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Is there an association between occlusion and periodontal destruction?: Yes—occlusal forces can contribute to periodontal destruction.

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POINT

Is there an association between occlusion

Yes—occlusal forces can contribute to periodontal destruction.

Stephen K. Harrel, DDS; Martha E. Nunn, DDS, PhD; William W. Hallmon, DMD, MS

Controversy over the relationship between occlusion and progression of periodontal destruction has been ongoing since the beginning of scientific studies of dental diseases. This controversy often has been heated. Some respected researchers have stated strongly that occlusal forces are a major factor in periodontal destructions and that treatment of occlusal forces is a major part of the successful treatment of periodontal disease. Other equally respected researchers have stated just as strongly that there is no relationship between occlusal forces and periodontal destruction and that there is little justification for occlusal treatment as a routine part of periodontal therapy.

There is evidence that the treatment of occlusal discrepancies should be considered an integral part of the overall treatment of periodontal disease.

This article presents a brief review of the literature concerning the relationship between periodontal disease and occlusal forces. Additionally, we will review recent research we have performed and compare it with past research findings. We also will discuss our conclusion that occlusal discrepancies are a significant risk factor for the progression of periodontal disease and our reasoning for suggesting that treatment of occlusal discrepancies should be a routine part of periodontal therapy.

HISTORICAL STUDIES

For more than a century, clinicians have postulated that a relationship existed between occlusal forces and the progression of periodontal disease. Karolyi,[1] in the early 20th century, was one of the first to publish on the relationship of occlusion to periodontal disease. He indicated that teeth undergoing excessive occlusal stress seemed to have more periodontal destruction than did teeth not experiencing occlusal stress. Also in the early 20th century, Stillman, one of the early pioneers of periodontal therapy, presented the proposition that excessive occlusal stress was the cause of periodontal disease. Stillman indicated that to treat periodontal disease successfully, the clinician must control occlusal forces.[2,3] Stillman's comments led to several studies aimed at determining whether occlusion did or did not play a causative role in periodontal disease.[4-6] These studies failed to produce conclusive results, and the controversy continued.

In the 1940s, Weinmann[7] published one of the first studies to evaluate the relationship of occlusion and periodontal disease at a cellular level. On the basis of his observations of human autopsy material, he felt that periodontal disease was related to progression of an inflammatory process that began at the gingival attachment and spread

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and periodontal destruction?

Only in limited circumstances does occlusal force contribute to periodontal disease progression.

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Examining the long-standing controversy about the role of occlusion in periodontal disease is a delightful look back at more than 100 years of periodontal theory and practice. The list of authors who have written on this topic in the past century reads like a "Who's Who" of some of the brightest minds in dentistry, and the debate has endured through several defined eras in the history of the specialty of periodontics. From the days when periodontics was dominated by those initially trained as pathologists, through the period when the specialty was led by master clinicians headquartered at certain universities, through an era characterized by meticulously controlled human and animal studies conducted both in the United States and abroad, up to the current period of evidence-based therapy, the debate has persisted. It is a reminder that even in this modern era, dentistry still is very much an art as well as a science.

Like most long-standing controversies, the debate about occlusion and periodontal disease has narrowed considerably over the years. For example, no one now believes that excessive occlusal force initiates periodontitis, nor does any credible person believe that occlusal force is incapable of causing periodontal injury. As the edges of the debate have been nibbled away over time, the crux of the remaining argument is this: Can occlusal forces exacerbate the progression of periodontitis, and is eliminating occlusal discrepancies appropriate or necessary in the treatment of the disease?

The purpose of this article is to outline the clinical and histological response of the periodontium to excessive occlusal force, to review the clinical studies that have examined the relationship between occlusion and periodontitis, and to reiterate a rational approach to managing occlusion within the context of periodontal therapy.

Treatment of occlusal trauma should be directed toward the specific instances in which occlusal trauma truly exists.

THE OCCLUSAL TRAUMA LESION

The term "occlusal trauma" (or "trauma resulting from occlusion") refers to the pathological or adaptive changes to the periodontium caused by the excessive occlusal force known as "traumatogenic occlusion."[1] Occlusal trauma, then, is an injury to the periodontium; traumatogenic occlusion is the etiologic factor causing the injury.

Similar in some respects to the tissue response to orthodontic forces, traumatogenic occlusion establishes distinct zones of tension and pressure within the periodontal ligament of the affected tooth. The location of these zones depends on the location and vector of the force, as well as on the

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into the surrounding bone, following the course of blood vessels. Weinmann did not see evidence that occlusion caused or influenced the progression of the inflammatory process.

Two decades later, Glickman and Smulow[8,9] also examined human autopsy material and agreed that inflammation appeared to begin at the gingival attachment and subsequently progressed into the surrounding periodontal supporting tissue. However, they suggested there was evidence that in teeth undergoing occlusal trauma, the inflammation progressed in a different manner than that in teeth that were not undergoing occlusal trauma. They termed this different progression of periodontal disease as an "altered pathway of destruction." They termed the combined effects of occlusal trauma and inflammation as "co-destructive factors" in periodontal disease.[8,9]

Other researchers did not agree with this theory of codestruction.[10,11] In the 1970s, Waerhaug,[12,13] again evaluating human autopsy material, felt that there was no evidence that occlusal forces played any role in periodontal destruction. He indicated that no differences in disease progression could be detected between teeth that were undergoing occlusal trauma and teeth that were not. Waerhaug found no evidence for Glickman and Smulow's "altered pathway of destruction" and indicated that all inflammation and bone loss were associated with the presence of bacterial plaque. Waerhaug showed evidence that bacterial plaque always was present in close proximity to the site of periodontal destruction. He also indicated that there was no evidence of the changes purported to be present in the altered pathway of destruction caused by occlusal trauma. Waerhaug's conclusion was that occlusal trauma played no part in periodontal destruction and plaque-related inflammation was the only cause of periodontal disease.

Most historical studies of the effect of occlusal forces on the progression of periodontal disease were aimed at showing that occlusion did or did not cause periodontal destruction. The desire to find a single cause of periodontal disease was rooted in the disease concepts of the late 19th century. The idea that a chronic process such as periodontal disease was the result of multiple risk factors did not fit the outlook of the first half of the 20th century. Glickman and Smulow's view of a codestructive action between bacterial inflammation and occlusal trauma was a step toward the modern concept of multiple risk factors' affecting the progression and severity of the disease process.

ANIMAL RESEARCH

Starting in the 1930s, multiple animal research projects were performed in an attempt to prove or disprove a relationship between occlusion and periodontal disease.[14-16] The most significant animal studies were performed in the 1970s by two research groups, one at Eastman Dental Center in Rochester, N.Y.,[17-21] and the other at the University of Gothenburg in Sweden,[22–25] and they often are referred to as the American and the Scandinavian occlusal studies, respectively. Both evaluated the effect of occlusal trauma and gingival inflammation in animals. The American group used repeated applications of orthodontic-like forces on the teeth of squirrel monkeys, and the Scandinavian group used occlusal forces similar to those of a "high" restoration in beagle dogs. Both groups evaluated the effects of these traumatic occlusal forces in animals: those in which good oral hygiene was maintained with little gingival inflammation and those in which a soft diet allowed the buildup of plaque and subsequent inflammation.

Despite major differences in the animal models and the types of excessive occlusal forces applied, the results of these two studies were similar in many respects. Within both animal models, researchers found that if oral hygiene was maintained and inflammation controlled, occlusal trauma resulted in increased mobility and loss of bone density, but no loss of attachment, during the length of the study. In no case in which inflammation was controlled was there any attachment loss or pocket formation. Furthermore, if the occlusal forces were removed, there was a return to pretreatment stability and bone volume. In animals in which plaque was allowed to accumulate and gingival inflammation was present, there was greater loss of bone volume and increased mobility, but still no attachment loss. Only in cases in which the bone support of beagle dogs was surgically decreased, inflammation was allowed to develop and occlusal stress was applied was there any evidence of attachment loss. The conclusion of both research groups was that

position of the alveolar crest.[2] The extent of the occlusal trauma lesion within the periodontal ligament space depends on the level of force. At low levels, the microscopic changes include increased vascularization, increased vascular permeability, vascular thrombosis, and disruption of fibroblasts and collagen fiber bundles. If the force is maintained, osteoclasts appear on the surface of the alveolus, leading to net bone resorption.[2] At higher levels, occlusal forces may cause necrosis of periodontal ligament tissue, including lysis of cells, disruption of blood vessels and hyalinization of collagen fibers.[3,4] Osteoclasts appear in marrow spaces adjacent to the alveolar bone, producing an undermining, rather than direct, resorption of bone.[2,5] In addition, resorption of the root surface may be a feature of the occlusal trauma lesion.[6-8]

The net effect of these microscopic changes is an adaptive response within the periodontium that allows it to compensate for the excessive force.[2,9] The density of the alveolar bone decreases and the width of the periodontal ligament space increases at the expense of both the socket wall and the root surface. This leads to the two most distinctive clinical signs of occlusal trauma: increased tooth mobility and a radiographic widening of the periodontal ligament space, which may be either uniform or accentuated at the alveolar crest.[4,5,10] An additional diagnostic sign of the occlusal trauma lesion is fremitus, or functional mobility, which refers to the palpable deflection of a tooth either on closure or during excursive movements.[11]

Not every mobile tooth suffers from occlusal trauma, but certainly every tooth with a sustained occlusal trauma lesion will become mobile.

The effect of occlusal forces on periodontal attachment levels has been well-studied in animal models. When imposed upon a healthy periodontium, even if reduced in height, traumatogenic occlusion does not cause pocket formation or loss of clinical attachment.[3,12] Though this finding is perhaps controversial, one of the two major research groups conducting animal studies of occlusal trauma observed that in certain circumstances, traumatogenic occlusion superimposed on pre-existing periodontitis lesions could lead to an increased loss of attachment.[9,10] Assuming this to be true, it is important to note that this loss of attachment was found only in conjunction with an actual occlusal trauma lesion.

CLINICAL STUDIES

Tooth mobility has been described as the "hallmark" of occlusal trauma.[5] Whether progressive as the injury occurs or simply increased after compensation has taken place, tooth mobility is a universally recognized component of occlusal trauma.[2-6,9,12] Not every mobile tooth suffers from occlusal trauma, but certainly every tooth with a sustained occlusal trauma lesion will become mobile. Most clinical studies that have examined the relationship between occlusion and periodontitis, however, have focused on teeth with occlusal discrepancies rather than teeth with traumatic lesions.

Yuodelis and Mann[13] reported on the relationship between periodontal parameters and molar nonworking contacts using the records, radiographs and study models of 54 patients with periodontal disease. Fifty-three percent of molar teeth had nonworking contacts, and the authors determined that probing depths and bone loss were greater for those teeth. Conversely, Shefter and McFall[14] looked at occlusal disharmonies in a group of 66 young patients with mild-to-moderate periodontitis. Seventy-eight percent had a deviation from centric relation to centric occlusion, and 56 percent had nonworking contacts in lateral movements. The authors found no relationship between the occlusal disharmonies and periodontal findings.

A more recent study by Nunn and Harrel[15] investigated the association between occlusal discrepancies and periodontitis in a private practice setting. These researchers compared 41 patients who received all recommended treatment, including adjustment of occlusal discrepancies, with 48 patients who received partial treatment or no treatment. They found that 56 (62.92 percent) of the 89 total patients and 307 (13.35 percent) of 2,147 teeth had occlusal discrepancies; these discrepancies were listed as a vertical slide greater than or equal to 1 millimeter from a premature contact and balancing contacts in lateral movement. The authors reported that teeth with occlusal discrepancies had significantly deeper

without inflammation, occlusal trauma does not cause irreversible bone loss or loss of attachment. On the basis of the collective results of these studies, it appears that in animals, occlusal trauma is not a causative agent of periodontal disease.

The cited animal research seems to suggest that occlusal forces are not a factor in the progression of periodontal destruction. However, several questions remain concerning the application of these results to humans. Naturally occurring periodontal disease is virtually unknown in monkeys, and it usually occurs only in much older dogs than those used in these studies. Furthermore, in humans, most periodontal destruction resulting in attachment and bone loss occurs relatively slowly over a much longer period than that used in the animal studies. Both the use of animal models and the relatively short duration of the studies leave questions concerning the application of these results to periodontal destruction occurring in humans.

HUMAN STUDIES

Human research on occlusion has yielded mixed results. One study evaluated teeth with balancing or nonworking contacts in relation to teeth without balancing contacts.[26] Teeth with nonworking contacts showed greater periodontal destruction and pocket depths. Another similar study showed no difference between the two groups.[27] The researchers conducting these studies used existing records such as periodontal charting and study models to determine which teeth were undergoing occlusal trauma, and they did not conduct direct patient examinations. Other human studies have yielded similar conflicting results.[28,29] Furthermore, these studies were epidemiologic in nature and looked at a general population rather than patients with periodontal disease.

Burgett and colleagues[30] used a controlled clinical trial to evaluate the effect of treating the occlusion on healing outcomes after periodontal treatment. In this trial, one-half of the patients received occlusal adjustment by means of selective grinding before undergoing surgical and nonsurgical periodontal therapy. The other one-half did not receive occlusal adjustment. After an extended healing period, the group that received occlusal adjustment before periodontal treatment showed consistently and statistically significantly better healing, in the form of improvements in attachment levels, when compared with patients who did not receive occlusal adjustment. This well-controlled study demonstrated that in a group of patients with existing periodontal disease, there was improved healing if occlusal trauma was minimized by occlusal adjustment.

As part of a large study on prognosis, McGuire and Nunn[31,32] reviewed the change in prognosis and in the number of teeth lost by patients with periodontal disease who had parafunctional habits. In patients with parafunctional habits that had not been treated with an occlusal appliance, there was no improvement in prognosis despite periodontal therapy. Also, more teeth were lost in the untreated group than in a group that received occlusal appliances. This study indicated that for patients with periodontal disease, the treatment of occlusal trauma improved treatment outcomes and that the lack of treatment resulted in greater tooth loss.

The consensus of the 1996 World Workshop in Periodontics indicated that there was inadequate information to determine whether a relationship exists between occlusion and the progression of periodontal disease.[33] Another review article published in the mid-1990s stated a similar viewpoint.[34] More recently, the 1999 Consensus Report on Periodontal Disease Classification agreed that occlusal trauma represented injury resulting in tissue changes within the attachment apparatus as a result of occlusal force(s). This report also agreed that excessive occlusal forces alone do not initiate plaque-induced gingival disease or loss of connective tissue associated with periodontitis.[35]

RECENT HUMAN STUDIES

The results of a large retrospective study performed by two of the authors (S.K.H. and M.E.N.) that evaluated the effects of occlusal discrepancies on the progress of periodontal disease have refocused attention on this area of periodontal therapy.[36-38] In that study, the authors evaluated a group of private practice patients referred for the treatment of active periodontal disease. All patients had advanced periodontal disease with clinically detectable bone loss. For inclusion in the study, the patients had to have been recommended to receive both nonsurgical and surgical periodontal treatment. All cases could be classi-

initial probing depths, more mobility and poorer prognoses than teeth without discrepancies.

Other clinical studies aimed specifically at evaluation of teeth with occlusal trauma lesions have failed to make this connection. Pihlstrom and colleagues,[16] in a study of various clinical parameters of the maxillary first molars of 300 patients, found that while 60.4 percent of teeth had wear facets, 66.4 percent had centric relation contacts and 7.5 percent had nonworking contacts, only 4.2 percent had a widened periodontal ligament space and functional mobility associated with occlusal trauma. They concluded that teeth with occlusal contacts in centric relation and in working, nonworking or protrusive positions had no more severe periodontitis than did teeth without these contacts.

Jin and Cao[17] examined 32 patients with moderate-to-advanced periodontitis to determine the reliability of several selected signs of occlusal trauma. Since the total number of teeth examined is not included in the article, it is difficult to determine the percentage of teeth with occlusal discrepancies versus the number with more objective signs of occlusal trauma. That said, the authors reported no significant differences in pocket depths, attachment levels or alveolar bone height between teeth with and without various abnormal occlusal contacts.

The determining factor of whether an occlusal contact produces occlusal trauma is the presence of periodontal injury, not the physical manifestations of the teeth, temporomandibular joints or muscles of mastication.

The evidence linking occlusal adjustment to improvements in periodontal parameters is extremely limited. Burgett and colleagues[18] randomly assigned 50 patients with periodontitis into two groups based on occlusal adjustment. As part of the initial therapy, 22 patients received occlusal adjustment, with the goal of achieving even and stable contacts in centric occlusion, freedom in centric occlusion, smooth gliding contacts and elimination of balancing interferences. The remaining 28 subjects did not receive occlusal adjustment. All patients then received definitive surgical or nonsurgical periodontal therapy. Two years after treatment, the occlusal adjustment group had a slightly greater (0.4-mm) gain in attachment level than did the no-adjustment group. The authors noted that there was no difference in posttreatment probing depth reduction or mobility levels between the two groups.

In a clinical trial nine years later, Harrel and Nunn[19] reported on the response to treatment of 89 untreated, partially treated and fully treated patients with periodontitis. Patients in each group were divided further on the basis of the presence or absence of occlusal discrepancies (premature contact with vertical slide 1 mm or greater or balancing contact in lateral movement) and whether occlusal adjustment was performed as part of treatment. Each patient received a follow-up examination at least 12 months after undergoing treatment or, for those electing not to receive treatment, the initial examination. Reporting their results only on the basis of the occlusal status, the authors observed a difference in probing depth changes after treatment, with a mean increased probing depth of 0.066 mm per year at sites with untreated occlusal problems, compared with a decreased probing depth of 0.048 mm per year at sites with no occlusal problems and 0.122 mm per year at sites with treated occlusal problems.

Though both Burgett and colleagues[18] and Harrel and Nunn[19] suggested a slight positive effect of occlusal therapy on the clinical outcome, the use of these studies as an endorsement for routine occlusal adjustment during the initial treatment of periodontitis is questionable.

DISCUSSION

There are several possible physiologic responses to excessive occlusal contact between teeth, and it is possible that two or more of these may occur simultaneously. The path of mandibular closure may be altered to avoid the excessive contact, the occlusal or incisal surfaces may wear leaving facets or even enamel fractures, pulpal symptoms may occur or the force may cause injury to the periodontium known as occlusal trauma.[20] When discussing the relationship between occlusion and periodontal disease, however, it is important to remember that the determining factor of whether an occlusal contact produces occlusal trauma is the presence of periodontal injury, not the physical manifestations of the teeth, temporo-

fied as periodontal case type III or IV. All patients had to have complete initial periodontal records, including a full occlusal analysis consisting of a recording of an initial contact point, measurement of any slide existing between a retruded position (centric relation) and maximum intercuspation (centric occlusion), lateral working and balancing contacts, and protrusive contacts. Furthermore, to be included, the patients had to undergo a second examination at least one year after the initial examination, at which time another complete periodontal evaluation was performed and the results recorded. We recorded other pertinent data such as pocket depth, mobility (according to the Miller[39] index), fremitus, width of gingiva and treatment performed. For this study, we defined occlusal discrepancies as teeth with a slide between centric relation and centric occlusion of 1 millimeter or greater or the presence of nonworking contacts. We placed all data in a database so that we could use general estimating equations to analyze the data.

We need to make clear that our study evaluated the effects of occlusal discrepancies on the progression of periodontal disease. We did not attempt to make a diagnosis of "occlusal trauma." The diagnosis of occlusal trauma can be made only by the histologic evaluation of the periodontium. This makes it impossible to verify the diagnosis of "occlusal trauma" for a tooth that is to be retained. Proposed surrogate markers of occlusal trauma, such as mobility or tooth wear, are problematic because of inconsistencies in presentation. Some teeth with severe wear facets may have no detectable mobility, while very mobile teeth may have no detectable occlusal wear. It even is possible to find mobile teeth that are not in occlusal function. We studied occlusal discrepancies because they can be consistently identified clinically without extraction of the tooth. The teeth identified as having an occlusal discrepancy may or may not have received a histologic diagnosis of "occlusal trauma." All data from these studies should be interpreted as demonstrating the effects of occlusal discrepancies and not necessarily the effects of "occlusal trauma."

We recorded all data on an individual-tooth basis. Recording and analyzing data in this manner allowed the comparison of teeth that had occlusal discrepancies with teeth that did not. Analysis of individual teeth according to occlusal discrepancy sets this study apart from most previous studies that have made comparisons between patients with and without occlusal trauma. While making such comparisons enables one to use traditional statistical tools for analysis, the "all-or-none" measure for describing each patient is a crude instrument for making comparisons. In addition, success or failure at individual sites is the measure by which patients and practitioners most often judge the outcome of periodontal therapy, and by using the measure of individual teeth for assessing occlusal discrepancy, each patient's occlusion can be put on a continuum—something that normally is not possible when patients are simply classified as having or not having an occlusal problem.

We entered data regarding 89 patients and 2,147 teeth into the database. The patients fell into three groups based on the type of treatment performed. In all groups, patients were selected randomly for inclusion and had self-selected the treatment that was performed.

- The first group was seen for a periodontal examination but elected to not receive any of the recommended treatment. The patients in this group voluntarily returned for another complete periodontal examination at least one year after the initial collection of data. We designated this group the "untreated" group and felt they represented how occlusal interferences could affect the progression of untreated periodontal disease.
- The second group completed the initial nonsurgical phase of the recommended treatment but did not complete the recommended surgical treatment. All patients in this group received at least root planing and oral hygiene instructions. Some patients in this group had occlusal adjustment performed. We designated this group the "nonsurgically treated" group.
- The third group was selected randomly from patients who had completed all recommended periodontal therapy, including surgery, and were in a periodontal maintenance program.

In evaluating the initial data of all patients within the study, we found that teeth with an occlusal discrepancy had pocket depths approximately 1 mm deeper than those of teeth with no occlusal discrepancy. This difference was highly statistically significant ($P \leq .0001$) and was true regardless of age, sex, smoking status or other risk factors. In addition to having deeper probing

mandibular joints or muscles of mastication.[4] If the periodontium is reduced enough, even a normal occlusal contact may produce occlusal trauma. Similarly, it is possible that even the worst deflective contact or balancing interference does not cause a traumatic lesion. Since the term "occlusal trauma" refers to the tissue injury rather than the occlusion, an increased occlusal force is not traumatic if no injury is present.[4]

The fact that not every occlusal discrepancy causes occlusal trauma is important when one considers that occlusal discrepancies are quite common in the general population.[21,22] In fact, both the Yuodelis and Mann[13] and Shefter and McFall[14] studies described earlier reported that more than one-half of the patients had occlusal discrepancies and one-half of all molar teeth had balancing contacts. Certainly not all of those contacts required occlusal adjustment to maintain periodontal health. Sixty-eight percent of all teeth with occlusal discrepancies in the Nunn and Harrel[15] patient group were nonmobile and, therefore, likely did not manifest an occlusal trauma lesion. It is difficult to understand the purpose of occlusal adjustment for these teeth.

Occlusion has been proposed as a risk factor for periodontitis.[23] We believe it is possible that in certain cases, traumatogenic occlusion can exacerbate periodontal destruction, and, therefore, occlusal adjustment occasionally is indicated as part of periodontal therapy. However, we also believe that since not every tooth with an occlusal discrepancy is suffering from occlusal trauma—and, in fact, most are not—not every occlusal discrepancy in a patient with periodontitis needs adjustment. This philosophy is best summed up by Ramfjord and Ash,[24] who stated that "the need for adjustment should be based on a definite diagnosis of a traumatic lesion rather than the location of some occlusal interferences which may be of no significance."

CONCLUSION

A treatment philosophy not calling for the early adjustment of occlusal discrepancies does not necessarily ignore the potential role of occlusion in periodontitis. We believe, as has been stated by others,[5,22] that the initial treatment of the periodontitis lesion should focus on control of inflammation by means of patient oral hygiene and nonsurgical therapy. In situations in which an obvious occlusal discrepancy is directly related to a clinically and/or radiographically evident traumatic lesion, it may be appropriate to adjust the occlusion at this stage. Conversely, occlusal discrepancies that are not accompanied by signs or symptoms of occlusal trauma generally do not require adjustment. After initial therapy, the dentist should re-evaluate the patient to assess the results. At this time, if indicated by persistent hypermobility or patient discomfort, further occlusal therapy may be indicated. In our view, this is the approach best supported by the available evidence, and it is the best way to ensure that treatment of occlusal trauma is directed toward the specific instances in which occlusal trauma truly exists. ■

The views expressed in this article are those of the authors and are not to be construed as official or as reflecting the views of the U.S. Air Force or Department of Defense.

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TABLE

Statistics for initial clinical parameters individually, by initial occlusal status.*

PARAMETER	OCCLUSAL STATUS		*P*†
	No Occlusal Discrepancy	Occlusal Discrepancy	
Initial Probing Depth (n)	1,991	156	
Mean (± standard deviation)	4.77 (± 1.31)	5.53 (± 1.51)	
Median	5.0	5.0	
Range	2.0 to 9.0	3.0 to 9.0	< .0001
Initial Prognosis (n)	1,993	307	
Good	896 (45%)	71 (23%)	
Fair	1012 (51%)	198 (65%)	
Fair to poor	41 (2%)	20 (7%)	
Poor	36 (2%)	14 (5%)	
Hopeless	8 (< 1%)	4 (1%)	< .0001
Initial Mobility (n)‡	1,894	281	
0	1467 (77%)	192 (68%)	
1	382 (20%)	74 (26%)	
2	34 (2%)	12 (4%)	
3	11 (1%)	3 (1%)	.0316

* Adapted with permission of the American Academy of Periodontology from Harrel and Nunn.[38]
† *P* values based on simple general estimating equation regression models using an exchangeable working correlation matrix.
‡ According to the Miller Mobility Index.[39]

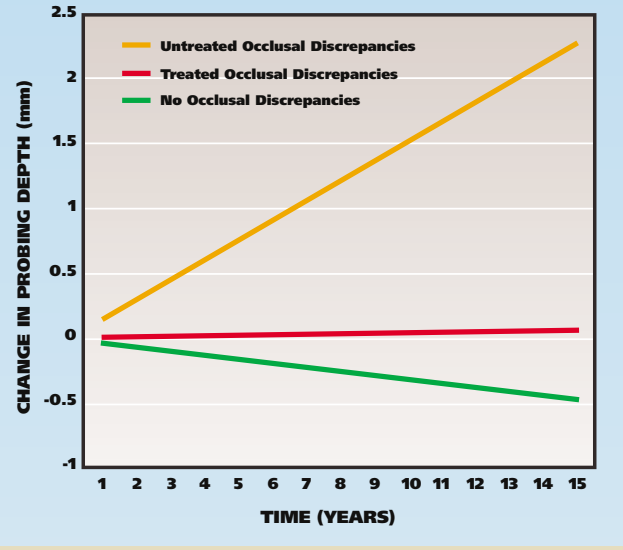

Figure 1. Change in probing depth over time for all subjects. General estimating equation regression model with median follow-up of 2.7 to 8.7 years; range of follow-up, 0.8 to 21.2 years. mm: Millimeters. Adapted with permission of the American Academy of Periodontology from Harrel and Nunn.[38]

depth, teeth with occlusal discrepancies had statistically greater mobility, as well as a prognosis statistically worse than that for teeth without occlusal discrepancies. The presence of occlusal discrepancies was a statistically significant predictor of deeper pocket depths, greater mobility and a poorer prognosis. When we evaluated only patients with good oral hygiene, occlusal discrepancies were a better predictor of pocket depths, mobility and poor prognosis than were any other risk factors evaluated, including smoking. These data are shown in the table.

We evaluated the progression of pocket depth over time for all patients in all treatment groups. We found that teeth with untreated occlusal discrepancies experienced a significant increase in pocket depth per year when compared with teeth with no occlusal discrepancies or teeth with treated occlusal discrepancies. Teeth with no occlusal discrepancy showed little change in pocket depth, and teeth with treated occlusal discrepancies showed improvement in pocket depth. Figure 1 shows these results. When we evaluated patients from the untreated group, we found that teeth both with and without occlusal discrepancies experienced increasing pocket depth over time. This is not surprising, as these patients had been diagnosed with advanced periodontal disease and elected not to have their disease treated. However, we determined that the teeth with occlusal discrepancies experienced a greater increase in pocket depth than did the teeth without occlusal discrepancies. Figure 2 (page 1390) shows these results. When we evaluated the patients who underwent nonsurgical treatment, we once again found that teeth both with and without occlusal discrepancies experienced increased pocket depth. However, the teeth with occlusal discrepancies experienced a greater increase in pocket depth than did teeth with no occlusal discrepancies. Figure 3 (page 1390) shows these results. As a control for patients who were not compliant with oral hygiene recommendations, we evaluated a subgroup of the nonsurgical treatment group who had good oral hygiene.

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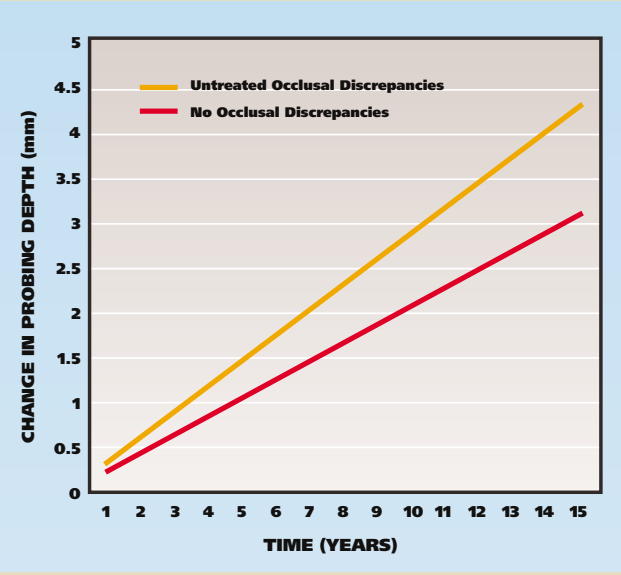

Figure 2. Change in probing depth over time for untreated subjects. General estimating equation regression model with median follow-up of 2.7 to 8.7 years; range of follow-up, 0.8 to 21.2 years. mm: Millimeters. Adapted with permission of the American Academy of Periodontology from Harrel and Nunn.[38]

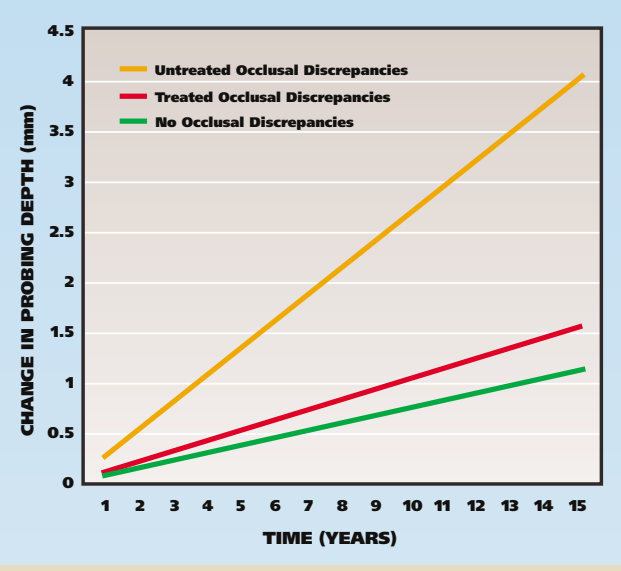

Figure 3. Change in probing depth over time for subjects who received nonsurgical treatment. General estimating equation regression model with median follow-up of 2.7 to 8.7 years; range of follow-up, 0.8 to 21.2 years. mm: Millimeters. Adapted with permission of the American Academy of Periodontology from Harrel and Nunn.[38]

Within this subgroup, we again determined that teeth both with and without occlusal discrepancies showed increasing pocket depth over time. And once again, we noted that the teeth with occlusal discrepancies experienced a greater increase in pocket depth than did those without occlusal discrepancies.

We evaluated the increase or decrease in the width of gingivae to determine if occlusal discrepancies contributed to a decrease in the width of this tissue consistent with recession. Occlusal discrepancies did not contribute to a decrease in the width of gingivae and, furthermore, treatment of occlusal discrepancies did not cause an increase in the width of gingivae. We determined that occlusal discrepancies were not a factor in the width of attached gingivae and did not appear to contribute to recession.[40]

Our study should be viewed in the context of its design. It does not meet the level of what is considered the gold standard of clinical research: the controlled clinical trial. Ideal research is prospective in nature, with a double-blind design in which neither the patients nor the evaluators know what treatment the patients did or did not receive. Our study was retrospective in nature, a single practitioner performed all treatment and the same practitioner performed all evaluations and data gathering. Furthermore, the patients' oral hygiene and maintenance compliance was not standardized. All of these are significant concerns regarding our research design.

However, we need to point out that the only way to fulfill the parameters of a controlled clinical trial would be to first diagnose periodontal disease and evaluate the patients for occlusal discrepancies, then follow the patients' status for many years without performing any treatment for their diagnosed periodontal disease. This clearly is unethical and would violate all standards for human research. We feel that our research, with its admitted flaws, represents the most valid and complete evaluation of the relationship between periodontal disease and occlusal forces published to date. The results of our studies demonstrate very strong statistical evidence that occlusal discrepancies are a significant risk factor in the progression of periodontal disease. We feel that the strong statistical relationship between occlusal discrepancies and the progression of periodontal disease is clinically valid, and that this positive correlation may be independent of the classic histologic diagnosis of "occlusal trauma."

SUMMARY

The exact effect of occlusal discrepancies/occlusal trauma on the progression of human periodontal

disease remains unknown. However, all studies performed to date strongly indicate that occlusion is not a causative factor in periodontal disease. On the basis of this finding, we should state categorically that there is no justification in the literature for prophylactic adjustment of the occlusion to prevent periodontal disease. It also can be stated that research involving humans has shown that occlusal discrepancies may be a significant risk factor for the progression of existing periodontal disease and that the treatment of occlusal discrepancies significantly improves the outcome achieved with periodontal treatment.

Periodontal disease is a multifactorial disease that affects only a limited number of people within a population. Our current understanding of periodontal disease is that it occurs in these susceptible people in the presence of multiple risk factors, such as bacterial plaque and smoking. Periodontal disease does not appear to be due to a single cause such as a specific bacterial species, but rather to be a result of multiple risk factors. This disease model is relevant to many chronic inflammatory diseases. Just as smoking does not cause periodontal disease but is a significant risk factor in the progression of periodontal disease, occlusal discrepancies do not cause periodontal disease but may be a significant risk factor in the progression of periodontal disease.[41,42] Removing the risk factor of occlusal discrepancies through selective grinding and/or occlusal appliances during periodontal therapy has been shown to produce significant changes in the progression of the disease and improve the results from treatment of the inflammatory component of the disease. On this basis, we feel there is evidence that the treatment of occlusal discrepancies should be considered as an integral part of the overall treatment of periodontal disease and should be included in the comprehensive treatment of this disease. ■

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