Occlusion in a Periodontal Context

Mariano Sanz, MD, DDS, Dr.Med

Mariano Sanz is a professor of periodontology and the dean of the Faculty of Odontology, Universidad Complutense de Madrid, Spain. He received his specialty training at the University of California at Los Angeles and was awarded the Jens Waerhaug Research Prize by the Scandinavian Society of Periodontology in 1984. He has made extensive contributions to the specialty literature and has given over 200 invited lectures to national and international dental audiences.

Trauma from occlusion is a term used to describe the pathological alterations or adaptative changes that develop in the periodontium as a result of undue force produced by the action from the jaw muscle system. These pathological changes are expressed clinically and radiologically as increased tooth mobility and a widening of the periodontal ligament space. In addition to producing damage in the periodontal tissues, excessive occlusal force may also cause injury leading to temporomandibular disorders or unwanted wear of tooth structure. These traumatizing forces may act on an individual tooth or on a group of teeth in premature contact relationship. They may occur in conjunction with parafunctions, such as clenching and bruxism, or they may act on a healthy periodontium or on a periodontium reduced as a consequence of destructive periodontal disease. In the latter case, occlusal trauma may be aggravated by the migration of teeth in the posterior dentition with accompanying spread of the anterior teeth of the maxilla, the so-called bite collapse syndrome.

Trauma from occlusion was defined classically as a condition in which injury results to the supporting tissues of teeth by the act of bringing the jaws into a closed position, resulting in microscopic periodontal ligament pathology leading to reversible tooth mobility. It was once claimed that apart from increased tooth mobility, the presence of angular bony defects in the affected teeth was also an important symptom of trauma from occlusion. The validity of this suggestion, however, was questioned and discarded after both animal experimental studies as well as clinical studies in humans.

In spite of many years of investigation aiming to prove this relationship between traumatic occlusion and periodontal pathology, clear evidence still does not exist. Early studies on trauma from occlusion typically used autopsy material and provided no information on the periodontal conditions and occlusal forces that occurred before the studies. By their nature, cross-sectional studies failed to prove any cause-and-effect relationship between trauma from occlusion and the severity of plaque-induced periodontal disease. The prospective studies lacked well-defined experimental models of trauma from occlusion, and the periodontal conditions were usually poorly controlled. Nevertheless, experiments carried out in humans as well as animals have produced evidence that neither unilateral forces nor jiggling forces applied to teeth with a healthy periodontium result in pocket formation or loss of connective tissue attachment; therefore, it is clear that trauma from occlusion cannot induce periodontal tissue breakdown. Trauma from occlusion does, however, result in resorption of alveolar bone, which leads to increased transient or permanent tooth mobility.

In teeth with progressive plaque-associated periodontal disease, trauma from occlusion may, under certain conditions, enhance the rate of progression of the disease. However, whether tooth hypermobility and trauma from occlusion act as co-destructive factors in the progression of periodontitis is a subject of debate. It is also controversial whether these factors interfere with healing in the treatment of periodontal diseases, although controlled clinical trials have recently shown that severe tooth mobility can negatively affect the clinical outcomes of periodontal regenerative techniques. Increased tooth mobility can indeed be observed in conjunction with trauma from occlusion. It may, however, also be the result of a reduction of the height of the alveolar bone caused by plaque-associated periodontal disease.

Trauma from Occlusion and Tooth Hypermobility in the Treatment of Periodontal Disease

Since increased tooth mobility may indicate that the periodontal structures have become adapted to an altered functional demand, in cases where the resulting widened periodontal ligament has a normal tissue composition indicating a lack of pathology, controversy still exists regarding when to treat hypermobile teeth.

Traditionally, a reduction in tooth mobility has been considered beneficial for the periodontal tissues, but the need for occlusal equilibration and fixation of the hypermobile teeth as integral parts in the treatment of periodontal disease is still controversial. Therefore it is still not clear whether taking measures to reduce tooth mobility when teeth are hypermobile has any advantage in regard to healing and improved tooth prognosis. Studies, mainly animal studies, using different approaches to experimentally induce trauma from occlusion have had controversial results when assessing the histologic response to periodontal therapy. Studies in the beagle dog demonstrated that the histologic gain in connective tissue attachment after periodontal therapy in teeth subjected to jiggling trauma from occlusion was not as favorable as in nontraumatized controls.

Similarly, in recent clinical studies in humans, probing attachment level gains were not as favorable in hypermobile teeth as in nonmobile controls, and occlusal adjustment to reduce tooth mobility prior to initial periodontal treatment was correlated with an increase in probing attachment gain following therapy.

However, in another study with a split-mouth design, occlusal adjustment and splinting of the test teeth revealed no differences when the splinted teeth were compared with the nonsplinted controls after scaling and root planing of all teeth or after subsequent periodontal surgery. One might therefore argue that occlusal adjustment for reduction in tooth mobility should not be done until after periodontal treatment. Well-designed prospective studies are needed to prove the impact of treating occlusal trauma or hypermobility within the different periodontal treatment modalities. However, since periodontal diseases are infectious in nature, the main thrust in their treatment and prevention must be the reduction in total bacterial mass or the reduction and possible elimination of specific micro-organisms that have been proven to be specific pathogens. In addition to systemic factors, which may affect host response to bacterial antigens, local factors such as trauma from occlusion and tooth hypermobility must be considered for their potential role as cofactors in the progression of periodontal diseases and their possible interference with optimal treatment of the diseases.

Considering the aforementioned research, perhaps only progressively increasing tooth mobility which may occur in conjunction with trauma from occlusion and which is characterized by active bone resorption should be considered pathological and treated. In these situations, any occlusal forces making a furcationally involved tooth hypermobile should be eliminated by selective grinding. This might reduce the negative impact of tooth mobility as a potential cofactor in the progression of poorly controlled periodontitis.

Conclusions

Excessive occlusal forces may or may not cause tooth hypermobility.

1. Characteristics of occlusal forces other than magnitude, such as velocity, duration, and frequency, may be of greater significance for the development of tooth hypermobility.
2. Hypermobility produced by occlusal forces does not cause gingival inflammation or aggravate the severity of chronic, nonprogressive gingivitis, nor does it act as a primary etiologic factor for connective tissue attachment loss.
3. Occlusal forces producing tooth hypermobility may accelerate attachment loss in progressive periodontitis.
4. Occlusal forces producing tooth hypermobility may interfere with optimal healing in the treatment of periodontal diseases. The role of occlusal equilibration and stabilization of hypermobile teeth within periodontal therapy remains controversial.
5. Tooth hypermobility and trauma from occlusion may act as codestructive factors in the progression of periodontitis. In these circumstances, occlusal adjustment could be an effective therapy to reduce increased tooth mobility and mprove the prognosis of the affected tooth.
6. Increased tooth mobility as a result of reduced height of the alveolar bone can be accepted and splinting avoided, provided that the occlusion is stable and that the degree of mobility does not interfere with the patient's chewing ability or comfort. Splinting may be considered when the periodontal support is so reduced that the mobility of teeth is progressively increasing.

Suggested Reading

1. Ericsson I, Lindhe J. Lack of effect of trauma from occlusion on the recurrence of experimental periodontitis. J Clin Periodontol 1977;4:115–127.
2. Ericsson I, Lindhe J. Effect of long-standing jiggling forces on experimental marginal periodontitis in the beagle dog. J Clin Periodontol 1982;9:497–503.
3. Fleszar TJ, Knowles JW, Morrison EC, Burgett FG, Nissle RR, Ramfjord SP. Tooth mobility and periodontal therapy. J Clin Periodontol 1980;7:495–505.
4. Galler C, Selisky H, Phillips C, Ammonds WF. The effect of splinting on tooth mobility. 2. After osseous surgery. J Clin Periodontol 1979;6:317–333.
5. Glickman I, Smulow JB. Adaptative alterations in the periodontium of the rhesus monkey in chronic trauma from occlusion. J Periodontol 1968;39:101–105.
6. Hanamura H, Houston F, Rylander H, Carlsson GE, Haraldson T, Nyman S. Periodontal status and bruxism. A comparative study of patients with periodontal disease and occlusal parafunctions. J Periodontol 1987;58:173–176.
7. Jin LJ, Cao CF. Clinical diagnosis of trauma from occlusion and its relationship with severity of periodontitis. J Clin Periodontol 1992;19:92–97.
8. Kegel W, Selipsky H, Phillips C. The effect of splinting on tooth mobility. I . During initial therapy. J Clin Periodontol 1979;6:45–48.
9. Kerry GJ, Morrison EC, Ramfjord SP, et al. Effect of periodontal treatment on tooth mobility. J Periodontol 1982;53:635–638.
10. Lindhe J, Ericsson I. The influence of trauma from occlusion on reduced but healthy periodontal tissues in dogs. J Clin Periodontol 1976;3:110–122.
11. Lindhe J, Svanberg G. Influence of trauma from occlusion on progression of experimental periodontitis in the beagle dog. J Clin Periodontol 1974;1: 3–14.
12. Lindhe J, Nyman S, Ericsson I. Trauma from occlusion. In:.Lindhe J, Karring T, Lang NP (eds). Clinical Periodontology and Implant Dentistry. Oxford: Blackwell, 2003.
13. Philstrom BL, Anderson KA, Aeppli D, Schaffer EM. Association between signs of trauma from occlusion and periodontitis. J Periodontol 1986;57:1–6.
14. Polson AM. Trauma and progression of marginal periodontitis in squirrel monkeys. J Periodontal Res 1974;9:108–113.
15. Svanberg Gk, King GJ, Gibbs CH. Occlusal considerations in periodontology. Periodontol 2000, 1995;9:106–117.