

Republic OF Iraq
Ministry of Higher Education
and Scientific Research
University of Baghdad
College of Dentistry



Tooth Wear

A Project Submitted to the Pedodontics and Preventive Dentistry
department in the college of Dentistry / University of Baghdad in
partial Fulfillment of the Requirement to Award the Degree of
B.D.S.

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B.D.S., M.Sc.

2022

1443

Dedication

I am dedicate this project to my lovely mother, father, brother, sister for their great support and for always believing in me.

To my best friends who supported me during my study

To my fiancée, Naba.

To my supervisor who encourages me to keep go.

All , I could not do all this without you.

Acknowledgment

First of all, I thank "Allah" almighty for granting me the will and strength to accomplish this research and I pray that his blessings upon me may continue throughout my life.

Deep thanks to Assist. Prof. **Dr. Raghad Abdulrazzaq AL Hashimi**, Dean of the College of Dentistry-University of Baghdad for his support to accomplish this review.

Deep thanks to **Prof Dr. Ali I. Al-Bustani**, the associate dean for scientific affairs I would like to thank Assist. **Prof. Dr. Ahlam Taha**, Head of the Department of Pedodontics and Preventive Dentistry for her Kindness and help.

I am indeed internally thankful to my supervisor **Lecturer Juman D. Al-Khayuon** for her continuous guidance, generous advice, and without their encouragement and wise supervision; the present dissertation would not see the light of the day. My great appreciation and thanks to all teaching staff in Department of Pedodontics and Preventive Dentistry. Finally, to all those whom I forgot to mention their names for their kind efforts helping me to compose this review of literature.

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List of abbreviations

Abbreviation	Criteria
CHX	Chlorhexidine
ECM	Extracellular matrix
MMPs	Matrix metalloproteinases
S	Seconds
CO₂	Carbone dioxide
Nm	Nanometer

Introduction

Dental wear is a term referring to a group of commonly observed dental tissue disorders, namely, erosion, attrition, abrasion, and abfraction (Karan et al., 2009).

Although these conditions are all characterized by loss of mineralized tissue, unrelated to bacterial action, they show morphological and etiological differences (Nguyen et al., 2008).

All dental wear is either mechanical or chemical. Abrasion, attrition, and abfraction fall into the category of mechanical wear, while erosion is chemical wear. Erosion is the gradual but irreversible dissolution of dental tissue caused by acidic agents (Johansson et al., 2012).

Its causes are closely connected with lifestyles, habits, and disorders that are common nowadays and whose identification is crucial in order to establish a correct treatment plan, which may involve not only the dentist but other specialists as well. The main causes of erosion can be divided into extrinsic and intrinsic factors (Ganss et al., 2001).

The former include intake of acid vitamin C supplements and the frequent consumption of acid foods and drinks (fruit juices, citrus fruits, soft drinks, etc.) (Lussi and Jaeggi, 2000).

The intrinsic factors include gastroesophageal reflux [Holbrook et al., 2009], eating disorders such as bulimia and anorexia, and conditions such as pregnancy and obesity that, causing increased gastric pressure, lead to acid reflux. Erosion lesions of the teeth have distinctive rounded edges; furthermore, thanks to the

protection afforded by crevicular fluids, part of the enamel on the cervical margins is typically preserved (Lussi et al ., 2004).

Attrition lesions present as large, shiny areas with clear margins; attrition defects match the morphology of the opposite teeth. Attrition wear is caused by excessive functional or parafunctional forces and bruxism (Kaidonis , 2008).

Abrasion, on the other hand, is due to the interaction between teeth and exogenous objects and substances, such as toothpicks, dental floss, highly abrasive toothpastes (in particular those with a whitening action), pipes, and toothbrushes (especially when these have hard bristles and too much pressure is applied when brushing) (Kaidonis , 2008). It may also be caused using toothpicks and miswaks, as well as the consumption of abrasive foods (Litonjua et al., 2019)

Abrasion is a type of wearing diseases that is caused by the sliding or rubbing of abrasive external objects against the tooth surfaces (Addy and Shellis, 2006).

Abfraction is loss of cervical tooth structure in the absence of caries is termed as a non-carious cervical lesion, stress-induced cervical lesion, or abfraction. Abfraction is a type of wasting diseases believed to be caused by tensile stress generated from non-axial cyclic occlusal forces (Chu et al., 2002).

Review of Literature

1. Tooth wear

The wasting diseases of teeth, namely attrition, abrasion and dental erosion have taken their toll in the population around the world due to the changing lifestyles, increase in the stress levels and many other factors that were persistent earlier but have suddenly increased drastically. Historically, the most common reason for tooth loss and dental hard tissue loss has been dental caries. Since the introduction of fluoride, the prevalence, incidence, and severity of caries has declined, and the dental life expectancy has increased. One of the most common problems associated with this prolonged dental life expectancy is tooth wear. Tooth wear is an irreversible, non-carious, destructive process, which results in a functional loss of dental hard tissue. It can manifest as abrasion, attrition, abfraction and erosion (Buzalaf et al., 2016).

2. Epidemiology of teeth wear

Millward et al.(1994) investigated, 4-year-old children from Birmingham, UK and reported that as high as 17percent showed involvement of dentine exposure.

The proportion of adults with severe tooth wear generally rises from approximately 3% in young people in their early 20s to 17% in those over the age 50 years that increasing tooth wear was observed with age particularly at the occlusal and incisal surfaces of teeth (Jones et al., 1995).

Epidemiological studies of young adults reported that prevalence of tooth wear ranged from 6 to 45% (Jones et al., 1995). In older individuals, the severity of tooth wear has been shown to be consistent.

A studies carried out on a population in northern Swedish country found excessive wear of maxillary anterior teeth in 14% of 35-year-old and 36% of wear in 65-year-old and in randomly selected Swedes of age 20–80 years, 41% of individuals showed dentinal exposure (Al-Malik et al 2002; Wiegand et al. 2006). The prevalence of tooth erosion, at least in European studies, would appear to be increasing especially among young adults and adolescents with a higher prevalence usually found in males (Nunn et al., 2003; Lussi, 2006; Arnadottir et al., 2010).

Al-Dafaai in 2007 studied dental erosion among 11-12 years old schoolchildren in Baghdad city. She concluded the prevalence of dental erosion in Baghdad city was relatively low. Sever dental erosion was associated with high frequency of carbonated drink intake and with night drinking habit (Al-Dafaai, 2007).

Al-Azawi and El-Samarrai, in 2014 studied the distribution of tooth wear among institutionalized residents (50-89 years old) in Baghdad city\Iraq (Cross-sectional study). This survey was accomplished on four private and one governmental institution. They concluded the occurrence of tooth wear among those subjects was high 100% thus they need oral health policy makers for promotion, prevention, and restorative care (Al-Azawi and El-Samarrai, 2014).

3. Classification of tooth wear

Tooth surface loss usually occurs due to three processes namely abrasion, attrition, and erosion. Abfraction is another process which may potentiate wear by abrasion or erosion (Buzalaf et al., 2016).

Experimental and clinical observations have demonstrated that individual wear mechanisms do not act alone but instead, cause loss of tooth surfaces by interacting with each other. One of the important interactions is the initiation of tooth abrasion by erosive damage caused onto the surfaces of the teeth. It appears that this interaction is a major factor in occlusal and cervical wear; however, the available evidence does not seem to be sufficient to establish the importance of abfraction as a major contributor to tooth wear in vivo (O'Sullivan et al., 2013).

4.1. Attrition

4.1.1. Definition

Attrition is a process in which tooth tissue is removed as result of opposing surfaces contacting during function or parafunction. Such direct contact occurs at proximal areas, on supporting cusps and on guiding surfaces during empty grinding movement (Marya, 2012).

4.1.2. Etiology

There are generally thought to be three principal theories regarding the etiology of attrition. In addition, there may also be modifying factors (often lifestyle factors) present, such as bone chewing (Hannig et al., 2011).

The theories of attrition are (Rees et al., 2013):

- Functional theory
- Parafunction initiated by occlusal interferences
- Central nervous system etiology

Functional theory suggests that tooth wear occurs due to prolonged contact of the teeth and the patient having a broad envelope of function. The seminal work of Lundeen et al., (1987) showed that some patients exhibit every extensive range of movement in their usual chewing pattern, analogous to a cow chewing which leads to attrition and tooth wear. Therefore, likely that any differences in the occlusal wear patterns seen between the 'grinding' and 'chopping' group would only increase as they aged.

The parafunction theory can be initiated by occlusal interferences and therefore managed clinically by occlusal adjustments or extensive rehabilitations has been present in the literature for many decades. Unfortunately, the evidence in the literature does not support this theory. For example, Clark et al., (1999) reviewed many animal and human studies and found that occlusal interferences could not cause bruxism or stop it. More recently a systematic review found no link between occlusal interferences and bruxism.

Over the last two to three decades, it has become evident that most of the bruxism is caused by a central nervous stimulus. It appears that bruxism can occur either when the patient is awake (awake bruxism) or when the patient is asleep (nocturnal bruxism). In awake bruxism the patient is naturally aware of jaw clenching, and this is a very common phenomena with a prevalence of around

20%. The etiology of awake bruxism is poorly understood but known risk factors are psychological stress and anxiety. In contrast, nocturnal bruxism is tooth grinding while the patient is asleep and the patient may be aware of this, or more likely, the patient's partner or family members are aware of this problem. The prevalence of nocturnal bruxism is reported as being 8–10% (Van't Spijker et al., 2009).

It has now been classified as a sleep-related movement disorder. Essentially, sleep bruxism occurs following sleep-related micro-arousals that originate in the brain stem.

These micro-arousals cause the heart rate to increase following which brain activity increases. This is followed by activation of the suprahyoid muscle and then this is followed by rhythmic masticatory muscle activity resulting in bruxism (Rees et al., 2013).

It therefore appears that bruxism is a neurological problem and the tooth damage that we see as dental professionals is a consequence of a neurologically initiated activity manifesting as grinding and tooth surface loss (Shellis and Addy, 2014).

Several factors are reported to predispose the occurrence of attrition. These factors include coarse porcelain on opposing natural teeth and occlusal collapse due to a lack of posterior support (Chu et al., 2002).

It is also important to realize that erosion may be superimposed or coexist with attrition/ abrasion, and this is often seen with patients who consume significant numbers of oranges for example. The citric acid within the oranges can cause

erosion while the fibrous structure of the fruit can cause abrasion, and this is often seen on lower molars. In this scenario, once the occlusal dentine is exposed then the tooth wear may accelerate as dentine tends to be lost two to five times faster than enamel (Grippio et al., 2018).



Figure 1: Examples of attrition cases (Kaidonis, 2008).

4.1.3. Clinical features

The signs and symptoms typically found in a patient presenting with attrition are outlined below (Grippio et al., 2018).

Symptoms

- Tooth grinding at night
- Jaw pain, fatigue and limited opening on waking
- Teeth feel loose (localized or generalized)
- Sore teeth or sore gums
- Headaches in the temporal region
- Grinding or clenching of the teeth while awake.

Clinical signs

- It is usually seen as well-defined wear facets on the surfaces of teeth in one jaw which match corresponding facets on opposing teeth in the other jaw. Attrition can be seen on cusps and guiding surfaces during empty mouth grinding movements as in parafunctional habits (i.e., bruxism). When the cause of attrition is active, the tooth wear is usually seen as shiny and well-defined facets (Kaidonis, 2008).
- Tooth fractures – natural teeth or restorations
- Tooth mobility
- Pulp necrosis – as loads cause limitation of blood supply
- Traumatic ulcers
- Linear alba .
- Masticatory muscle hypertrophy – particularly masseter and temporalis muscles
- Tongue indentations.

4.1.4. Clinical management

The clinical management of attrition is potentially problematic as the restorative treatment will not cure the attritional tendencies of the patient, as the drive for this is neurological, as discussed earlier. It is therefore imperative that the patient understands the etiology of this condition and is willing to commit to wearing a protective splint long term and potentially for a lifetime (Van't Spijker et al., 2009).

Treatment for bruxism can also be divided into an initial or diagnostic phase and a 'definitive' treatment phase, although it can be argued that the clinical management of a bruxism patient is never definitive as the risk for restoration failure is ever present. It is therefore often very useful to have an initial 'diagnostic' phase of treatment where the clinician can convince him/herself that the patient is compliant with splint wearing. The choice of splints available for bruxism patients are either hard splint, soft splints and, more recently, hybrid splints with a soft inner lining and a hard outer shell (Shellis and Addy, 2014) (Figure 2).

Splints can be used in either the upper or lower arch but there is little guidance in the literature as to which is best. Some clinicians believe it is potentially better to use a splint in the upper arch as the hard palate is available to distribute any occlusal loads more widely, but hard evidence for this recommendation is lacking (Shellis and Addy, 2014).

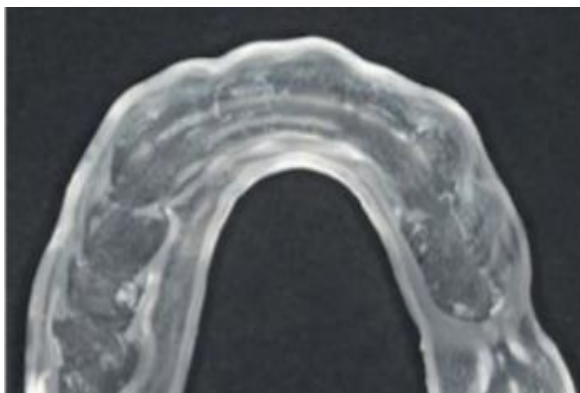


Figure 2: Hybrid hard and soft splint (Van't Spijker et al., 2009)

Hard (usually acrylic) splints (Figure 3) can also be used to allow any spasm in the muscles of mastication to resolve, which will allow recording of the returned contact position more easily. In addition to this, a hard splint can also be used to reversibly assess any increase of the occlusal vertical dimension if this is thought necessary, although many clinicians will now only use a splint in this way if increases in vertical dimension are large and in excess of 4–5 mm (Vlacic et al., 2007).

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Figure 3: Hard splint for management of teeth wear (Van't Spijker et al., 2009).

During this initial diagnostic phase, it is also important to explore any other potential contribution to the tooth wear presenting clinically, such as erosion. This may well involve the use of a diet history taken over at least three days together with the use of topical fluoride and modern desensitizing toothpastes. If toothbrush abrasion is thought to be a contributory factor, then advice about brushing in relation to consumption of any acidic food or drink at least an hour apart should be given (Grippio et al., 2018).

Generalized wear can also be managed using direct composite restorations (Featherstone et al., 2006). Attin et al. (2016) described a technique using clear silicone putty matrices for restoring the posterior teeth developed from an initial diagnostic wax up and following this the anterior teeth are restored. Attin et al., (2016) have also followed a small group of six patients treated in this way and the reconstructions have survived well to 5.5 years (Kurary Dental, Maiden Lane).

An alternative and conservative approach to managing full arch tooth wear is to use adhesive gold onlays on the molar teeth and use direct or indirect composite to restore the remaining teeth, increasing the oral vertical dimension as necessary as shown in (Figure 4). Adhesive gold onlays are rather more conservative than traditional gold crowns (Featherstone et al., 2004). Chana et al. (2000) reported an 89% survival probability at five years when cemented with Panavia cement.



Figure 4: Gold onlays used to manage generalised attritional wear (Rees and Somi,2018)

Finally, the more traditional way of dealing with a full arch or mouthrehabilitation is to use conventional crowns. These are usually placed using a mutually protected occlusal approach with canine guidance and anterior guidance protecting the posterior teeth in protrusive jaw movements. This traditional approach is both very destructive and very expensive. There is a relatively high possibility of pulpal death in around 10–15% of teeth and the longevity data for conventional crowns suggests a mean life span of around ten years (Lueckel et al., 2017).

4.2. Abrasion

4.2.1. Definition

Abrasion refers to the loss of tooth structure due to external agents which have an abrasive effect on the teeth for example, toothbrush bristles and dietary factors (Marya , 2012).

4.2.2. Etiology

Several factors are reported to cause such lesion. These factors include the use of an abrasive toothpaste, hard bristles, and a vigorous brushing technique. It may also be caused using toothpicks and miswaks, as well as the consumption of abrasive foods (Litonjua et al., 2019) as shown in (Figure 5).



Figure 5: Cervical abrasion lesion caused by the aggressive use of the chewing stick (Miswak) (Addy and Shellis, 2006).

Despite the general belief that cervical abrasions are caused by toothbrushes, toothpaste and brushing techniques, a definite conclusion is difficult to draw, as other factors such as erosion and abfraction may also play a role in the development of abrasion lesions. The orientation of the toothbrush influenced the wear of the teeth. Horizontal brushing was suggested as causing two to three times as much wear compared with vertical brushing. It was suggested that

toothpaste has more relevance to abrasion than does the toothbrush. Abrasion can occur also as a result of improper use of dental floss and toothpicks, or detrimental oral habits such as chewing tobacco; biting on hard objects such as pens, pencils or pipe stems; opening hair pins with teeth; and biting fingernails. Abrasion also can be produced by the clasps of partial dentures (Davies et al., 2002; Litonjua et al, 2005) (Figure 6).



Figure 6 : The effect of abrasion on the cervical surfaces of upper teeth (He et al .,2011).

Occupational abrasion may occur among tailors or seamstresses who sever thread with their teeth, shoemakers and upholsters who hold nails between their teeth, glassblowers , and musicians who play wind instruments (Addy and Shellis, 2006).

4.2.3. Clinical features

Abrasion lesions can be seen on the occlusal surfaces as wear areas rather than facets, which are characteristic of attrition. This is because occlusal abrasion involves the whole occlusal table. When compared with those caused by erosion, abrasion lesions are associated with relatively shallow cupping and exposed dentine that is not usually hypersensitive. The lack of hypersensitivity is attributed to the formation of a mechanical smear layer that blocks the exposed dentinal tubules (Kaidonis, 2008).

Furthermore, an acid attack on the teeth compromises their mechanical properties and makes them more susceptible to the other causes of wear, as in the case of abrasion. Clinically, cervical abrasions are commonly seen as V-shaped notches in the cervical regions of facial surfaces of one or more teeth. They are characterized by sharply defined margins and smooth surfaces (He et al., 2011).

4.2.4. Clinical management

Careful comprehensive treatment plan is required for each individual case. Indexing should be applied to rate abrasion. Generally, when patients do not show any interest in the treatment and in the absence of symptoms, a preventive approach seems to be more efficient- mainly when the patient doesn't present a dynamic wear process. However, for some other cases, restorative treatment may be required (Wetselaar et al., 2016).

Currently adhesive dentistry shows a considerable progress allowing advanced restoration techniques. Thus, many treatment options were proposed such as: direct/ indirect composite resin restorations and bonded ceramic restorations. It

is recommended that direct resin composite materials must be placed at a minimal increment thickness of 1.5 to 2.0 mm in all areas of functional loading to ensure appropriate longevity. Benefits of direct composite resin in abrasion management (Wetselaar et al., 2016):

- Acceptable esthetic outcome
- A conservative procedure applied with minimal or zero intervention
- Well tolerated by pulpal tissues
- Minimally abrasive to antagonistic tissues
- Acceptable quality / cost ratio
- Possibly applied in a single intervention
- Ease of restoration adjustment

4.3. Erosion

4.3.1. Definition

Erosion is a chemical process in which the tooth surface is removed in the absence of plaque. Erosive factors may be either intrinsic or extrinsic. Extrinsic sources include drinks such as fresh fruit juices, carbonated drinks and alcoholic beverages; and some foods and industrial processes. Intrinsic sources include gastroesophageal reflux and eating disorders(Marya , 2012) (Figure 7).



Figure 7: Example of teeth erosion (Holbrook, 2009).

Erosion is generally considered in Europe to be the most prevalent cause of tooth wear. The source of the acid can either be gastric (intrinsic) or dietary (extrinsic) acids. Dietary acids are a common part of modern diets, particularly the fruit acids. There is strong evidence to suggest that the manner in which the acid food or drink is consumed is more important than the overall quantity. Holding, swilling or retaining acidic drinks and foods in the mouth prolongs the acid exposure on the teeth increasing the risk of erosion (Moazzez et al ., 2000 ; Millward et al ., 1997).

4.3.2. Etiology

Hydrochloric acid produced by the parietal cells in the stomach causes intrinsic dental erosion. The acid reaches the mouth either through vomiting or by regurgitation. Vomiting disorders such as anorexia and bulimia nervosa have long been recognised as a factor in the development of dental erosion (Bartlett and Coward , 2001).

As the erosion continues, the destruction of enamel and dentine becomes more widespread and may not be limited to the palatal surfaces of the anterior teeth. The consequences of intrinsic erosion are often severe and require extensive restorative management to replace the lost tooth tissue. There are a number of conditions associated with the movement of the gastric acid from the stomach to the mouth. An underlying common feature of most is gastro-oesophageal reflux (Kidd and Smith, 1993).

This is the term used to describe the retrograde movement of the stomach acid past the lower oesophageal sphincter. The condition is associated with other known causes of intrinsic erosion including rumination, chronic alcoholism, and eating disorders. Unlike dietary acids, the pH and titratability of gastric juice is significantly greater and so the level of destruction is normally more severe (Bartlett and Coward , 2001).

The most common presenting dental sign of gastric acid is the development of tooth wear on the palatal surfaces of the upper incisor teeth. In some cases, it is possible to identify the cause of the wear and associate it with gastric acid, particularly with eating disorders, but for most the cause is less certain. In these circumstances, a thorough and comprehensive clinical examination is needed to

assess the most likely cause of the wear (Kidd and Smith, 1993).

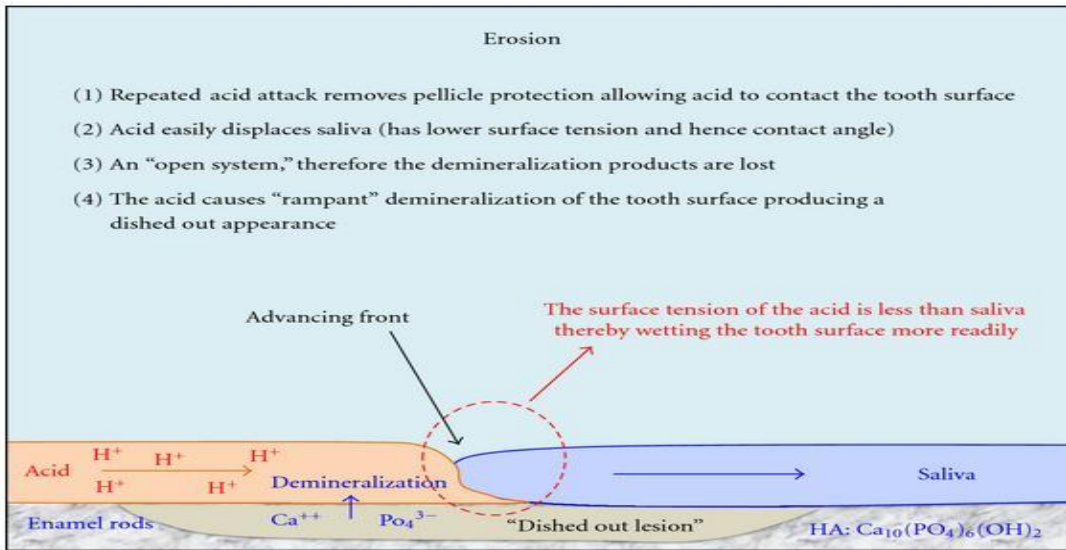


Figure 8 : when dental pellicle is removed by sustained endogenous acid attacks then demineralized tooth products are lost to the oral environment (Busscher et al ., 2000).

4.4. Abfraction

4.4.1. Definition

Abfraction is a term proposed to describe tooth substance loss by flexure and failure of tooth substance from occlusal loading at a location away from the loading, most typically the cervical enamel (Grippio , 1991).

Abfraction means 'to break away' and the term is derived from the Latin words 'ab,' or 'away' and 'fractio,' or 'breaking' by Grippio It is usually observed on the buccal surface at the cemento-enamel junction of the teeth, with prevalence ranging from 27 to 85% (Alexandaria et al., 2017).

The loss of cervical tooth structure in the absence of caries is termed as a non-carious cervical lesion, stress-induced cervical lesion, or abfraction. Abfraction is a type of wasting diseases believed to be caused by tensile stress generated from non-axial cyclic occlusal forces (Grippio, 1991 ; Chu et al., 2002).

4.4.2. Etiology

The theory of abfraction suggests that tooth flexure in the cervical area is caused by compressive occlusal forces and tensile stresses, resulting in microfractures of the hydroxyapatite crystals of the enamel and dentin with further fatigue and deformation of the tooth structure (Chu et al., 2002 ; Lee et al., 2002 ; Rees, 2006).

However, despite many efforts to demonstrate that occlusal forces are the main cause of abfraction, its etiology remains poorly understood and controversial (Michael et al., 2009).

It has been suggested that when a tooth is hyper-occluded, the masticatory forces are transmitted to this tooth, which in turn transfers this energy to the cervical region. However, it was found that the occlusal force during swallowing and mastication is only approximately 40 percent of the maximal bite force (Figure 8). Also, tooth contact occurs, on an average, for only 194 milliseconds during mastication and for 683 milliseconds during swallowing (Hur et al., 2011).

Lateral force produces compressive stress on the side toward which the tooth bends and the tensile stress is on the other side. These stresses create microfractures in the enamel or dentin at the cervical region. These fractures

propagate in a direction perpendicular to the long axis of the tooth, leading to a localized defect around the cemento-enamel junction (Dionysopoulos et al., 2019).

The lesion is formed by combined bending and barreling deformations. This leads to alternating tensile and compressive stresses, resulting in weakening of the enamel and dentin. If the forces reach a fatigue limit, the tooth cracks or breaks. At the same time, the opposite region is under compressive stress. When the direction of the force changes, the tooth bends in the opposite direction, and the stresses correspondingly reverse at this cervical area. Thus, side-to-side bending of the tooth results in fatigue and fracture of the most-flexed zone. These interocclusal forces create physical microfractures or abfractions at the cervical region (Hur et al., 2011).

4.4.3 Clinical management

Determination of the activity of an abfraction lesion can be done by using No. 12 scalpel blade. Loss of a scratch made by the blade signifies an active abfraction lesion. When an abfraction lesion is less than 1 mm in depth, only monitoring at regular intervals is sufficient, otherwise restoring it improves the maintenance of oral hygiene by the patient. It also helps in decreasing thermal sensitivity, improving esthetics, and strengthening the teeth. Along with restoration, a variety of treatment strategies have also been proposed like occlusal adjustments, occlusal splints, elimination of parafunctional habits, altering tooth brushing techniques, and the like (Dionysopoulos et al., 2019).

For restoring abfraction, many materials and techniques have been tried to date. The following materials are indicated for restoring the lesions: Glass

ionomer cements, resin-modified glass ionomer cements, polyacidmodified resin-based composites (compomers), composite resins, and a combination of the techniques. Resin-modified glass ionomer cements should be the first preference. A resin-modified glass ionomer cements liner or a base with a resin composite should be used wherever esthetics is required (Turner et al., 2019).

5.Preventive strategies of tooth wear

After the analysis of the dietary report provided by the patient, further questioning using the information listed in Fig. 9 should be undertaken. It has to be kept in mind that acidic candies and herbal teas (such as black currants, loganberries, cranberries, lemons and raspberries) may have an erosive potential and aggravate erosive lesions (Phelan and rees ,2003; Jensdottir et al ., 2005). Possible intrinsic acid exposure should also be taken into account. Based on these analyses, an appropriate preventive program may be suggested to the patients.

5.1 Fluoride

In the 1970s, (Graubart et al.,1972) showed an in vitro protective effect of a 2% sodium fluoride solution on the erosive process. Less wear of softened teeth was produced in vitro in the presence of fluoride toothpaste than in the presence of non-fluoride toothpaste with an otherwise identical formulation (Bartlet et al ., 1994).

Amaechi et al., (2003) have shown that xylitol and fluoride have an additive effect in reducing the erosive potential of orange juice in in vitro studies. Larsen, (2001) showed that the protective effect against erosion of fluoride added to soft drinks was minimal. Clearly some more research is required in this area to resolve

these differences, perhaps through the development of agreed test systems to evaluate erosive potential. It is known that tooth brushing shortly after drinking an erosive beverage causes an increase in tooth wear. Topical fluoride appears to protect against this subsequent tooth wear following acid challenge. This is especially helpful in reducing dentine wear in previously eroded teeth.

5.2. Saliva

Exposure to saliva has been shown to be effective for rehardening eroded enamel. The mechanism for this is thought to be that once the erosive agent is neutralized or cleared from the tooth surface, the deposition of salivary calcium and phosphate may lead to rehardening of the acid softened enamel (Gedalia et al., 1991 ; Amaechi and Higham , 2001).

Enamel specimens eroded by citric acid for 2 hours were immersed in artificial saliva and showed partial rehardening after 1 to 4 hours. These specimens remineralized for 6 to 24 hours and demonstrated complete rehardening (Eisenburger et al., 2001).

Saliva is an important biological factor in the prevention of erosion. It has been speculated that saliva stimulation will enhance the formation of the acquired salivary pellicle. It is known that the pellicle forms rapidly and has some protective effect against erosion(Hannig et al., 2004 ; Meurman and Frank, 2006).

Procedures that remove or reduce the thickness of the salivary pellicle may compromise its protective properties and therefore accelerate the erosion process. Procedures such as toothbrushing with abrasive dentifrice products, professional cleaning with prophylaxis paste, and tooth whitening will all remove

or weaken the pellicle and may render teeth more susceptible to erosion (Zero and Lussi , 2006). Acidic beverages may interfere with the pellicle formation and thus further modify the protective barrier (Finke et al ., 2002).

5.3. Sealant restorations

The application of dentine bonding agents and fissure sealant to eroded areas may be helpful in providing some level of protection and reduce dentinal hypersensitivity. Whilst the results of a study by(Sundaram et al.,2011). showed the longevity of sealants in the form of dentine bonding agents applied to teeth displaying severe wear to be relatively short lived, they may help to reduce the rate of wear on applied surfaces by up to a period of nine months following application Similarly, glass ionomer cements can be readily applied to worn surfaces for the same purposes (Sundaram et al.,2011).

5.4. Drink Modification

Considering the increasing prevalence of tooth erosion, specially in young children and teenagers and the strong association between consumption of acidic drinks and tooth erosion, it still seems logical to develop drinks with low erosive potential. Drink modification has been developing in recent years with varying success. Addition of calcium lactate to soft drinks has been shown to reduce the erosive potential of the most of erosive beverages. A successful attempt to reduce the erosive potential of soft drinks by the addition of calcium citrate malate was reviewed by Grenby in (2008), but a later in vivo investigation by (Rugg-Gunn et al., 2011) found no difference in the amount of erosion seen in

enamel slabs treated with plain or modified orange drinks. The low pH soft drink has been modified with the addition of calcium and has been shown in in situ and in vitro studies to be less erosive than soft drink drinks without added calcium and also less erosive than orange juice (Nilson et al., 2018) .

Conclusion

Tooth wear is a natural, dynamic and inevitable process that leads to gradual loss of dental hard tissue. It is considered pathological when exceed the acceptable normal level . Careful diagnosis and monitoring of progress are important and the underlying etiological factors of teeth wear should be corrected wherever possible. The etiology of tooth wear is often complex but individualized prevention can usually only be initiated once the disease has started. This is largely aimed at limiting progression of tooth wear in the affected individual.

Several preventive strategies for patients suffering from tooth wear are included dietary advice, stimulation of salivary flow, optimization of fluoride regimens, modification of erosive beverages and adequate oral hygiene measures. However, clinical trials are required to confirm the relevance of these measures. As tooth wear cannot be prevented totally with the recommended strategies, further research is necessary to develop new measures with higher protective capabilities and good clinical acceptance.

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