The Role of Occlusion in the Etiology and Therapy of Periodontal Disease

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Occlusal function and its relation to periodontal disease has been debated since Karolyi[1] in 1901 first suggested such an interaction. From that time on, innumerable reports have appeared in the dental literature which, by clinical or experimental procedures, attempted to delineate the role of occlusal trauma in periodontal breakdown.

Occlusal trauma has been defined as "that force or forces caused by mandibular movement and resultant tooth percussion capable of producing pathologic changes in the periodontium. Occlusal traumatism is the term applied to the tissue changes which occur as the result of this trauma. These tissue changes are limited primarily to the attachment apparatus . . . and are noninflammatory in character."[2] At the site of pressure, they essentially consist of: 1) resorption of bone and sometimes, cementum, 2) necrosis of the periodontal fibers, 3) hemorrhage and 4) thrombosis of vessels. At the site of tension one notes histologically 1) opposition of bone and, at times, cementum and 2) alterations and elongation of the periodontal fiber apparatus. These changes have been observed in humans and, also, in animals in which excessive occlusal overloading was experimentally produced.[3, 4] However, gingival inflammation was not induced by these experimental procedures. In experimental animals, repair of this injury occurred rather rapidly and adaptation of the periodontal structures to the newly-created functional demands usually took place.[5]

Descriptions as cited above, while based on extensive evidence, do not focus sufficiently on the dynamic interaction that must take place within the periodontal tissues when teeth come into contact, since they do not evaluate such variables as a) duration of contact, b) severity of contact and c) response potential of the host at time of contact. Once these and other variables are accepted as significant factors affecting the ultimate response of the tissue to the force of contact, our static descriptions become more limited in significance. It is within such a framework that we wish to evaluate current findings and, as therapists, need to define criteria for diagnosis and treatment of a malfunctioning occlusion.

Traditionally, there are certain tissue changes by which we recognize periodontal pathology. Among these are gingival inflammation, tooth mobility, loss of alveolar support, and pocket formation. In this discussion we must ask whether malocclusion initiates or accelerates any of these periodontal changes.

Gingival Inflammation

It has been demonstrated that abnormal forces placed on teeth do not initiate gingival inflammation. Whether or not they contribute to an altered or accelerated rate of infiltration is not clearly understood.

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Tooth Mobility

While, at this time, we cannot define the exact point at which mobility exceeds the adaptive capacity of the periodontium and becomes pathogenic, we have data available which deal with mobility responses to a variety of local and metabolic factors. For example, horizontal mobility showed a decreasing trend from morning to night and was increased after a period of sleep.[6] These findings support the concept that prolonged hypofunction increases tooth mobility.[7] Tooth mobility ranges also were influenced by changes in capillary tone and blood circulation found during pregnancy, and other metabolic factors affecting circulation.[7]

From a local point of view, removal of labial alveolar bone resulted in mobility increments which, however, were significantly less drastic than those observed following the application of an interdental wire ligature left in situ for four hours.[7] Yet, even these more dynamic findings must be tempered by observations that tube-type feeding, which required no mastication, caused no change of tooth mobility in four out of five human subjects over a thirty-three day period. Therefore, as concluded by the authors, "occlusal forces received by the teeth during swallowing are sufficient to maintain them in their proper place." However, stress conditions which induced bruxism increased tooth mobility, even though the individuals were maintained on a tube-type diet.[8] These examples are cited to depict the variability of factors that must be taken into account when tooth mobility studies are undertaken and results interpreted. If results from such studies ever need to be compared, it becomes obvious that experiments must use comparable devices for measuring of tooth mobility and include in their methodology an awareness of its multifaceted causation.

In this relation interest has recently been shown in mechanisms for variations in tooth mobility. An intriguing concept has suggested that the immediate response of the tooth to applied forces may be due to fluid changes rather than cellular activity with blood acting as a shock absorber to counteract applied forces.[9] In experimental animals such changes have indeed been demonstrated in the early stages of periodontal tissue responses to pressure.[10, 11] In one study increased vascularization has even been observed to persist as long as 120 days after initiation of the experimental occlusal trauma.[12] If vascular changes are the primary responses to the application of forces to teeth, one may wonder if generalized changes in vascular structures alter the capability of the periodontium to respond to these applied forces and whether this, in turn, would affect the integrity of the periodontal tissues.

Taking this concept one step further, we might speculate that other host factors altering tissue capacity to adapt and repair may also affect the periodontal tissue's ability to withstand forces applied to teeth. Using excessive occlusal forces, delayed repair has been demonstrated in animals suffering from alloxan diabetes,[13] generalized debilitation[14] and nutritional deficiencies.[15, 16]

From a therapeutic point of view we must now ask of what relevance is altered tooth mobility to the maintenance of functioning teeth? No clear-cut evidence is currently available to answer this question and the initiation of such longitudinal studies is urged. The data at hand suggest that, in humans, "increased mobility is not synonymous with increased bone resorption" nor does duration of occlusal stress accelerate bone loss. It has also been shown that tooth mobility may be considerably more influenced by oral hygiene patterns than occlusal stress. Lovdal et al.,[17] therefore,

conclude that their study "does not exclude the possibility that trauma in singular cases may contribute to the destruction of the supporting bone. However, such cases do not seem to bring traumatic occlusion in the range of important etiologic factors in periodontal disease. Therapeutic measures which are based on the supposition that the destruction of supporting structures is caused by traumatic occlusion should, consequently, be used with caution." Tooth mobility has also been reduced by local periodontal therapy including selective grinding[18] and in patients in whom gingival irritants alone were removed,[7] while gingivectomy increased tooth mobility in the early stages after surgery.[19] These studies suggest a possible association between tooth mobility and gingival inflammation in which tooth mobility may be a periodontal response to inflammation. Extensive clinical evaluations of this relation are not yet available.

Loss of Alveolar Support and Pocket Formation

Glickman has recently conceptualized that trauma from occlusion when combined with inflammation can produce infrabony pockets and angular or crater-like osseous defects.[20] These data are based on monkey experiments in which teeth "subjected to long-term excessive pressure such as that produced by marked attrition, both moderate and severe pressure changes persisted in the periodontium. In some instances, these were observed in relation to the same tooth surface. When the moderate changes were located in the crestal area, the inflammation from the gingiva extended directly into the altered periodontal membrane."[21] In human autopsy specimens the same authors noted the relationship of occlusal forces and the spread of gingival inflammation into the supporting periodontal tissues and concluded that "when gingival inflammation and trauma from occlusion occur together they produce specific types of periodontal pathology such as angular bone destruction and infrabony pockets."[22] In a subsequent article the authors, again using autopsy specimens, suggested that the degree of inflammation and trauma from occlusion may be significant determinants whether or not angular defects or infrabony pockets occur.[23] This emphasis on degree is of particular interest, since alterations in the pathway of inflammation may be due to anatomic variants of the alveolar crest.[24] Furthermore, other studies have not been able to demonstrate such alterations in the inflammatory pathway. For example, Ramfjord attempted to overload anterior teeth in humans by extraction of posterior teeth (as part of immediate denture construction) and studied the effects of this altered function histologically. He concluded that the most stable periodontal structures, with regard to functional changes in occlusal stress, appeared to be the transeptal fibers. Most of the adaptation to the altered occlusal function seemingly took place at the surface of the alveolar bone and the middle zone of the periodontal membrane.[25] These comments are particularly important since fenestrations of the labial alveolar plate are usually seen in its midzone,[26] and the etiology of these breaks in alveolar continuity is unknown. Yet, their presence may have significant effect on the outcome of various aspects of periodontal surgery.

Other human studies have also been unable to histologically demonstrate a cause and effect relation of forces applied to teeth and infrabony pocket formation. This summary statement is based on 1) our analysis of histologic changes in four adult surgical specimens,[27] 2) experiments in which abnormal occlusal stresses by means of high inlays were created in a young

individual over a 9 month period and the tooth and alveolus subsequently extracted[28] and 3) twenty premolar specimens in seven children in whom the teeth were orthodontically tipped and movement effected at 5 to 6 day intervals using forces between 100 to 150 g. over a 6 week period. Half of the teeth together with the buccal bone and mucosa were removed at that time. The remaining teeth were maintained for a three-months retention period and then extracted, again with removal of the buccal plate and mucosa. Tooth mobility increased during orthodontic movement and decreased during retention. Analysis of histologic findings showed no apical migration or pocket formation.[29] Finally, a recent experiment[30] used high crowns to produce a jiggling effect and overhanging margins to intensify local inflammation in adult monkey premolars over a 13 week period. These procedures failed to induce periodontal pockets, but did cause the adaptive responses previously reported in high filling experiments. It thus appears that no conclusive statements can be made regarding the effects of combined inflammation and occlusal trauma on the etiology of infrabony pockets. Since these lesions and their therapy are of great interest to periodontists, studies attempting such evaluations must crucially examine their methodology in order to evaluate and control the degrees of inflammation and applied forces, as well as the status of the host. Otherwise, conflicting reports will continue to appear in the literature and findings derived from different investigations will not be comparable. Such conflicts create confusion for the clinician and may either delay efficacious therapy or unnecessarily complicate it.

An evaluation of data summarized thus far clearly indicates the need for precise quantitation in experiments designed to evaluate the effects of tooth contact on the periodontium. Recently, intraoral telemetric systems have been developed.[31, 32] Obviously, the use of such devices is extremely limited. Yet, they are providing data on tooth contacts in a most physiologic environment. Results from such studies have indicated that most chewing and swallowing contacts may occur in functional rather than posterior position (centric) occlusion, while bruxism consists of regular repetitive grinding tooth contacts which differ from the haphazard pattern recorded during mastication.[33, 34] Furthermore, the effect of a single occlusal interference on the number and type of occlusal contacts during mastication did not lead to avoidance, but rather to an immediate adaptation of the stomatognathic system to the occlusal obstacle, since contacts were not reduced. These results, of course, do not rule out the possibility that long-range adaptation to an occlusal interference might not lead to a change of chewing patterns.[35] Such studies need further amplification. However, they do indicate the adaptive range of the periodontium.

Finally, in a clinical survey of patients suffering with periodontitis, an attempt was made to relate periodontal disease severity with such occlusal abnormalities as crowding of teeth and rotation of teeth. No such correlation could be established. A correlation between localized periodontal disease severity and teeth in crossbite or tilted teeth was demonstrated.[36] However, one wonders if poorer oral hygiene in such areas could not have contributed to this association. These findings are similar to those observed in children, where no correlation between gingivitis and malocclusion could be established.[37]

Summary

The basic question facing the periodontally-oriented dentist is "Does an

abnormal or excessive occlusal force detrimentally affect the periodontal health of the teeth involved?"

Our current state of the art indicates that, to date, experiments have often been static in conception. Yet, they have been useful in demonstrating clinical and histologic responses to overloading, namely, a range of noninflammatory adaptations of the periodontal structures to the newly created environment. Since findings have been similar in both humans and experimental animals, the data appear sufficient for such descriptive information.

The above cited experiments, however, did not measure degree of overloading or provide a record of the dynamic flow of responses once the irritant was placed. Such information is vitally needed since it might provide us with the "breaking point" at which adaptation ceases and pathology begins. We therefore need to apply more sophisticated measuring devices, such as telemetry, for the prolonged study of responses to overloading in conjunction with evaluation of changes visualized by clinical measurements and histologic analysis. Obviously, such findings are cumbersome to obtain and are limited to a small sample size. But they may provide the key data which could then be confirmed by less complicated techniques, such as mobilometry, in which larger samples can be used.

Until precise data are obtained determining the effects of various forms of overloading on the adaptive capacity of the periodontium and specific types of overloading can be indicted as exceeding this adaptive capacity, extensive therapeutic alterations of occlusal patterns for the "improvement of periodontal health" can be questioned.

One might further suggest that therapists undertake evaluations of the effects of clinically-induced occlusal changes on the periodontium, using experimental designs of similar sophistication as suggested for the nontreated dentition. Such information would provide us with the "therapeutic trial" and should expand our knowledge of the role of occlusal patterns in maintaining periodontal health.

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