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TRAUMA FROM OCCLUSION-AN ENLIGHTENING ENCHIRIDION

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ABSTRACT

A fleeting century has elapsed since the pioneering publication of Karolyi elucidating the panjandrum that occlusal forces exert over the periodontal tissues; yet lucidity still clouds over the explicit bearing that occlusion has on myriad states of periodontal health and disease. With the advent of occlusion into the boulevard of traumatism, some studies profess that initiation and progression of periodontal disease might ensue, while conflicting studies abnegate the same. The periodontium is in possession of a plethora of mechanoreceptors which function as a key sentinel heralding the regiment of neuromuscular co-ordination of the masticatory system. These mechanoreceptors in turn are reliant on diligently directed occlusal forces for their activation and erudite functioning. Any departure from the threshold of normalcy leave a profound impact on the health and functioning of periodontium. The denouement can culminate in turn in a state of masticatory dysfunction besides accelerating the propagation of pre-existing periodontal diseases. This review is an attempt to elucidate a cogent vade mecum on trauma from occlusion, right from tracing its history, classification, aetiology, molecular mechanisms, diagnosis and its impact on various structures of periodontium and implants.

KEYWORDS: Trauma from occlusion, periodontitis, occlusion, jiggling forces, yes-associated protein.

INTRODUCTION

Occlusal trauma is a lesion of the periodontal ligament, cementum and supporting alveolar bone, and it ensues excessive traumatic occlusal forces. It is a term used to elucidate the

pathologic alterations or adaptive changes which develop in the periodontium as a result of undue force produced by the muscles of mastication.[1]

Classification of Trauma from Occlusion

Glickman's classification (1953)

According to duration of cause.

i. Acute TFO

ii. Chronic TFO.

According to nature of cause

i. Primary TFO

ii. Secondary TFO.

Box's classification

Physiologic occlusion

It is defined as a condition, in which the systems of forces acting upon the tooth during the occlusion are in a state of equilibrium, and they do not and cannot change the normal relationship existing between the tooth and its supporting structures.

Traumatic Occlusion

The damage produced in the periodontium is due to the overstress produced by the occlusion and such an occlusion is termed traumatic.

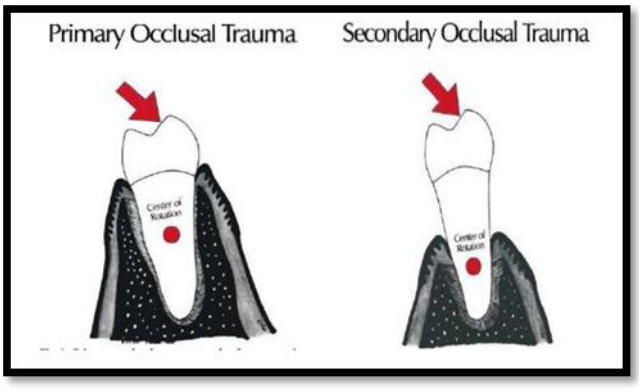

Figure 1: Primary and secondary occlusal trauma.

ETIOLOGICAL FACTORS

Ross has classified the factors causing chronic destructive periodontal disease into two groups.

a. Precipitating factors: Precipitating factors are the irritants and the destructive occlusal forces that further facilitate destruction of the tissues weakened by predisposing factors.

b. Predisposing factors: Factors which contribute to the histopathologic lesion and they are further enlisted as developmental factors, functional mechanisms, and the systemic component.[2]

Precipitating Factors

Destructive occlusal forces are the primary precipitating factors of trauma from occlusion. These forces are usually elucidated and characterized in terms of magnitude, direction, duration of application, and frequency of application.

• Magnitude: When the magnitude of occlusal forces exceeds the normally tolerated range of forces for a tooth, due to natural adaptive response, some morphological changes can be appreciated in the periodontal ligament (PDL) as exemplified by widening of the PDL space, an increase in the number and width of PDL fibers and an increase in the density of alveolar bone.

• Direction: The principal fibers of the PDL play a sapient role in withstanding the occlusal forces and transferring them to the alveolar bone. As the direction of occlusal forces is altered, the optimal orientation of the PDL fibers is also disrupted and they are thus rendered incapable of efficiently bearing the occlusal forces, and hence injury results.

• Duration of force application: If the tooth is subjected to abnormal occlusal forces for an extended duration of time, they cause injury to the periodontal apparatus, which can be detected histologically

• Frequency of force application: Frequent application of abnormal occlusal forces inflicts greater damage to the periodontium.

Predisposing Factors

They are classified as intrinsic and extrinsic factors.

Intrinsic Factors

• The orientation of the long axis of the teeth in relation to the forces that act on them. Axially inclined forces are more tolerable than nonaxially inclined forces irrespective of whether it is functional or parafunctional. With malaligned teeth, the effect of excessive force are particularly deleterious.

• The morphological characteristics of the roots: the size, shape, and number of the roots determine how the occlusal forces thrust are dissipated. In general, short, conical, slender, or fused roots are considered to be more vulnerable to TFO.

• The morphology of the alveolar process, i.e., the quality and the quantity of the alveolar bone play an important role in bearing the brunt of the occlusal forces.

Extrinsic Factors

• Irritants: Microbial plaque, food impaction that results in positive pressure on the tissues, overhanging restorations, improperly contoured crowns and bands, and ill‑fitting partial denture clasps.

• Neuroses that result in parafunctional activities like bruxism. These factors are the most pertinent causes of abnormal occlusal stresses.

• Loss of supporting bone: Periodontitis, injudicious bone resection, inadvertent trauma, and systemically related diseases leading to loss of bony support are the chief causative factors.

• Loss of teeth resulting in disproportional overloading of the remaining teeth, for example, posterior bite collapse. The effects of tooth loss are not as a rule restricted to the immediate vicinity of the edentulous region. As a result of loss of first permanent molars, there are occlusal discrepancies which in turn lead to increased slide from centric relation to centric occlusion; the anterior teeth hit each other with increased force during mastication. This functional disharmony causes labial drifting of anterior teeth, thereby resulting in an open contact relationship of the anterior segment. The next catastrophic phase usually is further bite collapse and loss of alveolar bone.[2]

• Iatrogenically induced functional malocclusion.[3]

• Injudicious periodontal surgery: Loss of alveolar support caused by either periodontal disease or surgical interventions to correct defects may seriously aggravate occlusal traumatism. In many severe cases, the benefits that pocket elimination by bone resection stand to offer must be weighed carefully against the risk that ensues in terms of decrease in support. In such situations, functional forces previously within a physiologic range may become excessive and irreversible breakdown may occur. The most significant factor in determining whether bone resection will predispose to occlusal traumatism or not is the amount and location of bone loss around the tooth before surgical intervention.[2]

Acute occlusal trauma is most often sequalae of precipitous impact of immoderate masticatory forces (e.g. chewing on an olive pit). It is accompanied by acute pain, percussion

sensitivity and increased tooth mobility, fremitus, sensitivity to thermal irritation, tooth fracture. Chronic occlusal trauma on the other hand is an upshot of changes in periodontal tissues associated with gradual discrepancies in occlusion due to tooth abrasion, migration and extrusion of teeth in combination with parafunctional factors (e.g. bruxism) and non-carious cervical lesions. In radiographic examination it is characterized by hour glass shaped widening of the periodontal ligament space and root resorption, as well as angular bony defects. Vertical bone loss synonymous with traumatic occlusion is neither accompanied by loss of clinical attachment level and periodontal pocket formation in the dearth of plaque induced inflammation nor is there an increase in gingival crevicular fluid.

Trauma from occlusion can lead to change in the morphology of the alveolar ridge secondary to adaptation to the so-called jiggling forces—pressure and tension on either side of the tooth exemplified by corresponding histological changes. Occlusal trauma is reversible as the health of the periodontium is restored when the pathology is eliminated. However, in cases of plaque-induced periodontal inflammation the presence of secondary occlusal trauma perpetuates and aggravates the condition, steering it away from the helms of reversibility.

Causes of primary occlusal trauma

- Localized, primary occlusal trauma most typically is a result of a “high” restoration, a common sequela of the placement of a new restoration in an individual tooth that has been extensively instrumented under anaesthesia in a patient whose mouth has been opened wide for a protracted interval of time thus rendering him unable to detect such an early deflective contact. Selective grinding usually suffice for correcting the problem; however, severe discrepancies may best be resolved by replacing the restoration.
- Malaligned teeth also may be subjected to primary occlusal trauma, especially when they are deviated and locked in facio- or linguoversion. Though selective grinding may resolve minor malalignments, orthodontic movement is often the definitive treatment choice. In severe crowding, however, selective extraction of teeth may be an alternative yet simple and satisfactory remedy.
- Generalized, primary occlusal trauma has a distinct etiology :clenching and grinding habits including bruxism (night grinding). Occlusal adjustment may resolve some problems; however it is also imperative to address the accompanying psychological components which deems a referral to psychiatric counselling. As the process of

resolution thus progresses, an occlusal splint (night guard) can be used to correct the damage ensuing grinding in the interim.

- Another less common etiology is "occupational" bruxism/clenching, wherein certain occupations cause patients to exert undue occlusal stresses onto their teeth. Changing jobs may not be a feasible option for the patient; however, an occlusal splint often can be worn during work hours and additionally, biofeedback has also been demonstrated to be beneficial in aiding the patient to become cognizant of these parafunctional habits.
- Recreational bruxism is a term used to describe the extreme gnashing of teeth that accompanies the use and abuse of some "recreational" drugs. The obvious solution to this malady is discontinuation of the drugs which offers wide ranging benefits beyond the scope of periodontology. Counselling may be of assistance in correcting such habits. Occasionally, an occlusal splint may be helpful if the patient is unable to ascend on the path of drug withdrawal.
- Post-orthodontic clenching may occur when a major occlusal discrepancy occurs following the completion of orthodontic therapy. A so-called double bite may be created if the patient has a maximal intercuspation position a half tooth or more ahead of a centric relation occlusion. As the patient navigates between these two positions, severe trauma usually occurs. If it is detected prior to the inception of periodontitis, occlusal adjustment may be somewhat helpful; however, the discrepancies often are too great to be ground out thus warranting the need for further orthodontic treatment or even orthognathic surgery for complete resolution. Though a night guard may be useful in controlling damage; a lifetime of wearing such a device is rarely a palatable alternative. Full mouth reconstruction to establish a stable, centric occlusion is another alternative.

Causes of secondary trauma from occlusion

Secondary occlusal trauma occurs when a tooth has effacement of attachment and bone support to the extent that trauma to the periodontal attachment apparatus occurs even with physiologically normal occlusal loading. The main perplexity associated with maintenance of periodontally compromised teeth subjected to secondary occlusal trauma is that bone loss inevitably occurs and it can be a source of masticatory discomfort. At some point, the clinician needs to consider strategic extraction so that adequate bone volume may be preserved to facilitate implant placement and the eventual rehabilitation with an implant supported prosthesis.

- Localized secondary occlusal trauma has a better prognosis relative to generalized secondary occlusal trauma. If the adjacent teeth have a sound support, selective grinding of the tooth that has lost support may minimize function on the tooth by delegating the occlusal load on to the teeth with good support. If good abutment teeth are available, the tooth that has lost support may be splinted with fixed bridgework to the abutments, thus alleviating the ostensibly arduous load on the weakened tooth. The greater the number of remaining periodontally sound abutment teeth, the better the prognosis. In situations where trauma exists, but the mobility is exiguous, the tooth can be relieved out of occlusion and may respond to regenerative procedures. Alternatively, if secondary mobility cannot be controlled, patient comfort may warrant strategic extraction.
- If no good adjacent abutment teeth remain, but the compromised tooth is amenable to periodontal regeneration, the formerly elucidated approach cannot be employed because the tooth cannot be stabilized; therefore, the tooth should either be extracted or maintained with full recognition that its prognosis is hopeless.
- If generalized secondary occlusal trauma is present, permanent splinting of all teeth with fixed bridgework may be sufficient to facilitate stabilization by engaging all the compromised teeth together as a unified unit. This approach is strictly contraindicated if all teeth have extensive bone loss. In such heavily involved generalized cases, temporary splinting may allow for interim maintenance of the teeth for shorter periods. Another alternative is the placement of an occlusal splint, especially if the patient is in possession of the habit of nocturnal grinding. As periodontal regeneration on large numbers of teeth is not currently practical or predictable; extraction is the only reasonable alternative if teeth have lost substantial support.[4]

The forces exerted on the periodontal structures may be in singular direction (orthodontic forces) or may be 'jiggling' forces. Forces in one direction cause tipping of the tooth in the opposite direction or tooth displacement parallel to the direction of application of force resulting in a 'bodily movement'.

In the periodontal ligament, zones of compression and zones of tension are detected histologically, inducing increased resorption. The clinical result is a transient increase in mobility. However, there are no changes in the orientation of supracrestal fibres, no loss of periodontal attachment, or an increased probing pocket depth. The increased tooth mobility reflects functional adaptation to the forces exerted on that tooth. If the forces surpass the

zenith of adaptation level, an aseptic necrosis in the tension zone of the periodontal ligament occurs, characterised by hyalinisation, bone apposition and rupture of the collagen fibres also occur. In the compression zone, pressure stimulates osteoclasts in the adjacent bone and the alveolar wall is resorbed until a new connection is established with the hyalinised bone ('undermining resorption'). After removal of the force the periodontal ligament is reorganised and after some time develops a normal histological appearance. If the applied forces are too high, root resorption occurs in the middle of the hyalinised tissues and this continues for a variable time, resulting in shorter roots, frequently seen following orthodontic treatment.

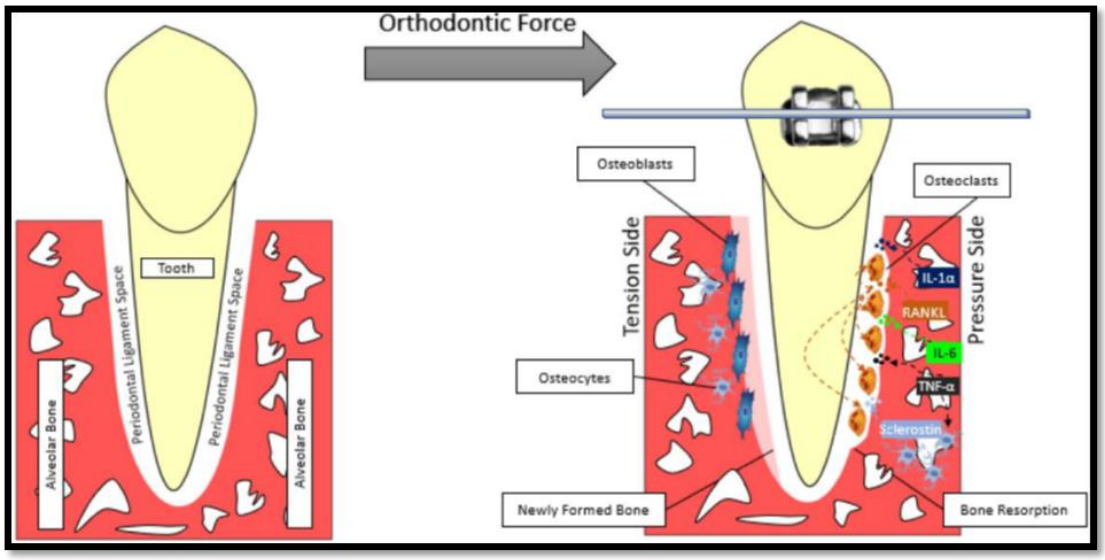

Figure 2: Biomechanics of tooth movement.

Jiggling forces, coming from different and opposite directions, mediate the manifestation of more complex histological changes in the ligament. Theoretically the same events (hyalinisation, resorption) occur, however, they are not clearly delineated as characterised by paucity of distinct zones of pressure and tension. Histologically, there is apposition and resorption on either side of the periodontal ligament, resulting in a widening of the periodontal ligament space.

The clinical phenomena accounting for increased mobility are not only dependent on the magnitude of the forces, but also on the crown-root relationship, the position in the arch, the direction of the long axis, and the pressure of tongue and orofacial musculature. Hypermobility is therefore not an insignia of an ongoing process, but may be the result of a previous jiggling forces. The long-term prognosis of teeth with increased mobility is poor, and is a complicating factor if the teeth are to be used as abutments in prosthodontic

reconstruction. Successful periodontal treatment leads to healthy but reduced periodontal structures and jiggling forces exerted on the teeth in this state of existence result in a pronounced increase in tooth mobility because the point of rotation (fulcrum) is closer to the apex than normal. This is distressing for the patient and might be an indication for splinting of teeth.[1] Secondary occlusal trauma comprises tissue alterations due to normal or excessive occlusal forces in a tooth or teeth which is housed in a compromised periodontium. Occlusal trauma does not cause periodontitis, but it can accelerate periodontal destruction in the presence of pre-existing periodontal inflammation.[5]

Occlusal disease is analogous to periodontitis in that it is generally not reversible (an exception for periodontitis might be osseous grafting), however, similar to periodontitis, is often maintainable. It does however render itself amenable to treatment and when restorative dentistry is utilized, it becomes, in that sense, reversible. Forces caused by occlusal trauma could reduce perfusion of the PDL, resulting in ischemia and necrosis of the PDL cells when the adaptive capacity of the PDL is exceeded. In this case, bacterial inflammation of the periodontium could go unchecked and progress faster because of the lower tissue resistance or tissue integrity.

Hence, TFO dons the role of a catalyst for the destructive processes initiated by bacterial periodontal inflammation.[3] Occlusal therapy is an integral component of periodontal treatment and can lead to reduction of tooth mobility and enhancement of patients' comfort and their masticatory function. It can decelerate the progression of periodontal disease and improve the overall prognosis.[5]

TRAUMA FROM OCCLUSION AND PERIODONTITIS-A BRIEF HISTORICAL OVERVIEW

AUTHOR	DEFINITION
Stillman (1917)[6]	Trauma from occlusion as a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position.
Glickman (1974)[7]	As the injury that results when the occlusal forces exceed the adaptive capacity of the tissues
WHO (1978)[8]	Defined it as damage in the periodontium caused by stress on the teeth produced directly or indirectly by the teeth of the opposing jaw.
Lindhe, Nyman, Ericsson (1978)[9]	"Pathologic alteration or adaptive changes which develop in the periodontium as a result of undue forces produced by masticatory muscles".
"Glossary of	Christened it "Occlusal trauma" and defined it as "An injury to the

periodontics terms" American Academy of Periodontology (2001)	attachment apparatus as a result of excessive occlusion forces".

In 1901, **Karolyi et al** posited a relationship between "trauma from occlusion" and "alveolar pyorrhea" and this in turn marked the commencement of the chapter of trauma from occlusion in the realm of periodontology**. Box in 1935 and Stones in 1938** undertook experiments in sheep and monkeys to validate the promulgations of Karolyi and subsequently ascertained that in teeth displaying periodontitis with vertical pocket formation, trauma from occlusion could be held culpable of causing the same. However, the results thus obtained were marred with fact that in these experiments there were no controls and also the design of these experiments could not veritably produce a justifiable conclusion.

Glickman's concept

Glickman (1965, 1967) proclaimed that the trajectory of spread of plaque- associated gingival lesion might be altered when occlusal forces of exceeding magnitude act on the diseased tooth. Extrapolating his findings thus, he claimed that in such teeth subjected to the dual action of occlusal stress and plaque associated sequalae, what ensued was infrabony pocket coupled with angular bone loss as an upshot of the uneven characterisation of periodontal destruction.

The periodontal structures were divided into two zones by Glickman as follows.

1. Zone of irritation

2. Zone of co-destruction

The zone of irritation encompasses the marginal and interdental gingiva. The soft tissue of this zone, by virtue of being bordered by hard tissue (the tooth) only on one side, are effectively emancipated from being devoured by abnormal occlusal forces. Any lesion in this zone is thus indelibly ascribed to plaque and the pathogenicity of its inhabitants. In non-traumatized tooth, any lesion thus incurred in this zone, propagates in an apical direction, first to the alveolar bone and later to the periodontal ligament thus resulting in horizontal bone loss.

The zone of co-destruction refers to cementum, alveolar bone and the soft tissue wedged between these two mineralized tissues, the cardinal periodontal ligament. The coronal extent of this zone is bordered by the trans-septal fibers and dentoalveolar collagen fibre bundles. Its

location and topography thus renders it highly vulnerable to the ramifications of traumatic occlusal forces.

The fiber bundles thus serving as the common denominator separating these adjacent zones could be affected from two directions as stated below.

1. From the inflammatory gingival lesion emanating from the zone of irritation
2. From trauma-induced changes taking root from the zone of co-destruction.

Having thus been ambushed from both the directions, the fibre bundles might be dissolved and take on an altered orientation aligning themselves parallel to the tooth surface. Boundaries thus dissolved, the gingival lesion from the zone of irritation directly spreads int the periodontal ligament taking a detour from its erstwhile path of navigation involving a pitstop at the alveolar bone prior to extending to the terrain of PDL. The result thus was angular bone loss and infrabony pockets thus ascertaining the role of trauma from occlusion as a co-destructive factor in the development of periodontitis.

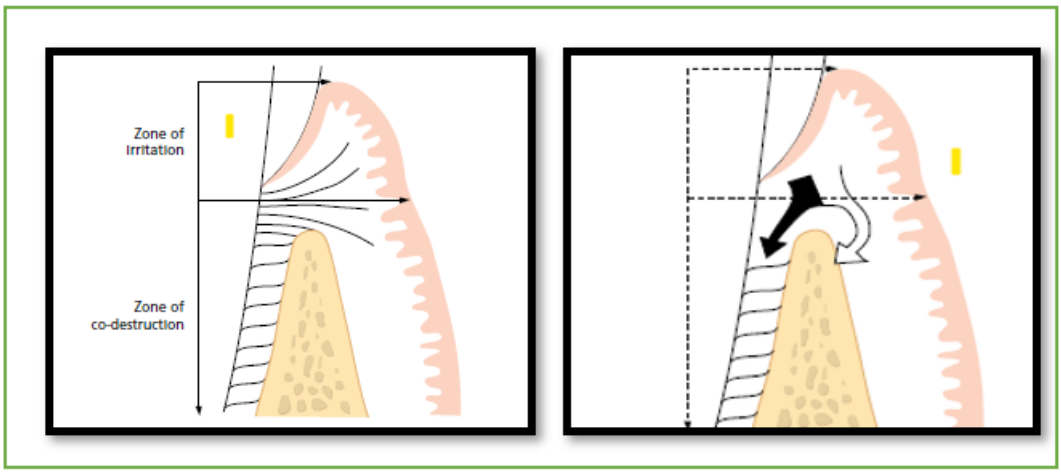

Figure 3: Glickman's concept.

Waerhaug's concept

Waerhaug (1979) examined autopsy specimens confluent to Glickman, but supplemented the same with additional measurements as follows.

the distance between the microbial plaque and (1) the periphery of the associated inflammatory cell infiltrate and (2) the surface of the adjacent alveolar bone.

He concluded from his measurements that angular bony defects and infrabony pockets showed no disharmony in terms of occurrence occur in teeth subjected to trauma from

occlusion and those abnegated from the same. Backed by his observations thus, he vehemently disregarded Glickman's hypothesis. The attachment loss and alveaolar bone loss in teeth, according to Waerhaug, was solely attributed to subgingival plaque associated inflammatory lesion and he was further of the view that angular bone loss and infrabony pockets manifested when there was a discrepancy in the apical extension of plaque between two adjacent teeth and when the alveolar bone housing the roots of the teeth had a substantially large volume.[10] Waerhaug found support in **Prichard (1965) and Manson(1976),** who diligently reaffirmed his affirmation stating that the pattern of loss of supporting structures is the consequence of an interplay between the form and volume of the alveolar bone and the apical extension of the microbial plaque on the adjacent root surfaces.[9]

Animal studies

The most significant animal studies were performed in the 1970s by two research group, one at **Eastman Dental Centre in Rochester, New York by Polson** et al wherein they refuted claims that trauma from occlusion could potentially inflict damage upon the periodontium.; and the other at the **University of Gothenburg in Sweden by Lindhe et al** whose experiments in beagle dogs led him to conclude that jiggling forces did not result in connective tissue breakdown in animals with normal gingiva or marginal gingivitis; and that occlusal trauma resulted in increased tooth mobility and accelerated destruction of periodontal structures in animals with periodontitis. These studies are often fondly referred to as the American and the Scandinavian occlusal studies, respectively.[9]

Human studies

Burgett et al, having studied the upshot of occlusal adjustment as a comprehensive part of periodontal therapy surmised that those patients who received combination therapy as opposed to those bereft of occlusal adjustment, an additional 0.5 mm gain of attachment was noted.[11]

As part of a large study on prognosis, **McGuire and Nunn** reviewed the change in prognosis and in the number of teeth lost by patients with periodontal disease in possession of parafunctional habits. They observed that those patients with parafunctional habits who were precluded from being given an occlusal appliance demonstrated no escalation in the assigned grade of prognosis despite receiving periodontal therapy.[12,13]

The consensus of the **1996 World Workshop in Periodontics** indicated that there was a lacuna of credibility in the available literature to gain lucidity over the proposed relationship between occlusion and periodontal disease progression.[14]

The **1999 consensus report** however conceded that occlusal trauma resulted in tissue changes resounding injury within the periodontal attachment apparatus. This report was also of the view that occlusal trauma could not independently initiate periodontal disease in the absence of plaque.[15]

Nunn and Harrel (2001) stated there existed a formidable association between occlusal discrepancies and the myriad clinical parameters widely employed to determine the state of health or disease of the periodontium.[16,17]

DIAGNOSIS OF TRAUMA FROM OCCLUSION

Radiographic signs

1. Widening of the PDL space, often accompanied by thickening of the lamina dura.
2. Vertical destruction of the interdental septum resulting in the genesis of infrabony defects
3. Radiolucency and condensation of the alveolar sockets
4. Root resorption

Clinical signs

Box and Stillman implicated TFO as the causative factor for the following signs of incipient periodontal disease.

1. Traumatic crescent – A crescent‑shaped bluish red zone detected in gingiva confined to about one‑sixth of the circumference of the root.
2. Recession of the gingiva, which is often asymmetrical and found to be present in associated with resorption of the alveolar crest.
3. Stillman's clefts – Apostrophe shaped indentations extending from and into the gingival margins found to occur generally on the facial surface of the tooth.
4. McCall's festoons – Discrete life preserver shaped semilunar enlargement of the marginal gingival more predisposed to occur in the canine premolar area.
5. Teeth mobility in traumatic occlusion which is distinguishable from the one in periodontitis as its incidence here is bereft of the occurrence of periodontal pocket.
6. Joint signs – Arthralgia, stiffness of TMJ, joint noises and radiological signs manifesting as narrowing of the joint space and presence of uneven radiotransparency, irregular osseous

contours, deformation of mandibular condyles, atrophy of articular tubercle, osteophytes and loss of the characteristic oval shape of mandibular condyles (arthrosis).

7. Muscular signs – Myositis, myospasm, localised myalgia, and myofibrotic contractions.

Symptoms

• Persistent discomfort upon mastication.

• Thermal sensitivity: Although the exact effect of occlusal trauma on dental pulp are still not clearly delineated, studies have demonstrated that there are changes in the pulp and hence tooth becomes abnormally sensitive to thermal changes.

• Muscle hypertonicity: The patient complains of jaw fatigue, especially after rising in the morning and at the end of the day. It is a consequence of the occlusal interferences inducing muscle spasm and discomfort.[3]

Because TFO is defined and diagnosed on the basis of histologic changes in the periodontal supporting structure, definitive diagnosis is impossible without block section biopsy. However diagnosis in clinical scenarios can be affirmed by assessing the dentition as follows.

1. Increased tooth mobility - assessed by mechanical and electronic instrument (Miller's classification).

2. Tilting and migration of individual tooth or of complete segments. In addition, the percussion of teeth on tapping with a blunt instrument changes from a resonant note in a tooth with a healthy supporting structure to a dull note if there is primary TFO.

3. Careful palpation of the muscles of mastication to ascertain whether there is hypertrophy or sign of hypertonicity with possible spasm of any of the muscles of mastication.

4. Palpation of TMJ and observation of any deviation of the mandible as it simulates the various movements enclosed in the envelope of its range of motion.

5. Fremitus test: Fremitus is a measurement of the vibratory patterns of the teeth when the teeth are placed in positions of contact and movements. A dampened index finger is placed along the buccal and labial surfaces of the maxillary teeth, and patient is asked to tap the teeth together in the maximum intercuspation and grind meticulously in the lateral, protrusive, and lateral protrusive contacting movements and positions. In general, this is limited to the maxillary teeth; however, in situations of edge-to-edge occlusion or when there is reduced overjet, mandibular teeth can also be assessed.[2]

TISSUE RESPONSE TO TRAUMA FROM OCCLUSION

Stage 1: Injury

Tissue damage is a result of injurious forces and depends on its magnitude, frequency, and direction. When subjected to the forces of occlusion, a tooth rotates around a fulcrum or axis of rotation that is located in single‐rooted teeth at the junction between the middle third and the apical third of the clinical root. This creates areas of pressure and tension on opposite sides of the fulcrum and different lesions are produced by pressure and tension variably although if jiggling forces are exerted they may coexist in the same area.[10] Slightly excessive pressure induces osteoclastic resorption of alveolar bone which in turn leads to widening of the PDL space. This area is then characterised by a decrease in the number of blood vessels. With further increase in pressure, the PDL fibres are subjected to compression, which leads to thrombosis of the blood vessels and hemorrhage. Subsequently, there is hyalinization and necrosis of the PDL fibres. Within 30 minutes, vascular stasis sets in, and within 2 to 3 hours, sequestration of erythrocytes are found which subsequently fragment and in the following 1-7 days fragmentation of the blood vessels ensued by the release of their contents into the surrounding milieu follows. When the pressure aggrandizes, undermining resorption follows. On the side of slightly excessive tension, the periodontal fibers elongate and there is apposition of alveolar bone with enlarged blood vessels being present in this area. Severe tension causes widening of PDL, thrombosis, stretching of PDL fibres to the point of tearing, and alveolar bone resorption. A transient diminution is seen in the mitotic activity and the rate of proliferation and differentiation of fibroblasts, collagen, and bone formation, which return to the state of normalcy following dissipation of forces.

Stage 2: Repair

Tissue repair is a constant and continuous process. The damaged tissues are removed, and new connective tissue cells and fibres, bone, and cementum are formed in an attempt to renew the injured periodontium. Forces will continue to be deemed traumatic as long as the damage produced exceeds the reparative capacity of the tissue. A sapient feature of reparative process is buttressing bone formation. When the bone is resorbed due to excessive occlusal forces, the thinned trabecular bone is reinforced with the new bone along the facial surfaces **(buttressing bone formation).** When the endosteal cells engage in bone formation resulting in increased trabeculae and reduced marrow spaces, it is referred to as central buttressing bone formation whereas deposition of bone peripherally results in altered facial or lingual

contours referred to as peripheral buttressing bone formation. The latter may sometimes produce a shelf-like thickening of the alveolar margin, referred to as **lipping.**

Stage 3: Remodelling of the periodontium

In order to create harmonious structural symphony between the ongoing destruction and repair, the periodontium is remodelled such that the forces are no longer injurious to the tissues. To cushion the impact of the offending forces, the PDL is widened and the adjacent bone is resorbed subsequent to which mobility sets in. The results are a widened PDL, which is characteristically funnel‑shaped at the crest, and angular defects in the bone. After the establishment of adaptive remodelling of the periodontium, the cycle of resorption and formation return to normal.[3]

TRAUMA FROM OCCLUSION AND ITS TRYST WITH FURCATION

Glickman et al (1964) was one of the erudite pioneers to posit that furcation areas rank high in terms of vulnerability to periodontal breakdown while being subjected to excessive occlusal forces and attributed this escalated propensity of the furcation to the characteristic orientation of PDL fibers in this area which in turn doubled as a low resistance checkpoint to curb the advancing inflammatory front. **Waerhaug** however strictly promulgated that tooth mobility was rather a late ramification rather than the etiology helming furcation invasion. Though enshrouded in enigma and awaiting a final verdict, it is believed that trauma from occlusion is suspected to orchestrate the occurrence of isolated furcation defects and it is only prudent to relieve any heavy occlusal forces on the molar, lest it might result in accelerated periodontal destruction.[18]

TRAUMA FROM OCCLUSION AND IMPLANTS

It is understood that the establishment and maintenance of balanced occlusal relationships is important both in natural dentition and in rehabilitation with osseointegrated implants.Endosseous implants have no periodontal ligament as an intimate implant-alveolar bone interface exists ('functional ankylosis'). Implant failure can occur not only because of bacterial infection (peri-implantitis) but also because of occlusal overload (biomechanical failure) working in cahoots with immunological host factors **(Esposito et al 1998).**

Occlusal overload results in 'osseodisintegration' over the complete implant surface in the absence of clinically detectable pocket formation or signs of inflammation. Clinical measurement of implant mobility is not an accurate and reliable tool for evaluating

osseointegration or disintegration of an implant until late in the pathological process.[1] Assessing the occlusal ability to detect occlusal interferences, it was found that the tactile perception of interference on natural teeth and implants with antagonists was approximately 20 and 48 μm, respectively. In another study, it was detected values of tactile perception for implants was 8.75 times higher (100.6 g) than that of the natural teeth (11.5 g). These results demonstrate that dental implants, devoid of the cushioning effect of the PDL, may be more susceptible to occlusal overload.[3]

GINGIVAL TRAUMA FROM OCCLUSION

Gingival recession may be elicited by direct impingement of the teeth on the gingiva, as in severe overbite, where the maxillary incisors damage the buccal gingiva of the mandibular incisors. This panacea for this may involve orthodontic treatment, orthognathic surgery or extensive prosthetic rehabilitation requiring the need to raise the vertical dimension. It has also been postulated that gingival recession occurs with functional overload and/ or premature contacts, as in the buccal surface of maxillary canines where there is a steep lateral canine guidance. The corresponding cervical surfaces also show enamel abfraction. There is still a looming controversy regarding whether or not overload is an aetiological factor in the recession, and consequently whether or not the occlusion and the lateral guidance should be altered.[1] Gingival recession and clefts can be attributed to various factors. **Stillman in 1971**, became the pioneer to chart an association between occlusal trauma and gingival clefts for the first time and it was thus christened **Stillman's clefts**. The unchartered territory of exploring the association between gingival clefts and occlusal trauma was demystified by **Solnit** when he revealed that gingival clefts underwent spontaneous repair following occlusal analysis and subsequent adjustment. He attributed the occurrence of gingival clefts in upper jaw to working side interferences and those in lower jaw to balancing side contacts. In direct contradiction to this proposition, **Emslie** found no substantial to bind gingival clefts to occlusal trauma.[19] **Parfit and Major** observed occlusal trauma in only 15% of the mandibular incisors presenting with recession.[20] **Trot and Love** found trauma from occlusion to coexist with gingival recession in only 10% cases[21] while **Gorman** declared a slightly higher percentage of observation of such association at 20%.[22] In a study conducted by **Krishna Prasad et al**, it was inferred that gingival recession was more commonly associated with group function (60%)than canine guided occlusion(40%). In subjects having canine guided occlusion, gingival recession was noted along the labial surface of the anteriors (75 %) whereas in group function occlusion, recession was equally prevalent on the facial

surfaces of the anteriors and posteriors.[19] There was a binding relation noted between mutually protected occlusion and the incidence of gingival recession and gingival clefts. When there was no mutually protected occlusion by extension , it is inferred that there is a dearth of disclusion in the anterior teeth during maximum intercuspation, and during protrusion there was no posterior disclusion.[23,24] All these situations constituted the envelope of pathological malocclusion which in turn adversely affected the occlusal surface of the teeth, periodontium, neuromuscular system and the temporomandibular joint.[25,26,27] This form of pathological malocclusion is also attributed to gingival recession and gingival clefts. Absence of anterior disclusion in maximum intercuspation can exert deleterious influence which may manifest as gingival recession along the labial surface of mandibular anteriors. Gingival clefts were generally seen on the maxillary teeth and they had a predilection to be located in the posterior region. The same occlusal interferences which were related to gingival recession were also implicated in the etiology of gingival clefts.[19]

The mechanism involved in the development of recession in response to occlusal overloading has been meticulously elucidated by **Consolaro.** Occlusal overloads generate pressure and tensional forces which in turn to the compression of periodontal fibers accompanied by the diminishing vascular diameter and subsequent disruption of fibers and periodontal cells. This results in the local influx of mediators that orchestrate the process of bone reconstruction. During the phase of primary occlusal trauma periodontal tissues adapt themselves to the excessive occlusal forces through the condensation of alveolar cortical bone, irregular extension of periodontal space and increased bone density, which is detected on the X-ray image. However, elevated and prolonged overload leads to an excessive stretching and compression of periodontal ligament differentially in the cervical region. This can cause a markedly increased level of mediators responsible for bone resorption, ultimately leading to bending and loss of lamina dura in the V shape. This observation is particularly made in the interdental space and along the buccal surface. The alveolar bone lining the buccal surface is frequently very thin. Its loss makes way for establishment of a nexus between the periosteum and periodontal ligament, producing an elongated connective tissue attachment. This modified and elongated connective tissue- epithelium apparatus can maintain the gingival level at the physiological height only for a certain period of time while being subjected to occlusal trauma, ultimately resulting in the genesis of decreased volume of tissues, tooth root dehiscence and the development of V-shaped recessions. This process exemplifies gingival tissue adaptation to the absence of osseous support. The decrease in the volume is

indemnified by a permanent physiological reconstruction of tissues, which is aimed at restoring right proportions between gingival sulcus epithelium, junctional epithelium, gingival connective tissue and alveolar bone. Consolaro reports that the V-shaped recessions are associated with occlusions contrary to the U-shaped recessions, which are sequalae of periodontitis or an improper tooth brushing method.[28]

YAP- UNEARTHING THE MOLECULAR PLAYER COUPLING OCCLUSAL TRAUMA AND PERIODONTITIS

Hippo pathway is an avant garde discovery and it exerts a marked panjandrum over intricate processes as in conserved cell signal transduction pathway, which in turn orchestrates regulation of organ size, tissue homeostasis and tumor development **(Zhao et al., 2011)**. The Hippo pathway is subjected to activation by myriad upstream signals such as G protein-coupled receptor signalling, mechanical forces, cytoskeletal signalling and so on. Phosphorylation of the downstream effector protein YAP by core kinases, effects an alteration in its intracellular distribution, regulating downstream gene transcription, further affecting various biological functions **(Gaspar and Tapon, 2014).** Serving as a cardinal stanchion holding down the bridge of mechanical stimulation, the Hippo pathway participates in mechanical transduction processes, regulation of cell morphology and various stress perceptions **(Aragona et al., 2013).** Several studies have revealed that the interaction between c-Jun N-terminal kinase (JNK) and YAP(Yes-associated protein) can promote the activity of AP-1,a downstream effector of JNK **(Liu et al., 2016).**

Furthermore, cross-talks between Hippo-YAP, Wnt, JNK and other signalling pathways that in turn exert influence over the downstream expression of genes that regulate osteogenic and host immune responses **(Codelia et al., 2014).**

It is conceivable that occlusal trauma exerts an abnormal mechanical force exceeding its threshold for adaptability in the periodontium. The Hippo-YAP pathway is associated with mechanical signals, bone remodelling, and inflammatory reactions in cells **(Piccolo et al., 2014)**. In addition, bone resorption and inflammatory reactions are characteristic of periodontal diseases (**Hajishengallis, 2014**).[29]

Yes-associated protein (YAP) is the most important sentinel in the regiment of the Hippo signaling pathway. In a dephosphorylated state, YAP can enter the nucleus and form a transcriptional co-activation state, initiating a cascade of reactions culminating in the

activation of inflammatory response. In the acute inflammatory phase, dephosphorylation-activated YAP can interact with Jun N-terminal kinases (JNK) proteins and catalyse the inflammatory onslaught. Extrapolating the same, it can be stated that inhibition of YAP dephosphorylation can effectively avert occlusal trauma from aggravating periodontitis.[30]

CONCLUSION

In the field of periodontics, it is imperative to prevent, treat, and maintain the periodontal supporting apparatus, which in turn insurmountably contributes to the long-term stability of teeth. In due acknowledgement of this dogma, periodontal treatment should be directed at the preservation of a healthy and sound periodontium, the homeostatic behaviour of which is in turn entrenched in the proper distribution and neutralization of applied mechanical forces.

Occlusion is one of the nonpareil factors affecting the behaviour of the supportive periodontal tissues. This being stated, a sound knowledge regarding the physiology and anatomy of occlusion and periodontia, the occlusal aberrations and its ramifications on the periodontium are sapient to aid in adept diagnosis and charting a comprehensive treatment plan to protect, preserve and allow for the flourish of the precious periodontal tissue in the pertinent zenith of health. This in turn will secure peerlessly the masticatory function and hence will markedly upheave the quality of life.

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