TRAUMA FROM OCCLUSION

Types of occlusal forces:

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.

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.

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

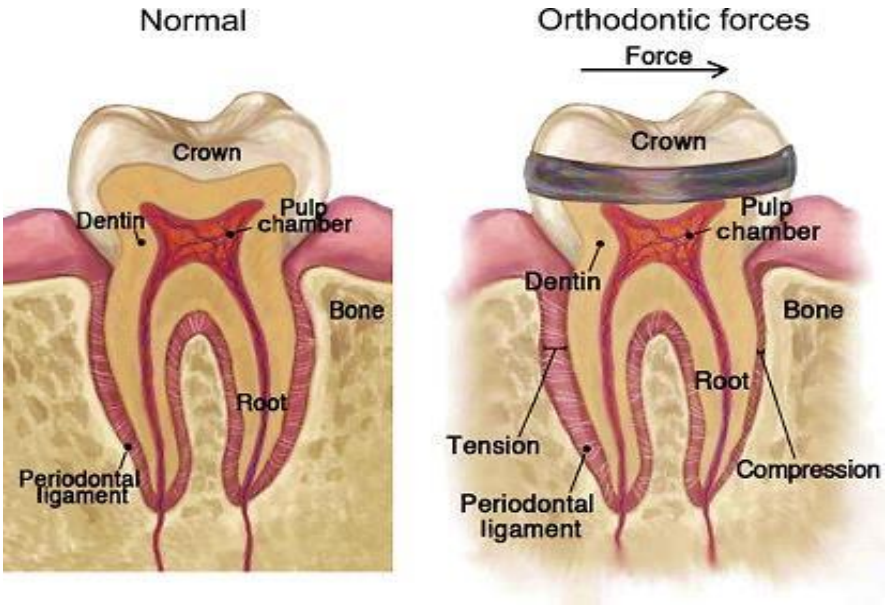

Fig. :- Effect of orthodontic force.

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. 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. 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**.

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 disrupt the a-function of the masticatory musculature 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. Malocclusion is not necessary to produce trauma; 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 **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

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 TFO**. When it results from the reduced ability of the tissues to resist the occlusal forces, it is known as **secondary TFO**.

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.

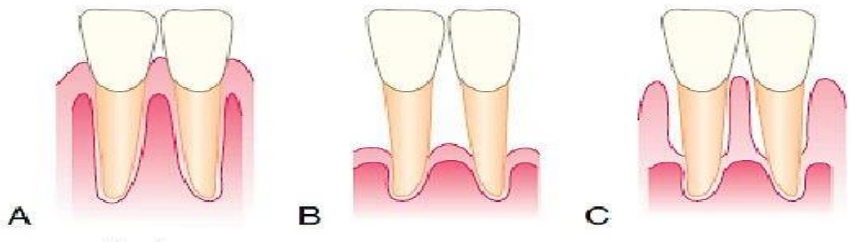

Fig.:- Traumatic forces can occur on (A) normal periodontium with normal height of bone, (B) normal periodontium with reduced height of bone, or (C) marginal periodontitis with reduced height of bone.

The first case is an example of primary trauma from occlusion, whereas the last two represent secondary trauma from occlusion.

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.

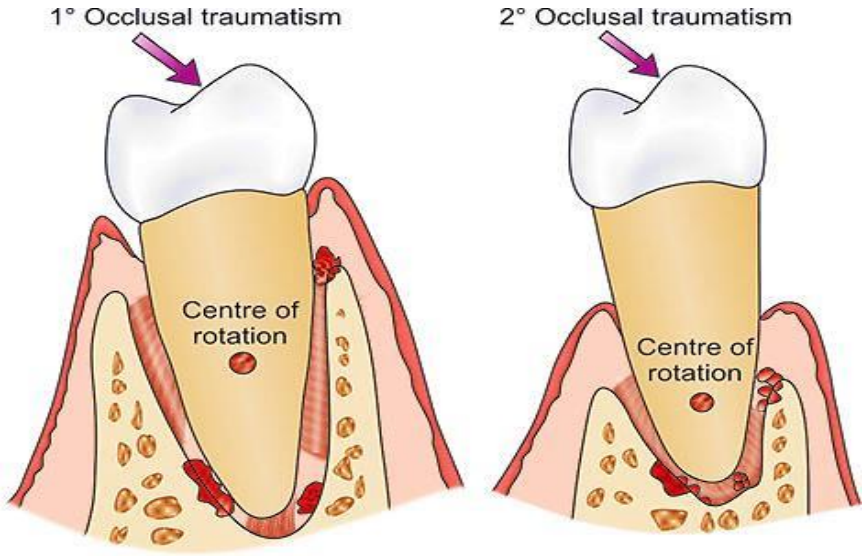

Fig.:- Traumatic forces occur in normal periodontium or in reduced periodontium with different center of rotation.

- ☒ **Slightly excessive pressure** stimulates resorption of the alveolar bone, with a resultant widening of the periodontal ligament space. In areas of increased pressure, the blood vessels are numerous and reduced in size.
- ☒ **Slightly excessive tension** causes elongation of the periodontal ligament fibers and the apposition of alveolar bone.; in areas of increased tension, the blood vessels are enlarged.
- ☒ **Greater pressure** produce a 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.

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

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**.

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),** Buttressing bone formation occurs within the jaw **(central buttressing)** and on the bone surface **(peripheral buttressing)**. Depending on its severity, peripheral buttressing may produce a shelf like thickening of the alveolar margin, which is referred to as **lipping** in the contour of the facial and lingual bone**.**

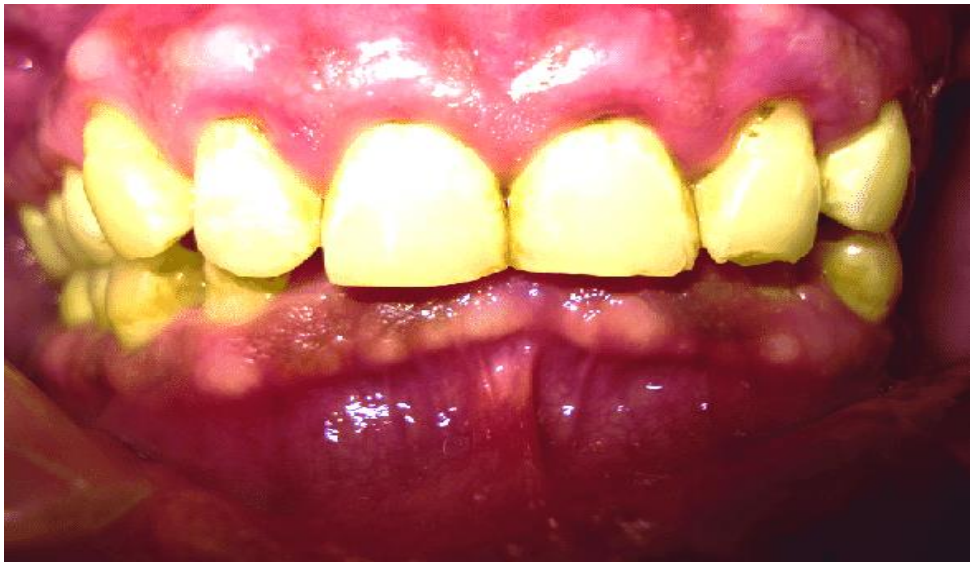

Fig.:- Peripheral buttressing bone formation.

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

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.

Concepts of relationship between trauma from occlusion and periodontal disease

It has been 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 .

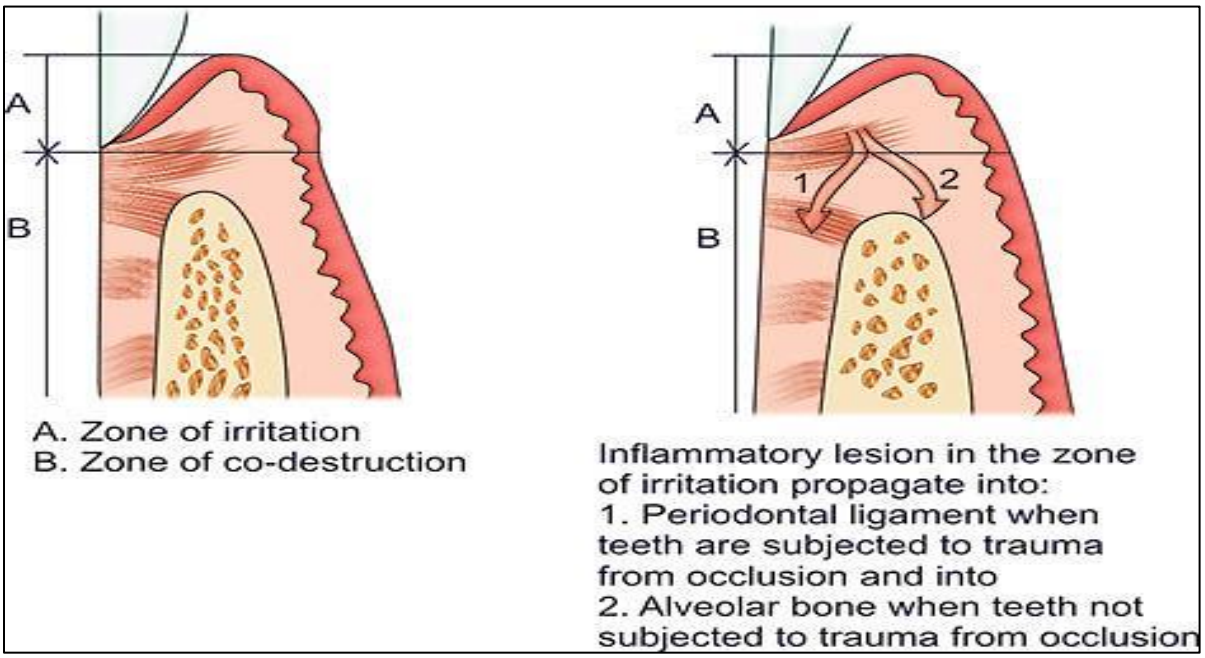

Fig.:- Glickman's concept

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
- ☒ Fractures, chipping tooth.

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.
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 & TMJ problems remain or worsen.