MOLECULAR MECHANISMS AND PATHOPHYSIOLOGY OF TRAUMA FROM OCCLUSION: A SCOPING REVIEW

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Abstract

This scoping review's goals were to thoroughly explore the existing understanding of the cellular and molecular mechanisms that underlie and regulate trauma, to explain the actual concept of TFO along with its background, etiology, signs, and symptoms, relation to periodontal elements, peri-implantitis, and cutting-edge diagnostic techniques, to identify knowledge gaps, and to provide the groundwork for more effective prevention and treatment. There are two schools of thought regarding the impact that trauma from occlusion has on the periodontium with respect to periodontal disease. One contemplates that occlusion-related stress plays a co-destructive role in the development of periodontal disease. The other reasons it is not.

Keywords: Trauma from occlusion, attachment apparatus, occlusal traumatism, periodontal disease, peri implantitis, traumatic occlusion, molecular mechanism, pathophysiology of trauma.

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INTRODUCTION:

Occlusal forces that exceeds beyond its adaptive capacity, tissue injury results and the resultant injury is known as Trauma from occlusion (TFO). Thus, TFO refers to the tissue injury and not the occlusal forces. An occlusion which causes such injury is called Traumatic occlusion. Decades of literature regarding TFO just clarifies that there is two school of thoughts, some literatures directly point out that TFO just simply cannot be the etiological factor for periodontal disease whereas the others are believed to consider TFO as a co-factor for causing periodontal disease. Either way, any literature does not depict that TFO could be a primary source to correspond TFO.

Although these concerns, nearly all of these early investigations concurred that occlusal damage did not result in pocket formation or loss of connective tissue connection. It is clear that the effects of high occlusal force and the periodontium's destructive, adaptive, and reparative responses have been hampered by a relative dearth of evidence based on well controlled prospective human investigations.[1,2,3]

The current review of the literature aims to address the histological and clinical effects of aberrant occlusal forces on other teeth and periodontium, as well as to give diagnosis and clinical aspects, classification, tissue response, molecular mechanisms and TFO with Implants for the same.

TFO HISTORY- Trip Down the Memory Lane

Karolyi was the first to raise the most contentious problem, establishing the concept of bruxism as a crucial role in the pathogenesis of periodontitis in 1901. It's referred to as the "Karolyi effect."
Box et al. conducted research on sheep's teeth, indicating that TFO causes vertical bone defects. Stillman was the first to identify traumatic occlusion as a cause of periodontitis. Trauma is caused by repeated abnormal pressures of one tooth on another. He emphasized that noninfectious alterations are directly caused by traumatic occlusion.[3]

Early in the 1960s, *Glickman and Smulow*[4,5,6] put up the hypothesis that a traumatogenic occlusion can contribute to the development of periodontitis. The "co destructive theory" is the name of this hypothesis. *Goldman*[7] demonstrated that soft tissue

lesions like Stillman's clefts and McCall's festoons were not caused by occlusal trauma. The involvement of TFO in the pathophysiology of Infrabony pockets was established *by Waerhaug*[8,9]. Squirrel monkeys served as the model animal for *Polson.*[10,11]

According to *Houston et al.,*[12,13] there is no connection between bruxism and periodontal disease because neither condition frequently affects the same person and neither condition and occlusal status are strongly related.

No discernible difference was seen between the adjusted and nonadjusted groups in the reduction of tooth mobility, according *to Burgett et al*.[14] "A periodontium remained healthy despite the persistent forces that caused the drifting of the teeth and significant changes in occlusion," according to *Wolffe et al.*[15]
Splinting did not prevent plaque growth or delay attachment loss, as demonstrated *by Ericsson et al.*[16] He demonstrated that jiggled teeth lost some bone despite having healthy gingival tissues and had deeper probing than non-jiggled teeth.

CLASSIFICATION:

Box's classification:
1. **Physiological occlusion.**
2. **Traumatic occlusion.**

PHYSIOLOGIC OCCLUSION:
- Occlusion will be in equilibrium.
- They cannot and will not be changed the normal relationship between the tooth and supporting structures.
- Here, the occlusal stress on the tooth is counteracted by the resistance of periodontal tissues.

TRAUMATIC OCCLUSION:
- Here the damage will be done to the periodontium due to the overloading stress initiated by traumatic occlusal forces.

Glickman Classification (Based on Duration):
1. **Acute TFO**
2. **Chronic TFO**

ACUTE TFO:
- Results from the abrupt change in occlusal forces such as that of biting a hard object.
- Restoration or prosthesis that could change the direction of forces.

CHRONIC TFO:
- Results from a trauma that took place over tissue that develops from gradual changes (transitional phase)
- Produced by tooth wear, drifting or extrusion of teeth combined with parafunctional habits such as bruxism.

Depending Upon the Nature of Cause:

1. **Primary TFO**
2. **Secondary TFO**

PRIMARY TFO*:*
- Occlusal forces are etiological factors
- All the forces are within adaptive capacity.
- **Causes:** restoration, faulty prosthesis, extrusion or drifting
- **Effects:**

a. No connective tissue changes
b. No pocket formation
c. This is because the supra crestal attachment still being intact.

Secondary TFO:
- Here traumatic forces are factor.
- Occlusal forces exceed the adaptive capacity.
- Supra crestal attachment is not subtle.
- Degenerative bone, showing angular bone loss.
- Apical migration of the epithelium.

ETIOLOGY:
- **Precipitating factors.**
- **Pre disposing factors.**

PRECIPITATING FACTORS:
They depend upon:

Magnitude:
- When the magnitude of the forces exceeds or changes, it has an effect on periodontal (pdl) fibres.
- Increase in pdl fibres width is seen.
- Increase in number of fibres.
- Increase in density of alveolar bone.

Direction:
These are the forces transmits from pdl to alveolar bone (AB)

Duration:
Is how long the force is applied.

Frequency:
It is how often the force is applied.

Distribution:
It is how the force is spread over.

PRE-DISPOSING FACTORS:
a. Intrinsic factors.
b. Extrinsic factors.

EXTRINSIC FACTORS:
- Long axis of the teeth in relation to the force applied.
- Morphology of root.
- Morphology of Alveolar Bone.

INTRINSIC FACTORS:
- Local irritants: plaque
- Neurosis by bruxism.
- Loss of supporting osseous structure.
- Malocclusion.
- Loss of supporting tooth.

CLINICAL SIGNS AND SYMPTOMS: (Box and Stillman)

CLINICAL SIGNS:
- Traumatic crescent.
- Recession
- Mobility
- McCall's Festoons.
- Stillman's cleft.
- Joint signs

None of these changes have been show conclusively to be associated with trauma to the periodontium.

SYMPTOMS:
- Persistent discomfort in eating
- Thermal sensitivity
- Muscle hypertonicity.

CLINICAL FEATURES OF TFO:
1. No periodontitis
2. Tooth wear (mild faceting or marked attrition)
3. Fractures of the enamel or restorations
4. Occlusal interferences (either from the retruded contact position to intercuspal position (ICP) or in lateral excursions/protrusive movements)
5. Ridging of buccal mucosa
6. Indentations in lateral border of the tongue
7. Reddening of the tip of the tongue.

TRAUMA FROM OCCLUSION AND PERIODONTAL DISEASES:
GLICKMANS CONCEPT (1969) (Figure 1)
Excessive occlusal forces changed the pathway of spread of inflammation, i.e. extending directly into the pdl leading to angular resorption and Infrabony pocket. According to Glickman, even destruction of periodontium and alveolar bone (horizontal bone loss) only occurs at non-complicated plaque associated sites, which are exposed to abnormal occlusal forces, will develop angular bony defect and infrabony defect.

Conclusion: Occlusal trauma is an aggravating factor (co-destructive factor) in periodontal disease.

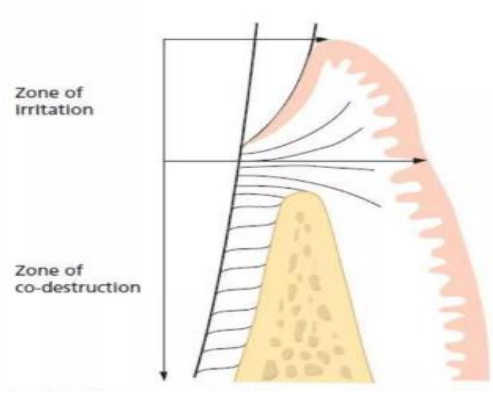


Figure 1: Glickman's concept

TWO ZONES FOR PERIODONTAL STRUCTURES:

1. Zone of Irritation:
- Includes: marginal gingiva, interdental papilla and gingival fibres.
- Local factors stimulate the inflammation in this zone.
- Degeneration and necrosis of gingival connective tissue, epithelial ulceration and suppuration are its most severe destructive effect.
- Uniform(even) bone destruction.

2. Zone of Co-Destruction: (Figure 2)
- Includes: PDL, Alveolar bone, root cementum.
- Demarcated by transeptal collagen fibre bundles.

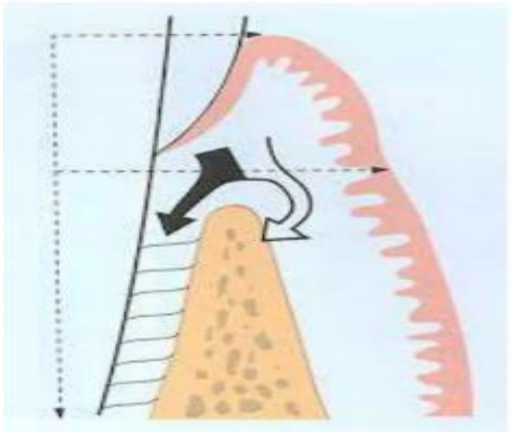


Figure 2: Zone of co destruction

WAERHAUGS CONCEPT (1979)

- Occlusal forces do not play a role in periodontal destruction.
- Angular /vertical bone loss and infra-bony pockets have similar occurrence even at sites with no trauma from occlusion.
- No evidence on "altered pathway of destruction" and said that inflammation and bone loss is due to presence of bacterial plaque.
- Waerhaug always insisted the presence of plaque wherever inflammation persisted.

Conclusion: TFO played no part in periodontal disease and that plaque was only the factor initiating periodontal disease.

CURRENT CONCEPT:

Marginal gingivitis is caused by plaque. (Trauma from occlusion does not affect the blood supply of gingiva). Trauma from occlusion affects the supporting tissues/periodontal ligament (Trauma from occlusion obliterates the blood vessels in the periodontal ligament).

When gingival inflammation spreads into the supporting periodontal tissue, trauma accelerates the progress and severity of bone loss. With elimination of trauma from occlusion bone loss reverses. However, in the presence of inflammation, bone loss does not reverse.

JIGGLING FORCES:

Jiggling forces are intermittent type of forces subjected to teeth or tooth in more than one direction, like in cases with premature contacts such as improper prosthetics or high unresolved restorations.

In conjunction with "jiggling type trauma" no clear-cut pressure and tension zones can be identified but rather there is a combination of pressure and tensions on both sides of the jiggled tooth. This phenomenon has increased mobility without pocket formation, migration and tipping.

Hypermobility will be there as long as the forces are exerted on the tooth. It is not indicative of adaptation nor a sign of ongoing process, but can be result of a previous jiggled force.

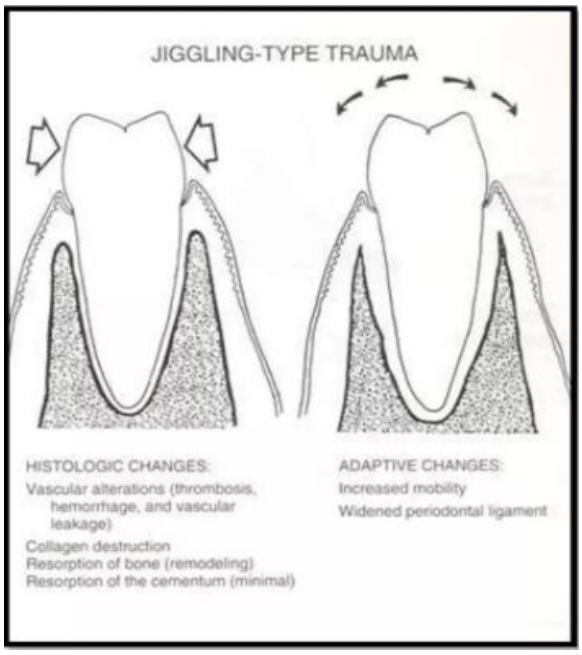


Figure 3: Jiggling Forces

TISSUE RESPONSE TO TFO: (Figure 4)

Tissue response by periodontal elements to trauma from occlusion can be discussed in 3 stages:

1. Tissue Injury.
2. Repair.
3. Adaptive Remodelling.

TISSUE INJURY:

a. Excessive Tension.
b. Excessive Pressure.

Excessive Pressure: (Figure 5)

- When excessive pressure is applied, osteoclastic resorption occurs.
- Pdl space is widened, numerous blood vessels observed during pressure which will ultimately decrease in size.
- When pressure is increased, PDL is compressed and thrombosis of blood vessel occur.
- When pressure gets severe, undermining resorption takes place.

Excessive Tension: (Figure 5)

- During Tension, periodontal fibres elongates and there is bone apposition.
- Blood vessels seems to be enlarged.
- Severe tension may cause widening of PDL, Thrombosis, tearing of PDL and bone resorption.
- Temporary depression is seen in the levels of mitotic activity and rate of proliferation and differentiation of:

a. Fibroblast.
b. Collagen fibres.
c. Bone formation, which returns back to normal after dissipation of the forces.

REPAIR:

- Tissue repair is a continuous process.
- Diseased tissue is removed and new connective tissue, cementum, bone is laid down for restoration of injured periodontium.
- Forces will continue to be traumatic as long as the damage capacity exceeds the reparative capacity of the tissue.
- Important feature in repair is buttressing bone formation.
- When bone is resorbed due to exceeding occlusal forces, a circumferential thin trabecular bone is formed along with new bone. This attempt to compensate for the lost bone is called buttressing bone formation.

REMODELLING:

- To create harmony between continuous damage and repair process, REMODELLING stage occurs for the overhaul of periodontium.
- To cushion the impact of offending forces, PDL is widened and bone resorbed.
- Also. Funnel shaped crest and angular defect in bone is observed.
- After adaptive Remodelling, resorption formation returns to normal.

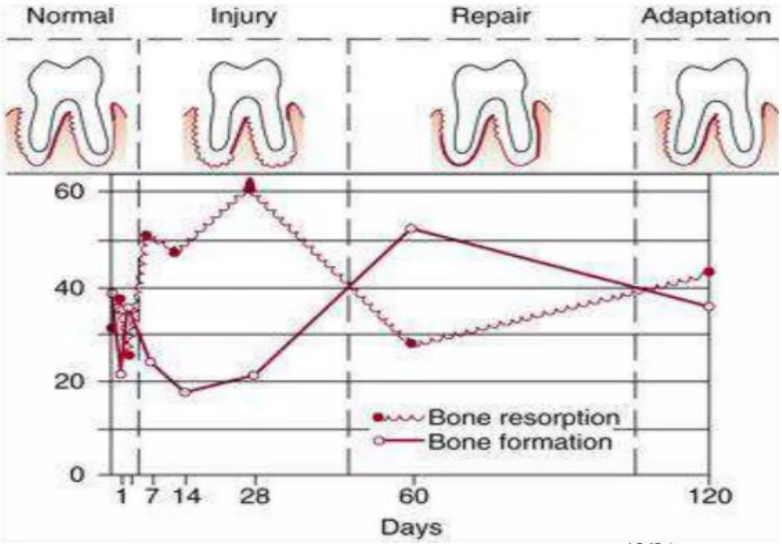


Figure 4: tissue response

MOLECULAR MECHANISMS OF ALVEOLAR BONE RESORPTION CAUSED BY FORCED APPLICATION:

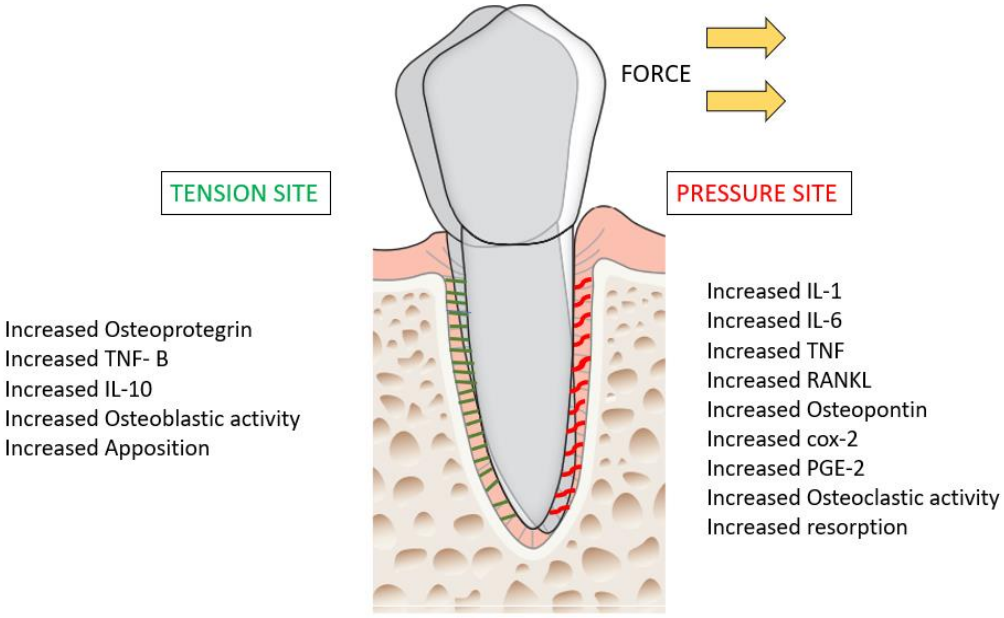
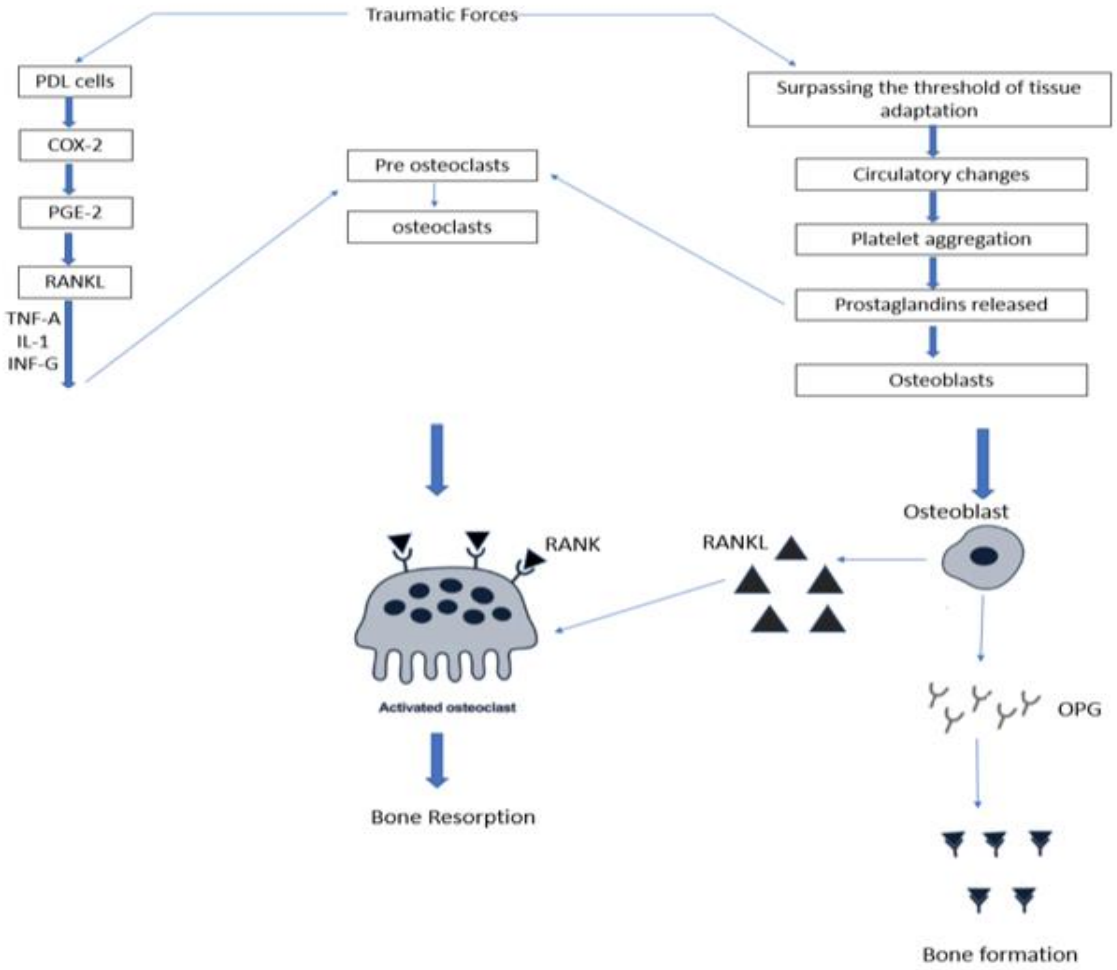


Figure 5: Molecular mechanism

PHYSIOLOGICAL AND PATHOLOGICAL MECHANISM FOR RESORPTION AND FORMATION IN BONE



IS TRAUMATIC OCCLUSION REVERSIBLE?

Occlusal diseases are very much like periodontal disease, they cannot be reversible, except when osseous grafting comes into question in relation to periodontal disease, it is in that way reversible.

Occlusal disease, however, like periodontal disease are maintainable.

It does itself lend to the treatment and when restorative dentistry is utilized, it becomes in that sense reversible.

When there are excessive occlusal forces, pdl might lose it perfusion resulting in ischemia and necrosis of pdl cells, when the adaptive capacity of the pdl is exceeded.

In this case, bacterial inflammation is faster because of lower tissue resistance or integrity.

Hence TFO might be a catalyst for the destructive forces initiated by bacterial periodontal inflammation.

TFO AROUND IMPLANTS:

The injuries caused by occlusal forces on periodontal tissues, can also be affect peri implant tissues.

It is established that obtainment and maintenance of the occlusal stability is important for both-Implants and natural dentition.

Nevertheless, the role of occlusal trauma in installation and worsening of periodontal/peri implant disease remains controversial.

Misch CE., conducted a study to verify the co relation between occlusal trauma and peri implant bone loss that compromised the osseointegration. Authors concluded that there is no enough evidence to prove any co relation between occlusal forces and osseointegrated peri implant bone loss.[17]

Stern RJ., 1993- perception of interference of natural and implant with antagonist teeth forces were 20 and 48 micrometers, respectively.[18]

Hammerlee et al., 1995- tactile perception of implants are 8.75 times more than natural teeth which concluded that implants, without the receivers of PDL, are more susceptible to occlusal forces, which is justified by its decreased adaptive capacity and distribution of forces.[19]

DIAGNOSIS:

TFO is characterised and diagnosed based on histologic alterations in the periodontal supporting

structure, hence block section biopsy is required for diagnosis. Often, occlusal analysis includes Angle's categorization. The structural relationship between the mandible and maxilla was the focus of the Angle classification, which had little to do with the occlusal relationships that occur between different cusp surfaces. Therefore, the most crucial feature of occlusion is the interaction between the opposing cusps. It is quite challenging to relate tooth mobility and wear patterns to occlusal contacts. Mobility is a condition that can be impacted by a variety of additional circumstances, such as loss of attachment.

1. Increased tooth movement is the major symptom of primary TFO. Instruments that are mechanical and electrical can be used to measure mobility. Mobility is evaluated subjectively using the Miller classification and given a score between 0 and 3.
2. The tilting and migration of specific teeth or whole segments. If there is primary TFO in the attachment apparatus, the percussion of teeth on tapping with a blunt object transforms from a resonant note with a healthy supporting framework to a dull note.
3. Careful examination of the masticatory muscles to check for signs of hypertrophy or hypertonicity, as well as potential spasms of one muscle group.
4. Assessing the TMJ and looking for any deviations in the mandible's numerous closure routes
5. Fremitus test: Fremitus is a measurement of the vibratory patterns of the teeth when the teeth are placed in contacting positions 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 ICP and grind systemically in the lateral, protrusive, and lateral protrusive contacting movements and positions. The teeth that are displaced by the patient in these jaw positions are then identified. In general, this is limited to the maxillary teeth; however, in cases of edge-to-edge occlusion or when there is little overlap of the teeth, mandibular teeth can also be assessed.[20,21,22]

The following classification system is used:
• Class 1: Mild vibration or movement detected
• Class II: Easily palpable vibration but no visible movement
• Class III: Movement visible with the naked eye.

Practical Clinical Guidelines:

- Traumatic occlusion causes some teeth in a healthy, non-inflamed dentition to become hypermobile; if this occurs, as well as radiologically enlarging the periodontal ligament space or evident cervical abfraction, the occlusion should be examined and repaired. Most of the time, simple, straightforward techniques are sufficient to restore a physiological situation and to lessen hypermobility.
- It should be understood that tooth mobility might be decreased by occlusal adjustment in cases of a healthy but diminished periodontium. Based on the mechanical circumstances, mobility is nevertheless improved in such situations. Splinting the teeth could be required to improve functional comfort and prevent immediate breakage. This could involve teeth that are extremely movable and have a healthy but reduced periodontium, which complicates the clinical treatments.
- Treating the inflammation is crucial in situations of subsequent occlusal damage and should come first in treatment planning. According to the literature, prematurity may contribute to the development of periodontitis.
- If necessary, occlusal correction ought to be a part of the first stage of periodontal therapy. This causes a greater increase in attachment level during periodontal therapy and may help the periodontal tissues repair more effectively. Premature tooth contacts may worsen the prognosis of periodontally affected tissues, according to some evidence.
- If some teeth do not respond to traditional periodontal therapy as anticipated, additional research should also include a more thorough occlusal study in addition to periodontal reexamination and microbiological testing.
- Undergraduate and graduate training should cover how occlusal trauma and periodontal disease are related (progression, treatment results, prognosis).

CONCLUSION:

The extensive search and in-depth analysis of the existing literature found a body of knowledge as well as redundant data, conflicting conclusions, knowledge gaps, a dearth of supporting data, and the need for additional study. There are several claims made without supporting evidence, which shows that many facets of this subject have not been well investigated and comprehended. This can be caused by the complexity of the regulatory procedures, the low level of problem awareness, and the dearth of appropriate study models. Therefore, root resorption is still frequently detected at a late stage, which can even lead to tooth loss, frequently in a crucial growth and aesthetic zone, with fairly unfavorable long-term effects for patients that could have been avoided. Understanding etiological components and

differentiating whether they can be modified or not indicates therapeutic methods for clinical practice. Therefore, more thorough understanding of the cervical resorption pathomechanisms may result in more efficient prevention and treatment. Another major problem with replacement resorption is that it cannot be stopped once it has started. Tetracycline and/or corticosteroids[23] can block clastic cells, which can delay and reduce the extent of resorption.

Future research on local pharmaceutical strategies to stop dentine from Remodelling into bone has the potential to have significant positive effects on trauma victims. Establishing animal models to investigate dentine resorption, such as through the ectopic implantation of dentine chips following various pre-treatments, may aid in determining the impact of various parameters on the resorptive process and in the development of methods to slow down or stop resorption. The prevention, early detection, and optimal treatment of resorption in patients who have had dental trauma will be made possible by more systematic teaching and rigorous training of dental students and young professionals.

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