

TRAUMA FROM OCCLUSION: DOES IT AFFECT THE PERIODONTIUM? A REVIEW

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ABSTRACT

Mastication is the major function of the dentition and since the periodontium constitutes the supporting mechanism which enables the teeth to fulfil this function, consideration of the interrelation between occlusal forces and the periodontium is basic in periodontology. The etiology of periodontal disease is multifactorial. One of the factors, thought to be responsible for the initiation of inflammatory periodontal disease, is occlusal trauma. Although a variety of occlusal conditions have reportedly been related to inflammatory periodontal disease (e.g. bruxism, malocclusion, abfraction etc.).

Keywords: Trauma, Occlusion, Periodontium

BACKGROUND

For many years, the role of occlusion and its dynamic interactive impact on the periodontium has been an issue of controversy and extensive debate. Although a variety of occlusal conditions have purportedly been related to this interaction, the central focus has been on occlusal trauma resulting from excessive forces applied to the periodontium. In an attempt to clarify and better understand this condition; early investigators used human necropsy specimens and a variety of animal models as a basis for clinical and histological studies. Findings were often diverse and somewhat contradictory.^{1,2}

TERMINOLOGIES USED IN RELATION TO TRAUMA FROM OCCLUSION

1. Occlusal traumatism: The term traumatic occlusion, introduced by Stillman, denoted abnormal stress capable of producing injury to dental or periodontal tissues.

2. Trauma from occlusion: It is a term used to describe pathologic alterations or adaptive changes which develop in the periodontium as a result of undue force produced by the masticatory muscles. TFO was defined by Stillman as “a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position.” The WHO in 1997 defined TFO as “damage in the periodontium caused by stress on the teeth produced directly or indirectly by teeth of the opposing jaw.” In “Glossary of

Periodontics terms” (American Academy of Periodontology 1986), occlusal trauma was defined as “An injury to the attachment apparatus as a result of excessive occlusal force.

3. Fremitus: A palpable or visible movement of a tooth when subjected to occlusal forces (also known as functional mobility).

4. Occlusal adjustment: Reshaping of the occlusal surfaces of teeth by grinding to create harmonious contact relationships between the upper and lower teeth, or orthodontic movement of the teeth to create more harmonious contact relationship.

5. Occlusal interference: Any contact that inhibits the remaining occluding surfaces from achieving stable and harmonious contacts.

6. Occlusal traumatism: The overall process by which a traumatogenic occlusion produces injury in the periodontal attachment apparatus.

7. Parafunction: Abnormal or perverted function.

8. Premature occlusal contact: A condition of tooth contact that diverts the mandible from a normal path of closure.

9. Traumatogenic occlusion: Any occlusion that produces forces which cause an injury to the attachment apparatus.

10. Occlusal trauma: An injury to the attachment apparatus as a result of excessive occlusal forces.

FACTORS CAUSING CHRONIC DESTRUCTIVE PERIODONTAL DISEASE

• Intrinsic factors

1. Morphologic characteristics of the roots. Such factors as their size, shape, and number are of prime importance. Teeth with short, conical, slender, or fused roots rather than divergent roots are more predisposed to occlusal traumatism when subjected to prolonged excessive force than are those with normal structure.³
2. The manner in which occlusal surfaces and the roots are oriented in relation to the forces to which they are exposed. Axially inclined forces are more tolerable than are nonaxially inclined forces, which may be functional or parafunctional. When teeth are badly aligned, the effect of excessive force can be deleterious.
3. Morphologic characteristics of the alveolar processes. If the quantity or quality of alveolar bone is inherently lacking, the effects of prolonged parafunctional forces may result in rapid loss of the remaining support.^{2,3}

• Extrinsic factors

Among the extrinsic factors that may seriously increase the rapidity of loss of supporting alveolar bone are the following:

1. Irritants: Microbial plaque is implicated as the most serious irritant. Other irritants that may have similar effects are food impaction that results in positive pressure on the tissues, overhanging restorations, poorly contoured crowns and bands, and ill fitting partial denture clasps.
2. Neuroses that result in parafunctional activities, for example, bruxism. These factors are the most prevalent and serious causes of abnormal occlusal stresses.
3. Loss of supporting bone: Periodontitis, injudicious bone resection, inadvertent trauma, and systemically related diseases are the chief causative factors.
4. Loss of teeth resulting in overloading of the remaining teeth, for example, posterior bite collapse.
5. Iatrogenically created functional malocclusion.

RATIONALE FOR REDUCING OCCLUSAL TRAUMA IN PERIODONTITIS

The scientific rationale for how occlusal trauma may contribute to increased periodontal bone loss has received some attention over the past decade. Central to occlusal trauma-induced (and periodontitis-induced) bone resorption seems to be stimulation of receptor activator of nuclear factor kappa B ligand (RANKL), the primary driver of osteoclast activation. RANKL interacts with RANK

receptor on osteoclasts to initiate bone resorption. In a rat model where occlusal trauma was initiated by raising the occlusal surface, immunohistochemistry demonstrated RANKL associated with osteoclasts and osteoblasts.⁴ A similar occlusal trauma protocol combined with lipopolysaccharide-induced periodontitis extended the length of time that osteoclasts were present on the interproximal bone surface, and the increased expression of RANKL in osteoclasts, endothelial cells, inflammatory cells, and periodontal ligament cells. Human periodontal ligament cells exposed to mechanical stress also increased interleukin-6 production, a potent stimulator of RANKL, periodontal inflammation, and bone resorption.^{4,5}

HISTORICAL EVIDENCE

Karolyi was the first one to start the most controversial issue by introducing in 1901 the concept of bruxism as a significant factor in the pathogenesis of periodontitis. It is known as the “Karolyi effect.”³

Occlusal trauma was defined as an injury (usually a histologically demonstrable lesion) to the attachment apparatus or tooth as a result of excessive occlusal forces. Common symptoms of occlusal trauma were described to include pain or discomfort, dental hypersensitivity, tooth mobility (fremitus), or pathologic migration of teeth.

Box et al. did study on sheep's tooth suggesting that TFO produces vertical bone defect. **Stillman** was the first to emphasize traumatic occlusion as a cause of periodontal disease. He suggested abnormal pressures of one tooth on another produce traumatic injury and also pointed out that there are noninfectious changes that are directly produced by traumatic occlusion. **Glickman and Smulow** proposed the theory in the early 1960s that a traumatogenic occlusion could act as a cofactor in the progression of periodontitis. This theory is known as the “co destructive theory.”^{4,5,6}

Goldman proved that occlusal trauma was not the cause of soft tissue lesions such as Stillman's clefts and McCall's festoons. **Waerhaug** proved the involvement of TFO in the pathogenesis of infrabony pockets.¹

Wang and colleagues reported that mobile teeth had significantly more attachment loss during the maintenance years than the nonmobile teeth.¹

Jin and Cao found no significant difference in probing depth, clinical attachment level, or bone height loss in teeth with or

without abnormal occlusal contact, but teeth with mobility or widened periodontal ligaments had greater probing depths, more attachment loss, and increased alveolar bone loss.²

Support for abnormal occlusal contacts, which may lead to occlusal trauma, as a risk factor in periodontitis progression and eventually tooth longevity was found in a series of retrospective studies by Harrel and Nunn. These authors reported that teeth with occlusal discrepancies had significantly deeper probing depths and worse prognoses and mobility. Occlusal discrepancies were defined as differences between retruded position (centric relation) and maximum intercuspation (centric occlusion), and working and balancing contacts in lateral and protrusive movements. After adjusting for other risk factors, such as smoking and poor oral hygiene, occlusal discrepancy continued as an independent contributor to increased probing depths.^{1,2,3}

IMPACT OF OCCLUSAL TRAUMA ON PERIIMPLANT BONE LOSS

With the surge of dental implant placements over the last few years, the role of occlusal trauma or overload in periimplant bone resorption has been investigated. It was proposed that excessive occlusal force may contribute to bone loss around implants.

Monkey studies found that, as with natural teeth, occlusal overload did not induce periimplant inflammation but did cause bone resorption around implants. However, when inflammation was added to the occlusal overload by withholding tooth brushing, no acceleration of bone resorption was noted. A subsequent review paper of cellular biomechanics, engineering principles, bone mechanical properties, animal studies, clinical reports, bone physiology, and implant design biomechanics reported that occlusal overload on implants may increase the incidence of marginal bone loss.⁷

A systematic review of animal studies on the effects of occlusal overload on periimplant tissue revealed only two appropriate controlled trials (in dogs), which concluded that overload alone is not associated with periimplant tissue breakdown, but when combined with plaque accumulation is key to increased pocket depths and loss of bone-to-implant contact. A more recent review came to the same conclusion, whereas, another pointed to the poor level of evidence and conflicting results.⁷

In natural teeth, the periodontal ligament has

neurophysiological receptor function, which transmits information from nerve endings to the central nervous system. The presence or absence of periodontal ligament function determines a great difference in the phase of detection of occlusal force. While, in teeth, the excessive force can be caught early, in the case of implants, the absence of proprioception of the periodontal ligament can lead to significant bone loss.² The periimplant bone loss has two major etiological factors: inflammation caused by microorganisms and occlusal overload. The first is already well established in the literature as a causal factor. The second, however, is controversial. A study was conducted in order to verify the correlation between occlusal trauma and periimplant bone loss that compromised osseointegration. The authors concluded that there is not sufficient evidence to confirm the correlation between occlusal overload and periimplant bone loss.^{5,7}

CONCLUSION

It is universally believed that the bacterial plaque is the causative agent for periodontal destruction. A risk factor for periodontal disease appears to be an environmental or host factor that predisposes a patient to periodontal breakdown from bacterial plaque. The risk factor for excessive occlusal forces is one that can be minimized with existing clinical armamentarium, and, as is the case for all risk factors that can be ameliorated by treatment, treatment of excessive occlusal forces to minimize the effect of this risk factor may need to be a part of routine periodontal treatment.

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