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Biology of Tooth Movement

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ABSTRACT

Tooth movement by orthodontic force application is characterized by Remodeling changes in dental and paradental tissues, including dental pulp, periodontal ligament (PDL), alveolar bone, and gingiva. These tissues, when exposed to varying degrees of magnitude, frequency, and duration of mechanical loading, express extensive macroscopic and microscopic changes. Orthodontic tooth movement differs markedly from physiological dental drift or tooth eruption. The former is uniquely characterized by the abrupt creation of compression and tension regions in the PDL.The aims of this study is to shed light on the biological effects of force of the orthodontic treatment and the effect of light and heavy force on the periodontal ligament and the alveolar bone.

Keywords: PDL, Osteogenesis, Chemical mediators, Osteoblasts, Osteoclast

INTRODUCTION

The Periodontal Ligament and Orthodontic tooth movement Each tooth is attached to and separated from the adjacent alveolar bone by a heavy collagenous supporting structure, the periodontal ligament (PDL). Under normal circumstances, the PDL occupies a space approximately 0.5 mm in width around all parts of the root. By far the major component of the ligament is a network of parallel collagenous fibers, inserting into cementum of the root surface on 1 one side and into a relatively dense bony plate, the lamina dura, on the other side.**(Figure 1)[1,2]**

These supporting fibers run at an angle, attaching farther apically on the tooth than on the adjacent alveolar bone. This arrangement, of course, resists the displacement of the tooth expected during normal function. **[3]**

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Although most of the PDL space is taken up with the collagenous fiber bundles that constitute the ligamentous attachment, two other major components of the ligament must be considered. These are the cellular elements, including mesenchymal cells of various types along with vascular and neural elements, and the tissue fluids. **[4,5]**

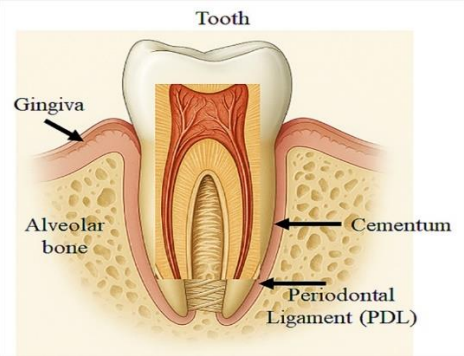

Figure 1: Cross-section of a healthy tooth

The image shows the key components of the periodontium:Gingiva, alveolar bone, cementum, and periodontal ligament (PDL), which anchors the tooth root to the bone and absorbs mechanical forces.

Both play an important role in normal function and in making orthodontic tooth movement possible. The principal cellular elements in the PDL are undifferentiated mesenchymal cells and their 1 progeny in the form of fibroblasts and osteoblasts. The collagen of the ligament is constantly being remodeled and renewed during normal function. The same cells can serve as both fibroblasts, producing new collagenous matrix materials, and fibroclasts, destroying previously produced collagen. Remodeling and recontouring of the bony socket and the cementum of the root is also constantly being carried out, though on a smaller scale, as a response to normal function. Fibroblasts in the PDL have properties similar to osteoblasts, and new alveolar bone probably is formed by oteoblaststhat differentiated from the local cellular population. **[1-9]**Bone and cementum are removed by specialized osteoclasts and cementoclasts, respectively. These multinucleated giant cells are quite different from the osteoblasts and cementoblasts that produce bone and cementum. PDL contain some blood vessels and cells from the vascular system. Nerve endings are also found within the ligament, both the unmyelinated free endings associated with perception of pain and the more complex receptors associated with pressure and Charge positional information (proprioception). PDL space is filled with fluid; this fluid is the same as that found in all other tissues, ultimately derived from the vascular system. A fluid-filled chamber with retentive but porous walls could be a description of a shock absorber, and in normal function, the fluid allows the PDL space to play just this role. Bone bending in response to normal function generates piezoelectric currents that appear to be an important stimulus to skeletal regeneration and

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repair. This is the mechanism by which bony architecture is adapted to functional demands. **[1-12]**

Very little of the fluid within the PDL space is squeezed out during the first second of pressure application. If pressure against a tooth is maintained, however, the fluid is rapidly expressed, and the tooth displaces within the PDL space, compressing the ligament itself against adjacent bone. Pain is normally felt after 3 to 5 seconds of heavy force application, indicating that the fluids are expressed and crushing pressure is applied against the PDL in this amount of time . The resistance provided by tissue fluids allows normal mastication, with its force applications of 1 second or less, to occur without pain. **[1-12]**

Kohno T. et al. in 2002 carried out studies to investigate experimental tooth movement under light orthodontic forces: rates of tooth movement and changes of the periodontium. Forty male Sprague–Dawley (SD) strain rats were used in this study. All animals were fed on powder form fodder and drinking water. In addition to orthodontic forces, occlusal force,and cheek and tongue 4 pressure may act on teeth during experimental tooth movement. In this study, all of the mandibular left molars were extracted at 4 weeks of age in order to eliminate the influence of occlusal forces caused by occlusal contact with the opposing side. The rats were intraperitoneally anaesthetized with ketamine hydrochloridecontaining 20 per cent xylazine hydrochloride as a muscle relaxant, after anaesthetization by inhaling diethyl ether. To assess whole body effects the animals 4 were weighed before each treatment. The tooth movements were started when rats were 5 weeks of age. Forces were applied to the maxillary left first (M1) and second (M2) molars in the mesio-distal 3 direction reciprocally for 14 days. **[13]**

Buccopalatal enamel grooves 3.2 mm apart were cut in the occlusal surface of M1 and M2 with a steel bar under cooling with an air spray. The enamel around the grooves was etched with 65 per cent phosphoric acid for 30 seconds. Both ends of an orthodontic wire were set into the groove and attached 4 with light cure resin. Work-hardened titanium-nickel alloy wires, 0.152 mm in diameter and 12 mm in length were used to exert the orthodontic forces. The wires were bent so as to exert initial forces , and 10 gf when the distance of the both ends was 3.2 mm. The initial force was measured by load cell . **[6-13]**

The load produced as a result of displacement was measured with the load cell by simulating 14 days of tooth displacement. Rats of the same age were used for the sham operations, their mandibular left three molars were extracted, and the grooves in the occlusal surface of the M1 and M2 were cut and 4 filled with the same resin, but without wire fixation.

The results showed :

1. Weight change of animals:

2. Macro-findings of tooth displacement :

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3. Aspects of tooth displacement:

4. Change of the periodontium: The average weights of the rats increased continuously in both the experimental and control groups. No significant difference in weight was found among groups. Three weeks after the extraction of mandibular molars, the inter-proximal area between M1 and M2 of the sham-treated controls, was in contact with no remarkable elongation . After 14 days of tooth movement, mesial inclination of the M1 and distal inclination of the M2 were evident in the buccal view. Changes of distance of tooth displacement in each group. At the first 56 hour measurement, the average tooth displacement among the four groups was 0.2 mm, and there were no significant differences in tooth displacement among groups. From the second 56 hour measurement through day 14, the displacement was increased, but that of the group receiving 1.2 gf was significantly smaller than that of the other groups.Although there was no significant difference, the groups receiving larger initial force tended to show greater tooth displacement than those receiving lower initial force. The orthodontic forces during the experimental period diminished slightly and almost linearly as the tooth displacement increased. On days 7 and 14 in the groups receiving 1.2 and 3.6 gf force, the widths of the PDL were slightly narrow, but no hyalinized tissue was evident..Multinucleated cells, which were considered to be osteo clasts, were seen on the surface of the alveolar bone and direct bone resorption was observed subjacent to these cells. **[7-13]**

A narrow PDL, limited hyalinization and frontal bone resorption were all evident on days 7 and 14 in the 6.5 gf group. At day 7 in the 10 gf group, the root touched the alveolar bone and hyalinization with local undermining bone resorption was observed in the surrounding area. On day 14 in the 10 gf group, however, only a very limited area of hyalinization was seen, and direct bone resorption took place rather than undermining bone resorption . The root resorption seen in these experiments was mainly in the cementum lacunae. The largest lacunae were found in the 10 gf group on day 14, but no severe resorptions, which were observed with forces of higher magnitude, were seen. **[1-13]**

The aims of this study is to shed light on the biological effects of force of the orthodontic treatment and the effect of light and heavy force on the periodontal ligament and the alveolar bone.

Tissue Response in Periodontium:

In 1905, Carl Sandstedt's studies in dogs convincingly demonstrated that tooth movement is a process of resorption and apposition. He gave the first description of the glasslike appearance of the compressed tissue, termed hyalinization, which has been associated with a standstill of the tooth movement. It was not until the 1950s that tooth movements attracted wider

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attention with Kaare Reitan's classic study The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function .**[14-18]**

Bohl M. V. Et. al . in 2004, .**[19]** conducted a study to evaluate histological changes in the periodontal structures of beagle dogs after using high and low continuous forces during experimental tooth movement. An orthodontic appliance was placed on the second premolar and the first molar by exerting a continuous and constant reciprocal force of 25 cN on one side and 300 cN on the other side of the mandible. Tooth movement was recorded weekly. Dogs were sacrificed after one, four, 20, 40, and 80 days for histological evaluation. Hematoxylin and eosin (HE) staining was used for tissue survey, staining for alkaline phosphatise as a marker was used for active osteoblasts, and tartrate resistant acid phosphatase staining was used for osteoclasts. After 24 hours, the remodeling processhad already started at the pressure and tension side, and in some samples hyalinization was found. In contrast to earlier studies, hyalinization was found throughout the entire experimental period, both in molars and in premolars. In the periodontal ligament of some teeth, small patches of hyalinization were found at the pressure side, mostly located buccally or lingually of the mesiodistal plane, whereas others showed large areas of necrotic tissue. It is concluded that hyalinization limits tooth movement, but there is no relationship with the force level. .**[14-21]**

Initial Period of Tooth Movement

Application of a continuous force on the crown of the tooth leads to tooth movement within the alveolus that is marked initially by narrowing of the periodontal membrane, particularly in the marginal area. After a certain period, osteoclasts differentiate along the alveolar bone wall, as occurs in young humans after 30 to 40 hours. All permanent alterations depend on cellular activity. When conditions are favorable, the cells increase in number and differentiate into osteoclasts and fibroblasts.**[14-18]**

The width of the membrane is increased by osteoclastic removal of bone, and the orientation of the fibers in the periodontal membrane changes, as does the arrangement of the ground substance fibroblasts not only are capable of synthesizing fibrous tissue and ground substance but also play an important role in the breakdown of connective tissue. These processes occur simultaneously.

Hyalinization Phase

During the crucial stage of the initial application of force, compression in limited areas of the membrane frequently impedes vascular circulation and cell differentiation, causing degradation of the cells and vascular structures rather than proliferation and differentiation. The tissue reveals a glasslike appearance in light microscopy, which is termed hyalinization. It is caused partly by anatomic and partly by mechanical factors and is almost unavoidable in

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the initial period of tooth movement in clinical orthodontics. Hyalinization represents a sterile necrotic area, characterized by three main 5 stages: degeneration, elimination of destroyed tissue, and establishment of a new tooth attachment.

Degeneration starts where the pressure is highest and the narrowing of the membrane is most pronounced, that is, around bone spicules. Degeneration may be limited to parts of the membrane or extend from the root surface to the alveolar bone. Electron microscopy has shown that advanced cellular and vascular changes may occur within a few hours of the application of the force. Retardation of the blood flow is followed by disintegration of the vessel walls and degradation of blood elements, all occurring by mechanisms different from those seen during physiologic breakdown.

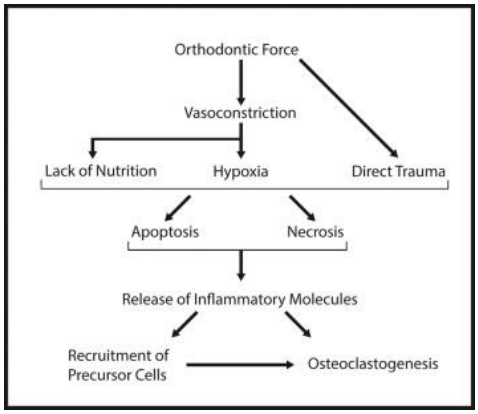

Table 1;Biological Mechanisms to Accelerate Tooth Movement.

The cells undergo a series of changes, starting with a swelling of the mitochondria and the endoplasmic reticulum and continuing with rupture and dissolution of the cytoplastic membrane. This leaves only isolated nuclei between compressed fibrous elements (pyknosis) and is the first indication of hyalinization. In hyalinized zones, the cells cannot differentiate into osteoclasts and no bone resorption can take place from the periodontal membrane. Tooth movement stops until the adjacent alveolar bone has been resorbed, the hyalinised structures are removed, and the area is repopulated by cells. A limited hyalinized area occurring during the application of light forces may be expected to persist from 2 to 4 weeks in a young patient. When bone density is high, the duration is longer. Theperipheral areas of the hyalinized compressed tissue are eliminated by an invasion of cells and blood vessels from the adjacent undamaged PDL. The hyalinised materials are ingested by the phagocytic activity of macro phages and are removed completely. The adjacent alveolar bone is removed by indirect resorption by cells that have differentiated into osteoclastson the surfaces of adjacent marrow spaces or, if the alveolar wall and the outer cortical bone are fused, on the surface of the alveolar process. Reestablishment of the tooth attachment in the hyalinised

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areas starts by synthesis of new tissue elements as soon as the adjacent bone and degenerated membrane tissue have been removed. The ligament space is now wider than before treatment started, and the membranous tissue under repair is rich in cells.

Hyalinized Zone and Root Resorption 3-5

A side effect of the cellular activity during the removal of the necrotichy alinized tissue is that the cementoid layer of the root and the bone are left with raw unprotected surfaces incertain areas that can readily be attacked by resorptive cells.

Root resorption then occurs around this cell-free tissue, starting at the border of the hyalinized zone. Some of these small resorption lacunae are visible only by the scanning microscope. According to Kvam organic tissue tends to remain in the resorbed area, which can be exposed more clearly by removing the organic components. These initial injuries are small and insignificant .Light and transmission electron microscopy have shown that root resorption occurs near the hyalinised zone in close proximity to a rich vascular network**(Figure 2 A-D)**.

This has been verified by Brudvik and Rygh, who showed occurrence of small lacunae in the cementum at the coronal and apical peripheries of the hyalinized zone. Their results indicated an association between root resorption and active removal of the hyalinised necrotic tissue.

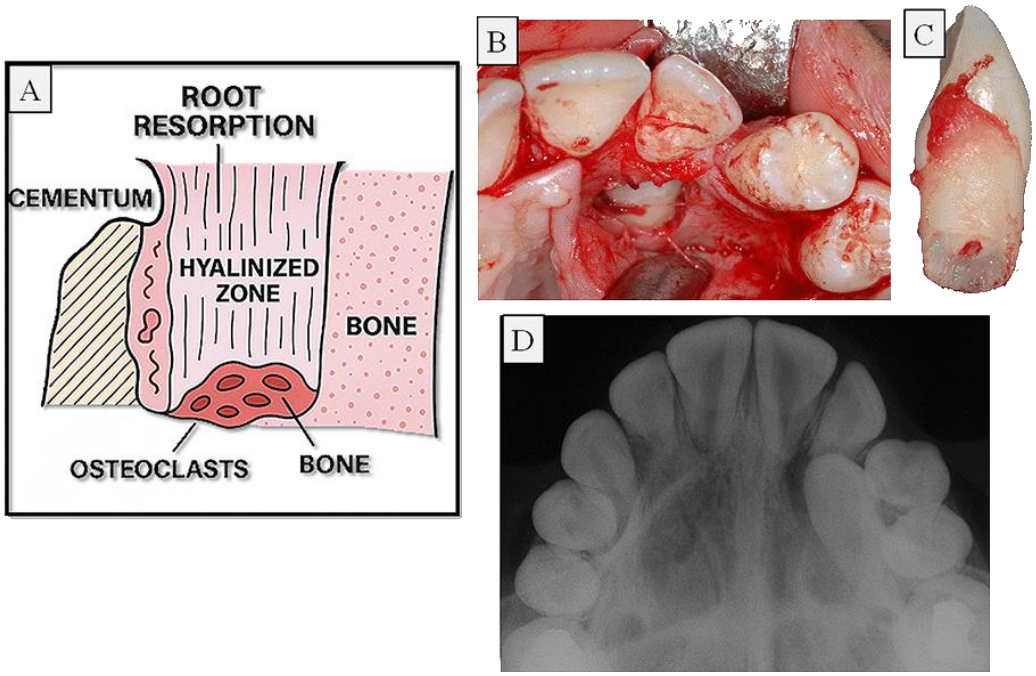

Figure 2 A–D: *Schematic and clinical representation of the hyalinized zone and root resorption.*

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A: Schematic illustration of a cell-free hyalinized area within the periodontal ligament (PDL), resulting from orthodontic pressure. Adjacent bone and root resorption are visible, mediated by osteoclast activity.
B–D: Clinical images of a resorbed lateral incisor demonstrating advanced orthodontically induced root resorption.

The first sign of root resorption(initial phase)

Was defined as a penetration of cells from the periphery of the necrotic tissue where mononucleated fibro blast-like cells, stained negatively by tartrate resistant acid phosphatase (TRAP), started removing the precementum/ cementum surface. Root resorption by TRAP-positive cells in the resorption lacunae still was observed in areas where hyalinised tissue existed. After the hyalinised tissue was eliminated, fibroblast-like cells invaded the active resorption site.

After termination of force and inthe absence of hyalinized necrotic tissue in the PDL,repair on the resorption lacunae occurred. The first signwas synthesis of collagenous fibrillar material by fibroblastand cemento blast-like cells, followed by reestablishment of the new PDL. To fully clarify the factors leading to transition of an active process of resorption into one of repair.

A reduction of root resorption is likely provided byminor trauma and can be repaired during periods of noforce or possibly during periods of extremely low force application. resorption by TRAP-positive cells in the resorption lacunae still was observed in areas where hyalinised tissue existed. After the hyalinised tissue was eliminated, fibroblast-like cells invaded the active resorption site. After termination of force and in the absence of hyalinized necrotic tissue in the PDL, repair on 5 the resorption lacunae occurred. The first sign was synthesis of collagenous fibrillar material by fibroblast and cemento blast like cells, followed by reestablishment of the new PDL.

Further studies are needed, however, to fully clarify the factors leading to transition of an active process of resorption into one of repair. A reduction of root resorption is likely provided by minor trauma and can be repaired during periods of no force or possibly during periods of extremely low force application**(Figure 3)**.

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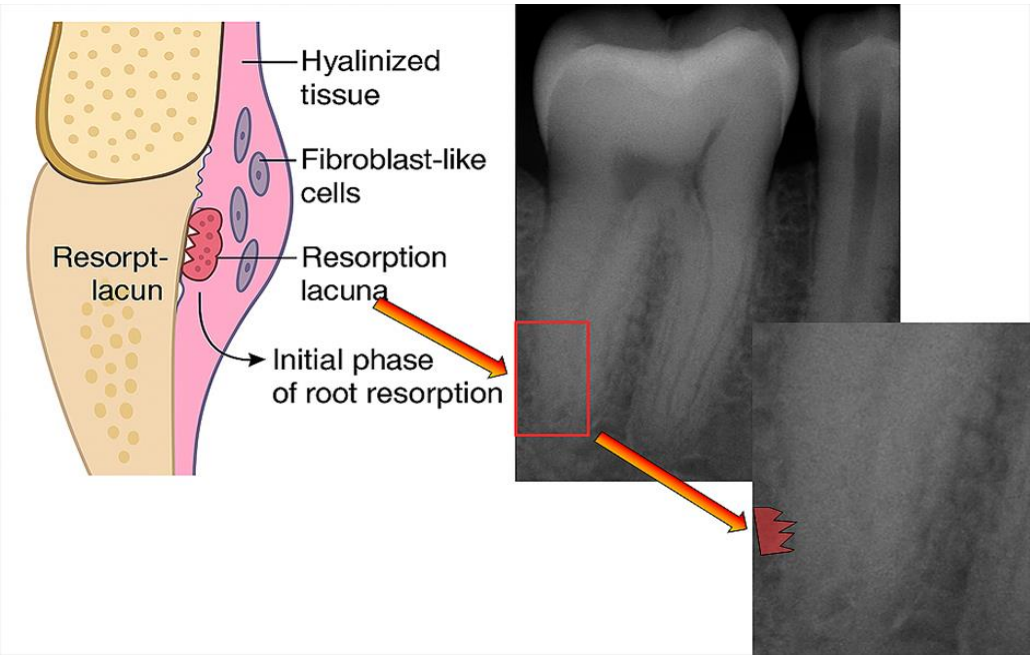

Figure 3: *Initial phase of root resorption – schematic and radiographic view.**Left:** Illustration of a resorption lacuna on the root surface with adjacent hyalinized tissue and fibroblast-like cells involved in the repair phase.**Right:** Radiograph of the tooth roots showing periodontal structures in the early stage of orthodontic treatment.*

Secondary Period of Tooth Movement

In this period, the PDL is considerably widened. The osteoclasts attack the bone surface over a much wider area. As long as the force is kept within certain limits or gentle reactivation of the force is undertaken, further bone resorption is predominantly direct. The fibrous attachment apparatus is reorganized by the production of new periodontal fibrils. When the application of a force is favorable, a large number of osteoclasts appear along the bone surface on the pressure side and tooth movement is rapid. Modern histologic techniques reveal that complete reorganization of the fibrous system takes place throughout the membrane. The main feature is the deposition of new bone on the alveolar surface from which the tooth is moving away(tension side). Cell proliferation usually occurs after 30to 40 hours in young human beings.

The newly formed cells, osteoblasts with darkly stained nuclei, have a characteristic appearance. Osteoblasts may be observed along stretched fiber bundles. Shortly after cell proliferation has started, osteoid tissue is deposited on the tension side. The formation of this new osteoid depends to some 5 extent on the form and thickness of the fiber bundles. The original periodontal fibers become 5 embedded in the new layers of pre-bone, or osteoid, which mineralizes in the deeper parts. New bone is deposited until the width of the membrane has returned to normal limits, and simultaneously the fibrous system is remodeled. The

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original stretched fibers are not broken down to the same extent as is the case on the pressure side, and the remodeling involves resorption and replacement of collagen, leading to a lengthening of the fibers, the mechanism of which is largely unknown. Concomitantly with bone apposition on the periodontal surface on the tension side, an accompanying resorption process occurs on the spongios a surface of the alveolar bone that tends to maintain the dimension of the supporting bone tissue**(Figure 4)**.

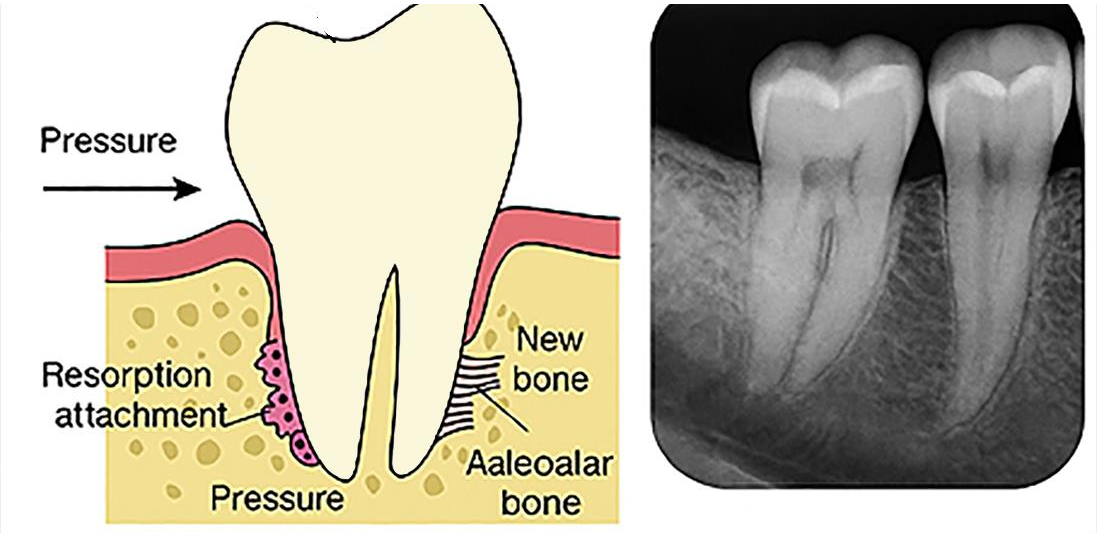

Figure 4: *Schematic and radiographic representation of the secondary phase of tooth movement.* ***Left:*** *Illustration of biomechanical responses in the periodontal space during orthodontic force application. On the pressure side: osteoclastic activity and bone resorption. On the tension side: bone apposition by osteoblasts along stretched fiber bundles.* ***Right:*** *Intraoral radiograph showing tooth roots and alveolar bone in a typical clinical setting for assessing orthodontically induced remodeling processes*

Correspondingly, during the resorption of the alveolar bone on the pressure side, maintenance of the alveolar lamina thickness is ensured by apposition on the spongios a surface. These processes are mediated by the cells of the endosteum, which cover all the internal bone surfaces and dental alveoli. Extensive remodeling takes place in the deeper cell-rich layers of the periosteum, incident to the 3 orthodontic forces, a reaction that tends to restore the thickness of the supporting bone. The observation that orthodontic tooth movement involves many inflammation-like reactions is important in that this, in turn, has enhanced an understanding that the whole cascade of factors involved in inflammation may be part of the reactions to orthodontic forces in the tooth-supporting tissues: that is, extracellular break down of collagen by collage nases, produced by leukocyte– fibroblast interaction. The term inflammation should not be confused with the term infection, as is often the casein popular

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use. In orthodontics, inflammation is a process occurring in a local environment when a rapid response is needed for a stress that is felt transiently by the cells as being too heavy.

Optimal Orthhodontic force :

Orthodontic forces are categorized as “light” or “heavy,” and it was assumed that light forces are gentler and therefore more physiologic than heavy forces. Therefore it can be stated that, to produce adequate biological response in the periodontium, light forces are preferable. Unlike light forces, heavy forces often cause necrosis (hyalinization) of the PDL and undermining bone resorption, and 6 have been implicated in root resorption. According to Schwarz, forces below optimum force produce no reaction, whereas forces above that level lead to tissue necrosis, thus preventing frontal resorption of the alveolar bone.

Reitan K. in 1957**[23]** made attempts to discuss some of the factors involved and, by a few examples, to illustrate the use and significance of carrelating histologic findings with practical observations. A closer investigation of the problem forces in orthodontics reveals that some of the main factors are the individual variation in tissue reaction,the type of force applied, and the mechanical principles involved. It is found that these factors are closely linked together. They can be discussed separately only to a limited extent**(Figure 5)**.

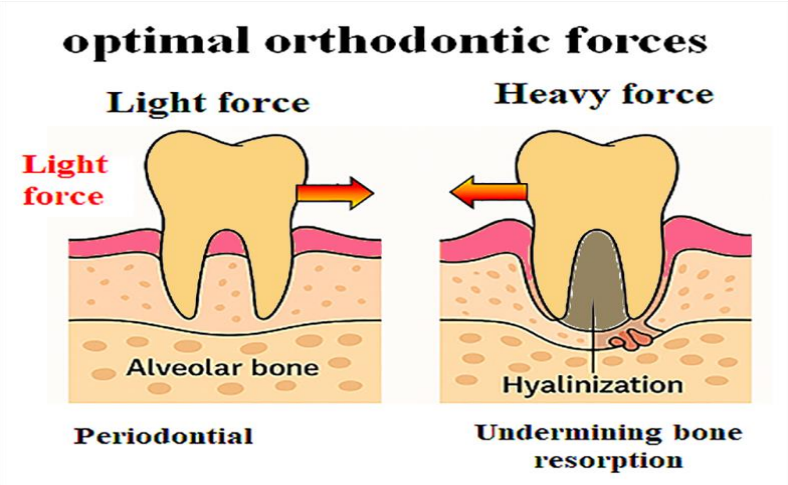

Figure 5: *Schematic representation of the effect of optimal orthodontic forces.*

Left: ***Light force*** *results in uniform periodontal loading with direct bone resorption – physiologically favorable. Right:* ***Heavy force*** *causes hyalinization of the periodontal tissue and indirect bone resorption by osteoclasts – increased risk of root resorption.*

The Individual Variatioin Tissue Reaction

Some variation in tissue characteristics is found among persons of a similar age group. It has been shown, furthermore, that considerable variation exists in the normal tissue characteristics between young and adult structures.

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This is seen clearly in control material taken from the jaws of untreated subjects. In 12 year old children a many-celled periodontal membrane a bone crest lined with uncalcified osteoid tissue, and marrow spaces containing a loose fibrous tissue arc found. In an adult between 30 and 40 years of age there are not so many cells in the periodontal membrane. Thick periodontal fiber bundles are observed, and along the inner bone surface there is a darkly stained resting line. Frequently no osteoid tissue and no osteoblasts are found. The consequence of these observations is of importance especially during the initial stage of tooth movement.

When an orthodontic force is applied, one may note that it usually takes a certain period of time before the cells on the tension side begin to multiply and finally reach a proliferation stage favourable for further tissue changes. The time required for attaining this proliferation stage is dependent upon various factors, including the age of the patient. During this transitory, initial stage of tooth 6-7 movement, it will be practical to apply light forces.

Adult structures especially will need some time to overcome the static condition. While proliferation of new cells is initiated after one or two days in young persons, cell proliferation and formation of osteoid tissue do not start until after about eight days in adult structures. In both cases, nothing would have been gained by applying strong initial forces.

There is one special reason for applying light forces during the initial stage of tooth movement, namely, the tendency to formation of cell-free, hyalinized areas. Hyalinization of periodontal fiber bundles on the pressure side occurs more frequently during the initial stage than later on. This is caused partly by the density of the inner alveolar bone, along which extensive cell-free areas may be formed.

The tendency to such formation decreases as soon as this compact bone plate has been eliminated by resorption. In a great number of cases, formation of cell-free areas is unavoidable. Continuous forces, measurements were made of the force applied as well as of the distance through which the teeth were moved fairly exact, estimation of the distance of labial movement of an upper first premolar can be obtained by inserting occlusal amalgam fillings in the experimental tooth and the control tooth of the opposite side of the jaw. A tiny hole drilled in each filling, should be made so as to fit the metal points indicating the time and the degree of movement, shows the sequence of events in such a case, where continuous force was 30 grams.

It is found that hyalinization remained for nineteen days or as long as the experiment lasted. Fibrous tissue is not removed by osteoclastic resorption. Osteoclasts will remove the bone around the cell-free area. ln this case bone resorption will continue for a few days more until the cell-free area is completely undermined. Then the tooth will suddenly move. The degree

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of movement, also including a period after the cell-free area was undermined. This tooth had a long root. Here hyalinization lasted for about, two weeks.

A considerable amount of new bone has been formed at the outer bone plate as a result of tooth movement. Hyalinized tissue is again reorganized and invaded by new cells. In general, light initial forces art desirable in such cases, because the cell-free area will be less extensive and more readily invaded by cellular elements. If the pressure is strong, new hyalinized areas will be created as soon as the initial cell-free area has been eliminated.

In a continuous tooth movement of individual teeth, it is practical to apply an initial force as light as around 25 grams in adults and around 40 grams in younger patients. After this first stage, the force applied for further continuous tipping of the tooth may be somewhat increased. This is justified by the fact that stretched fibre bundles on the tension side will exert a certain amount of resistance and by the position of the tooth and the type of movement. This can be observed in experimental material. The tooth in the dog that most frequently causes direct bone resorption when moved labially is the second lower incisor.

Direct bone resorption may take place on the pressure side because of the anatomic form of the root and because of its narrow position, by which compression of periodontal fibre bundles is avoided. On the other hand, parallel movement, between root surface and bone surface, occasionally observed during rotation also causes direct resorption. This leads to the second factor in evaluating orthodontic forces namely the type of force to be applied.

The Type of Force

Intermittent forces may be divided into removable appliances in general as distinguished from 8 functional appliances. An example of interrupted continuous forces may be illustrated by tying the tooth to a slightly expanded labial arch. This type of force is favourable especially when it can be kept light enough, A hyalinized area may be created on the pressure side in this instance as well, but, as soon as it has been eliminated by bone resorption the force will decrease rapidly as the tooth moves.

Finally it is retained passively by the arch wire, during which period there will be time for calcification of newly formed preosseous structures laid down on the tension side. On the pressure side, it is characteristic of an interrupted continuous tooth movement for new bone layers to be formed in the many celled tissue at the entrance of open marrow spaces, as soon as tooth movement, is 8 terminated.

Intermittent forces will create a favourable tissue reaction, especially on the pressure side. The resorption process following the application of intermittent or removable appliances is characterized 9 by the interruption of the active force as the plate is moved out, of position. Functional appliances will move more or less in accordance with the movement of the jaws. Thus, in a treatment with intermittent forces the nutritional conditions on the pressure side are

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favoured, to a varying degree, by an increased blood circulation frequently manifested by an augmentation in the number of cells. Functional intermittent of around 70 to 100 grams may cause formation of cell free areas, but these are less extensive and of a shorter duration than in a continuous tooth movement. The degree of tooth movement is considerably increases in accordance with the contact created between the appliance and the tooth surface.

Functional intermittent of around 70 to 100 grams may cause formation of cell free areas, but these are less extensive and of a shorter duration than in a continuous tooth movement. The degree of tooth movement is considerably increases in accordance with the contact created between the appliance and the tooth surface, as well as in accordance with the time period during which the forces were active. A tooth with a short root will be tipped considerably and its supporting tissues will react more favorably to an interrupted continuous force or intermittent forces than to continuous forces. In a comparison of adult teeth with teeth of young persons, it is noted that the adult tooth is tipped less during movements owing to its strong apical fiber bundles. Consequently, direct resorption may be found in the apical area.

Light continuous forces may lie applied in such cases. The current concept means that there is a force of certain magnitude and temporal characteristics (continuous v/s intermitted, constant v/s declining)capable of producing a maximal rate of tooth movement, without tissue damage, and with maximum patient comfort. According to this concept, the optimal force might differ for each tooth and for each patient**(Figure 6)**..

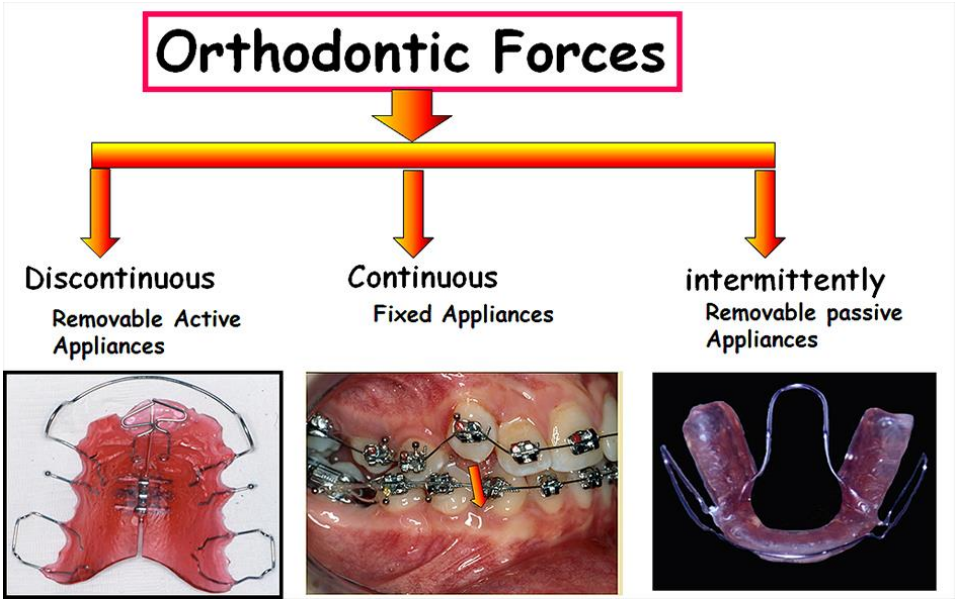

Figure 6: *Classification of orthodontic forces based on appliance type and mode of action.*

The diagram illustrates three types of orthodontic force application: ***Discontinuous:*** *Removable active appliances (e.g., expansion plates) that exert force only when worn.*

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Continuous: *Fixed appliances (e.g., multibracket systems) that apply constant forces over time.* ***Intermittent:*** *Removable passive appliances (e.g., functional regulators) that deliver force only during functional activity such as speaking or swallowing.*

Constant versus dissipating forces in orthodontics:

the effect on initial tooth movement and root resorption :

Weiland et. al. in 2003 conducted a clinical and confocal laser scanning microscopic study, to compare the effects of two frequently used archwires on tooth movement and root resorption. **[30]** A total of 84 premolars in 27 individuals (10 boys, 17 girls, with a mean age of 12.5 years) was moved buccally with an experimental fixed orthodontic appliance. In a split mouth experimental design the premolar on one side was activated with a stainless steel wire with a buccal offset of 1 mm, which was reactivated every four weeks and the contralateral premolar was moved with a superelastic wire with a 10 force plateau of 0.8–1 N. This wire had an initial activation of 4.5 mm and was not reactivated during the 12-week experimental period. At the end of the experimental period the teeth were extracted. Six 10 premolars were used as control teeth and were extracted before the experiment started. Tooth displacement was studied three-dimensionally on dental casts with a co-ordinate measuring machine. The depth, perimeter, area, and volume of the resorption lacunae was measured using three dimensional digitalimages made with a confocal laser scanning microscope . **[21-36]**

On these images the resorbed portions of the root surface were 'reconstructed' mathematically. The results show that the teeth activated with the superelastic wire moved significantly more than the teeth with the steel wire during the experimental period. The depth of the resorption lacunae did not differ significantly between the groups; however, perimeter, area, and volume of the resorption lacunae on the teeth of the 'superelastic group' were 140 per cent greater than on the teeth of the 'steel 10 10 group'. It may be concluded that a greater amount of tooth movement occurred with super elastic wires, offering a force level of 0.8–1 N compared with stainless steel wires, with initially higher but rapidly declining forces in an experimental set up for a period of 12 weeks. The amount of root resorption was significantly larger in the superelastic group**(Figure 7)**..

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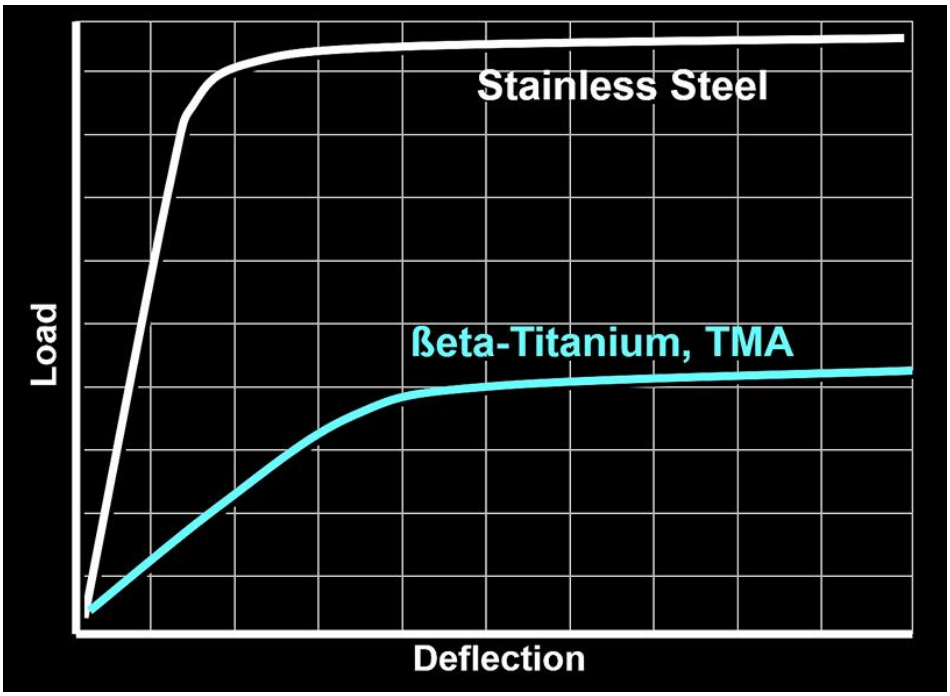

Figure 7: *Force-deflection diagram of orthodontic archwires.*

The diagram illustrates: ***X-axis:*** *Wire deflection,* ***Y-axis:*** *Applied force. Superelastic wires (e.g., NiTi) maintain a nearly constant force over a wide range (plateau), whereas stainless steel wires show a steadily increasing force with deflection.*

Effects of Initially Light and Gradually Increasing Force on Orthodontic Tooth Movement :

Light continuous force results in a relatively smooth progression of tooth movement by frontal resorption traditional orthodontic appliances are not suitable for generating light force because of their material properties, and the force decreases as the tooth moves. We have reported the effect of initially light and gradually increasing force generated by magnets on tooth movement in a previous 5 study. Magnets generate initially light force that depends on the distance between magnets, and the force gradually increases as the magnets move closer to each other. Although application of initially heavy force followed by gradual increasing force showed a lag phase in the initial stage, application of an initially light force followed by a gradually increasing force induced tooth movement without a lag phase. Orthodontic tooth movement is dependent on the ability of periodontal cells to react to the mechanical stimuli. For tooth movement, osteoclast recruitment and activation must be induced to remove bone from the area adjacent to the pressure side of the periodontal tissue. A correlation has been reported in young rats between the number of osteoclasts and the rate of tooth movement. On the other hand, hyalinization in periodontal tissue will limit tooth movement. Hyalinization

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not only inhibits the osteoclastic recruitment in the compressed area for frontal resorption, but also strongly induces the undermining resorption. This degenerative change is caused by excessive force for the periodontal tissue.

Von Bohl et al .**[25]**showed that areas of hyalinization are associated with all applied force levels and also suggested that the development and removal of necrotic tissue is a continuous process during tooth displacement. Delay of the alveolar bone resorption was induced by the removal of hyalinized tissue and, moreover, is suggested to have a correlation with root resorption. Although the histology and biomechanics of many kinds of orthodontic forces have been described, the histological response to initially light and gradually increasing forces is not fully clarified. In a clinical investigation,

Iwasaki et. al. **[28]** concluded that effective tooth movement can be produced with lower forces. It was then hypothesized that smooth tooth movement by initially light and gradually increasing forces could be a result of less hyalinization and increased recruitment of osteoclasts in the compressed area without undermining resorption. The current study histologically evaluated effects of the initially light and gradually increasing force on orthodontic tooth movement in rats.

Tomizuka R. et. al. **[29]** in 2007 conducted a study to investigate histologically the effect of initially 10 light and gradually increasing force on tooth movement in the rat. In this study cuboids made of neodymium-iron-boron magnets (experimental groups) or titanium (control group) were bonded to the lingual surface of the right and left maxillary first molars of 18-week-old male Wistar rats. The initial distances between materials were 1.0 mm generating 4.96 gf (experimental group 1) and 1.5 mm generating 2.26 gf (experimental group 2). In three groups, rats were killed 1, 3, 7, 10, or 14 days 10 10 after treatment. Histological sections were prepared and stained with hematoxyl in and eosin or for tartrate-resistant acid phosphatase (TRAP) activity. The number of TRAP-positive osteoclasts was counted, and the relative hyalinized area was measured on the pressure side of periodontal 10 ligament. There were significant differences in the number of osteoclasts among the three group. On days 1 and 3, the numbers of osteoclasts in experimental group 2 were greater than in experimental 10 group 1. There were significant differences in the relative hyalinized area between the control group and experimental group 1 and between experimental groups 1 and 2 . On days 1 and 3, the hyalinized area in experimental group 1 was larger than in experimental group 2. Therefore it was concluded that Initially light and gradually increasing force induced tooth movement without the lag phase and showed smooth recruitment of osteoclasts and inhibition of hyalinization.

Periodontal Ligament and Bone Response to Sustained Force

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The response to sustained force against the teeth is a function of force magnitude: heavy forces lead to rapidly developing pain, necrosis of cellular elements within the PDL, and the phenomenon of "undermining resorption" of alveolar bone near the affected tooth. Lighter forces are compatible with survival of cells within the PDL and a remodeling of the tooth socket by a relatively painless "frontal resorption" of the tooth socket. In orthodontic practice, the objective is to produce tooth movement as much as possible by frontal resorption, recognizing that some areas of PDL necrosis and undermining resorption will probably occur despite efforts to prevent this.

Biological Mechanisms of Orthodontic Tooth Movement :

V. Krishnan and Z. Davidovitch in 2009 published a review with aims at identifying events that affect the sequence, timing, and significance of factors that determine the nature of the biological response of each para dental tissue toorth odontic force. **[31]** The purpose of this review was expanding the overview of the orthodontic tooth movement process, by delineating reactions occurring in mineralized (alveolar bone) and non-mineralized (PDL and gingiva) para dental tissues, and their associated neurovascular networks. It presents known information about the mechanism of cell signaling in response to mechanical loading, including mechanosensing, transduction, and cellular responses. . **[31-35]**

Furthermore, the various components of this ECM/cellular interrelated chain of responses are presented in an organized sequence, highlighting the links between clinical events and knowledge derived from basic research. **[2,31]**

Extracellular Matrix Remodeling:

Application of external mechanical loads to teeth may alter the effects of gravitational and internal forces acting on resident cells, leading to changes in gene expression, and production of proteins that ultimately alter the structure and function of the ECM, as well as the jaw bones. The most important ECM macromolecules in determining its mechanical properties are collagen (the most abundant molecule), proteoglycans, laminin, fibronectin, elastin, and hyaluronic acid. These molecules bind to . cell adhesion foci, consisting mainly of integrins, to transfer the signals intracellularly Thus, ECMs can be considered to be multi-component tissues that enable internal and external mechanical strains to effect changes in organ structure and function, through mechano transduction. . **[31-37]**

The Concept of Tensegrity

The structure and shape of cells are determined by cytoskeletal molecular structures—microfilaments, microtubules, and intermediate filaments—which link the nucleus to the cell surfaceadhesion receptors. Since microfilaments are chemically attached to proteins in the cellular and nuclear membranes, they are ideal candidates for transfer of tractional forces from inside the cell. These mechanical signals are transferred to the ECM through integrins,

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and to adjacent cells through cadherins, and are resisted and balanced by the same attachment entities. This physiological pre-existing tensile stress in the cell is known as 'tensegrity' (tension-dependent cellular integrity), and is considered to be essential for the normal function of cells, tissues, and organs, as well as for . proper growth and survival. In the absence of this internal tension, cells often undergo apoptosis. **[33]**

The physical properties of the ECM also plays a significant role in cell behavior, by determining the transmission of stress from the outside to the inside of the cell. According to the tensegrity model of Ingber, a stiffer cytoskeleton is advantageous in sensing external mechanical loading, in contrast to a completely relaxed cytoskeleton. The cells are more sensitive to dynamic changes in mechanical stress when the effective external and internal stresses are similar in magnitude. In contrast, the cells in a completely relaxed state are unable to sense an external stress, because it is not matched by an internal counterstress. **[33-35] (Figure 8)**

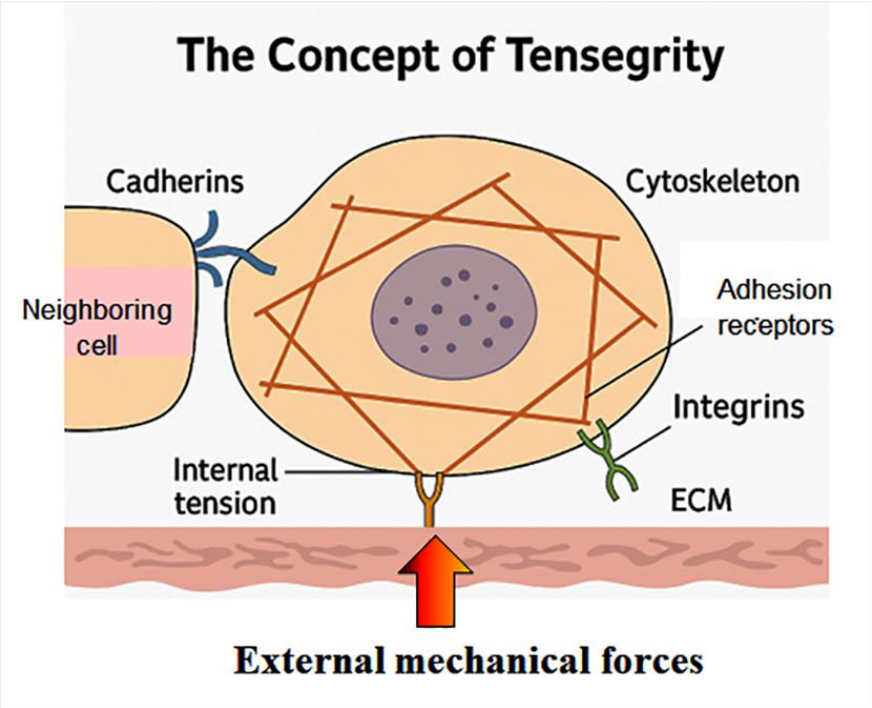

Figure 8: Fluid Dynamic Theory

This diagram illustrates tooth movement through fluid dynamics in the periodontal ligament: ***Compression side:*** *Reduced oxygen, high ion concentration, and bone resorption; blood vessels are narrowed.* ***Tension side:*** *Lower pressure promotes bone apposition; vessels are widened.* ***Hydraulic effect:*** *Interacting fluid systems dampen force like a shock absorber*.**[2]**

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Mineralized Tissue Responses to Applied Mechanical Loads

Alveolar Bone Osteocytes as Mechanosensors. Bone cells (osteoblasts, osteoclasts, and osteocytes) are sensitive to their mechanical environment, and their adaptive response can alter both the mass and the morphology of bones. The important role played by these bone cells, especially osteocytes, as part of mechanosensing or transducing mechanisms in response to applied mechanical stress, has been detailed in the literature. **[3642]**

Osteocytes are connected to each other and to cells of the bone surface by cytoplasmic processes ordendrites. Within a few minutes of the onset of mechanical loading, glucose 6-phosphate dehydrogenase (a marker of cell metabolism)is elevated in osteocytes, and an increase inc-FOS mRNA is observed within 2 hrs. **[36-39]**

Soon thereafter, by 4 hrs, transforming growthfactor â and insulin-like growth factor mRNA expression areincreased. Research reports demonstratingincreases in osteocyte-specific markers 16 have also beenpublished, including E11/gp , dentin matrix protein 1 (found in a tooth movement . model) and sclerostin**.[4143]**

Anabolic signals, such as nitric oxide, prostaglandins, and ATP, are released within seconds of osteocyte loading. All these signals activate bone-surface cells and osteoblasts, by the network ofcell cell communications. It was proposed recently that theWnt/â-catenin pathway initiates, while the SOST/sclerostinpathway inhibits, new bone formation. Osteocytes are the onlycells secreting sclerostin—the product of the SOST gene. **[43-52]**

Sclerostin potently antagonizes and negatively regulates several members of the bone morphogenetic protein (BMP) family of proteins, and also binds to LRP5/LRP6, preventing canonical Wntsignaling. Both BMPs and Wnts are critical for osteoblasto genesis, since they provide the initial and essential stimulus for the commitment of multi potential mesenchymal progenitors to the osteoblast lineage. **[53-60]**

This makes sclerost in a likely candidate as an osteocyte-derived regulator of osteoblast function. SOST transcripts and sclerost in protein levels were dramatically reduced by mechanical loading. The fluid flow hypothesis, describing a mechanism by which osteocytes respond to mechanical forces, states that locally evoked strain derived from the displacement of fluid in the canaliculi is very important. **[53-58]**

When loading occurs, interstitial fluid is squeezed through the thin layer of non-mineralized matrix surrounding the cell bodies and cell processes, resulting in local strain at the cell membrane and activation of the affected osteocytes. Osteocytes may alsosend signals for activation of the bone resorption cascadethrough expression of activator for NF-êB ligand (RANKL),secretion of macrophage colony-stimulating factors (M-CSF),and through their own apoptosis at the sites of 20 micro-damage or-cracks. **[61-70]**

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Gene expression analysis suggests that osteocytes control osteoclast differentiation indirectly through modulation of RANKL expression in osteoblasts. In addition, humoral factors produced and released through canaliculi into the bone marrow may regulate the differentiation and activity of osteoclasts . It has been reported that monocytes are differentiated to osteoclasts in the presence of 2RANKL and M-CSF . It is therefore reasonable to conclude that osteocytes act as chief mechano sensors in bone, which has been recently confirmed by targeted ablation of osteocytes in a mousemodel . **[20,68]**

Alveolar Bone Remodeling in Response to Strain Applied mechanical stress causes flow of bone interstitial fluid, evoking shear stress in the mineralized ECM and deformation of the alveolar bone osteocytes in the lacunae and of the dendrites in the canaliculae.This deformation perturbs the integrin molecules, which act as a tethering protein, opening hemichannels in strained osteocytes, allowing for the release of prostaglandins and facilitating mechano transduction. Mechanical forces alter the metabolic state of osteocytes, which in turn secrete osteotropic factors, such as MLO-Y4, c apable of stimulating surface-lining osteoblasts. The importance of physical contacts between osteocytes and surface-lining osteoblasts through gap junction intercellular communications, formed by members of a family of proteins known as Connexins (Cx), for a proper bone-forming response has been stressed. **[20,68]**

Researchers have detected an increase in the release of PGE2, modulated by hemichannels formed by Cx43, enhancing osteoblastic activity as part of alveolar bone response to mechanical loading . The outcomes of these structural modifications are profound changes in cytoskeletal organization, as evidenced by increases in alpha actin(29%), filamin (185%), and vimentin (15%), altering the metabolic state of the cell, activating signalling molecules inside the cellular cytoplasm, as well as extracellular nucleotides (i.e., ATP and UTP). This promotes a significant stimulation of Runx2 DNA binding activity via a mechanism involving protein kinase C and distinct mitogen-activated proteinkinase cascades. . **[20,68]**

Up-regulation(BMP2, BMP6, ALP, SOX9, MSX1, and VEGFA) and down regulation(BMP4 and EGF) of some osteogenic specific genes in response to cyclic tensile forces have been reported. Time-dependent up-regulation of osterix, anosteoblast-specific transcription factor, which enhanced alkaline phosphatase activity, and the mRNA expression of all osteogenic marker genes (osteopontin, bone sialoprotein, osteocalcin, and collagen) have been observed.

The secreted bone matrix proteins consist of type I collagen (about90% of the organic matrix), which provides strength, structure, and elasticity to mature bone tissue. Type I collagen also regulates expression/ secretion of other non-collagenous proteins, such as osteocalcin, osteopontin, osteonectin, and bone sialoprotein. It is apparent that bone

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formation is coordinated by the expression of many molecules, such as growth factors, transcription factors, and anti-inflammatory cytokines. . **[20,68-73]**

Committed osteoblasts induce/stimulate osteoclastogenesis through the RANK/RANKL/OPG pathway, or become highly differentiated cells, persisting for a very long period of time inside the bone matrix as osteocytes. In physiologic conditions, RANKL is released only by osteoblasts, but in cases of inflammation (e.g., rheumatoid arthritis, orthodontic tooth movement, periodontitis), these molecules areproduced in abundance by T-lymphocytes In addition to RANKL, TNF-á has also been shown to promoteosteoclastogenesis in conditiThis effect is achieved either by stimulation of RANKL release from osteoblasts, or through directactivation of osteoclast precursors. TNF-á apparently inducesbasal osteoclastogenesisvia TNF receptor type 1 (p55), while suppressing it via TNF receptor type 2 (p75), as has been demonstrated in a tooth movement model . Synthesis of other inflammatory mediators and chemokines such as RANTES/ CCL5 and MCP-1/CCL2, capable of hemichannels in strained osteocytes, allowing for the release of prostaglandins and facilitating mechano transduction.ons of inflammatory osteolysis. . **[68-74]**

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These findings strongly suggest that TNF-á plays a pivotal role in the bone resorption process that facilitates orthodontic tooth movement . Bone degradation and terminal osteoclastogenesis require physical contact between osteoclasts and bone matrix. Once differentiated, the capacity of a matrix osteoclast to resorb bone depends on its ability to synthesize and mobilize a series of degradative enzymes. The way osteoclasts perform the bone degradation process has been well-reviewed, and readers are referred to these excellent articles for details .

Non-mineralized Tissue Responsesto Mechanical Loading

Fibroblasts as Mechanosensors and Transducers

The changes elaborated in connective tissues in responseto mechanical loading are largely mediated through fibroblasts. Fibroblasts are considered to be mechano responsive, in that the mechanical signals transmitted from the ECM via integrin receptors influence their morphology, cytoskeletal organization, proliferation, differentiation, and gene expression. Proteins located at focal adhesion kinases(FAK) in the inner cell surface act as strain gauges that ‘sense’stress from the ECM, as well as strain generated by cytoskeletal reorganization .These stretch-induced conformational changes might expose previously hidden

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phosphorylation sites. These newly modified sites are recognized by other signaling molecules (growth factors and cytokines), which in turn activate small GT Pases of the Rho and Ras families, which have multiple effects on cytoskeletal dynamics, gene transcription, and cell division and differentiation. Thus, focal adhesions, together with the actincytoskeleton, have been suggested to serve as mechano sensors in fibroblasts. Fibroblasts in the Mechanically Stressed Periodontal Ligament Various investigators studied the mechano response by gene expression in PDL fibroblasts . . **[88-92]**

PDL fibroblasts exist in an active mechanical environment, filling the gap between the dental rootcementumand the alveolar bone, and are periodically subjected to compressive, tensile, and shearing forces of both short (during mastication) and long duration (during orthodontic treatment).In the PDL, integrin binds to fibronect in in the ECM and total in intra cellularly, as part of establishing a signal transduction pathway. Applied mechanical forces stretch the fibronect in molecules in ECM at sites of PDL tension, straightening the initial nodular assembly with quaternary structure, and unfolding fibronect in type III molecules. The signals transferred to the inside of the fibroblast result in re-organization of the cell's cytoskeleton, leading to changes in cell shape and mobility, induction of gene expression in the nucleus, and secretion of newly synthesized proteins, such as fibronect in and collagen. . **[88-97]**

A quantity of these newly synthesized collagen fibers is incorporated into the newly formed osteoid, where as the rest are embedded in the PDL to form new Sharpey's fibers. It has been demonstrated that, after orthodontic force application, the density of cells expressing positive signalsfor type I collagen mRNA is greater in the PDL in tension sites, followed by expression of type XII collagen. Synthesis of all other collagen types has also been demonstrated(types III, V, and VI) in both tension and compression sites. With recognition of several binding sites, the response of fibronect in to mechanical force is important in the mechano sensing and transduction pathway, leading toECM remodeling. Degradation of collagen and other macromolecules in the ECM as part of the force-induced PDL remodeling is performed by several enzymes, such as serine proteases, aspartate proteases, cysteine proteases, and the matrix metallo proteinases (MMPs; collagenase, gelatinase, and stromelysin). Specific up-regulationof MMP-8 and -13 mRNA in PDL compression and tensionsites during orthodontic tooth movement has been demonstrated, stressing the role of ECM degradationin the remodeling process. . **[2]**

Gingival Fibroblasts and Mechanical Forces Gingival fibroblasts play an important role in determining tissue architecture, by virtue of their attachment to the a cellular cementum in the cervical part of the dental root, and to the gingival fibrous matrix at the other end of the fibers. It has been proposed that if there is any disruption in the chemico-physical connectivity of the marginal gingiva to the dental root, the intracellular tensile forces of gingival fibroblasts are abruptly lowered, changing the balanced physiological strains

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existing in the cells (tensegrity). In response, molecular changesmay occur, activating propagation of intracellular messengers that start remodeling responses in the gingiva, as well as in the alveolar bone. In this situation, the marginal gingiva acts as a mechano sensor apparatus, recognizing and responding to abrupt modifications in physical stimuli. Once intracellular and cell-ECM strains are lowered, a molecular signaling pathway, with alocal release of ATP, and up-regulation of a specific p2x4purinoreceptor in the gingival fibroblasts are initiated. These events lead to an increase in intracellular calcium influx, transducing extracellular signals to the interior, and subsequently to the genome (Blood Vessel Re-organization and Neo vascularization in Orthodontic Tooth Movement Blood vessels in the PDL, which provide gases and nutrients required for all metabolic activities of their surrounding tissues, in health and disease, are active participants in the tissue remodelling associated with orthodontic treatment. Furthermore, these vessels usher immune cells into the strained PDL, extending a helping hand for their role in the tissue-remodeling process. They also permit migration into the strained paradental tissues of hormones and a variety of molecules derived from food and drugs consumed by the individual. . **[88-92]**

All these molecules-thosethat originate from native paradental cells or migratory leukocytes, or from remote endocrine glands, or from consumed nutrients and drugs—may have profound and long lastingeffects on the nature of the force-induced tissue remodeling and the degree of success of orthodontic treatment The ECM surrounding the vessels provides critical support for the vascular endothelium. Adhesion of the endothelial cell tothe ECM is required for its proliferation, migration, morphogenesis, survival, and blood vessel stabilization, to maintain tissue viability and neo vascularisation. During angiogenesis, with the help of the ECM (which serves as a three-dimensional malleable scaffold), proliferating and migrating endothelial cells organize to form new three dimensional capillary networks. By generating mechanical contractile forces within the ECM, endothelial cells, which acquire shape changes with the help of the COL1A1 gene, establish tension based guidance pathways that allow them to form inter connected cords . **[20,68]**

A collagentype I gene is involved in acquiring shape changes by the endothelial cells. Over the course of several days, inter connected cords mature to form hollow tubes through a process involving the development and coalescence of intracellular vacuoles. The key angiogenic cytokine, vascular endothelial growth factor (VEGF), which induces sprouting angiogenesis, also induces microvascular endothelial cells to express integrins . ligation to integrins in endothelial cells suppresses cAMP, thereby suppressing The sequence of neo vascularisation:

Collagen type I PKA activity of cAMP mediated protein kinase A (PKA). activity suppression leads to a marked induction of act in polymerization, leading to the formation of prominent stress fibers and endothelial cell contractility.

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and GTPase Rhothrough interaction with integrins, which also results incord formation. Applied mechanical stresses develop strain and activate signalling pathways, mainly through integrin-dependent mechanisms, followed by act in polymerization, activation of type I collagen genes, neo vascularization, and remodeling of blood vessels. Theentire process is self-limiting, once sufficient changes are obtained to nourish the associated structures. With the help of light and transmission electron-microscopic observations ,showed a reduction in the number of blood vessels after 72 hrs of treatment and an increase in their number after 7 days of treatment. Modifications of immunofluorescence staining patterns of tested proteins revealed angiogenesis and reparative processes, along with thickening of fibrillar matrix, as a defensive reply to mechanical stress. Through a quantitative representation of PDL vascular reactions in rats, discovered that prolonged force applications lead to increased vascularity, which is age-related. In young rats, increased numbers of both small and large blood vessels were found, while adult rats exhibited an increase in the number of only small blood vessels. These results confirm earlier findings on the responses of blood vessels to applied mechanical forces, typified by increased permeability, which enhances fluid extra vasation from capillariNeural Responses to Mechanical Forces Innervations of dental and paradental tissues are mainly constitutedby neurons that originate in the trigeminal ganglia (located in the cervical and middle root areas) and mesencephalic trigeminal nucleus (apical periodontal area). The nerve endings consist mainly of mechano receptors (Ruffini-like endings) and nociceptors. Under normal physiological conditions, these receptors remain quiescent, but contain various neuropeptides, such as calcitonin gene-relatedpeptide (CGRP) and substance P(SP). Orthodontic tooth movement alters the neutral state of both mechano receptors and nociceptive PDL nerve fibers, effecting the release of biologically active proteins, leading to local neurogenic inflammation and the release of neuropeptides such as CGRP and SPes into the interstitial space. . **[88-92]**

Since most PDL neurons are wrapped around blood vessels, including capillaries, endothelial cells are probably the first to interact with neuropeptides, which in turn bind circulating leukocytes, facilitating their migration from the capillaries. Chemokines activate integrins on the leukocyte cell surface to promote firm adhesion. The migration of leukocytes by the chemo attractant gradient and their arrival in the PDL signify the on set of an acute inflammation, which is essential for tooth movement into newer locations. Signaling molecules released by these migrating leukocytes (cytokines, growth factors, and colony-stimulating factors) interact with various dental and para dental cells and stimulate them to initiate and sustain the tissue remodelling. Monocytes, lymphocytes, and mast cells express receptors for neuropeptides, which transduce signals intra cellularly and evoke cellular responses, such as cytokine release, production of other neuropeptides, changes in the expression of mediators, or direct release of inflammatory mediators. . **[88-91]**

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Moreover, CGRP and SP also serve as vasodilators, increasing vascular permeability, flow, and plasma extra vasations. Often, this plasma contains molecules derived from the diet, medications, remedies drugs, hormones, and inflammatory mediators that originated in remote diseased organs, which might influence the course of mechano therapy, by virtue of their interaction with cells. In addition to this peripheral action, the affected neurons release neuro transmitters centrally, in the trigeminal nucleus, causing pain shortly after appliance activations in persons undergoing orthodontic treatment . **[2]**

The short delay in the onset of pain is due to the initial resistance of the tissue fluids. The gradual movement of these fluids from areas of compression to zones of tension facilitates the development of ECM strain and the entire process of mechano transduction. **[2]**

Pressure–Tension in Periodontal Ligament ;

Tooth movement as currently understood from a "wet-fingered" clinical perspective is that when an orthodontic appliance applies a force, there is little movement initially but after a month or so teeth begin to move. **[20]**

A clinician's understanding is that of a cell mediated process - that the periodontal ligament (PDL) experiences "pressure" in the direction of movement and "tension" on the opposite side. The PDL is recognized as the major site of activity with some contribution from undermining resorption on the medullary side of the lamina dura. This model is uncomplicated and visual, explains and predicts tooth movement based upon over a century of observations, aids in clinical decision making, and can be comprehended in its entirety because this pressure-tension model contains only those structure and process features that are of primary importance. Teeth move at a rate that is somewhat predictable, comprehensive orthodontic treatment takes 21–27 months for non-extraction therapy and 11 months for extraction treatment. . **[62-68]**

The pressure–tension theory, the classic theory of tooth movement, relies on chemical rather than 1 electric signals as the stimulus for cellular differentiation and ultimately tooth movement. Chemical messengers are important in the cascade of events that lead to remodeling of alveolar bone and tooth movement, and both mechanical compression of tissues and changes in blood flow can cause their release. Sustained pressure against a tooth causes the tooth to shift position within the PDL space, compressing the ligament in some areas while stretching it in others. The mechanical effects on cells within the ligament cause the release of cytokines, prostaglandins, and other chemical messengers. Blood flow is decreased where the PDL is compressed while it is maintained or increased where the PDL is under tension . These alterations in blood flow also quickly create changes in the chemical environment. For instance, oxygen levels certainly would fall in the compressed area and carbon dioxide (CO_2) levels would increase, while the reverse might occur on the tension

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side. These chemical changes, acting either directly or by stimulating the release of other biologically active agents, then would stimulate cellular differentiation and activity. **[62-78]**

Cellular and Molecular Behavior in Sites of PDL Tension and Compression;

Orthodontic forces (continuous, interrupted, or intermittent) create strains in the teeth and paradental tissues. These strains are not uniform throughout the applied region, but rather develop a 'quilt' of areas where tension or compression prevails, creating favorable conditions for tissue remodeling. Great clinical interest exists regarding the behavior of cells in these strained locations, since it determines the orthodontic treatment outcome. An insight into this puzzle may be obtained from investigations on the responses of paradental cells to conditions of tension, compression, or torque. It has been reported that PDL cells respond differently to tensile and compressive strains, in 26 terms of synthesis and degradation of ECMcomponents . A 10% compressive strain (0.5 Hz) decreased collagen (COL1A1) mRNA, type I collagen, and fibronect in levels in these cells. In contrast, a 10% tensile strain increased COL1A1 mRNA, MMP2 mRNA, and TIMP2 mRNA .

At the same time, tensile forces result in the synthesis of similar levels of both MMP and TIMP, promoting anabolic activity, whereas synthesis of MMP is increased under compressive loads without a change in TIMP, thus promotingdegradation . Recently, an increase in type I collagen immuno fluore scence staining intensity was demonstrated in PDL pressure sites, while tension sites displayed a diminution of staining after72 hrs of treatment

Type IV collagen staining was reduced in both sites, increasing gradually after 7 days of treatment .Orthodontic forces induce an aseptic inflammatory response, rather than a reaction to an invasion by micro-organisms through a wound. Nonetheless, the classic signs of inflammation (redness, swelling, pain, and reduced function) are similar in both cases. Inflammatory cytokines produced by leukocytes, platelets, and native cells, which include lymphocyte-and monocyte derived factors, colony-stimulating factors, growth factors, and chemotactic chemokines, are often involved in tissue reactions associated with orthodontic 26 forces .Inperiodontal tissues, cytokines are produced by immune cells, fibroblasts, and osteoblasts, and participate in regular tissue turnover and in induced bone-remodeling events. Both these procedures are mediated by pro- as well as anti-inflammatory cytokines under the influence of distinct cytokine subsets. Elevatedlevels of IL-1â, SP, and PGE2 in gingival crevicular fluid fromboth compression and tension sites of moving teeth have beenidentified, with higher concentrations found in tension sites. **[62-78]**

Recently, the differential expression patternsof 3 cytokines (TGF-â, IL-10, and TNF-á) in PDL 26 compressionand tension sites during tooth movement have been reported. An augmented expression pattern forTGF-â mRNA has been reported at sites of both PDL compression and tension, which induces proliferation and chemotaxis of PDL cells, up-

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regulates COL-I, recruits osteoblast precursors, inducing their differentiation, down-regulates MMPs, and up regulates TIMPs, enhancing 26 production of bone matrix proteins. In areas of compression, TGF-â inhibitsrecruitment of osteoclast precursor cells, suppressing osteoclastic activity. **[20]**

Compression-site PDLcells have been reported to express increased amounts of TNF-á,which stimulate the production of MMPs, elevating the levels of RANKL to be directly involved in bone resorption. In contrast, simultaneous up-regulation of OPG and down-regulation of RANKL expression by the anti-inflammatorycytokine IL-10 inhibit bone resorption. The dual role of cytokines in the remodeling ofmineralized and non-mineralized connective tissues can bedescribed as that of a pro-inflammatory cytokine, such as IL-1and TNF-á, which promotes resorption and inhibits apposition,while anti-inflammatory cytokines promote apposition and inhibit resorption. . **[20]**

Chemokines, the superfamily of chemotactic cytokines, are small structurally related heparin binding proteins classified into 4 subfamilies based on the configuration of cysteine residues near the N-terminal, depending on whether the first 2cysteines are separated (CXC, CX3C) or not (CC, C). They are synthesized by a variety of cell types, such as endothelial, epithelial,and stromal cells, 26 fibroblasts, mast cells, bone cells, and leukocytes. . **[68]**

Functionally, chemokines are divided into homeostatic and inflammatory molecules, which target all types of leukocytes and lymphocytes through their binding to selective G-protein-coupled receptors. The activation of these receptorstriggers several intracellular signaling pathways, resulting in reorganization of the cytoskeleton and cell adhesion, causingcells to extend pseudopodia and crawl up the chemo attract antgradient. . **[70]**

Chemokine-driven cellmigration is thought to be an important step in orthodontic force induced paradental tissue remodeling, especially in bone remodeling and angiogenic events. Osteoclastprecursors express chemokine receptors such as CCR2 andCCR5 and signals provided by chemokines, such as CCL2/MCP-1, CCL3/MIP-1á, and CXCL12/SDF-1, which are essentialfor the differentiation of osteoclast progenitors into matureosteoclasts. High expression patterns have been demonstrated for MCP-1, CCL5/RANTES, and MIP-2 in bone resorption areas associated with PDL compression. . **[70-76]**

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Osteoblasts also express receptors, such asCXCR4, CXCR5, and CCR5, which function by binding to specific chemokines, such as SDF-1 and BCA-1/CXCL13,resulting in proliferation and 26 type I collagen mRNA expression. Moreover, osteoblasts can secretechemokines such as MCP-1, SDF-1 (involved in bone remodeling),KC/CXCL1, LIX/CXCL5, CINC-1/CXCL1, and BCA 1/CXCL13 (involved in the recruitment of different leukocytes) in response to mechanical force application. **[73-84]**

These findings suggest an interesting role for osteoblasts in the development of aseptic inflammatory reaction in the PDL following orthodontic force application. Chemokines, the known regulators of cell-trafficking and tissue remodeling, play a crucialrole in the migration of cells into specific organs and tissues. **[73-82]**

In this capacity, chemokines initiate signal transduction events, leading to other biological processes such as angiogenesis, cell proliferation, apoptosis, and host Defense. Chemokines of the CXC family areunique in promoting angiogenic (CXCL1/GROá, CXCL2/GROâ, CXCL3/GROã, CXCL5/ENA 78, CXCL6/GCP-2,CXCL7/NAP-2, and CXCL8/IL-8) and angiostatic (CXCL4/PF4, CXCL9/Mig, CXCL10/IL-10, CXCL11/I-TAC, andCXCL14/BRAK) activities associated with any sort of chronic inflammation. Hence, chemokines appear to emerge as important factors, in more than one way, in the remodeling of tissues subsequent to the application of orthodontic forces. It can be concluded that orthodontic forces are capable of creating aseptic and transitory inflammation at both PDL compression and tension sites. Although a clear-cut microscopic and/or computerized to mographic demarcation between are a sunder compression and tension is not always possible, because these zones often overlap, the bulk of research outcomes to date points to the fact that the reaction a teach of these areas differs clearly on the molecular and cellular levels. Future research will continue to increase our knowledge about this dichotomy. **[73-82]**

Drawback There are two major conceptual problems associated with the hypothesis. First, does stretching of the principal fibre bundles generate tension and second, can differential pressures be developed within the tissues of the periodontium. During the 1970s, the mechano biological pressure-tension model was well established as it became more apparent that mechanical strain activates multiple cell internal signaling pathways and/or second messengers in order to modulate the behavior of all cells responsible for tooth movement within the hydrodynamic periodontal ligament and alveolar bone. 26 Knowledge of these molecular signalling systems in the biomedical sciences exploded at an 26 alarming rate during the “long 1970s.” Krishnan expanded the overview of the orthodontic tooth movement process, by delineating reactions occurring in mineralized (alveolar bone) and nonmineralized (PDL and gingiva) paradental tissues and their associated neuro vascular networks. The authors presented known information about the mechanism of cell signaling in

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response to mechanical loading, including mechanosensing, transduction, and cellular responses. They presented the various components of this extracellular matrix (ECM)/cellular interrelated chain of responses in an organizedsequence, highlighting the links between clinical events and knowledge derived from basic research. The pressure-tension concept is currently, and has been, the essence of and central to explanations of tooth movement. Meikle indicated that the idea that pressure and tension sites are generated within the PDL is firmly embedded in the orthodontic subconscious and it continues to play a key role in organizing our ideas, as well as advancing our understanding of a complex biological process**(Figure 9)**. **[73-86]**

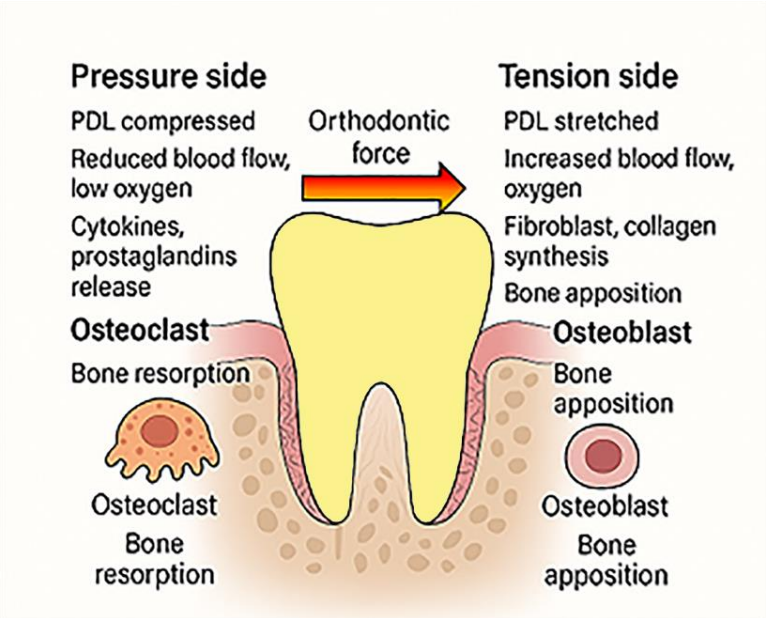

Figure 9: *Schematic illustration of the pressure–tension theory in orthodontic tooth movement. The diagram depicts the biological response in the periodontal ligament (PDL) under orthodontic force:* ***Pressure side (left):*** *Compression of the PDL leads to reduced blood flow, decreased oxygen levels, and the release of cytokines and prostaglandins activation of osteoclasts bone resorption.* ***Tension side (right):*** *Stretching of the PDL results in increased blood flow and oxygen supply activation of fibroblasts and osteoblasts → new collagen fiber synthesis and bone apposition.*

After 100+ years, the orthodontic community has a reasonably good understanding of the sequence of events involved in orthodontic tooth movement at the tissue and cellular levels on both the tensile and compression sides of the periodontium. Scholarly literature suggests that much is known about bone apposition and resorption and “that” understanding explains tooth movement. The pressure-tension model, as represented in the scholarly literature, is depicted as a natural physical event. Orthodontists heretofore regard pressure-tension as an

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event that is characterized by osteoclastic resorption on the pressure side and osteoblastic apposition on the tension side – that to understand these essentials is to understand tooth movement. Such a view of the pressure-tension construct is spatiotemporally restricted to the PDL and is insensitive to tissue strain variation except that too much biomechanical force will hyalinize the PDL and inhibit tooth movement for a time. **[82-92]**

Fluid dynamic theory

Was given by Bien in 1966 also called blood flow theory of tooth movement occurs as a result of alterations in fluid dynamics in periodontal ligament. The tooth oscillates in its socket in response to intrusive forces of short duration. The force, which damps these oscillations of the tooth in its socket, has been shown by Bien and Ayers to be a hydraulic damping effect. Within the geometry and structures of the periodontium, fluid systems act in the transmission and damping of forces acting on a tooth. **[2]**

There are three distinct but interacting fluid systems that are involved in damping oscillation of the tooth in its socket; first, the vascular system enclosed within the blood vessel and lymph vessel walls; second, the system of the periodontal membrane comprised of cells and periodontal fibers; and third, the interstitial fluid continuum that permeates the spaces betweThese systems are analyzed in terms of their viscous and inertial characteristics. The dynamic activities of the fluid systems operating in the periodontium exhibit separate but interrelated functional properties. Peridontal space is a confined space and passage of fluid in and out of this space is limited. A hydrodynamic condition is created that resembles a hydraulic mechanism and a shock absorber. On application of force 'squeeze film effect' results. en the cells, fibers, blood vessels, tooth, and bone**(Figure 10)**. **[73-82]**

This results in reduced oxygen level on the compression side, escaping of blood gases into interstitial fluid creating a favourable environment for resorption. Rapid decalcification in the pressure areas would throw a high ion concentration into the local interstitial fluids. During chewing, these fluids fill up the tension areas with a free expansion at reduced pressure, showing the Venturi effect of hydraulic jump. Further support for these views on the relatively lower pressure on the tension side is given by the findings of Zaki and Van Huysen that the blood vessels in the periodontium on the pressure side of moving teeth are smaller than those on the tension side. During chewing, these fluids fill up the tension areas with a free expansion at reduced pressure. **[82-92]**

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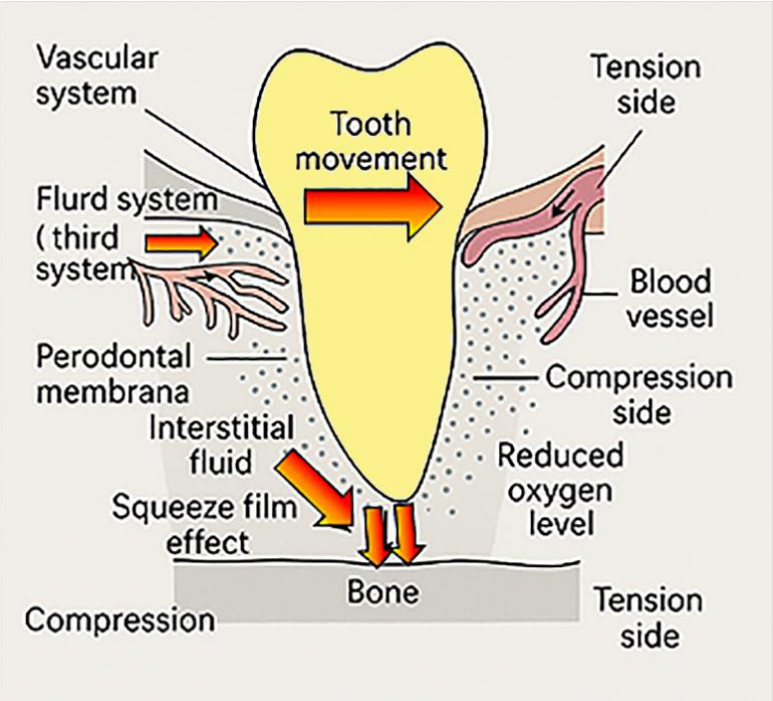

Figure 10: *Fluid Dynamic Theory. This diagram illustrates tooth movement through fluid dynamics in the periodontal ligament:* ***Compression side:*** *Reduced oxygen, high ion concentration, and bone resorption; blood vessels are narrowed.* ***Tension side:*** *Lower pressure promotes bone apposition; vessels are widened.* ***Hydraulic effect:*** *Interacting fluid systems dampen force like a shock absorber.*

The free expansion at reduced pressure accompanying slight tooth movements would account for the increased size of the vessels on the tension side, whereas the vessels on the compression side would be of relatively smaller diameter because of the greater pressure. Filling the interstitial spaces in tension areas with the fluid containing high ion concentrations from the pressure areas establishes a favorable climate for the formation of a calcification front. Without contributing factors other than the central heart pump system the circulation of blood in bone would come to a standstill according to Trueta. He shows muscular involvement in osseous circulation, Trueta points out that bone deposition and removal are related to such vascular changes as rate, rather than primarily in cellular activity. **[82-94]**

The bone-bending theory ;

Farrar was the first to suggest, in 1888, that alveolar bone bending plays a pivotal role in orthodontic tooth movement. According to these authors, when an orthodontic appliance is activated, forces delivered to the tooth are transmitted to all tissues near force application. These forces bend bone, tooth, and the solid structures of the PDL. Bone was found to be

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more elastic than the other tissues and to bend far more readily in response to force application. **[82-92]**

Biphasic Theory of Tooth Movement:

Understanding the molecular and cellular events during orthodontic tooth movement can greatly impact daily orthodontic practice. Selecting the most appropriate force magnitude, knowing precise tooth movement, optimizing activation intervals, preventing side effects, and, most importantly, developing techniques that increase the rate of tooth movement are all influenced by this understanding.

These events can be divided into two main phases, a catabolic phase, where osteoclast-driven bone resorption determines the rate of tooth movement, and an anabolic phase, where osteoblast-driven bone formation re-establishes and maintains alveolar bone integrity of the new occlusion. These two phases are not simultaneous or independent – the catabolic phase is required and always precedes the anabolic phase. We call this biological phenomenon the Biphasic Theory of Tooth Movement . **[82-92]**

While cytokines play an important role in initiating the catabolic phase, interaction between osteoclasts and osteoblasts regulates the anabolic phase. Therefore, to increase the rate of tooth movement, acceleration techniques must focus first on producing higher cytokine activity and second on enhancing osteoclast and osteoblast interactions to expand the boundary of tooth movement and maintain the integrity of alveolar bone in the newly established occlusion. **[68]**

Three types of bone cells play a significant role in the biology of tooth movement: osteoblasts, osteocytes, and osteoclasts. Osteoblasts are mononuclear cells found along the surface of bones. They are derived from mesenchymal stem cells in the bone marrow and synthesize collagenous and non collagenous proteins that comprise the organic bone matrix, the osteoid. Inactive osteoblasts that cover bone surfaces, particularly in the adult skeleton, are called bone lining cells. These cells are quiescent until growth factors or other anabolic stimuli induce their proliferation and differentiation into cuboidal osteoblasts. Osteoblasts are the main cells participating in the anabolic phase of orthodontic tooth movement with a limited role during catabolic phase. **[82-92]**

Osteocytes are mature osteoblasts embedded in lacunae within the bone matrix. Although immobile, osteocytes possess exquisitely fine processes, which traverse the mineralized matrix in tunnels called canaliculi, to make contact with other osteocytes, as well as with osteoblasts residing on the bone surface. Given their preponderance in the bone, and their intricate three-dimensional network, osteocytes are key mechano sensors that recognize mechanical load and, by regulating osteoclast and osteoblast activity, reshape the bone to fit the mechanical demand. **[92-97]**

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The mechanism by which mechanical stimulation activates osteocytes is not clear. Loading of bone under physiologic condition results in strain, or deformation, in the bone matrix and the lacunae and canaliculi that surround the osteocytes. Some authors suggest that it is the magnitude of the matrix deformation (strain) that triggers bone remodeling . Conversely, others argue that load itself is not the main ostoeogenic component of mechanical stimulation, but, instead, load by-products such as strain rate , strain distribution, or fluid flow are the primary remodeling initiators. While this controversy remains under active investigation, there is consensus that mechanical stimulation is detected by osteocytes via fluid shear stress produced by increased fluid flow in the lacuno canalicular system and electrical strain potentials. These responses to mechanical load activate osteocytes to secrete key factors, such as prostaglandins, nitric oxide, or insulin-like growth factors (IGFs), which then activate osteoclasts and osteoblasts in a tightly synchronized biological phenomenon called bone remodeling. Osteocytes are critical for normal bone remodeling, the precise role they play in the biology of tooth movement is unknown. **[82-92]**

They may play a role in the catabolic phase of movement by activating osteoclasts. However, it is more probable that they play a role in the anabolic phase by coordinating osteoblast activation . The last cell type that plays a significant role in orthodontic tooth movement is the osteoclast, which is the major bone resorbing cell.

Osteoclasts are specialized monocyte/ macrophage family members, formed by the fusion of numerous monocytic precursors to create giant multi nucleated cells .Activation of osteoblasts by osteoclasts is observed during tooth movement where the bone resorption phase of tooth movement is followed by a bone formation phase to prevent bone loss during tooth movement. Similar phenomenon can be stimulated during movement of a tooth into an area of alveolar bone loss . These areas usually are occupied with thick cortical bone that is short in height and narrow in width. Moving a tooth in this area is slow, can cause root resorption, and usually results in tilting the crown into the edentulous space without significant root movement. In the Biphasic Theory of Tooth Movement , osteoclasts play an important role in the activation of osteoblasts. This is in agreement with numerous studies that suggest osteoclasts are principle regulator of osteoblast activity. In healthy individuals, osteoclast activation is tightly coupled to osteoblast activation. **[2]**

This effect can occur through different pathways :

1)Osteoclasts release paracrine factors that directly recruit and activate osteoblasts,

2) Osteoclasts activate osteoblasts through direct cell-cell interaction, and

3) Bone resorption by osteoclasts exposes bone matrix proteins that then attract and activate osteoblasts

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While these pathways differ fundamentally, they do share an important feature. In each case, osteoclast activity precedes osteoblast activity. This directionality is seen any time osteoclasts are activated and is best visualized in the remodeling cone where the head of the cone is occupied by osteoclasts and the tail of the cone is filled with osteoblasts. By harnessing this repeatable and predictable sequential process, we can increase the anabolic effect of orthodontics in both trabecular and corticalbones**(Figure 11)**. **[86-92]**

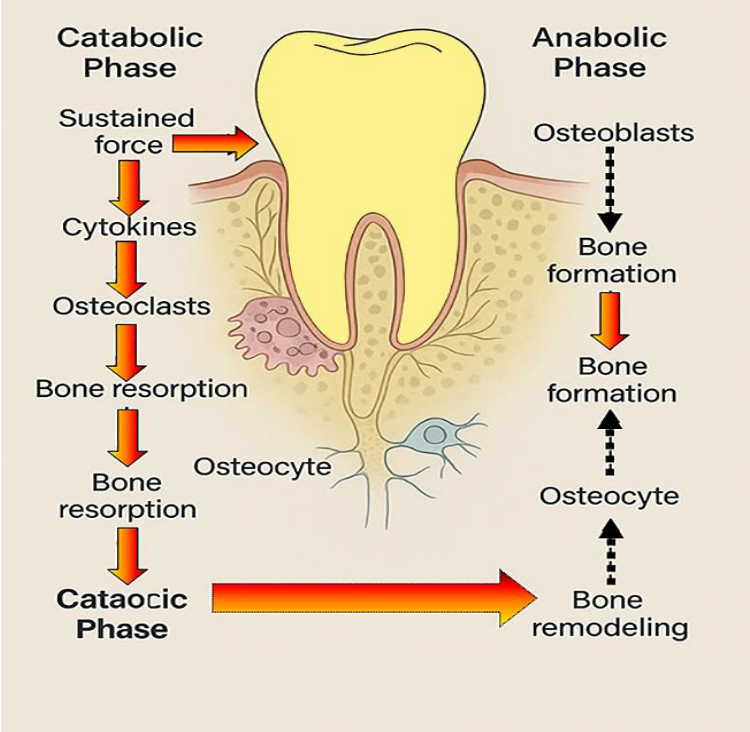

Figure 11: Biphasic Theory of Tooth Movement. *This diagram depicts the biological process of orthodontic tooth movement in two phases:* ***Catabolic Phase:*** *Osteoclasts resorb bone on the pressure side, enabling the tooth to move.* ***Anabolic Phase:*** *Osteoblasts rebuild bone on the tension side to stabilize the new tooth position. Osteocytes act as mechanosensors, detecting mechanical load and coordinating the activity of osteoclasts and osteoblasts.*
The two phases occur sequentially and are essential for controlled and stable orthodontic tooth movement.

How Do Teeth Move Orthodontically?

Orthodontic forces strain ECM and cells of the alveolar bone, PDL, gingiva, and associated blood vessels and neural elements, the initial effect of which is physical in nature, followed closely by a biological response. **(Figure 12)**

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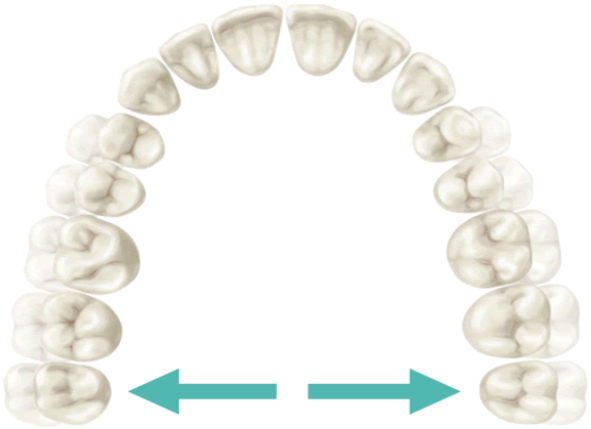

Figure 12: Expansion is moving posterior teeth outwards away from the midline.

This interaction generates profound changes in the structure and function of the ECM, cell membrane, cyto skeletal elements, nucleus, and several other cytoplasmicorganelles that synthesize and mobilize a variety of molecules inside and outside the cells. Cell adhesion molecules, like the integrins, transmit tensile, compressive, and shear stresses directly from the ECM into the cell and vice versa, and are considered essential for cellular survival, growth, and mobility. **(Figure 13) [82-92]**

This transmission helps to maintain the cells in active form(tensegrity), capable of responding rapidly to various mechanical, physical, and other challenges. Mechano sensing is a process by which cells sense structural changes in the ECM, caused by external mechanical loading. This sensing triggers biochemical reactions that generate energy, in addition to the energy provided by the mechanical load, required for the response to the elevated environmental demands .Each cellular system in paradental tissues is equipped with mechano sensors thus, each mechanical stimulus may activate multiple mechano sensors, followed by downstream cellular events. Subsequent changes in cytoskeletal protein structure and function propagate the signaling process into the nucleus by a process known as mechano transduction, defined as the process by which mechanical energy is converted to biochemical and/or electrical signals. **(Figure 14) [82-97]**

In addition, signalling proteins generated in the cell cytoplasm, such as hedgehog and transforming growth factors, as well as calcium ions, reach the nuclear matrix and, ultimately, the genome. The outcome of the entire chain of events is enhanced or suppressed gene expression, transferring he signal back to the cytoplasm through mRNAs, reaching the ribosomes, generating protein synthesis and secretion, mitosis, cell motility, and programmed cell death The unique feature of this scheme in orthodontics is the interaction between various tissues, both mineralized and non-mineralized, and their

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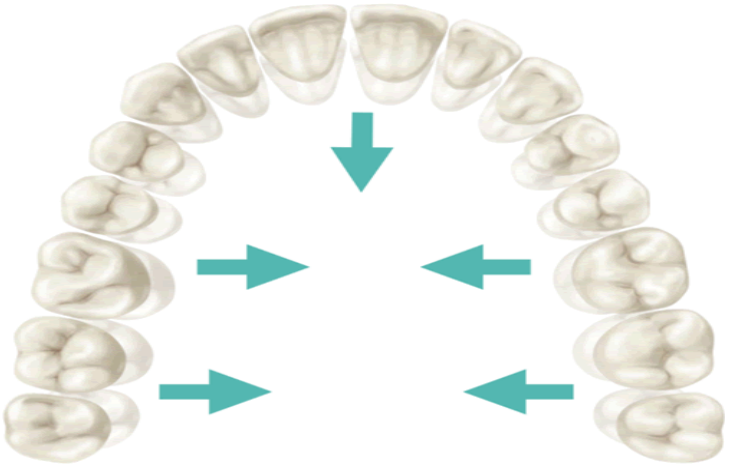

Figure 13: Lingualization is moving teeth towards the tongue side of the arch.

associated Neurovascular elements, for effecting tooth movement. For such an event to happen, remodeling of various tissues—performed by cells of the alveolar bone(osteoblasts, osteoclasts, and osteocytes), PDL, and gingival fibroblasts, blood vessels (endothelial cells),and neural tissues (dendritic and neural cells)—mustoccur. Additional cell types essential to the body's response to applied mechanical loads are derivatives of the immune system (inflammatory cells and osteoclast progenitors). Each of these tissues follows its own response pattern, as far as the mechano sensing, transduction, and response mechanisms are 34 concerned . Orthodontic forces bend the alveolar bone, and compress and stretch the PDL. Consequently, there will be an alteration in the electrical neutrality of the alveolar bone, as the compressed PDL becomes the site of intense bone resorption, while stretched PDL are as interface with sites of active osteogenesis. Osteocytes are key participants in this process, being acutely sensitive and responsive to applied mechanical loads. Drugs, Hormones and Orthodontic tooth Movement . **[2,20,59,68]**

Their elaborate network of cellular projections facilitates communications with neighboring osteocytes, as well as with alveolar bone surface-lining cells and bone marrow cavity cells. Osteoblasts, which maintain direct contact with osteocytes, respond to these signals and initiate appositional changes. The activated osteoblasts proceed to convey signals to approaching osteoclasts, enticing these cells to start resorbing the alveolar bone, and informing them on the proper time to cease their resorptive activities. The origin of osteoclasts for bone resorption is attributed to either conversion ofmonocytes/macrophages or migration of progenitor cells from alveolarbone marrow cavities to the strained PDL . Important components of this osteocyte-osteoblast-osteoclastchain are PGs, TNF-á, and the RANK/RANKL/OPG system. Gingival and periodontal fibroblasts play different roles inparadental tissue remodeling. While PDL fibroblasts are predominantly involved in the synthesis and degradation of theirown ECM, gingival fibroblasts participate in bone

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remodelling events. Up regulation of 85 genes and down-regulation of 23genes associated with protein synthesis in the PDL have been identified . **[54,68]**

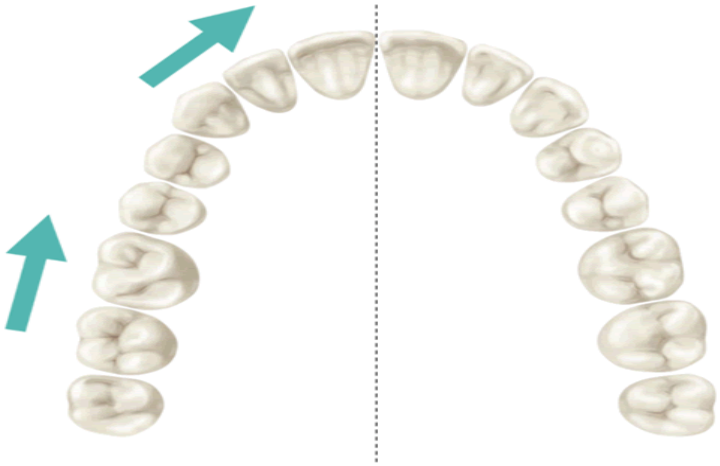

Figure 14: Mesialization is moving a tooth along the occlusal plane towards the midline.

Diminution of collagen production(type I and IV) in the compressed PDL has been reported, as well as increased type IV collagen production at PDL tension sites after 72 hrs of force application .The role played by blood vessels in orthodontic tooth movement has received much recent attention. Angiogenesis and remodeling of existing blood vessels help them adapt to the new environment created by mechanical forces. An initial reduction and a later increase in the number of PDL blood vessels following orthodontic force application have been reported . These blood vessels play a major role in the mechanical force induced aseptic inflammation byacting as sources of cytokines and chemokines. Increasedexpression of IL-1â, IL-1 receptor, IL-6, IL-6 receptor, IL-8receptor, IL-11, and TNF-á has been demonstrated in the compressedPDL, confirming the presence of aseptic inflammation substance P, CGRP, VIP, and other released neurotransmitters interact with endothelial cells of the paradental capillary network, enticing them to bind circulating leukocytes, promoting their migration into the paradental ECM. Upon entering these tissues, the migratory leukocytes produceample amounts of chemokines and cytokines. Along with the cytokines produced by the native paradental cells (fibroblasts and osteoblasts), they evoke and maintain the remodeling events of the PDL and alveolar bone. The released neuropeptides also act centrally to produce pain, which in turn restricts the use of the jaws and teeth by the individual, whether for mastication or speech. **[2,20,68] (Figure 15)**

These reduced movements of contraction and relaxation of the PDL might provokea loss of transmission of mechanical stresses to the ligament, delaying the cellular responses due to the state of inactivity and loss of tensegrity. Increasing the magnitude and frequency of

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functional force application to orthodontically treated teeth may restore the pace of tooth movement during these painful periods. A similar effect can be achieved by physical, chemical, or surgical means**[2,20,68]**. This phenomenon further emphasizes the fact that physical forces of gravity, hemodynamic stresses, and movement play a critical role in tissues, since the cells use tensegrity architecture for their structuralorganization It can be concluded that orthodontic tooth movement is produced by mechanical means that evoke biological responses. These two entities, mechanics and biology, act in concert to produce desirable and predictable alterations in the form and function of the dentoalveolar complex. The actual performers of this force induced remodeling are the native cells of the treated teeth and their surrounding tissues. **[68-76]**

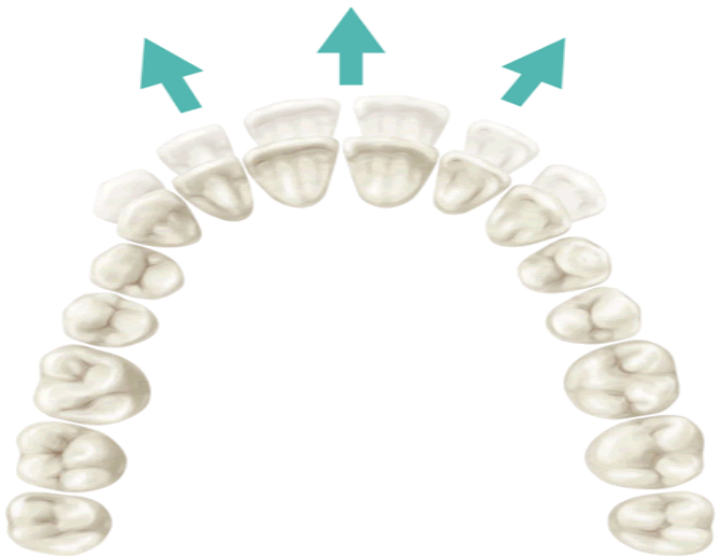

Figure 15: Proclination is tipping the crown of anterior teeth labially.

Other important cellular participants in this remodeling process are derivatives of the neuro vascular and immune systems. Cells and tissues use mechano sensing, transduction, and response phenomena to respond to applied mechanical forces. This reaction is typified by the aseptic inflammatory reaction, which is initially acute,becoming chronic a few days after the activation of the orthodontic appliance. Acute inflammation is re-introduced to the paradental tissues each time the appliance is reactivated. Hence, it may be suggested that mechano response and inflammation are both essential for achieving the clinical effect of tooth movement. If both mechanisms indeed unfold in concert, orthodontists might be able to accelerate the velocity of tooth movement by utilizing additional external stimuli, whether physical, chemical, or surgical . Knowledge pertaining to the subject of mechanics and biology in orthodontics is expanding constantly, creating an ever-stronger basis for success in the clinical realm. **[77-97]**

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Conclusion

From This review we conclude that:

The applied force from orthodontic appliance affect and change the homeostasis of the periodontium and alter the blood flow and this will activate enzymes and mediators that are responsible for the differentiation of osteoblast and Osteoclast on the pressure and tension side. Heavy force causes hyalinization of the tissues surrounding the tooth .Light force is better for Orthodontic tooth movement . The Optimal orthodontic force depends on the type of the tooth and on the patient condition

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