

Periodontal-Occlusal Interrelationships: A Perspective

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Introduction

The role of occlusal trauma in the initiation and progression of periodontal disease has been an ongoing debate for decades. While still unsettled, our knowledge base has matured in recent years affording us an opportunity to draw some legitimate conclusions and to define more clearly the existing controversies. The literature pertaining to this subject can be clearly divided into two groups. Early writings described occlusal trauma as a primary etiology of periodontal disease. Bone loss and pocket formation were believed to be a direct consequence of injury to the periodontal ligament. The more recent literature examines occlusal trauma as a possible co-factor in periodontal disease.

Early evidence supporting occlusal trauma as a primary cause of pocket formation was largely empiric. Karolyi¹, Stillman², Box³, Stones⁴ and McCall⁵ made important observations on the nature of the injury to the supporting periodontal structures and suggested a possible correlation to the formation of vertical osseous defects. The present body of information on the pathogenesis of periodontal disease, however, overwhelmingly supports a bacterial etiology to periodontitis.^{6,7}

The role of occlusal trauma in the progression of periodontitis remains to be clarified. This paper will focus on the research designed to separate the variables of inflammatory periodontal disease from the lesion of occlusal trauma. The objective is to help the practitioner formulate a working strategy to incorporate these concepts into clinical practice.

Current Concepts of Occlusion Forces

Glickman^{8,9} in the 1960's described an alteration in the pathway of inflammation as a result of the influence of occlusal trauma. He suggested that periodontal ligament and bone damage facilitated the movement of the inflammatory process into the ligament, resulting in angular and crater-like bone loss. Glickman's work stimulated a series of animal experiments directed at reproducing his findings.

Lindhe and Svanberg¹⁰ in 1974 designed an experiment utilizing Beagle dogs that compared attachment levels in periodontally involved sites with and without

occlusal trauma. The results showed a loss of periodontal ligament attachment in the test teeth but not in the controls, supporting in many ways the experiments of Glickman. Another Beagle dog study¹¹ also demonstrated that occlusal trauma alone does not lead to pocket formation. The authors subjected healthy, non-periodontally involved teeth to excessive occlusal trauma, yet no loss of attachment was noted. Other investigators duplicated this experiment using other animal models and conditions with virtually the same results.^{12,13,14,15}

Meitner et al.,¹⁶ in 1975, designed a similar experiment combining occlusal trauma and periodontitis. Meitner found loss in attachment levels in only one out of the four specimens, leading him to his conclusion that occlusal trauma does not contribute to increased attachment loss. His study differed from the Lindhe/Svanberg studies in that Squirrel monkeys were used, a shorter period of occlusal trauma was employed, and the method of traumatizing the teeth was different.

The evidence suggests that the lesion of occlusal trauma is an injury to the periodontal ligament structure (the cementum, the periodontal ligament proper and the associated alveolar bone). Excessive forces can lead to pathologic changes in the periodontal ligament. Reported findings include vasculitis, disorganization of cells and fibers, bone and cementum resorption, necrosis of collagen fibers and hyalinization of the periodontal ligament.^{17,18} The type of damage depends upon the direction, duration and magnitude of the force and is limited to the ligament region and not the gingiva (Figure 1). Orthodontic or parafunctional forces or forces of mastication may be capable of promoting these tissue changes.

Formation of pockets is unrelated to occlusal forces since periodontitis is initiated in the gingival apparatus (the epithelial attachment, the sulcular epithelium, and the surrounding connective tissue) which is contiguous with the tooth and can be displaced if an external force is applied. This is further supported by studies showing trauma from occlusion does not initiate nor alter gingival inflammation, nor does it have an effect on subgingival bacterial compositions.^{13,18}

The controversy remains whether the combination of occlusal trauma and periodontal inflammation will lead to a greater loss of attachment than would be expected from periodontitis alone.

Orthodontic tooth movement serves as an illustration of this question.¹⁹ Trauma, in the form of tension and compression of the periodontal ligament, must be introduced in order to initiate tooth movement. Resorption of bone results in a widened periodontal ligament. Bodily movement of teeth tends to produce uniform widening of the periodontal ligament while tipping forces usually produce a funnel effect described as an increased ligament dimension on the coronal aspect of the marginal bone. This v-shaped lesion, seen radiographically, may or may not be the infrabony defect often associated with trauma from occlusion depending on whether there has actually been attachment loss.

Let's take, for example, a patient with existing untreated periodontal disease who is undergoing orthodontic therapy. Is this patient likely to have accelerated attachment loss now that occlusal trauma, in the form of orthodontic therapy, is superimposed on an existing periodontitis? The majority of patients do not develop severe periodontal progression during active therapy. Yet some degree of increased periodontal disease has been reported.²⁰⁻²⁴ This unpredictability may be correlated to the way periodontal disease progresses.

Current Concepts of Periodontal Disease Progression

Studies introduced over the past five years have enhanced our understanding of the progression of periodontal disease. Sites with clinical signs of inflammation do not seem to lose attachment in uniform increments over long periods of time. Rather, it is believed that periodontal disease progresses episodically and is site specific. Active disease is characterized by rapid bone and attachment loss followed by long periods of inactivity.²⁵ It is thought that when activity does occur, large increments of attachment are lost.²⁶ The prediction of disease activity is still a mystery. If periodontal disease is not linear, then, with the introduction of occlusal trauma, attachment loss may not occur simply because a particular site is not presently active. How-

ever, if the site does become active, then increased loss may be possible. The bone loss may be more pronounced in the combined lesion than what would be expected from periodontitis alone.^{10,27} From this information one may feel inclined to rethink the conclusion of Meitner.¹⁶ It is possible that only the monkey which showed loss of attachment had the combined lesions of occlusal trauma and active periodontitis.

Let's return to the adult orthodontic patient with pre-existing periodontitis. If the periodontitis is not controlled, this patient may experience severe attachment and bone loss because certain sites may become active during tooth movement. The turn on of activity may be coincidental to the orthodontic therapy or may be the result of increased plaque accumulation around the orthodontic appliances. Periodontal breakdown, secondary to orthodontic therapy, is becoming more prevalent as more adults are being treated.²⁰⁻²¹

It is important that we distinguish the periodontally susceptible patients from the resistant ones. We gauge susceptibility to periodontal disease by the patient's current periodontal status with reference to the patient's age, local factors, dental history and systemic influences. It is far easier to classify a patient's susceptibility as they increase in age because we can assess their ability to defend against local factors (plaque, calculus) over a long period of time. Older patients may present chronic marginal gingivitis, yet their failure to progress into periodontitis allows us to classify them as resistant. We would categorize the young patient who presents with significant periodontitis as susceptible.

Clinical Signs of Occlusal Trauma

The diagnosis of occlusal trauma requires an accurate dental history, a thorough clinical and radiographic examination, and, in many instances, time. Knowledge, experience and clinical intuition may be of equal importance. The terms primary and secondary occlusal trauma have been used to describe clinical signs of occlusal trauma and are based on the presence of significant clinical mobility. Primary occlusal trauma is defined as "trauma resulting from excessive occlusal forces applied to a tooth with normal supporting structure."²⁸ Significant clinical mobility exists, but the con-

dition can be reversed by re-establishing a normal occlusal load on the tooth either through elimination of the tooth away from the force or by the migration of the tooth away from the force. Teeth that have lost periodontal support may be subject to secondary occlusal trauma, defined as “normal occlusal forces,” which may cause trauma to the attachment apparatus of a tooth or teeth with inadequate support.²⁸ Significant clinical mobility is present, yet the mobility cannot be reversed due to lack of osseous support on the involved tooth.

Mobility, if not progressive, represents a physiologic adaptation of the periodontal ligament to a directed force. The periodontal ligament will widen until its increased dampening ability is in equilibrium with the forces applied. Once this occurs, evidence suggests there is no longer pathologic change to the ligament on a histologic level. Studies have indicated that the progression of periodontal disease is unrelated to the width of the periodontal ligament and its associated mobility.^{14,15,29} It is quite common for patients to exhibit long-standing clinical mobility. This probably depicts a healthy adaptive periodontal ligament and not traumatic occlusion.

It may be more appropriate to change the terms primary and secondary occlusal trauma to primary and secondary occlusal mobility. It is quite evident that most cases with mobility are not associated with trauma. The only true determinant of occlusal trauma is histologic examination of the structures. The distinction between simple mobility and mobility with concurrent trauma is very difficult, yet the clinician is charged with making that diagnosis and providing appropriate therapy.

Indication for the Treatment of Occlusal Trauma

The finding of progressive mobility is a strong indication for treatment.³⁰ By being progressive, the occlusal forces are constantly exceeding the ability of the periodontium to adapt. To determine increasing mobility, the clinician must be able to use an appropriate scale³¹ and measure mobility over at least two points in time. We are limited by the subjectivity of mobility scales and that we must allow the condition to worsen before we can diagnose progression.

The presence of abnormal forces manifesting as significant mobility on a normal (primary) or a reduced (secondary) periodontium should also be considered for treatment. One is unsure that this finding repre-

sents occlusal trauma. Abnormal occlusion in the form of parafunctional habits, fremitus (a palpable or visible movement of a tooth when subjected to occlusal forces),²⁸ or occlusal interferences implies an occlusal scheme that deviates from the ideal. The resultant mobility patterns may compromise periodontal therapy. Fleszar et al.²⁹ reported that teeth associated with clinical mobility did not respond as well to periodontal therapy over an 8-year period as did firm teeth with the same degree of disease severity.

The practitioner should treat a dentition with significant mobility if the mobility interferes with the patient's comfort.³⁰ This is frequently associated with a severely compromised dentition and a patient with poor masticatory function. Splinting is usually indicated as treatment.

Conclusion

We currently are limited in our ability to diagnose active periodontal disease. The treatment of periodontal disease is based on reducing the risk of activity by controlling plaque related inflammation and potential co-factors. Clearly, occlusal trauma does not initiate pocket formation, but significant information exists that when coupled with periodontitis, the potential for severe attachment loss does exist.

If we were capable of maintaining the periodontal patients in a state of ideal periodontal health, free of marginal inflammation, then we may not need to concern ourselves with potential co-factors. However, since clinical practice frequently deviates from conditions of the research laboratory, and marginal inflammation does exist in even our most compliant and motivated patients,³² we are obligated to provide the best environment for maintaining our patients' dentition.

The evidence suggests that we consider treating occlusal trauma on our patients with concurrent periodontitis. The evidence is less clear on the non-periodontally involved patient. The clinician must be able to carefully assess the patient's periodontal status and degree of susceptibility in order to make sound judgments on treatment. The difficulty in the diagnosis of occlusal trauma makes the potential for overtreatment or undertreatment a reality. The decision to treat occlusion must be founded on scientific data as well as our clinical experience. Our ultimate goal remains the retention of our patients' dentition with the preservation of function and comfort for their lifetime.

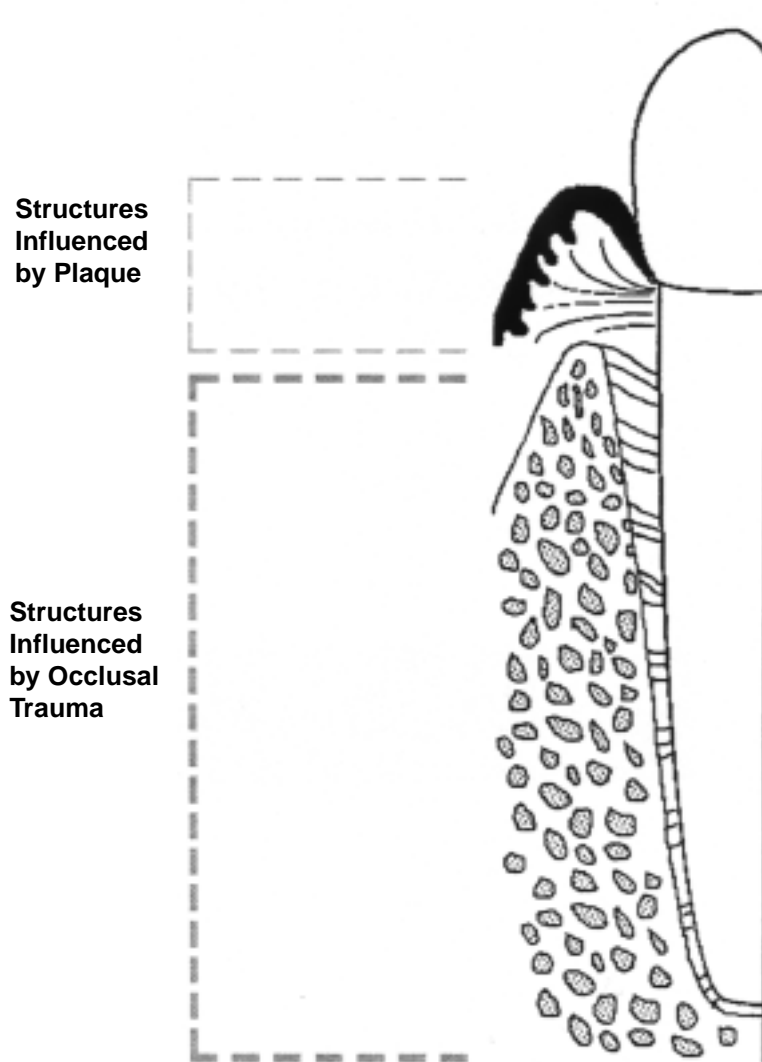
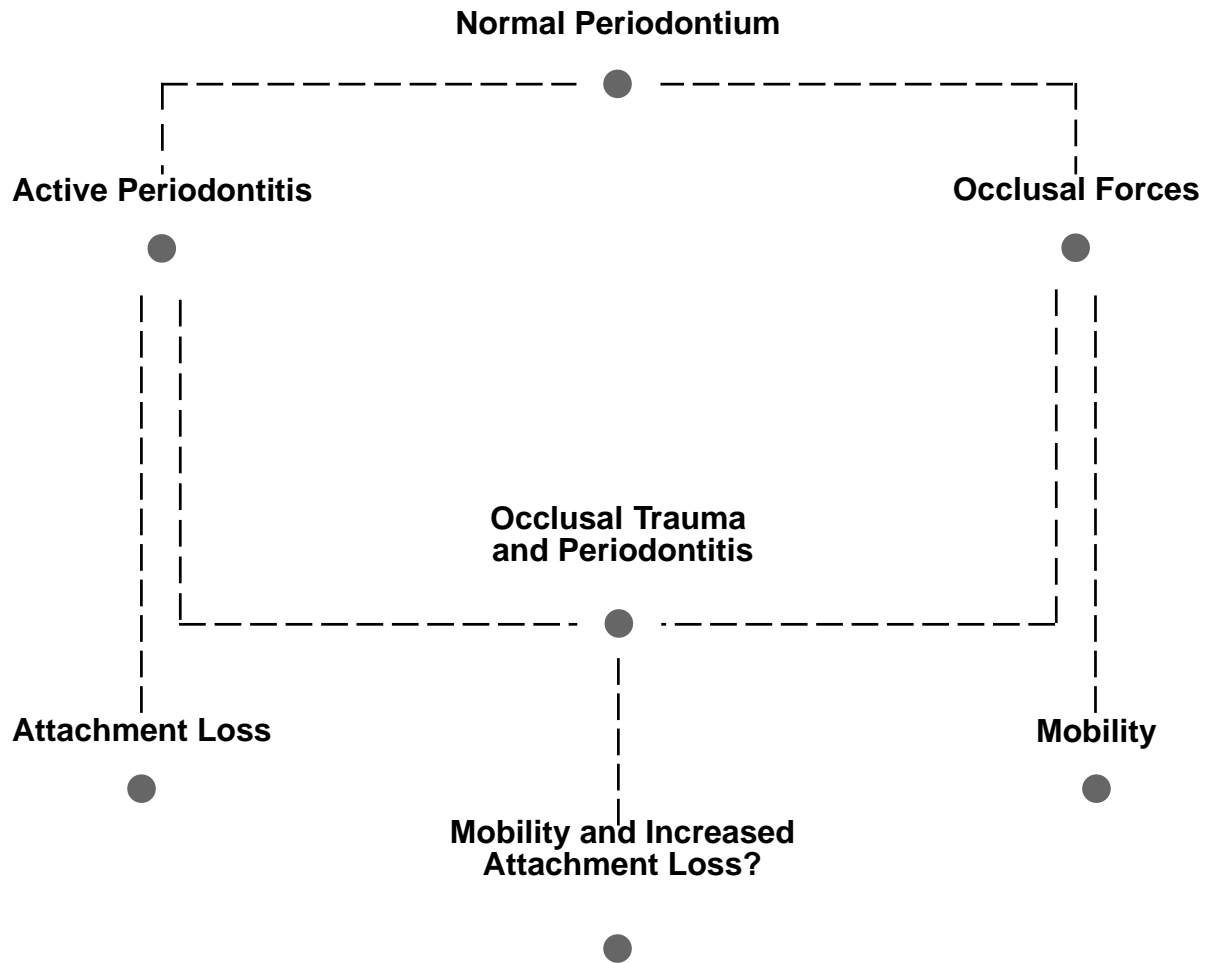


Figure 1. The separate lesions of inflammation and occlusal trauma are thought to act on distinct anatomical structures.

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