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Bruxism and prosthodontics treatment

A Project Submitted to
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سُورَةُ الأَنفَالِ

Certification of the Supervisor

I certify that this project entitled ***Bruxism and prosthodontics treatment*** *was prepared by the fifth year student* ***Fatima Jameel Abd Ali*** *under my supervision at the College of Dentistry/University of Baghdad in partial fulfilment of the graduation requirements for the Bachelor Degree in Dental surgery.*

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Dedication

I *dedicate this project to all of my family members. It would not be possible to complete it without their support.*
And to my close friends who poured me with confidence and faith to believe that I can do it.

Finally ،I want to thank myself for reaching this stage and still continuing with courage and steadfastness.

Fatima Jameel Abd Ali

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SB	**Sleep bruxism**
AB	**Awake bruxism**
TMD	**Tempromandibular disorder**
TMJ	**Tempromandibular joint**
CNS	**Central Nervous system**
TSL	**Tooth surface Loss**
EMG	**Electromyography**
CBT	**Cognitive Behavioural Therapy**
PSG	**Polysomnography**
BTX	**Botulinum toxin**
BTX A	**Botulinum toxin type A**
SNAP-25	**Synaptosomal protein -25**
NSAID	**Non steroidal anti inflammatory drug**
BU	**Botox Unit**
IU	**International Unit**
FDP	**Fixed Dental Prostheses**
LED	**Light Emitting diode**

Introduction

The term bruxism refers to a non-functional contact of mandibular and maxillary teeth often resulting in the clenching or grinding of teeth**. (Glaros and Rao, 1977)**

This dyskinesia most often occurs during sleep although it also may occur while awake. **(Bader, 1997).**

Typical symptoms are abrasion of the dental hard substance, chipping or even fractures of teeth and prostheses, pain in the affected muscles and joints, and teeth which are sensitive to biting. **(Greene ,1998)**.

Bruxism can be divided into idiopathic and iatrogenic types.

The idiopathic form, which includes clenching and grating as well as nocturnal bruxism, is not linked to neurologic or psychiatric disorders **(Glaros,2006).** The prevalence of bruxism during infancy is 14–20%. Orofacial dyskinesia affects about 8% of adolescents (**Egermark , 2003)** and 8% to 9% of adults **,** a percentage that decreases to less than 3 in the age group of 60 years and older (**Ohayon , 2001) .**

This review is concerned with the relationships that may directly or indirectly exist between bruxism and prosthetic treatment. Although certain occlusal conditions and/or incorrectly prosthetically modified occlusions were historically believed to be potential causes of bruxism, this has largely ceased to be the case. **(Lobbezoo, 2010)**.

Also, the assumption that correction of such occlusal conditions could reverse bruxism has also been discredited. **(Lobbezoo, 2010).**

What is important in the present context, however, is the possible effect of bruxism on prosthetic restorations.A relationship upon which the dental literature would appear not to be conclusive.

The treatment of bruxism may include various therapy options.

Aim of the Review

The aim of review is to identify the aetiology of Bruxism,types ,treatment and effects of bruxism on prosthetic restorations and natural teeth.

Chapter One

Review of literature

Definition of the Bruxism

Bruxism is an oral condition of great interest to both researchers and clinicians in the dental, neurological and sleep medicine domains (**Lavigne ، 2005).**

Bruxism is a diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing and grinding of the teeth. (**De Leeuw , 2008)**

In addition, this definition employs diurnal and nocturnal as indicators for the conditions effects circadian relationships, while sleep and awake are to be preferred for their unbiased nature (some of us sleep during the day and are awake at night). **(De Leeuw, 2008).**

The inclusion of bracing is interesting, although linking this term to the teeth makes bracing a mere synonym of clenching.Bruxism is a repetitive jaw muscle activity char acterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. **(De Leeuw, 2008).**

Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism).(**De Leeuw, 2008)**.

1.2 Aetiology and pathophysiology

1.2.1 Aetiology

Researchers have suggested that there may be some degree of inherited susceptibility towards sleep bruxism symptoms progression. **(Heyat, 2021)**
It has been reported that 21–50% of people with sleep bruxism have a close family member who had SB in childhood, suggesting the involvement of genetic factors **(Caivano, 2021)**.With that in mind, Rintakoski et al,
Tried looking for any genetic or environmental factors that may play a role in the phenotypic variance of bruxism **(Rintakoski 2012).**

They demonstrated that there is a sex-independent genetic component that occurs more frequently in phenotypic SB variations than in other tempromandibular disorder .Based on that, they reported the heredity of bruxism, although they emphasised that the emancipation is dependent on age and environment, and so for example the older a pair of twins, the greater the differences that are observed between them. **(Maciejewska, 2021).**

Thus, although the genetic factor has been found, the entire mechanism behind it remains unknown. **(Maciejewska, 2021)**.

On the other hand, one of the most commonly used explanations of causes of bruxism is stress (**Przystańska , 2019)(Chattrattrai, 2022).**

We can clearly observe an increasing tendency of patients in the COVID-19 pandemic period to look at articles on the topic teeth grinding at night on the internet **(Kardeş , 2022).**

Increased stress during the pandemic resulted in an elevated number of bruxism episodes in society. **(Kardeş , 2022).**

That, combined with more difficult access to doctors in the same period resulted in people looking for explanations for their symptoms on the internet, leading to growing public awareness (**Wetselaar , 2019)** .

Recently, researchers have focused more on younger people such as college students, noting a significant increase in the occurrence of bruxism in this studied group (**Quadri, 2015).**

This serves to highlight the presence of a correlation between age and incidents of bruxism as epidemiologically variable, which indicates the need for local research to isolate vulnerable groups and implement appropriate preventive measures.

1.2.2 Pathophysiology

Bodily functions disturbed by bruxism can include, among other things, increased activity of the masticatory muscles, masticatory muscle hypertrophy

(especially of the masseter), scalloped or burning tongue, linea alba on the cheeks along the bite line, tooth tissue damage (enamel cracks, abfractions, excessive tooth wear beyond what is expected with age) (**Koyano, 2008)(van Selms, 2013)** .

Repeated damage to prosthetic restorations, disturbances of the amount and composition of secreted saliva, an often severe manifestation of craniofacial pain, stiffness of the TMJ**(Svensson , 2008)(Carra , 2012) .**
Gingival fluid secretion increase ,bone exostoses, and periodontal leases (increased tooth mobility, gingival recessions) **(Svensson , 2008)**
Yet, in most cases, the origin of symptoms is unknown.

The vast majority of researchers agree that the direct trigger of bruxism symptoms comes from the Central Nervous System (CNS) (**Klasser , 2015).**

It has been proved in metanalysis that bruxism seems to be associated with distinct differences in the neural pathways related to the control of the jaw closing muscles(**Boscato , 2022) .**

Disturbances in the concentration of catecholamines, especially dopamine, which affect the mandibular motor dysfunctions, are thought to be of great importance in the mechanism of bruxism **(Mascaro , 2005).**
The first evidence of such a correlation came from a case report in which a patient suffering from Parkinson's syndrome was treated for grinding his teeth with L-3,4-dihydroxypheny lalanine (L-DOPA), a dopamine precursor. In a series of con- trolled studies in young, healthy SB patients, L-DOPA has been shown to cause a slight but noticeable reduction in episodes of SB compared to a placebo group. **(Lobbezoo,1997)**.

While bromocriptine, a dopa mine antagonist, has been shown not to affect expected rhythmic activity in the masticatory muscles (**Lavigne, 2001)**. Given the pre- sumed role of norepinephrine in bruxism, experimental trials have been conducted with propranolol and clonidine **(Huynh , 2006)**. Propranolol, a non-selective beta-blocker, has not caused a significant

reduction in SB, but clonidine, an alpha-agonist acting on the CNS, has significantly reduced SB score when compared to a placebo.

This effect was partially related to the concomitant reduction in cardiovascular sympathetic dominance preceding the rhythmic activity of the masticatory muscles. **(Przystańska, 2019)**

Simultaneously, fluctuations in the level of dopamine and disorders of its receptors in the brain are often associated with chronic stress (**Dahoun , 2019).**

The pathophysiological mech anism in which stress affects the occurrence of bruxism has been explained by evidence that individuals with increased levels of neuroticism and anxiety disorders tend to release their emotional tension by engaging in activities related to bruxism. (**Serra-Negra , 2017**) Increased concentration of stress hormones can also be noted in many chronic diseases.

Psychosocial factors such as state anxiety and trait anxiety as well as alexithymia are also among the reported causes of the occurrence and maintenance of AB **(Przystańska , 2019).**

Bruxism-related TMD can be explained by the craniofacial anatomy.

As there is a fixed relationship between the maxillary teeth and the base of the skull, and at the same time the man dibular teeth have a set position in relation to the TMJ, this means that misaligned contact of the upper and lower occlusal surfaces may have a direct impact on the position and motor function of the mandible head in the TMJ (**Murali , 2015)**.

In acute and subacute TMD, It has been shown that mandibular neck weakness and craniofacial symptoms are more common in women with bruxism than in a control group (**Piekartz, 2019)**.

Clear indications of bruxism have also been observed in children with asthma and sleep apnoea (**Buske-Kirschbaum, 2003)**.

It is also possible that drugs such as dopamine antagonists, dopamine agonists, tricyclic antidepressants, and selective serotonin inhibitors, as well as alcohol, cocaine and amphetamines, can contribute to the progression of bruxism symptoms. **(Buescher , 2007)**.

Studies have shown that reducing the dose clearly reduces the symptoms **(Buescher , 2007)(Quadri, 2015)** .Dystonias and other muscular problems, as well as developmental disorders including various types of autism and neurological diseases that may have environmental and traumatic causes, can further be exemplified as bruxism-related conditions (**Goldstein, 2017).**

1.3 Risk factors

1. **Age**: Bruxism is more common in young children and noted to decrease by adulthood.
2. **Stress**: Increased stress and anxiety can cause bruxism.
3. **Personality**: Aggressive, competitive and hyperactive type of behaviour and personality can increase the chance of teeth grinding.
4. **Family history:** Sleep bruxism tends to give a family history, other members also may have teeth grinding or a history of it.
5. **Medications and habits**: Certain antidepressants can result in bruxism as an uncommon side effect. Habits like smoking, tobacco chewing, drinking caffeinated beverages may increase the risk of bruxism.
6. **Other factors-** Bruxism can be associated with medical problems like epilepsy, sleep related disorders, dementia, parkinson's disease and gastroesophageal reflux disorder **.(Kanathila, 2018).**

1.4 Signs and symptoms of Bruxism

1. Pain in the teeth and sensitivity to heat and cold.

2. Chronic muscular facial pain with tension headaches, caused by intense muscle contraction.

3. The noise noticed by parents, friends or relatives, that occurs as the teeth are ground together.

4. An abnormal alignment of the teeth, caused by uneven tooth wear

5. Flattened and worn tooth surfaces, which may reveal the underlying yellow dentine layer.

6. Micro-fractures of the tooth enamel.

7. Broken or chipped teeth

8. Loose teeth with possible damage to the tooth sockets

9. Stiffness and pain in the jaw joint (temporomandibular joint)that cause restricted opening and difficult chewing; sometimes the jaw joint may suffer damage that is slow to heal.

10. Earache**. (Lavigne , 2003).**

1.5 Types of Bruxism

1.5.1 Bruxism and sleep

Sleep bruxism is classified by the International Classification of Sleep Disorders as a sleep related movement disorder.**(Rosenberg, 2013).**

Sleep bruxism appears to occur as a reaction to microarousals during sleep, three to ten seconds long episodes of increased heart rate and muscle tone, occurring eight to 15 times per hour in healthy subjects(**Lavigne , 2008).**

Most SB episodes occur in clusters during these microarousals(**Miyawaki , 2003**) and subjects with SB exhibit more microarousals than controls**(Carra, 2012**).

Sleep bruxism may be concomitant with other sleep disorders for example, sleepwalking, sleep terrors and sleep-talking. (**Shochat , 2007**). which are also associated with arousal from sleep or confused wakening. **(Shochat , 2007**).

Around 60% of subjects without SB also exhibit masticatory muscle activity during sleep, but to a much lesser extent and without tooth contact.

Sleep bruxism is considered to be an extreme manifestation of normal muscle activity during sleep. (**Shochat , 2007**).

Subjects with SB may also exhibit more whole body movements (for example, twitches, jerks during sleep than controls, **(Bader , 2001)** with studies finding that body movements were associated with SB in 65–78.9% of subjects23 and 93% of SB episodes(**Oksenberg , 2002).**

1.5.1.1 Possible function of sleep Bruxism

Bruxism is often considered as a disorder, or a parafunction.

A disorder is characterised by lack of normal functioning of physical or mental processes, and a parafunction is a disordered function, for example, a normal function done to an excessive extent, which has led to the consideration by some of bruxism as a parafunction. **(Lobbezoo , 2013)**.

It has been postulated that sleep bruxism may have a protective role during sleep, which may relate to airway maintenance(**Lobbezoo , 2013)**or in stimulating saliva flow to lubricate the oropharynx.(**Miyawaki, 2003).**

1.5.1.2. Diagnosis of sleep bruxism

A diagnosis of bruxism may be made by the following:

1.5.1.2.a Patient report and clinical interview.

1.5.1.2.b Clinical examination.

1.5.1.2.c Intraoral appliances.

1.5.1.2.d Electromyography (EMG).

1.5.1.2.e Polysomnography (PSG).

1.5.1.2.a Patient report and clinical interview

The questions relating to awake bruxism may be used to help distinguish SB from other behaviours.(**Lobbezoo , 2017).**

However, diagnosis via questionnaire alone may be inaccurate, for example due to lack of awareness of current behaviours (for example, grinding noises in subjects sleeping alone). **(Lavigne, 2008)**.

Up to 80% of patients may be unaware of bruxism.(**Thompson, 1994)** Reports by sleeping partners of grinding noises during sleep are particularly suggestive of SB**(Carra, 2012)** since normal orofacial muscle activity does not cause noise.

Painful TMD and bruxism whilst any patients are unaware of having SB, conversely some patients who do not have SB may falsely believe they do have the condition, because they have been told by their dentist that they do. This is probably based on the erroneous assumption by the dentist that painful masticatory muscles are caused by bruxism. **(Smith, 2009).**

A dentist may, therefore, conclude that a patient suffering from a painful temporomandibular disorder (TMD) must be a bruxist.

Significantly more subjects with painful TMD self-reported bruxism than controls (55% vs 15%), although there was no difference in prevalence of SB as diagnosed by PSG (around 10%). **(Smith, 2009).**

Interestingly, 39% of those with TMD pain had been told by their dentist that they had SB.(**Raphael , 2012).**

1.5.1.2.b Clinical examination

Many of the given observations, for example tooth surface loss (TSL), are subjective and/or may represent signs of historical bruxism.**(Lobbezoo , 2008).**

Studies have failed to demonstrate an association between tooth surface loss and bruxism or TMD **(Baba, 2004)**.

Tooth surface loss will be a combination of normal physiological functional wear, wear associated with bruxism, plus erosion from dietary or gastric sources. **(Lavigne, 2008)**.

Tooth surface loss may be historical and cannot be used to indicate static clenching activity. Extent of tooth wear would be influenced by factors such as dietary and gastric acids, enamel quality and quantity and lack of posterior tooth support. As such, TSL is described as a weak indicator of bruxism. **(Lavigne, 2008)**.

However bruxofacets that is, tooth wear in an eccentric position of closure, may seem more convincing evidence of bruxism than wear in the intercuspal position. (**Koyano, 2008)**.

Tooth surface loss alone should not, therefore, be regarded as a reliable indicator of active bruxism, but should be used in conjunction with other clinical indicators. (**Koyano, 2008)**.

1.5.1.2.c Intraoral devices

Some intra-oral appliances aim to detect SB, such as via the incorporation of electric devices detecting forces applied during clenching/ grinding(**Baba, 2004).**

The use of intra-oral appliances in diagnosis relies on patient tolerance and on the assumption that the insertion of the device will not affect bruxist activity. **(Koyano, 2008).**

The observation of wear facets on intra oral splints has been observed in the literature and anecdotally, although this has not been validated in detection of bruxism.**(Koyano, 2008).**

1.5.1.2.d Electromyography (EMG)

EMG records the electrical activity of muscles generated during movement, and will provide information on extent, duration and force of muscle activity. **(Lavigne , 1996).**

EMG uses sensors attached to the skin overlying the masseter or temporalismuscles. Recordings can be made using ambulatory devices, so are also suitable for detection of AB as well as SB. **(Lavigne , 1996).**

.EMG cannot detect grinding noises, nor can it distinguish between bruxism and other orofacial activities such as swallowing, talking, lip biting/sucking, which represent around 85% of EMG recordings in controls.**(Lavigne , 1996).**

1.5.1.2.e Polysomnography (PSG)

Polysomnographyincorporates various recordings including EMG, electro encephalogram, electrocardiogram and audio-visual recordings.

These detailed evaluations allow arousal from sleep to be assessed, and the presence of other sleep disorders to be ruled out.(**Koyano , 2008).**

Bruxist episodes can be distinguished more readily from other orofacial movements**(Koyano , 2008).**

PSG with audio visual recording is the gold standard mode of assessment and diagnosis of sleep movement disorders and SB.

Although it is not without disadvantages relating to its complexity and requiring specialist equipment and is generally carried out in the sleep laboratory setting for research purposes only**(Lobbezoo , 2008).**

This investigation is clearly out with the scope of dental practice.

1.5.2 awake Bruxism

Many oral habits exist that can cause damage to the stomatognathic system. Several of these are well documented or readily identified by the patient. Many people, however, clench or grind their teeth while they are awake without realizing they are doing so.(**Panek, 2012).**

There are multiple kinds of diurnal oral parafunctions that can affect the stomatognathic system, including but not limited to nail-biting, chewing on cheeks or other mucosa, and chewing on pens or other objects **(Panek, 2012).**

Although clinicians should be aware of these conditions and educate patients about the effects, for brevity, we will focus specifically on awake bruxism tooth to tooth contact while the patient is awake.**(Panek, 2012).**

In our clinical experience, this is a relatively common habit that frequently results in the need for dental work (Figure 1.1). Compounding the problem, there is a lack of awareness specific to awake tooth clenching and grinding.

Figure(1.1) Awake bruxism habit

A. this 46-year-old man showed continued wear on his anterior teeth.

B. On questioning, we determined that the patient had an awake bruxism habit—grinding his anterior teeth in times of stress **(Goldstein & Clark, 2017).**

1.5.1 IDENTIFICATION

For clinicians to treat awake bruxism, they first must diagnose it.

A simple first screening step is to ask patients (on a medical history or examination form) whether they clench or grind their teeth.

Much parafunctional activity is not accompanied by noise, however, which can make self-awareness difficult. (**Rouse , 2010)** .

There is encouraging evidence, however, that once a person is made aware of waking oral habits, he or she is more likely to give accurate feedback **(Glaros, 2012; Kaplan , 2016).**

This initial screening can serve as an opening dialogue for clinicians to educate and inform patients about awake bruxism. Another critical way for clinicians to identify a patient with a parafunctional habit is by damage to tooth structure. **(Goldstein, 2017)**

This damage includes wear facets, fractured teeth and restorations, craze lines, abfractive lesions, and ultimately loss of teeth (Figure1.2)**(Kawakami , 2014; Goldstein, 2017)**.

Although many times these are readily identifiable through a clinical examination, clinicians should use mounted diagnostic models to detect these defects more accurately and thoroughly. (**Shetty, 2010).**

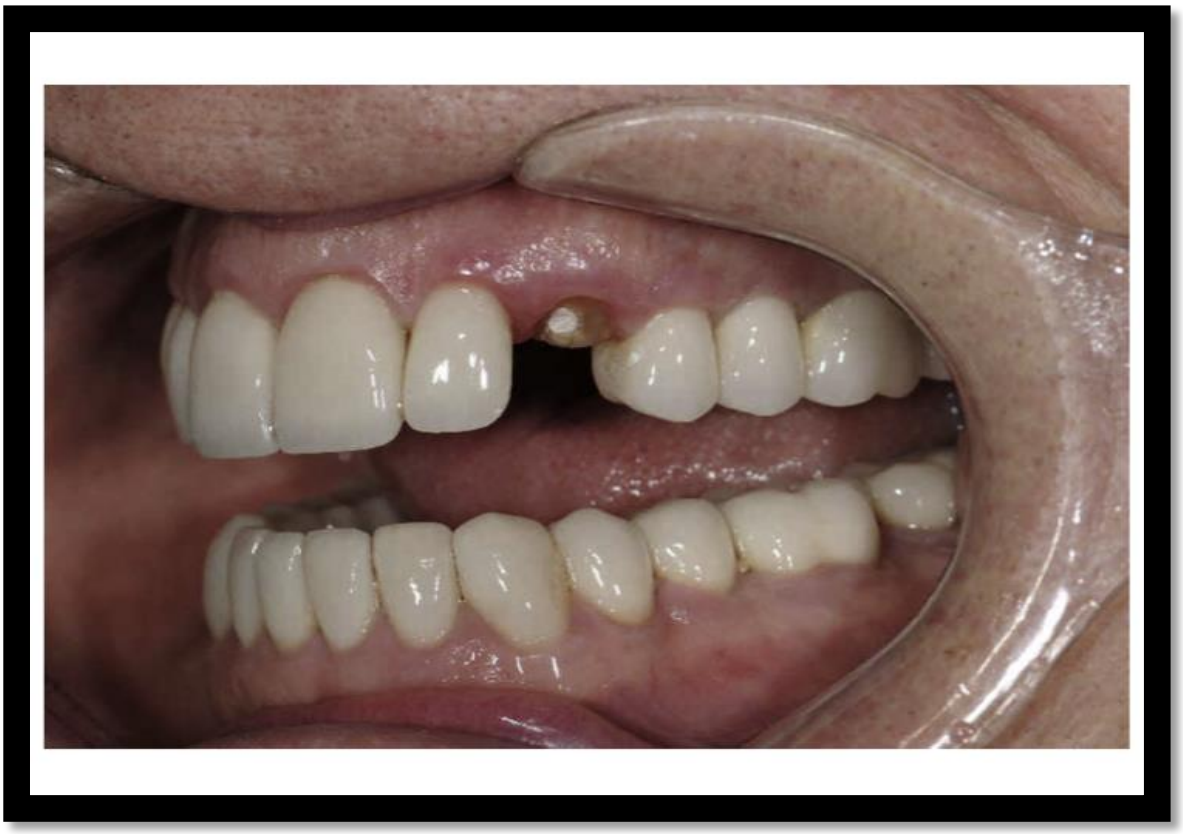
Other intraoral signs include indentations along the side of the tongue, as well as bony exostoses or tori.**(Shetty, 2010).**

Periodontal changes, including widening ofthe periodontal ligament, tooth mobility, and recession, also may occur(**Machado, 2007).**

If clinicians note this kind of damage, they should discuss the damage with the patients and review their medical history to determine the cause. When the clinician suspects a patient has a sleeping or waking parafunctional habit, it is important to discern what that habit specifically is.To differentiate be- tween sleep and awake bruxism, for example, the clinician can mea- sure the electromyographic activity of the masticatory muscles(**Piquero, 2000).**

The dentist can prescribe easy to use electromyographic devices to use during sleep to confirm muscle activity (for example, BiteStrip, Great Lakes Orthodontics).

After initial screening and suspicion of a parafunctional habit, more in depth patient ques- tionnaires (with clear definitions) and at home journaling can help further identify what type of parafunctional activities the patient engages in and how often.**(Kaplan ,2016**).



Figure(1.2) Awake and sleep BruxismWith fracture in his right maxillary canine (Goldstein, 2017).

The patient who had a history of awake and sleep bruxism previously had received metal ceramic restorations. While clenching his teeth during a strenuous situation, he fractured his right maxillary canine through the fiber post and core.

1.6 therapy for Bruxism

1.6.1 Educational therapy

The purpose of this is to make the patient aware of the existence of parafunction and its harmfulness, as well as to interrupt pathological reactions. The patient is recommended to conduct regular physical activity, maintain muscle and joint balance (correct posture), to avoid gum chewing, to not clench their teeth, and to maintain correct nasal breathing with the correct position of the tongue. **(Carlson , 2001)**.

He/she is also advised to use physical self-regulatory tech- niques.
The goal of this method is to produce physiological changes that will reduce pain, fatigue and physical overload. **(Carlson , 2001).**
However, only a small number of well-designed studies into the effectiveness of Cognitive Behavioural Therapy are available at the moment, meaning that further exploration is needed in order to unequivocally state that CBT has a positive tive effect in SB management. **(Minakuchi, 2022).**

1.6.2 Biofeedback therapy

This technique is based on the premise that patients with bruxism can consciously unlearn their pathological behaviour **(Amorim, 2018).**
It uses positive feedback as a teaching strategy focused on lowering patients stress levers, potentially ensuing in longterm behavioural changes aiming at the reduction or elimination of symptoms.**(Lobbezoo, 2018)**.
This is, however, a controversial method of remedying bruxism, as studies have shown that significant results have been achieved with AB but not yet with SB .(**Lobbezoo, 2018) (Wang , 2014)**.

Furthermore, proper sleep hygiene (i.e. relaxation before bedtime, caffeine reduction, etc.) is recommended for SB control.
Although the therapeutic effect has not been proven **(Mesko , 2017).**

Mobile phone applications such as BruxApp are available to download and may be helpful in tracking occurrences of AB as well as control attempts. They should make patients aware of the scale of the problem along with helping to cut down the number of episodes of AB. **(Colonna , 2020**).

The effectiveness of biofeedback therapy in studies is in general highly rated, but the patient often needs to carry a portable device that's either continuously monitoring muscle tension or frequently reminds the carrier to consciously relax their masseters throughout the day. **(Minakuchi , 2022).**

1.6.3 Muscle relaxation and posture exercises

The proper mandibular resting position is as follows: lips together, teeth having contact only when swallowing. To balance the overactivity of the masticatory muscles and stretch them, muscle relaxation exercises are recommended. **(Amorim, 2018)**.

These ex-ercises are also aimed at teaching the patient the correct resting position of the tongue, the correct path of nasal breathing, and how to avoid parafunction. **(Amorim, 2018)**.

For example, myorelaxation exercises have been used with good results in our clinic. A single cycle of relaxation exercises includes five steps:I. Setting the mandible in a relaxed position II. Protrusive movement to reach 'tete-a-tete' teeth contact III. Opening mouth .IV. Closing mouth until -a-tetecontact V. Returning jaw to starting position.Exercises are recommended three times a day, with 20 rep- etitions each time in a half-sitting position with supported head, straight and uncrossed legs. Patients should exercise daily for a month, freely but without force, roughly at the speed of their heartbeat **(Amorim, 2018)**.

1.6.4 Occlusal splint therapy

To prevent negative consequences of SB, stabilising and repositioning splints can be used **(Jokubauskas, 2018)(van der Zaag, 2005).**

These splints eliminate occlusal obstacles, relax the chewing and neck muscles by passive stretching, improve the occlusive and neuromuscular stabilisation, and reposition the mandibular heads and articular discs. **(de Paula Gomes, 2015).**

Showed that the use of splint therapy in the treatment of SB improves quality of life. They showed that the use of occlusal splints creates a biomechanical balance between the physiological load and that generated by stress. Occlusal splints can lead to the stabilisation of bruxism by reducing deformities and deviations in the temporomandibular joint, reducing the load on the joint **(Gholampour, 2019).**

Different types of splints vary slightly in their effectiveness, but due to the low level of commitment needed from the patient, the relatively low price, and noticeable pain reduction, they are often the chosen option.

We must underline nevertheless that most splints only mechanically minimise tooth destruction, and so do not affect bruxism itself. **(Minakuchi, 2022).**

Additionally, in the case of SB, they have to be worn at night, which for some patients is a significant inconvenience **(Minakuchi, 2022)**.

1.6.5 Short-term medications

Analgesics, sedatives, anxiolytics, antidepressants, and muscle relaxants can be used in the pharmacotherapy of bruxism **(Barth , 2021)**.

Whereby the NSAIDs are recommended to use for pain relief.In order to reduce the increased skeletal muscle tone, patients are administered tolperisone (e.g. mydocalm 50 mg 3 x 1). **(Kulkarni, 2020).**

For anxiolytic and sedative treatment, most often 25 mg of hydroxyzine 1 hour before bedtime is prescribed **(Murali , 2015).**

However, recent studies have shown that there is insufficient evidence based data to draw definite conclusions concerning medications attenuating SB and/or AB **(de Baat , 2021).**

1.6.6 Botulinum toxin

Botulinum toxin has been widely used both in the treatment of various diseases and in aesthetic medicine. It is a strong neurotoxin an exotoxin produced by strictly anaerobic bacteria (bacilli called Clostridium botulinum, and a few other representatives of the genus Clostridium). Type A botulinum toxin is the most potent toxin known (**Rasetti-Escargueil , 2019).**

It binds permanently to the neuromuscular plate, and paralyses neuromuscular conduction by fragmenting the SNAP-25 protein necessary in the process of acetylcholine release from presynaptic terminals. After intramuscular injection, there is initially rapid binding to specific, high affinity cell surface receptors for the toxin, followed by the toxin being transported across the cell membrane by receptor mediated endocytosis, and eventually the toxin is released into the cytosol. **(Kwon, 2019)**.

This process is accompanied by a progressive inhibition of acetylcholine release. **(Botzenhart, 2019)**.

The results begin to be noticeable after 2–3 days the maximum effect occurs around 2–6 weeks after the injection and lasts 2-4 months.

BTX-A indirectly reduces the bite force (i.e., the force generated by the masticatory muscles measured at the contact point of antagonist teeth during biting) by 20–40% in the masseter muscle **(Botzenhart, 2019).**

Fernandez-Nunez (2019) reported that an injection of BTX-A can reduce the frequency of bruxism episodes, lower the level of pain, and weaken the bite strength.**(Fernández-Núñez , 2019)**.

Kwon (2019) have recommended the administration of BTX-A at a dose of 25–30 BU in the masseter per side, at three injection sites, and in the temporalis muscle recommended a dose of 15–20 BU per side at 2–3 sites **(Kwon,2019)**

It is advised to aspirate each time in order to avoid intravenous administration.

Jost(2019) recommended in his book a dose of 50–100 U in the masseter per side at three injection sites, and in the temporalis muscle a dose of 50–100 U per side at 3–4 sites as well. **(Matusz, 2022).**

Monroy(2006) have stated that treatment of bruxism with botulinum toxin can cause dysphagia, slight pain at the injection site, and temporary excessive salivation, but that these symptoms appeared only in patients who received a dose above 100 IU or who had other systemic diseases present. **(Monroy, 2006)**.

1.6.7 Psychological care

Psychological factors vastly influence the aetiology and treatment of bruxism (**Fluераşu, 2022).** The maximum bite force decreased in an experimental group (administered with BTX-A) and in control and placebo groups as well. (**Murali, 2015).**

The lack of differences between the control group and the placebo group may indicate a substantial role of psychological intervention in the treatment of bruxism.

Psychological counselling, self-suggestion, hippo therapy or psychotherapy, relaxation techniques and stress management are all endorsed in the treatment of bruxism.**(Imbriglio, 2020).**

1.6.8 Biostimulation

Electro-galvanic stimulation relaxing the muscles is currently used as a therapy for bruxism. Despite its simplicity, this is not a widespread method, and there are still only a few publications regarding its efficiency **(Lal, 2022)**. Another inexpensive new method is photobiomodulation with light emitting diode **(Kobayashi, 2019),** which has been used, with very promising results, in the treatment of fibromyalgia .

Despite little research, we can already find optimistic results reporting biostimulation to have a magnifying effect on other forms of treatment, for

example in patients with splints having their trigger points irradiated **(Demirkol, 2015).**

1.6.9 Correction of short-circuit disturbances

In some cases, correction of malocclusion is sufficient. Improvements are achieved by integrated dental work including orthodontic prosthetic and conservative treatment **(Ribeiro-Lages, 2020)**.

Obtaining evenly distributed occlusal forces on the surfaces of the teeth will allow harmonious work of the muscles and, as the outcome, their relaxation **(Ghafournia , 2012).** However, opinions remain divided.

Some scientists consider the above claims to be true, while others highlight the fact that in order to properly remodel patient's occlusion, a dentist has to interfere, even minimally, with already worn teeth.

Both parties agree though that the problem does not start with the teeth, but rather with the nerves and muscles **(Shetty, 2010)**.

1.7 Effects of bruxism on prosthetic restorations on natural teeth

Fixed dental prostheses are successful prosthetic restorations in partially dentate patients. Systematic reviews have demonstrated survival rates of conventional FDPs of 94% after 5 years and 89% after 10 years (**Pjetursson, 2007**), the most common technical failures reported included loss of retention and fracture of material.

It is often suggested that the occurrence of such failures is greatest in patients with bruxing habits(**Bra gger, 2001).**

For example, when prosthetic restoration is being provided for a worn dentition (usually with teeth having short clinical crowns), it will be difficult to achieve adequate mechanical retention and resistance forms for conventionally cemented restorations. **(Bra gger, 2001).**

Furthermore, the potentially greater load on restorations if there is bruxism, heavy chewing forces, or unfavourable loading directions between teeth, means that great caution is needed in the design of the restoration if the risk of mechanical failure is to be reduced. **(Eliasson, 2007).**

We found no controlled study in this regard, although several reports have noted the possible association between bruxism and survival of FDPs (**Bra gger, 2001)**.

reports of wear on natural teeth and prosthetic restorations opposing various materials have appeared, and a few examples of such occurrences are shown in **(Figs 1. 3 and 1.4)**

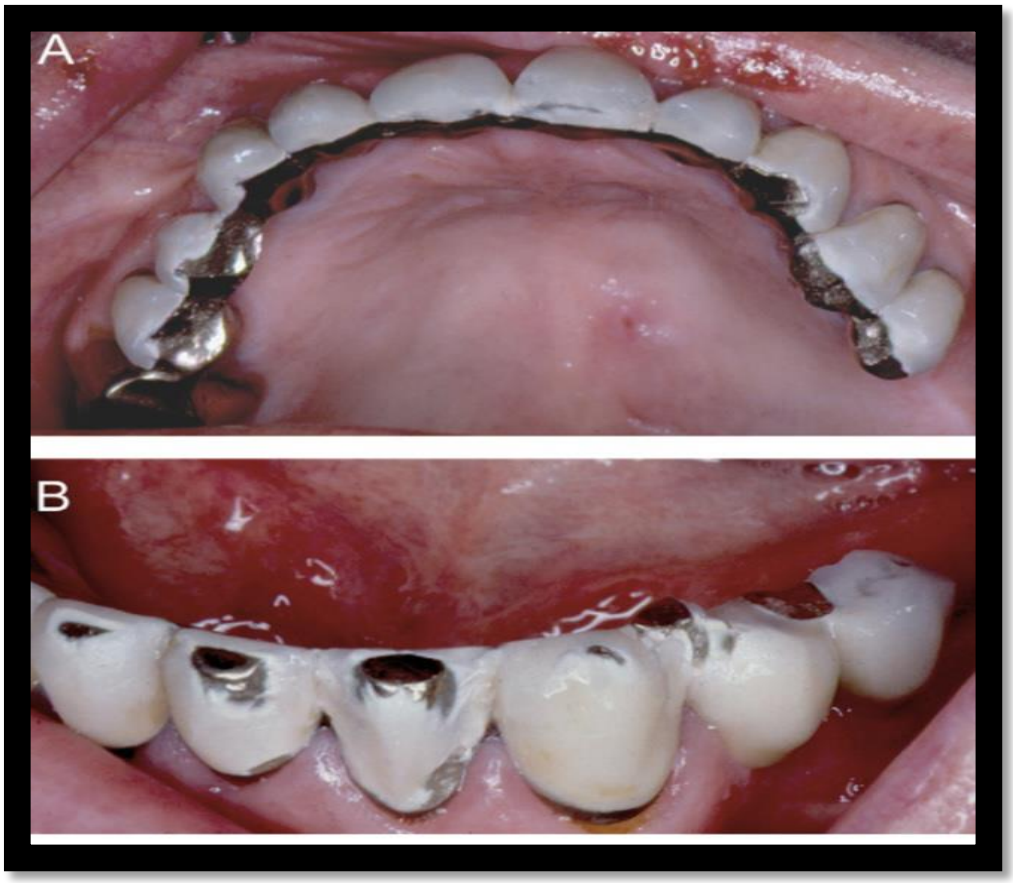


Fig.(1.3) (A and B)Extensive porcelain fractures (metal–ceramic fixed dental prostheses (FDPs) in both maxillary and mandibular jaws.
(Johansson, 2011).

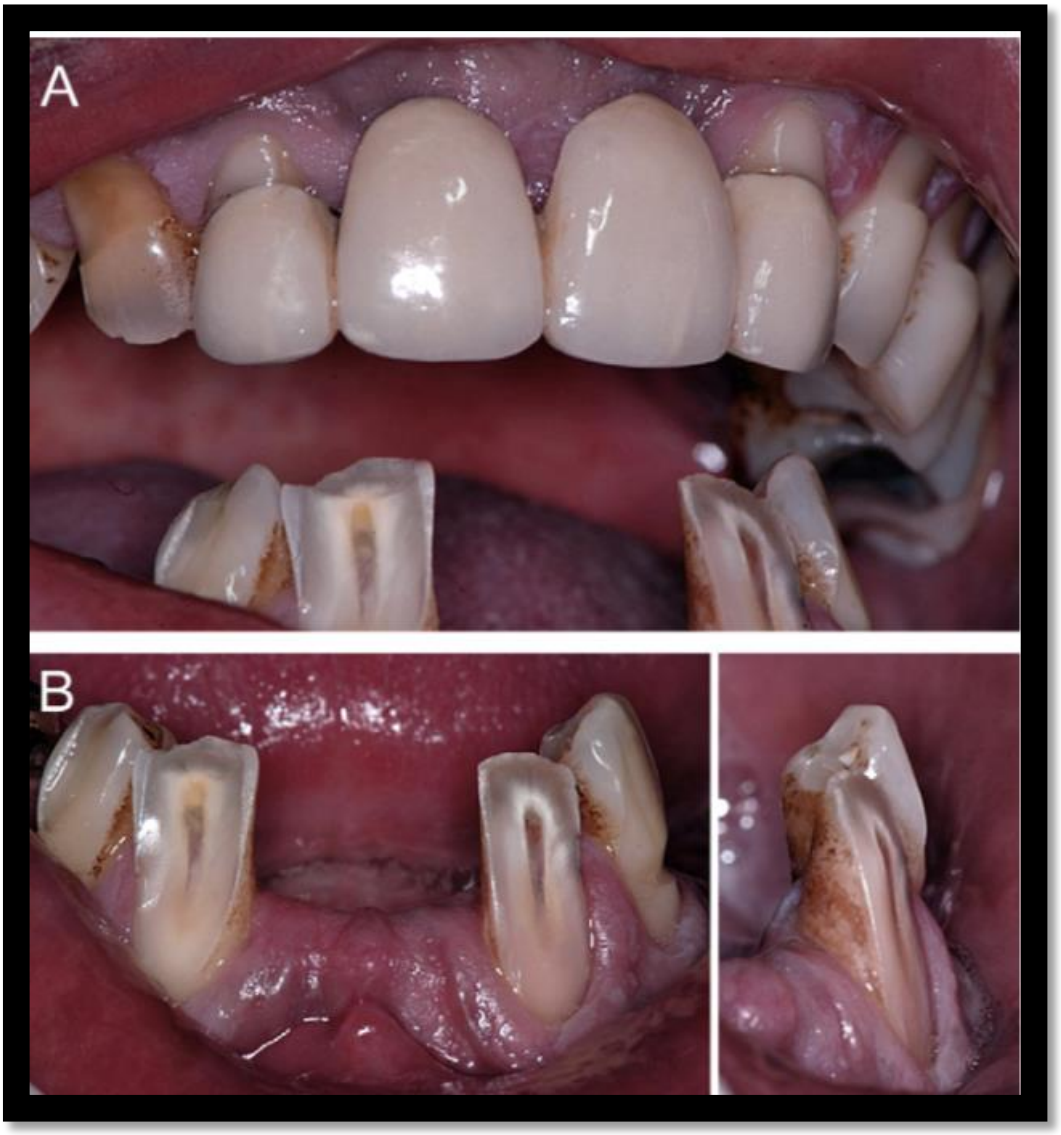


Fig.1.4 (A and B) severe lower anterior tooth wear caused by a combination of different factors (Johansson, 2011).

The process of wear that affects restorative materials is almost always studied experi-mentally in laboratory trials. Results are then extrapolated to the extremely variable intraoral conditions, whereas only long-term clinical investigations can demonstrate the true outcome **(Bayne, 2007).**

With an opposing occlusion of tooth enamel, most clinicians and researchers agree that a metal occlusal surface, and preferably one of high noble content. Is preferred in order to minimize wear of the natural dentition

Unpolished ceramics could be especially hazardous to opposing natural teeth. It is also necessary to consider other factors which influence the wear resistance of natural teeth, salivary secretory and lubricatory factors, among others **(Yip, 2004)**.

In cases of heavy occlusal load such as, for example, in bruxers, the situation becomes very complex as we need to consider not only the risk for wear of the restorative material itself and the opposing dentition, but also the need for sufficient strength in all the components of the superstructure to be able to withstand the applied load **(Yip, 2004)**.

Besides the risk of mechanical failures and loss of retention under conditions of excessive load, biological failures are even more likely, e.g caries, marginal degradation, and endodontic problems **(Yip, 2004)**.

The sequence of these events may be difficult to determine, and it may be that loss of retention occurs first and is then followed by caries and the other biological problems **(Karlsson, 1989).**

All things considered, metal or metal–ceramic restorations seem to be the safest choice in cases of high load conditions (**Dahl ,1996).**

Although under extreme conditions, there is no material that will last for too long (**Figs. 1.5and 1.6).**

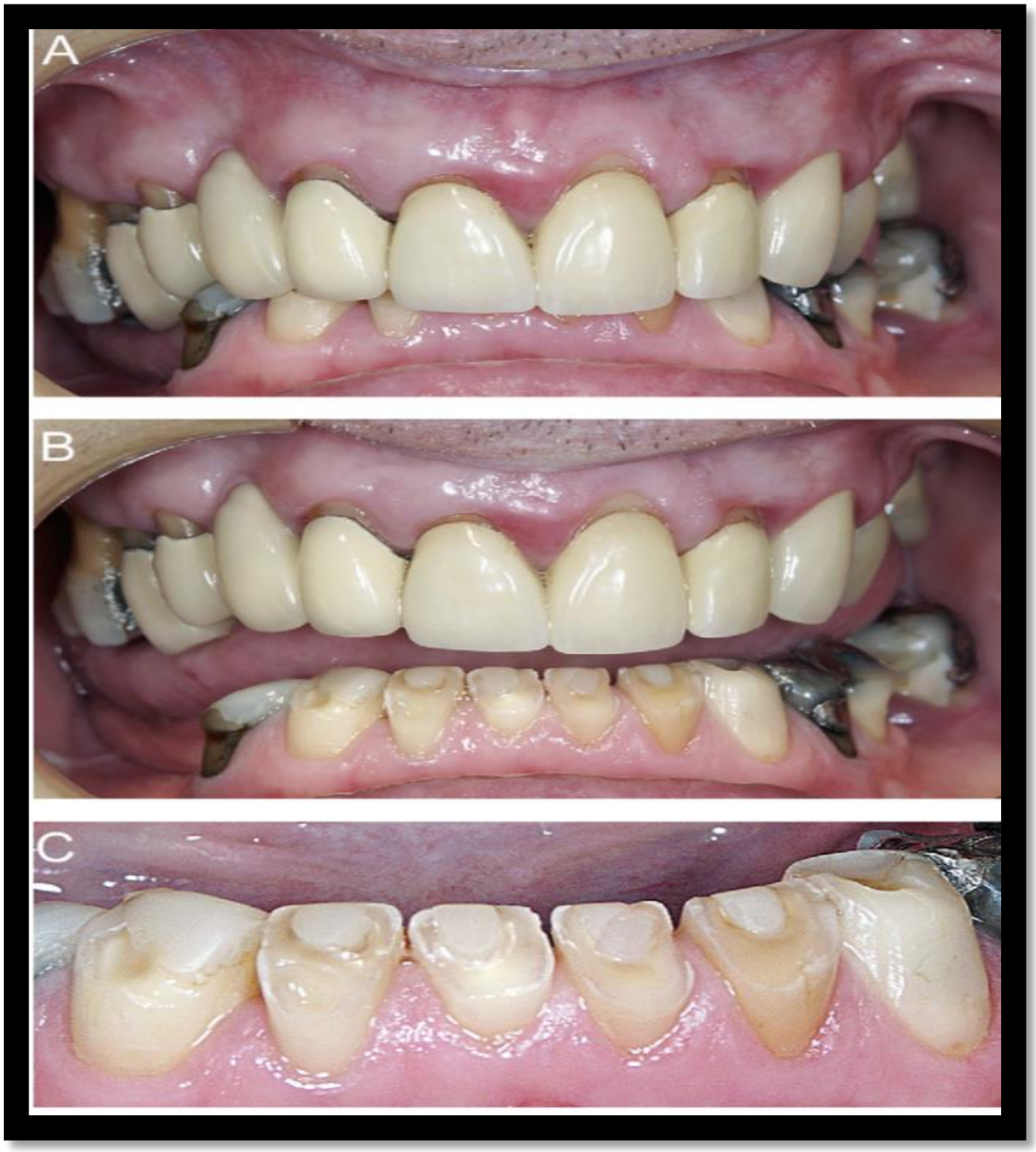


Fig. 1.5 (A, B and C) maxillary metal–ceramic crowns , a deep bite and excessive wear seen on the mandibular incisors (Johansson, 2011).

55-year-old man with maxillary metal–ceramic crowns and a deep bite. Heavy load due to bruxism and an absence of posterior support, opposing porcelain crowns, in combination with dental erosion have most likely contributed to the excessive wear seen on the mandibular incisors**.**

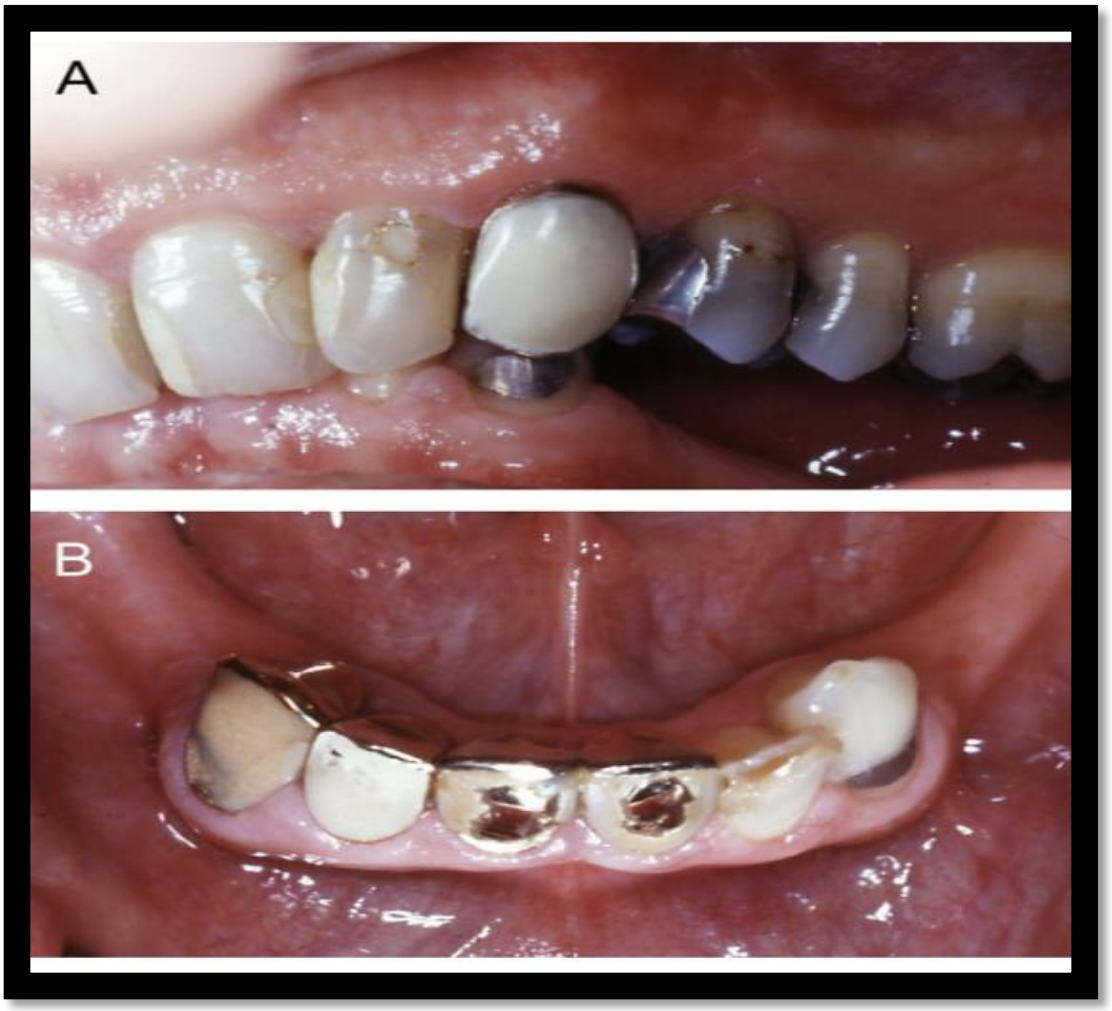


Fig (1.6) Aand (Aand B)Wear of metal crown veneered With acrylic Opposing natural teeth (Johansson, 2011).

Because of the risk of chipping of ceramic veneers in metal–ceramic restorations, many clinicians prefer gold– acrylic FDPs for heavy bruxers. The few clinical studies published on wear of materials in bruxers indicate only small differences in wear resistance of gold and ceramic materials, whereas resin-based materials showed 3–4 times more substance loss than gold or ceramics **(Dahl , 1996).**

During the last few years, new ceramics, for example zirconia, have demonstrated improved mechanical properties in laboratory studies and may be promising in the treatment of Bruxism related tooth wear (**Vagkopoulou 2009).**

However, a systematic review of zirconia FDPs has shown that there are complications when the material meets clinical reality. Improvement of the veneering systems is especially required as chipping was the most frequent mechanical complication **(Schley , 2010)**.

1.8. Effects of bruxism on implant restorations

Early papers on survival of fixed prostheses on osseointegrated implants often referred to bruxism and heavy occlusal loading as the cause of implant failures But, inprospective 15- year follow-up study of mandibular implant supported fixed prostheses, smoking and poor oral hygiene had a significant influence on bone loss, while occlusal loading factors such as bruxism, maximal bite force and length of cantilevers were of minor importance **(Lindquist, 1996).**

Further, a study using occlusal wear as a proxy for bruxism, gave no indication that implants in patients with occlusal wear have an increased rate of bone loss or higher Periotest value **(Engel, 2001).**

Systematic reviews have concluded that a causative relationship between occlusal forces and loss of osseointegration has never been demonstrated **(Carlsson, 2009).**

Although bruxism was included among risk factors, and was associated with increased mechanical and/or technical complications, it had no impact on implant survival **(Salvi, 2009).**

However, several studies have indicated that patients with bruxism have a higher incidence of complications on the superstructures of both of fixed and removable implant-supported restorations **.(De Boever, 2006).**

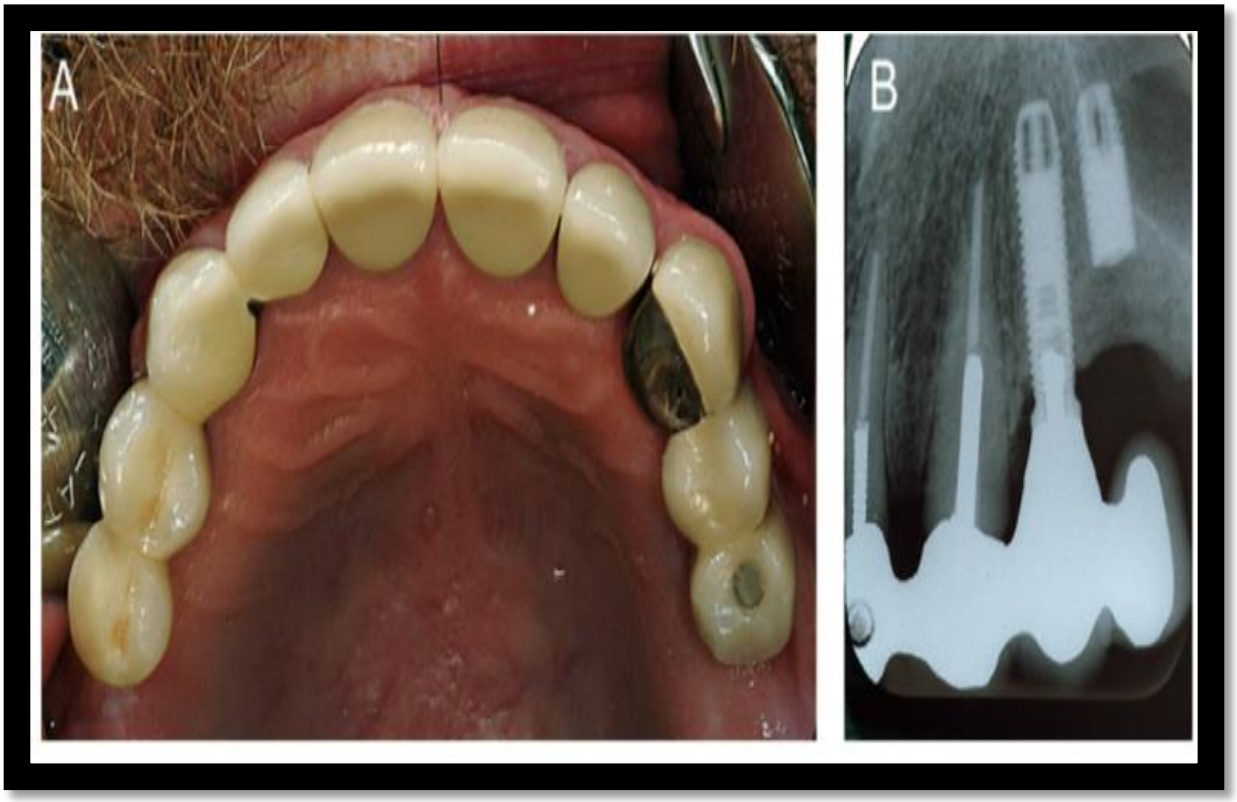


Fig. 1.7 implant fracture A-clinical picture ,B- peri apical X-ray (Johansson, 2011).

Chapter two

2.1 Conclusions

1. The etiology of bruxism is not well known, but it is agreed that it is multifactorial.
2. There is no specific treatment available at this time to stop bruxism, so that the focus has been to reduce the adverse effects of the habit.
3. The use of interocclusal appliances is the most common and accepted way to prevent wear of teeth and prosthodontic restorations in spite of lack of strong evidence for its efficacy.
4. The role of bruxism in the multifactorial process of tooth wear is not clear, but it is in general not the major cause.
5. The force of untreated Bruxism may cause failure of implant.

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