Review Article

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Trauma from Occlusion an Etiological Factor or Co-Factor??? An Insight

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ABSTRACT

Background: From many years it is been a controversy that the role of trauma from occlusion in periodontal diseases as primary or as a co-factor and has been an issue of controversy and extensive debate. Findings of various experimental studies were often diverse and somewhat contradictory. Despite the foregoing concerns, the majority of these early studies agreed that occlusal trauma in and of itself failed to result in pocket formation or loss of connective tissue attachment. The current review of literature is an attempt to clear the controversy whether trauma from occlusion is prime etiological factor or a co-factor for periodontal diseases.

Keywords: Trauma from occlusion, Occlusal trauma, Periodontal disease.

INTRODUCTION

For many years the role of occlusion and its dynamic interactive impact on the periodontium has been an issue of controversy and extensive debate. Although a variety of occlusal conditions have purportedly been related to this interaction, the central focus has been on occlusal trauma resulting from excessive forces applied to the periodontium. In an attempt to clarify and better understand this condition early investigators used human necropsy specimens and a variety of animal models as a basis for clinical and histological studies. Findings were often diverse and somewhat contradictory. In the animal studies factors of concern included differences among animals, forces applied and lack of controls. Retrospective descriptive observations of the effect of excessive forces on the periodontium were derived from human necropsy materials. The selection of study sites was based on occlusal wear, patterns of pocket formation and presence of attachment loss leaving some question as to the presence of ongoing occlusal trauma.

Despite the foregoing concerns, the majority of these early studies agreed that occlusal trauma in and of itself failed to result in pocket formation or loss of connective tissue attachment. It is apparent that the effects of excessive occlusal force and the destructive, adaptive and reparative response of the periodontium have been complicated by a relative lack of evidence based on well controlled prospective studies in human beings.

This is to review the literatures in an attempt to conclude whether trauma from occlusion is an etiological factor or co-factor for periodontal diseases.

1. What evidence exists that establishes a relationship between excessive occlusal forces and the initiation, development, and/or exacerbation of periodontal diseases?

Excessive occlusal forces have been demonstrated to cause defined lesions and responses in the attachment apparatus. These lesions and responses are referred to as “Occlusal

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Trauma" and listed in the classification of periodontal diseases in Current Procedural Terminology for Periodontics and Insurance Reporting Manual. Studies which evaluated the interactions of excessive occlusal forces with inflammatory periodontal diseases agree that these forces do not initiate gingivitis or periodontitis. These studies disagree on the effect of excessive occlusal forces on the exacerbation or progression of periodontitis. Due to conflicting studies, non-standardized experimental design, and the variety of experimental models used, the effect of excessive occlusal force on the progression of periodontitis is inconclusive.

2. What evidence exist that occlusal therapy can modify the progression of periodontitis or aid in the treatment of periodontitis?

The progression of periodontitis can be halted by the control of the putative periodontal pathogens that are the primary etiologic factors of the disease process. Animal studies indicate that tooth mobility which may exist after resolving inflammation does not result in any further loss of connective tissue attachment. Thus, a subsequent addition of occlusal therapy to the therapeutic process does not aid in halting the progression of periodontitis. However, occlusal adjustment may reduce mobility, increase stability and function, and aid in regaining bone that was lost due traumatic occlusal forces. It needs to be realized that the potential for reductions tooth mobility may be limited because the hyper mobility may be due to function of reduced periodontal support, rather than recessive occlusal forces.

3. What are the effects of occlusal forces on wound healing following various periodontal surgical procedures; e.g., guided tissue and guided bone regeneration, mucogingival surgery, osseous regeneration, etc?

Limited data have demonstrated a small but statistically significantly greater gain in attachment level when occlusal adjustment was combined with modified Widman flap surgery or with scaling and root planning for the treatment of adult periodontitis. The effects of occlusal forces on guided tissue regeneration (GTR), guided bone regeneration (GBR), mucogingival surgery, and osseous regeneration have not been adequately evaluated to draw conclusions concerning these modes of therapy.

4. What evidence exists that "preventive" occlusal adjustment is justified for the health of the periodontium?

No controlled studies have documented the benefit of preventive occlusal adjustment for the purpose of controlling inflammatory periodontal disease.

5. What are the future directions for clinical practice and research on occlusal therapy in relation to periodontal health?

Questions that should be evaluated concerning the effect of traumatic occlusal forces (TOF), occlusal adjustment (OA), and tooth mobility (TM) including the following.

1. What is the effect of TOF/OA/TM on regenerative periodontal procedures?
2. What is the effect of TOF/OA/TM on mucogingival surgery?
3. Are excessive occlusal forces a risk factor for the progression of periodontitis?
4. Is there a relationship between psychological stress, occlusal Trauma, and inflammatory periodontal disease?

The concept of trauma from occlusion had undergone changes with time. Earlier it was considered as a primary etiological factor; then it was believed to be a co-factor and still later it was considered as a predisposing factor. With this background the review of literature has been divided into three parts:

EARLIEST STUDIES SUPPORTING THE CONCEPT TRAUMA FROM OCCLUSION – AN ETIOLOGIC FACTOR[2]

There is no aspect of periodontology that has received more attention than traumatic occlusion. The literature on the subject is voluminous, a great portion of it being devoted to argumentation about whether it is a primary or secondary causative factor in periodontal disease, but rather an aggravating factor when some form of

periodontal disease already exists. The first comprehensive study of the role of occlusal stress on teeth in relation to periodontal disease was made by Talbot[3], who pointed out that man is predisposed to disease of the supporting tissues of the teeth because jaw function has been greatly decreased by modern methods of food preparation.

Stillman [4] is credited with being the first to emphasize traumatic occlusion as a cause of periodontal disease. Repeated abnormal pressure of one tooth on another produces traumatic injury. He pointed out that there are noninfectious changes that are directly produced by traumatic occlusion.

McCall [4] supported Stillman in his belief that, once traumatic occlusion is established, irritation and periodontal congestion results. In studies of human teeth, Orban and Weinmann found signs of trauma in every case of missing teeth.

Gottlieb and Orban [5] studied the effects of traumatic occlusion in dogs. They found that the periodontal tissue in the area of pressure became thinner and bone resorption enlarged the periodontal space and on the other side, tension stimulated bone building.

Box [6] experimented on sheep to observe the excessive occlusal stress and found that continued excessive occlusal stress tended to move teeth out of line. He differentiated between traumatic and traumatogenic occlusions, pointing out that "an occlusion which, under biting pressure was capable of producing or has produced injury to periodontal tissues."

Breitner [7] pointed out that histologic studies have shown that natural abrasion of the teeth is a protective factor against damage to the tooth-supporting apparatus, since it reduces forces acting laterally on teeth. He said that, vertical forces are easily assimilated whereas lateral forces can be tolerated to some extent.

Leonard[8] stated that, uneven wear of the teeth produces changes in the direction of forces that hold the teeth in equilibrium.

McCoy[9] stressed the importance of integrity of arch and relationship between teeth. Tipping of the teeth as a result of tooth loss was, therefore stated as a causative factor of occlusal trauma.

Gratzinger [10] believed that the damaging effects to periodontal tissues do not have the character of a traumatic injury and are not produced by some definite type of occlusion. It is produced by an imbalance in the musculature of lip, cheek, tongue and masticatory muscles. Since the forces not related to occlusion constitute the irritation, Gratzinger referred the term "dynamic irritation" to traumatic occlusion.

Lundquist [11] demonstrated that normal functional demands needs normal bony structure and increased functional demands might become excessive and cause injury to the periodontal tissues. Such traumatic injury could be considered an aseptic wound.

Macmillan [11] disagreed with the opinion that traumatic occlusion is the predominating causative factor in periodontal disease. The maximum amount of stress which the periodontium can withstand has not been determined. Nor has there been a determination of the critical point at which normal stimulation ends and retrogressive changes as a result of masticatory overstress begin. He concluded that the degenerative process attributed to traumatic occlusion could not be produced on a perfectly healthy tooth by masticatory forces.

Blayney disagreed with the concept that too much pressure produces alveolar bone and cementum resorption. He said that lack of function causes bone resorption, and an intermittent pressure as produced by occlusal function would result in bone building rather than resorption, since the latter requires a constant pressure.

Leirche and Policard stated that constant pressure produces bone resorption and intermittent pressure favors bone formation.

Kronfeld was of the opinion that in persons with high resistance, even a great amount of occlusal trauma does not produce resorption of root, while in susceptible ones, normal mastication may occasionally produce progressive root resorption.

Rule insisted that occlusal overstress is not a primary factor, but a secondary predisposing factor in the initiation of periodontal disease.

Merritt noted that trauma is a symptom of periodontal disease and not the cause of it. He used

the term "occlusal traumatosis" to designate the effects of traumatic occlusion.

Muhlemann [12] pointed out that occlusal trauma should not be used in the meaning of an abnormal occlusal stress which is "capable of producing or has produced injury to periodontal tissues" and suggested that a distinction be made between

1) the traumatic occlusal situation, initiating abnormal occlusal stress

2) the occlusal trauma, meaning the microscopic lesion of the periodontal tissues.

World Workshop in Periodontics, 1966 [12], postulated that "…Muhlemann's suggestion to describe occlusal trauma as a lesion is much better…the clinical signs rest on measurements of mobility".

Coolidge summarized the local causes of occlusal trauma as unrestored loss of posterior teeth with tooth drift, malposition of teeth, cuspal interferences, uneven wear of the teeth, atrophy of supporting tissues of the teeth, excessive overbite, uneven eruption rate, abrasion with loss of vertical dimension, defective occlusion as induced by ill-constructed restorations and orthodontic appliances, and injurious oral habits.

Goldman[13] pointed out that systemic factors may influence and complicate these local causes of traumatic occlusion.

Orban [14] concluded that general physical state may be an important determinant of tissue changes in traumatic occlusion. The clinical picture of periodontal traumatism has been described by Orban as a loosening of teeth due to widening of periodontal space. There is sensitivity to percussion and thermal change. If trauma is in a single direction, the tooth may move out of occlusion.

Box [15] and **Leonard** [16] have recorded several other symptoms of periodontal disease resulting from traumatic occlusion including tenderness to biting stress, traumatic crescents, congestion of marginal gingiva, McCall's festoons (marginal thickening of the gingiva), Stillman's clefts (slits in the gingival margin over a pocket) and changes in tooth stability. Roentgenographically, thickenings of lamina dura, refraction, condensation and hypercementosis in some cases and root resorption in others have been seen.

The pathologic changes occurring as a result of occlusal trauma have been summarized by **Orban1**[17]. Trauma may act in vertical (axial) or in horizontal (lateral) direction. Vertical overloading may not lead to degenerative changes, since the fiber arrangement of the periodontal ligament compensates the overstress. Lateral or horizontal forces compress the PDL between the tooth root and alveolar wall. Small amounts of forces produce moderate inflammation whereas severe traumatic forces produce thrombosis, hemorrhage and eventually necrosis of periodontal tissues. Extensive bone resorption occurs and periodontal space becomes widened.

Gottlieb [18] showed that the resorption of principal fibers adjacent to the epithelial attachment permits apical proliferation of the epithelium.

Becks [19] expressed the opinion that long standing occlusal trauma results in extensive cemental resorption, which is always less extensive bone resorption and usually subsides once the trauma is over.

Kronfeld [20], and **Orban** [21], agreed that, if tissue resistance is high, pathologic changes in the gingiva are not likely directly from traumatic occlusion.

Thoma stated that, gingival changes do not, as a rule, occur as a result of abnormal stress, since increased function is more likely to influence the gingiva favorably. **Hill** however listed occlusal trauma as one of the causes of gingivitis.

Since the **World Workshop in periodontics in 1966,** the term TFO commonly has been used to designate a traumatic injury related to occlusion in any part of the masticatory system, but with the main stress on the periodontal manifestations. [22]

NEWER STUDIES SUPPORTING THE CONCEPT OF TRAUMA FROM OCCLUSION – A CO - FACTOR

In the 1960's the role of microorganisms as the cause of periodontitis became more accepted. This led to a switch in theory in which traumatogenic occlusion was no longer considered as a causative agent but as a cofactor in the progression of periodontitis. A cofactor was defined

as a factor that by itself that does not cause a disease but could modify the course of expression of a disease process.

Glickman and Smulow [23,24,25] in the early 1960s were the principal proponents of the theory that a traumatogenic occlusion could act as a cofactor in the progression of periodontitis. This theory known as the "co-destructive theory" was proposed, based on a zone of irritation (marginal/ interdental gingiva; gingival fibers) and zone of co-destruction (transseptal/ alveolar crest fibers, periodontal ligament, cementum, bone). This theory suggested that occlusal trauma in the presence of plaque induced inflammation may result in alteration of the normal pathway of inflammation, and development of angular bony defects with infrabony pockets, but that occlusal trauma, in and of itself, did not cause gingivitis and periodontitis. The traumatic occlusal forces would cause damage to the periodontium resulting in what is known as "lesion of occlusal trauma", which could accelerate the progression of periodontitis and direct the inflammatory process into the periodontal ligament (PDL), leading to the development of intra osseous defects.

Yuodelis and Mann [26], using charts from previously examined patients, reported that teeth with nonworking contacts were associated with significantly greater mobility, bone loss, and pocket depth but not with any particular pattern of bone loss.

Nascimento A and Sallum AW [27] studied the changes that occur in periodontal supporting tissues of lower canine and premolar of marmosets (Callithrix Jacchus), due to experimental modification of occlusal relationships in the region of the molar. The results clearly showed areas of tension with stretched periodontal fibers in the distal region of the canine and areas of pressure with osseous and radicular resorption in the mesial region of the first premolar. The tissue changes may be considered to result from transmission of abnormal forces through the proximal contacts of teeth associated with displacements and minor modifications in their occlusal relationships. Similar changes may occur in a tooth distant from the teeth exposed to primary excessive occlusal forces.

These early papers relied on retrospective analysis of occlusal wear patterns, mobility patterns, autopsy material from human and animal models, and patterns of attachment loss and pocket formation to link traumatogenic occlusion as a cofactor in the progression of periodontitis. Research projects using animal models were able to demonstrate that traumatic occlusal forces produced a lesion of occlusal trauma characterized by hemorrhage, necrosis, bone destruction, widening of periodontal ligament, and increased tooth mobility.

Wentz et al [28], however, also reported that despite the traumatic lesions noted in the PDL, no pathologic changes were noted in the gingival tissues or the epithelial attachment. They also noted that the tissues responded to the trauma through an increase in the width of PDL and healing of all necrotic areas. This was considered to be an adaptation to the traumatic forces.

STUDIES THAT QUESTIONED THE CO-FACTOR THEORY

Ramfjord and Kohler [29] in a human block section study reported that experimental occlusal trauma led to bone resorption, root resorption, hyalinization followed by widening of the PDL spaces in areas of compression, and bone apposition in areas of tension. They also reported that the alveolar crest fibers of the PDL remained stable during the period of experimental occlusal trauma.

Akiyoshi and Mori [30] and **Macapan and Weinmann** [31] evaluated histologic specimens to determine the pathway of inflammation in an inflamed and traumatized periodontium. These authors reported that inflammatory cells were found following the perivascular pathways and not travelling directly into the periodontal ligament because of the less dense tissues in the pathways. This contradicted the theory that occlusal trauma acted as a cofactor to change the pathway of inflammation into the PDL.

Goldman [32] observed the effect of occlusal trauma on gingival blood flow. He reported that although occlusal forces did affect blood flow in the PDL, they had no significant effect on the blood supply to the gingival tissues. This indicated that occlusal trauma was not likely to be the cause of soft tissue lesions such as Stillman's clefts and McCall's festoons.

Stahl [33] and **Comar** [34] evaluated the interactions between occlusal trauma and plaque induced periodontal inflammation. Both of them concluded that occlusal forces led to traumatic changes in the PDL; however, they did not believe that occlusal trauma was affecting the plaque induced inflammatory lesion in the gingiva.

These studies not only questioned the effect of occlusal forces on the progression of periodontitis, but also suggested that the various patterns of attachment loss and intraosseous defect formation could be explained by the local anatomy, the location of dental plaque, and the inflammatory lesion that it has produced.

CONCLUSION

Despite decades of debate and multiple publications that discuss the theory of occlusion, occlusal design, and equilibration techniques, there have been few well-designed human studies that can help answer the question "does occlusal trauma modify the progression of attachment loss due to inflammatory periodontal disease". The articles reviewed clearly demonstrate that occlusal forces are transmitted to the periodontal attachment apparatus and those forces can cause changes in the bone and connective tissue. Current research articles are concluding with references that trauma from occlusion acts as co-factor for periodontal diseases and not an etiological factor.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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