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INTRODUCTION

Occlusal trauma has been associated with periodontal disease for over 100years. In 1917 and 1926 stillman indicated that excessive occlusal forces must be controlled in order to prevent and treat periodontal disease.

The periodontium tries to accommodate to the forces exerted on the crown. This adaptive capacity varies in different persons and in the same person at different times. The effect of occlusal forces on the periodontium is influenced by the magnitude, direction, duration and frequency of the forces

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

Change in the direction of occlusal forces causes a reorientation of the stresses and strains with in the periodontium. The principal fibres of the periodontium are

arranged so that they best accommodate occlusal forces along the long axis of the tooth. Lateral (horizontal) forces 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 to the periodontium.

SYNONYMS: 1) Occlusal trauma
2) Traumatogenic occlusion
3) Overload
4) Occlusal disharmony
5) Periodontal traumatism.
6) Traumatism.
7) Functional imbalance
8) Occlusal dystrophy.

DEFINITION: when the occlusal forces exceed the adaptive capacity of the tissues, tissue injury results. The resultant injury is termed "trauma from occlusion". (Carranza)

"Injury resulting in tissue changes with in the attachment apparatus as a result of occlusal forces (Annals of periodontology 1999; 4: 102-107)

"Pathologic alterations or adaptive changes with which develop in the periodontium as a result of undue forces produced by the masticatory muscles" is termed trauma form occlusion".

"A condition where injury results to the supporting structures of teeth by the act of bringing the jaws into a closed position" (Stillman 1917).

"Defined as damage in the periodontium caused by stress on the teeth produced directly or indirectly by teeth of the opposing jaw" (WHO 1978).

An occlusion that produces such injury is termed "traumatic occlusion" (Bhaskar S.N, Orban, 1955).

Excessive occlusal forces disrupt the function of the masticatory musculature and cause painful spasms, injure the TMJ or produce excessive tooth wear, but the term 'TFO' is used in connection with injury in the periodontium.

TFO is divided into—

I) a. Acute TFO
b. Chronic TFO
II) a. Primary TFO
b. Secondary TFO

A) ACUTE TFO: results from an abrupt occlusal impact, such as that produced by biting on a hard object (e.g. an olive pit), restoration or prosthetic appliances that interfere with or alter the direction of occlusal forces on the teeth may induce acute trauma.

This result in—tooth pain

---sensitivity to percussion

---increased tooth mobility

---- Cemental tears.

If the force is dissipated by a shift in the position of the tooth or by wearing away or correction of the restoration, the injury heals and symptoms subside.

If not treated periodontal injury may worsen and develop into necrosis accompanied by the periodontal abscess formation or remain as a symptom free chronic condition.

a) **CHRONIC TRAUMA FROM OCCLUSION:** develops from gradual changes in occlusion produced by tooth wear, drifting movement and extrusion of teeth, combined with Para functional habits, e.g. bruxism and clenching, rather as a sequlae of acute periodontal trauma. Chronic TFO is more common than acute form and is of greater clinical significance.

The criterion that determines whether an occlusion is traumatic is whether it produces periodontal injury, not how the teeth occlude. Any occlusion producing periodontal injury is traumatic. Malocclusion is not necessary to produce trauma, periodontal injury may occur when the occlusion appears normal.

Traumatic occlusal relationships are referred by such terms as occlusal disharmony, occlusal dystrophy and functional imbalance. Trauma from occlusion may be caused by alterations in occlusal forces, reduced capacity of the periodontium to withstand occlusal forces or both.

PRIMARY TRAUMA FROM OCCLUSION: it is defined as "when the TFO is the result of alterations in occlusal forces is called primary TFO" (Carranza).

Or as "injury resulting in tissue changes from excessive occlusal forces applied to a tooth or teeth with normal support" (periodontology 2000; vol 2004).

It occurs in the presence of—

1. Normal bone levels
2. Normal attachment levels
3. Excessive occlusal force.

Etiology: - -- insertion of a high filling

--insertion of a prosthetic appliance that creates excessive forces on abutment and antagonistic teeth.

--the drifting movement/extrusion of teeth into spaces created by unreplaced missing teeth

--the orthodontic movement of teeth into functionally unacceptable positions.

Changes produced by primary TFO do not alter the level of connective tissue attachment and do not initiate packet formation. As the supracrestal gingival fibers are not affected and therefore prevent apical migration of junctional epithelium.

SECONDARY TRAUMA FROM OCCLUSION: "occurs when the adaptive capacity of the tissues to withstand occlusal forces is impaired by bone loss resulting from marginal inflammation" (Carranza).

Or it can be defined as "injury resulting in tissue changes from normal or excessive occlusal forces applied to a tooth or teeth with reduced support".

It occurs in the presence of-

1) Bone loss
2) Attachment loss
3) Normal/excessive occlusal forces.

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.

Systemic disorders can reduce tissue resistance and previously tolerable forces may become excessive.

Traumatic forces can occur on---

A) Normal periodontium with normal height of bone
B) Normal periodontium with reduce height of bone
C) Marginal periodontitis with reduced height of bone.

TISSUE RESPONSE TO INCREASED OCCLUSAL FORCES:

Stages of tissue response:--

Tissue response occurs in three stages.

Stage I---Injury
Stage II—Repair
Stage III---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, the periodontium is remodeled to cushion its impact. The ligament is widened at the expense of the bone resulting 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, in single rooted teeth is located at the junction between middle third and apical third of clinical root. It creates areas of tension and pressure on opposite sides of the fulcrum. Different lesion is produced by different degrees of pressure and tension. If jiggling forces are exerted, these different lesions may co-exist in the same area.

Jiggling forces: in humans, the occlusal forces act alternatively in one and the in the opposite direction. Such forces have been termed jiggling forces.

There is combination of pressure and tension on both sides of the jiggled tooth. For example, second premolars exposed to juggling forces are represented here. The combined pressure and tension zones (encircled areas) are characterized by signs of acute inflammation including collagen resorption, bone resorption and cementum resorption.

As a result of bone resorption, periodontal space increases gradually on both sides and in apical region. When the effect of force applied has been compensated by the increased with of periodontal space, the ligament tissue shows no signs of inflammation.

The supraalveolar connective tissue is not affected by jiggling forces and there is no apical down growth of dentogingival epithelium. After occlusal adjustment width of periodontal ligament is normalized and teeth are stabilized. This reveals with certain limits, a healthy periodontium with reduced height has a capacity similar to that of periodontium with normal height to adapt to altered functional demands.

The area of periodontium most susceptible to injury from excessive occlusal forces is the furcation (Glickman, 1961). Injury to the periodontium produces a temporary depression in mitotic activity, the rate of proliferation and differentiation of fibroblasts. In collagen and bone formation. These return to normal after dissipation of forces.

Slightly excessive pressure: stimulates resorption of the alveolar bone, with a resultant widening of the PDL fibers and apposition of 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 in the PDL, 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 the area. Vascular changes are also produced within thirty minutes, retardation and stasis of blood flow occur at 2to 3 hours, blood vessels appear to be packed with erythroblasts which start to fragment; and between 1 and 7 days, disintegration of the blood vessels 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 PDL, thrombosis, hemorrhage, tearing of the PDL, and resorption of alveolar bone.

Pressure severe enough to force the root against bone: causes necrosis of the PDL and bone. The bone is resorbed from viable PDL adjacent to necrotic areas and from marrow spaces, a process called “undermining resorption”.

Stage II—Repair: repair is constantly occurring in the periodontium and TFO stimulates increased reparative activity. In the process of repairing

the damaged tissues are removed. New connective tissue cells, fibers, bone and cementum are formed in an attempt to restore the injured periodontium.

Forces remain traumatic, 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” and is an important feature of the reparative process associated with TFO. 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). In central buttressing the endosteal cells deposit new bone, which restores the bony trabeculae and reduces the size of the marrow spaces. The 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, referred to as lipping or a pronounced bulge in the contour of the facial and lingual bone. Cartilage like material sometimes develops in the PDL space as an aftermath of the trauma. Formation of crystals from erythrocytes has also been shown.
Stage---III Adaptive remodeling of the periodontium: of 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 result in a thickened PDL, which is funnel shaped at the crest and angular defects in the bone, with no pocket formation. The involved teeth become loose. Increased vascularization has also been reported. These three stages in the evolution of traumatic lesions have been differentiated histometrically by means of the relative amounts of periodontal bone surface undergoing resorption or formation.

The injury phase shows and increased in areas of resorption and increased bone formation. After adaptive remodeling of the periodontium, resorption and formation return to normal.

TISSUE CHANGES CAUSED BY PERIODONTAL TRAUMATISM

Periodontal ligament: in acute phase of trauma, there is compression on the pressure side of the PDL, crushing, hemorrhage, thrombosis and necrosis may occur. Stretching takes place on tension side, this may cause thrombosis of the vessels and tearing of ligament.

In chronic phase PDL widens which increases mobility, hyalinization and a subsequent formation of fibrocatilage takes place. Ankylosis may also occur.

Cementum

In acute phase cemental tears and fractures, cemental and dentinal resorption can occur.
In chronic phase—Cemental hyperplasia

--- Formation of cemental spurs

---Resorbed areas repaired by cemental apposition can occur.

Alveolar bone

To moderate pressure---resorption will occur

To moderate tension---apposition will occur

In acute phase; necrosis of bone may occur in compression areas along with funnel shaped ligament.

Gingiva

No evidence of gingival changes has been notices. Early explanation for marginal festooning and gingival clefts was occlusal traumatism and impingement on the blood supply reaching the gingiva through PDL.

Pulp

1. Odontoblastic activity may be stimulated and secondary dentin may be formed.
2. Pulp chamber and canal may become narrower and even obliterated
3. Pulp stones may be formed
4. In some cases pulpitis and loss of pulpal vitality may occur.

EFFECTS OF INSUFFICIENT OCCLUSAL FORCE

Insufficient occlusal forces may also be injurious to the supporting periodontal tissues. Insufficient stimulation cases thinning of the PDL atrophy of the fibers, osteoporosis of the alveolar bone, and reduction in bone height. Hypo function can result from an open bite relationship, an absence of the functional antagonists, or unilateral chewing habits that neglect on side of the mouth.

REVERSIBILITY OF TRAUMATICV LESIONS

TFO is reversible. When trauma is artificially induced, the teeth move away or intrude into jaw, when force is relieved, the tissue undergoes repair. Through it is reversible, it does not always correct itself, the injurious force must be relieved for repair to occur, if not periodontal damage persists and worsens. The presence of inflammation in the periodontium due to plaque accumulation, may impair the reversibility of traumatic lesions.

EFFECTS OF ECESSIVE OCCLUSAL FORCES ON DENTAL PULP

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

INFLUENCE OF TFO ON PROGRESSION OF MARGINAL PERIODONTITIS

The accumulation of bacterial plaque that initiates gingivitis and results in periodontal pocket formation affects the marginal gingiva, but TFO occurs in the supporting tissues and does not affect the gingiva. The marginal gingiva is unaffected by TFO as its blood supply is sufficient to maintain it, even when the vessels of the periodontal ligament are obliterated by excessive occlusal forces. TFO does not cause pocket or neither gingivitis

nor it increased GCF flow, mobile teeth in human harbor significantly higher proportion of campylobacter rectus and Peptostreptococcus micros than nonmobile teeth.

When the inflammation extends from the gingiva into the supporting periodontal tissues (that is when gingivitis becomes periodontitis), plaque induced inflammation enters the zone influenced by occlusion, which Glickman has called the "zone of co-destruction".

Glickman's concept (1965-1967): Glickman claimed that the pathway of the spread of plaque associated gingival lesion can be changed if forces of an abnormal magnitude are acting on teeth harboring subgingival plaque. He says that character of the progressive tissue destruction of the periodontium at a "traumatized tooth" will be different from that characterizing a "non-traumatized tooth".

Instead of even destruction (that is, suprabony pocket and horizontal bone loss) at uncomplicated plaque-associated lesion, sites exposed to abnormal occlusal force will develop angular bony defects and infrabony pocket.

As Glickman's concept regarding TFO is often cited, a more detailed presentation of this theory is pertinent. The periodontal structures can be divided into two zones"

1. Zone of irritation_ from inflammatory lesion by plaque
2. Zone of co-destruction-from trauma induced changes.

1) **ZONE OF IRRITATION**: includes marginal and interdental gingiva. Soft tissue of this zone is bordered by hard tissue (the tooth) on one side and is not affected by forces of occlusion. This means gingival inflammation cannot be induced by TFO but is the result of microbial plaque.

The plaque associated lesion at a "non-traumatized tooth propagates in apical direction by first involving the alveolar bone and only later the PDL area. This progression of lesion will result in the even (horizontal) bone destruction.

2) **ZONE OF CO-DESTUCTION**: includes PDL, root cementum and alveolar bone and coronally demarcated by transseptal (interdental) and dentoalveolar collagen fiver bundles, the tissue in this zone may become the seat of lesion caused by TFO.

The fiber bundles which separate the zone of co-destruction from zone of irritation can be affected by two different directions.

a) Form the inflammatory lesion maintained by the plaque in the zone of irritation
b) From the trauma induced changes in the zone of co-destruction.

Through this exposure from two different directions the fiber bundles may become dissolved and oriented in direction parallel to root surface. The spread of an inflammatory lesion from the zone of irritation directly down into periodontal ligament and not via interdental bone, results in the direction of angular defects.

Glickman (1967) in a review paper stated; TFO is an etiologic factor (co destruction factor) to importance in situations where angular bony defects combined with infrabony pockets are found at one or several teeth.

WAERHAUG'S CONCEPT

Conducted autopsy specimens similar to Glickman but in addition measured the distance between the subgingival plaque, the periphery of associated inflammatory cell infiltrate in the gingiva and the surface of the adjacent alveolar bone. He reported the hypothesis that TFO played a role in spread of gingival lesion into "zone of co-destruction". He concluded that angular bony defects and infrabony pockets occur when the subgingival plaque of one tooth has reached more apical level than the micro biota on the neighboring tooth and when the volume of alveolar bone surrounding the roots is comparatively large.

Waerhaug's observation support the findings presented by Prichard (1965) and Manson(1976) that pattern of loss of supporting structures is the result of interplay between the form and volume of alveolar bone and apical extension of microbial plaque on the adjacent tooth surfaces.

When TFO is eliminated, a substantial reversal of bone loss occurs, except in the presence of periodontitis. This indicates that inflammation inhibits the potential for bone regeneration. Thus it is important to eliminate the marginal inflammatory component in cases of TFO, because the presence of inflammation effects bone regeneration after the removal of the traumatizing contacts.

TFO also tends to change the shape of alveolar crest. Change in the shape consists of widening of marginal PDL space, narrowing of interproximal alveolar bone, a shelf like thickening of the alveolar margin

So TFO does not alter inflammation process, it changes the architecture of area around the inflamed site. Thus in absence of inflammation, response of TFO is limited to adaptation of increased forces.

However, in presence of inflammation, the changes in the shape of alveolar crest may be conducive to angular bone loss and existing pockets may become intrabony.

CLINICAL SIGNS OF TFO

May include one or more of the following

1) Mobility (progressive)
2) Occlusal discrepancies
3) Fremitus
4) Wear facets
5) Tooth migration
6) Fractured tooth/teeth
7) Thermal sensitivity

8) Pain in TMJ
9) Recession.

MOBILITY

The most common clinical sign is increased mobility in the case of TFO. In injury state destruction of PDL fivers occur that results in increased mobility of tooth. In final stage, accommodation of periodontium to increased forces results in widening of PDL, which also leads to increased tooth mobility.

Though this mobility is greater than the so called normal mobility, it cannot be considered pathologic because it is an adaptation to altered functional demand and not due to disease process. When it becomes progressively worse, it can be considered pathologic.

Other causes for tooth mobility are

1. Advanced bone loss
2. Inflammation of PDL
3. Osteomyelitis of jaw tumors, abscesses, cysts
4. PDL surgeries
5. Pathologic migration
6. Orthodontic treatment.

FREMITUS

Definition: "fremitus is a measurement of the vibratory patterns of teeth where the teeth are placed in contacting positions and movements".

After placing damp finger the patient is asked to tap the teeth together in the maximum intercuspal position and then grind systematically in lateral, protrusive movements and position. The teeth that are displaced by the patient in these jaw positions are then identified.

Classification: class I—mild vibration or movement detected.

Class II—easily palpable vibration but not visible.

Class III—movement visible with naked eye.

RADIOGRAPHIC SIGNS OF TFO

1) Increased width of PDL space
2) Thickening of lamina dura in apical region and bifurcation areas.
3) A vertical rather than horizontal destruction of bone loss in interdental septum.
4) Radiolucence and condensation of alveolar bone.
5) Root resorption.

TREATMENT OF TFO

1) Selective grinding/occlusal therapy
2) Occlusal bite guard e.g. night guards
3) Orthodontic treatment
4) Splinting
5) Prosthetic reconstruction
6) Extraction of extruded teeth.