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Occlusal trauma and excessive occlusal forces: Narrative review, case definitions, and diagnostic considerations

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Abstract

Objectives: This narrative review determines the effects of occlusal trauma and excessive occlusal forces on the periodontium, including the initiation and progression of periodontitis, abfraction, and gingival recession. Case definitions, diagnostic considerations, and the effects of occlusal therapy are also reviewed and discussed.

Importance: The role of occlusal trauma in the initiation and progression of periodontitis remains a controversial subject in periodontology. Because occlusal trauma can only be confirmed histologically, its clinical diagnosis depends on clinical and radiographic surrogate indicators which make clinical trials difficult.

Findings: Investigations have generally agreed that occlusal trauma and excessive occlusal forces do not initiate periodontitis or loss of connective tissue attachment. When plaque-induced periodontitis and occlusal trauma are present at the same time, there is weak evidence that the occlusal trauma may increase the rate of connective tissue loss. Occlusal therapy is indicated as part of periodontal therapy to reduce mobility and increase patient comfort and masticatory function. Existing data do not support the existence of abfraction as a cause for gingival recession.

Conclusions: Occlusal trauma does not initiate periodontitis, and there is weak evidence that it alters the progression of the disease. There is no credible evidence to support the existence of abfraction or implicate it as a cause of gingival recession. Reduction of tooth mobility may enhance the effect of periodontal therapy.

KEYWORDS

attachment loss, classification, diagnosis, disease progression, esthetics, gingival recession, periodontal biotype

The literature concerning the relationship between periodontal diseases and occlusal forces is reviewed. In addition, studies that have examined effects of excessive occlusal forces, abfraction, and gingival recession are reviewed. Finally, this information is used to consider the revision of the classification of periodontal diseases and conditions.

MATERIALS AND METHODS

For this narrative review, a literature search was conducted using PubMed and Web of Science. A search strategy for the database was performed to find studies that matched the following terms: (periodontal disease OR periodontitis OR periodontium) AND

(traumatic dental occlusion OR traumatic dental occlusions OR occlusal force OR occlusal forces OR occlusal discrepancies OR occlusal discrepancy OR occlusal interference OR occlusal interferences OR occlusal trauma OR occlusal traumatism); (occlusal) AND (non carious cervical lesion OR non carious cervical lesions); (occlusal) AND (abfraction OR abfractions); and gingival recession AND occlusal. Databases were searched without language restrictions using MeSH terms, key words, and other free terms, and Boolean operators (OR, AND) were used to combine searches. Randomized controlled clinical trials, cohort studies, case control studies, case series, review articles, guidelines, animal research, and in vitro research were eligible for inclusion in this review. Databases were searched up to February 2017, with no limits on the year of publication.

Manual searches of *Journal of Periodontology*, *Journal of Clinical Periodontology*, *Periodontology 2000*, *Journal of Periodontal Research*, and *International Journal of Periodontics and Restorative Dentistry* were also conducted. Initially, one reviewer screened the titles and abstracts of articles. Articles that indicated a possible match were obtained for full review for potential inclusion. Important historic articles were included. To complement the search, reference lists of main articles related to this narrative review were also assessed. Due to the heterogeneity of the studies, a meta-analysis was not conducted. A total of 93 articles were included in the review and included both human and animal studies.

CASE DEFINITIONS AND DIAGNOSTIC CONSIDERATIONS

Excessive occlusal force is defined as occlusal force that exceeds the reparative capacity of the periodontal attachment apparatus, which results in occlusal trauma and/or causes excessive tooth wear (loss).[1–3]

Occlusal trauma is a term used to describe injury resulting in tissue changes within the attachment apparatus, including periodontal ligament, supporting alveolar bone and cementum, as a result of occlusal force(s).[4] Occlusal trauma may occur in an intact periodontium or in a reduced periodontium caused by periodontal disease.

Primary occlusal trauma is injury resulting in tissue changes from excessive occlusal forces applied to a tooth or teeth with normal periodontal support.[4] It occurs in the presence of normal clinical attachment levels, normal bone levels, and excessive occlusal force(s).

Secondary occlusal trauma is injury resulting in tissue changes from normal or excessive occlusal forces applied to a tooth or teeth with reduced periodontal support.[4] It occurs in the presence of attachment loss, bone loss, and normal/excessive occlusal force(s).

Fremitus is a palpable or visible movement of a tooth when subjected to occlusal forces.[4]

Bruxism or tooth grinding is a habit of grinding, clenching, or clamping the teeth.[4] The force generated may damage both tooth and attachment apparatus.

Despite the consensus on the definition of primary and secondary occlusal trauma, specific criteria to distinguish between "normal" and "reduced" periodontal support have not been identified from controlled studies. In an in vitro study, periodontal ligament stress increased significantly after reducing 60% of bone support.[5]

Because trauma from occlusion is defined and diagnosed on the basis of histologic changes in the periodontium, a definitive diagnosis of occlusal trauma is not possible without block section biopsy. Consequently, multiple clinical and radiographic indicators are used as surrogates to assist the presumptive diagnosis of occlusal trauma. Clinical diagnosis that occlusal trauma has occurred or is occurring may include progressive tooth mobility, fremitus, occlusal discrepancies/disharmonies, wear facets (caused by tooth grinding), tooth migration, tooth fracture, thermal sensitivity, root resorption, cemental tear, and widening of the periodontal ligament space upon radiographic examination (Table 1).[6,7] These clinical signs and symptoms may indicate other pathoses. For instance, loss of clinical attachment can affect the severity of mobility. Also, it is often very difficult to determine whether the wear facets are caused by functional contacts or parafunctional habits, such as bruxism. Therefore, differential diagnoses should be established. Supplementary diagnostic procedures, such as pulp vitality tests and evaluation of parafunctional habits, may be considered.

Non-carious cervical lesions (NCCLs) involve loss of hard tissue at the cervical third of the crown and subjacent root surface, through processes unrelated to caries.[8] Gingival recession is defined as location of the gingival margin apical to the cemento-enamel junction.[4] NCCLs are usually accompanied by gingival recession.[9] NCCLs are a group of lesions and the etiology is multifactorial.[10] Abfraction, a hypothetical tooth–surface lesion caused by occlusal forces, is one of the proposed etiologies for NCCLs, and other etiologies include abrasion, erosion, corrosion, or a combination.[4,8,11] The lesion of abfraction has been described as wedge-shaped defects that occur at the cemento-enamel junction of affected teeth as a result of flexure and eventual fatigue of enamel and dentin.[8,12–14] Excessive occlusal forces have long been proposed to be a causative factor in the development of abfraction and gingival

TABLE 1 Proposed clinical and radiographic indicators of occlusal trauma

1. Fremitus	7. Thermal sensitivity
2. Mobility	8. Discomfort/pain on chewing
3. Occlusal discrepancies	9. Widened PDL space
4. Wear facets	10. Root resorption
5. Tooth migration	11. Cemental tear
6. Fractured tooth	

PDL, periodontal ligament.

recession.[2,3,11–16] Because abfraction is not currently supported by appropriate evidence, a definitive diagnosis is not possible. NCCLs may result from abrasion, erosion, or corrosion. Therefore, in cases of NCCLs, toothbrushing habits, diet, eating disorders as well as occlusal relationships and parafunctional habits should be thoroughly evaluated.

NARRATIVE REVIEW

Effects of occlusal trauma on the initiation and progression of periodontitis

Histologically, a tooth affected by occlusal trauma demonstrates distinct zones of tension and pressure within the adjacent periodontium. The location and severity of the lesions vary based on the magnitude and direction of applied forces.[2] On the pressure side, these changes may include increased vascularization and permeability, hyalinization/necrosis of the periodontal ligament, hemorrhage, thrombosis, bone resorption, and in some instances, root resorption and cemental tears. On the side of tension, these changes may include elongation of the periodontal ligament fibers and apposition of alveolar bone and cementum.[3,17–19]

Collectively, the histologic changes reflect an adaptive response within the periodontium to occlusal trauma.[2,20] As a result of sustained occlusal trauma, the density of the alveolar bone decreases while the width of the periodontal ligament space increases, which leads to increased tooth mobility and often a radiographic widening of the periodontal ligament space, either limited to the alveolar crest or through the entire width of the alveolar bone.[17,18,21] In addition, fremitus, or palpable functional mobility of a tooth, is another significant clinical sign of occlusal trauma.[22]

Historic studies

In the early 20th century, a report indicated an association between excessive occlusal forces and pyorrhea alveolaris (i.e., periodontitis).[1] It was further suggested by other early investigators that excessive occlusal force was the cause of periodontitis.[2,3,23,24] They felt that occlusal forces had to be controlled to successfully treat periodontitis.[3,17]

In the1930s to the 1940s, the role of excessive occlusal forces in the progression of periodontitis was disputed.[25,26] Using human autopsy material, it was concluded that gingival inflammation extending into the supporting bone was the cause of periodontal destruction. In a subsequent animal experiment, it was found that the excessive occlusal forces caused changes in the direction of the periodontal membrane fibers so that gingival inflammation passed directly into such areas.[18] Later, another study based on human autopsy material agreed that inflammation appeared to begin in the gingiva and subsequently progressed into the adjacent periodontal supporting tissue[19,20] It was further proposed that inflammation progressed in an altered pathway in teeth subjected to occlusal trauma. The combined effect of occlusal trauma and bacterial plaque–induced inflammation was termed "co-destruction." This theory was then challenged by other investigators.[27–29] Using human autopsy material again, the altered pathway of destruction was questioned because bacterial plaque was always present in close proximity to the site of periodontal destruction, and this suggested that inflammation and bone loss were associated with the presence of bacterial plaque rather than excessive occlusal forces. The historic studies used autopsy material that provided little or no information on the periodontal conditions and occlusal conditions of these study subjects. It was after the co-destruction theory was presented that researchers started to examine the concept of multiple risk factors that resulted in the initiation and progression of periodontal diseases.

Animal studies

By their nature, historic observations failed to prove any causal relationship between occlusal trauma and the initiation or progression of periodontal disease. In an attempt to prove a relationship between occlusion and periodontal disease, multiple animal studies with strict controls and designs were performed in the 1970s. There were two significant groups, one from Eastman Dental Center in Rochester, NY,[30–34] and the other one from the University of Gothenburg in Sweden.[35–38] The effects of occlusal trauma and gingival inflammation in animals were investigated. The Eastman group used repeated applications of orthodontic-like forces on the teeth of squirrel monkeys, and the Gothenburg group used occlusal forces similar to those of a "high" restoration in beagle dogs. Both groups examined the effects of excessive occlusal forces on the periodontium with a duration from a few weeks up to 6 months in the presence and absence of bacterial plaque-induced periodontitis.

Despite the different animal models and the different types of occlusal forces applied, the results of these two studies were similar in many respects. When oral hygiene was maintained and inflammation was controlled, occlusal trauma resulted in increased mobility and loss of bone density without loss of connective tissue attachment, during the length of the study. If the occlusal forces were removed, the loss of bone density was reversible. In contrast, in the presence of plaque-induced periodontitis and occlusal trauma, there was greater loss of bone volume and increased mobility, but loss of connective tissue attachment was the same as on teeth subjected to periodontitis alone in the squirrel monkey.[31] In the beagle dog model, when occlusal trauma was superimposed on periodontitis, there was an accelerated loss of connective tissue attachment.[35] Based on the findings of these studies, it was concluded that without plaque-induced inflammation, occlusal trauma does not cause irreversible bone loss or loss of connective tissue attachment. Therefore, occlusal trauma is not a causative agent of periodontitis.

Using rat models, more recent studies re-examined the association of occlusal trauma and periodontal bone loss.[39–41] Occlusal trauma was induced by either placing inlay or metal wire bonding to raise the occlusal surfaces. The receptor activator of nuclear factor-kappa B ligand (RANKL) is an important factor in osteoclast

differentiation, activation, and survival.[42] RANKL interacts with RANK receptor on osteoclasts to initiate bone resorption. During excessive occlusal loading, the destruction of the periodontal ligament was observed, and the RANKL associated with osteoclasts and osteoblasts was demonstrated via immunohistochemistry.[39] In the presence of lipopolysaccharide-induced inflammation, the expression of RANKL on endothelial cells, inflammatory cells, and periodontal ligament cells was enhanced by occlusal trauma.[40] It was suggested that RANKL expression on these cells was closely involved in the increase of osteoclasts induced by occlusal trauma. Further, loss of connective tissue attachment at the onset of experimental periodontitis was increased when inflammation was combined with occlusal trauma.[41] In addition, estrogen deficiency, nicotine, and diabetes were all shown to enhance bone loss in rats with combined with occlusal trauma and ligature-induced periodontitis.[43–45]

None of the animal studies were able to reproduce all aspects of human periodontitis. In addition, the animal studies used excessive forces and were conducted for a relatively short duration (a few weeks to a few months). Nonetheless, the results from animal studies suggested that occlusal trauma does not cause periodontitis, but it may be a cofactor that can accelerate the periodontal breakdown in the presence of periodontitis.

Clinical studies

Tooth mobility has been described as one of the common clinical signs of occlusal trauma.[3,17,18,20,25,28] However, increased tooth mobility may result from inflammation and/or bone loss or attachment loss alone. Progressive mobility may be suggestive of ongoing occlusal trauma, but assessments at different time points are necessary to make this determination.[46] In an epidemiologic study, a group of subjects was re-examined for loss of periodontal clinical attachment after 28 years. It was found that baseline tooth mobility was a factor related to clinical attachment loss.[47] In addition, mobile teeth with a widened periodontal ligament space had greater probing depth, more attachment loss, and increased alveolar bone loss than non-mobile teeth.[7] Tooth mobility was also found to affect the results following periodontal therapy.[48,49] It was shown that teeth with mobility did not gain as much clinical attachment as those without mobility following periodontal treatment.[48] Further, teeth with increased mobility demonstrated significantly more clinical attachment loss during the maintenance period.[49] A recent study on regenerative surgery indicated that mobile teeth treated with regeneration did not respond as well as non-mobile teeth. However, no association was drawn between mobility and occlusal forces.[50]

The relationship between cusps is an important factor in the transmission of occlusal forces to the periodontium.[51] Due to the limitations of clinical diagnosis of occlusal trauma and ethical considerations, most clinical studies have focused on teeth with occlusal discrepancies/disharmonies, which are defined as "contacts of opposing surfaces of teeth that are not in harmony with each other or with the anatomic and physiologic control of the mandible."[4] In an early retrospective study, the relationship between periodontal parameters and molar non-working contacts was examined.[52] It was found that molar teeth with non-working contacts had greater probing depths and bone loss compared with those without non-working contacts. Conversely, other studies looked at occlusal disharmonies in patients with periodontitis and failed to find any correlation between abnormal occlusal contacts and periodontal parameters, including probing depth, clinical attachment level, and bone loss.[6,7,53] Nevertheless, teeth with frank signs of occlusal trauma, including fremitus and a widened periodontal ligament space, demonstrated greater probing depth, clinical attachment loss, and bone loss.[7]

A series of retrospective studies investigated the association between occlusal discrepancies and the progression of periodontitis in a private practice setting.[54,55] All patients included had moderate to severe chronic periodontitis. These studies found that teeth with occlusal discrepancies had significantly deeper initial probing depths, more mobility, and poorer prognoses than those teeth without occlusal discrepancies.[54] Teeth with occlusal discrepancies demonstrated a significant increase in probing depth and a worsening prognosis with time. Multiple types of occlusal contacts, including premature contacts in centric relation, posterior protrusive contact, non-working contacts, combined working and non-working contacts, and the length of slide between centric relation and centric occlusion were associated with significantly deeper probing depths and increased assignment to a less favorable prognosis.[55] In a more recent cross-sectional epidemiologic study, the non-working side contact was also associated with deeper probing depth and more clinical attachment loss.[56]

Based on those observations, if occlusal trauma has any relationship to the progression of periodontitis, then its elimination should improve clinical periodontal conditions. Occlusal adjustment is defined as "reshaping the occluding surfaces of teeth by grinding to create harmonious contact relationships between the maxillary and mandibular teeth."[4] The evidence linking occlusal adjustment to improvement in periodontal parameters is limited. In an earlier study, the flow rate and quality of gingival crevicular flow (GCF) after removal of occlusal interferences was examined in patients with advanced periodontitis.[57,58] It was found that occlusal adjustment reduced the protein content and collagenase activity without affecting the quantity of GCF. Later, a well-controlled clinical trial was conducted to evaluate the effect of the occlusal adjustment on healing outcomes after periodontal treatment.[59] In this study, half of the patients received occlusal adjustment by selective grinding before receiving surgical or non-surgical periodontal therapy. The other half did not receive occlusal adjustment. After healing, the group that received occlusal adjustment before periodontal treatment gained 0.4 mm improvement in mean clinical attachment levels compared with those without pre-treatment occlusal adjustment. However, it was noted that the post-treatment reduction of probing depth and mobility were comparable. During long-term periodontal maintenance, the parafunctional habits that are not treated with a bite guard and the presence of mobility were both associated with increased clinical attachment loss and tooth loss.[60,61] In another study conducted in a private practice, the response of patients with periodontitis and occlusal discrepancies to occlusal adjustment

was examined. Regardless of the periodontal treatment status, the probing depth of teeth with untreated occlusal discrepancies was increased by a mean of 0.066 mm/year while a decreased probing depth of 0.122 mm/year was noted on teeth with occlusal adjustment.[62]

Collectively, these clinical studies demonstrated the added benefit of occlusal therapy in the management of periodontal disease, but they do not provide strong evidence to support routine occlusal therapy. Clearly, occlusal therapy is not a substitute for conventional periodontal treatment for resolving plaque-induced inflammation. However, it may be beneficial to perform occlusal therapy in conjunction with periodontal treatment in the presence of clinical indicators of occlusal trauma, especially relating to the patient's comfort and masticatory function. The patient's occlusion should be carefully examined and recorded before and after treatment. The occlusion of periodontally compromised teeth should be designed to reduce the forces to be within the adaptive capabilities of the reduced periodontal attachment. Overall, in the presence of occlusal trauma, occlusal therapy may slow the progression of periodontitis and improve the prognosis.

Excessive occlusal forces and abfraction

In the late 1970s, excessive occlusal loading was first proposed to cause cervical stress that results in the formation of non-carious cervical lesions (NCCLs).[15] This purported occlusally generated lesion was termed abfraction.[11,13] Although there is theoretic evidence in support of abfraction, predominantly from finite element analysis (FEA) studies, caution is advised when interpreting results of these studies because FEA does not replicate a clinical situation.[63–69] In FEA models, different researchers have assumed significantly different physical properties of the dental tissues. Also, arbitrary magnitudes, directions, and durations of forces have been used, which makes comparison between studies difficult. Cross-sectional studies have indicated associations between NCCLs, bruxism, and occlusal factors, such as presence of occlusal wear facets, group function, and premature contacts, but these investigations do not confirm causal relationships.[9,70–74] Despite the positive association, the size of NCCLs and the extent of occlusal wear was not correlated.[9]

Only a few studies have sought evidence for a causal relationship between occlusion and NCCLs.[75–77] An increased incidence of NCCLs was associated with presence of occlusal wear facets after a 3-year follow-up in a group of dental students.[75] To the contrary, in a split-mouth design, it was shown that the elimination of excursive interferences by occlusal adjustment did not decrease the progression NCCLs.[76] More recently, a 5-year prospective clinical trial found that progression of NCCLs was associated with relative occlusal forces in maximum intercuspation position, but not diet, toothbrushing, presence of occlusal wear facets, group function, or parafunctional habits.[77] If excessive occlusal forces were contributing to the etiology of NCCLs, it would be expected that parafunctional habits, such as bruxism and clenching, would exacerbate the progression of NCCLs. Two studies have reported a correlation between self-reported bruxism and NCCLs.[78,79] Although some studies suggested an association, the causal relationship between excessive occlusal forces and the progression of NCCLs is still uncertain. Therefore, abfraction is still a biomechanically based theoretic concept, and it is not supported by appropriate clinical evidence.

Effects of excessive occlusal forces on gingival recession

Historically, it has been suggested that excessive occlusal force might be a factor in gingival recession and the loss of gingiva.[2,3] The term “Stillman's cleft” is defined as narrow, triangular-shaped gingival recession on the facial aspect of the tooth. It was postulated that excessive occlusal force caused the Stillman's cleft. However, these historic references are based on uncontrolled clinical observations.

By examining teeth with gingival recession, no correlation was identified between mobility and gingival recession.[80] Compared with contralateral teeth without recession, teeth with recession showed either no or similar mobility. In a clinical investigation on the etiology of gingival recession, a positive association between occlusal trauma and gingival recession was reported;[16] however, this association disappeared when tooth malposition was present. In evaluation of the relationship between incisor inclination and periodontal status, labial gingival recession of the mandibular incisors was related to linguoversion.[81] However, there was no further analysis of the functional occlusal relationship. A recent retrospective study also failed to establish a relationship between the presence of occlusal discrepancies and initial width of the gingival tissue or between occlusal treatment and changes in the width of the gingiva.[82] Hence, existing data do not provide any solid evidence to substantiate the effects of occlusal forces on NCCLs and gingival recession.

Effects of orthodontic forces on the periodontium

Clinical studies have demonstrated that with good plaque control, teeth with a reduced but healthy periodontium can undergo successful tooth movement without compromising the periodontal support.[83,84] However, a non-controlled orthodontic force can negatively affect the periodontium and result in root resorption, pulpal disorders, and alveolar bone resorption.[85,86]

The long-term effects of orthodontic forces on the periodontium have been controversial.[87–91] A recent systematic review demonstrated that orthodontic therapy was associated with 0.03 mm of gingival recession, 0.13 mm of alveolar bone loss, and 0.23 mm of increased pocket depth when compared with no treatment.[92] Overall, the existing evidence suggested that orthodontic treatment has minimal detrimental effects to the periodontium.

CONCLUSIONS

Animal and human studies have indicated some association between occlusal trauma/occlusal discrepancies and progression of

periodontal disease. Nevertheless, all investigators agreed that excessive occlusal forces do not initiate plaque-induced periodontal diseases or loss of periodontal attachment, and more recent studies support this conclusion. In addition, based on the existing data, there does not appear to be any scientific evidence to prove that excessive occlusal forces cause abfraction or gingival recession.

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