BIOLOGY OF TOOTH MOVEMENT

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INTRODUCTION

The essence of orthodontic treatment is the movement of teeth through bone to obtain a more perfect dental occlusion.

Orthodontic tooth movement has been defined by Proffit as the result of a biological response to an interference in the physiological equilibrium in the dentofacial complex by an externally applied force.

Accurate and precise control of tooth movement can be optimized with the proper use of mechanics and knowledge of the subsequent tissue response.

TOOTH SUPPORTING TISSUES

Orthodontic Treatment involves the use and control of forces acting on the teeth and associated structures. During tooth movement changes in the periodontium occur, depending on the magnitude, direction and duration of the force applied, as well as the age of the orthodontically treated patient.

The Periodontium (pert=around, odontos=tooth) comprises the following tissues:

-the *gingiva*, the *periodontal ligament (PDL)*,

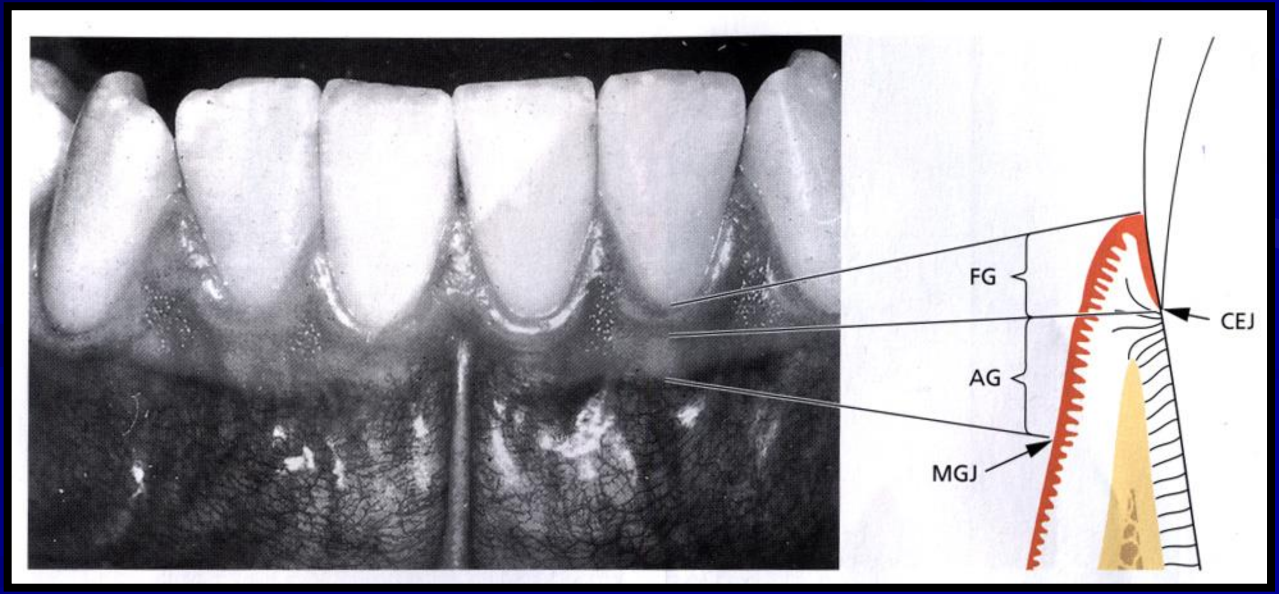
-the *root cementum*, and

-the *alveolar bone*.

Gingiva:

The gingiva is further differentiated into the *Free* and *Attached Gingiva*. In clinically healthy gingiva the *free gingiva* is in close contact with the enamel surface, and its margin is located 0.5 to 2mm coronal to the cementoenamel junction after completed tooth eruption. The *attached gingiva* is firmly attached to the underlying alveolar bone and cementum by connective tissue fibres and is therefore comparatively immobile.

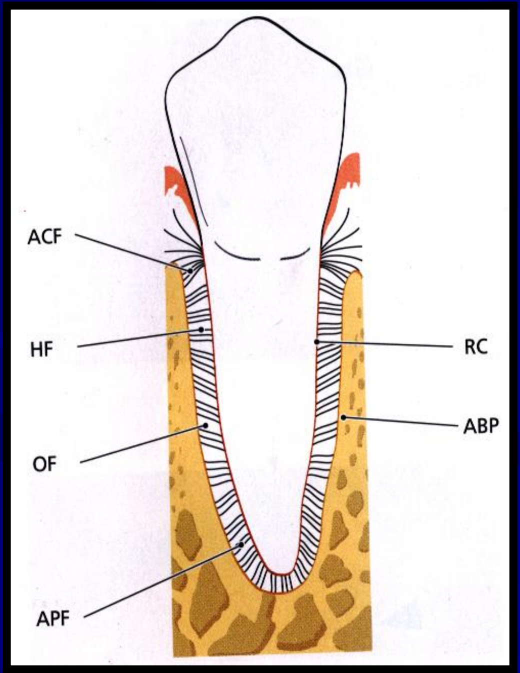
The predominant tissue component of the gingival is the Connective Tissue, which consists of Collagen Fibres (66%), Fibroblasts (5%) and Vessels, Nerves & Matrix (35%)

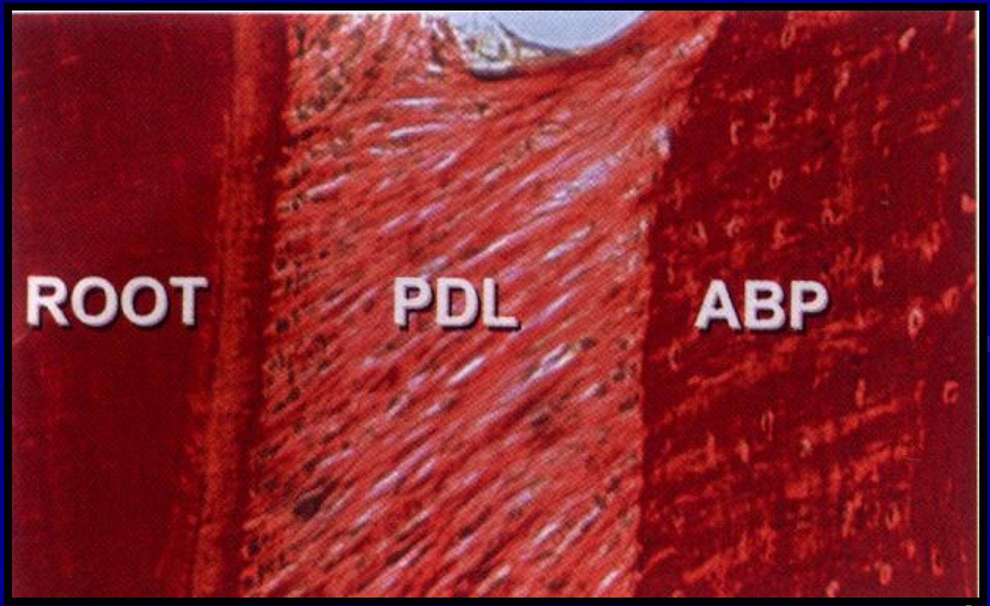


Periodontal Ligament:

The Periodontal Ligament (PDL), approximately 0.25mm wide, is the soft, richly vascular and cellular connective tissue that surrounds the roots of the teeth and joins the root cementum with the lamina dura or alveolar bone proper. The presence of a PDL makes it possible to distribute and resorb the forces elicited into the alveolar process through the alveolar bone proper.

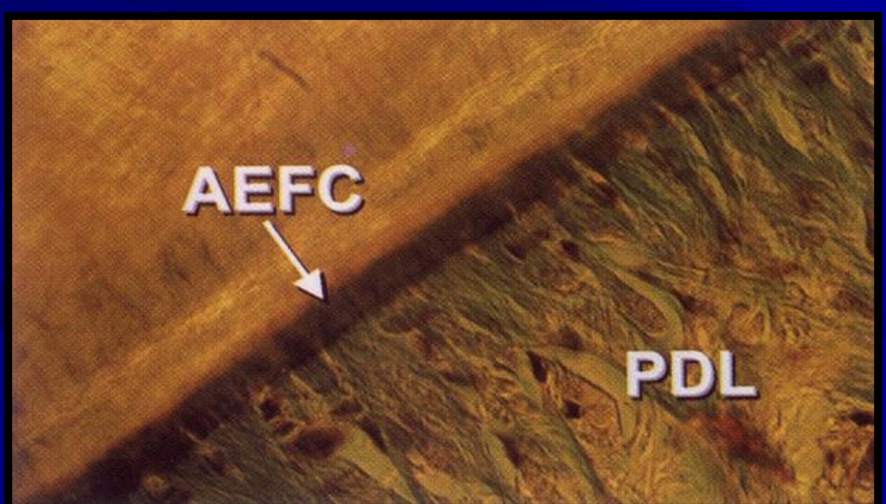
The true periodontal fibres, the *Principal Fibres*, develop in conjunction with the eruption of the tooth. When the tooth has reached contact in occlusion and is functioning properly, the principal fibres associate into the following well-oriented groups: *Alveolar Crest Fibres(ACF)*,*Horizontal Fibres(HF)*, *Oblique Fibres(OF)* and *Apical Fibres(APF)*. The fibrils of PDL are embedded in a ground substance which contains connective tissue polysaccharides (Glycosylaminoglycans), salts and water.

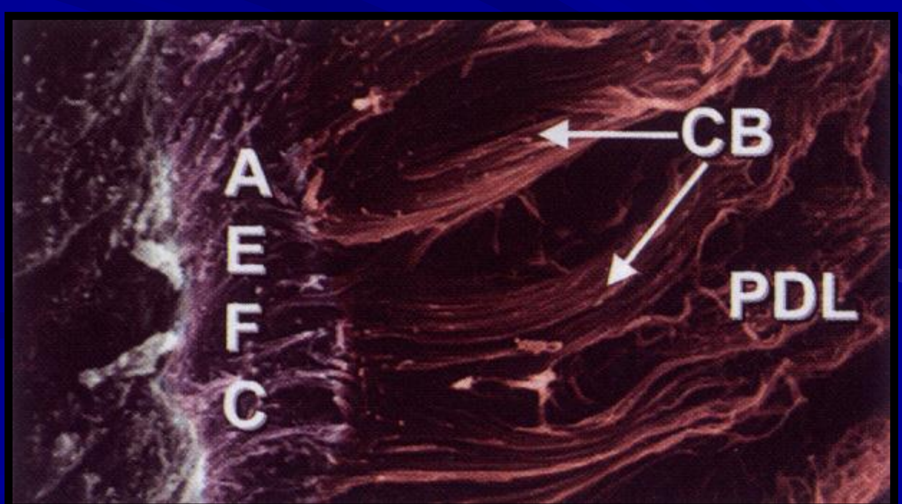




Root Cementum:

The root cementum is a specialized mineralized tissue covering the root surface. The cementum does not contain any blood vessels, has no innervations, does not undergo physiologic resorption or remodelling, but is characterized by continuing deposition throughout life. The cementum attaches the PDL fibres to the root and contributes to the process of repair after damage to the root surface (e.g., during orthodontic treatment). During root formation a primary cementum is formed. After tooth eruption and in response to functional demands, a secondary cementum is formed, which in contrast to the primary cementum contains cells.





(AEFC-acellular extrinsic fibre cementum) (CB-collagen bundles)

Alveolar Bone:

Alveolar bone surrounds the tooth to a level approximately 1mm apical to the CEJ. This part of the alveolar bone that covers the alveolus is referred to as *lamina dura*, a cortical bone. The alveolar bone is constantly renewed in response to functional demands with the help of bone-forming *Osteoblasts* and *Osteoclasts*. The Osteoblasts produce *Osteoid*, consisting of collagen fibres and a matrix that contain mainly proteoglycans and glycoproteins. The bone is covered with the *Periosteum*, which functions as an osteogenic zone throughout life.

The alveolar bone further consists of two components, the *alveolar bone proper* and the alveolar process.

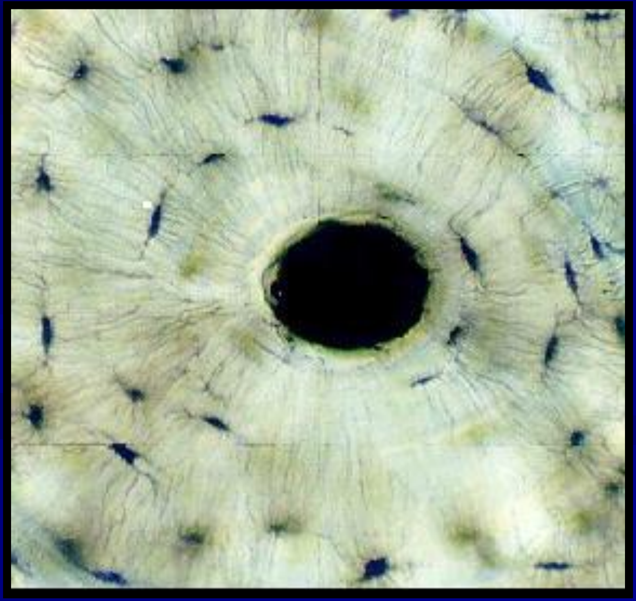
BONE TISSUE

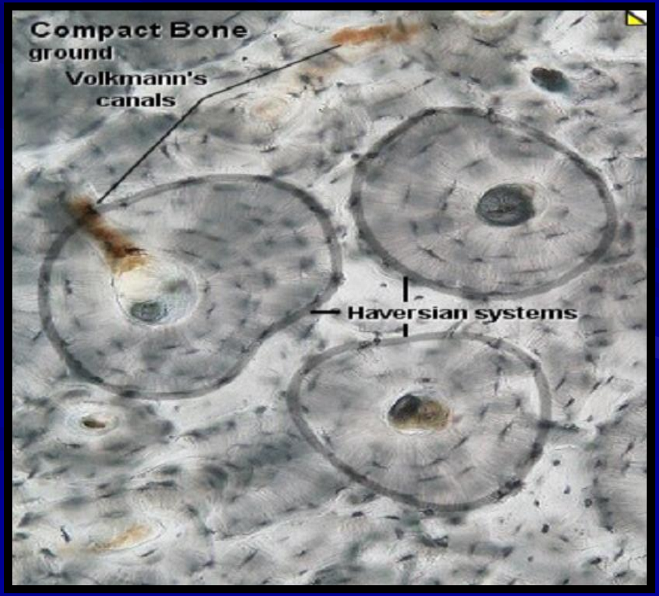
Bone is a specialized mineralized connective tissue made up of an organic matrix of collagen fibrils embedded in an amorphous substance with mineral crystals precipitated within the matrix.

The main functions of bone are two fold:

Function of Support &

Reservoir Function



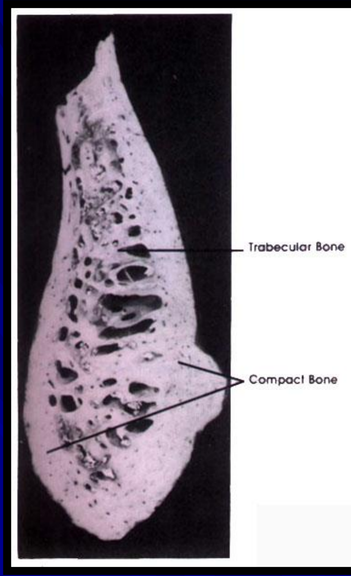


Classification:

>>Based on Structure.

1)*Compact Bone* or *Cortical Bone:* the dense outer shell of the skeleton.

2)*Cancellous Bone* or *Trabecular Bone* - comprises of a system of plates, rods, arches and struts traversing the medullary cavity encased within the shell of compact bone.



>>Based on the arrangement of collagenous matrix.

1)*Immature Bone:* This is further subdivided into:

Woven Bone: Relatively weak, disorganized and poorly mineralized. The first bone formed in response to orthodontic loading usually is the woven type.

Bundle Bone: is a functional adaptation of lamellar structure to allow attachment of *Sharpey's fibers*.

2)*Mature Bone :* This is further subdivided into:

Lamellar Bone: is a strong, highly organized, well-mineralized tissue. Adult human bone is almost entirely of this remodeled variety. The full strength of lamellar bone that supports an orthodontically moved tooth is not achieved until approximately 1 year after completion of active treatment.

Composite Bone: is an osseous tissue formed by the deposition of lamellar bone within a woven bone lattice. It is an important intermediary type of bone in the physiologic response to orthodontic loading.

BONE MODELING AND REMODELING

Wolff's Law as stated in 1892:

"Every change in the form and function of bone or of their function alone is followed by certain definite changes in their internal architecture, and equally definite alteration in their external conformation, in accordance with mathematical laws."

Both trabecular and cortical bone grow, adapt, and turn over by means of two fundamentally distinct mechanisms: *Modeling* and *Remodeling*.

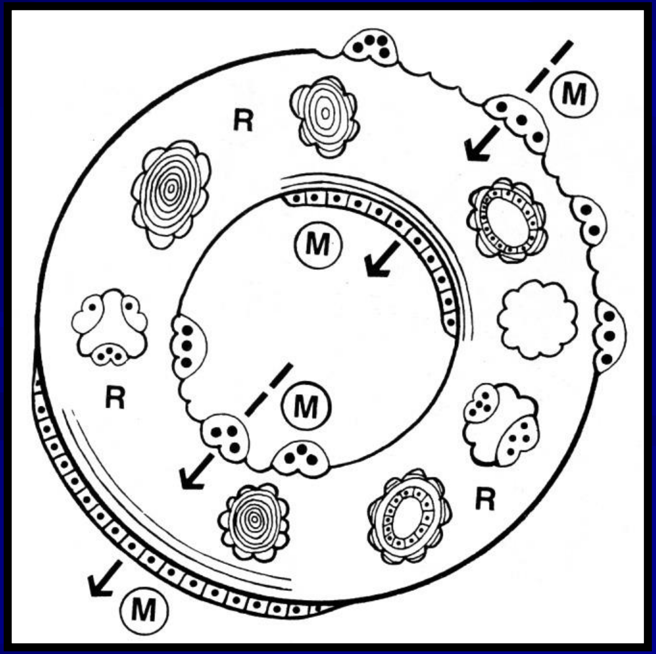
Because bone is a relatively rigid material, incapable of internal expansion or contraction, changes in osseous structure are via cell-mediated resorption and formation.

In *Bone modeling*, independent sites of resorption and formation change the form (i.e., shape or size or both) of a bone. In other words it is a process of uncoupled resorption and formation. In *bone remodeling* a specific, coupled sequence of resorption and formation occurs to replace previously existing bone. From an orthodontic perspective the biomechanical response to tooth movement involves an integrated array of bone modeling and remodeling events.

Bone modeling is the dominant process of facial growth and adaptations to applied loads such as head gear, RPE, and functional appliances. Modeling changes can be seen on cephalometric tracings, but remodeling events are apparent only at the microscopic level.

Constant *remodeling* mobilizes and redeposits calcium by means of 'coupled' resorption and formation: bone is resorbed and redeposited at the same site. Osteoblasts, Osteoclasts, and possibly their precursors are thought to communicate by chemical messages known as *Coupling Factors*. Transforming Growth Factor beta (TGF-beta) is thought to be a possible coupling factor.

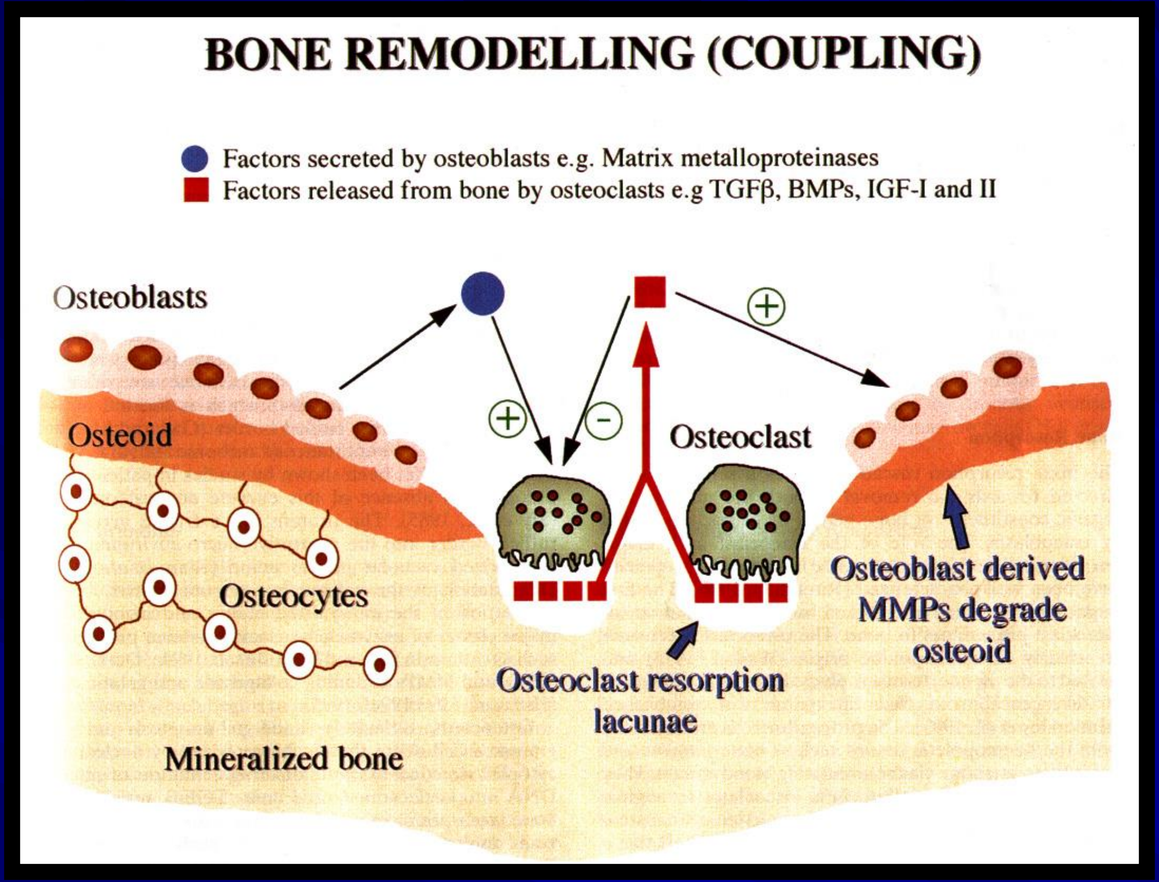
The remodeling process has evolved a vascularized multicellular unit for removing and replacing cortical bone which is called a *cutting/filling cone*. Remodeling of cortical bone will result in the formation of *secondary osteons*. These vascularized multicellular units of osteoclasts and osteoblasts are essential for metabolic, biomechanical, and postoperative remodeling. The entire coupled sequence to form a new secondary osteon requires about 6 months in man.



Schematic cross section through a bone showing the physiological relationship of bone modeling and remodeling.

-Modeling(M) changes the size or shape of a bone by forming or resorbing bone along periosteal and endosteal surfaces.

-Remodeling(R) is the internal bone turnover to form new secondary osteons.



Diagrammatic representation of coupling of osteoclastic bone resorption followed by osteoblastic bone formation. The initial event involves the synthesis and release of matrix metalloproteinases (MMPs) by osteoblasts which are responsible for degrading the osteoid, thus exposing the mineralized ,matrix which may be chemotactic to osteoclast. The osteoblast also directly stimulates osteoclast activity. During the resorption process growth factors are released from the matrix which then activate osteoprogenitor cells. The osteoprogenitor cells mature into osteoblasts and ultimately replace the resorbed bone. [The mechanism by which osteoclasts are directed to form bone only in the resorption lacunae may be due to the presence of molecules such as TGF-β and BMPs which are left behind during osteoclastic activity.]

TOOTH MOVEMENT

PATHOLOGIC TOOTH MOVEMENT

Carranza defined it as 'displacement that results when the balance among the factors that maintain physiological tooth position is disturbed by periodontal disease'.

It occurs most frequently in the anterior region, but posterior teeth may also be affected.

PHYSIOLOGIC TOOTH MOVEMENT

'The term physiologic tooth movement designates, primarily, the slight tipping of the functioning tooth in its socket and, secondarily, the changes in the tooth position that occur in young persons during and after tooth eruption.' Contrary to the relatively short eruption period, the teeth and their supporting tissues have a life-long ability to adapt to functional demands and hence drift through the alveolar process, a phenomenon called *physiologic tooth migration*. This physiologic drift is essential to maintain stomatognathic form and function.

ORTHODONTIC TOOTH MOVEMENT

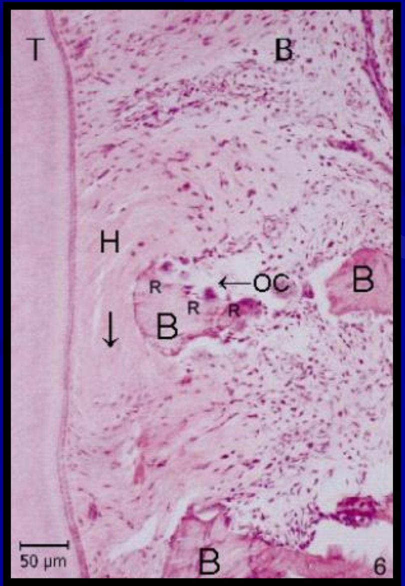
No great difference exists between the tissue reactions observed in physiologic and those observed in orthodontic tooth movement. However, since the teeth are moved more rapidly during treatment, the tissue changes elicited during orthodontic forces are consequently more marked and extensive.

Hyalinization: Hyalinization is a form of tissue degeneration characterized by formation of a clear, eosinophilic homogenous substance. A hyalinized zone is a local cell free area of overcompressed periodontal tissue. The conventional pathologic process of hyalinization is an irreversible one; however, hyalinization of the periodontal ligament is a reversible process. Hyalinization is caused partly by mechanical factors and is almost unavoidable in the initial period of tooth movement in clinical orthodontics.

The changes observed during formation of hyalinized zones can be summarized as follows:

- There is a gradual compression of the periodontal fibres leading to shrinkage and disappearance of cell nuclei and, subsequently, an exchange of degraded capillaries and fibrils as well.
- Osteoclasts are formed in marrow spaces and adjacent areas of the inner bone surface after a period of 20 to 30 hours.
- There is a gradual increase in the number of young connective tissue cells around the osteoclasts and in areas where the pressure is relieved by undermining bone resorption. This change in appearance before and after hyalinization is especially marked in the adult periodontal ligament. The general increase in cell number will facilitate bone resorption during the secondary stage of tooth movement.

A semihyalinized zone may occur due to lower local pressure with more viable tissue and a smaller risk of adjacent root resorption, or it may be a preliminary stage to full hyalinization.



Photomicrograph showing focal hyalinization of the PDL at the pressure side of a second premolar.
T-tooth, B-Bone, H-Hyalinization, OC-Osteoclast, R-undermining Resorption

Tissue Response in Periodontium:

The most dramatic remodelling changes incident to orthodontic tooth movement occur in the PDL. 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.

If the duration of movement is divided into an *initial* and a *secondary period*, direct bone resorption is found notably in the secondary period, when the hyalinized tissue has disappeared after undermining bone resorption.

During the crucial stage of *initial* application of force, the tissue reveals a glass like appearance in light microscopy, termed *hyalinization*. It represents a sterile necrotic area, generally limited to 1 or 2mm in diameter. The process displays three main stages: Degeneration, Elimination of destroyed tissue, and Establishment of a new tooth attachment.

In the *secondary period of tooth movement* the PDL is considerably widened. The osteoclasts will attack the bone surface over a much wider area and, provided the force is kept within certain limits, further bone resorption will be predominantly of the direct type. The fibrous attachment apparatus is somewhat reorganized by the production of new periodontal fibrils. These are attached to the root surface and parts of the alveolar bone wall where direct resorption is not occurring by the deposition of new tissue, in which the fibrils become embedded.

The main feature is the deposition of new bone on the alveolar surface from which the tooth is moving away. Cell proliferation is usually seen after 30 to 40 hours in young human beings. Shortly after cell proliferation has started, osteoid tissue is deposited on the tension side. The original periodontal fibres become embedded in the new layers of osteoid, which mineralizes in the deeper parts. New bone is deposited until the width of the membrane has returned to normal limits, and simultaneously fibrous system is remodelled. Concomitantly with bone apposition on the periodontal surface on the tension side, an accompanying resorption process occurs on the spongiosa surface of the alveolar bone. This tends to maintain the dimension of the supporting bone tissue.

PHASES OF TOOTH MOVEMENT

Classification of Three Phases of tooth movement by Burstone, Reitan, and Storey:

	FIRST PHASE (An initial period of Tooth Movement following application of Force)	SECOND PHASE (Slight movement or no movement)	THIRD PHASE (Tooth movement resumes at Slow or Rapid Rate)
Burstone, C.J. (1962)	Initial Phase	Lag Phase	Post-Lag Phase
Reitan, K. (1975)	Initial Period, First, or Pre-Hyalinization Stage	Initial Period, Plateau, or Hyalinization Stage	Secondary or Post-Hyalinization Period
Storey, E. (1973)	First Phase	Second Phase	Third Phase

Storey inferred from his animal studies and graphed analyses that,

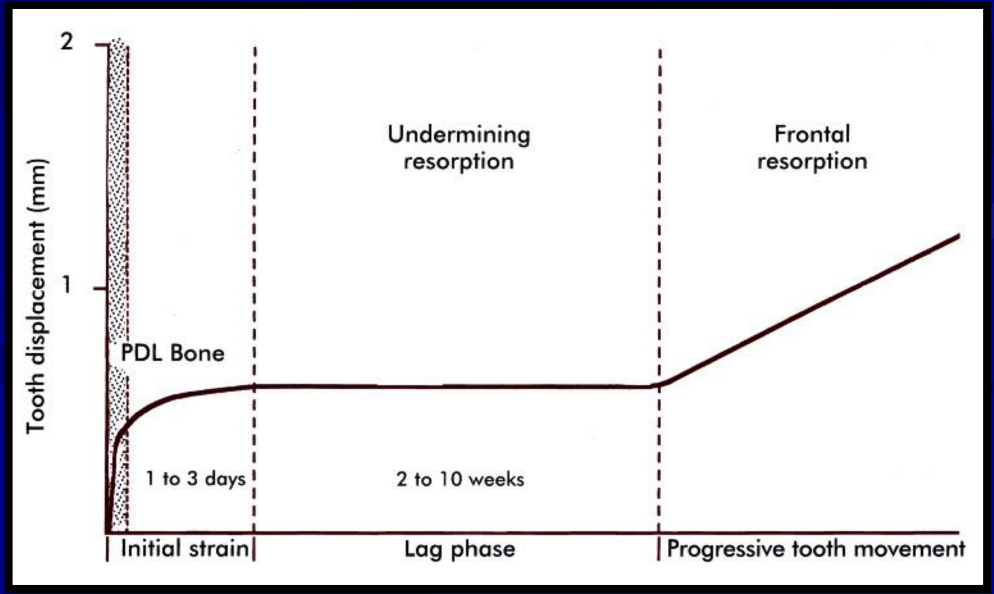
"In general, each curve has three phases: the first, where rapid movement takes place through the periodontal ligament space; the second, where movement occurs relatively slowly, or not at all, with the heaviest forces; and finally a stage where teeth begin to move rapidly……"

The Initial Phase: There is mechanical displacement of the tooth within the periodontal membrane space. This movement may be a crown tipping or a bodily movement, and is frequently a combination. The initial movement maybe regarded as including mechanical displacement following deformation of supporting bone. Tissue compression and bone deformation in this phase, which ordinarily lasts six to eight days, can result in rapid movement.

The Lag Phase: According to Reitan, this is the plateau or hyalinization stage in which little or no tooth movement occurs. It is characterized by cell free zones on the pressure side of the root and undermining resorption on the periodontal side of the alveolar wall. This stage usually lasts from one to three weeks.

The Post-Lag Phase: There occurs a mechanical displacement of the tooth associated with cellular activity of resorption and deposition. This may be any type of tooth movement and may be rapid or slow. It occurs spontaneously at the conclusion of the hyalinization period or lag phase without additional force input.

Interrupted Lag Phase: Reitan initially observed that little or no movement occurs during the hyalinization period or lag phase. But movement can occur following reactivation of spring force before undermining resorption has eliminated the hyalinized areas resulting from the previous activation. This might be termed an *interrupted lag phase*.



BIOLOGICAL CONTROL MECHANISMS IN TOOTH MOVEMENT

(What makes the tissue respond and what are the control elements involved in tooth movement ??)

Two mechanisms have been proposed:
The *pressure-tension theory* relates tooth movement to *biochemical-responses* by the cells and extracellular components of the PDL, and alveolar bone.
The *bio-electric theory* deals with tooth movement as a *bioelectric phenomenon* that may occur as a result of mechanical distortion of collagenous matrices, mineralized or nonmineralized, in the alveolar bone, the PDL, and the teeth.

-Pressure Tension theory: This classic hypotheses on the mechanism of tooth movement, based on the work of Oppenheim(1911), Sandstedt(1905), and Schwarz(1932), postulate the movement of the tooth within the periodontal space, generating a "pressure" side and a "tension" side. Schwarz hypothesized that the PDL space is a continuous hydrostatic system, and forces applied to this environment by means of mastication or orthodontic appliances create a hydrostatic pressure that would be, in accordance with Pascal's law, transmitted equally to all regions of the PDL. On the "pressure" side, cell replication is said to decrease as a result of vascular constriction, causing bone resorption. On the "tension" side, cell replication is said to increase because of the stimulation afforded by the stretching of the fibre bundles of the periodontal ligament (PDL), thus causing bone deposition. In terms of fibre content, the PDL on the "pressure" side is said to display disorganization and diminution of fibre production, while on the "tension" side, fibre production is said to be stimulated.

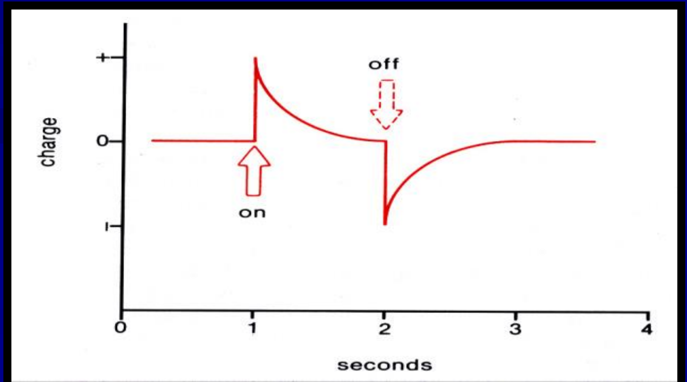
-The Bio-electric Theory: (Farrar 1876)

It has been shown that distortion of cells and extracellular matrix is associated with alteration in tissue and cellular electric potentials. Bones generally have a remarkable ability to remodel their structure in such a way that the stress is optimally resisted. It has been hypothesized that mechanical deformation of the crystalline structure of the hydroxyapetite and the crystalline structure of collagen induce migration of electrons that generate local electric fields. This phenomenon is called *piezoelectricity*.

*Such signals die away quickly even though the force is maintained.

* But when the force is released and the crystal lattice returns to the original shape, a reverse flow of electrons occurs. Rhythmic activity would cause a rhythmic flow of electrons in both directions.

Cells are sensitive to this piezoelctric effect. It has been assumed that bending of bone may create negative fields occurring in the concave aspect of the bone surface leading to deposition. Areas of convexity are associated with positive charges and evoke bone resorption. Further, ions in the fluids surrounding the living bone interact with these electrical fields. These currents of small voltages are called *streaming potentials*. Recent in vivo experiments conducted by Roberts et al (1981-JDR) have revealed that a negative electrical field is created in the areas where the PDL is widened.



-Fluid Dynamic Theory: (Bien 1966)

This theory is also called the Blood Flow Theory as proposed by Bien. According to this theory tooth movement occurs as a result of alterations in fluid dynamics in the PDL. The periodontal space contains a fluid system made up of interstitial fluid, cellular elements, blood vessels and viscous ground substance in addition to periodontal fibres. It is a confined space and passage of fluid in and out of this space is limited. The contents of the PDL thus create a unique hydrodynamic condition resembling a hydraulic mechanism and a shock absorber. When the force is removed, the fluid is replenished by diffusion from capillary walls and recirculation of interstitial fluid. But when a force of greater magnitude and duration is applied such as during orthodontic tooth movement, the interstitial fluid in the periodontal space gets squeezed out and moves towards the apex and cervical margins and results in decreased tooth movement. This is called the "*squeeze film effect*" as proposed by Bien.

When an orthodontic force is applied, it results in compression of the PDL. Blood vessels of the PDL gets trapped between the principal fibres and this results in their stenosis. Bien suggested that there is an alteration in the chemical environment at the site of vascular stenosis due to a decreased oxygen level in the compressed areas as compared to the tension side. The formation of these aneurysms and vascular stenosis causes blood gases to escape into the interstitial fluid thereby creating a favourable local environment for resorption.

GENETIC CONTROL MECHANISMS

Several Genes, linked to mechanical activation of bone, produce enzymes such as *glutamate/aspartate transporter (GLAST), inducible nitric oxide synthetase (iNOS)* and *prostaglandin G/H synthetase (PGHS-2)*. Inducible gene products compose an intricate series of edocrine, paracrine, and autocrine mechanisms for controlling bone modeling.

-Parathyroid Hormone (PTH) and PTH-related protein (PTHrP) enhance expression of insulin-like growth factor I (IGF-I).

-In situ hybridization under conditions of physiologic tooth movement in rats demonstrated site-specific expression of mRNAs for *osteonectin(Osn), osteocalcin (Ocn), and osteopontin (Opn)* (JHC-1994). In response to orthodontic force, Opn mRNA is elevated within the tissue by 12hrs and can be demonstrated at 48hrs by in situ hybridization in >50% of osteoclasts and >87% of osteocytes in the interdental septum of maxillary molars (JBMR-1999).

-*Msx1* is a regulator of bone formation during development and postnatal growth. It is involved in the control of neural crest cell migration but also appears to be important for bone modeling activity.

-Osteoclast differentiation and activation is controlled by a group of genes related to tumour necrosis factor (TNF) and its receptor (TNFR). Genes involved are *osteoprotegerin (OPG), receptor activator of nuclear factor (RANK), and RANK ligand (RANKL)*.

[Colony stimulating factor 1 (CSF1) and RANK induce differentiation of haematopoietic precursor cells, which results in osteoclast precursors with RANK receptors. Local bone related cells secrete RANKL, which binds with RANK on the preosteoclast cell surface to induce the development of a functional osteoclast. As a feedback control, the same regulatory cells produce OPG, which blocks the RANK receptor and thus downregulates osteoclasts.]

-In addition to the well established 'RANK-RANKL-OPG axis', another gene (*TREM-2)* has been implicated in control of bone modeling.

-Another gene, the P2X7 receptor, has been reported to play an important role for initiating and sustaining all types of anabolic bone modeling.

BIOCHEMICAL REACTIONS TO ORTHODONTIC TOOTHMOVEMENT

ORTHODONTIC FORCE

↓

BIO-PHYSICAL REACTIONS

Bone deformation
Compression of PDL
Tissue Injury

↓

PRODUCTION OF FIRST MESSENGERS

Hormones (e.g.. PTH)
Prostaglandins
Neurotransmitters

↓

PRODUCTION OF SECOND MESSENGERS

C amp, C gmp, Ca^{++}

↓

Increase in cells of Resorption (osteoclasts)
Increase in cells of Deposition (osteoblasts)

↓

Bone Remodelling

↓

ORTHODONTIC TOOTH MOVEMENT

Inflammation due to tissue injury

Activation of Collagenase

ORTHODONTIC FORCES

Orthodontic forces comprises those that are brought to bear on the PDL and alveolar process, whereas *orthopaedic forces* are more powerful and act on the basal parts of the jaws. The decisive variables regarding these forces at the cellular level are application, magnitude, duration, and direction of force.

Types of Forces:

Two different types of force exist: *Continuous (fixed appliances)* and *Intermittent (removable appliances)*

-Continuous Forces: Modern fixed appliance systems are based on light continuous forces from the arch wire. However, a continuous force may be *interrupted* after a limited period (*interrupted continuous force*). e.g. the movement that occurs when a tooth is ligated to a labial arch, the tooth being held in position after the force is no longer acting ; torquing movement performed by an edgewise archwire. Although the typical continuous force acts for longer periods, the interrupted force is of comparatively short duration (3 to 4 weeks on average).

[*In clinical orthodontics an interrupted tooth movement may have certain advantages. Because of the increase in the number of cells, osteiod tissue is deposited in open marrow spaces on the pressure side and in other areas not undergoing direct resorption. On the tension side a gradual calcification and reorganization of newly formed tissue occurs during the rest period. Hence the tissues are given ample time for reorganization and the cell proliferation is favourable for further tissue changes when the appliance is again activated.*]

***Intermittent Forces*:** Such a force act during a short period and is induced primarily by removable appliances, especially functional appliances. This also applies to springs resting on the tooth surface that produces impulses and stimuli of short duration as the appliance moves during speech and swallowing. The intermittent action may then to a varying extent result in less compression on the pressure side and shorter hyalinization periods, unlike elicited by continuous forces. Experiments have shown that movement effected by an intermittent force depends on the length of time of application and on the magnitude of force. The disadvantage of intermittent tooth movement is the mode of displacement, which always occurs in the form of tipping.

MAGNITUDE AND DURATION OF FORCES

It is generally considered that a light force over a certain distance moves a tooth more rapidly and with less injury to the supporting tissues than a heavy one. A light or heavy force depends on the mode of application and the mechanical arrangement of the recipient tooth units. Experimental studies have shown that heavy, continuous loads results in a vertical reduction of the height of the approximal alveolar bone.

The purpose of applying a light force is to increase cellular activity without causing undue tissue compression and to prepare the tissues for further changes. Generally, the magnitude of force determines the duration of hyalinization. This is shorter within the light force level. Another reason is that it results in less discomfort and pain to the patient.

The duration of force, equivalent to treatment time, is often considered to be a more crucial factor than the magnitude of the force with regard to adverse tissue reactions, especially in connection with long treatment periods and in cases with high density of alveolar bone.

Time		
Light pressure	Heavy pressure	Event
< 1 sec		PDL* fluid incompressible, alveolar bone bends, piezoelectric signal generated
1-2 sec		PDL fluid expressed, tooth moves within PDL space
3-5 sec		Blood vessels within PDL partially compressed on pressure side, dilated on tension side; PDL fibers and cells mechanically distorted
Minutes		Blood flow altered, oxygen tension begins to change; prostaglandins and cytokines released
Hours		Metabolic changes occurring: chemical messengers affect cellular activity, enzyme levels change
~4 hours		Increased cAMP levels detectable, cellular differentiation begins within PDL
~2 days		Tooth movement beginning as osteoclasts/osteoblasts remodel bony socket
	3-5 sec	Blood vessels within PDL occlused on pressure side
	Minutes	Blood flow cut off to compressed PDL area
	Hours	Cell death in compressed area
	3-5 days	Cell differentiation in adjacent narrow spaces, undermining resorption begins
	7-14 days	Undermining resorption removes lamina dura adjacent to compressed PDL, tooth movement occurs

Physiological response to a sustained pressure against a Tooth

Time course of events after application of orthodontic force:

Heavy vs. Light Force.

TYPES OF TOOTH MOVEMENT and TISSUE REACTIONS

(in supporting tissues)

Initially only minor tooth movements occurs within the periodontal space. The larger long-term tooth movements are the result of such minor movements, depending on the pattern of socket remodelling.

>>Tipping

This simplest form of orthodontic tooth movement is produced when a single force is applied against the crown of a tooth. When this is done, the tooth rotates around its Centre of Resistance, a point located about halfway down the root.

>With light continuous forces, tipping results in a greater movement within a shorter time than that obtained by any other method.

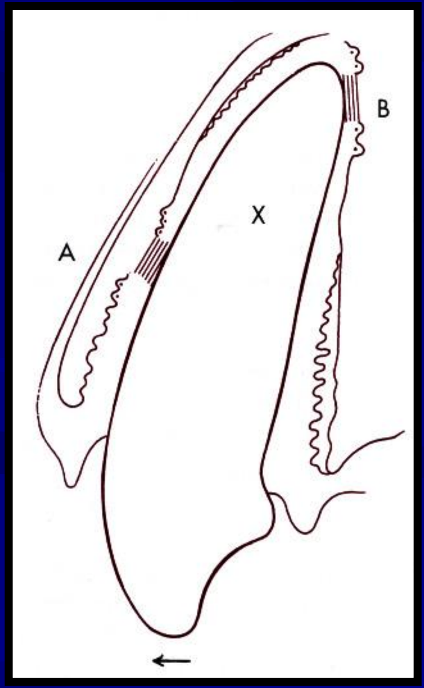
>Prolonged tipping may result in apical root resorption even if the force is light.

>The PDL is compressed near the root apex on one side and at the crest of the alveolar bone on the opposite side. Thus maximum pressure in the PDL is created at the alveolar crest and at the root apex.

>It results in the formation of a hyalinized zone slightly below the alveolar crest (when the tooth has a short undeveloped root) or at a short distance from the alveolar crest (when the root is fully developed).

>In young patients, bone resorption resulting from a moderate tipping movement is usually followed by compensatory bone formation. The degree of such compensation depends primarily on the presence of bone-forming osteoblasts in the periosteum.

>Tipping of adult teeth in a labial direction may result in bone destruction of the alveolar crest, with little compensatory bone formation.



A = Secondary Hyalinized Zone

B = Compressed PDL

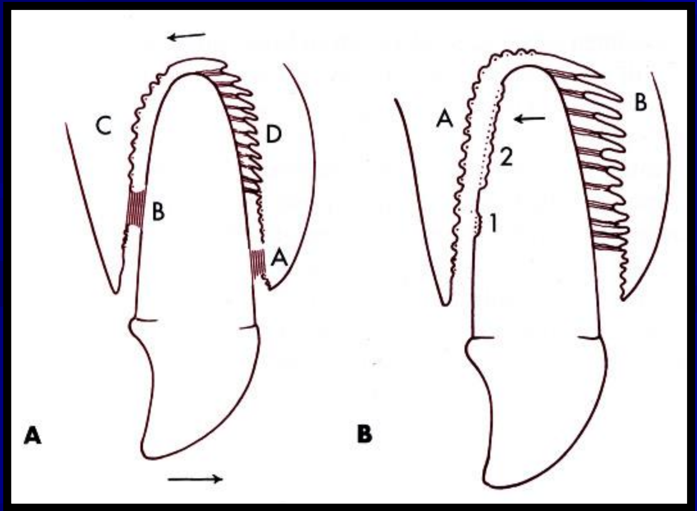
X = Fulcrum

>>Torque

>During the initial movement of torque, the pressure area is located close to the middle region of the root. This occurs because the PDL is normally wider in the apical third than in the middle third.

>After resorption of bone areas corresponding to the middle third, the apical surface of the root gradually begins to compress adjacent periodontal fibres and a wider pressure area is established.

>Experimental studies by Reitan and Kvam(1971-AO) have shown that 50gms of force was sufficient to cause root movement without any undermining resorption.



>>Bodily Movement

Bodily tooth movement is obtained by establishing a couple of forces acting along parallel lines and distributing the force over the whole alveolar bone surface. This is a favourable method of displacement provided the magnitude of force does not exceed a certain limit.

>It is characteristic of the initial bodily movement that the hyalinization periods are shorter than in tipping movements.

>Hyalinization occurs largely as a result of mechanical factors. Shortly after the movement is initiated there is compression on the pressure side with formation of a hyalinized zone between the marginal and middle regions of the root.

>The short duration of hyalinization results from an increased bone resorption on both sides of the hyalinized tissue, especially in the apical region of the pressure side.

>The PDL on the pressure side is considerably widened by the resorption process.

>There is gradually increased stretching of the fiber bundles on the tension side, which tends to prevent the tooth from further tipping. New bone layers are formed on the tension side along these fiber bundles.

>>Rotation

Pure Rotation of a tooth requires a couple. No net force acts at the CRes, so only rotation occurs. Clinically this movement is most commonly needed for movement as viewed from the occlusal perspective.

>In rotation of a tooth around its long axis the force can be distributed over the entire PDL rather than over a narrow vertical strip, whereas larger forces can be applied than in other tooth movements.

>Histologically, the tissue transformation that occurs during the rotation is largely influenced by the anatomic arrangement of the supporting structures.

>After rotation of the tooth, the stretch of the free gingival tissue may cause displacement of collagen, elastic, and oxytalan fibres located even some distance from the tooth being moved.

>Most teeth to be rotated create two pressure sides and two tension sides.

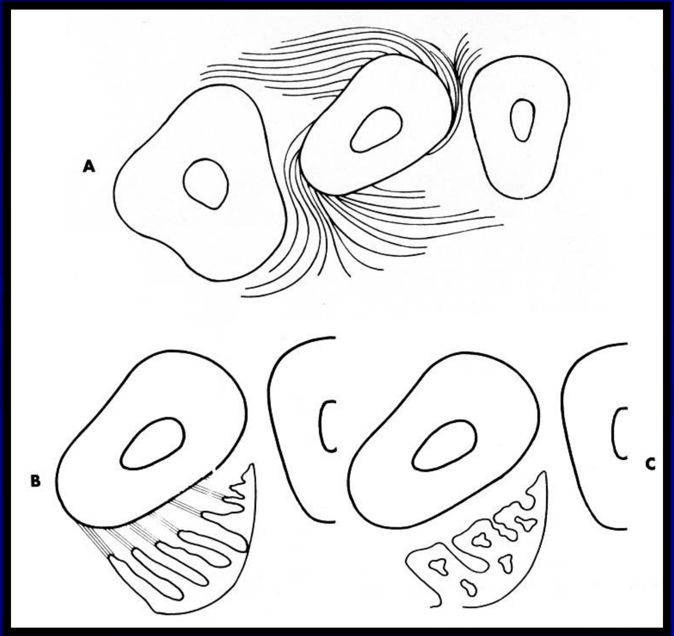
>Occasionally, hyalinization and undermining bone resorption takes place in one pressure zone while direct bone resorption occurs in the other.

>After rotation for 3 to 4 weeks, the undermining resorption is usually completed and direct bone resorption prevails on the pressure side.

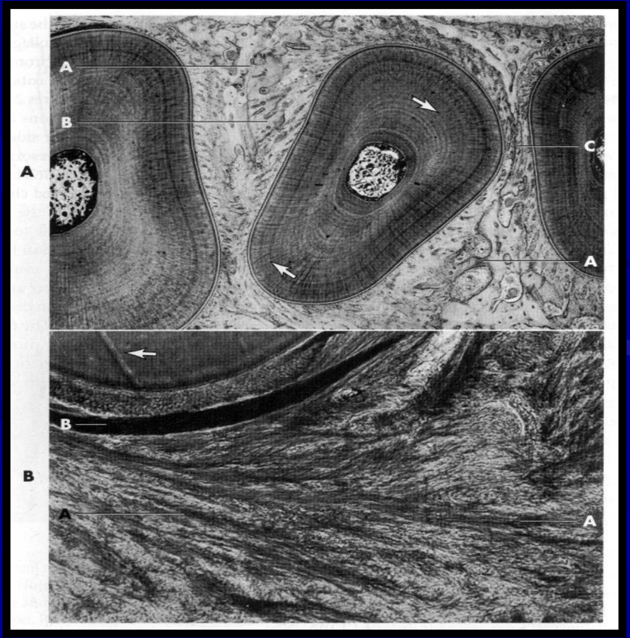
>On the tension side of the middle third, new bone spicules are formed along stretched fibre bundles arranged more or less obliquely.

>Furthermore, the periodontal space is considerably widened by bone resorption after rotation.

>The fiber bundles and the new bone layers of the middle and apical thirds rearrange themselves after a fairly short retention period (Reitan K. AO-1959). However, the free gingival fibres remain stretched and displaced for as long as 232 days and possibly longer. [Therefore overrotation has been recommended.]



Arrangement of gingival fibers and new bone layers formed on the tension side, after rotation.



>>Extrusion (Bodily displacement of a tooth along its long axis in an apical direction)

>Extrusive movements ideally does not produce any areas of compression within the PDL, but only tension.

>Varying with the individual tissue reaction, the periodontal fibre bundles elongate and new bone is deposited in areas of alveolar crest as a result of the tension exerted by these stretched fibre bundles.

>In young individuals, extrusion of a tooth involves a more prolonged stretch and displacement of the supraalveolar fibre bundles than of the principal fibres of the middle and apical thirds. They will be rearranged after a fairly short retention period.

>In adult patients the fibre bundles also are stretched during extrusion, but they are less readily elongated and rearranged after treatment.

>The force exerted must not exceed 25 to 30cN because extrusion constitutes the type of tooth movement that requires minimal force.

>>Intrusion (Bodily displacement of a tooth along its long axis in an occlusal direction)

>Light force is required because the force is concentrated in a small area at the tooth apex. Primarily the anterior teeth are intruded.

>Stretch is exerted primarily on the principal fibres.

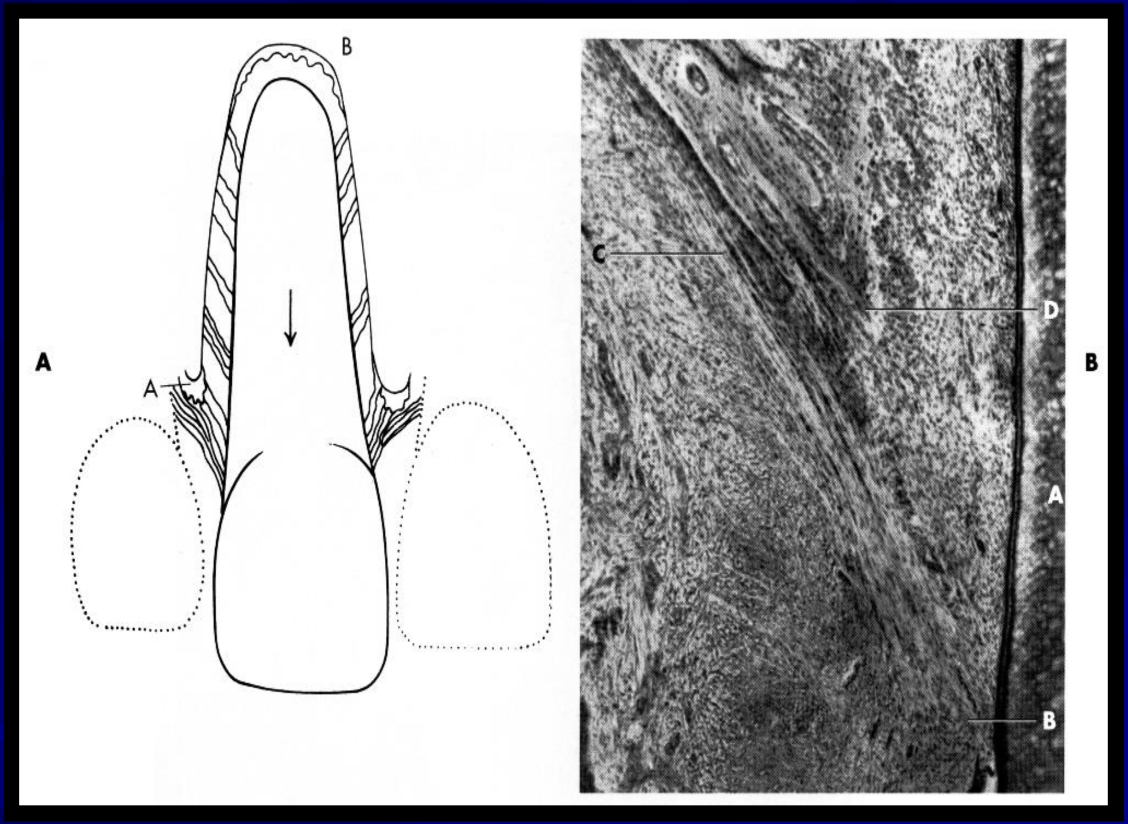
>An intruding movement may therefore cause formation of new bone spicules in the marginal region. These new bone layers occasionally become slightly curved as a result of the tension exerted by stretched fibre bundles.

>Rearrangement of the principal fibres occurs after a retention period of 2 to 3 months.

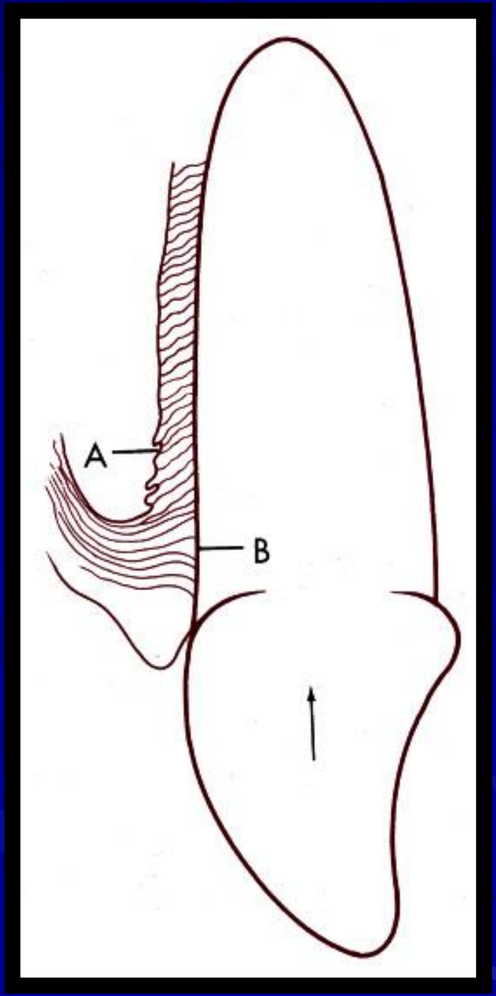
>Unlike extruded teeth, intruded teeth in young patients undergo only minor positional changes after treatment.

>Relapse usually does not occur, partly because the free gingival fibre bundles become slightly relaxed.

>In adults, however, relapse after intrusion may occur, particularly when the retention period has been too short.



Arrangement of fiber bundles during or after extrusion of U1.



Relaxation of Free Gingival fibers during intrusion.

TOOTH MOVEMENT by EXTRA-ORAL FORCES

Generally extra-oral forces can be divided into two categories.

>In the first group strong extra-oral forces can be applied for early control of facial bone growth – *Orthopaedic Forces*. Not only tooth position but also the direction of bone growth is influenced during treatment.

>The second type of extra-oral treatment consists of movement of individual teeth. Hence it is impractical to apply forces that are too strong. A change in the direction of force applied may alter and modify the reaction of the tissues involved. The mechanics involved in an extra-oral force treatment were duplicated in animal experiments (Rietan K. AO-1964). Since the interseptal bone in the dog is predominantly spongy and therefore similar to interseptal areas of human alveolar structures, it is suggested that these experiments might illustrate fairly well what happens in tooth movement performed with extra-oral forces.

....

[Strong forces of about 400gms were applied with elastics placed between hooks on the upper canine and second incisor bands in the dog.

Direct bone resorption was observed on the distal side of the third incisor whereas semihyalinization occurred on the pressure side of the tooth moved. Semihyalinization implies that osteoclasts are formed subjacent to the hyalinized fibres. The reason behind this would be that interseptal bone contains a system of marrow spaces whereby osteoclasts are formed in many areas. The additional thickness of the band material and the fact that the tooth was moved remained in firm contact with the proximal tooth also tend to prevent complete hyalinization. A variation is also caused by the tissue characteristics of various experimental animals. Joho in his studies of the monkey (AJO-1973) observed that extra-oral forces acting continuously may cause appreciable root resorption between the middle and apical thirds of the root, but no definite shortening of the apical portion is evident. This lack of apical shortening is usually observed in most cases of extra-oral tooth movement.]

FACTORS INFLUENCING TOOTH MOVEMENT

ANALGESICS

>Acetominophen is the preferred 'over-the-counter' medication for orthodontic patients because it acts on the CNS and does not interfere with localized inflammatory processes. However, numerous studies have demonstrated that >NSAIDs are superior to acetominophen and aspirin for relief of orthodontic pain. NSAIDs are effective orthodontic analgesics, but they may reduce the rate of tooth movement, and they should not be administered for long periods of time to orthodontic patients. Strain-induced catabolic modelling(bone resorption) at bone/PDL interface limits the rate of tooth movement. However, short-term treatment (≤3days), particularly if the initial dose is administered before applying force, is a very effective orthodontic analgesic, and it is unlikely to significantly increase treatment.

INFLAMMATORY AGENTS

Focus also has been on the effects of *Prostaglandins*, *IL-1β*, and *Leukotrines* relative to orthodontic tooth movement.

>*Inflammatory Cytokines* have been administered to enhance orthodontically induced bone modelling. Similar effects have been demonstrated with >*Prostaglandin E2 (PGE2)* administration to primates, and the results have been confirmed clinically (AJO-1984). Most prostaglandin studies have demonstrated an increased risk of root resorption that is proportional to the increase in the rate of tooth movement.

>*Misoprostol*, a prostaglandin E1 analog, has been used to enhance orthodontic tooth movement in rats (AJODO-2002). At a dose of 10μg/day for 14 days, oral misoprostol increased the amount of orthodontic tooth movement in all the experimental groups compared with the appliance control group. These results indicate that oral Misoprostol can be used to enhance the rate of tooth movement with less risk of increased root resorption than PGE2.

>*Prostaglandin E2*, with or without simultaneous administration of Calcium Gluconate (Ca), was tested over a 21-day period of experimental tooth movement in rats (EJO-2003). An acceleration in tooth movement was noted after PGE2 injection. The addition of Ca moderated the increase in the rate of tooth movement due to PGE2, but most importantly, the increase in root resorption, observed in the PGE2 only group, was negated by simultaneously administering Ca. It was concluded that Ca ions stabilize teeth against root resorption when the rate of tooth movement is enhanced by PGE2.

>Recent studies observed that with both light continuous force and interrupted force for a duration of 24hrs, there was a significant elevation in both *IL-1β* and *PGE2* levels (AJODO-Feb.2004)

>The effects of local administrations of PGE2 and 1,25-dihydroxycholecalciferol (1,25-DHCC) on orthodontic tooth movement was compared in a recent study (AJODO-May2004), and 1,25-DHCC was found to be more effective in modulating bone turnove during orthodontic tooth movement.

SURGICAL ENHANCEMENT

Orthodontists have long noted increased rates of tooth movement following orthognathic surgical procedures; this effect is usually attributed to a post operative acceleration of bone remodelling. >*Maxillary Corticotomy* is now a routine procedure for surgically assisted rapid palatal expansion. However, *Alveolar Corticotomy* to enhance the rate of toot movement has developed more slowly, largely because of concern about periodontal outcomes.

>Wilcko et al (IJPRD-2001) introduced a new surgical procedure that involves buccal and lingual full-thickness flaps, selective partial decortication of the alveolar cortex, concomitant bone grafting/augmentation, and primary flap closure. Two cases of Class I malocclusion with relatively narrow arches were reported. In both instead of en bloc movement of bony segments, the rapid expansion of arches to correct crowding was attributed to the postoperative Regional Acceleratory Phenomenon (RAP), originally described by Frost.

>In periodontally compromised patients, surgical augmentation , immediately before intrusion and alignment of incisors compromised by periodontitis, resulted in increased osseous support.

>Recent studies have concluded that a surgical augmentation immediately before orthodontics may offer advantages for arch expansion in a healthy dentition, for alveolar cleft management, or to increase osseous support for periodontially compromised teeth (Dent.Update1999).

DISTRACTION OSTEOGENESIS

>Distraction Osteogenesis is a method for generating new bone by progressively distracting healing surfaces, following the complete osteotomy of a bone. Essentially it is a bone modelling procedure that produces perivascular woven bone, which then condenses and remodels to mature lamellar bone.

The method is currently being developed for orthodontic applications such as cuspid retraction,
molar intrusion,
segmental translation,
recovery of ankylosed teeth, and
interdental expansion.

NITRIC OXIDE

Localized *Nitric Oxide (NO)* production is a known mediator of osteoclastic induction in an inflammatory environment, that is, in the presence of cytokines such as IL-1β, IL-6, and TNFα. Cuzzocrea et al, provided evidence that *inducible nitric oxide synthetase (iNOS)*, a receptor that controls NO production, also mediates bone loss systemically in estrogen-deficient mice (Endocrinology 2003). Estrogen exhibits anti-inflammatory activity by preventing the induction of iNOS and other inflammatory components. Recent studies have suggested that NO is an important biochemical mediator in the response of periodontal tissue to orthodontic force (AJODO-2002).

SYSTEMIC DISEASES

>*Rheumatoid Arthritis-* It is a relatively common disease in prospective orthodontic patients. The relatively high doses of corticosteroids used to treat these patients can inhibit bone growth and the rate of tooth movement.

>*Cystic Fibrosis(CF)-* It is often associated with low bone mineral density. Therefore bone formation rate at tissue level is significantly lower.

>*Osteomalacia-*(excessive unmineralized osteoid) is a complicating factor when there is concomitant VitD deficiency. In the absence of Osteomalacaia, CF patients are viable candidates for orthodontic treatment.

>*Primary Hyperparathyroidism (PHP)-*is a high turnover metabolic bone disease. Surgical treatment of severe cases results in dramatic improvement in bone metabolic parameters.

Other systemic diseases include:- *Parathyroid Carcinoma, Hyperparathyroidism Jaw tumour Syndrome (HPT-JT syndrome), Kidney disorders, Diabetes, Osteopenia and Osteoporosis, Osteogenesis Imperfecta etc.*

IATROGENIC RESPONSE OF SUPPORTING TISSUES IN ORTHODONTICS

Various Clinical, Radiological and Histological investigations have been conducted from time to time to assess the damage to root substance and supporting tissues.

Damage to Periodontal Tissues

>**Gingival Inflammation:** The initial and most important factor causing gingival inflammation is bacterial plaque at the gingival margin. Patients with fixed appliances have increased retention sites for microbial samples and therefore significantly higher total numbers of Strep. Mutans and Lactobacilli.

A greater plaque index; tendency for bleeding; increased pocket depth and greater interproximal loss of attachment have been observed more frequently for molars with orthodontic bands.

>**Alveolar Bone Loss:** Compressed gingiva in the extraction sites (between teeth that have moved together) can produce a long-lasting epithelial fold, or invagination. The surrounding connective tissue exhibits loss of collagen. The mechanical forces employed can cause sublethal damage and stimulate a hyperplastic tendency in the tissue components. It has been shown that orthodontic treatment may in fact aggravate a pre-existing plaque induced gingival lesion and cause loss of alveolar bone and periodontal attachment.

Experimental studies in the *beagle* also have shown that it is possible for orthodontic tipping forces to shift a supragingivally located plaque into a subgingival position, resulting in the formation of infrabony pockets. (JCP 1977)

>**Marginal Bone Recession:** It is the displacement of the soft tissue margin, apical to the CEJ, with subsequent exposure of the root surface. This is associated with localized plaque induced inflammatory lesions and sometimes in combination with orthodontic therapy.

Alterations occurring the gingival dimensions and marginal tissue position in conjunction with orthodontic therapy are related to the direction of tooth movement. Labial and Buccal movements results in reduced facial gingival dimensions, whereas an increase is observed after lingual movement.

[The presence of an alveolar bone dehiscence is considered to be a prerequisite for the development of marginal recession. With respect to orthodontic treatment, this implies that as long as tooth is moved exclusively within the envelope of the alveolar process, the risk of harmful side effects in the marginal tissue is minimal, irrespective of the dimensions and quality of the soft tissue (JCP 1981,87).]

>**Pulpal reaction:** Although Pulpal reactions to orthodontic treatment are minimal there is a modest transient *inflammatory response* within the pulp, at least at the beginning of the treatment. This may contribute to the discomfort that patients often experience for a few days after appliances are activated, but the mild pulpitis has no long term significance.

As demonstrated by Stenvik and Mjör (AJO-1970), vacuolization of the odontoblast layer constitutes the most characteristic tissue alteration.

Devitalization may occur when the pulp structures have become degraded due to the teeth being subjected to trauma or severe pressure before treatment period.

Since the response of the PDL, not the Pulp, is the key element in orthodontic tooth movement, moving endodontically treated teeth is feasible.

Some evidence has indicated that endodontically treated teeth are more prone to root resorption during orthodontics than are teeth with normal vitality. But recent studies suggest that this is not so (Spurrier et al. AJO-1990).

>**Root resorption:** The first comprehensive study on root resorption after orthodontic treatment was conducted by Ketcham (IJO-1929). Since then extensive research has elucidated the mechanism of external root resorption.

Two types of root resorption may occur in connection with orthodontic treatment:

Superficial Resorption, that undergo repair and resorption in the apical area, which may lead to permanent root shortening.

As with osteoid, cementoid tends to decrease in thickness on the side of compression. If the pressure is continuous for a long period, root resorption may start even if the root was initially protected by uncalcified tissue. Root resorption that occurs during orthodontic treatment is frequently preceded by hyalinization of the PDL. During the remodelling process of the hyalinized zone the necrotic hyalinized tissue and alveolar bone wall are removed by phagocytic cells such as macrophages, foreign body giant cells, and osteoclasts. As a side effect of the cellular activity during the removal of the necrotic PDL tissue, the cementoid layer of the root and the bone are left with raw unprotected surfaces in certain 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.

Apical Resorption: Experiments have revealed tat anatomic environment constitutes an important factor during tipping movement and intrusion. If the root surface is well calcified and the predentin layer is thin, tipping movement may lead to resorption of the outer side of the apical portion as well as along the inside of the root canal. The apical side resorption is preceded by a short hyalinization period. The anatomic environment and duration and direction of movement constitute the determining factors in apical root resorption.

Factors affecting Root Resorption:

Individual tooth vulnerability, Endodontically treated teeth, Age, Orthodontic appliances, Magnitude of Force, Duration of Force and Direction of Tooth Movement: Intrusion and Torque are probably the most detrimental to the tooth involved. Experiments in monkeys and dogs have shown that alveolar bone dehiscences maybe induced by uncontrolled *labial movement* of incisors through the cortical bone plate (EJO- 1983). Tooth movements in such a direction also initiate root resorption. Using *rapid palatal expansion techniques,* premolars and molars are pressed in a buccal direction against the thin cortical plate with risk of similar damage.

POSTTREATMENT STABILITY

Not all orthodontically achieved changes remain stable, although the question of *relapse* is related to the objective of treatment. *Retention* is designed to maintain the occlusion during remodelling of the periodontal tissues and further aging of the occlusion, i.e. the transitional changes in growth, dentoalveolar development and muscular adaptation. Retention is thus a continuation of orthodontic treatment.

If orthodontic tooth movement has not been followed by re-modelling of the supporting tissues, the tooth tends to return to its former position. Correct positioning of the entire tooth and good intercuspation are the main contributors to eventual stability.

An orthodontic movement that is opposed to the direction of functional tooth migration is more liable to relapse than one in which the directions correspond. Several factors are essential for the reestablishment of an adequate supporting apparatus during and after tooth movements, and conversely for an eventual lack of stability after treatment.

>The main remodelling of the PDL takes place near the alveolar bone. Unlike the PDL, the supraalveolar fibres are not anchored in a bone wall that is readily remodelled and therefore they have less chance of being reconstructed. Further more the remodelling of the gingival connective tissue is not as rapid as that of the PDL.

>The transseptal fibre system stabilizes teeth against separating forces and may actually maintain the contacts of adjacent teeth in a state of compression. This interproximal force is increased with occlusal loading and may help to explain physiological migration and long term incisor crowding.

>Certain fibre groups offer particular resistance to alterations in tooth position. Besides transseptal and dentoperiosteal fibres of the gingiva, the fibrils connecting heavy maxillary frenulum attachments to the alveolar process need a very long period of remodelling.

>Experimental studies have found that the stretched fibre bundles on the tension side tend to become functionally arranged according to the physiological movement of the tooth (AO-1964). During retention, new bone fills in the space between the bone spicules. This rearrangement and calcification of the new bone spicules result in a fairly dense bone tissue, which for a certain period prevents relapse of the tooth moved. Therefore, to avoid relapse, a tooth should be retained until total rearrangement of the structures involved has occurred.

>The most persistent relapse tendency is caused by the structures related to the marginal third of the root, whereas relatively little relapse tendency exists in the area adjacent to the middle and apical thirds.

>Periodontal structures undergo significant remodelling and rearrangement in cases where space closure of extraction sites has been achieved orthodontically. These extraction sites will retain a tendency to reopen. The compressed gingival tissue in the extraction sites may produce a long lasting epithelial fold or invagination. The increased amount of glycosaminoglycans may be responsible for possible relapse after orthodontic treatment, i.e. reopening of the extraction sites.

>Relapse tendency varies with the individual reaction pattern, a fact that calls for immediate insertion of a retention device. The duration of the retention period should vary according to the treatment that has been performed, from 1year to permanent retention.

CONCLUSION

Tooth movement is a highly conserved physiological mechanism for continuous adaptation of the dentition. Orthodontic tooth movement is a biomechanical exploitation of the physiologic mechanisms for developing and maintaining optimal occlusal function. The tooth continues to move until it achieves equilibrium with natural and applied loads.

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