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Trauma From Occlusion - Periodontal Traumatism

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ABSTRACT

Patients suffering from occlusal trauma may experience tooth mobility, temperomandibular joint pain, masticatory pain, and periodontal disease. An early diagnosis, a proper treatment plan, and malocclusion correction can all lead to a successful outcome. In patients with occlusal trauma, a lack of awareness of orthodontic treatment can result in tooth structure loss. The effects of occlusal trauma and excessive occlusal forces on the periodontium are investigated in this narrative review, which includes the onset and progression of periodontitis, abfraction, and gingival recession.

Keywords: trauma from occlusion, jiggling forces, coronoplasty, splinting, peri-implant occlusal load

INTRODUCTION

The periodontal ligament acts on forces applied to the teeth as a way of accommodating the forces exerted on the crown because of the elastic character of the periodontal ligament and all the teeth that have a regular bone support with physiological movement in all directions, when occlusal strength increases. Thus, it has a cushioning effect on forces exerted on the tooth in all directions. It is primarily determined by the magnitude, direction, duration, and frequency.[1] When occlusal forces are increased in magnitude, the periodontium responds by widening the periodontal ligament space, increasing the number and width of periodontal ligament fibres, and increasing the density of alveolar bone. Changing the direction of occlusal forces causes the stresses and strains within the periodontium to reorient. The principal fibres of the periodontal ligament are arranged to best accommodate occlusal forces along the tooth's long axis. Lateral (horizontal) and torque (rotational) forces are more likely to cause periodontal injury. The duration and frequency of occlusal forces affect the response of alveolar bone, with constant pressure being more damaging than intermittent forces. The more frequently an intermittent force is applied, the more damaging it is to the periodontium.[1]

TRAUMA FROM OCCLUSION

Whenocclusal forces exceed the adaptive capacity of the tissues, tissue injury results. The resultant injury is termed trauma from occlusion. Trauma from occlusion refers to the tissue injury, not the occlusal force. The term trauma from occlusion is generally used in connection with injury in the periodontium.An occlusion that produces such injury is called a traumatic

occlusion.Excessive occlusal forces may also disrupt the function of the masticatory musculature and cause painful spasms, injure the temperomandibular joints, or produce excessive tooth wear.[2]

ETIOLOGY

The etiology of occlusion-related trauma includes both intrinsic and extrinsic factors. Intrinsic factors include the orientation of the teeth's long axis in relation to the forces to which they are subjected, the morphology of the roots, and the morphology of the alveolar process. Extrinsic factors include plaque, the fabrication of long span bridges on a few teeth, injudicious bone resection during surgical procedures, parafunctional habits, food impaction, overhanging fillings, poorly contoured crowns and bridges, and ill-fitting dentures.[1]

CLASSIFICATION OF TRAUMA FROM OCCLUSION

There are four main classification of trauma from occlusion discussed in the article which include, Box et al classification (1930) who classified into Physiologic occlusion was defined by *Box et al* as a condition in which the systems of forces acting on the tooth during occlusion are in a state of equilibrium and do not and cannot change the normal relationship between the tooth and its supporting structures and traumatic occlusion - The periodontium suffers damage as a result of the occlusion's overstress.[3] The second one being Glickman's classification (1953)[1]Trauma from occlusion can be classified as acute or chronic depending on the duration of the cause. It can be divided into primary and secondary trauma from occlusion based on the nature of the cause.[3]The third classification being Hamp, Nyman, and Lindhe's classification (1975), in this classification is based on a horizontal component of tissue destruction in the inter-radicular area, such as the degree of horizontal root exposure or attachment loss.

Degree I refer to horizontal loss of periodontal tissue support that is less than one-third the width of the tooth. Degree II refers to horizontal loss of periodontal support that is greater than one-third the width of the tooth. Degree III refers to horizontal through and through destruction of periodontal tissue in the area of the furcation.[4]

The fourth and final classification being the latest2017 AAP classification[5] where the occlusal trauma is classified into three types: primary occlusal trauma, secondary occlusal trauma, and orthodontic force trauma.[5]

ACUTE TRAUMA AS A RESULT OF OCCLUSION

The result of an abrupt occlusal impact, such as biting on a hard object (e.g., olive pit) is termed as acute trauma from occlusion. Acute trauma can also be caused by restorations or prosthetic appliances that interfere with or change the direction of occlusal forces on the teeth. Acute trauma can also cause cementum tears if the force is dissipated by a shift in tooth position or by wearing away or correcting the restoration and the injury heals and the symptoms go away. Otherwise, periodontal injury may worsen and progress to necrosis, accompanied by the formation of a periodontal abscess, or it may persist as a symptom-free, chronic condition.[1]

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Tooth pain, sensitivity to percussion, and increased tooth mobility were all observed in cases of acute trauma. While it is more common and significant in cases of chronic trauma. Gradual changes were observed, such as tooth wear, drifting movement and extrusion, and parafunctional habits (bruxism, clenching).[1]

Occlusal disharmony, functional imbalance, and occlusal dystrophy are all terms used to describe traumatic occlusal relationships. When trauma from occlusion is caused by changes in occlusal forces, it is referred to as "primary trauma from occlusion." It is referred to as "secondary trauma from occlusion" when it occurs as a result of the tissues' reduced ability to resist occlusal forces.[1]

I. **Primary trauma from occlusion**

Primary occlusal trauma can occur due to following reasons which include inclusion of a "high-filling", inclusion of prosthesis replacement that creates excessive force for abutment and antagonistic teeth; movement or extrusion of teeth into spaces created by missing teeth; primary trauma changes do not alter the level of connective tissue attachment and do not initiate pocket formation. This is most likely due to the fact that the supracrestal gingival fibres are unaffected, preventing apical migration of the junctional epithelium.[1]

II. **Secondary trauma from occlusion**

Secondary trauma from occlusion occurs when bone loss caused by marginal inflammation impairs the tissues' adaptive capacity to withstand occlusal forces. This reduces the area of periodontal attachment and changes the leverage on the remaining tissues. The periodontium becomes more prone to injury, and previously tolerable occlusal forces become traumatic.[1]
Traumatic forces can occur on the following surfacesnormal periodontium with normal bone height, normal periodontium with reduced bone height, or marginal periodontitis with reduced bone height. The first case is an example of primary occlusion trauma, while the last two are examples of secondary occlusion trauma.

STAGES OF TISSUE RESPONSE TO INCREASED OCCLUSAL FORCES

Stages of tissue response to increased occlusal forces include 3 stages. They are Injury, Repair and Adaptive remodelling of the periodontium.

I. Stage I: Injury

Tissue injury is produced by excessive occlusal forces. The body then attempts to repair the injury and restore the periodontium. If the offending force is chronic, the periodontium is remodelled to cushion its impact. The ligament is widened at the expense of the bone, resulting

in angular bone defects without periodontal pockets, and the tooth becomes loose. Under the forces of occlusion, a tooth rotates around a fulcrum or axis of rotation, which in single rooted teeth is located in the junction between the middle third and the apical third of the clinical root. This creates areas of pressure and tension on opposite sides of the fulcrum. Different lesions are produced by different degrees of pressure and tension.[1]

Occlusion trauma causes vascular changes in the periodontium within 30 minutes. Within 2-3 hours, stasis and vasodilation occur. Between 1 and 7 days, the blood vessel walls disintegrate and the contents are released into the surrounding tissue, accompanied by pain and hypersensitivity. Later changes result in bone loss lining the socket, widened periodontal ligament, and increased tooth mobility. Furthermore, increased alveolar bone resorption and tooth surface resorption occur.[1]

TISSUE DESTRUCTIVE CHANGES INDUCED BY TRAUMA FROM OCCLUSION

Occlusion trauma causes vascular changes in the periodontium within 30 minutes. Within 2-3 hours, stasis and vasodilation occur. Between 1 and 7 days, the blood vessel walls disintegrate and the contents are released into the surrounding tissue, accompanied by pain and hypersensitivity. Later changes result in bone loss lining the socket, widened periodontal ligament, and increased tooth mobility. Furthermore, increased alveolar bone resorption and tooth surface resorption occur. Frontal resorption of cortical bone begins in the area of pdl compression and progresses to spongy bone tissue, resulting in periodontal ligament widening (24-38hrs).[1]

MOLECULAR MECHANISMS OF BONE RESORPTION INDUCED BY FORCE APPLICATION

Traumatogenic occlusal forces that slightly exceed the tissue adaption threshold cause very rapid circulatory changes in the periodontal ligament, resulting in platelet aggregation and prostaglandin release, which activates osteoclasts. During orthodontic movement, there is a significant increase in the levels of IL-1 and PGE2 in gingival crevicular fluid in 10 patients, which returns to baseline within 7 days (*Grieve et al* 1994).[6]

Mechanical stimuli on teeth generate free proteins within the periodontal ligament, which are induced either by cell compression and mechanical stress or by cellular destruction and collagen fibredisorganisation following blood vessel collapse. These proteins cause mast cell degranulation in the periodontal ligament. It takes about 90 minutes after applying force to the periodontal ligament for an inflammatory infiltrate to form, eliciting an acid pH that promotes the attraction and accumulation of osteoclasts. Bone resorption began 12 hours after the application of orthodontic force and reached a peak 48 hours after the start of orthodontic movement.[1]

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RANKL expression on endothelial cells, inflammatory cells, and periodontal ligament cells is linked to inflammatory bone resorption. According to ***Walker et al*2008**, occlusal trauma causes an increase in osteoclasts, which is related to the expression of RANKL.[7] Traumatogenic occlusion resulted in the expression of both RANKL and osteopontin in the hyperocclusion mouse model. According to ***Passos et al*2009**, osteopontin and RANKL in bone resorption are related to trauma from occlusion.[8]

II. Stage II: Repair

Normal periodontium is constantly repairing itself, and trauma from occlusion stimulates increased reparative activity. In an attempt to restore the injured periodontium, the damaged tissues are removed, and new connective tissue cells and fibres, bone, and cementum are formed. When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned bony trabeculae with new bone, a process known as buttressing bone formation. It can also happen when bone is destroyed by inflammation or osteolytic tumours that are buttressing the bone.[1]The formation of buttressing bone occurs both within the jaw (central buttressing) and on the bone surface (peripheral buttressing). Endosteal cells deposit new bone, which restores the bony trabeculae and reduces the size of the marrow spaces, resulting in central buttressing. Peripheral buttressing occurs on the alveolar plate's facial and lingual surfaces. Peripheral buttressing can cause a shelf-like thickening of the alveolar margin, known as "lipping," or a pronounced bulge in the contour of the facial and lingual Bone, depending on its severity.[1]

III. Stage III: Adaptive Remodeling of the Periodontium

If the repair process is unable to keep up with the occlusion's destruction. The periodontium is remodelled to form a structural relationship that results in a thickened periodontal ligament, a funnel-shaped crest, and angular defects in the bone, but no pocket formation. Increased vascularization has also been reported as the involved teeth become loose. The relative amounts of periodontal bone surface undergoing resorption or formation have been used to differentiate the three stages in the evolution of traumatic lesions. The injury phase is characterised by increased resorption and decreased bone formation, whereas the repair phase is characterised by decreased resorption and increased bone formation after adaptive remodelling of the periodontium, when resorption and formation return to normal.[1]

CLINICAL AND RADIOGRAPHIC SIGNS OF TRAUMA FROM OCCLUSION

The clinical signs include increased tooth mobility; destruction of periodontal fibres in the injury stage; fractured tooth; widening of the periodontal ligament in the final stage, which also leads to increased tooth mobility; thermal sensitivity; fremitus; wear facets; occlusal discrepancies; tooth migration; discomfort/pain on chewing and cemental tear. The radiographic signs include:[1]periodontal space width, frequently associated with thickening of the lamina dura along the lateral aspect of the root, in the apical region, and in bifurcation areas, interdental septum

destruction that is “vertical” rather than “horizontal”,alveolar bone radiolucency and condensationand resorption of the roots.[1]

EFFECTS OF INSUFFICIENT OCCLUSAL FORCE

Inadequate occlusal force can also be harmful to the supporting periodontal tissues. Inadequate stimulation causes periodontal ligament thinning, fibre atrophy, alveolar bone osteoporosis, and bone height reduction. Hypofunction can be caused by an open-bite relationship, a lack of functional antagonists, or unilateral chewing habits that ignore one side of the mouth.[1]

REVERSIBILITY OF TRAUMATIC LESIONS

Occlusion-related trauma is reversible. If the teeth are unable to escape or adapt to excessive occlusal force, periodontal damage will persist and worsen. Inflammation in the periodontium caused by plaque accumulation may impair the reversibility of traumatic lesions.[1]

RELATIONSHIP BETWEEN PLAQUE-INDUCED PERIODONTAL DISEASES AND TRAUMA FROM OCCLUSION

he bacterial plaque accumulation that causes gingivitis and periodontal pocket formation affects the marginal gingiva, but trauma from occlusion occurs in the supporting tissues and has no effect on the gingiva. Human mobile teeth contain significantly more *Campylobacter rectus* and *Peptostreptococcus micros* than nonmobile teeth. Plaque-induced inflammation enters the zone of co-destruction when it spreads from the gingiva into the supporting periodontal tissues. There have been other theories proposed to explain the interaction of trauma and inflammation.[1]

In 1974, the Eastman Dental Center group in Rochester, New York examined squirrel monkeys that had been subjected to trauma caused by repetitive interdental wedging and mild to moderate gingival inflammation; the experiments lasted up to 10 weeks. Periodontitis-induced attachment loss was not exacerbated by the presence of trauma.[9]

The University of Gothenburg group in Sweden experimented with beagle dogs in 1974, inducing severe gingival inflammation by placing cap splints and orthodontic appliances. Periodontal destruction caused by periodontitis is exacerbated by occlusal stresses.[10]

Trauma from occlusion may alter the pathway of gingival inflammation extension to the underlying tissues, according to other theories proposed to explain the interaction of trauma and inflammation. The bone loss that results would be angular, with pockets that could become intrabony. Deeper lesions may develop as a result of trauma-induced areas of root resorption revealed by apical migration of the inflamed gingival attachment. Supragingival plaque can become subgingival if the tooth is tilted orthodontically or if it migrates into an edentulous area, causing a suprabony pocket to become an intrabony pocket. Increased mobility of traumatised weakened teeth can pump metabolites of plaques, boosting their dispersion.[1]

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INFLUENCE OF TRAUMA FROM OCCLUSION ON PROGRESSION OF MARGINAL PERIODONTITIS

The clinical impressions of early investigators and clinicians assigned an important role to trauma from occlusion in the aetiology of periodontal lesions.[1]Numerous studies have since been performed to determine the mechanisms by which TFO may affect periodontal disease.

These studies can be classified broadly into three categories: - Human autopsy material, Clinical trials and Animal experiments

I. Human Autopsy Material:

Certain criteria were evaluated using human autopsy material. They were based on histopathology of the lesions, presence of apical extension of microbial deposits, mobility and occlusion. Based on these findings, two concepts were proposed: Glickmans concept 1965 and Waerhaug concept 1979

Glickmans concept, 1965

If forces of an abnormal magnitude occur in the form of subgingival plaque, the path of spreading the plaque-related gingival lesion may shift. The periodontal structures can be split into two zones, irritation zone and co-destruction zone, on the basis of this notion. The marginal and interdental gingiva comprise the irritation zone. Only on the side of the soft tissue in this zone are hard tissues bounded, and the occlusion forces do not influence this area. Gingival inflammation is therefore caused not by trauma of occlusion but by irritation from microbial deposits.[11]

The PDL, root cement and alveolar bone are included in the co-destruction zone. The transeptal and the dentoalveolar fibre bundles are coronally defined. There are two possible pathways for fibre bundles separating the two areas: alterations in the co-destruction area caused by the inflammatory lesion maintained by the plaque in the irritation zone and by trauma. The bundle of fibres can be disintegrated and/or parallel to the surface of the root. Altering the normal propagation path results in angular osteoarthritis.[11]

Waerhaugs concept, 1979

The distance between the subsidingives plate and the associated inflammatory periphery of the infiltrate cell on the surface of the adjacent bone was measured by Waerhupp, and he concluded that angular ion defects and infrastructural pockets are equally observed which are not affected by a TFO as in the case of traumatised loss of teething tissue and resorption of the teeth of the bone. Corner ossic deficiencies and infrabony pockets occur once the subgingival plaque of the tooth is more than the microbiota of the surrounding tooth and the volume of the root alveolar bone is relatively high.[12]

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II. Clinical trials

In human patients, various writers have performed clinical studies. ***Rosling et al* 1976** demonstrated that hypermobile teeth had the same level of healing as those near to hard teeth in the infrabony pocket.[13]***Fleaszar et al*1980** showed that clinically mobile teeth with the same disease activity as hard teeth do not respond to periodontal treatments.[14] The teeth of enhanced mobility and extended PDL widening had in fact more pockets, more attachment and less bone support than the teeth with no these symptoms (***Philstrom et al* 1986**).[15]***Burgett et al* 1992** showed that patients who underwent scaling and occlusal adjustment, the probability of an attachment increase was on average approximately 0.5mm higher than in patients who did not receive an occlusal adaptation.[16]***Neiderud et al*1992** substantiated tissue changes with clinically healthy gingiva in movable teeth may lower the resistance to testing supplied by periodontal tissue.[17]

III. Animal experiments

The positioning of high crowns or restorations on the teeth of dogs or monks leading in one direction to a continuous and intermittent stress. When a tooth is exposed to unilateral forces that do not withstand and disperse its periodontal tissue while keeping the tooth stability in such magnitude, frequency or duration, certain well-defined reactions occur in periodontal structures in accordance with changed functional demand. The tooth tilts towards the force when horizontally oriented pressures are applied, causing pressure and tension zones inside the marginal and apical regions of the tooth to grow briefly and migrate to new positions and heals.[2]

When horizontally directed pressures are applied, it tilts towards the force that causes the development of pressure and stress zone in the marginal and apical areas, and the tooth is briefly hypermobile. In experimental animals with jiggling force, traumas of occlusion were examined, usually induced with the help of a high crown paired with an orthodontic instrument. In another way, the teeth were inter-proximally separated by wooden or elastic material to move a tooth to the opposite side. It took 48 hours to remove the wedge and repeat the procedure on the other side.[2]

Alternative traumatic forces are administered orally and lingually or mesially and distally. The PDL space increases on each side, leading to inflammatory alterations, active sound resorption and increasing movement, and there are combinations of pressure and tension zones. At one stage, when the rising breadth is equal to strength, the teeth are hypermobile yet mobility is not progressive any more. A healthy periodontium with low height is capable of adapting to modified functional requirements within specific limits, like that of a periodontal with a normal height.

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Due to the removal ("occlusal adjustment") of the giggling forces, the width of the periodontal ligament will be normalised in this circumstance. The tissue could not adjust in the area of pressure/tension in the presence of plaque-associated periodontal disease and the harm in the areas of co deterioration had an increasingly permanent character. The following alterations were noted: continual destruction of alveolar bone, steady mobility of teeth, fusion of the irritant area and co-destruction zone, apically proliferating dentogingival epithelium, and periodontal disease.[2]

TRAUMAFROM OCCLUSIONPERI-IMPLANT TISSUES

Trauma from occlusion in peri – implant tissues can occur due to various reasons which includeorthodontic loading and alveolar bone,bone reactions to functional loading, excessive occlusal load on implants, static and cyclic loads on implants, load and loss of osseointegration, masticatory occlusal forces on implants
and tooth-implant supported reconstructions

I. Orthodontic loading and alveolar bone

Excessive loading was studied in *Adell et al* 1981, a prevalent cause of implant loss.[18] Early charging seen by *Sagara et al* 1993 may hinder osseointegration.[19]*Isidor et al* 1997 studied non-axial implant forces as an osseointegration risk factor.[20] The bone surrounding an implant – biological reaction to mechanical stress below the threshold in *Asikainen et al* 1997, whereas loss of the marginal bone or osseointegration due to mechanical stress outside the threshold.[21]

II. Bone reactions to functional loading

After long term functional load, *Berglundh et al* 2005 discussed the reactivity of the peri-implant bone in comparison to unloaded controls.[22]AstraTech implants are implanted on the one hand after extraction and Branemark on the other. Results demonstrated that functional implant loading may improve osseointegration.[22]

III. Excessive occlusal load on implants

***Heitz-Mayfield et al*2004** studied on dogs using 2 titanium plasma sprayed implant and 2 sandblasted acid etched implants on each side of the mandible.[23] In the test side, implants were placed after 6months gold crowns are placed in supraocclussion. Result showed that occlusal overload does not result in loss of implant stability.[23]

IV. Static and cyclic loads on implants

Scheppers*1977** demonstrated bone tissue reaction to axial loading and was evaluated using conventional three-unit FDPs in the mandible of Beagle dogs, and compared with that to non-axial loading provoked by installing a distal cantilever of two implants.[24]Gotfredsen et al*2001**analyzed bone reactions around osseointegrated implants to static load in three studies in dogs.[25] Results showed that lateral static load resulting in an adaptive remodeling of the

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periimplant bone.[25]***Duycket al*2001** showed that dynamic load on implants resulted in the establishment of marginal crater defects.[26]

V. Load and loss of osseointegration

Isidor et al*1997** studied excessive occlusal load leading to osseointegration along the entire length of implant that causes implant mobility.[20]Lindhe et al* 1992** used cotton ligatures for increased plaque retention placed around implants. It resulted in mucositis and later peri-implantitis.[17]

VI. Masticatory occlusal forces on implants

***Lundgren et al*1989** examined 8 strain gauge tranducers mounted bilaterally in maxillary complete denture to occlude with mandibular implant suported fixed cantilever prosthesis.[27]Larger closing and chewing forces were measured over the cantilever than implant supported areas.[27]

DIAGNOSIS

Increased tooth movement is cardinal symptom of primary TFO. The mechanical and electronic equipment can assess mobility. Mobility subjective evaluations are carried out as allocated from 0 to 3 in the Miller classification. Tilting and migration of single teeth or whole segments. If there's primary TFO in a fixing device, the pounding of the teeth with an unobtrusive instrument changes from the resonating note with a healthy support structure to a dull note careful examination of chewing muscles to see whether hypertrophy or hypertonic signs are present with probable spasm of a muscle group.TMJ palpation and observation on numerous routes for closure of any deviation from the mandible. The fremitus test is a measurement of the vibrational patterns of the teeth when the tooth is placed in the position and motion of the contact.[1]

MANAGEMENT OF TRAUMA FROM OCCLUSION

Occlusal therapy should concentrate primarily at building stable functional links, including periodontium, favorable to dental health in patients.[1]For the management of TFO the following procedures are used:occlusal adjustment – coronoplasty andorthodontic treatment.

Occlusal analysis is done by using occlusal registration steps, occlusal indicator wax, marking ribbon, red, green, blue nylon ribbon and using articulating paper.

Coronoplasty

Coronoplasty is a selective reduction in occlusal areas with the main objective of affecting mechanical contact and sensory input neuronal pattern.

Objectives of coronoplasty includes changes in the pattern and degree of afferent impulses, lessening of excessive tooth mobility, multiple simultaneous contact spread over the occlusal scheme to effect occlusal stabilization, beneficial change in the pattern of chewing or swallowing

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function, multidirectional mandibular moving patterns and verticalization of occlusal forces on implants.[1]

The following are the steps followed in coronoplasty. The first stepbeing the removal of extensive prematurities and eliminate the deflective shift from retrudedcuspal position (RCP) to intercuspal position (ICP). The second step indicates adjustment of ICP to achieve stable simultaneous, multi-pointed, widely distributed contact . The third Step involves thetest for excessive contact on the incisor teeth. The fourth step codes for the removal of posterior protrusive super contacts and establish contacts that are bilaterally distributed on the anterior teeth. The fifth step ensures removal of lessen mediotrusive interferences. The next following step involves reduction of excessive cusp steepness on the laterotrusive contacts. The seventh step involves elimination of gross occlusal disharmonies. The eighth step being rechecking of tooth contact relationships The last and final step involves polishing of all rough tooth surfaces. [1]

Invasive, irreversible intervention and no reduction in mobility or pocket depth are the disadvantages of coronoplasty. Time for tissue healing must be allowed to take precautions and periodic reassessment should be carried out.

ORTHODONTIC TREATMENT

The orthodontic treatment indications include anterior functional crossbit, tapped teeth and single rooted extruded teeth. Timing of orthodontic treatment: 1) Until the teeth are completely scaled, planned and the patient is trained in oral hygiene, should be commenced. 2) Completed before full correction of the occlusal and periodontal operations. 3) Bonded orthodontic retainers that stabilise the teeth can ensure ideal health and bone regeneration conditions for periodontal treatment.[1]

I. Temporary and Provisional Splinting:

A splint is a rigid appliance used to stabilize and protect an injured part. A temporary splint is used to reduce occlusal forces and stabilize teeth for a limited period of time. A provisional splint is used in borderline cases in which the final results of the periodontal treatment cannot be predicted with certainty at the time of the initial treatment planning. It is beneficial in cases of progressive TFO during the post-operative healing phase.[2]

II. Biteplanes:

A flat maxillary biteplane with cuspid rise is the best universal appliance for prompt temporary elimination of TFO. It is especially useful in patients with bruxism and advanced periodontal disease, since it will induce muscle relaxation and eliminate the bruxism.[1]

CONCLUSION

The primary management is the elimination of abnormal occlusal forces as well as the stabilization of the involved tooth/teeth. Except in the presence of periodontitis, when TFO is

removed, bone loss is reversed. The elimination of periodontal inflammation as well as TFO is critical for complete periodontal health and improving tooth prognosis.

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