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# Trauma and the Periodontal Tissues: A Narrative Review

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## Abstract

The health of the periodontium including the soft tissues – gingiva and periodontal ligament; and the hard tissues – cementum and alveolar bone is of key importance in the overall homeostasis of the dentition. Injury to the periodontal tissues in any form such as microbial, physical, thermal, chemical, mechanical, occlusal and habitual injury affects the harmony of the periodontal attachment apparatus thereby altering the entire functioning of the dentition. The type of tissue damage when trauma is unintentional and iatrogenic tends to be acute and self-limiting. On the other hand, mechanical and occlusal forces could result in chronic damage. This book chapter provides a review on the various forms of non-microbial trauma to the periodontal tissues, their clinical manifestations and its management.

**Keywords:** periodontal trauma, gingival trauma, chemical injury, trauma from occlusion, iatrogenic trauma

## 1. Introduction

A traumatic dental injury represents acute transmission of energy to a tooth and its supporting structures, which results in fracture and/or displacement of the tooth and/or separation or crushing of the supporting tissues (gingival, periodontal ligament, PDL and bone) [1, 2]. Dental injuries can be divided into different categories based on their genesis, anatomy, pathology, or treatment implications (**Figure 1**) [3, 4].

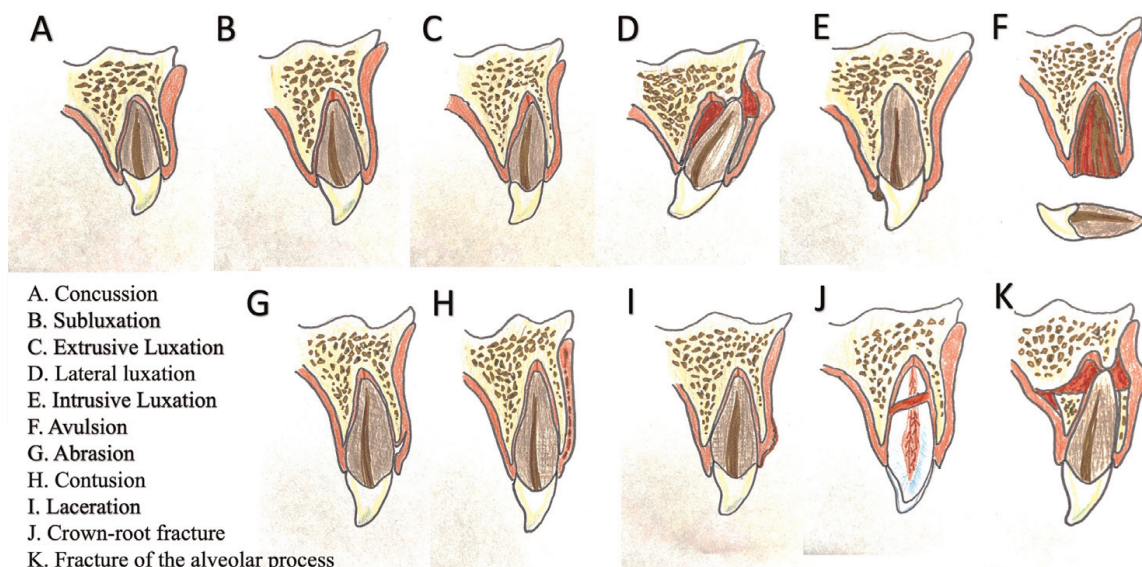
### 1.1 Injuries to the hard dental tissues: pulp, periodontal ligament, alveolar process and supporting tissues

#### 1.1.1 Crown-root fracture

A fracture involving enamel, dentin and cementum. It may or may not expose the pulp.

#### 1.1.2 Root fracture

A fracture involving dentin, cementum and the pulp. Root fractures can be further classified according to displacement of the coronal fragment.



**Figure 1.**  
*Traumatic injuries to the periodontal tissues.*

### 1.1.3 Fracture of the alveolar socket wall

A fracture of the alveolar process which involves the alveolar socket.

### 1.1.4 Fracture of the alveolar process

Fracture of the alveolar process that may or may not involve the alveolar socket.

### 1.1.5 Concussion

An injury to the tooth- supporting structures without abnormal loosening or displacement of the tooth, but with marked pain to percussion.

### 1.1.6 Subluxation (loosening)

An injury to the tooth-supporting structures resulting in increased mobility, but without displacement of the tooth.

### 1.1.7 Extrusive luxation (peripheral dislocation, partial avulsion)

Partial displacement of the tooth out of its socket.

### 1.1.8 Lateral luxation

Displacement of the tooth in a direction other than axially. Displacement is accompanied by comminution or fracture of either the labial or the palatal/lingual alveolar bone.

### 1.1.9 Intrusive luxation (central dislocation)

Displacement of the tooth into the alveolar bone. This injury is accompanied by comminution or fracture of the alveolar socket.

#### *1.1.10 Avulsion (exarticulation)*

The tooth is completely displaced out of its socket.

#### *1.1.11 Abrasion*

A superficial wound produced by rubbing or scraping of the skin or mucosa leaving a raw, bleeding surface.

#### *1.1.12 Contusion*

A bruise without a break in the skin or mucosa. Subcutaneous or submucosal haemorrhage in the tissue. A contusion may be isolated to the soft tissue but may also indicate an underlying bone fracture.

#### *1.1.13 Laceration*

A shallow or deep wound penetrating into the soft tissue, usually produced by a sharp object. May disrupt blood vessels, nerves, muscles and involve salivary glands. Most frequently seen in lips, oral mucosa and gingiva. More seldom the tongue is involved.

#### *1.1.14 Soft tissue avulsion*

Avulsion (loss of tissue) injuries are rare but seen with bite injuries or as a result of a very deep and extended abrasion.

## **2. Thermal traumatic injuries (TTI)**

The use of overheated handpieces and ultrasonic scalers can iatrogenically result in thermal traumatic injuries to the gingiva. Thermal burns, however, have been listed as one of the potential side effects that could be brought on by using ultrasonic scalers [5]. TTI can also be brought on by consuming unusually hot/cold meals or drinks. However, persons with psychiatric illnesses have been documented to experience severe cases of intraoral and laryngopharyngeal burn brought on by hot foods or beverages [6]. Because the supporting periodontal tissue is unconstrained by space and has a larger blood supply than the pulp, heat-generating devices are more likely to cause a temperature increase in the periodontal ligament than the pulp [7].

## **3. Mechanical traumatic injuries (MTI)**

Accidents, assaults, falls during play or sports, and convulsions can all lead to mechanical traumatic injuries (MTI) to the gingival tissue. It has also been noted that malocclusions such a deep bite or an enhanced overbite can harm the gingiva [8, 9]. Dental appliances that were manufactured incorrectly and defective dental restorations might cause iatrogenic mechanical stress [10, 11]. MTI can also occur when one bites or sucks on a fingernail or other hard, sharp object, improper flossing [12].

Gingivitis artefacta are the most frequently described self-inflicted MTI to the gingival tissue [13]. According to reports, gingivitis artefacta minor is more prevalent, whereas gingivitis artefacta major is more severe and spreads to the periodontium's deeper tissues. In gingivitis artefacta minor, there is typically a previous source of irritation, such as a habit of biting one's fingers or eating foods that are abrasive. As a result, the condition can be treated effectively by removing the irritant from its source. On the other hand, gingivitis artefacta major requires specialised treatment approaches because the condition may have an emotional and extraoral component [14].

Stewart and Kernohan [15] grouped gingival injuries resulting from self-inflicted physical trauma into three categories: Type A injuries are those that are added on top of pre-existing lesions (or irritations) where the patient continues to cause harm to the area, Type B injuries are those that are caused by habits like fingernail biting or finger sucking, and Type C injuries are those with unknown and/or complex etiologies that are typically brought on by emotional disturbances [15]. Oral piercing is another MTI to the oral soft tissues that is self-inflicted [16]. The prevalence of oral and peri-oral piercings among young adults ranged from 0.8 percent to 12 percent, according to a comprehensive review [17].

Oral and peri-oral tissues are perforated in order to place jewellery on various facial features, including the tongue, lips, cheek, and labial frenum. However, complications could develop after surgery [17–20]. In addition to increased plaque and calculus production, gingival inflammation may also be one of the main post-operative periodontal problems [17, 20]. Chronic bad oral hygiene, particularly in people who smoke a lot, can lead to secondary post-operative periodontal problems. Additionally, when the jewellery material is porous, there may be a change in the bacterial population and a rise in the pathogenic potential of the periodontopathogens bacteria leading to mucogingival defects and abscesses of the supporting tissues [17, 20].

While the only reasons for getting an oral piercing may be a trend, group identification, attractiveness, other types of self-inflicted MTI to the gingival tissue may be caused by potentially dangerous oral behaviour [16, 21]. Neuroses, occupational habits, and other habits were used to categorise these oral behaviour [22]. Neuroses include biting the lips, cheeks, pencils, pens, and fingernails. Occupational habits include holding nails in the mouth by upholsterers, carpenters and biting thread by tailors. Miscellaneous habits include smoking pipes or cigarettes, thumb sucking, mouth breathing, and using the wrong toothbrushing technique [22].

### **3.1 Clinical presentations and management of trauma to the gingiva**

The numerous factors already mentioned lead to a wide range of potential clinical manifestations. Depending on the unique characteristics of each instance, the history elicited will vary from case to case. Patients who engage in self-harming behaviour may have a history of continuous gingival irritation, which leads to picking or scratching of the gingiva, as well as a history that may point to an emotional disorder or psychological imbalance [13, 14, 23]. A history may also indicate long-standing behaviour like compulsive toothbrushing, digit sucking, or biting on potentially harmful things, as well as interaction with other potentially harmful mechanical, chemical, or thermal objects.

When examined, there may be significant ulcerative lesions affecting the lips, tongue, and gingival oral mucosa in addition to intense burns, scorching, and bleeding [13]. The ulcer may occasionally appear as coagulative necrosis coated in slough. In

factitious injuries, bite marks on the lips and nail marks on the gingiva can both be seen [14]. Usually, there is gingival recession, which can be severe and widespread, affecting numerous teeth. Depending on the affected location, mouth opening may be restricted and chewing may be challenging. It's possible that there is not any laboratory or radiologic proof of an underlying systemic illness.

Complete re-epithelialization of the gingival tissue, complete or considerable root covering, and an expansion of the zone of keratinized gingiva are the treatment's primary objectives [24]. Additionally, it's crucial to stop a recurrence by getting rid of any identified causal factors. Therefore, the first step in treatment is to cure any improper oral hygiene habits and get rid of any dangerous substances. To eliminate the accumulated plaque, gentle mechanical oral hygiene techniques are implemented; if necessary, this may be done with topical or local anaesthetic medications. In the beginning, regular brushing in the other areas may be continued while chemical plaque management is advised as the only oral hygiene measure in the affected area. Two times a day, chlorhexidine mouthwash may be recommended. Warm saline mouth wash and Betadine has also been found helpful [25, 26].

Supportive symptomatic care is provided to preserve the patient's overall health, and this includes the recommendation of a non-spicy, soft food, as well as multivitamins and topical analgesics such triamcinolone acetate and benzocaine to ease discomfort [26]. Topical steroid triamcinolone should be administered in conjunction with carboxymethyl cellulose in cases of significant tissue injury [25]. To eliminate the infection, antibiotic treatment could be required. Factitious situations may necessitate professional psychiatric care, which may involve the use of antidepressants and/or antianxiety medications [14]. A significant loss of gingival tissue may need the use of a free gingival graft to repair the damage [12].

### **3.2 Trauma to periodontal ligaments**

The soft complex connective tissue that serves as an interface between the cementum covering the roots and the inner wall of the alveolar bone is vascular, highly cellular, and specialised [27, 28]. The periodontal ligaments may become overextended and inflamed in circumstances where there are too many bilateral opposing vector stresses. The term "sprained tooth syndrome" is sometimes used to describe this (STS.). This can happen when someone unintentionally bites down on a hard object, when a tooth is improperly or excessively filled, when teeth are drifting, or in cases of sinusitis or allergies. The aberrant outward lateral pressure from the tongue to the teeth that results in temporary orthodontic pressure and outer movement outward and abnormal tooth mobility has been linked to STS in cases of upper respiratory tract infection.

Every effort must be made to avoid iatrogenic forms of trauma to the periodontal ligament because its attempts at healing may have unpredictable outcomes. Periodontal ligament may heal favourably without resorption or with repair related surface resorption and it can also heal unfavourably with osseous replacement resorption (ankylosis) or with inflammatory resorption [29].

Treatment for sprained teeth focuses mostly on reversing the opposing vector forces that caused the strain. A broken tooth, on the other hand, can wait a few days to see whether it heals by itself. Rest is necessary for the treatment of a sprained tooth because using the injured teeth for chewing and speaking causes further discomfort [23].

Radiographic evaluation, tooth realignment, surgical or orthodontic extrusion of the afflicted tooth, splinting, occlusal correction, antimicrobial therapy, endodontic

treatment, and follow-up are all necessary for the management of invasive luxation [30]. All of the aforementioned procedures are likewise necessary for lateral and extrusive luxation, with the exception of tooth extrusion. While concussion simply needs a radiographic evaluation and follow-up to monitor the tooth over time, sub-luxation only needs a radiographic evaluation, splinting, and follow-up [30]. Only if there is no contamination and the tooth was transported and stored in a manner that ensured the health of the periodontal ligaments could an avulsed tooth be successfully reimplanted. After reimplantation, the tooth will be immobilised, and endodontic procedure may be initiated later [31].

It has been established that higher temperatures have detrimental effects on the periodontal ligament [32]. Clinical signs of protein denaturation in periodontal ligaments, disruption of the blood supply to the periodontal ligament, and tooth ankylosis may result from thermal injury to periodontal disorders [32, 33]. Thermal injuries are mostly managed by prevention. Using enough cooling water for tooth preparation or when dry cutting is required, applying gentle pressure, and limiting the bur-contact duration to less than 20 seconds at a time are just a few of the clinical recommendations that have been made to help prevent thermal injuries during dental treatment [17].

## 4. Chemical burns

### 4.1 Classification

Chemical burns majorly occur by the action of irritants on the mucosal tissues (Table 1). Mucosal damage caused by chemical burns could be

1. Iatrogenic as in irrigation performing endodontic procedures
2. Inappropriate application of chemicals and non-therapeutic agents by the patients.

#### 4.1.1 Alendronate

It is a constituent of the diphosphonate family and has been used to treat osteoporosis caused on by glucocorticoids as well as a number of other bone disorders. Alendronate may have side effects, such as esophagitis on the mucosa of the upper

Dental materials	Medications	Nontherapeutic agents	Drugs
<ul style="list-style-type: none"> <li>• Arsenic [34]</li> <li>• Calcium hydroxide [35, 36]</li> <li>• Cavity varnish [37]</li> <li>• Chromic acid [38]</li> <li>• Dentine-bonding agent [39]</li> <li>• Ferric sulfate [40]</li> <li>• Formocresol [41]</li> <li>• Iodine [42]</li> <li>• Paraformaldehyde [42]</li> <li>• Eugenol [43, 44]</li> </ul>	<ul style="list-style-type: none"> <li>Alendronate [45, 46]</li> <li>Aspirin [47, 48]</li> <li>Chlorpromazine [49]</li> <li>Promazine [49]</li> <li>Tetracycline hydrochloride [50–52]</li> </ul>	<ul style="list-style-type: none"> <li>• Arrack [53]</li> <li>• Battery acid [49]</li> <li>• Denture cleansers [54, 55]</li> <li>• Garlic [56]</li> <li>• Gasoline [57]</li> <li>• H<sub>2</sub>O<sub>2</sub> [58, 59]</li> <li>• Minard's Liniment [60]</li> <li>• Mouthwashes [61–64]</li> <li>• NaOCl [65, 66]</li> </ul>	<ul style="list-style-type: none"> <li>Amphetamine [67]</li> <li>Cocaine [68–72]</li> <li>MDMA [72]</li> </ul>

**Table 1.**  
*Aetiology for chemical burns of the oral mucosa.*

aerodigestive tract is the most typical one. Patients must be instructed to take the drug with a glass of water, not to chew or suck the tablet, and to stand up straight for around 30 minutes in order to avoid this side effect [45].

#### 4.1.2 Aspirin

Acetylsalicylic acid, popularly known as aspirin, is a frequently prescribed drug for the treatment of pain, fever, and inflammation. Aspirin and its derivatives, commonly prescribed for alleviating oral and tooth pain, are responsible for the majority of chemical burn incidences when they are sucked, administered as a gel, mouthwash, powder, or as a tablet near to a sore tooth in an effort to relieve pain [73]. When aspirin is applied to the mucosa for a long time, it exerts a caustic impact. A cell-mediated response might result in an aphthous-like ulceration of the oral mucosal layer due to the acidic nature of acetylsalicylic acid (pH 3.5–5.0) [47]. Further, aspirin's organic and inorganic components adhere to the oral cavity epithelium, causing denaturation and coagulative necrosis [48].

#### 4.1.3 Calcium hydroxide

Calcium hydroxide ( $\text{Ca}(\text{OH})_2$ ) is frequently used in the field of endodontics due to its notable characteristics of mineralisation induction and promotion, antimicrobial capabilities, and necrotic material disintegration. Its adverse effects include cellular damage, epithelial damage, necrosis of bone, and cytotoxicity.

#### 4.1.4 Cocaine (benzoylmethylecgonine) ( $\text{C}_{17}\text{H}_{21}\text{NO}_4$ )

It is an alkaloid obtained from *Erythroxylum coca* leaves. Europe has the second-highest prevalence of cocaine use, with about 910,000 persons taking it. Cocaine's nonionized form diffuses across the lipid membranes of neurons. It transforms back into the active cationic form in the axoplasm, binds to the sodium channels, and stops the action potential from forming, producing a reversible anaesthetic effect [72].

#### 4.1.5 Denture cleansers

Patients clean their dentures with a variety of homemade and store-bought cleansers. Potassium monopersulfate, sodium perborate, sodium carbonate, surfactant, sodium bicarbonate, citric acid, and an additive are ingredients in immersion-type denture cleansers sold as tablets or powders. The perborate breaks down into an alkaline peroxide solution when the tablet or powder is dissolved in water, and it continues to break down to release oxygen. Debris is mechanically loosened by this process. The newly formed oxygen may interact with substances required for cell metabolism, interact with cell structures, or accelerate metabolism at the expense of cell growth [54].

#### 4.1.6 Eugenol

Eugenol has a scorching flavour, a strong carnation aroma and is a pale yellow liquid. It has been included in a number of products, including dental cement, endodontic sealants, impression pastes, and dressings for dry sockets. Unprocessed eugenol is combined with zinc oxide to create zinc oxide-eugenol, which demonstrates a



combination of physical and therapeutic qualities and can be used as a foundation material, root canal filler, and temporary restorative material. The tissue reaction caused by eugenol's byproducts might range from mild local allergic reactions to the uncommon catastrophic anaphylactic reactions [43, 44].

#### 4.1.7 Formocresol

Ever since Buckley introduced formocresol to dentistry in 1904, it has been frequently utilised in paediatric dentistry. In pulpotomy, formocresol is employed as a medication because of its ability to repair tissue when exposed to pulp. In the field, there has been a lot of concern stated and debated regarding the safety of using formocresol. There have been reports of widespread necrosis of soft tissues in the oral cavity due to improper formocresol use.

#### 4.1.8 Garlic burn

*Allium sativum*, also known as garlic, is regarded as a valuable herbal remedy and has been used for ages to treat a variety of illnesses. Additionally, studies have indicated that garlic has fibrinolytic, antihypertensive, and lipid-lowering properties. It has antiviral, antifungal, and antibacterial effects. Garlic's most frequent adverse reactions are nausea, diarrhoea, heartburn, and digestive distress. Rhinitis, asthma, anaphylaxis, contact dermatitis, and pemphigus are manifestations of garlic allergy. In 1987, Parish et al. reported the first instance of garlic burn. Garlic's precise ingredients that cause skin lesions are still a mystery. Allicin, diallyl disulfide, and allyl propyl disulfide are presumed to be the causes of chemical burn [56].

#### 4.1.9 Hydrogen peroxide

The method by which  $H_2O_2$  exerts its antimicrobial effects is owing to the release of nascent oxygen, which is harmful to anaerobes. The action of  $H_2O_2$  on bacterial cell wall debridement is the other antibacterial property mechanism [58].  $H_2O_2$  is used in mouthrinses (1–3%) and as a bleaching agent (3–5%) among other applications. Oral use of solutions containing 3 percent  $H_2O_2$  may result in nausea, minor mucosal irritability, and burns to the mouth, throat, oesophagus, and stomach. Ingestion of greater concentrations (>10 percent) can have more hazardous side effects, like burns to the gut mucosa and mucous membranes [59]. It is because it can directly react with proteins to create conjugates and reactive haptens that it triggers localised hypersensitivity reactions to oral mucosa, known as “contact stomatitis,” and to the dermis, known as “contact dermatitis,” in lower doses [43, 44].

#### 4.1.10 Sodium hypochlorite

NaOCl is a transparent, straw-coloured solution with 5% accessible chlorine in it. It creates chloramines upon ionisation, those are providing the antibacterial properties. The drawback of NaOCl is that if it is used outside of a root canal, it can lead to soft-tissue irritation and necrosis [40]. It reacts with the oral mucosa's proteins and lipids, which could cause subsequent infections [42]. NaOCl should be used as an irrigant in the root canal at a concentration of between 0.5 percent and 5.2 percent. Localised or widespread tissue necrosis has resulted from NaOCl's extension into the periradicular tissue. In the epidermis and subcutaneous tissues, a significant acute inflammatory

response causes rapid intraoral and extraoral tissue swelling. It causes acute sinusitis if it spreads into the maxillary sinus [65].

## 4.2 Clinical features

The degree of tissue damage, the causative agent's destructive qualities, and the method of application all affect how chemical burns look clinically [56]. Clinical lesions can range in severity from mild to severe depending on the substance used, pH level, chemical agent concentration, quantity used, method and length of tissue contact, depth of tissue penetration, and mechanism of action. Chemical burns on the mucosa manifest as diffuse erosive lesions that can range in severity from conventional desquamation to full mucosal detachment with penetration into the submucosa [50]. Chemical exposure causes changes in the vascularity, colour, texture, and consistency of the tissues. The general chemical burn appears as a shallow, wrinkled lesion that ranges in colour from white to yellow.

The characteristics of coagulative necrosis are seen through histopathological analysis [56]. Salivary gland duct involvement may result in temporary obstructive sialivary glanditis, but subsequent ductal opening scarring may result in chronic obstruction. Excision of the duct or gland may be necessary in cases of chronic sialadenitis [41]. Chemical burns frequently have a localised distribution and are not always restricted to the anatomic distribution of the masticatory mucosa [74].

### 4.2.1 Alendronate

Clinically, it may present as ulcers located on the palate, tongue, and lower lip. The ulcers cause intense pain [45].

### 4.2.2 Aspirin

It causes a localised white scurf with a reddened and thickened border [47, 73], while chromic acid produces a characteristic yellow lesion with a flat border [74].

### 4.2.3 Calcium hydroxide

Patients present with a swollen lip and mucosa, no history of pain and an extensive necrotic zone on gingiva with perforation [37].

### 4.2.4 Cocaine

Lesions develop at the site of application. A white slough, which could easily be removed, showing underlying ulceration and erythema seen on the gingiva. Patients may report painful, retracted gingiva [72].

### 4.2.5 Denture cleansers

Denture cleansers on chewing and swishing it around the mouth show burning sensation, swelling of the floor of the mouth and the salivary glands, inflammation of the soft palate and tongue. The orifices of the submandibular glands can also present with red and ulcerated with sloughing of the mucosa. There have been several reports of upper digestive tract injury associated with the accidental ingestion of denture

cleansers. Furthermore, it can depict as diffuse erosive lesions ranging from simple desquamation to complete obliteration of the oral mucosa with extension past the basement membrane into the submucosa. On ingestion, it also presented with perioral, glossal, and laryngeal edema and respiratory stridor leading to death. On autopsy, it showed extensive hemorrhagic bronchopneumonia and widespread gastrointestinal ulceration [54].

#### *4.2.6 Eugenol*

Eugenol burns usually presents with burning sensation and pain over the exposed area. Patient also complains of itching sensation. Intraoral examination may reveal allergic reaction “contact stomatitis” over the gingiva and adjacent mucosa [43].

#### *4.2.7 Formocresol*

Formocresol burns usually presents with pain and swelling on the exposed area. Extensive ulcerative lesion extending along exposed surface will appear like coagulative necrosis covered by slough. Patient also presents with symptoms of restricted mouth opening and reduced food intake [41].

#### *4.2.8 Garlic*

Garlic burns are clinically manifested as an area of slough and mucosal ulceration extending along the area of placement. The lesion may be painful on palpation [56].

#### *4.2.9 Hydrogen peroxide*

H<sub>2</sub>O<sub>2</sub> burns present as extensive areas of ulceration and erythema involving the alveolar mucosa and the marginal and attached gingival regions. Focal areas of ulceration and sloughing with necrosis of the surface layers of the epithelium may be seen [59].

#### *4.2.10 Sodium hypochlorite*

NaOCl burns manifest clinically as soft-tissue inflammation and necrosis if it is expressed outside the confines of root canal. The swelling may be edematous, hemorrhagic, or both and may extend beyond the region that might be expected with an acute infection of the affected tooth. The sudden onset of pain is a hallmark of tissue damage and may occur immediately or be delayed for several minutes or hours. Associated bruising and ecchymosis of adjacent tissues may occur due to bleeding into interstitial spaces [65].

#### *4.2.11 Tetracycline hydrochloride*

Tetracycline hydrochloride burns are manifested as loosely adherent yellowish white slough on gingiva. It may show erythematous margins, and patients complain of severe pain [38].

### 4.3 Treatment

A correct clinical history must be obtained in order to diagnose a chemical burn because the patient may not be aware of the relevance of these potentially harmful chemicals [41]. Early patient screening and prompt implementation of therapeutic measures will guarantee a speedy recovery and potentially stop additional mucosal damage [74].

The reason might be entirely eliminated by just getting rid of the agent permanently. Multiple irrigations with sterile water or betadine. If needed, analgesics, corticosteroids and benzocaine applied topically. Dietary supplements in the form of multivitamins would accelerate the recovery. When necessary, medications to stop secondary infections. Encourage the patient to follow a soft, chilled diet devoid of spices for a week. After a week, recall.

## 5. Occlusal trauma

The role of occlusion and how it interacts with the periodontium has been extensively researched. Occlusal trauma, also known as traumatism or trauma from occlusion (TFO), is an injury that causes tissue alterations inside the attachment apparatus as a result of occlusal force. Clinicians' knowledge of the impacts of damaging occlusal forces and the periodontium's adaptive, reparative, and destructive responses is mostly based on retrospective observations of human postmortem specimens and laboratory animal investigations. Unfortunately, there is a scarcity of scientific information from well-controlled prospective trials in people, making it difficult to comprehend this intricate host interaction [75].

### 5.1 Definitions

- Trauma from occlusion was defined by Stillman [76] as “a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position.”
- According to Carranza (AAP, 1986) [77], when occlusal forces exceed the adaptive capacity of the tissues, tissue injury results, the resultant injury is termed trauma from occlusion.
- WHO in 1978 defined trauma from occlusion as “damage in the periodontium caused by stress on the teeth produced directly or indirectly by teeth of the opposing jaw.”
- In the “Glossary of Periodontic Terms” (AAP 1986); occlusal trauma was defined as “An injury to the attachment apparatus as a result of excessive occlusal force”.

### 5.2 Glickman's concept

Glickman asserted that a plaque-associated gingivitis can be altered by abnormal occlusal forces [28]. That suggests that the Periodontal apparatus is gradually being affected by these forces. The response of a traumatised tooth with subgingival plaque

is different when compared to tissues around a tooth without deliterious forces. He suggested that there exists two distinct zones (**Figure 2**).

*Zone of irritation:* The interdental gingival papillae and marginal gingiva make up the zone of irritation, which is restricted by the gingival fibres. In this area, local irritations cause inflammation. The most harmful effects are epithelium ulceration, suppuration, and gingival connective tissue degeneration and necrosis. Even (horizontal) bone loss is could occur [78].

*Zone of co-destruction:* This contains the alveolar bone, the root cementum, and the periodontal ligament. Transseptal collagen fibre bundles (interdental and dentoalveolar) demarcate it coronally. The formation of an angular bony defect may result from the progression of inflammation from the zone of irritation directly into the periodontal ligament [79].

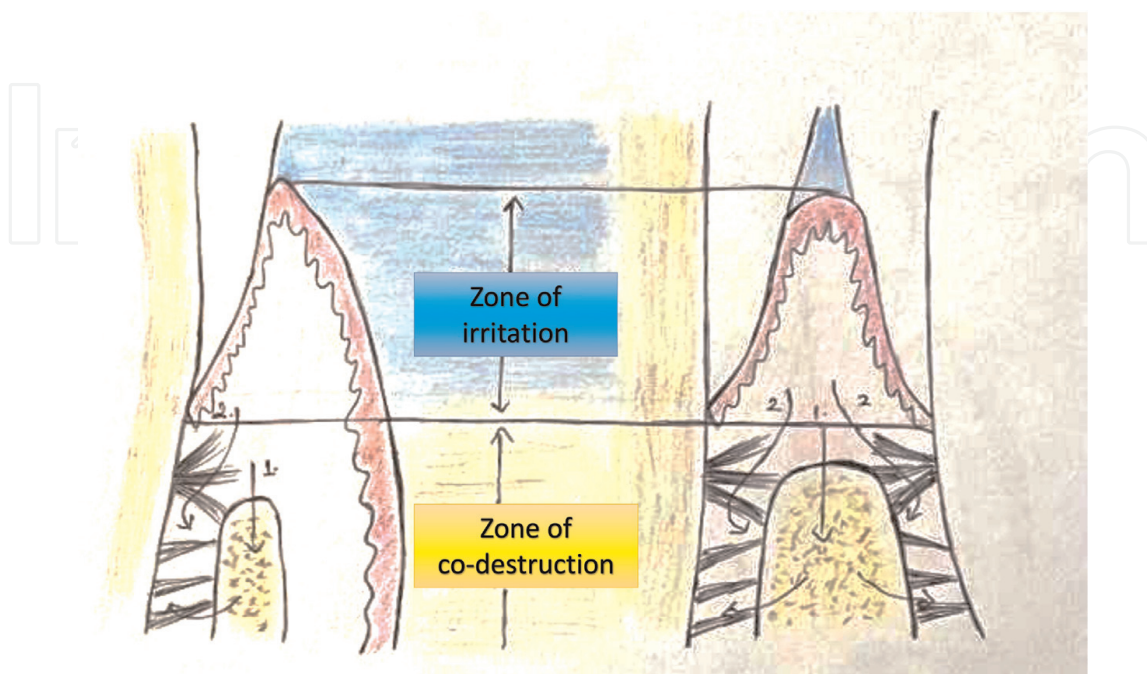
### 5.3 Adaptive capacity of the periodontium to occlusal forces

Adaptive capacity varies from person to person and from time to time within the same person. The size, direction, duration, and frequency of occlusal forces on the periodontium all determine their effect (**Figure 3**).

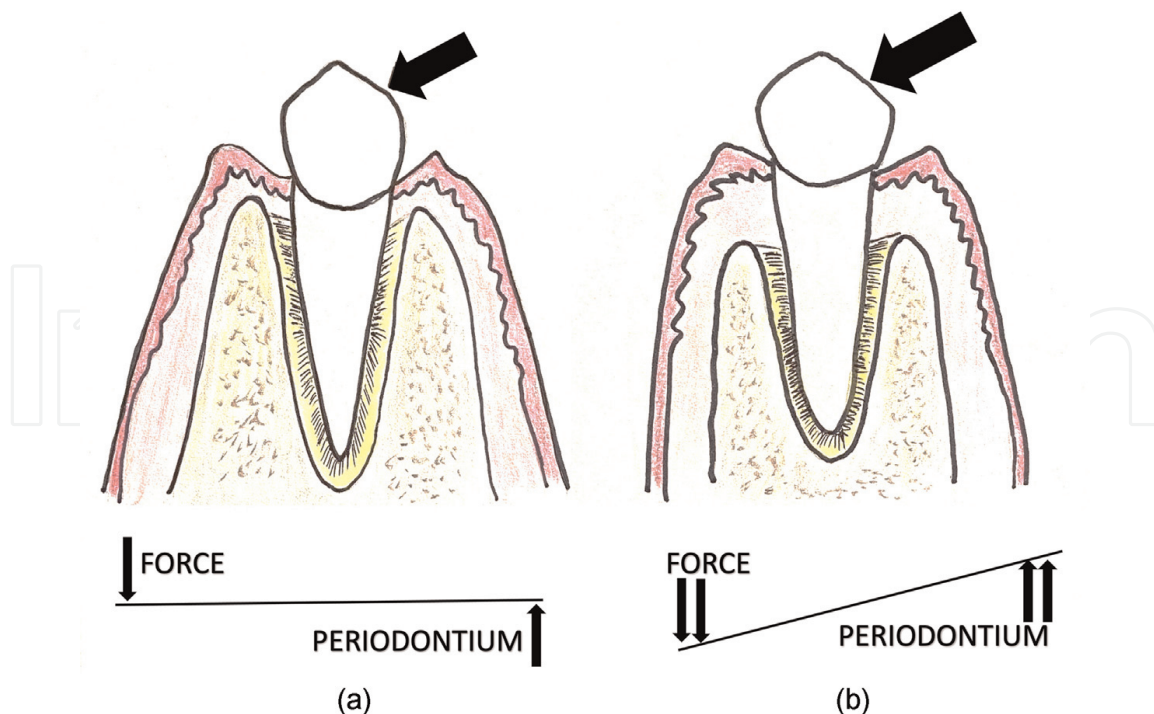
When occlusal stresses are raised in amplitude, the periodontium responds with

1. The periodontal ligament space is being widened.
2. The quantity and width of PDL fibres significantly increased.
3. An increase in the alveolar bone density [76].

The reorientation of stress and strains inside the periodontium is caused by changing the direction of occlusal pressures. The main fibres are positioned along the long



**Figure 2.** Spread of inflammation in TFO-Glickman Concept-zone of irritation, zone of co-destruction [28].



**Figure 3.**  
*Response to occlusal forces (a) normal periodontium can withstand normal occlusal forces, and (b) excessive occlusal forces causes damage to the periodontium [28].*

axis of the tooth to best tolerate occlusal stresses. The periodontium is more prone to be injured by lateral (horizontal) and torque (rotational) forces.

## 5.4 Trauma caused by occlusion

### 5.4.1 Acute vs chronic TFO

#### 5.4.1.1 Acute TFO

Acute TFO is a type of TFO that occurs suddenly. It is usually caused by

1. Biting down on a hard object causes an abrupt occlusal impact.
2. Prosthetic appliances that interfere with or change the direction of occlusal force are known as restorations [80].

*Characteristics:* Tooth pain, percussion sensitivity, and increased tooth movement.

*Consequence:* If the force is removed by a shift in the position of the tooth or by wearing away or correcting the restoration, the injury heals and the symptoms disappear if not, injury may worsen, leading to necrosis and the creation of a periodontal abscess. In some instances, it may persist as a symptomless chronic illness or end up in formation of cemental tears.

#### 5.4.1.2 Chronic

It's more common and has a higher clinical impact. Rather than developing as a result of acute TFO, it develops as a result of gradual alterations in occlusion caused by

tooth attrition combined with parafunctional habits like as bruxism and clenching [81].

*Characteristics:* Increasing mobility is one of the clinical characteristics.

#### 5.4.2 Primary vs secondary TFO

##### 5.4.2.1 Primary TFO

Occlusion trauma is the key etiologic cause in periodontal deterioration, and occlusion trauma is the only local modification to which a tooth is susceptible. Insertion of a “high filling” insertion of a prosthetic replacement that places excessive forces on the abutment and antagonist teeth drifting movement or extension of teeth into spaces created by unreplaced missing teeth drifting movement or extension of teeth into functionally unacceptable positions drifting movement or extension of teeth into spaces created by unreplaced missing teeth.

The amount of connective tissue attachment is not altered by original trauma, and pocket development is not initiated. This is likely because the supracrestal gingival fibres are unaffected, preventing the junctional epithelium from migrating apically [81].

##### 5.4.2.2 Secondary TFO

Occurs when bone loss caused by marginal inflammation impairs the tissues’ adaptive capacity to bear occlusal stresses. This changes the leverage on the remaining tissues by reducing the periodontal attachment area. The periodontium becomes more prone to injury, and occlusal forces that were previously tolerated become traumatic [82].

### 5.5 Tissue response to increased occlusal forces

#### 5.5.1 Effect on periodontal ligament

##### 5.5.1.1 Stage I: injury

Excessive occlusal forces result in tissue destruction. The periodontium is modified to buffer the impact of the offending force if it is chronic. The ligament widens at the expense of the bone, resulting in angular bone defects and loose teeth without periodontal pockets. The fulcrum or axis of rotation, which in single-rooted teeth is located in the junction of the middle third and the apical third of the clinical root, rotates under the stresses of occlusion. On opposite sides of the fulcrum, pressure and tension are created. These various lesions may coexist in the same region if jiggling pressures are applied.

Slightly too much pressure induces alveolar bone resorption, resulting in a widening of the periodontal ligament space. Blood vessels become more numerous and smaller in places with increased pressure.

Excessive tension promotes elongation of the PDL fibres and alveolar bone opposition. The size of blood vessels expands in locations of high strain.

Greater pressure caused a gradation of alterations in the periodontal ligament, beginning with the compression of the fibres, resulting in areas of hyalization. Following insult to the fibroblasts and other connective tissue cells, portions of the ligament undergo necrosis.

Within 30 minutes, blood vessels appear to be constricted and stagnant with erythrocytes, which begin to fragment at the end of 3 hours.

Disintegration of the blood vessel walls and discharge of the contents into the surrounding tissue occurs between 1 and 7 days.

Resorption of alveolar bone and root surface also occurs.

Severe strain causes periodontal ligament expansion, thrombosis, bleeding, ripping of the periodontal ligament, and alveolar bone resorption.

Necrosis of the periodontal ligament and bone occurs when there is enough pressure to drive the tooth against the bone. Undermining resorption occurs when bone is resorbed from viable periodontal ligament close to necrotic areas and from marrow gaps.

The furcations are the parts of the periodontium that are most vulnerable to harm from high occlusal stresses. When the periodontium is injured, there is a temporary decrease in mitotic activity and fibroblast proliferation, as well as in the creation of collages and bone. After the forces have dissipated, these restore to normal levels.(6).

#### 5.5.1.2 Stage 2

Repair TFO induces greater reparative activity in the typical periodontium, which is always occurring. In order to heal the injured periodontium, the damaged tissues are eliminated and new connective tissue cells and fibres, bone, and cementum are created. The body seeks to reinforce the thinned bony trabeculae with new bone when bone is damaged by high occlusal forces. This attempt to compensate for missing bone is known as buttressing bone growth, and it is a key part of the TFO recovery process. Within the jaw, buttressing bone production occurs when endosteal cells deposit new bone, which repairs the bony trabeculae and minimises the size of the marrow gaps. On the bone, buttressing bone development can also occur [77].

#### 5.5.1.3 Stage3: periodontal adaptive remodelling

If the repair process is unable to keep up with the occlusion's damage, the periodontium is reformed in an attempt to establish a structured relationship in which forces are no longer harmful to the tissues. This causes a thicker periodontal ligament with a funnel-shaped crest, as well as angular flaws in the bone and no pocket formation. The teeth that are implicated grow loose. There has also been evidence of increased vascularization [83].

Apart from occlusal forces, the forces such as orthodontic forces, jiggling forces etc. also play a role in remodelling (**Figures 4–7**).

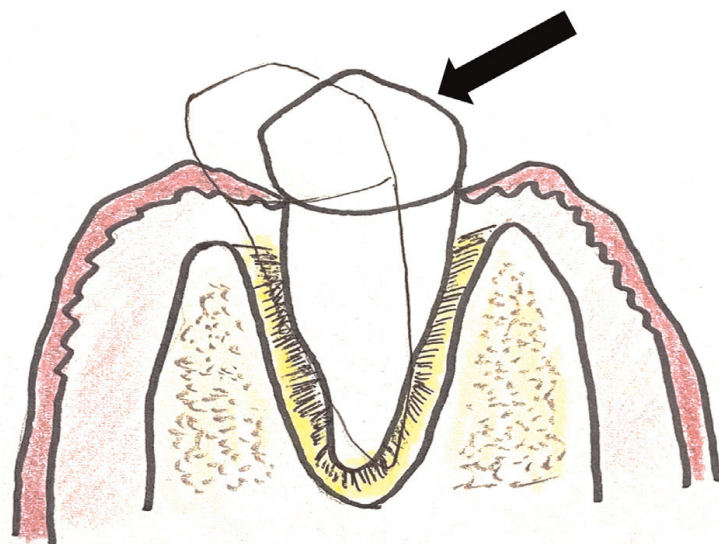
#### 5.5.2 Pathologic tooth destruction

Each tooth is expected to have a force threshold at which an occlusal traumatic lesion in the attachment apparatus occurs. This threshold force may be extremely strong, but it may be greater than a tooth's resistance to wear. Wear on the occlusal and incisal surfaces. As a result of the trauma, cartilage-like material might form in the periodontal ligament space. It has also been demonstrated that erythrocytes can form crystals [84].

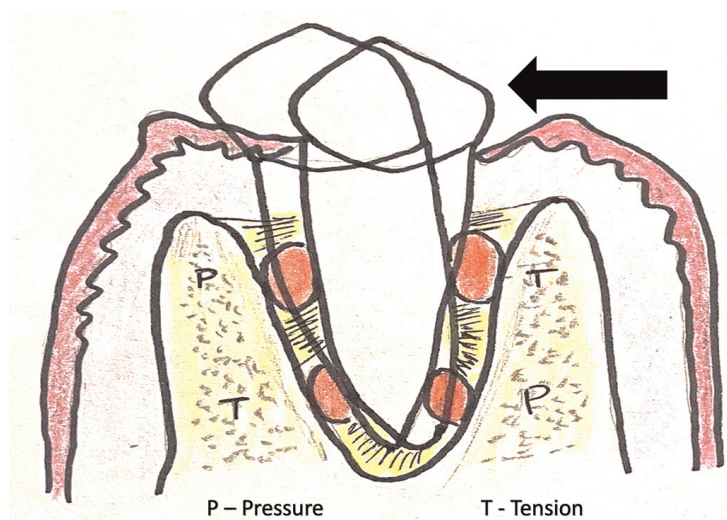
#### 5.5.3 Soft-tissue effects

Because occlusal forces only affect the attachment apparatus of the periodontium, they have no effect on the supracrestal soft tissues of the periodontium. Excessive





**Figure 4.** Physiological response, intra-socket adaptation of the tooth to the normal occlusal forces [28].



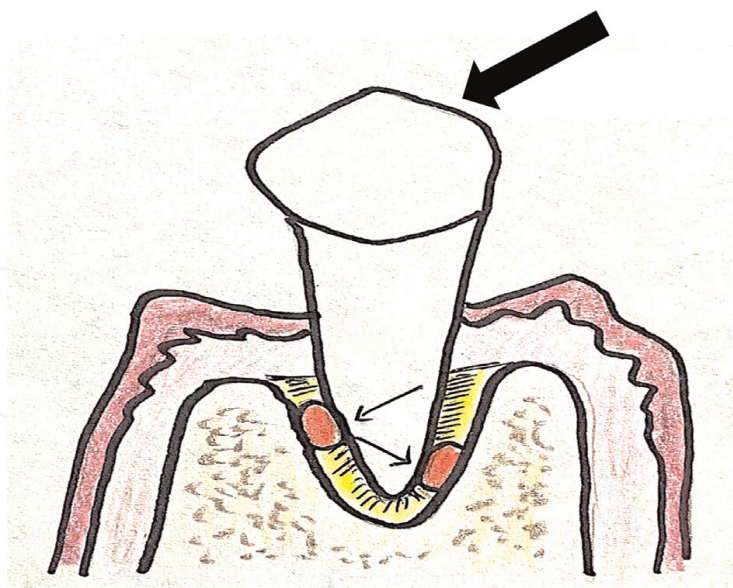
**Figure 5.** Response to orthodontic forces—results in areas of pressure and tension [28].

force on a tooth does not harm the attachment of gingival soft tissues coronal to the bone (the C.T. attachment and JE). Pocketing and recession are marginal disease entities (occlusal trauma is not a marginal illness) that are initiated at the gingival margin by plaque-related pathosis [84]. As a result of the trauma, cartilage-like material might form in the periodontal ligament space. It has also been demonstrated that erythrocytes can form crystals.

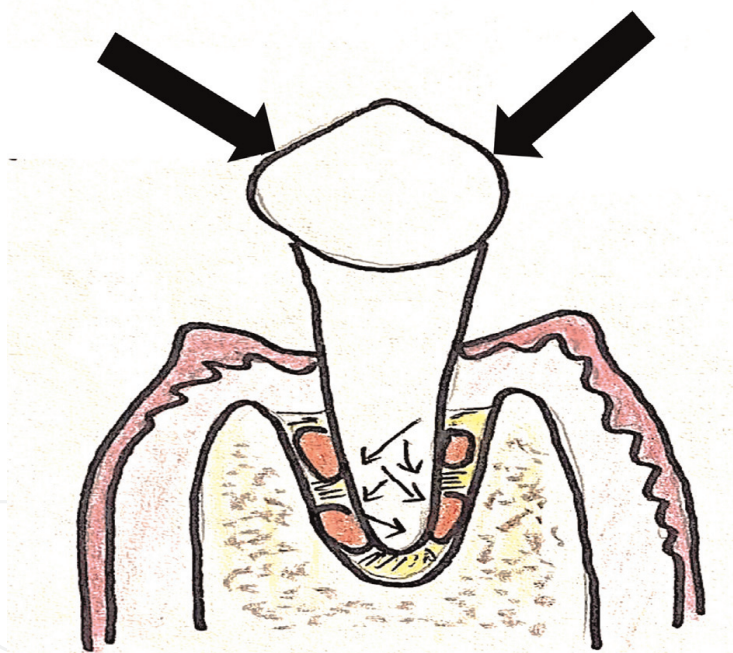
## 5.6 Clinical and radiographic

### 5.6.1 Signs

Increased tooth mobility is the most prevalent clinical symptom of periodontal damage. Destruction of PDL fibres increases mobility in the early stages.



**Figure 6.**  
*Response to excessive occlusal forces by a compromised periodontium results in tooth mobility [28].*



**Figure 7.**  
*Periodontal response to jiggling forces results in multiple areas of pressure and tension causing extensive tissue damage [28].*

Accommodation of PDL to force widening of PDL to promote mobility is not pathologic in the ultimate stage. It becomes pathogenic when it gets worse over time [85].

### 5.6.2 Radiographic features

The following radiographic features of TFO may be seen:

1. Wider periodontal space, often accompanied with thickening of the lamina dura along the lateral face of the root, in the apical region, and at bifurcation points.

These changes may be due to thickening and strengthening of the periodontal ligament and alveolar bone as a positive reaction to increasing occlusal stresses, and so are not necessarily detrimental.

2. Interdental septum damage that is “vertical” rather than “horizontal.”

3. Alveolar bone radiolucence and condensation.

4. Resorption of the roots.

### **5.7 Traumatic lesions can be reversed**

Occlusion-related trauma is reversible. When the artificially induced force is removed from experimental animals, the tissues begin to heal. TFO, on the other hand, does not always fix itself (by going away or intruding), hence it is not always transitory or of minor clinical relevance. For repair to take place, the injurious force must be alleviated. Periodontal injury persists or worsens in people if the teeth are unable to escape or adapt to severe occlusal force. Inflammation may make this reversibility more difficult [86].

Occlusal trauma cannot be identified without a block section biopsy since TFO is characterised and diagnosed based on histologic abnormalities in the periodontal supporting system. Because this is manifestly unfeasible in periodontics therapy, the doctor must rely on clinical symptoms of possible occlusal trauma. These are some of the indicators:

1. Fremitus

2. Flexibility (progressive)

3. Occlusal inconsistencies

4. Additional signs such as wear facets.

5. Migration of teeth

6. Broken tooth/teeth

7. Sensitivity to heat

The method in which the teeth make contact can also be used to assess the occlusal relationship of the teeth. This is accomplished by gradually retruding the patient’s jaw (retruded occlusion/centric relation) and softly closing the patient’s mouth, until the first tooth-to-tooth contact is made. After that, the patient is asked to close their mouth to a comfortable intercusped position (central occlusion/habitual occlusion). The centric relation to centric occlusion slide (or CR/CO shift) is the distance the patient moves from the retruded position to the greatest intercuspatation. It is possible to capture both the initial contact point and the approximate amount of slide. Tooth interactions are also documented in eccentric jaw positions (i.e. lateral and protrusive jaw positions). Contacts are frequently noticed and may also be

documented with a thin inked silk ribbon or Mylar film to record tooth contact during an examination.

## 5.8 Occlusal therapy

After nonsurgical treatment is completed, occlusal treatment is frequently carried out. When the periodontal supporting structures become inflamed, the teeth's mobility often increases. The periodontal ligament may be inflamed, resulting in more movement. When inflammation is under control, teeth are less dynamic, resulting in a more stable occlusal association after occlusal therapy. To make the patient more comfortable, occlusal treatment may be recommended as the first stage of periodontal therapy in these circumstances [87]. If occlusal therapy is started before inflammation is controlled, it will almost certainly be essential to do additional occlusal treatment once the inflammation has been controlled. Prior to starting treatment, the patient should be advised about this.

There are two primary techniques to occlusal therapy.

(1) Using a bite device (bite guard) and/or (2) Altering the occlusal connections between the teeth to adjust the occlusion.

Orthodontic therapy or selective occlusal surface grinding can permanently alter the relationship between teeth.

Coronoplasty/Selective grinding- Selective grinding is a process that involves modifying the occlusal surfaces of teeth to improve the overall contact pattern. Tooth structure is eliminated selectively until the reshaped teeth make contact in a way that achieves the treatment goals [88].

The following are some of the goals of coronoplasty that have been met as a consequence of occlusal adjustment:

1. Afferent impulse pattern and intensity change.
2. Excessive tooth movement is reduced.
3. To provide occlusal stabilisation, multiple simultaneous contacts are dispersed across the occlusal scheme.
4. A beneficial adjustment in chewing or swallowing habits.
5. Mandibular movement patterns in multiple directions.
6. Occlusal stresses on implants are verticalized.

The occlusion is generally modified once gingival inflammation and periodontal pockets have been removed in treatment planning for the following reasons:

1. Evidence of aetiology and healing features suggests that coronoplasty effects are incomplete unless inflammation is addressed initially.
2. When the inflammation is gone, the teeth frequently shift back to their original place. As a result, if the occlusion is modified before the inflammation has subsided, it may need to be corrected again once gingival health has been restored.

This treatment sequence is altered if the following conditions are met:

- a. In infrabony pockets, where excessive occlusal stresses are vital in deciding the pattern of osseous defects, the occlusion is altered to give best conditions for development of bony flaws.
- b. Bacterial plaque is often thought to be the cause of periodontal disease. It's possible that a localised mechanism interacts with bacterial plaque when high occlusal stresses are applied. It's also possible that high occlusal forces produce an environment in which bacterial plaque's harmful effects are amplified, or that there's a whole new process at work that has not been discovered yet. Treatment of the occlusion to reduce occlusal interferences, in combination with other modalities of periodontal treatment, may, however, have a positive impact on the progression and treatment of periodontal deterioration. Excessive occlusal forces is a risk factor that can be reduced using current clinical tools, as can any risk factors that can be reduced using current clinical tools.

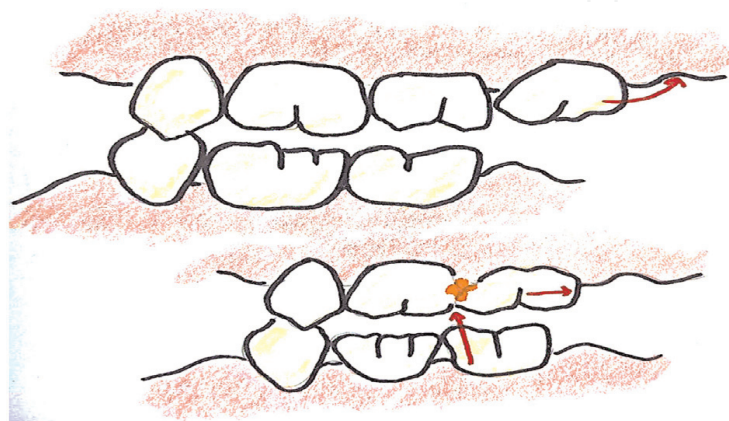
## 6. Food impaction

Food impaction is the forceful wedging of food into the tooth supporting tissues by occlusal force. It may occur interdentally or in the buccal and palatal surfaces. It is perceived to be the most common cause of gingival and periodontal inflammation. If left unidentified it could alter the pathogenesis and leads to adverse therapeutic outcomes [89]. Food is typically prevented from being forced into the mouth by the integrity and placement of the proximal contact, the contour of the marginal ridge and developing grooves, and the contour of the lingual and facial surfaces (**Figure 8**).

### 6.1 Types of food impaction

#### 6.1.1 Vertical impaction

- A. Open contacts
- B. Irregular marginal ridge



**Figure 8.** Food impaction—wedging of food into the deeper tissues by plunger cusp [89].

C. Plunger cusp: Cusp that tend to forcibly wedge food interproximal region of opposing teeth.

### *6.1.2 Horizontal/lateral food impaction*

Periodontal disease/gingival recession results frequently in enlargement of gingival tissues in the interdental embrasure area which is subjected to forces from the lips, cheek and tongue resulting in food lodgment in the proximal areas.

## **6.2 Management**

### *6.2.1 Periodontal treatment*

Curettage, interproximal brushing, flossing, and scaling.

### *6.2.2 Occlusal adjustment*

Plunger cusp—Go around these angular plunger cusp peaks. The palatal cusp of maxillary teeth, the buccal cusp of mandibular teeth, and occasionally the palatal incline of the maxillary buccal cusp and the buccal incline of the lingual cusp are the functional cusps that make up these plunger teeth. Additional advantages could result from reviewing the study cast to assess the relationship between the lingual cusps. Equalise the marginal ridges' occlusal heights.

## **7. Iatrogenic injury**

In 1912, Black recognised the strong connection between iatrogenic causes and periodontal degeneration [90]. It has been well established for many years that dental restorations and periodontal health are related. The position of the restoration in relation to the gingival margin, the presence of overhangs, the presence of marginal leakage, the roughness of the surfaces, and the type of restorative material are a few examples of the various aspects of the periodontal-restorative interaction that have received attention in numerous studies [91]. The most consistent way that dental restorations can harm marginal health is by increasing plaque formation, but overhanging metal restorations have also been linked to changes in the subgingival microbiota, including an increase in potential periodontal pathogenic microflora [92].

The areas of restorative dentistry and periodontics that overlap are the marginal periodontium. In contrast, special attention should be paid to how the periodontium reacts to the irritants caused by negligent procedures, which might start or exacerbate gingival inflammation already present. If the illness is not identified and treated in its early stages, loss of periodontal support and subsequent tooth loss may follow. Particularly when they are subgingivally positioned, dental restorations or appliances are usually linked to the development of gingival inflammation. This may be true for orthodontic bands, crowns placed onlays, fillings, and subgingivally. By being positioned deep inside the sulcus or within the junctional epithelium, restorations may have an impact on the biologic width. With apical migration of the junctional epithelium and re-establishment of the attachment apparatus at a higher apical level, this may encourage inflammation and loss of clinical attachment.

## **7.1 Margins of restorations**

The following characteristics of dental restorations and detachable partial dentures are important for maintaining periodontal health:

1. Where the gingival margin should be placed for the restoration
2. The distance between the restoration's edge and the unprepared tooth
3. The design of restorations
4. The occlusion
5. The restoration's components
6. The practical restoration process
7. The removable partial denture's design

When positioning the restorative margins, especially in the aesthetic zone where the major treatment objective is to cover the junction of the margin with the tooth, the biologic width information should be used. The placement of the restoration margin depends greatly on:

1. Aesthetics.
2. The need for extra restorative retention.
3. The level of oral hygiene.
4. The person's vulnerability to root caries.
5. The marginal gingiva's susceptibility to irritants.
6. The marginal gingiva's morphological features.
7. The severity of gingival recession.
8. Significant cervical abrasion.
9. Dental restorations with overhanging margins.

Overhanging dental restoration margins increase the severity of periodontal disease by changing the gingival sulcus' ecological balance to one that favours the growth of disease-associated organisms (mostly gram-negative anaerobic species) at the expense of healthy organisms (primarily gram-positive facultative species) [93] and by preventing the patient's access to remove accumulated plaque.

## 7.2 Placing the matrix/rubber dam

After cavity preparation, a correctly planned and contoured matrix needs to be implanted so that it may be accurately adjusted to the margins without harming the biologic width. For class II restorations, extra interdental wedge placements are necessary for a well-contoured restoration, but they must be done carefully. To repeat proper form and avoid intracrevicular overhangs, the matrix should be stiff and well-contoured (**Figure 9**).

## 7.3 Hypersensitivity to dental materials

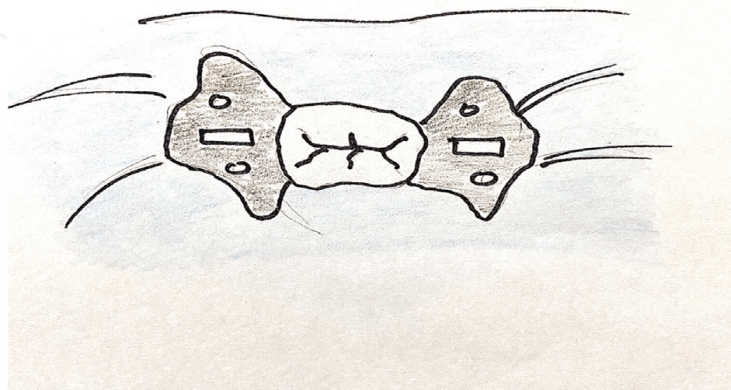
Nonprecious alloys used in dental restorations have been linked to inflammatory gingival reactions, according to reports [94]. Although the frequency of these events is debatable [95], the reactions have typically been to alloys containing nickel. Rarely do precious alloys cause hypersensitivity reactions, and these alloys offer a simple fix for the issues with nonprecious alloys.

## 7.4 Marginal fit

Marginal fit has unmistakably been linked to the periodontium's inflammatory response. According to research, the degree of gingival inflammation might rise in direct proportion to the degree of marginal opening [96]. Significantly open margins (more than a few tenths of a millimetre) are capable of hosting huge numbers of bacteria and may be the cause of the observed inflammatory response. However, the periodontium is significantly more affected by the quality of the marginal finish and the location of the margin in relation to the attachment than by the distinction between a 20-m fit and a 100-m fit [97].

## 7.5 Crown contour

The preservation of periodontal health has been said to depend heavily on restoration contours [98]. Access for hygiene is made possible by proper contour, which also



**Figure 9.**  
*Improper rubber dam placement can cause injury to the periodontal tissues.*



has the capabilities to create the ideal gingival form and has a pleasant visible tooth contour in places that matter aesthetically.

Studies on both humans and animals conclusively show a link between gingival inflammation and over-contouring, whereas under-contouring has no negative effects on periodontal health [99]. Inadequate tooth preparation by the dentist is the most frequent cause of over-contoured restorations, which compels the technician to create a bulky restoration to make place for the restorative material. A flatter contour is always acceptable in parts of the mouth where aesthetic concerns are not important.

## **8. Discussion**

There is a potential risk that the periodontium's soft tissues will sustain accidental, intentional, or fictitious trauma, which can have a negative impact on periodontal health. Also, studies on animals and humans have suggested a connection between periodontal disease progression and occlusal trauma/occlusal differences [100]. However, all researchers came to the consensus that high occlusal forces do not cause plaque-induced periodontal disease or loss of periodontal attachment, and more recent research confirms this [101]. The treatment strategy for traumatic dental injuries differs depending on the amount of damage to the teeth and supporting tissues. It should be remembered that the treatment strategy must be properly developed in this situation. By doing so, additional traumas that might exacerbate the prognosis—which is not always favourable—would be avoided. Because it is impossible to determine the exact extent of damage to the tooth and supporting structures, follow-up after any form of trauma is crucial [102].

## **9. Conclusion**

In order to maintain the general homeostasis of the dentition, the periodontium, which includes the cementum and alveolar bone as well as the soft tissues gingiva and periodontal ligament, must be in good health. The harmony of the periodontal attachment system is affected by injury to the periodontal tissues in any form, including microbiological, physical, thermal, chemical, mechanical, occlusal, and habitual injury, which changes how the dentition functions as a whole. Prior to receiving any other dental treatment, the periodontium must first be restored which comprehends that gingival irritants are eliminated, functional and occlusal interferences are fixed, morphologic and pathologic gingival problems are treated, and bone abnormalities of the supporting periodontium are corrected.

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## References

- [1] Gottrup F, Andreasen JO. Wound Healing Subsequent to Injury. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 4th ed. Oxford: Blackwell; 2007. pp. 1-61
- [2] Andreasen JO, Andreasen FM, Andersson L, editors. Textbook and Color Atlas of Traumatic Injuries to the Teeth. John Wiley & Sons; 2018
- [3] Andreasen JO, Andreasen FM. Classification, etiology and epidemiology. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 1994; 3:151-179
- [4] Glendor U, Marcenes W, Andreasen JO. Classification, epidemiology and etiology. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 2007;4: 217-254
- [5] Thennukonda RA, Natarajan BR. Adverse events associated with ultrasonic scalers: A manufacturer and user facility device experience database analysis. Indian Journal of Dental Research. 2015;26(6):598
- [6] Iyama K, Ueki T, Yamano S, Tajima G, Inokuma T, Hirao T, et al. An adult case of laryngopharyngeal burn by drinking hot water. Acute Medicine & Surgery. 2017;4(2):198-201
- [7] Kwon SJ, Park YJ, Jun SH, Ahn JS, Lee IB, Cho BH, et al. Thermal irritation of teeth during dental treatment procedures. Restorative Dentistry & Endodontics. 2013;38(3):105-112
- [8] Ustun K, Sari Z, Orucoglu H, Duran I, Hakki SS. Severe gingival recession caused by traumatic occlusion and mucogingival stress: A case report. European Journal of Dentistry. 2008;2(02):127-133
- [9] Gupta ND, Maheshwari S, Prabhat KC, Goyal L. A critical review of the management of deep overbite complicated by periodontal diseases. European Journal of General Dentistry. 2012;1(01):2-5
- [10] Meeran NA. Iatrogenic possibilities of orthodontic treatment and modalities of prevention. Journal of Orthodontic Science. 2013;2(3):73
- [11] Safa'a AA, Fadel EI. Hazards of prosthodontic devices and materials. Tanta Dental Journal. 2017;14(1):7
- [12] Dilsiz A, Aydin T. Self-inflicted gingival injury due to habitual fingernail scratching: A case report with a 1-year follow up. European Journal of Dentistry. 2009;3(02):150-154
- [13] Chalkoo AH, Peerzada GY, Makroo NN. Case report of gingivitis artefacta (self-injurious behavior). Journal of Indian Academy of Oral Medicine and Radiology. 2016;28(3):317
- [14] Mazumdar U, Mazumdar B, Rajkumar IR. Gingivitis artefacta-self injurious behaviour in adult patients of depression report of 2 cases. Journal of Orofacial Research. 2011;1(1):45-48
- [15] Stewart DJ, Kernohan DC. Self-inflicted gingival injuries. Gingivitis artefacta, factitial gingivitis. The Dental Practitioner and Dental Record. 1972;22(11):418-426
- [16] Khalia N, Vemanaradhya GG, Mehta DS. Oral piercing: Style statement or a state of disharmony? International Journal of Oral Health Sciences. 2013;3(2):84
- [17] Hennequin-Hoenderdos NL, Slot DE, Van der Weijden GA. The

prevalence of oral and peri-oral piercings in young adults: A systematic review. *International Journal of Dental Hygiene*. 2012;**10**(3):223-228

[18] Bone A, Ncube F, Nichols T, Noah ND. Body piercing in England: A survey of piercing at sites other than earlobe. *BMJ*. 2008;**336**(7658):1426-1428

[19] Hickey BM, Schoch EA, Bigeard L, Musset AM. Complications following oral piercing. A study among 201 young adults in Strasbourg, France. *Community Dental Health*. 2010;**27**(1): 35-40

[20] Gill JB, Karp JM, Kopycka-Kedzierawski DT. Oral piercing injuries treated in United States emergency departments, 2002–2008. *Pediatric Dentistry*. 2012;**34**(1):56-60

[21] Mishra P, Gupta M, Shrivastava K, Singh N, Singh P. Oral tattoos: “beauty lies in the eyes of the beholder”. *Advances in Human Biology*. 2015;**5**(2): 17-21

[22] Bathla S. *Periodontics Revisited*. India: JP Medical Ltd; 2011

[23] Odai ED, Ehizele AO. Trauma to the soft tissues of the periodontium: A review. *Nigerian Journal of Dental and Maxillofacial Traumatology*. 2019;**2**(1&2):7-14

[24] Griffin TJ, Cheung WS, Zavras AI, Damoulis PD. Postoperative complications following gingival augmentation procedures. *Journal of Periodontology*. 2006;**77**(12):2070-2079

[25] Girish MS, Anandakrishna L, Chandra P, Nandlal B, Srilatha KT. Iatrogenic injury of oral mucosa due to chemicals: A case report of formocresol injury and review. *IOSR Journal of Dental and Medical Sciences*. 2015;**14**:1-5

[26] Gonçalves PS, Mendez DA, Santos PS, Damante JH, Rios D, Cruvinel T. The diagnosis and treatment of multiple factitious oral ulcers in a 6-year-old boy. *Case Reports in Dentistry*. 2017;**2017**

[27] Mcculloch CA, Lekic P, Mckee MD. Role of physical forces in regulating the form and function of the periodontal ligament. *Periodontology* 2000. 2000;**24**(1):56-72

[28] Lindhe J, Lang NP, Karring T. *Clinical Periodontology and Implant Dentistry*. 5th ed. Pan America, Mexico: Wiley-Blackwell; 2012. pp. 3-49

[29] Goswami M, Chaitra TR, Chaudhary S, Manuja N, Sinha A. Strategies for periodontal ligament cell viability: An overview. *Journal of Conservative Dentistry: JCD*. 2011;**14**(3):215

[30] Pedrini D, Panzarini SR, Poi WR, Sundefeld ML, Tiveron AR. Dentists' level of knowledge of the treatment plans for periodontal ligament injuries after dentoalveolar trauma. *Brazilian Oral Research*. 2011;**25**:307-313

[31] Trope M. Clinical management of the avulsed tooth. *Dental Clinics of North America*. 1995;**39**(1):93-112

[32] Kocak FZ, Talari AC, Yar M, Rehman IU. In-situ forming pH and thermosensitive injectable hydrogels to stimulate angiogenesis: Potential candidates for fast bone regeneration applications. *International Journal of Molecular Sciences*. 2020;**21**(5):1633

[33] Gluskin AH, Ruddle CJ, Zinman EJ. Thermal injury through intraradicular heat transfer using ultrasonic devices: Precautions and practical preventive strategies. *The Journal of the American Dental Association*. 2005;**136**(9):1286-1293

- [34] Maron FS. Mucosal burn resulting from chewable aspirin: Report of case. *Journal of the American Dental Association* (1939). 1989;**119**(2):279-280
- [35] Santos-Pinto L, Campos JA, Giro EM, Cordeiro R. Iatrogenic chemical burns caused by chemical agents used in dental pulp therapy. *Burns: Journal of the International Society for Burn Injuries*. 2004;**30**(6):614-615
- [36] De Bruyne MA, De Moor RJ, Raes FM. Necrosis of the gingiva caused by calcium hydroxide: A case report. *International Endodontic Journal*. 2000;**33**(1):67-71
- [37] Murrin JR, Abrams H, Barkmeier WW. Chemical burn of oral tissue caused by dental cavity varnish. Report of a case. *Illinois Dental Journal*. 1978;**47**(10):580-581
- [38] Dayakar MM, Pai PG, Sooranagi RP, Vijayan V, Waheed A. Chemical burns of gingiva and its management. *SRM Journal of Research in Dental Sciences*. 2018;**9**(4):174
- [39] Harris JH. Soft-tissue burns from a dentin bonding agent. *General Dentistry*. 1991;**39**(5):350-351
- [40] Loe H. Reaction of marginal periodontal tissues to restorative procedures. *International Dental Journal*. 1968;**18**:759-778
- [41] Ozcelik O, Haytac MC, Akkaya M. Iatrogenic trauma to oral tissues. *Journal of Periodontology*. Oct 2005;**76**(10):1793-1797
- [42] Pindborg JJ. *Atlas of Diseases of the Oral Mucosa*. Copenhagen: Munksgaard; 1980
- [43] Deshpande A, Verma S, Macwan C. Allergic reaction associated with the use of eugenol containing dental cement in a young child. *Austin Journal of Dental*. 2014;**1**(2):1-3
- [44] Sarrami N, Pemberton MN, Thornhill MH, Theaker ED. Adverse reactions associated with the use of eugenol in dentistry. *British Dental Journal*. 2002;**193**(5):257-259
- [45] Gonzalez-Moles MA, Bagan-Sebastian JV. Alendronate-related oral mucosa ulcerations. *Journal of Oral Pathology & Medicine*. 2000;**29**(10):514-518
- [46] Krasagakis K, Krüger-Krasagakis S, Ioannidou D, Tosca A. Chronic erosive and ulcerative oral lesions caused by incorrect administration of alendronate. *Journal of the American Academy of Dermatology*. 2004;**50**(4):651-652
- [47] Dellinger TM, Livingston HM. Aspirin burn of the oral cavity. *Annals of Pharmacotherapy*. 1998;**32**(10):1107
- [48] Ramalingam K, Abdalla KA. Traumatic chemical oral ulceration—Aspirin burn—A case report from Libya. *CIBTech Journal of Surgery*. 2016;**5**(2):2319-3875
- [49] Neville BW, Damm DD, Allen CM, Bouquot JE. *Oral and Maxillofacial Pathology*. 2nd ed. Philadelphia, PA: Saunders; 2002. pp. 337-369
- [50] Dayakar MM, Pai PG, Madhavan SS. “Tetracycline hydrochloride chemical burn” as self-inflicted mucogingival injury: A rare case report. *Journal of Indian Society of Periodontology*. 2012;**16**(2):282
- [51] Sánchez AR, Rogers RS III, Sheridan PJ. Tetracycline and other tetracycline-derivative staining of the teeth and oral

- cavity. *International Journal of Dermatology*. 2004;**43**(10):709-715
- [52] Kapoor A, Malhotra R, Grover V, Grover D. Systemic antibiotic therapy in periodontics. *Dental Research Journal*. 2012;**9**(5):505
- [53] Baruchin AM, Lustig JP, Nahlieli O, Nader A. Burns of the oral mucosa: Report of 6 cases. *Journal of Cranio-Maxillofacial Surgery*. 1991;**19**(2):94-96
- [54] Murdoch-Kinch CA, Mallatt ME, Miles DA. Oral mucosal injury caused by denture cleanser tablets: A case report. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics*. 1995;**80**(6):756-758
- [55] Bundgaard T. Chemical burns in the oral cavity and esophagus after accidental intake of a Steradent denture-cleansing tablet. *Tandlaegebladet*. 1985;**89**(19):711-713
- [56] Bagga S, Thomas BS, Bhat KM. Garlic burn as self-inflicted mucosal injury—A case report and review of the literature. *Quintessence International*. 2008;**39**(6)
- [57] Isenberg SR, Hier LA, Chauvin PJ. Chemical burns of the oral mucosa: Report of a case. *Journal (Canadian Dental Association)*. 1996;**62**(3):262-264
- [58] Putt MS, Proskin HM. Custom tray application of peroxide gel as an adjunct to scaling and root planing in the treatment of periodontitis: Results of a randomized controlled trial after six months. *The Journal of Clinical Dentistry*. 2013;**24**(3):100-107
- [59] Shetty K. Hydrogen peroxide burn of the oral mucosa. *The Annals of Pharmacotherapy*. 2006;**40**(2)
- [60] Moghadam BK, Gier R, Thurlow T. Extensive oral mucosal ulcerations caused by misuse of a commercial mouthwash. *Cutis*. 1999;**64**(2):131-134
- [61] Bernstein ML. Oral mucosal white lesions associated with excessive use of Listerine mouthwash: Report of two cases. *Oral Surgery, Oral Medicine, Oral Pathology*. 1978;**46**(6):781-785
- [62] Flötra L, Gjermo PE, Rölla G, Waerhaug J. Side effects of chlorhexidine mouth washes. *European Journal of Oral Sciences*. 1971;**79**(2):119-125
- [63] Gagari E, Kabani S. Adverse effects of mouthwash use. A review. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics*. 1995;**80**(4):432-439
- [64] Kowitz GM, Lucatorto FM, Cherrick HM. Effects of mouthwashes on the oral soft tissues. *Journal of Oral Medicine*. 1976;**31**(2):47-50
- [65] Singhal A, Vinaya V, Ahuja T, Longani P. Sodium hypochlorite: Complications and management. *Journal of Dental Sciences and Oral Rehabilitation*. 2013;**4**(1):7-10
- [66] Mathew ST. Risks and management of sodium hypochlorite in endodontics. *Journal of Oral Hygiene & Health*. 2015;**3**(3):1-5
- [67] Parry J, Porter S, Scully C, Flint S, Parry MG. Mucosal lesions due to oral cocaine use. *British Dental Journal*. 1996;**180**(12):462-464
- [68] Gandara-Rey JM, Diniz-Freitas M, Gandara-Vila P, Blanco-Carrion A, Garcia-Garcia A. Lesions of the oral mucosa in cocaine users who apply the drug topically. *Medicina Oral: Organo Oficial de la Sociedad Espanola de Medicina Oral y de la Academia Iberoamericana de Patologia y Medicina Bucal*. 2002;**7**(2):103-107

- [69] Blanksma CJ, Brand HS. Cocaine abuse: Orofacial manifestations and implications for dental treatment. *International Dental Journal*. 2005;**55**(6): 365-369
- [70] Kapila YL, Kashani H. Cocaine-associated rapid gingival recession and dental erosion. A case report. *Journal of Periodontology*. 1997;**68**(5):485-488
- [71] Brazier WJ, Dhariwal DK, Patton DW, Bishop K. Ecstasy related periodontitis and mucosal ulceration—a case report. *British Dental Journal*. 2003; **194**(4):197-199
- [72] Brand HS, Gonggrijp S, Blanksma CJ. Cocaine and oral health. *British Dental Journal*. 2008;**204**(7):365-369
- [73] Gilvetti C, Porter SR, Fedele S. Traumatic chemical oral ulceration: A case report and review of the literature. *British Dental Journal*. 2010;**208**(7): 297-300
- [74] Touyz LZ, Hille JJ. A fruit—Mouthwash chemical burn: Report of a case. *Oral Surgery, Oral Medicine, Oral Pathology*. 1984;**58**(3):290-292
- [75] Kar KM. Beobachtungen uber Pyorrhoea alveolaris. *Osterreichisch-Ungarische Vierteljahresschrift fur Zahnheilkunde*. 1901;**17**:279
- [76] Stillman PR. Early clinical evidence of disease in the gingiva and pericementum. *Journal of Dental Research*. Dec 1921;**3**(3):25-31
- [77] Pihlstrom BL, Anderson KA, Aepli D, Schaffer EM. Association between signs of trauma from occlusion and periodontitis. *Journal of Periodontology*. 1986;**57**:1-6
- [78] Glickman I, Smulow J. The combined effects of inflammation and trauma from occlusion in periodontics. *International Dental Journal*. 1969;**19**: 393
- [79] Glickman I, Sumlow JB. Effect of excessive occlusal forces upon the pathway of gingival inflammation in human. *Journal of Periodontology*. 1965; **36**:141-147
- [80] Stillman PR. What is traumatic occlusion and how can it be diagnosed and corrected. *Journal of the American Dental Association (1939)*. 1925;**12**:1330-1338
- [81] American Academy of Periodontology. Glossary of Periodontal Terms. 2001. Available from: <https://www.perio.org>
- [82] Reinhardt RA, Pao YC, Krejci RF. Periodontal ligament stresses in the initiation of occlusal traumatism. *Journal of Periodontal Research*. 1984;**19**:238-246
- [83] Jin LJ, Cao CF. Clinical diagnosis of trauma from occlusion and its relation with severity of periodontitis. *Journal of Clinical Periodontology*. 1992;**19**:92-97
- [84] Piotrowski BT, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfraction-like cervical lesions in a population of U.S. veterans. *Journal of the American Dental Association (1939)*. 2001;**132**:1694-1701
- [85] Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. *Journal of Prosthetic Dentistry*. 1984;**52**:374-380
- [86] Nakatsu S, Yoshinaga Y, Kuramoto A, et al. Occlusal trauma accelerates attachment loss at the onset of experimental periodontitis in rats. *Journal of Periodontal Research*. 2014; **49**:314-322

- [87] McCauley LK, Nohutcu RM. Mediators of periodontal osseous destruction and remodeling: Principles and implications for diagnosis and therapy. *Journal of Periodontology*. 2002;**73**:1377-1391
- [88] Fleszar TJ, Knowles JW, Morrison EC, Burgett FG, Nissle RR, Ramfjord SP. Tooth mobility and periodontal therapy. *Journal of Clinical Periodontology*. 1980; **7**:495-505
- [89] Khairnar M. Classification of food impaction-revisited and its management. *Indian Journal of Dental Advancements*. 2013;**5**(1):1113-1119
- [90] Black A. Preventive treatment of periodontal diseases. *NW Dental Journal*. 1912;**10**:60-73
- [91] Waerhaug J. Effect of rough surfaces upon gingival tissue. *Journal of Dental Research*. 1956;**35**:323-325
- [92] Lang NP, Kiel RA, Anderhalden K. Clinical and microbiological effects of subgingival restorations with overhanging or clinically perfect margins. *Journal of Clinical Periodontology*. 1983;**10**:563-578
- [93] Icon A. Amalgam restorations and periodontal disease. *British Dental Journal*. 1976;**140**:377
- [94] Pierce LH, Goodkind RJ. A status report of possible risks in base metal alloys and their components. *The Journal of Prosthetic Dentistry*. 1989;**62**:234
- [95] Pelton L. Nickel sensibility in the general population. *Contact Dermatitis*. 1979;**5**:2-7
- [96] Felton DA, Be K, Bayne SC, et al. Effect of *in vivo* crown margin discrepancies on periodontal health. *The Journal of Prosthetic Dentistry*. 1991;**65**: 357
- [97] Marcum JS. The effect of crown margin depth upon gingival tissue. *The Journal of Prosthetic Dentistry*. 1967;**17**: 479
- [98] Hochman N, Yaffe A, Ehrlich J. Crown contour variation in gingival health. *The Compendium of Continuing Education in Dentistry*. 1983;**4**:360
- [99] Parkinson CF. Excessive crown contours facilitate endemic plaque niches. *The Journal of Prosthetic Dentistry*. 1976;**35**:424
- [100] Reinhardt RA, Pao YC, Krejci RF. Periodontal ligament stresses in the initiation of occlusal traumatism. *Journal of Periodontal Research*. 1984;**19**: 238-246
- [101] Flores MT, Andersson L, Andreasen JO, Bakland LK, Malmgren B, Barnett F, et al. Guideline for the management of traumatic dental injuries. I. Fractures and luxations of permanent teeth. *Dental Traumatology*. 2007;**23**(2):66-71
- [102] Andreasen JO, Andreasen FM. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*. Copenhagen: Munksgaard; 1994