Occlusal forces as a risk factor for periodontal disease

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The relationship between occlusal forces and the initiation or progression of periodontal disease has been controversial for over a century. Early in the 20th century, excessive occlusal forces were considered to be a major causative factor of periodontal destruction. In 1901, Karolyi (28) indicated that there appeared to be a correlation between excessive occlusal forces and periodontal destruction. In 1917 and 1926, Stillman (56, 57) stated that excessive occlusal forces were the primary cause of periodontal disease and that occlusal therapy was mandatory for the control of periodontal disease. Stillman further advocated the use of occlusal adjustment to prevent periodontal destruction. This set the stage for a controversy that continues to this day: What roles, if any, do excessive occlusal forces play in the initiation and progression of periodontal disease?

Historical perspective

As noted above, several early authors indicated that occlusal forces played a significant role in the initiation and progression of periodontal destruction. However, these statements were based on clinical observations rather than scientific evaluation. In the 20 years following Stillman’s statement that excessive occlusal forces were the primary cause of periodontal destruction, several animal studies on sheep and monkeys were carried out in an attempt to elucidate the response of the periodontium to occlusal forces (1, 58). The results of these studies were interpreted to show that excessive occlusal forces were at least a contributing factor in the progression of periodontal disease. At the end of the 1930s it was still felt that excessive occlusal forces were a major cause of periodontal disease, that occlusal adjustment should be part of periodontal treatment, and that occlusal discrepancies should be prophylactically treated to prevent periodontal disease (2, 3, 35).

Orban & Weinman, in 1933, used the histologic observation of human autopsy material as a methodology for the evaluation of the effect of excessive occlusal forces on the periodontium (40, 66). They concluded that occlusal forces did not have a major effect on periodontal destruction and felt that there was no indication that occlusal forces played a part in periodontal destruction. Instead, they interpreted this material as demonstrating that gingival inflammation extending into the supporting bone was the cause of periodontal destruction.

During the 1950s and 1960s, further animal research was performed. These studies used rats, monkeys, and dogs to evaluate the effect of occlusal forces on the periodontium (5, 9, 34, 43, 67). These studies, by their use of untreated controls, controlled occlusal forces, experimentally induced periodontitis, and larger number of subjects, introduced a new level of sophistication in the evaluation of the effect of occlusal forces. None of these studies gave support to the concept that excessive occlusal forces were a primary causative agent of periodontal destruction and many of the studies indicated that there was little or no correlation between occlusal forces and periodontal destruction.

During this period Glickman and co-workers performed a series of animal model and human autopsy studies which strongly influenced the perception of the relationship between excessive occlusal forces and periodontal destruction. Animal studies where a “high” occlusal contact was created by overcontouring a restoration were performed utilizing dogs and monkeys (16, 22). These studies showed no evidence that occlusal contacts initiated periodontal destruction. However, in a study on Rhesus monkeys, a phenomena that was described as an “altered pathway of destruction” was noted on teeth where excessive occlusal forces were present (19). This altered pathway of destruction was described as a change in the orientation of the periodontal and gingival fibers
which occurred in the presence of excessive occlusal forces that allowed gingival inflammation to extend along the periodontal ligament. This altered pathway of destruction was felt to result in vertical bony defects where bone loss followed the periodontal ligament. Another animal study (21) showed that bifurcation areas were most prone to stress from occlusal forces and it was postulated that bone loss in the furcation area could be related to excessive occlusal forces. From these studies and later works by Glickman came the concept that vertical bony defects and furcation defects were often associated with excessive occlusal forces.

Glickman and coworkers (4) also performed a series of studies using human autopsy material. One of these studies looked at the histomorphology of infrabony pockets. The authors felt that the histologic material showed evidence that occlusal forces affected the attachment apparatus apical to the bony defect. Later studies (17, 18) using several human block sections were interpreted to show that occlusal forces and gingival inflammation were two separate processes and that occlusal forces altered the environment in which gingival inflammation affected the bone. They felt that the occlusal forces changed the alignment of the periodontal ligaments, allowing for an altered path way of destruction with the end result being vertical bony defects. Because two separate pathologic processes worked together to cause bone loss, the process was termed a "co-destructive" effect.

Glickman and coworkers summarized their work in a series of review articles (12–15, 20). These articles indicated that excessive occlusal forces (trauma from occlusion) were a co-destructive force which in the presence of gingival inflammation could lead to vertical osseous defects. Based on this, the use of occlusal adjustment was advocated as part of the treatment for existing periodontal disease. Because no evidence existed that excessive occlusal forces initiated periodontal disease, occlusal adjustment to prevent periodontitis was not advocated.

Waerhaug (62–64) performed studies that utilized a large number of human autopsy specimens in which he evaluated the relationship of the morphology of the bone pocket with the plaque level and presence or absence of excessive occlusal forces. He found that the "plaque front" (i.e., the border of the subgingival plaque) was always in very close approximation to the epithelial attachment level, always resembled the morphology of the bony defect, and that the relationship of the plaque level between adjacent teeth (either at the same of different apico-coronal levels) would yield either horizontal or vertical interproximal bone loss. He also observed that excessive occlusal forces bore no relationship to the underlying bony defect and that vertical defects were found equally around traumatized and non-traumatized teeth.

Waerhaug’s conclusion was that bone loss was always associated with the downgrowth of plaque and there was no relationship between excessive occlusal forces and vertical bone loss.

It should be pointed out that a common difficulty encountered in almost all of the occlusal studies that used human autopsy material is a lack of knowledge of the patient’s occlusal relationship in life. While certain inferences can be derived from wear patterns, there is no assurance that the teeth actually occluded in the assumed manner. Therefore, any statement, based solely on autopsy material, that a particular tooth was undergoing excessive occlusal forces at the time of death can be questioned and any conclusions about the effects of excessive occlusal forces on the periodontal supporting structures cannot be definitive. A single study (55) evaluated the patient’s occlusal relationship prior to the removal of the jaws for cancer therapy. Only a single bony defect was found in the block sections and no obvious relationship between occlusion and periodontal pocketing could be determined from this limited material.

The results of two extensive animal studies were published beginning in the 1970s. These studies were aimed at evaluating the effect of plaque and excessive occlusal forces in the animal models utilized. Stringent scientific controls and design were used in all of these studies. These studies were performed separately by Polson and coworkers (27, 41, 42, 44, 45, 47–51) and Lindhe and coworkers (6–8, 30–32, 38, 39, 59–61). Polson used squirrel monkeys and mesial-distal compression forces comparable to orthodontic forces, while Lindhe used beagle dogs and applied buccal-lingual forces using a high occlusal contact and a finger spring. Both groups looked at excessive occlusal forces with and without plaque as well as histologic healing which occurs when excessive occlusal forces are applied for a period of time and then removed. In the beagle dog study, surgical defects were created and the effect of excessive occlusal forces and plaque were evaluated.

The results of these studies were very similar despite the differences in the animal model and the manner of applying excessive occlusal forces. Excessive occlusal forces not in the presence of plaque were found to cause loss of bone density and mobility of the tooth but no evidence was found that excessive occlusal forces alone would cause attachment loss. When the excessive occlusal forces were...
removed it was found that the loss of bone density was reversible. In the presence of plaque, inflammation of the gingiva and periodontal supporting structures were noted and in the presence of excessive occlusal forces and plaque together there was an indication that more bone density was lost in both animal models. In the beagle dog model there was evidence of attachment loss when plaque and excessive occlusal forces were both present. This was not found in the squirrel monkey model.

These two series of studies were exhaustive in their evaluation of the relationship of occlusal forces and plaque in an animal model. Both studies concluded that there was no evidence that excessive occlusal forces alone caused loss of attachment. The studies by Lindhe showed that in special circumstances that there may be attachment loss when plaque and excessive occlusal forces were both present. Both studies agreed that the control of plaque and gingival inflammation would stop the progress of periodontal disease in the presence or absence of excessive occlusal forces. These studies helped to establish that bacterial plaque is the initiating factor and the main cause for progression of periodontal disease.

Human studies

There is a paucity of studies evaluating the effect of occlusion in humans. This is due in part to ethical difficulties related to the non-treatment of diagnosed periodontal disease. In order to perform a controlled clinical trial, the gold standard of clinical research, it is necessary to compare treatment methods. However, in evaluating the combined effects of excessive occlusal forces and periodontal disease, it would be necessary to treat one group of patients and leave the other group untreated. This approach would be ethically unacceptable due to the known deleterious effects of the non-treatment of periodontal disease. The World Workshop in Periodontics stated “Prospective studies on the effect of occlusal forces on the progression of periodontitis are not ethically acceptable in humans” (11). As a result, most occlusal studies in humans have been descriptive and/or retrospective in nature.

It has been reported that patients who have occlusal discrepancies have no more severe periodontal destruction than do patients without occlusal discrepancies (26, 29, 42, 52–54). However, it has also been reported that molars with furcation invasion and mobility have greater pocket depths than molars that are not mobile (65). The increased mobility noted in this study may have been due to occlusal factors or to greater loss of bony support due to the furcation involvement. Due to the inability to determine whether occlusal factors or bone loss was first present, from this study, it is impossible to draw a clear relationship between occlusion discrepancies, mobility, and pocket depths. Other studies reported that patients who received occlusal adjustment as part of their periodontal therapy had greater attachment gain than patients who did not receive occlusal adjustment (3, 10). This study may be an indication that occlusal adjustment should be performed, where indicated, as a part of periodontal treatment. A series of reports on risk factors for periodontal destruction indicated that mobility and parafunctional habits that are not treated with a biteguard are associated with increased attachment loss, worsening prognosis, and tooth loss (36). These studies seem to indicate that untreated (i.e., no biteguard) parafunctional habits may contribute to increased periodontal breakdown.

A recent retrospective study evaluated patients from a private practice who were diagnosed with advanced periodontal disease and had a comprehensive treatment plan recommended that included occlusal adjustment if occlusal discrepancies were detected. Some of these patients self-selected to not have any periodontal treatment performed (untreated group). Other patients had only non-surgical periodontal treatment performed (partially treated group). Others followed through with all recommended periodontal treatment including surgery (fully treated group). The effect of occlusal discrepancies was studied in each of these groups using the individual tooth as the experimental unit (24, 25, 37). This means that the progression of periodontal destruction or the improvement of the periodontium for each tooth was followed over time. This study design allowed for the evaluation of teeth with occlusal discrepancies versus teeth without occlusal discrepancies rather than comparing patients with occlusal discrepancies vs. patients without occlusal discrepancies. This experimental approach differs from most past studies where the patient was the experimental unit and the changes in pocket depth or attachment levels were expressed as the “patient mean”. Using the patient mean may tend to mask changes that are occurring at the more active sites and, thereby, may give results that do not reflect what is actually occurring during localized disease progression.

These studies found that teeth with occlusal discrepancies had deeper presenting pocket depths and
a worse prognosis than those teeth that did not have occlusal discrepancies. Further, when teeth with occlusal discrepancies were followed over time, a significant increase in pocket depth and a worsening of prognosis was noted when compared to teeth without occlusal discrepancies. Additionally, teeth in the partially treated group that had received occlusal adjustment showed a slowing of the progression of periodontal destruction when compared to teeth with occlusal discrepancies that had not had occlusal adjustment. It was concluded that occlusal discrepancies appear to be a significant risk factor that contributes to more rapid periodontal destruction and that treatment of occlusal discrepancies seemed to slow periodontal destruction. The authors postulated that the reason for the difference in their findings and those of previous studies was the use of the individual tooth as the experimental unit, which they felt yielded a more accurate assessment of the effect of occlusal discrepancies on the periodontium.

Summary of literature review

Most early research on the effects of occlusion on the progression of periodontal disease focused on a cause and effect relationship. Stillman clearly felt that excessive occlusal forces were the cause of periodontal disease and that treatment of the occlusion was the primary method of effective periodontal treatment. As it became evident that bacterial plaque was an integral part of the periodontal disease process, the role of occlusal forces became less clear. Eventually this led to viewing occlusion as a cause of specific types of periodontal destruction. This was described by Glickman as the co-destructive roles of occlusion and bacterial plaque in the formation of vertical osseous defects and furcation bone loss. Glickman’s theory of Co-Destruction continued to hold to the thesis that occlusion was, in concert with bacterial plaque, a causative factor in periodontal attachment loss and bony destruction. Glickman described an altered pathway of destruction in an attempt to articulate a functional mechanism for the formation of the specific morphology of attachment and bone loss thought to be caused by the co-destructive action of occlusal forces and bacterial plaque. The altered pathway of destruction still held to the concept that occlusion directly changed the disease process and was thereby, in the presence of bacterial plaque, a causative agent for periodontal destruction.

The animal studies on squirrel monkeys and beagle dogs began to shed light on the effect of occlusal forces on the periodontal attachment structures at a cellular level. From these studies it was clear that within these animal models, occlusion had an effect on the periodontium in the form of bone rarefaction, which resulted in the clinical manifestation of mobility. However, it was equally clear that, within the animal models, loss of attachment and thereby periodontal destruction did not occur in the presence of excessive occlusal forces only. Loss of attachment occurred only in the beagle dog model and then only in the presence of excessive occlusal forces and bacterial plaque. While these animal studies gave us an exhaustive insight into the effect of excessive occlusal forces on the periodontal supporting structures of the studied animals, it must be remembered that these studies were performed using animal models that show little or no tendency toward periodontal destruction under natural conditions. The application of the information obtained from these animal models to the periodontal destruction that occurs in humans must be approached with caution. It is probable that these animal studies give us a picture only of the physiologic response of the periodontium to excessive occlusal forces with and without bacterial plaque. It is unlikely that these animal studies give us significant information about the pathophysiology that may occur when excessive occlusal forces are present in humans who may be genetically prone to periodontal destruction and who may also have additional risk factors for periodontal disease beyond occlusal forces and bacterial plaque.

Human studies begin to give us some indication of the effect of excessive occlusal forces on the progression of periodontal disease in those patients who show a tendency toward periodontal destruction. While there are many apparently contradictory findings from human studies, there appears to be a trend toward evidence that excessive occlusal forces may play a role in periodontal destruction and the response of the periodontium to periodontal treatment. While the available information suggests a relationship between excessive occlusal forces and progression of periodontal disease, the 1999 International Workshop for Classification of Diseases and Conditions indicated that there was no clear evidence that occlusal forces were a factor in plaque-induced gingival disease or connective tissue loss (23). Since the 1999 Workshop, studies have shown that occlusal interferences have a negative effect on the periodontium and tend to cause more rapid pocket formation and poorer prognosis when
compared to teeth that do not have occlusal interference.

Occlusal interferences as a risk factor

Recent human research seems to show a relationship between occlusal discrepancies and periodontal destruction (24, 25, 37). If the relationship found in these studies does exist, then excessive occlusal forces may need to be classified as a risk factor for periodontal destruction. Much of the confusion that exists about the role of occlusal forces in periodontal disease is based on over 100 years of research and controversy that has focused, in one way or another, on a causative role for occlusal forces in periodontal disease. The concept of risk factors for periodontal disease, as opposed to causative factors, is a relatively recent occurrence. When the body of information on occlusal forces in periodontal disease is reviewed in terms of occlusal forces as a potential risk factor instead of a causative or co-destructive agent, many of the contradictory results of past research may be more reconcilable. A large body of research exists which indicates that smoking is a significant risk factor for periodontal destruction, that there is a much higher incidence of periodontal destruction in smokers vs. non-smokers, and that smokers do not respond as well to periodontal treatment when compared to non-smokers. Despite the overwhelmingly strong link between smoking and periodontal destruction, smoking is not viewed as a causative or co-destructive agent for periodontal disease. Instead, smoking is viewed as a cumulative risk factor for periodontal destruction. Existing research makes a case for viewing occlusion from a similar prospective.

It is universally believed that bacterial plaque is the causative agent for periodontal destruction (33). 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 exact mechanism by which each individual risk factor affects this predisposition is unknown. The effect of the risk factor may be to alter the host response or change the bacterial environment or effect. It may be that some form of localized alteration in the host environment might be the mechanism by which excessive occlusal forces interact with bacterial plaque. It is also possible that excessive occlusal forces create an environment whereby the deleterious effects of bacterial plaque are enhanced, or there may be a completely different mechanism that has not been noted in the past. Under any circumstances, recent human research indicates that treatment of the occlusion to minimize occlusal interferences may, in concert with other forms of periodontal treatment, positively affect the progress and treatment of periodontal destruction. The risk factor of 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.

References