BIOLOGY OF TOOTH MOVEMENT
Biology

The science of life of living organisms, including their structure, function, growth, origin, evolution and distribution.
Role Of Biology In Orthodontic Practice

Forces applied to the teeth

Evoke cellular responses in the teeth and their surrounding structures, including the PDL, alveolar bone, and gingiva.

Orthodontic tooth movement
Orthodontist should know these details that unfold during tooth movement, because some of these details may differ from one person to another, due to variables such as age, gender, nutritional habits and drug consumption.
Orthodontics is a field of endeavor where mechanics and biology are integrated.
What should be the meaning of “Biology” to the orthodontist?

A thorough knowledge and consideration of

1) Patient’s own physiological and anatomical features.

2) Specific pathological conditions present in individual patients.

3) Nutrients and drugs consumed regularly by each patient.
4) The details of the cellular and molecular effects of mechanical forces.

5) The capabilities and limitations of orthodontic treatment due to the interactive effects of above factors.

6) The ability of the tooth movement to cause irreversible damage, especially root resorption.
As a clinical profession, orthodontics must be based on a commanding knowledge of mechanics, biology, physiology and pathology.
Biological variations may be the foundation of the differences that are frequently observed in the outcomes of the treatment between patients with similar malocclusions, treated identically.
Orthodontics = mechanics + biology
But do we learn biology in typodont course?
This set up entirely ignores the biological aspect of tooth movement.
Above all, it should always be remembered that the orofacial complex is an integral part of the human body.
Orthodontic treatment is based on the principle that if prolonged pressure is applied to a tooth, tooth movement will occur as the bone around the tooth remodels.
Bony response is mediated by the PDL, tooth movement is primarily a PDL phenomenon.
TOOTH-SUPPORTING TISSUES

Gingiva
Root Cementum

During root formation a *primary cementum* is formed. After tooth eruption and in response to functional demands, a *secondary cementum* is formed that, in contrast to the primary cementum, contains cells.
Alveolar Bone
Periodontal Ligament
During masticatory function, the teeth and their periodontal structures are subjected to intermittent heavy forces.
Tooth contact lasts for 1 second or less, forces are quite heavy ranging from 1-2 kg while soft substances are chewed up to as much as 50 kg against a more resistant object.
When a tooth is subjected to heavy loads of this type, quick displacement of the tooth within the PDL space is prevented by the tissue fluid.

Instead the force is transmitted to the alveolar bone, which bends in response.
Physiologic response to heavy pressure against a tooth

<table>
<thead>
<tr>
<th>Time (seconds)</th>
<th>Event</th>
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<tbody>
<tr>
<td>&lt;1</td>
<td>PDL* fluid incompressible, alveolar bone bends, piezoelectric signal generated</td>
</tr>
<tr>
<td>1-2</td>
<td>PDL fluid expressed, tooth moves within PDL space</td>
</tr>
<tr>
<td>3-5</td>
<td>PDL fluid squeezed out, tissues compressed; immediate pain if pressure is heavy</td>
</tr>
</tbody>
</table>
The resistance provided by the tissue fluids allows normal mastication, with its force applications of 1 second or less, to occur without pain.
Pain is normally felt after 3-5 seconds of heavy force application, indicating that the fluids are expressed and crushing pressure is applied in the PDL in this amount of time.
Although the PDL is beautifully adapted to resist forces of short duration, it rapidly loses its adaptive capacity as the tissue fluids are squeezed out of its confined area.
Prolonged force, even of low magnitude, produces a different physiologic response – remodeling of the adjacent bone.

Orthodontic tooth movement is made possible by the application of prolonged forces
Resting pressure from the lips or cheeks and tongue are usually not balanced.

In some areas as in the mandibular anterior, tongue pressure is greater than the lip pressure.

In other areas as in the maxillary incisor region, lip pressure is greater.

The ability of the PDL to generate a force (active stabilization) and thereby contribute to the set of forces that determine the equilibrium, probably explains this.
Active stabilization implies a threshold for orthodontic force, since forces below the stabilization level would be expected to be ineffective.
The current concept is that active stabilization can overcome prolonged forces of a few grams at most, perhaps up to 5-10 gm/sq.cm often observed as the magnitude of unbalanced soft tissue resting pressures.
Theories of tooth movement

Two possible control elements, biologic electricity and pressure tension in the PDL that affects blood flow, are contrasted in the two major theories of orthodontic tooth movement.
From a contemporary perspective, it appears that both mechanisms may play a part in the biologic control of tooth movement.
Biologic electricity

The bioelectric theory relates tooth movement at least in part to the changes in bone metabolism controlled by the electric signals that are produced when alveolar bone bends.
Bone bending theory in 1969 by Baumrind – hypothesis states that orthodontic forces routinely produce alveolar bone deflection and these strains are accompanied by resultant changes in PDL.
Piezoelectricity

Piezoelectricity is a phenomenon observed in many crystalline materials in which a deformation of the crystal structure produces a flow of electric current as electrons are displaced from one location to another location. Farrar (1888)
Piezoelectricity can be observed in bone, collagen, and fibrous proteins as these are crystalline in nature.
Piezoelectric signals has two unusual characteristics –

1) quick decay rate – when a force is applied, a piezoelectric signal is created in response that quickly dies away to zero even though the force is maintained.

2) production of an equivalent signal, opposite in direction, when the force is released.
Not only the application of force cause distortion of crystalline structure and with it an electric signal, application of an electric field can cause a crystal to deform and produce force in doing so

-reverse piezo electricity
Streaming Potential

Ions in the fluids that bathe living bone interact with the complex electric field generated when the bone bends, causing temperature changes as well as electric signals.
As a result both convection and conduction currents can be detected in the extra cellular fluids and the currents are affected by the nature of fluids.

The small voltages that are observed are called the “streaming potential”. 
Stress generated signals are important in the general maintenance of the skeleton.

Without such signals, bone mineral is lost and general skeletal atrophy ensues - a situation that has proved troublesome for astronauts whose bones no longer flex in a weightless environment.
Signals generated by the bending of alveolar bone during normal chewing almost surely are important for maintenance of the bone around the teeth.
Sustained force of the type used to induce orthodontic tooth movement does not produce prominent stress–generated signals.
When the force is applied, a brief signal is created; when it is removed, the reverse signal appears.

As long as the force is sustained, nothing happens.
If stress generated signals were important in producing the bone remodeling associated with orthodontic tooth movement, a vibrating application of pressure would be advantageous.
Experiments indicated little or no advantage in vibrating over sustained force for the movement of teeth.
It appears that stress generated signals, important as they may be for normal skeletal function, probably have little if anything to do with the response to orthodontic tooth movement.
Another endogenous electric signal, which is called the “bioelectric potential” can be observed in bone that is not being stressed.

Metabolically active bone or connective tissue cells (in areas of active growth or remodeling) produce electronegative charges that are proportional to how active they are; inactive cells are electrically neutral.
Although the purpose of this bioelectric potential is not known, cellular activity can be modified by adding exogenous electric signals.

Both human and animal experiments indicate that when low voltage DC is applied to the alveolar bone, modifying the bioelectric potential, a tooth moves faster than its control.
In animal experiments, a pulsed electromagnetic field increased the role of tooth movement, apparently by shortening the initial “lag phase” before tooth movement begins.

Electromagnetic fields can be induced within tissues by adjacent magnets, and bone healing has been shown to be enhanced by certain types of fields.
It is possible that this effect can be utilized in the future to enhance orthodontic tooth movement and/or alter jaw growth.
Perhaps a conclusion is that even though stress generated electrical signals do not explain tooth movement, electric and electromagnetic influences can modify the bony remodeling on which tooth movement depends and may yet prove useful therapeutically.
The classic theory of tooth movement relies on chemical rather than electric signals as the stimulus for cellular differentiation and ultimately tooth movement.
The significance of mechanical stimuli in the maintenance and structure of skeletal tissues has been recognized since the middle of the 19th century (von Meyer, 1867; Wölff, 1892), and is the traditional starting point for any discussion of bone remodelling and tooth movement.
The original histological investigation that forms the foundation of our present knowledge was carried out by Sandstedt (1904, 1905) on dogs and published shortly after his death.
• Oppenheim (1911) study the tissue changes in the bone, incident to orthodontic tooth movement.

• Schwartz (1932) is said to be the author of this theory.
In this theory, alteration in blood flow within the PDL is produced by the sustained pressure that causes the tooth to shift position within the PDL space, compressing the PDL in some areas and stretching it in others.

Alterations in blood flow quickly create changes in the chemical environment.
For instance, oxygen levels certainly would fall in the compressed area, but might increase on the tension and the relative proportions of other metabolites would also change in a matter of minutes.
These chemical changes acting either directly or by stimulating the release of other biologically active agents, then would stimulate cellular differentiation and activity.
In essence, this view of tooth movement shoes three stages:

1) Alterations in blood flow associated with pressure within the PDL

2) The formation and/or release of chemical messengers, and

3) Activation of cells.
Animal experiments indicate that increased levels of CAMP, the second messenger for many important cellular functions including differentiation, appear after about 4 hrs of sustained pressure.

This amount of time to produce a response correlates well with the human response to removable appliances.
If a removable appliance is worn less than 4-6 hrs per day, it will produce no orthodontic effects.

Above this duration threshold, tooth movement occurs.
Experiments have shown that prostaglandin and interleukin-1 beta levels increase within the PDL within a short time after the application of pressure, and it now seems clear Pg E is an important mediator of the cellular responses.
Histologic changes seen during tooth movement varies according to the amount and duration of force applied.
Histologic changes seen during orthodontic tooth movement can be studied under two headings.

1) Changes following application of mild force.
2) Changes following application of extreme force.
After several weeks both osteoblasts and osteoclasts are present in Cancellous bone where the internal structure being reoriented by -resorption and deposition.

Trabecular pattern instead of lying in a vertical direction becomes oriented in a predominantly horizontal direction. This process reverses when the force is removed during the retention phase of the treatment.
CHANGES FOLLOWING APPLICATION OF MILD FORCE

- **CHANGES IN PRESSURE SIDE**

  Periodontal ligament gets compressed and a change in capillary blood supply is seen.

  After several days of force application, large multinucleated called Osteoclasts are seen along the walls of the socket.

  Osteoclasts are seen in crescent shaped evacuations of bone called howships lacunae.
Osteoclasts cause resorption of bone.

When the forces applied is minimal, the bone resorption is in the alveolar plate immediately adjacent to the ligament.

This kind of resorption is called frontal resorption.
CHANGES IN THE TENSION SIDE

Area of the tooth opposite to the direction of force is called the tension side.

Tension side shows stretched periodontal membrane.

Stretching of the periodontal fibres causes a raised vascularity in the tension side.

Raised vascularity causes mobilization of cells such as fibroblasts and osteoblasts in the area.
SECONDARY REMODELLING CHANGES

Whenever a force is applied to move teeth, the bone immediately near the teeth shows osteoclastic and osteoblastic activity in the pressure and tension side respectively.

In addition to bony changes immediately near the tooth, bony changes also take place elsewhere to maintain the width or thickness of the alveolar bone. These changes are called secondary remodelling changes.
Whenever extreme forces are applied on to tooth it results in crushing or total compression of the periodontal ligament.

Compression of the periodontal ligament may result in occlusion of the blood vessels. The ligament is deprived of its nutritional supply leading to regressive changes called hyalinization.
Bone cannot resorp in the frontal portion adjacent to the teeth. Rather the bone resorption occurs in the adjacent marrow spaces, the alveolar plate below, behind and above the hyalinized zones. This kind of resorption is called UNDERMINING OR REARWARD RESORPTION.
Over stretching of the periodontal tissue can lead to tear of the blood vessels and ischemia.

When extreme force is applied there is a net increase in osteoclastic activity as compared to bone formation.

Tooth becomes loosened in its socket. Pain and hyperaemia of the gingiva may occur due to application of extreme forces during orthodontic tooth movement.
### Physiologic Response to Sustained Pressure Against a Tooth

<table>
<thead>
<tr>
<th>TIME</th>
<th>Light pressure</th>
<th>Heavy pressure</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;1 sec</td>
<td>PDL* fluid incompressible, alveolar bone bends, piezoelectric signal generated</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-2 sec</td>
<td>PDL fluid expressed, tooth moves within PDL space</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3-5 sec</td>
<td>Blood vessels within PDL partially compressed on pressure side, dilated on tension side; PDL fibers and cells mechanically distorted</td>
</tr>
<tr>
<td>Minutes</td>
<td></td>
<td></td>
<td>Blood flow altered, oxygen tension begins to change; prostaglandins and cytokines released</td>
</tr>
<tr>
<td>Hours</td>
<td></td>
<td></td>
<td>Metabolic changes occurring: Chemical messengers affect cellular activity, enzyme levels change</td>
</tr>
<tr>
<td>~4 hours</td>
<td></td>
<td></td>
<td>Increased cAMP levels detectable, cellular differentiation begins within PDL</td>
</tr>
<tr>
<td>~2 days</td>
<td></td>
<td></td>
<td>Tooth movement beginning as osteoclasts/osteoblasts remodel bony socket</td>
</tr>
<tr>
<td></td>
<td>3-5 sec</td>
<td></td>
<td>Blood vessels within PDL occluded on pressure side</td>
</tr>
<tr>
<td>Minutes</td>
<td></td>
<td></td>
<td>Blood flow cut off to compressed PDL area</td>
</tr>
<tr>
<td>Hours</td>
<td></td>
<td></td>
<td>Cell death in compressed area</td>
</tr>
<tr>
<td>3-5 days</td>
<td></td>
<td></td>
<td>Cell differentiation in adjacent narrow spaces, undermining resorption begins</td>
</tr>
<tr>
<td>7-14 days</td>
<td></td>
<td></td>
<td>Undermining resorption removes lamina dura adjacent to compressed PDL, tooth movement occurs</td>
</tr>
</tbody>
</table>
Hyalinization is a form of degeneration characterized by formation of a clear, eosinophilic homogenous substance.

Periodontal ligament hyalinization denotes a locally compressed and degenerated periodontal ligament.

Conventional pathologic process of hyalinization is an irreversible one. But the periodontal ligament hyalinization is a reversible process.

Experimental evidences show that the periodontal ligament hyalinization on the pressure side occurs in some areas of almost all forms of orthodontic tooth movement. But the areas are wider when the force applied is extreme.
Presence of a hyalinized zone indicates that the ligament is non functional and therefore bone resorption cannot occur.

Thus the tooth is not capable of further movement until the local tissue damage has been removed and the adjacent alveolar bone wall resorbed.
Changes observed during formation of hyalinized zones are as follows.

Gradual shrinkage of periodontal ligament fibres.

Cellular structures become indistinct. Some nuclei become smaller (PYCNOTIC) while some nuclei disappear.

Compressed collagenous fibres gradually unite into a more or less cell free mass.

In addition certain changes also occur in the ground substance.

Break down of the blood vessel wall leading to spilling of its contents.

Osteoclasts are formed in marrow spaces and adjacent areas of inner bone surface after a period of 20-30 hours. They do not attack the cell free fibre bundles of the hyalinized tissue.
Elimination of hyalinized tissue occurs by two mechanisms.

By resorption of the alveolar bone by osteoclast differentiating from the peripheral intact periodontal membrane and in adjacent marrow spaces.

Invasion of cells and blood vessels from the periphery of the compressed zone by which the necrotic tissue is removed. The invading cells penetrate the hyalinized tissue and eliminates the unwanted fibrous tissue by enzymatic action and phagocytosis.
Experiments have shown that orthodontic appliances should not be reactivated more frequently than 3 week intervals.

A 4 week appointment cycle is more typical in clinical practice.

Undermining resorption requires 7-14 days.
Optimal orthodontic forces

According to Schwarz (1932), ‘optimal force is the force leading to a change in tissue pressure that approximated the capillary vessels’ blood pressure, thus preventing their occlusion in the compressed periodontal ligament.’ (capillary blood pressure is 20-25 gm/cm² of the root surface area).
Optimum orthodontic force should produce fast tooth movement without any harmful effects to the tooth and its supporting tissues with least discomfort to the patient.

The optimal force value varies according to the root surface area and the type of tooth movement.

For example, tipping of the canine distally requires less force than the one desired for translation.
Effects of Force Distribution and Types of Tooth Movement

<table>
<thead>
<tr>
<th>Type of movement</th>
<th>Force* (gm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tipping</td>
<td>35-60</td>
</tr>
<tr>
<td>Bodily movement (translation)</td>
<td>70-120</td>
</tr>
<tr>
<td>Root uprighting</td>
<td>50-100</td>
</tr>
<tr>
<td>Rotation</td>
<td>35-60</td>
</tr>
<tr>
<td>Extrusion</td>
<td>35-60</td>
</tr>
<tr>
<td>Intrusion</td>
<td>10-20</td>
</tr>
</tbody>
</table>
**Blood flow theory**

Fluid dynamic theory - by Bien.

Tooth movement occurs as a result of alteration in fluid dynamics.

When a force of short duration is applied to a tooth, the fluid in the PDL escapes through tiny vascular channels.
When the force is removed, the fluid is replenished by diffusion through capillary walls and recirculation of interstitial fluid is established.

A force of greater magnitude and duration causes the interstitial fluid in the PDL space to get squeezed out and move towards the apex and cervical margins. This is called "squeeze film" effect.
When an orthodontic force is applied, stenosis of the blood vessels of PDL occurs on the pressure side.

Vessels above the stenosis balloons forming aneurysms.

Alteration in the chemical environment at the site of stenosis due to decreased oxygen level in the compressed areas when compared to the tension side.
Formation of these aneurysms and vascular stenosis causes blood gases to escape into the interstitial fluid thereby creating a favourable environment for resorption.
Theories of tooth movement

Two possible control elements, biologic electricity and pressure tension in the PDL that affects blood flow, are contrasted in the two major theories of orthodontic tooth movement.
Recap

- Pressure tension theory
- Hyalinization
- Optimum orthodontic forces
- Blood flow theory – squeeze film effect
Burstone categorises the stages as -

Initial Phase
Lag Phase
Post Lag Phase
INITIAL PHASE

During the initial phase very rapid tooth movement is observed over a short distance and then stops.

This movement represents displacement of tooth in the periodontal membrane space and probably bending of alveolar bone.

Studies have shown that the light and heavy forces displace the tooth to the same extent during this initial phase of tooth movement.
LAG PHASE

During this phase little or no tooth movement occurs

Characterized by formation of hyalinized tissue in the periodontal ligament. This hyalinized zone should be resorbed before further tooth movement can occur

The duration of the lag phase depends on the amount of force used to move the tooth. If light forces are used the area of hyalinization is small. Thus frontal resorption is seen. If heavy forces are used the area of hyalinization is large. Thus rear resorption should occur to eliminate the hyalinized tissue. Thus longer lag period occurs.
POST LAG PHASE

After the lag phase the tooth movement progresses rapidly as the hyalinized zone is removed and the bone undergoes resorption.

During this post lag period the osteoclasts are found over a large area resulting in direct resorption of bony surface facing the periodontal ligament.
Study by Pilon divided the curve of tooth movement into 4 phases.

The first phase lasts 24 hours to 2 days and represents the initial movement of the tooth inside its bony socket.

It is followed by a second phase, when the tooth movement stops for 20 to 30 days.

After the removal of necrotic tissue formed during the second phase, tooth movement is accelerated in the third phase and continues into the fourth linear phase. The third and fourth phases comprise most of the total tooth movement during orthodontic treatment.
Drug Effects on the Response to Orthodontic Force

Orthodontic tooth movement largely depends on paradental tissue remodeling, and optimal force application is thought to elicit optimal responses.
The tooth and related tissues are influenced by disease processes that occur locally, such as periodontitis, as well as systemically, such as rheumatoid arthritis.
Drugs consumed by orthodontic patients can have a wide range of effects on the tooth movement process, either slowing it down or accelerating it, depending on the medications effects on cells involved in bone and periodontal ligament (PDL) remodeling.
Editorial by Turpin emphasized the importance of obtaining a proper drug intake history from all prospective orthodontic patients, so that any untoward effects on the course of orthodontic treatment is well managed.
Until recently, bronchospasm of the smooth airway musculature was considered to be a major cause of bronchial asthma, and beta-adrenergic agonists were used for its management.
With recent research, it has become increasingly clear that ongoing chronic inflammation of the bronchial wall plays a prominent role in the disease process, with histamine as an important mediator.
Complex interplay of mediators, such as leukotrienes, prostaglandins, and platelet-activating factor may lead to a chronic inflammation.
Based on this new disease concept, anti-inflammatory drugs, especially corticosteroids, have become the mainstay of therapy even in mild-to-moderate asthmatics.

Beta-adrenergic agonists remain the most important drug for the relief of acute bronchospasm.
Basic research has shown that patients with asthma have an imbalance between 2 groups of lymphocytes—the T helper 1 and the T helper 2 cells.

T Cells responsible for the synthesis and release in the lung of many inflammatory mediators, such as interleukin (IL)-4, IL-5, IL-6, IL-10, and IL-13.
These cytokines attract many inflammatory cells to the lung airways, including eosinophils, which secrete large amounts of histamines, prostaglandins, and leukotrienes, causing blood vessels to leak and lung tissues to swell and secrete large amounts of mucus.
These signal molecules can enter the circulation and reach the PDL, where they can interact with target cells involved in tissue remodeling and tooth movement.
The immune system of chronic asthmatic patients is always active and there will be an increased production of osteoclasts and odontoclasts, both multinucleated cells involved in bone as well as root resorption, respectively.
Therefore, it is recommended to defer orthodontic treatment in patients who report symptomatic disease or who have frequent flare-ups despite being adequately medicated.
Kalia et al. found noticeable change in the bone turnover rate and a differential response to short-term and long-term steroid therapy.
In the group that was prescribed with a short-duration drug therapy, orthodontic tooth movement was not affected, but at a tissue level, the remodeling process seemed delayed.
However, in the chronic group the tooth movement rate did increase, possibly as a result of the induction of a secondary hyperparathyroidism.
So it is recommended that delaying orthodontic treatment in patients with short-term corticosteroid treatment, until the intake of medication is over, and expect a faster rate of tooth movement with more alveolar bone loss in patients with a long-term steroid therapy, as in chronic asthmatics.
Meh et al demonstrated increased alveolar bone density in rats after administration of cetrizine along with orthodontic force application.

They observed a reduction in tooth movement by indirectly blocking recruitment and differentiation of osteoclasts by blocking H1 receptors on osteoblasts.
Cholesterol-Lowering Drugs and Orthodontic Tooth Movement

Statins are the most common and widely prescribed drugs for their high cholesterol-lowering activity
It has been noted in animal experiments that statins may play a dual role in that in normal dosages they promote bone formation more than bone resorption, increasing the BMD, whereas in lower doses, they are more catabolic than anabolic.
Han et al in a recent report showed the beneficial effects of simvastatin in preventing relapse after orthodontic treatment with the help of measurement on study models as well as immunohistochemistry.
On injection of simvastatin at 2.5 mg/kg body weights for 4 days posttreatment, they observed reduction in bone-resorbing activity of osteoclasts while stimulating bone formation, probably by controlling the ratio of local osteoprotegerin to receptor activator for nuclear factor kappa B ligand (RANKL) in the periodontal tissues, in the postorthodontically treated rat model.
Although all this research points to the beneficial effects of statins during the retention period, by reducing bone resorption, the anabolic effects of statins might be a drawback while performing mechanotherapy.
In addition, when attempting orthodontic treatment in patients who are on statin medication, the clinician should always be aware of the antiangiogenic property of these agents, which in turn can lead to osteonecrosis of the jaws.
Treatment with Bisphosphonates: The Recent Orthodontic Concern!

Much of the recent orthodontic literature related to drug usage among patients revolves around bisphosphonates, which are the mainstay treatment modality for bone cancer, osteoporosis, osteopenia, and bone- and calcium-related disorders.
Bisphosphonates act by suppressing osteoclastic activity, slowing down the bone remodeling process, thereby increasing BMD and reducing the risk of fractures in patients with osteopenia or osteoporosis.
The drug inhibits resorption of bone, formation of capillaries in the alveolus, and, consequently, it slows down the velocity of orthodontic tooth movement.

However, by inhibiting bone resorption, this drug may have positive effects on periodontal health.
The reported experimental and clinical research outcomes indicate that intake of bisphosphonates poses a significant challenge for orthodontic treatment planning, because of the possible pharmacologic inhibition of tooth movement, in addition to a potential for the development of jaw osteonecrosis.
Recently, usage of these drugs for reducing the rate of tooth movement, with the aim of enhancing anchorage conservation, and reducing post treatment relapse has been investigated by various researchers, who reported promising results.
Among the drugs investigated, risedronate appears to be the most effective in reducing orthodontic tooth movement, followed by 4-amino-1-hydroxybutylidene-1,1-bisphosphonate, and then clodronate.
It is further hypothesized that local delivery of these drugs may possibly be used in human patients in the future for anchorage reinforcement and root resorption prevention. However, the validity of these concepts has yet to be demonstrated clinically in human subjects.
Alcohol and Nicotine: Do They Affect Tooth Movement

Chronic alcohol consumption results in an osteopenic skeleton and an increased risk for osteoporosis.

These patients are prone for delayed fracture healing when compared with nonalcoholics.
Alcohol consumption during adolescence reduces peak bone mass and can result in relatively weak adult bones.

This occurs as a direct result of inhibited Wnt signaling and activation of peroxisome proliferator-activated receptor-pathways in mesenchymal stem cells by fatty acids as the result of oxidative stress.
Alcohol-induced oxidative stress as the result of increased nicotinamide adenine dinucleotide phosphate oxidase activity in bone cells also results in enhanced RANKL-RANK signaling to increase osteoclastogenesis.
Therefore, orthodontists treating patients with chronic alcoholism should be aware of the bone remodeling response and take necessary precautions to avoid excessive force application leading to tooth mobility.
Cigarette smoking is considered to be an important risk factor in lung cancers and is often implicated in the progression of atherosclerosis and cardiovascular diseases.
Smoking has injurious intraoral effects, such as increases in the progression of periodontal disease, as well as carious lesions.
The increase in bone resorption observed with nicotine is mediated through COX enzyme, which converts arachidonic acid to prostaglandins.

Nicotine causes an increase in the expression of the COX-2 gene and prostaglandin E2 release from human gingival fibroblasts in a time- and dose-dependent manner.
Orthodontists treating patients with a chronic smoking habit should discourage the habit and keep in mind that nicotine increase in bone resorption
The Effects of NSAIDs in Orthodontic Tooth Movement

Sterile inflammatory process after mechanical force application is considered to be of paramount importance as far as orthodontic tooth movement is concerned.
Various researches suggest that prostaglandin inhibition by NSAIDs triggers a cascade of events, leading to a reduction in the numbers of osteoclast-like cells, Howship lacunae, and blood vessels throughout all treatment periods.
Most of the reported studies have concentrated on prostaglandin inhibition, largely ignoring the fact that there are other mediators involved, as far as orthodontic tooth movement is concerned, such as leukotriens, ILs, cyclic adenosine monophosphate (cAMP) and guanosine monophosphate (cGMP), and calcium, to name a few.
With this in mind, researchers have proposed short-term administration of acetaminophen or acetylsalicylic acid, as these drugs act as modulators of prostaglandin production, helping to control pain and discomfort rather than inhibiting total prostaglandin synthesis, whereas other mediators of bone resorption continue to operate normally in the periodontal tissues.
The randomized control trial by Kohli and Kohli showed that premedication with 20 mg of piroxicam, 1 hour before the procedure, is more effective than 400 mg of ibuprofen in reducing orthodontic pain after separator placement.
All these research reports point to the fact that NSAIDs are effective in reducing postoperative orthodontic pain without affecting the tooth movement process.
The long-held notion that prostaglandin inhibitors significantly reduce the tooth movement process no longer stands valid as new pathways and molecules associated with tooth movement are discovered.
The main drawback of all these studies is their short-term nature, while the long-term effects as in patients with rheumatoid arthritis, who consume these drugs for a longer duration, are not available.
However, it can be clearly stated that these drugs have no effect on the orthodontic tooth movement process in the short-term, facilitating their usage for immediate pain relief after orthodontic appointments.
Relaxin, a pregnancy hormone—works by both reducing collagen synthesis and increasing collagen breakdown.

Preliminary data in rats showed faster tooth movement with Relaxin treatment.

University of Florida—did not show a positive effect.
Several other classes of drugs can affect prostaglandin levels, and therefore could affect the response to orthodontic force.
Tricyclic antidepressants (doxepin, amitriptyline, imipramine),

Antiarrhythmic agents (procaine),

Antimalarial drugs (quinine, quinidine, chloroquine),

Methyl xanthines
In addition, the anticonvulsant drug phenytoin has been reported to decrease tooth movement in rats, and some tetracyclines (e.g., doxycycline) inhibit osteoclast recruitment, an effect similar to bisphosphonates.
It is quite possible that pharmacologic agents to manipulate tooth movement in both directions will come into common use.
Direct injection of prostaglandin into the periodontal ligament has been shown to increase the rate of tooth movement, but this is quite painful and not very practical.
It now is possible in periodontal therapy to place miniature spheres that release a specific antibiotic into the gingival sulcus and in periodontal pockets.
If a prostaglandin inhibitor or any of mentioned drugs were placed in similar mini-spheres and could be maintained in the sulcus around teeth that were to serve as anchors, the improved anchorage would allow more effective movement of the teeth whose movement was desired.
Drugs that inhibit tooth movement, however, already are encountered frequently, though not yet prescribed for their tooth-stabilizing effect.
It seems likely that at some point in future, drugs to facilitate tooth movement will become clinically useful – but there is no way to know how long it will take to develop them.
Continuous, interrupted, and intermittent forces

Force magnitude decreases as the tooth moves and there is a decline from the desired force level between two patient appointments. This is called force decay.

Orthodontic forces can be classified as continuous, interrupted and intermittent.
Continuous force means that the force magnitude is maintained at almost the same level in the period between two activations.

Interrupted force declines to zero between activations.
Fixed appliances produce continuous and interrupted forces.

It is not always possible to distinguish between continuous and interrupted movements.
Intermittent force falls to zero when the appliance is removed and it also shows force decay with tooth movement.

Intermittent forces act as an impulse for short periods with a series of interruptions.
Intermittent forces are produced by removable appliances and headgears.
Light continuous forces produce efficient tooth movement with the least harmful effects.

Heavy forces are physiologically acceptable if they act as interrupted ones with a rest period in between.
The rest period between appliance activations is the time used by the tissues for reorganization.

This rest can promote favourable cell proliferation for further tissue changes when the appliance is activated again.
Light continuous and heavy interrupted forces are clinically acceptable whereas heavy continuous forces should be avoided.

Orthodontic appliances should not be reactivated more frequently than 3-week intervals.

A 4-week appointment cycle is more typical in a clinical practice.
Deleterious effects of Orthodontic forces

Mobility and Pain Related to Orthodontic Treatment

Effects on the Pulp

Effects on Root Structure

Effects of Treatment on the Height of Alveolar Bone
Mobility and Pain Related to Orthodontic Treatment

Orthodontic tooth movement requires not only a remodeling of bone adjacent to the teeth, but also a reorganization of the PDL itself.

Fibers become detached from the bone and cementum, then reattach at a later time.
Radiographically it can be observed that the PDL space widens during orthodontic tooth movement.

The combination of a wider ligament space and a somewhat disorganized ligament means that some increase in mobility will be observed in every patient.
Excessive mobility is an indication that excessive forces are being encountered. This may occur because the patient is clenching or grinding against a tooth that has moved into a position of traumatic occlusion.
If heavy pressure is applied to a tooth, pain develops almost immediately.

If appropriate orthodontic force is applied, the patient feels little pain immediately.

Several hours later, however, pain usually appears.
The patient feels a mild aching sensation, and the teeth are quite sensitive to pressures, so that biting a hard object hurts.

The pain typically lasts for 2 to 4 days, then disappears until the orthodontic appliance is reactivate.
It is commonly noted that there is a great deal of individual variation in any pain experience, and this is certainly true of orthodontic pain.

Some patients report little or no pain even with relatively heavy forces, whereas others experience considerable discomfort with quite light force.
The pain associated with orthodontic treatment is related to the development of ischemic areas in the PDL that will undergo sterile necrosis - hyalinization.
If the source of pain is the development of ischemic areas, strategies to temporarily relieve pressure and allow blood flow through compressed areas should help.

In fact, if light forces are used, the amount of pain experienced by patients can be decreased by having them engage in repetitive chewing (of sugarless gum, a plastic wafer placed between the teeth, or whatever) during the first 8 hours after the orthodontic appliance is activate
Presumably this works by temporarily displacing the teeth enough to allow some blood flow through compressed areas thereby preventing build-up of metabolic products that stimulate pain receptors.
It is rare but not impossible for orthodontic patients to develop pain and inflammation of soft tissues not because of the orthodontic force, but because of an allergic reaction.
Effects on the Pulp

In theory, the application of light sustained force to the crown of a tooth should produce a PDL reaction but should have little if any effect on the pulp.
Although pulpal reactions to orthodontic treatment are minimal, there is probably a modest and transient inflammatory response within the pulp, at least at the beginning of 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.
There are occasional reports of loss of tooth vitality during orthodontic treatment. Usually there is a history of previous trauma to the tooth.
Since the response of the PDL, not the pulp, is the key element in orthodontic tooth movement, moving endodontically treated teeth is perfectly feasible.
Although some evidence has indicated that endodontically treated teeth are more prone to root resorption during orthodontics than are teeth with normal vitality.

Recent studies suggest that this is not the case. Severe root resorption should not be expected as a consequence of moving a nonvital tooth that has had proper endodontic therapy.
One special circumstance is a tooth that experienced severe intrusive trauma and required pulp therapy for that reason.

If such a tooth must be repositioned orthodontically, resorption seems less likely if a calcium hydroxide fill is maintained until the tooth movement is completed, and then the definitive root canal filling is placed.
Effects on Root Structure

Orthodontic treatment requires resorption and apposition of bone adjacent to the root structure of teeth.
Rygh and co-workers have shown that cementum adjacent to hyalinized (necrotic) areas of the PDL is "marked" by this contact and that clast cells attack this marked cementum when the PDL area is repaired.
Root remodeling, in other words, is a constant feature of orthodontic tooth movement, but permanent loss of root structure would occur only if repair did not replace the initially resorbed cementum.
Shortening of the root occurs when cavities coalesce at the apex, so that peninsulas of root structure are cut off as islands. Then the repair process smooths over the new root surface, and a net loss of root length occurs.

This is why, although both the sides and the apex of the root experience resorption, roots become shorter but not thinner as a result of orthodontic tooth movement.
Shortening of tooth roots during orthodontic treatment occurs in three distinct forms that must be distinguished when the etiology of resorption is considered.

- Moderate Generalized Resorption
- Severe Generalized Resorption
- Severe Localized Resorption
Effects of Treatment on the Height of Alveolar Bone

Since the presence of orthodontic appliances increases the amount of gingival inflammation, even with good hygiene, potential side effect of treatment might seem even more likely.
Fortunately, excessive loss of crestal bone height is almost never seen as a complication of orthodontic treatment.

Loss of alveolar crest height in one large series of patients averaged less than 0.5mm and almost never exceeded 1mm with the greatest changes at extraction sites.
In short........
For many years it was thought that the root structure of the teeth was not remodeled in the same way as bone.

More recent research has made it plain that when orthodontic forces are applied, there is usually an attack on the cementum of the root, just as there is an attack on adjacent bone, but repair of cementum also occurs.
Alveolar bone resorption and deposition during orthodontic tooth movement is a cell-mediated process regulated by various factors. However, the mechanisms involved in conversion of O.F. into biologic activity are not completely understood.
The discussion on biological basis of tooth movement, invariably raises the following queries:........
How do cells distinguish between pressure and tension?

Do they have separate receptors for tensile and compressive forces?

If compressive force causes resorption why do different trajectories in maxilla and mandible have linear bone deposition?

In spite of compressive force in TMJ, the condyle grows, how?

The weight bearing joints of the body shows linear increment in size of bone at the compressive end, how?