Lec 8 TRAUMA FROM OCCLUSION

TYPES OF OCCLUSAL FORCES:

1. **Physiologically normal occlusal forces** in chewing and swallowing: small and rarely exceeding 5 N. They provide the positive stimulus to maintaining the periodontium and the alveolar bone in a healthy and functional condition.

2. Impact forces: mainly high but of short duration. The periodontium can sustain high forces during a short period; however, forces exceeding the viscoelastic buffer capacities of the periodontal ligament will result in fracture of tooth and bone.

3.Continuous forces: very low forces (for example, orthodontic forces), but continuously applied in one direction are effective in displacing a tooth by remodeling the alveolus.

Forces in one direction: orthodontic forces (bodily or tipping forces) produce distinct zones of pressure and tension

4. Jiggling forces: intermittent forces in two different directions (premature contacts on, for example, crowns, fillings) result in widening of the alveolus and in increased mobility

Periodontal Response to External Forces

Adaptive Capacity of the Periodontium to Occlusal Forces

The periodontal ligament has a cushioning effect on forces applied to teeth as means to accommodate forces exerted on the crown. Due to the elastic nature of the periodontal ligament, all teeth with normal bone support present with physiologic mobility in all directions. Physiologic tooth mobility varies among individuals and within the dentition of the same individual. In the absence of excessive occlusal forces or the absence of reduced bone support induced by inflammatory periodontal disease, tooth mobility remains unchanged due to the fact that physiologic forces are not able to induce changes to the periodontal tissues. When there is an increase in occlusal forces, changes occur in the periodontium in order to accommodate for such forces. **Changes in the periodontium depend on** the **magnitude, direction, duration, and frequency of increased occlusal forces**

When the **magnitude** of occlusal forces is increased, the periodontium responds **with a widening of the periodontal ligament** space, an increase in the number and width of periodontal ligament fibers, and an **increase in the density of alveolar bone.**

Changing the **direction** of occlusal forces causes **a reorientation** of the stresses and strains within the periodontium . The principal fibers of the periodontal ligament are arranged so that they best accommodate occlusal forces along the long axis of the tooth. **Lateral** (horizontal) and torque (**rotational**) forces are **more likely to injure** the periodontium.

The response of alveolar bone is also affected by the **duration and frequency** of occlusal forces. **Constant** pressure on the bone is **more injurious** than intermittent forces. The **more frequent** the application of an **intermittent force**, **the more injurious** the force is to the periodontium.

Trauma From Occlusion

Trauma from occlusion is defined as microscopic alterations of periodontal structures in the area of the periodontal ligament that become manifest clinically in the elevation of tooth mobility. An inherent "margin of safety" that is common to all tissues permits some variation in occlusion without adversely affecting the periodontium. However, when occlusal forces **exceed** the adaptive capacity of the tissues, **tissue injury** results. The resultant injury is termed trauma from occlusion, which is also known as occlusal trauma.

Thus trauma from occlusion **refers to the tissue injury rather than the occlusal force.** An occlusion that produces such an injury is called a traumatic occlusion.

Excessive occlusal forces may also,

a- disrupt the function of the masticatory muscules and cause painful spasms,
b-injure the temporomandibular joints, or
c-produce excessive tooth wear.

Classification of Trauma From Occlusion

Trauma from occlusion can be classified according to the injurious occlusal force(s) mode of onset (acute and chronic) or according to the capacity of the periodontium to resist to occlusal forces (primary and secondary).

Acute and Chronic Trauma From Occlusion

Acute trauma from occlusion refers to periodontal changes associated with an **abrupt occlusal impact** such as that produced by biting on a hard object (e.g., an olive pit). In addition, restorations or prosthetic appliances that interfere with or alter the direction of occlusal forces on the teeth may also induce acute trauma.

Acute trauma results in **tooth pain, sensitivity to percussion, and increased tooth mobility**. Acute trauma can also produce **cementum tears**. If the **force is dissipated** by a shift in the position of the tooth or by the wearing away or correction of the restoration, then **the injury heals**, and the symptoms subside. Otherwise, periodontal injury may worsen and develop into necrosis accompanied by periodontal abscess formation, or it may persist as a symptom-free chronic condition.

Chronic trauma from occlusion refers to periodontal changes associated with **gradual changes in occlusion** produced by tooth wear, drifting movement, and extrusion of the teeth in combination with parafunctional habits (e.g., bruxism, clenching) rather than as a sequela of acute periodontal trauma. Chronic trauma from occlusion is **more common** than the acute form and of greater clinical significance.

The **criterion** that determines if an occlusion is traumatic **is whether it produces periodontal injury**; the criterion is **not based** on how the teeth occlude. Any occlusion that produces periodontal injury is traumatic. Periodontal injury may occur when the occlusion appears normal. The dentition may **be anatomically and aesthetically acceptable but functionally injurious**. Similarly, not all malocclusions are necessarily injurious to the periodontium. **Traumatic occlusal** relationships are **referred** to by such terms as **occlusal disharmony, functional imbalance, and occlusal dystrophy**.

These terms **refer to the effect of the occlusion** on the periodontium rather than to the position of the teeth. Because trauma from occlusion refers to the tissue injury rather than the occlusion, an increased occlusal force is not traumatic if the periodontium can accommodate it.

Primary and Secondary Trauma From Occlusion

As mentioned previously, trauma from occlusion can also be classified according to the **capacity of the periodontium to resist occlusal forces** into primary and secondary trauma from occlusion.

In other words, trauma from occlusion may be caused by alterations in occlusal forces, a reduced capacity of the periodontium to withstand occlusal forces, or both. When trauma from occlusion **is the result of alterations in occlusal forces**, it is **called primary trauma from occlusion**. When it **results from the reduced ability of the tissues to resist the occlusal forces**, it is known as **secondary trauma from occlusion**.

Primary trauma from occlusion occurs if trauma from occlusion is **considered the primary etiologic factor** in periodontal destruction and if the only local alteration to which a tooth is subjected is a result of occlusion.

Examples include periodontal injury produced around teeth with a previously healthy periodontium after the following:

(1) the insertion of a “high filling”.

(2) the insertion of a prosthetic replacement that creates excessive forces on abutment and antagonistic teeth.

(3) the drifting movement or extrusion of the teeth into spaces created by unreplaced missing teeth. or

(4) the orthodontic movement of teeth into functionally unacceptable positions.

Most studies of the effect of trauma from occlusion involving experimental animals have examined the primary type of trauma. Changes produced by primary trauma **do not alter the level of connective tissue attachment and do not initiate pocket formation**. This is probably because the supracrestal **gingival fibers** are **not affected** and therefore prevent the apical migration of the junctional epithelium.

Secondary trauma from occlusion occurs when the adaptive capacity of the tissues to withstand occlusal forces is **impaired by bone loss** that results from marginal inflammation. This reduces the periodontal attachment area and alters the leverage on the remaining tissues. The periodontium becomes more vulnerable to injury, and previously well-tolerated occlusal forces become traumatic.

Traumatic forces can occur on (**A**) normal periodontium with normal height of bone, (**B**) normal periodontium with reduced height of bone, or (**C**) marginal periodontitis with reduced height of bone.

The **first** case is an example of **primary** trauma from occlusion, whereas the **last two** represent **secondary** trauma from occlusion. It has been found in experimental animals that **systemic disorders** can reduce tissue resistance and that previously tolerable forces may become excessive. This could theoretically represent another mechanism by which tissue resistance to increased forces is lowered, thereby resulting in secondary trauma from occlusion.

Stages of Tissue Response to Increased Occlusal Forces

Tissue response occurs in three stages: **injury, repair, and adaptive remodeling** of the periodontium.

Stage I: Injury

Tissue injury is produced by excessive occlusal forces. The body then attempts to repair the injury and restore the periodontium. This can occur if the forces are diminished or if

the tooth drifts away from them. If the offending force is chronic, however, the periodontium is remodeled to cushion its impact. **The ligament is widened at the expense of the bone, which results in angular bone defects without periodontal pockets**, and the tooth becomes loose.

Under the forces of occlusion, a tooth rotates around a **fulcrum or axis** of rotation, which in **single-rooted teeth** is located **in the junction between the middle third and the apical** third of the clinical root and in **multirooted teeth** in the middle of the **interradicular bone**. This creates areas of pressure and tension on opposite sides of the fulcrum. Different lesions are produced by different degrees of pressure and tension. If jiggling forces are exerted, these different lesions may coexist in the same area.

Slightly excessive pressure stimulates **resorption** of the alveolar bone, with a resultant **widening** of the periodontal ligament space.

Slightly excessive tension causes **elongation** of the periodontal ligament fibers and the **apposition** of alveolar bone. In areas of increased pressure, the blood vessels are numerous and reduced in size; in areas of increased tension, they are enlarged.

Greater pressure produces a gradation of changes in the periodontal ligament, starting with **compression of the fibers**, which produces areas of hyalinization. Subsequent **injury to the fibroblasts** and other connective tissue cells leads to **necrosis** of areas of the ligament.

Vascular changes are also produced:

within 30 minutes, impairment and stasis of blood flow occur; **at 2 to 3 hours**, blood vessels appear to be packed with erythrocytes, which start to fragment; and **between 1 and 7 days**, disintegration of the blood vessel walls and release of the contents into the surrounding tissue occur. In addition, increased resorption of alveolar bone and resorption of the tooth surface occur.

Severe tension causes widening of the periodontal ligament, thrombosis, hemorrhage, tearing of the periodontal ligament, and resorption of alveolar bone. Pressure severe enough to force the root against bone causes necrosis of the periodontal ligament and bone. **The bone is resorbed from viable periodontal ligament adjacent to necrotic areas and from marrow spaces; this process is called undermining resorption.**

The areas of the periodontium that are most susceptible to injury from excessive occlusal forces are the **furcations.**

Injury to the periodontium produces a temporary depression in mitotic activity, in the rate of proliferation and differentiation of fibroblasts, in collagen formation, and in bone

formation. These return to normal levels after the dissipation of the forces. The **injury phase shows an increase** in areas of resorption and **a decrease** in bone formation

Stage II: Repair

Repair occurs constantly in the **normal** periodontium, and trauma from occlusion stimulates increased reparative activity. The damaged tissues are removed, and new connective tissue cells and fibers, bone, and cementum are formed in an attempt to restore the injured periodontium. Forces remain **traumatic** only as long as the **damage produced exceeds the reparative** capacity of the tissues. **The repair phase demonstrates decreased resorption and increased bone formation**. When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned bony trabeculae with new bone. This attempt to compensate for lost bone is called **buttressing bone formation**, and it is an important feature of the reparative process associated with trauma from occlusion. It also occurs when bone is destroyed by inflammation or osteolytic tumors.

Buttressing bone formation occurs within the jaw (central buttressing) and on the bone surface (peripheral buttressing).

During **central buttressin**g, the endosteal cells deposit new bone, which restores the bony trabeculae and reduces the size of the marrow spaces. **Peripheral buttressing** occurs on the facial and lingual surfaces of the alveolar plate. Depending on its severity, peripheral buttressing may produce a **shelf like thickening** of the alveolar margin, which is referred to as **lipping,** or a **pronounced bulge** in the contour of the facial and lingual bone. **Cartilage-like** material sometimes develops in the periodontal ligament space as an aftermath of the trauma. The formation of crystals from erythrocytes has also been demonstrated

Stage III: Adaptive Remodeling of the Periodontium

If the repair process cannot keep pace with the destruction caused by the occlusion, the periodontium is remodeled in an effort to create a structural relationship in which the forces are no longer injurious to the tissues. This results in a **widened periodontal ligament, which is funnel shaped at the crest, and angular defects** in the bone, with **no** pocket formation.

The involved teeth become **loose**. **Increased v**ascularization has also been reported. After adaptive remodeling of the periodontium, resorption and formation of the bone return to normal.

Effects of Insufficient Occlusal Force

Insufficient occlusal force may also be **injurious** to the supporting periodontal tissues. Insufficient stimulation causes **thinning** of the periodontal ligament, **atrophy** of the fibers, **osteoporosis** of the alveolar bone, and a **reduction** in bone height. Hypofunction can result from an open-bite relationship, an absence of functional antagonists, or unilateral chewing habits that neglect one side of the mouth.

Reversibility of Traumatic Lesions

Trauma from occlusion is **reversible**. When trauma is artificially induced in experimental animals, the teeth move away or intrude into the jaw. When the impact of the artificially created force is relieved, the tissues undergo repair. **Although** trauma from occlusion is reversible under such conditions, **it does not always correct itself**, and therefore it is not always temporary or of limited clinical significance. The injurious force must be relieved for repair to occur. If conditions in humans do not permit the teeth to escape from or adapt to excessive occlusal force, periodontal damage persists and worsens.

The presence of **inflammation in the periodontium as a result of plaque** accumulation may impair the reversibility of traumatic lesions.

Effects of Excessive Occlusal Forces on Dental Pulp

The effects of excessive occlusal forces on the dental pulp have not been established. Some clinicians report the disappearance of pulpal symptoms after the correction of excessive occlusal forces. Pulpal reactions have been noted in animals subjected to increased occlusal forces, but these did not occur when the forces were minimal and occurred over short periods.

Relationship Between Plaque-Induced Periodontal Diseases and Trauma From Occlusion

Numerous studies have been performed that have attempted to determine the mechanisms by which trauma from occlusion may affect periodontal disease. Trauma from occlusion in humans, however, is the result of forces that act alternatively in opposing directions. These were analyzed in experimental animals with “jiggling forces,” which were usually produced by a high crown in combination with an orthodontic appliance that would bring the traumatized tooth back to its original position when the force was dissipated by separating the teeth. With another method, the teeth were separated by wooden or elastic material wedged interproximally to displace a tooth toward the opposite proximal side. After 48 hours, the wedge was removed, and the procedure was repeated on the opposite side. These studies resulted in a combination of changes produced by pressure and

tension on both sides of the tooth, with an increase in the width of the ligament and increased tooth mobility. **None of these methods caused gingival inflammation or pocket formation**, and the results essentially represented different degrees of functional adaptation to increased forces. To mimic the problem in humans more closely, studies were then conducted on the effect produced by jiggling trauma and simultaneous plaque-induced gingival inflammation. **The accumulation of bacterial plaque that initiates gingivitis and results in periodontal pocket formation affects the marginal gingiva**, but **trauma from occlusion occurs in the supporting tissues and does not affect the gingiva** . The marginal gingiva is **unaffected** by trauma from occlusion **because its blood supply is not affected**, even when the vessels of the periodontal ligament are obliterated by excessive occlusal forces.

It has been repeatedly proved that trauma from occlusion **does not cause** pockets or gingivitis and that it also **does not** increase gingival fluid flow. Furthermore, experimental trauma in dogs does not influence the bacterial repopulation of pockets after scaling and root planing. However, mobile teeth in humans harbor significantly higher proportions of Campylobacter rectus and Peptostreptococcus micros than do non mobile teeth.

1-Glickman's concept

Glickman and Smulow proposed the theory in the early 1960s that a traumatogenic occlusion **may act as a cofactor** in the progression of periodontitis. This theory is known as the **"co destructive theory**.

Glickman (1965, 1967) claimed that, if forces of an abnormal magnitude are acting on teeth harboring subgingival plaque, then the alley of the spread of a plaque-associated gingival lesion can be altered.

The periodontal structures can be divided into:

1. The zone of irritation.

2. The zone of codestruction.

The **zone of irritation consists of the marginal and interdental gingiva**. The soft tissue zone is surrounded by the hard tissue (the tooth) on one side and has **no impact** by occlusal forces. This means that gingival inflammation cannot be initiated by TFO but rather due to irritation from plaque.

The **zone of codestruction** consists of the **periodontal ligament, cementum, and alveolar bone** and is coronally delineated by the transseptal and the dentoalveolar collagen fiber bundles. The inflammatory lesion in the zone of irritation can, in teeth not subjected to trauma, propagate into the alveolar bone, while in teeth subjected to trauma

from occlusion, the inflammatory infiltrate spreads directly into periodontal ligament . In conclusion; **As long as inflammation is confined to the gingiva, the inflammatory process is not affected by occlusal forces**. When inflammation **extends** from the gingiva into the supporting periodontal tissues (i.e., when gingivitis becomes periodontitis), **plaque-induced inflammation enters the zone that is influenced by occlusion**, (zone of co-destruction).

2-Waerhaug's concept

Waerhaug and Glickman, both had examined autopsy specimens, but Waerhaug also measured the distance from the subgingival plaque to the periphery of the associated inflammatory cell infiltrate in the gingiva and the adjacent alveolar bone surface. **He came to the conclusion that angular bony defects and also infrabony pockets occur equally often at periodontal sites which are unaffected by TFO like in traumatized teeth**. In other words, he **refuted** the hypothesis that trauma from occlusion played a role in the spread of a gingival lesion into the "zone of co-destruction".

The **loss of connective attachment and the resorption of bone** around teeth are, according to Waerhaug, exclusively the **result of inflammatory lesions** associated with subgingival plaque. Waerhaug concluded that angular bony defects and infrabony pockets occur when the subgingival plaque of one tooth has reached a more apical level than the microbiota on the neighboring tooth, and when the volume of the alveolar bone surrounding the roots is comparatively large.

Clinical Signs of Trauma From Occlusion

Alone The most **common clinical sign** of trauma to the periodontium is increased **tooth mobility**. During the injury stage of trauma from occlusion, the destruction of periodontal fibers occurs, which increases tooth mobility. During the final stage, the accommodation of the periodontium to increased forces entails a widening of the periodontal ligament, which also leads to increased tooth mobility.

Although this tooth mobility is greater than the so-called normal mobility, it cannot be considered pathologic, because it is an adaptation and not a disease process. If it does become progressively worse, it can then be considered pathologic.

-Other causes of increased tooth mobility include advanced bone loss, inflammation of the periodontal ligament of periodontal or periapical origin, and some systemic causes (e.g., pregnancy). The destruction of surrounding alveolar bone, such as occurs with osteomyelitis or jaw tumors, may also increase tooth mobility.

Other clinical signs of TFO may include Fremitus (sensitive),Pain, Tooth migration, Attrition ,Muscle/joint pain and Fractures, chipping

Radiographic signs of trauma from occlusion may include the following:

1. Increased width of the periodontal space, often with **thickening** of the lamina dura along the lateral aspect of the root, in the apical region, and in bifurcation areas. These changes do not necessarily indicate destructive changes, because they may result from thickening and strengthening of the periodontal ligament and alveolar bone, thereby constituting a favorable response to increased occlusal forces.

2. A **vertical** rather than horizontal destruction of the interdental septum.

3. Radiolucency and condensation of the alveolar bone.

4. Root resorption ,hypercementosis

Treatment Outcomes

1. Reduce/eliminate tooth mobility 2. Eliminate occlusal prematurity's & fremitus 3. Eliminate parafunctional habits 4. Prevent further tooth migration 5. Decrease/stabilize radiographic changes

Therapy

Primary Occlusal Trauma:

Selective grinding, Habit control, Orthodontic movement, inter occlusal appliance

Secondary Occlusal Trauma:

Splinting, Selective grinding, Orthodontic movement,

Unsuccessful Therapy

1. Increasing tooth mobility **2.** Progressive tooth migration **3.** Continued client discomfort **4.** Premature contacts remain **5.** No change in radiographs/worsening **6.** Parafunctional habits remain **7.** TMJ problems remain or worsen

conclusion

Experiments carried out in humans as well as animals, have produced convincing evidence that neither unilateral forces nor jiggling forces, applied to teeth with a healthy periodontium, result in pocket formation or in loss of connective tissue attachment.

Trauma from occlusion cannot induce periodontal tissue breakdown. Trauma from occlusion does, however, result in resorption of alveolar bone leading to an increased tooth mobility which can be of a transient or permanent character. This bone resorption with resulting increased tooth mobility should be regarded as a physiologic adaptation of the periodontal ligament and surrounding alveolar bone to the traumatizing forces, i.e. to altered functional demands. In teeth with progressive, plaque-associated periodontal disease, trauma from occlusion may, however, under certain conditions enhance the rate of progression of the disease, i.e. act as a cofactor in the destructive process. From a clinical point of view, this knowledge strengthens the demand for proper treatment of plaque associated periodontal disease. This treatment will arrest the destruction of the periodontal tissues even if the occlusal trauma persists. A treatment directed towards the trauma alone, however, i.e. occlusal adjustment or splinting, may reduce the mobility of the traumatized teeth and result in some regrowth of bone, but it will not arrest the rate of further breakdown of the supporting apparatus caused by plaque