintenance, we will address these cellular and molecular reactions that allow the activation of the orthodontic movement[1,2].

## 2. MATERIAL AND METHODS

To carry out this study we following steps: 1) identification of the research question, followed by a search of the descriptors or keywords; 2) determining the

criteria for inclusion or exclusion of research in online databases; 3) categorization of studies, summarizing and organizing relevant information; 4) assessment of studies for critical analysis of the extracted data; 5) discussion and interpretation of the examination results, contextualizing theoretical knowledge and evaluating their applicability as; 6) presentation of the integrative review and synthesis of knowledge of each article reviewed briefly and systematic way.

In the present study the guiding question of the integrative review was: such as histological modification due to orthodontic movement

Bases (Latin American and Caribbean Literature on Health Sciences) LILACS, SciELO (Scientific Electronic Library on Line) and PubMed (NCBI US National Library of Medicine National Center for Biotechnology Information) were consulted. Studies that have addressed the thematic, published from 1972 to 2016, regardless of the languages of publication were included. The following controlled for the search and also used as keywords descriptors were used: Bone remodeling, fibroblasts, osteoclasts, neoformation.

## 3. LITERATURA REVIEW

Since inflammation is required for orthodontic movement, Eltman *et al*. (2001)[8] have schematized the phases following inflammation, which are caused by the mechanical forces involved. Continuous to the trauma, we obtain free proteins, free phospholipids, which undergo the enzymatic action of phospholipase, which transforms into arachidonic acid, the molecules of phospholipids. This substrate will undergo two enzymatic pathways: one is the enzyme cyclooxygenase, generating the formation of prostaglandins and thromboxanes; And the other is the enzyme lipoxygenase, resulting in the formation of hydroxyethoxytetraenoic (Hete) and leukotrienes[9].

The substances generated from arachidonic acid are called eicosanoids. Among these, as already mentioned we have prostaglandins (PG), and prostaglandin E (PGE), which play an important role in orthodontic movement. PGE is responsible for vasodilation, increased cellular permeability, regulation and synthesis of osteoblasts and fibroblasts, and alteration of cellular immune functions. PGE, basically speaking, alters the metabolism of fibroblasts and osteoblasts, which secrete collagenase and also PGE, altering its morphology[2].

The PGE synthesized by these cells attracts the osteoclasts to the site of the trauma. The collagen matrix may be degraded by collagenase enzymes, because these matrices are not mineralized, this being possible because they form channels between the osteoblasts[10,11]. Degradations of the periosteum expose the area of hydroxyapatite, which results in the chemotaxis of osteoclasts. Then, migration of the osteoblasts to the periphery of the bone occurs, and osteoclasts move to the hydroxyapatite region (PAH)[10].

Concomitantly, PGE performs vasodilation, allowing the migration of macrophages, and other cells. Macrophages release interleukins, which is a type of cytokine, essential in the inflammatory process. In addition, cytokines induce the production of PGE and collagenase by osteoblasts and fibroblasts, as well as can contribute to cell division and minimize bone formation in vitro[12].

Osteoblasts and macrophages together are called the hard tissue resorption unit (10). Metabolism and also migration of osteoclasts can be controlled by both osteoblasts. In the process of bone remodeling, due to the fact that odontoblasts and cementoblasts do not have cellular receptors for PGE, this chemical mediator is not involved in this mechanism.

The undifferentiated and perivascular cells of the mesenchyme may be attracted by the reabsorption process, due to this, releasing growth factors (peptides). These mesenchymal cells differentiate into osteoblasts and begin to synthesize and secrete constituents of the bone tissue, such as matrix components, proteoglycans, type I collagen, osteonectin, osteocalcin and osteopontin, chemical mediators that control: and synthesis as the Bone resorption; The presence of other constituents of the region; And actions of a systemic character, that is, that go beyond the region that is being manipulatedAmong the systemic factors, the main ones are growth hormones, thyroid hormones, parathyroid hormone, glucocorticoids, calcitonin, and vitamin D3. Among the most important local factors, such as prostaglandins E2 (PGE2), skeletal growth factor (SGF), fibroblast growth (FGF), platelet-derived growth (PDGF), epidermal growth (EGF) Beta transformer (TGF), gamainterferon (INF) and interleukin (IL-1)[2,5,10].

### Tissue reaction when in orthodontic movement

Faced with a mechanical stimulus obtained through orthodontic movement, tissue reactions occur in the periodontal tissues, mainly in the periodontium of sustentation. Studies have established that, generally, tooth movement occurs through two processes: by resorption in pressure areas and by formation in tension areas. The bone allows us this movement due to the dynamics of its fabric, which allows a constant remodeling. This change in bone structure is only achieved through biochemical reactions, allowing cellular interactions, and mechanical factors, thus allowing a tissue response. Several histological studies evaluating the movement through the application of intermittent forces, where the intensity of force gradually declines, requiring recurrent activations in biomechanics, proceeded the classic concepts on tissue reactions against orthodontic movement[13,14].

Several studies have documented that in areas

where there was pressure there was an increase in the number of osteoclasts and consequently bone resorption. In the periodontal ligament, changes occur in vascularization, collagen disruption and hyalinization[13,15].

The hyalinization of an aseptic necrotic air, due to the fact that it does not have cells in this region of the periodontal ligament, a limitation for orthodontic movement occurs. Thus, the process of hyalinization should be eliminated for continuity of the movement process, with the formation of new periodontal structures[13]. If this necrotic process is not deconstitution, permanent lesions such as root resorption can be generated[16]. Much research indicates that hyalinization of the periodontal ligament during the initial phase of orthodontic treatment is almost inevitable.

This repair process occurs when pressure is reduced and cells migrate to the site. In addition, new capillary vessels are formed, being an important factor in this repair process. However, root resorption may still occur where these areas of hyalinization are formed[9].

On the tissue changes in the areas of tension, there is an increase in the spaces between the structures of the periodontal ligament. The cells change shape, becoming longer, with their elongated axis, toward the traction. The osteoblasts that migrate and distribute in the alveolar surface region, characterize the first response of these cells to the orthodontic stimulus[21]. Then, there is an increase in the number of fibers and vascular changes, such as vasodilation. Bone deposition occurs over the entire surface of the alveolus, progressing with increased movement time[14].

### Root resorption

The lesion leading to root shortening or root resorption may be correlated with the force model applied, with the magnitude and type of this force, with the genetics, the type of malocclusion, some systemic condition, with sex, Age, with several factors that may result in root resorption[22,23].

In ideal conditions, orthodontic movement should involve alveolar bone and ligament. However, root resorption is an undesirable effect that occurs in some cases. It is believed that this resorption occurs adjacent to the areas of hyalinization, in the stage of removal. The elimination of this hyalinized area by macrophages and giant cells causes removal of the cementoid layer, leaving the root surface exposed[18,19] With the late occurrence of root resorption, at which time the "clasts" that resorb root structure (cementum and dentin) appear, and this reabsorption continues until all areas of hyalinization are removed[9,20].

The cement has the capacity of neoformation, but it can not be compared with the regenerative capacity of the bone. This allows the cement to regrow new layers at the affected site after resorption. However, when the dentin is reabsorbed, the cement in most cases will cover it with a thin layer, but without recovering its initial root shape. Because of this, root resorptions are considered irreversible when they reach the dentin. The periodontal tissue change in relation to the orthodontic movement depends on several factors, especially the mechanical factors that are divided into: force magnitude, force direction, type of force, period of incidence of the quantum and also the magnitude of the force of movement[9,20].

## 4. DISCUSSION

As previously shown, prostaglandins play a key role in orthodontic movement. But in addition to prostlaglandines, we have osteopontin (OPN), which is a non-collagenous protein found in large numbers in the matrix of mineralized tissues. Some authors suggest that this protein is produced by osteoblasts, others suggest that it is produced by osteoclasts. Osteopontin has great importance in the mechanism of bone remodeling suggesting that it participates in the phases of reabsorption and neoformation. Studies suggest that OPN synthesis increases in bone remodeling versus mechanical stimulation[24,25,26].

There are signs that OPN has an important role in relation to cement, since its absence leads to a decrease in the number of osteoclasts causing a decrease in root resorption when the mechanical force is applied to the bone tissue. In addition, it was observed in rats that root resorption was observed after approximately 10 days; Even so it did not cause any irreversible damage. However, it is possible that root resorption occurs after the period of application of force[27].

Krishnan & Davidovitch (2006)[28] in their studies report that during the early stages of tooth movement the application of force generates an acute inflammation, in which there is continuous flow of fluids in the periodontal ligament and in the bone tissue, which lead to cell and matrix distortion. As a consequence of the absence of bacterial invasion this reaction is considered as an aseptic inflammatory reaction, which involves a complex cellular response and is reintroduced with each activation of the orthodontic appliance and, because of this, the release of arachidonic acid from the cell surfaces and its metabolism through the lipoxygenase or cyclooxygenase pathway lead to the release of the first messengers (prostaglandins and leukotrienes), which activate or stimulate the release of the second messengers, The cyclic AMP. These second messengers provoke cellular reactions, leading to the remodeling of the bone and the periodontal ligament, where there is also intense angiogenesis and remodeling of the pre-existing blood vessels, through which the release of inflammatory mediators occurs. Therefore, the synthesis and local release of sev-

eral chemical mediators, such as interleukin-1 alpha (IL-1), interleukin-1 beta (IL-1), tumor necrosis factor alpha (TNF), interleukin- 6), and nitric oxide (NO), play an important role in the beginning and maintenance of the bone remodeling process, resulting in the clinical effect of tooth movement[29,30,31,32,33,34,37,38].

In order to verify which cells were involved in the removal of hyalinized tissue and in the reabsorption of root structures, after the application of orthodontic force, Brudvik & Rygh (1994)[6] moved the first right upper molar of 12 Wistar rats and, through the electron microscopy of Scanning, they observed that multinucleated giant cells as well as macrophage-like mononuclear cells were responsible for the elimination of formed hyalinized tissue as well as root resorption. Osteoclasts were only found in association with bone tissue. As a result, degraded fibrous tissue and cell debris appeared to be eliminated primarily by macrophages. Fibroblasts can also act as tissue eliminating cells[34,35,38].

Roberts & Chase (1981) 36 observed the presence of pre-osteoblasts that differentiated into osteoblasts in the periodontal ligament after orthodontic movement in rats. From its results, it could be concluded that osteoblasts could be derived from cells present in the periodontal ligament (mesenchymal cells). The production and mineralization of the matrix of any hard tissue are phasic processes that are characterized by periods of activity interspersed with periods of rest, and are morphologically reflected by perceptible incremental lines[28,29,30,31,37,38].

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## 5. CONCLUSION

The orthodontic movement occurs as a consequence of mechanical factors applied in the bone tissue and as a consequence of these, a biochemical cascade occurs, with actions of several enzymes in several cells and as a result cellular alterations arise and finally changes in the tissue itself, but all these mechanisms can Be affected by issues of applied force, but also due to physiological factors of each individual.

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