International Journal Dental and Medical Sciences Research
Volume 3, Issue 3, May - June 2021 pp 612-621 www.ijdmsrjournal.com ISSN: 2582-6018

Trauma from Occlusion: A Review Article

Dr. Aparna Srivastava[1], Dr. K. K. Chaubey[2], Dr. Raghu Raghav Agarwal[3], Dr. Ashima Agarwal[4], Dr. Jharna Bharali[5]

Submitted: 05-06-2021 Revised: 21-06-2021 Accepted: 24-06-2021

ABSTRACT: Occlusal force which goes beyond the adaptive capacity of our periodontium causes injury to periodontal structures and the resultant trauma is called trauma from occlusion (TFO). Several schools of thoughts are there, some do not consider TFO as an etiological factor while others consider it as a cofactor for the occurrence of periodontal diseases. This narrative review determines the effects of occlusal trauma and excessive occlusal forces on the periodontium, including the initiation and progression of periodontitis, its historical background, etiological factors, relevant terminologies, signs and symptoms, and advanced diagnostic methods.

Keywords - Trauma from Occlusion, Occlusal Trauma, Excessive Occlusal Forces

I. INTRODUCTION

The study of the relationship of occlusion to periodontal disease has long been one of the great mysteries of dentistry. It is generally accepted that plaque is the primary cause for the initiation and progression of periodontal disease.Occlusion effects are often listed among these modifying factors.[1]The dependence of the periodontal tissues on the trophic stimulation from occlusal forces in periodontal health makes occlusion a critical environmental factor. So, occlusion and local factors, may act independently or together in the pathogenesis of periodontal disease.[2] Thus, an understanding of the principles of occlusion and its relationship to oral health and disease is necessary.

Occlusion derived from latin word occlusion i.e., 'oc' up and 'clusion' means closing. Edward angle in1899 defines static concept of occlusion. This concept mainly told us about the occlusion means only how the max and mand teeth are coming in contact with each other.Occlusion is the normal relations of inclined planes of the teeth when the jaws are closed.While in 1930 factual period, dynamic conceptof occlusion came which stated Dynamic biological relationship of all components of the masticatory system to the contacting surfaces of the teeth in function and dysfunction i.e., integrated function of jaw muscles, TMJ and teeth.[3]

Criteria for optimum functional occlusion

a. Optimum orthopedically stable joint position
b. Optimum functional tooth position
c. Direction of force placed on the teeth
d. Amount of force placed on the teeth

It was believed for many years that trauma from occlusion was a primary etiological factor in periodontal disease and that; if it was present, periodontal disease would surely occur. Stillman and mMcCall defined traumatic occlusion as "an abnormal occlusal stress which is capable of producing or has produced an injury to the periodontium." Changing the direction of occlusion forces causes a reorientation of stresses and strains within the periodontium.[2] Excessive occlusion forces may disrupt the function of masticatory musculature, injury of the tempromandibular joint or produce excessive tooth wear, but the term trauma from occlusion generally refers to injury in the periodontium.[4] So various authors described it as –

AUTHORS	DEFINATION
Stillman (1917)[5]	Trauma from occlusion as a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position.
WHO (1978)[6]	Defined it as damage in the periodontium caused by stress on the teeth produced directly or indirectly by the teeth of the opposing jaw.
Glickman (1974)[7]	As the injury that results when the occlusal forces exceed the adaptive capacity of the tissues.
Lindhe, Nyman, Ericsson (1978)[8]	"Pathologic alteration or adaptive changes which develop in the periodontium as a result of undue forces produced by masticatory muscles".
"Glossary of periodontics terms"	"Occlusion trauma and defined it as "An injury to the attachment apparatus as a result of excessive occlusion forces".

International Journal Dental and Medical Sciences Research
Volume 3, Issue 3, May - June 2021 pp 612-621 www.ijdmsrjournal.com ISSN: 2582-6018

American Academy of Periodontology (2001)	

It is, however, important to understand that symptoms of trauma from occlusion may develop only in situations when the magnitude of the load elicited by occlusion is so high that the periodontium around the exposed tooth cannot properly withstand and distribute the resulting force with unaltered position and stability of the tooth involved. This means that in cases of severely reduced height of the periodontium even comparatively small forces may produce trauma or adaptive changes in the periodontium.[6]

Trauma from occlusion results in non-inflammatory changes in the tissue of the periodontal attachment apparatus. Forces of pressure and tension elicit different cellular responses within the periodontium. Pressure results in the resorption of bone and occasionally cementum, necrosis of periodontal fibers, haemorrhage and thrombosis of vessels. Tension in the absence of inflammation promotes the deposition of bone and sometimes cementum. It also causes various changes in the periodontal fibres.[9]

Stallard points out that the arterial blood supply for the periodontal ligament is derived from the alveolar bone.[10,11] This segmental arrangement tend to limit the spread of vascular destruction. Tissue damage resulting from traumatic forces can be localized within the periodontal ligament and alveolar bone. When the forces are withdrawn, the ligament and bone will either repair or regenerate, or both without the formation of periodontal pockets.[12]

When periodontitis is present in combination with occlusion trauma, initial therapy should attempt to resolve the inflammatory condition.[13] Glickman believed that if trauma from occlusion contribute to the evolution of periodontal disease, occlusion adjustments be considered an appropriate therapeutic modality of periodontal treatment.[14] He believed that occlusion equilibration should yield normal, functional interdental relations among teeth, which must be able to maintain a healthy periodontium that does not destroy itself in function.

II. HISTORICAL STUDIES

For more than a century, clinicians have postulated that a relationship existed between occlusion forces and the progression of periodontal diseases.

Karolyi,[8] in the early 20th century, was one of the first to publish on the relationship of occlusal to periodontal disease. He indicated that teeth undergoing excessive occlusal stress seemed to have more periodontal destruction than teeth not experiencing occlusal stress.

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.[5,15]Stillman's comments led to several studies aimed at determining whether occlusion did or did not play a causative role in periodontal disease.[16,17]These studies failed to produces conclusive results, and the controversy continued.

In the 1940s, Weinmann[18] 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 begin from the gingival attachment and spread into the surrounding bone, following the course of blood vessels.

Glickman (1965, 1967)[6] claimed that the pathway of spread of a plaque associated gingival lesion can be changed if forces of an abnormal magnitude are acting on teeth harboring subgingival plaque. This implies that the progressive tissue destruction of periodontium at a traumatized tooth will be different from that characterizing a non-traumatized tooth. Instead of an even destruction of the periodontium and alveolar bone (suprabony pockets and horizontal bone loss), which according to Glickman occurs at sites with uncomplicated plaque-associated lesion, sites which are also exposed to abnormal occlusal force will develop angular bony defect and infrabony pocket.

The periodontal structures are divided into two zones: (Fig-1)

A) Zone of irritation
B) Zone of co-destruction

A. **ZONE OF IRRITATION:** This consists of marginal gingiva and interdental gingival papillae and is bound by the gingival fibers. Local irritants stimulate inflammation in this zone. Degeneration and necrosis of gingival connective tissue, epithelial ulceration and suppuration are its most severe destructive effect. This progression results in even (horizontal) bone destruction.[6]

International Journal Dental and Medical Sciences Research
Volume 3, Issue 3, May - June 2021 pp 612-621 www.ijdmsrjournal.com ISSN: 2582-6018

B. **ZONE OF CO-DESTRUCTION:** This includes periodontal ligament, the root cementum and alveolar bone. It is demarcated coronally by transseptal (interdental and dentoalveolar) collagen fiber bundles. (Fig-2)

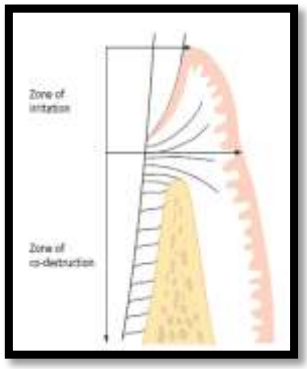


Fig. 1 Schematic drawing of Zone of irritation and the Zone of Co- destruction according to Glickman

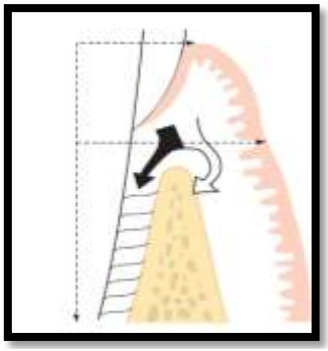


Fig. 2 The inflammatory lesion in the Zone of irritation can, in teeth not subjected to trauma, propagate into the alveolar bone (open arrow), While in teeth also subjected to trauma from occlusion, the inflammatory infiltrate spreads directly into periodontal ligament (filled arrow)

The spread of inflammation may occur directly into periodontal ligament from zone of irritation leading to development of angular bony defect.[19, 20]

Then Glickman and Smulow (1969) experimented by applying excessive occlusion forces combined with inflammation severe enough to involve the periodontal tissue, the fluid & cellular exudates followed the least resistant pathway. They suggested that excessive occlusion forces changed the pathways of spread of inflammation, i.e. extending directly to periodontal ligament, leading to angular resorption of alveolar bone and infrabony pocket formation.[19, 21]

In 1979, Waerhaug,[22] again evaluated human autopsy material and 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 occlusion 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 was always present in close proximity to site of periodontal destruction (Fig-3). He also indicated that there was no evidence of the changes purported to be present in the altered pathway of destruction caused by occlusion trauma. Waerhaug conclusion was that occlusal trauma played no part in periodontal destruction and plaque –related inflammation was the only periodontal disease.

International Journal Dental and Medical Sciences Research
Volume 3, Issue 3, May - June 2021 pp 612-621 www.ijdmsrjournal.com ISSN: 2582-6018

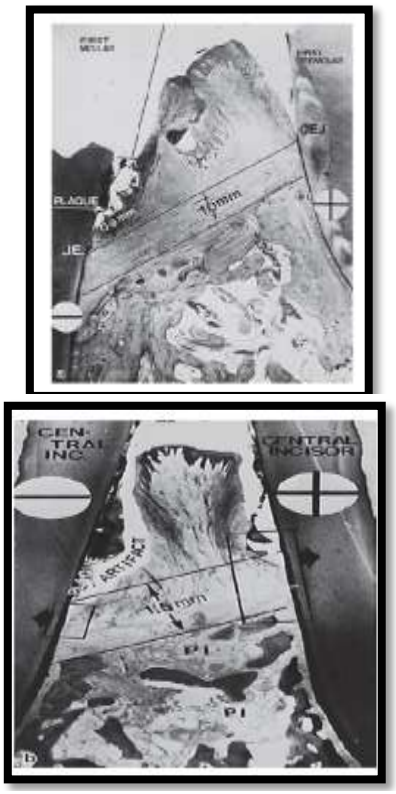


Fig-3: Microphotographs illustrating two interproximal areas with angular bony defects. "–" denotes a tooth not subjected and "+" denotes a tooth subjected to trauma from occlusion. In categories "–" and "+" the distance between the apical cells of the junctional epithelium and the supporting alveolar bone is about 1–1.5 mm, and the distance between the apical extension of plaque and the apical cells of the junctional epithelium about 1 mm.

Animal Research

The most significant animal studies were performed in the 1970s by two research group, one at Eastman dental centre in Rochester, New York by Polson et al[23-26];they found that occlusal forces trauma did not induce progressive periodontal tissue destruction under that condition.[27] and the other at the university of Gothenburg in Sweden By Lindhe et al[28-30]and they often are referred to as the American and the Scandinavian occlusal studies, respectively ; Working with Beagles doges, they observed that experimentally induced trauma from occlusion (jiggling forces) did not result in loss of connective attachment or the formation of periodontal pockets in animals with normal gingiva or even in animals with marginal gingivitis, occlusal trauma resulted in induction of increased tooth mobility and increased tooth mobility and an increased rate of tissue destruction in periodontium as well as angular osseous defects and infrabony pockets[28]. Both evaluated the effect of occlusal trauma and gingival inflammation in animals.

Human Studies

One study evaluated teeth with balancing contacts in relation to teeth without balancing contacts. Teeth with non-working contacts showed greater periodontal destruction and pocket depth.[31] Another similar study showed no difference between the two groups.[32]

A study by Fleszar et al in 1980 reported the influence of tooth mobility on healing after periodontal therapy, which includes both root debridement and occlusal adjustment.[33]In another study, however, Burgett et al studied the effect of occlusal adjustment in treatment of periodontitis; observed an average 0.5 mm more probing attachment gain in patients who received combined treatment (root debridement, flap surgery and comprehensive occlusal therapy) than patients in whom occlusal adjustment was not done.[34]

As part of a large study on prognosis, McGuire and Nunn[35,36] 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 the prognosis despite periodontal therapy.

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.[37]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 forces. This report also agreed that excessive occlusal forces alone do not initiate plaque induced gingival disease or loss of connective tissue associated with periodontitis.[38]

Nunn and Harrel (2001)[39] evaluated the relationship between occlusal discrepancies and periodontal disease.They concluded that there is strong association between occlusion discrepancies and various clinical parameters indicative of periodontal disease.

Harrel and Nunn (2001)[40]studied the association between occlusal forces and gingival recession. They did not find any statistically significant relationship between occlusal discrepancies and gingival recession. They evaluated the increase or decrease in the width of gingiva to determine if occlusal discrepancies contributed to decrease in the width of gingival tissue consistent with recession.

III. CLASSIFICATIONS

I. Box classification[41]

A. Physiologic occlusion:

it as a condition in which the occlusion are in a state of equilibrium and they do not and cannot change the normal relationship existing between the tooth and its supporting structures. In this, the occlusal pressure against the tooth is balanced by the resistance of periodontal tissues.

B. Traumatic occlusion:

The damage produced in the periodontium is due to the overstress produced by the occlusion.

II. Glickman Classification

A) Depending on the duration of cause:

a) Acute Trauma from Occlusion: It results from an abrupt change in the occlusal force such as that produced by biting on a hard object. In addition, restorations and prosthesis that interfere with or alter the direction of occlusal forces on the teeth may induce acute trauma.

b) Chronic Trauma from Occlusion:It may be produced by gradual changes like tooth wear, drift, extrusion or parafunctional habits like bruxism or clenching.

B) Depending on the nature of cause: (Fig-4)

a) Primary Trauma from Occlusion:

- Criteria are
  - Periodontium is previously healthy
  - Trauma from occlusion is considered as the prime etiologic factor.
  - The only local alteration to which the tooth is subjected is from occlusion.
- Causes are
  - High fillings
  - A prosthesis that creates excessive forces on abutments and antagonists.
  - Drift / extrusion of teeth into the place of missing but unreplaced teeth.
  - Orthodontic movement of teeth into functionally unacceptable positions.
- Effects are
  - No change in the level of connective tissue attachment.
  - No initiation of pocket formation.
  - The reason for this is that the supracrestal gingival fibers are not affected and therefore prevent the apical migration of epithelium.

b) Secondary Trauma from Occlusion

- Criteria are
  - Adaptive capacity of the periodontium to withstand occlusal forces is impaired
  - Periodontium becomes vulnerable to injury and previously well tolerated occlusal forces become traumatic.
  - Trauma from occlusion is considered a secondary cause of periodontal destruction.
- Causes are
  - Alveolar bone loss due to marginal inflammation reduces the periodontal attachment area. This increases the burden on the remaining tissues.
  - Systemic disorders may reduce tissue resistance and the forces previously tolerable may become excessive.

International Journal Dental and Medical Sciences Research
Volume 3, Issue 3, May - June 2021 pp 612-621 www.ijdmsrjournal.com ISSN: 2582-6018

Fig-4: Clinical Features of TFO


Fig-5: Radiographic Features of TFO

Signs and Symptoms:

These indicators of trauma from occlusion may include one or more of the following.[4]

1) Mobility (progressive)
2) Fremitus
3) Wear facets in the presence of other clinical indicators
4) Occlusal prematurity/discrepancies
5) Pain on chewing or percussion
6) Tooth migration
7) Chipped or fractured tooth (teeth)
8) Thermal sensitivity

Radiographic (Fig-5)

1) Widened PDL space
2) Bone loss (furcation; vertical; circumferential)
3) Root resorption

JIGGLING-TYPE TRAUMA

Jiggling forces are the forces which are produced by multi-direction displacement of a tooth in alternating buccolingual or mesiodistal directions.[6] The tissue reactions in the periodontal ligament provoked by the combined pressure and tension forces were found to be similar, however, to those reported for the pressure zone at orthodontically moved teeth, with one difference that the periodontal ligament space at jiggling gradually increased in width on both sides of the tooth.

Histologic features

These changes may include widening/compression of the periodontal ligament, bone remodelling (resorption/ repair), hyalinization-necrosis, increased cellularity, vascular dilatation/permeability, thrombosis, root resorption, and cemental tears.

STAGES OF TISSUE RESPONSE

Tissue response occurs in three stages[42]: injury, repair and adaptive remodelling of the periodontium.

STAGE I: INJURY

Tissue injury is produced by excessive occlusal forces. The body then attempts to repair the injury and restore the periodontium. This can occur if the forces are diminished or if the tooth drifts away from them. If the offending force is chronic, the periodontium is remodeled to cushion the effect.

STAGE II: REPAIR

Repair is constantly occurring in normal periodontium and trauma from occlusion stimulates increased reparative activity. The damaged tissues are removed, and new connective tissue cells and fibers, bone and cementum are formed in an attempt to restore the injured periodontium. Forces remain traumatic as long as the damage produced exceeds the reparative capacity of the tissues.

International Journal Dental and Medical Sciences Research
Volume 3, Issue 3, May - June 2021 pp 612-621 www.ijdmsrjournal.com ISSN: 2582-6018

Bone formation occurs as a part of the reparative process in trauma from occlusion, but because of its location is significance may extend beyond that of restoring injured tissue. The term "buttressing bone formation." Buttressing bone formation occurs centrally and peripherally. When it occurs centrally, within the jaw, it is located along the endosteal surfaces of the trabeculae and does not alter the gross morphology of bone. When buttressing bone formation occurs peripherally, on the external surface, it may produce a bulbous contour in the buccal or lingual plate or a pronounced ridge at the cervical bone margin referred to as "lipping".

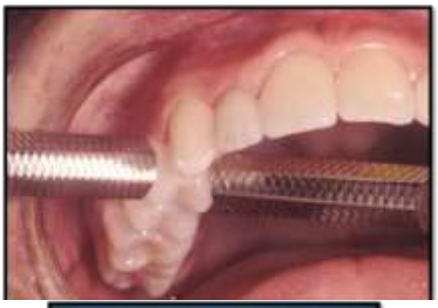
STAGE III: ADAPTIVE REMODELLING OF THE PERIODONTIUM

If the repair process cannot keep pace with the destruction caused by the occlusion, the periodontium is remodelled in an effort to create a structural relationship in which the forces are no longer injurious to the tissues. The result is the thickened periodontal ligament, which is funnel shaped at the crest and angular defects in the bone with no pocket formation. The involved teeth becomes loose.

Diagnosis

Assessment of tooth mobility constitutes a basic part of the comprehensive periodontal examination.[43] The mobility status of the dentition has traditionally been evaluated by

i. Visual assessment[44] (Fig-6)
ii. Periotest[45](Fig-7)
iii. Fremitus[46](Fig-8)



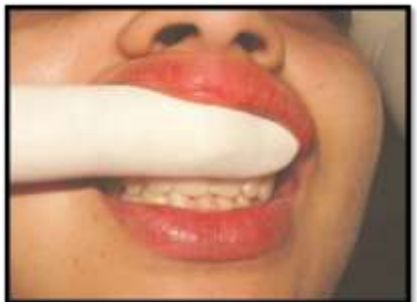


Fig-6: Assessing mobility using two Instrument handles
Fig-7: Periotest
Fig-8: Fremitus Test

Treatment modalities

The ultimate goal of any dental treatment should be to provide optimum oral health.[47] When optimum oral health is the goal, diagnosis and treatment planing can be condensed into two fundamental objectives:

1. Finding all factors that contribute in any way to deterioration of oral health.
2. Determining the best method of eliminating each factor of deterioration.

The goal of periodontal therapy in the treatment of occlusal traumatism should be to maintain the periodontium in comfort and function. In order to achieve this goal a number of treatment considerations may be considered including one or more of the following:[43]

i. Occlusal adjustments
ii. Temporary, provisional or long term stabilization of mobile teeth with removable or fixed appliances
iii. Orthodontic tooth movement
iv. Occlusal reconstruction
v. Management of parafunction habits
vi. Extraction of selected teeth

IV. CONCLUSION

Based on the extensive literature survey the following conclusions can be drawn that Occlusal trauma does not initiate periodontitis, and there is weak evidence that it alters the progression of the disease. There is no credible evidence to support the existence of abfraction or implicate it as a cause of gingival recession. Reduction of tooth mobility may enhance the effect of periodontal therapy.

REFERENCES

[1]. Serio FG, Hawley CE. Periodontal trauma and mobility. Dent Clin North Am 1999; 43:37-43.
[2]. Glickman I, Roeber FW, Brion M, Pameijer JH. Photoelastic analysis of internal stresses in the periodontium created by occlusal forces. J Periodontol 1963; 34:5-10.
[3]. Klineberg I: The lateral pterygoid muscle: some anatomical, physiological and clinical considerations. Ann R Australas Coll Dent Surg 11: 96e108, 1991
[4]. Glickman I. Role of occlusion in the etiology and treatment of periodontal disease. J Dent Res 1971 50:199-204.
[5]. Stillman PK. The management of pyorrhoea. Dent Cosmos, 1917, 59:405. Cited from Hallmon W, Harrel S. Occlusal analysis, diagnosis and management in the practice of periodontics. Periodontol 2000 2004; 34:151-164.
[6]. Lindhe J, Lang NP, Karring T. Clinical periodontology and implant dentistry. 5th ed. U.K. Blackwell Publishing Co.;2008.
[7]. Glickman I. Clinical periodontology, 10th Ed. Philadelphia. W. B Saunders Co, 2006.
[8]. Newman S, Lindhe J, Ericsson I. The effect of progressive tooth mobility on destructive periodontitis in the dog. J Clin Periodontol 1978; 7:351-360.
[9]. Wank GS, Kroll YJ. Occlusal trauma. An evaluation of its relationship to periodontal prostheses. Dent Clin North Am 1981, 25:511-532.
[10]. Burgett FG. Trauma from occlusion-periodontal concerns. Dent Clin North Am 1995; 39:301-311.
[11]. Stallard RE. Occlusion and periodontal disease. Dent Clin North Am 1969; 13:599.
[12]. Gerald S. Wank, Kroll YJ. Occlusal trauma-An evaluation of its relationship to periodontal prosthesis. Dent Clin North Am 1981, 25:511-532.
[13]. Zander H, Polson A. Present status of occlusal therapy in periodontics. J Periodontol 1977, 48:540.

[14]. Glickman I. Role of occlusion in the etiology and treatment of periodontal disease. J Dent Res
[15]. Stillman PR. What is traumatic occlusion and how can it be diagnosed and corrected. J Am Dent Assoc 1926; 12:1330-1338.
[16]. McCall J. Traumatic occlusion. J Am Dent Assoc 1939; 26:519-526.
[17]. Macapanpan LC, Weinmann JP. The influence of injury to the periodontal membrane on the spread of gingival inflammation. J Dent Res 1954; 33:263-272.
[18]. Weimann J. Progress of gingival inflammation into the supporting structure of teeth. J Periodontol 1941; 12:71-76.1971, 50:199.
[19]. Glickman I, Smulow J. The combined effects of inflammation and trauma from occlusion in periodontics. Int Dent J 1969, 19:393.
[20]. Glickman I, Sumlow JB. Effect of excessive occlusal forces upon the pathway of gingival inflammation in human. J Periodontol 1965, 36:141-147.
[21]. Stahl S. The response of the periodontium to combined gingival inflammation and occluso-functional stresses in four human surgical specimens. Periodontics 1968; 6:14-22.
[22]. Waerhaug J. The infrabony pockets and its relationship to trauma from occlusion and subgingival plaque. J Periodontol 1979;50(7):355-365.
[23]. Polson A, Kennedy J, Zander H. Trauma and progression of marginal periodontitis in squirrel monkeys, part I: co-destructive factors of periodontitis and thermally produced injuries. J Periodontol Res 1974; 9:100-107.
[24]. Poison A. Trauma and progression of marginal periodontitis in squirrel monkeys, part II: co-destructive factors of periodontitis and mechanically produced injuries. J Periodontol Res 1974; 9:108-113.
[25]. Poison A, Meitner S, Zander H. Trauma and progression of marginal periodontitis in squirrel monkeys, part III: adaption of inter-proximal alveolar bone to repetitive injury. J Periodontol Res 1976 11:279-289.
[26]. Poison A, Meitner S, Zander H. Trauma and progression of marginal periodontitis in squirrel monkeys, Part IV: reversibility of bone loss due to trauma alone and trauma superimposed upon periodontitis. J Periodontol Res 1976; 11:290-298.
[27]. Ericson I, Lindhe J. Lack of effect of trauma from occlusion on the recurrence of experimental periodontitis. J Clin Periodontol 1977; 4:115.
[28]. Lindhe J, Svenberg G. Influence of trauma from occlusion on the progression of experimental periodontitis in the beagle dogs. J Clin Periodontol 1974; 1:3-14.
[29]. Lindhe J, Ericsson I. The influence of trauma from occlusion on reduced but healthy periodontal tissue in dogs. J Clin Periodontol 1976; 3:110-122.
[30]. Lindhe J, Ericson I. The effect of elimination of jiggling forces on periodontally exposed teeth in the dog. J Periodontol 1982; 9:497-503.
[31]. Yuodelies RA, Mann WV Jr. The prevalence of and possible role of non-working contacts in periodontal disease. Periodontics 1965; 3:219-223.
[32]. Shefter GJ, McFall WT Jr. Occlusal relations and periodontal status in human adults. J Periodontol 1986; 57:1-6.
[33]. Philstorm B, Anderson K, Aeppli D, Schaffer E. Association between signs of trauma from occlusion and periodontitis. J Periodontol 1986; 57:1-6.
[34]. Burgett F, Ramfjord S, Nissle R, Morrison E, Charbeneau T, Caffesse R. A randomized trial of occlusal adjustment in the treatment of periodontitis patients. J Clin Periodontol 1992: 19:381-387.
[35]. McGuire MK, Nunn ME. Prognosis versus actual outcome, Part II: the effectiveness of clinical parameters in developing an accurate prognosis. J Periodontol 1996; 67:658-665.
[36]. McGuire MK, Nunn ME. Prognosis versus actual outcome, Part III: the effectiveness of clinical parameters in accurately predicting tooth survival. J Periodontol 1996; 67:666-674.
[37]. Gher M. Non-surgical pocket therapy: dental occlusion. Ann Periodontol 1996; 4:102-108.
[38]. Hallmon W. Occlusal trauma: Effect and impact on periodontium. Ann Periodontol 1999 4:102-107.
[39]. Nunn ME, Harrel SK. The effect of occlusal discrepancies on periodontitis. I . Relationship of initial occlusal discrepancies to initial clinical parameters. J Periodontol 2001, 72:485-494.
[40]. Harrel SK, Nunn ME. The effect of occlusal discrepancies on periodontitis. II. Relationship of occlusal treatment to

progression of periodontal disease. J Periodontol 2001, 72:495-505.
[41]. Sidney Sorrin .The practice of periodontia. CH.9: Occlusal equilibration: Habits and Food impaction: 224-298.McGraw Hill Book Co. 1960.
[42]. Carranza FA Jr. Histometric evaluation of periodontal pathology. J Periodontol 1970; 38:741.
[43]. American Academy of Periodontology Parameters of care. J Periodontol 2000; 71:873-875.
[44]. Miller SC. Textbook of Periodontia, 3"I ed. Philadelphia: The Blakeston Co.; 1950.
[45]. Schulte W, d'Hoedt B, Lucas D, Maunz M, Steppeler M. Periotest for measuring periodontal characteristics- Correlation with periodontal bone loss. J Periodontol Res 1992; 27:184-190.
[46]. Wilkins EM. Clinical practice of the dental hygienist. 2008
[47]. Dawson P.E. Occlusal Problems. Mosby Co. 1974; 103-106.