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EFFECT OF OCCLUSAL SPLINT THERAPY ON THE TEMPORALIS MUSCLE ACTIVITY DURING SLEEP BRUXISM

PhD Doctoral Thesis

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ABSTRACT

The aim of our study was to investigate the effect of the short term occlusal appliances therapy on the nocturnal electromyographic activity of the temporalis muscle during Sleep bruxism. This study performed on 37 patients Over 18 years of age that had sleep bruxism. Subjects varied in age between 21 and 37 years with a mean age of 28.29±5.16 years, 12 (38.7 %) females and 19 (61.3 %) male. However, 6 subjects were excluded from the statistical analysis because there is loss of electrode skin contact. The final statistical sample of subjects was reduced to 31 patients were divided into two groups; Treatment group 20 patients (64.5 %), and untreated control group 11 patients (35.5 %). The subjects are selected on the basis of questionnaire and clinical signs and the activity of bruxism was confirmed with acid etching technique and by using portable EMG recording device. For all subjects in treatment group, a full arch heat cure acrylic resin interocclusal appliance covering all of the mandibular teeth were fabricated on the mandibular casts with 3 mm thickness of acrylic between the maxillary and mandibular posterior teeth. All interocclusal appliance have bilateral anterior-posterior simultaneous contacts in centric relation with anterior and canine guidance in lateral and protrusive excursions. Electromyographic activity during sleep was recorded from the right side of anterior temporalis muscle with portable Electromyographic recording device. The patients were instructed to wear it every night over the 12 days of the study. During the first 6 consecutive nights, all patients used the device during sleep without the occlusal appliance to record the pre-treatment baseline data (number of EMG events/hour). During the next 6 consecutive nights, all patients in treatment group used the device during sleep with the occlusal appliance in, to record the treatment data (number of EMG events/hour). Our result showed a significant reduction in the EMG activity of temporalis muscles during sleep with occlusal appliances in situ. An individual variation in changes of sleep bruxism activities by wearing splints was observed in this study; 13 of 20 subjects showed statistical significant reduction in the sleep bruxism activity, 3 of 20 subjects showed reduction in the sleep bruxism activity however this reduction was not statistical significant, and 2 of 20 subject showed no difference between pre-treatment and post treatment sleep bruxism activity. On the contrary, 2 subjects showed increase in sleep bruxism activities while using occlusal appliance as compared to baseline data. From our study, we could conclude that, that the occlusal appliance

should be regarded as a management for rapidly relaxing masticatory muscles. However since some individual variations have been observe, the splint is not always effective in all bruxism patients, and the clinicians should always monitor the signs and symptoms reported by the patient when using an oral device for the management of sleep bruxism

Keywords: Bruxism, occlusal appliance, electromyography, masticatory muscle activity.

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TABLE OF CONTENTS

LIST OF ABBREVIATIONS

TMJ	Temporomandibular joint
TMD	Temporomandibular Joint Disorders
EMG	Electromyographic
GPT	Glossary of prosthodontic terms
MMA	Masticatory muscle activity
RMMA	Rhythmic masticatory muscle activity
EEG	Electroencephalograph
SB	Sleep bruxism
REM	Rapid eye movement
PLM	Periodic limb movement
MVC	Maximum voluntary contraction
AASM	American Academy of Sleep Medicine
PSG	Polysomnography
CNS	Central nervous system
NTI	Nociceptive trigeminal inhibition
MAD	Mandibular advancement devices

LIST OF TABLES

LIST OF FIGURES

1. BACKGROUND AND OBJECTIVES

The stomatognathic system consists of three main components; temporomandibular (TMJ) components, masticatory muscles, and dental occlusion. These components are interrelated and coordinated by the central nervous system. If one of the components is abnormal, the stomatognathic system will not be able to do its function properly.

In order to understanding of the masticatory system, many studies have attempted to analyze the normal functional, and parafunctional behaviour of the masticatory muscles. Parafunctional habits refer to oral habit other than common functions of chewing, swallowing and speaking. These habits may overload the temporomandibular joint and masticatory muscles, and contribute to TMJ dysfunction. They may include Bruxism (tooth-clenching or grinding), Tongue Thrusting, Mouth Breathing, Thumb sucking and biting objects.

Bruxism is an oral parafunctional habit involving clenching and grinding of the teeth that occurs mainly unconsciously, diurnally and nocturnally. Bruxism is considered to be one of the most significant parafunctional activities of the stomatognathic system (1). It is considered an important contributory factor in the etiology of temporomandibular disorders (TMDs). The incidence of bruxism is conservatively estimated to be between 5% and 10% of the adult population (2). The etiology of bruxism is suggested to be multifactorial, involving morphological, pathophysiological and psychological factors (3,4).

Many patients clench or grind their teeth are not aware of the activity when it occurs during sleep because there is no discomfort or pain. They remain ignorant of the tooth wear associated with non-functional clenching/grinding unless observed by a dentist during a consultation. The patients with uncomfortable symptoms like jaw-muscle pain or stiffness, headache, difficult mouth opening, increased tooth sensitivity usually consult the dentist who will diagnosis the problems and prescribe the use of an occlusal appliance to be worn during sleep.

Although the use of an occlusal appliance is effective for the prevention of further tooth wear during sleep, it does not necessarily eliminate the unconscious clenching/grinding activity nor the accompanying pain or discomfort (5).

The efficacy of the occlusal appliance to reduce masticatory muscle activity and overall response to occlusal appliance treatment remains questionable (6), some study reported to decrease masseter muscle activity in more than 50% of bruxism patients (6). Conversely, some studies have shown increased electromyographic (EMG) activity (7) and other studies have shown a variation in response with EMG levels decreased in 52%, increased in 20%, and unchanged in 28%, of the patients (8).

Aims of the study:

The literature review identified a need for further study on the effect of occlusal appliance therapy on the activity of muscle of mastication, The aim of this study is to compare the effect of occlusal appliance therapy on the electromyographic activity of temporalis muscle, before and after the application of the occlusal appliance for sleep bruxer patients using a portable EMG recording system.

2. INTRODUCTION

The term bruxism comes from the Old Greek word brychein- the grinding of teeth. In scientific and scholarly writings, the term bruxism was first used by Marie and Pietkiewitz in 1907. Not long before that, Black had described abnormal enamel wear connected with non-functional activity and Karoly had referred to the state as neuralgia traumatica. Other researchers spoke about Karoly's effect (Weski) and the neurosis of occlusal habits (Tishler). Miller makes a distinction between nocturnal gnashing-bruxism, and diurnal gnashing, which he calls bruxomania (4).

2.1. Definition of bruxism:

There is no consensus about the definition of bruxism (9), but there are many definitions to the Bruxism include:

- According to the fourth edition of the Oro-facial Pain Guidelines, published by the American Academy of Oro-facial Pain (10), Bruxism is a diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing and grinding of the teeth.
- American Sleep Disorders Association (11) defines Bruxism as "tooth grinding or clenching during sleep plus one of the following: Tooth wear, sounds or jaw muscle discomfort which cannot be attributed to any other disorder."
- According to glossary of prosthodontic terms (GPT) (12), bruxism is "parafunctional grinding of the teeth (or). An oral habit consisting of involuntary rhythmic or spasmodic nonfunctional gnashing, grinding or clenching of teeth, in other than chewing movements of the mandible, this may lead to occlusal trauma".

Bruxism according to the etiology is primary and secondary. In the absence of a medical cause, primary forms of bruxism include daytime clenching and Sleep Bruxism. Secondary forms of bruxism are associated with either neurologic, psychiatric or sleep disorders, or withdrawal of drugs (13).

Bruxism occurring during wakefulness should be differentiated from Sleep Bruxism, because the two conditions occur under different circumstances and have been considered different entities (14).

2.2. Epidemiology

Data on the prevalence of bruxism vary because of the use of different research methods, working definitions, clinical criteria and population samples. The prevalence of awake bruxism in the general population is approximately 20%, while the prevalence of sleep bruxism is about8% (15,16). Complaints of tooth grinding occurring during sleep decline over time, from 14% in children to 8%in adults to 3% in patients over 60 years of age (15,17).

The data regarding the distribution between the sexes vary. Some research studies show higher prevalence of parafunctions in women, while other studies deny any differences in the prevalence of the symptoms of bruxism between the sexes (18).

Other studies demonstrate that bruxism occurs in up to 90% in the general population. Therefore, determination of the actual prevalence of bruxism is difficult because this mandibular parafunctional behavior is performed at a subconscious level by most individuals. Because of this limitation, mostly an individual is dependent on family member or roommate or bed partner to ascertain by the sounds that can be generated from clenches or grinds the dentition (19,20). However, the validity of bruxism grinding sounds showed sensitivity of 78% and with specificity of 94% when it compared with polysomnography (21).

2.3. Etiology of bruxism

The etiology of bruxism behavior is, to a great part, unknown and controversial and many theories to explain it have been developed. Etiology is probably made up of many factors which overlapped, the factors involving anatomical-morphological, psychophysiological, pathophysiological factors, and other causes which are illustrated in Table 1 (22).

Table 1. Causes of bruxism

1. Anatomical-morphological factors
   Dental occlusion anomalies (malocclusion)
   - Various morphological types of malocclusion.
   - Functional malocclusion.

   Anomalies of the oro-facial region
   - Condyles height asymmetry.
   - Larger cranial and bizygomatic widths.
   - Rectangular form of dental arch of maxilla.
   - Rectangular morphology of face.
2. Psychophysiological factors
   - Stress (emotional, physical, psychosocial).
   - Anxiety (states of anxiety, psychosocial).
   - Emotional disorders.
   - Psychosomatic disorders.
   - Personality disorders (hyperactivity, rage, aggressiveness, perfectionist tendency).
3. Pathophysiological factors
   - sleep disorders
     - Poor quality of sleep.
     - Micro arousal episodes (short awakening).
     - Frequent movements of body.
     - Behaviour disorders during REM sleep.
     - Periodic movements of feet.
     - Sleep apnea syndrome.
     - Sleep epilepsy.
     - Sleepiness during the day.
   - Rhythmic muscular activity of masseter muscles.
   - Genetic predisposition.
   - Sensitivity disorders of central dopaminergic neurotransmission.
4. Other causes
   - Allergy (allergic rhinitis, bronchial asthma, swallowing of allergenic foods).
   - Hemifacial spasm.
   - Various syndromes (Gilles de la Tourette S., Rett S., Shy-Drager S.).
   - Neurological disease (brain haemorrhage, coma, Huntington disease, Parkinson disease).
   - Drug intake.
     - Antidepressants (SSRI) (Fluoxetine, sestraline, paroxetine).
     - Chronic use of neuroleptic and levodopa.
     - Amphetamine and analogous drugs (OCT).
   - Smoke and alcohol abuse.

2.3.1. Anatomical-Morphological factors

Initially, morphological factors, like occlusal discrepancies and deviations in the anatomy of the bony structures of the oro-facial region, have been considered the main causative factors for bruxism. More recently, these factors are thought to play only a small role, if at all present (23).

The occlusion concept was popularized in a classical article by Ramfjord in 1961(24), and later studies supported this concept, as occlusal corrections were reported to diminish or stop this sleep activity (25). Although Ramfjord also saw a role for `neurotic tensions' in the etiology of bruxism, he held certain occlusal characteristics mainly responsible for the initiation of the disorder. Especially discrepancies between retruded contact position and intercuspal position, and also the presence of mediotrusive (balancing side) contacts during articulation were thought to be involved in the etiology of bruxism. Ramfjord reported that occlusal adjustments (grinding procedures) always led to a disappearance of bruxism without any supported study other.

Rugh et al. in1984 (26) studied the influences of artificial occlusal interferences, incorporated in crowns in the molar region, on masticatory muscle activity (MMA) during sleep. The MMA was quantified by means of EMG recordings from the sleeping patient. In contrast with the Ramfjord hypothesis, artificial interferences caused a significant decrease of sleep related MMA in 90% of the cases.

In other studies, the elimination of interferences in occlusion and articulation was shown to have no influence on bruxism activities (27,28). Moreover, not every bruxer has occlusal interferences and not every person with such interferences is a bruxer (29). Therefore, there is no scientific proof for a role of occlusion and articulation in the etiology of bruxism (23).

Two studies have examined the possible relationship between bruxism and the anatomy of the oro-facial anatomical structures. Miller et al (30) examine the relationship between condylar asymmetry and parafunction in patients with temporomandibular disorders and parafunction and patients with temporomandibular disorders but without parafunction. He found a more pronounced asymmetry in condylar height in bruxers as compared with non-bruxers. Young et al (31) compare the craniofacial morphologies of bruxer and non-bruxers, he observed larger bizygomatic and cranial widths in bruxers. In both these studies, the presence or absence of bruxism, is assessed by self-report and a clinical examination, however it was not confirmed by

polysomnography, which can affect their results.

Menapace et al. (32) compare dentofacial morphology of bruxers with non-bruxers and found no differences, The absence of a polysomnography confirmation of the non-bruxers status also can affect their results. In another study by Waltimo et al (33) that primarily focused on tooth wear in relation to the morphology of the craniofacial structures, a more rectangular form of the maxillary dental arch was found in patients with severe dental attrition than in control subjects. In addition, these authors found that patients with severe attrition had a more rectangular facial morphology than controls, in combination with an anteriorly rotated mandible, a small anterior facial height and a large bimaxillary interincisal angle. Again, they did not use polysomnography to classify their patients.

Only one controlled study to the relationship between bruxism and morphological factors was performed with the use of polysomnography to confirm or refute the presence of bruxism. In that study, Lobbezoo et al. (34) compared 26 occlusal variables and 25 cephalometric variables between bruxers and non bruxers and found no differences between both groups.

Therefore, there is no proof for a role of factors related to the anatomy of the oro-facial skeleton in the etiology of bruxism.

2.3.2. Pathophysiological factors

Pathophysiological factors are suggested to be involved in the precipitation of bruxism, For example, bruxism has been linked to sleep disturbances, altered brain chemistry, the use of certain medications and illicit drugs, smoking, the consumption of alcohol, and certain traumata and diseases (4).

2.3.2.1. Sleep disturbances

An arousal response is a sudden change in the depth of sleep, during which the individual either arrives in a lighter sleep stage or actually wakes up(35). Such a response is accompanied by gross body movements (e.g. turning), the appearance of K complexes in the electroencephalograph (EEG) (single, biphasic potentials with relatively large amplitude), an increased heart rate, respiratory changes, peripheral vasoconstrictions and increased muscle activities. Macaluso et al. showed that in 86% of cases, bruxism episodes were associated with an arousal response (36). Besides the above mentioned characteristics of an arousal response, involuntary leg movements

were present in association with about 80% of the bruxism episodes. These observations suggest that bruxism is part of an arousal response. Therefore, bruxism can be classified among the parasomnias, a group of sleep disturbances that also includes sleepwalking, nightmares, sleep talking and enuresis (37).

2.3.2.2.Neurochemical factors

Certain disturbances in the central neurotransmitter system may be involved in the etiology of bruxism (34,38,39). It can be hypothesized that the balance between the direct and indirect pathways of the basal ganglia (a group of five subcortical nuclei that are involved in the coordination of movements) is disturbed in bruxers (18). The direct output pathway goes directly from the striatum (one of the five basal ganglia) to the thalamus, from where afferent signals project to the cerebral cortex. The indirect pathway, on the other hand, passes by several other nuclei before the thalamus is being reached. If there is an imbalance between both pathways, movement disorders are the result, like Parkinson's disease (40). The cause of such an imbalance can be found in the `nigrostriatal projection', a feedback loop within the complex of nuclei that constitute the basal ganglia. The imbalance goes with disturbances in the dopamine-mediated transmission of action potentials. In case of actual nigrostriatal degeneration, Parkinson's disease emerges because of a lack of endogenous dopamine, which can be influenced by pharmacological therapy (e.g. dopamine precursors, dopamine agonists). In case of bruxism, there may be an imbalance between both output pathways as well, however, without signs of degeneration of the nigrostriatal feedback loop. The acute (short-term) use of L-dopa, a dopamine precursor (38), and of bromocriptine, a D2 receptor agonist (39), inhibits bruxism activity in polysomnography studies. The chronic (long-term) use of L-dopa by Parkinson patients is known to cause bruxism (41). Similarly, the chronic use of neuroleptics by psychiatric patients gives rise to bruxism during wakefulness (42). Also medications that exert an indirect influence on the dopaminergic system, like selective serotonine reuptake inhibitors (34) may cause bruxism after long-term usage. It appears that there maybe two types of bruxism: an idiopathic type that can be suppressed by a short term treatment with dopamine agonists, and an iatrogenic type that is caused by the long-term application of several dopaminergic medicines. The only study so far that complicates this view is a recent one by Lavigne et al. (43), in which bruxism could not be influenced by the acute use of bromocriptine. A possible explanation for this deviant finding may be the fact that

bromocriptine was combined with domperidone to suppress peripheral side effects.

Tooth grinding that can be observed in relation to the abuse of amphetamine (44) (a substance that increases the dopamine concentration by facilitating its release) can be classified as iatrogenic bruxism in association with dopamine as well. The drug XTC (an amphetamine-like substance) has recently been associated with excessive tooth wear (45). Also nicotine stimulates the central dopaminergic activities, which might explain the finding that smokers of cigarettes report bruxism almost two times more than non-smokers (46) and that smokers show about five times more bruxism episodes per night than non-smokers (46) ,Also alcohol could lead to bruxism (47).

Gomez et al. (48) and Areso et al. (49) investigate the role of occlusal disharmonies on the alterations in central dopaminergic neurotransmission in experimentally induced bruxism in the rat, they found that an acrylic cap on the mandibular central incisors that was worn for a prolonged period of time resulted in an imbalance between hemispheres in dopa accumulation in the basal ganglia. However, it is difficult to compare artificial occlusal interferences in experimentally bruxer rats with `natural' ones in patients with sleep related bruxism.

Lobbezoo et al. (34) were not able demonstrate a similar phenomenon in man: no significant correlations were found between morphological factors and asymmetries in striatal D2 receptor expression in a group of patients with polysomnography confirmed sleep-related bruxism.

2.3.2.3.Genetic predisposition

Some clinicians find that bruxism runs in families. Hublin et al. (50) demonstrated in a questionnaire study with about 4000 twin pairs that the contribution of heredity to bruxism varies from 39 to 64%. In contrast, in a combined questionnaire and clinical study with almost 250 pairs of twins, Michalowicz et al. (51) concluded that there is no such contribution. Therefore, whether or not bruxism is more or less genetically determined remains unclear.

2.3.3. Psychological factors

Ramfjord (24) show a role for `neurotic tensions' in the etiology of bruxism. Stress and personality have been already implicated in the etiology of bruxism for many years. However, the exact contribution of these psychological factors to this etiology remains a subject of debate.

A questionnaire study by Olkinuora (52) demonstrated that bruxers can be considered emotionally out of balance and that they tend to develop more psychosomatic disorders. Their personalities would be characterized by perfectionism and an increased tendency towards anger and aggression. These findings were later confirmed by Kampe et al. (53), who also demonstrated more anxiety in a group of bruxers. An increased amount of aggression and somatization can already be found in bruxer 5- and 6-year olds (54). However, the use of a specific questionnaire for the amount of psychological disturbance, indicated that bruxers do not differ from non-bruxers within a population of patients with facial pain (55).

The EMG or polysomnography has been rarely used in the study as to the role of psychological factors in the etiology of bruxism. In EMG case study described by Rugh and Robbins (56),during a 6 month period, they continuously recorded the masticatory EMG activity of a young woman. In times of increased stress caused by, for example, exams and fights with her partner, she developed an increase in her sleep related MMA. However, it is still a case, and this relationship is less obvious in groups of bruxers.

Pierce et al. (57) investigate the relation between stress and bruxism, These authors investigated in 100 bruxers the amount of self-reported stress in relation to electromyographic recording taken from bruxer during the night before the stress report (anticipatory stress) and the night following the report (current stress). A total of 15 nights was recorded. For both anticipatory stress and current stress, significant correlations with bruxism were found in eight individuals only. For the entire sample, no association between stress and bruxism could be demonstrated.

Finally, in a polysomnographical study to vigilance and reaction time, an increased level of anxiety was found in sleep bruxers (58). Anxiety was the only psychological outcome variable to reach statistical significance in this study.

The role of psychological factors in the etiology of bruxism is not clear. It appears that this role differs between individuals and is probably smaller than previously assumed (47).

2.4. Classification of bruxism

Bruxism may be classified according to several criteria (59):

1. By when it occurs:
    a. Awake bruxism: This is presented when the individual is awake.
    b. Sleep bruxism (SB): This is presented when the individual is asleep.
    c. Combined bruxism: This is presented in both situations.
2. By etiology:
    a. Primary or idiopathic bruxism: For which no apparent cause is known.
    b. Secondary bruxism: Secondary to diseases (coma, ictus, cerebral palsy), medicinal products (e.g., antipsychotic medication, cardio-active medication), drugs (e.g., amphetamines, cocaine, ecstasy).
3. By motor activity type (Fig 1):
    a. Tonic.
    b. Phasic.
    c. Combined.
4. By current or past presence:
    a. Past bruxism.
    b. Current or present bruxism.

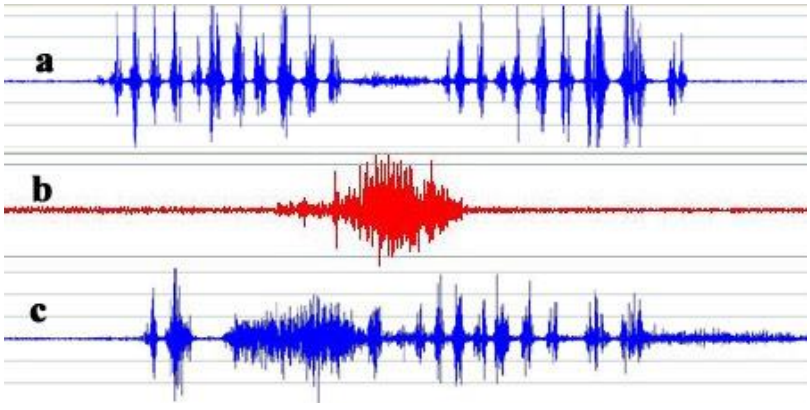


Fig 1: Types of bruxism episodes: a) phasic. b) Tonic. b) Mixed.

2.5. Sleep bruxism

Sleep bruxism is a parasomnia and an oral parafunctional activity during sleep that is characterized by either jaw clenching (tonic activity) and/or repetitive phasic jaw muscle activity that produces tooth grinding (60).

Sleep bruxism is a common disorder. Its prevalence has been reported in questionnaire studies to be the highest in childhood (about 14%) and decreases from about 8% in younger adults to about 3% in elderly people (15,17).

In awake individuals, bruxism is an involuntary activity of the jaw musculature that is characterized, by jaw clenching and, on rare occasions, by tooth gnashing and/or grinding. During sleep bruxism, both clenching and tooth grinding are observed. sleep bruxism can cause tooth destruction, temporomandibular dysfunction (e.g., jaw pain or movement limitation), occasional headaches, and the disruption of the bed partner's sleep due to the grinding sounds (4,13). Sleep bruxism can occur alone or concomitantly with awake bruxism. Some patients complain of jaw tightness and grinding sounds daily or nightly, whereas others report these infrequently (13).

The jaw elevator muscles commonly exhibit three basic patterns of activity during sleep bruxism (61):

- Phasic: rhythmic, chewing like movements, which are repetitive (three or more bursts of muscle contractions of 0.25 to 2 second of duration) (62).
- Tonic: prolonged, strong, isotonic contractions of the jaw closing muscles (contractions lasting more than 2 second) (62).
- Mixture of both types (21,62).

An analysis of electromyographic studies (EMG), shows that 52.5% of sleep bruxism episodes are phasic, 11.4% tonic and 36.1% are mixed type (21). Approximately 89% of the episodes of SB are phasic or combined, unlike in awake bruxism, where episodes are predominantly tonic.

The number and duration of bruxing event during sleep vary greatly with the same person over time and among the persons, sometimes even on a day-today basis, with subjects showing no activity during some nights and intense activity during others (63). In cases of severe and frequent sleep bruxism, the variation of the number of episodes per hour of sleep is 25%, and the variation of tooth-grinding frequency is 53.5% (63).

In normal subjects during sleep, nearly 60% show rhythmic masticatory muscle activity (RMMA), this occurs at a frequency of 1.8 episodes per hour of sleep. This is three times lower than in sleep bruxism patients, the muscle contractions are of lower amplitude, and no tooth-grinding sound is reported (64). In sleep bruxism, jaw muscle activity mainly occurs during light sleep (60-80%) at a mean frequency of 5.4 to 5.8 episodes per hour of sleep (36,65).

2.6. Sleep Physiology

In human adults, sleep is divided into two major types occurring in 3 to 6 cycles at an interval of 60-90 minutes: (1) Non-rapid eye movement (non- REM) sleep ('quiet' sleep) that includes light sleep (stages 1and 2) and deep sleep (stages 3 and 4 or delta sleep); and (2) REM sleep ('active' or 'paradoxical' sleep). The first third of the sleep period is characterized by a dominance of deep sleep (delta sleep), while the last period toward morning is characterized by a dominance of REM sleep. As humans pass from wakefulness to deep sleep, brain electrical (electroencephalographic, EEG) activity slows, and autonomic cardiac sympathetic activity (measured by spectral analysis of heart rate interval in the low frequency 0.05-0.15 Hz range) diminishes. During sleep stage 2, EEG traces are also characterized by EEG K-complexes (brief bipolar EEG waves) that occur from one to five times per minute and represent a cortical response to exogenous (e.g., sound) or endogenous (e.g., change in blood pressure) events. These events are clearly associated with periodic limb movement (PLM) during sleep (66) but not with sleep bruxism.

2.7. Physiology of Rhythmic Movements

Chewing is a repetitive motor activity, which like respiration, is driven by cellular networks within the central nervous system (67,68). These networks are termed the central pattern generator (CPG). They are organized to initiate and maintain the motor activity integration of influences from sensory inputs, such as those from muscle spindles, joint, mucosal, and periodontal receptors, is necessary to control or fine-tune rhythmic jaw movements (69,70).

2.8. Rhythmic masticatory muscle activity (RMMA) and sleep bruxism, and sleep architecture:

Sleep organization in sleep bruxism patients and in non- bruxism subjects with RMMA is usually normal in terms of sleep duration, sleep efficiency (The proportion of how much time subject spend asleep over time in spend bed), and sleep stage distributions (21,64). Most sleep bruxism episodes (60-80%) occur in light non-REM sleep (36,65). The presence of high amplitude EEG positive and negative signals, termed K-complexes, is considered a marker of transient EEG activation. It has been previously associated with sleep bruxism (71,72). However, in a recent analysis comparing sleep bruxism and controls, it was noted that sleep bruxism patients had significantly fewer K-EEG events than normal (73).

Sleep micro-arousal is an unconscious and transient 3 to 10 or 15 second burst of brain EEG activity, alone or sometimes with an increase in heart rate and muscle tone. Although the incidence of microarousal (number of events/hr of sleep) is moderately correlated with the high frequency of masticatory muscle activity, it is the magnitude(e.g., a more rapid onset in heart rate, a bigger rise in EMG activity, and a forceful tooth contact with grinding) of microarousal that seems to distinguish sleep bruxism patients from normal (36,64,71,74). This is consistent with the observation that most sleep bruxism episodes occur in a period of intense EEG and autonomic activation that has been termed a 'cyclic alternating period' (CAP) (36,75,76). Interestingly, when sleep bruxism episodes occur with an active CAP phase, very few K-complexes are observed (36).

An acceleration in cardiac rhythm, also a sign of an autonomic cardiac sleep arousal, has been previously observed in relation to sleep bruxism episodes (71,72,77,78).Although the rise in heart rate associated with sleep bruxism is important, it is not specific, since a similar increase (25%) is also observed with RMMA episodes in normal subjects (74).

However, in comparison with normal subjects, sleep bruxism patients do show a more rapid onset of heart rate (HR) increase (74). Although 90% of RMMA and sleep bruxism episodes have been observed in association with EEG and EMG related micro-arousal, a clear sequence of physiological activation occurs before the onset of 80% of these episodes. In the four-second period before RMMA, there is first a clear increase in the power of EEG activity, followed by a rise in heart rate. Again, this increase in heart

rate is not unique to RMMA and sleep bruxism, since it has been observed with another periodic motor manifestation during sleep, i.e., periodic limb movements (PLM) (4,79). The above evidence suggests that sleep bruxism episodes are closely related to the transient EEG cardiac and EMG activations that are part of sleep micro-arousal (4).

The proposal that sleep bruxism and RMMA are associated with sleep arousal (71) is further supported by the observation that tooth-grinding and RMMA can be evoked experimentally through manipulations that trigger microarousal (4,36,72).

In humans, auditory or photo-optic flash stimulation triggers tooth grinding episodes (71). In a study in which sleep arousal was induced by auditory or vibro-tactile stimuli, 11% of experimental sleep arousals were followed by RMMA, and 71% of these were associated with tooth-grinding in sleep bruxism patients (73,80).

2.9. Bruxism effect on stomatognathic system

The effects of bruxism on TMDs are based on a habit that can cause significant damage to every part of the masticatory system (62), but the exact role of bruxism in the etiology remains unclear.

2.9.1. Bruxism effect on masticatory muscle

Bruxism may have effect on muscles: masticatory muscle tenderness (62,81), and masticatory muscle hypertrophy (62,81).

In cases of protrusive (forward and backward) bruxism associated with grinding or jaw sliding between centric and eccentric positions, the lateral pterygoid muscle suffers a contraction under muscular lengthening during the slow condylar retrusion into the joint cavity. The same happens with the elevator muscles during laterotrusive bruxism (from right to left) or protrusive under grinding or jaw sliding between centric and eccentric positions. During this eccentric muscular work, the muscle develops different magnitudes of muscular tension (depending on the load torque) as it becomes larger. This is the position in which there may be greater probability of muscular damage (82).

The maximal masticatory force is about 60-70 kgf, during chewing and swallowing only a part of the maximal jaw force is used (about 30%), But during bruxism a force slightly less than or similar to the maximal force is developed. Also bruxers contract their masticatory muscles, specifically jaw elevator muscles, for longer periods than normal individuals (82). It is possible to conclude that the

magnitude and frequency of dental contacts during parafunctional activity will affect the structures of the stomatognathic system to a greater extent than during functional activity (82).

The main part of the functional activity (chewing swallowing, and phono-articulation) consists of a rhythmic pattern of alternating isotonic and isometric contractions, with relaxation pauses. This physiologic activity permits a good supply of oxygen to the muscles by aerobic glycolysis. Parafunctional activities, in contrast, consisting of eccentric contractions and, moreover, longer and more intense isometric contractions, which decrease the normal blood flow to the muscle leading to reduction of oxygen supply (82).

2.9.2. Effects on Soft Tissues

Linea Alba (Fig 2) is a whitish ridging or line which is parallel to the dental occlusion plane. It consists of hyperkeratinization of the cheek mucosal epithelium, and it will usually occur bilaterally (83).

Tongue indentation (Fig 3) this refers to the impressions made by the teeth on the both the upper and lower portions of the circumferential edge of the tongue (84).

Some authors consider that a scalloped tongue and the presence of linea alba are clear signs of bruxism (30,85–88).They are even considered to be the two most reliable clinical signs of active bruxism, and they are caused by the force exerted by soft tissues against the surfaces of the teeth. Some authors said that these signs are to regress once the parafunctional stops (89).

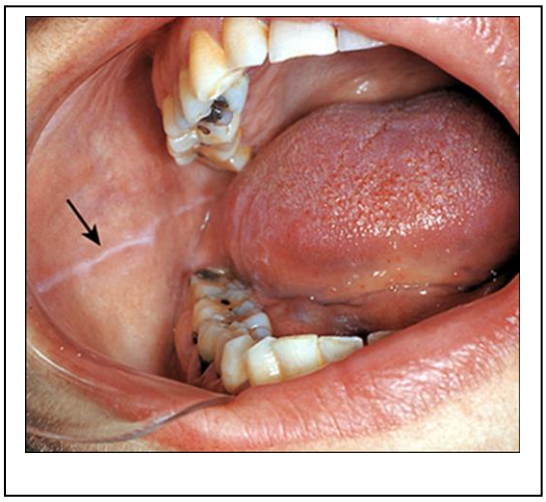


Fig 2: Linea Alba

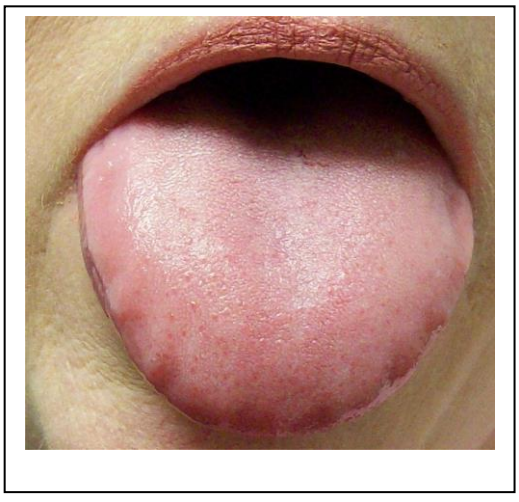


Fig 3: Tongue indentation

A study of a sample of individuals with a history of at least 5 years of active bruxism reported a linea Alba prevalence of 58.6% and a Tongue indentation prevalence of 41. 4 % (87).

Longer (88) suggested that tongue indentations is likely to be caused by a vacuum effect created between the tongue and the palate during sustained tooth clenching. He said that it is essential that lips be sealed in order for a vacuum to exist; from this, he concludes that without such a vacuum, no tooth clenching would be possible. He describes a device (a tube held between the lips extending out of the mouth to break the vacuum) that is intended to prevent the clenching of the teeth. It has not been scientifically validated so far (18).

Takagi and Sakurai (83) compared the pressure exerted by the cheeks of two groups of subjects; One group included subjects with linea Alba; the other group included subjects without linea Alba (control group). The recording tasks were: (i) silent reading at rest, (ii) light voluntary clenching, (iii) maximum voluntary clenching, (iv) holding the cheeks tightly against the teeth while light voluntary clenching, (v) holding the cheeks tightly against the teeth without tooth contact, (vi) pulling angle of mouth laterally while light voluntary clenching, (vii) pulling angle of mouth laterally without tooth contact and (viii) swallowing. No significant differences were found between groups in all the recording tasks except for the swallowing, at which significant difference was found. These forces were higher than the ones recorded during maximum tooth clenching, and were not observed in the members of the control group. Based on these results, the authors ascribe linea Alba formation to the forces exerted during swallowing, and not to tooth clenching. In this study no data about the potential presence of bruxism in both study groups were obtained and the linea Alba observed may result from force exerted by soft tissues against the surfaces of the teeth during tooth clenching (18).

Some others authors have suggested that tongue indentations are caused by macroglossia secondary to systemic amyloid disease (90,91). Yanagisawa and co-workers (84) do a study to clarify which physiological and anatomical factors were involved in the formation of tongue indentations. They studied a group of individuals with tongue indentations and another group of individuals without tongue indentations, by using a small intraoral appliance containing small pressure sensor. The appliances was placed at the lingual surfaces of the upper and lower right first molars. Lingual pressure was recorded under different conditions: during swallowing, during maximum

voluntary contraction (MVC) for 5, during 10% MVC for 1 min and, rest posture for 10 minutes. They also obtained casts of both dental arches and the tongue, in order to obtain the tongue/arch width ratio. No significant differences were found between groups during all the tests conducted. A higher tongue/arch width ratio was found in the group of individuals with Tongue indentation. They suggested that the lingual pressure exerted on the surface of the teeth is not related to the formation of tongue indentations, they consider tongue size to be a factor in the development of indentations. Also no data about the potential presence of bruxism in both study populations were obtained and the macroglossia observed in the group of subjects with Tongue indentation may result from tongue hypertrophy caused by tooth clenching (18).

2.9.3. Effects of Bruxism on Teeth and Teeth vitality

Tooth wear is a manifestation of loss of tooth structure due to non-carious processes such as attrition, erosion, and abrasion (92).

Bruxofacets (Fig 4) have been defined as atypical facets on teeth, with flat, smooth, shiny areas with sharp edges that correspond with similar opposing areas when the mandible is moved more than 3.5 mm from centric occlusion in a lateral excursion (93).

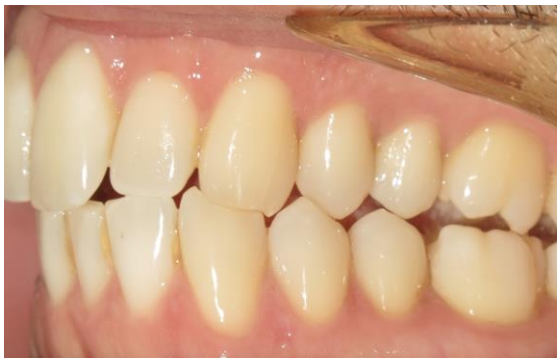


Fig 4: Bruxofacets

Excessive tooth wear is the most frequently cited sign of bruxism, and Bruxism was for long considered a major cause of tooth wear. A study of 15 bruxers and 15 non bruxers found that tooth wear progresses faster in bruxers than non bruxers (94).

A systematic review concluded that attrition seems to be co-existent with self-reported bruxism (95), and significant correlation reported between self-reported bruxism, tooth wear and/or TMD in several studies (96,97).

In contrast, in studies where the sleep bruxism has been diagnosed with polysomnography, no consistent relationship has been found between bruxism and tooth wear, or between bruxism and TMD. In fact, there have been suggestions that an inverse relationship may apply (98,99) .

Menapace et al. (32) reported that although tooth wear was present in 100% of sleep bruxism patients, it also occurred in 40% of asymptomatic individuals. Although sleep bruxism patients (young adults) presented with greater tooth wear than those with no history of tooth grinding or sleep laboratory evidence of sleep bruxism, tooth wear could not discriminate between patients with moderate–high versus low levels of sleep bruxism (100). Moreover, minimal wear has been reported in patients identified as active bruxers using electromyography (EMG) of the jaw closers during sleep (101).

The use of tooth wear as accurate sign for bruxism is controversial; while tooth wear clearly provides information about a history of forceful tooth to tooth contact, it does not prove current ongoing bruxism, and cannot indicate if the subject has static clenching activity. Considering these observations, tooth wear may not be sensitive or specific enough as a marker to capture current bruxism (18).

Nocturnal bite force during bruxism can exceed the amplitude of maximum voluntary bite force during the daytime (102). As a result of this excessive force, mild but continuous trauma will applied to the teeth, some teeth will present with degenerative or inflammatory processes that affect pulp health (103–107).

Two studies mentioned that the Excessive occlusal forces maintained for a long time may cause pulp alterations (104,105). These Excessive occlusal forces in many cases are accompanied by loss of small enamel structures on friction surfaces; this causes dentin to become exposed to the oral environment. This enamel loss frequently coexists with enamel and cement loss in the cervical region (abfraction). As a result of the excessive forces and tooth structure loss the pulp protect itself by producing reparative dentine "sclerotic dentin or reactional dentin " (108). This results in partial or total calcification of dentinal tubule lumen size.

Frequently, with formation of reparative dentine, mineralization of the pulp structure occurs too “pulp calcifications” (103,104,106). As a result of lack of space, pulp tissue decreases in size and starts to lose vitality. Clinically, apart from enamel loss, a change of color on the exposed dentin surface in the worn area may be observed (18); in slow evolution cases, such change of color is accompanied by reduced pulp sensitivity. In contrast, when wear progresses quickly, the pulp has no time to react and

defend itself; this will result in a pulp inflammatory state with increased sensitivity, which in some circumstances may require pulpectomy.

Groves Cooke (109) reports the case of a patient who presented with moderate pulp pain in the area of the maxillary incisors; he was diagnosed with occlusal trauma from protrusive bruxism. A night guard was made for the patient, and within a year pulpal symptoms had disappeared and pulp vitality was normal. Also Yu (110) describes two clinical cases where occlusal trauma resulted in the onset of pulp and periradicular alterations.

In histologic evaluation of 45 human permanent teeth with attrition (103); odontoblastic layer alterations were observed in all of them: pulp inflammation in 29 teeth, extensive degenerative changes in 13 teeth, and pulp necrosis in 3 teeth. In studied of 448 patients with dental wear (111); in 52 of them (11.6%) pulp almost reached the dental surface or was completely exposed. The palatal surface of anterior-superior teeth and the incisal surface of anterior inferior teeth were the most affected.

Rosenberg (106) considers abnormal occlusal stress to be the potential cause of internal and external dental resorptions.

Hethersay (107) proposed the bruxism as one of the predisposing factors to invasive cervical resorptions. He studied 222 patients with 257 teeth showing variable degrees of invasive cervical resorptions. The invasive cervical resorptions were associated with bruxism in six patients. Although nocturnal occlusal stress accounts for a low percentage of invasive cervical resorptions cases, its incidence cannot be dismissed. Rawlinson (112) reports on a mandibular first premolar affected by a severe occlusal trauma from bruxism showing severe apical and bone root resorption. Bone repair was achieved and root resorption stopped by means of endodontic treatment, occlusal adjustment, and the aid of a bite guard.

2.9.4. Effect of Bruxism on periodontium

Some authors have considered that the bruxism generates enough orthodontic forces to be able to move certain teeth, (113,114). Southardand co-workers (115) consider the increase in inter-proximal forces as a result of parafunctions to be one of the causes of tooth alignment disruption. In protrusive bruxism simulations, mandibular incisor displacements were observed which ranged from 100 to 700 μm, depending on the amount of force exerted during the simulation. These same researchers obtained similar results in lateral bruxism simulations (116).

The relationship between occlusal force and the periodontal disease has been a controversial topic. Stillman (117) and Box (118) suggested that excessive occlusal forces were the primary cause of periodontal disease. They recommended occlusal therapy to control tissue breakdown caused by periodontal disease.

In a study in 1954, Macapanpan and Weinmann (119) reported that both excessive pressure and tension produced alterations in the periodontal ligament, and that inflammation passed into that altered area. The authors considered that, provided severe force that cause necrosis of the periodontal ligament, this necrotic tissue would act as a barrier preventing the inflammation from extending into the underlying periodontal tissues (120).

In a study Later, authors confirm the relationship between excessive occlusal forces and the course of gingival inflammation (121). They also stated that excessive occlusal forces do not change the vascular and cellular features of gingival inflammation, but they do change the environment around the inflammation and consequently lead to changes in its direction. They postulated that, when inflammation spreads beyond the marginal gingiva and combines with occlusal trauma, they become interrelated co-destructive factors in periodontitis.

In contrast, Stahl (122) did not find such an association. He found that the inflammatory process spread into the crestal septum instead of spreading into the periodontal ligament space, and further suggested that "periodontal tissues may have adaptive capabilities to withstand abnormal occlusal forces which also limit inflammatory infiltration into the periodontal spaces from the marginal gingiva".

More recently, several authors agreed that there is not enough information available on the relationship between the progression of periodontal disease and trauma from occlusion (123,124). However, other authors disagree, since they consider occlusal discrepancies to be a risk factor for periodontitis (125,126).

Tooth migration has been defined by Carranza (127) as tooth displacement that occurs when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal disease. Periodontitis do not agree as to whether bruxism should be considered one of the causes of pathologic tooth migration (128).

A study of a population of patients suffering from periodontal disease failed to demonstrate pathologic tooth migration association with tooth wear and bruxism identified by means of questionnaires (129).

2.9.5. Bruxism and Temporomandibular Joint Dysfunction

Bruxism has been suggested as an initial and/or perpetuating factor in certain subgroups of temporomandibular disorder (TMD), but exact role of bruxism in the etiology remains unclear (130). The assumption is that chronic joint loading produces micro-trauma that leads to TMJ dysfunction.

In a study to investigate the prevalence of nocturnal bruxism, the author concludes that most TMD patients have a parafunctional habit, namely bruxism (131).

Clinicians believe that bruxism leads to signs and symptoms characteristic of TMD, but in a study to comparison of internal derangements of the temporomandibular joint with occlusal findings, the prevalence of bruxism and clenching are the same across diagnostic groups (132).

The diagnosis of sleep bruxism is based on a report of tooth grinding or clenching in combination with at least one of the following signs: abnormal tooth wear, sounds associated with bruxism, and jaw muscle discomfort (37). But the predictive value of these signs has been questioned (21,99).

Tooth wear is commonly considered to be a characteristic of bruxism, and it has been used in research as a predictor of patient's bruxism level to investigate the relationship between bruxism and TMD. Tooth wear provides information about a history of forceful tooth-to-tooth contact, but it does not validate current ongoing bruxism, nor can it indicate whether a subject has clenching activity. Tooth wear is considered to be an age related process (101), and it too has a multifactorial etiology that includes the presence of parafunctional habits, occlusal characteristics, diet, and salivary function and composition (133).Therefore, any conclusions about the relationship between bruxism and TMD symptoms based on tooth wear must be carefully interpreted (99).

The diagnosis of sleep bruxism is based also on the report of jaw muscle discomfort (37). Many theories tried to explain the musculoskeletal pain (25,134). Abnormal muscle function leading to muscle overwork and fatigue has long been regarded as a cause of pain (135).The theory of the vicious cycle ("pain - spasm – pain theory") was proposed to explain the role for the persistence of pain through a mutually reinforcing, vicious link between pain and muscle hyperactivity. Muscle hyperactivity has been suggested to be the cause of chronic pain conditions, such as tension-type headache, myofascial pain, and TMD (136).The pain model for TMD was, based on two

premises: that muscle hyperactivity can lead to pain, and pain leads to tonic hyperactivity. The first premise would appear to be true - when the muscles are voluntarily contracted for longer periods, the muscle fibers start to present fatigue; the second premise is questionable.

Camparis and Siqueira (137) did not found statistically significant differences for bruxism between patients with pain and without pain, even though patients without pain presented 20% more bruxism episodes than those with pain. The influence of pain on the bruxism pattern has been studied in patients with non-myofascial pain, compared with subjects without any facial pain, and it has been observed that patients with pain presented with 40% fewer bruxism episodes, suggesting that pain decreases the number of bruxism episodes (138).

Bruxism and/or oral parafunctions have been described as increasing loading forces in the TMJ that can contribute to dysfunction and pathology. Many different hypotheses have tried to explain the possible mechanism. The TMJ osteoarthrosis concept is that abnormal joint loading may change the equilibrium between cartilage degradation and synthesis, exceeding the functional adaptive capacity of the tissues in the joint. Excessive joint loading can lead to proteoglycan degradation, alterations in the synovium, inflammation, changes in the synovial fluid leading to impaired lubrication and nutrition of chondrocytes and ultimately cartilage degradation (139,140).

The "oxidative stress and degenerative TMJ hypothesis" has been proposed as a model for the molecular pathogenesis of degenerative TMJ disease. Abnormal mechanical stresses on the TMJ may lead to the generation of free radicals through hypoxia reperfusion injury, micro bleeding leading to hemoglobin deposition in intra-articular tissues, phospholipid catabolism, and other mechanisms. This accumulation of free radicals in the joint may lead to cartilage matrix degradation and the elaboration of an inflammatory response, ultimately affecting the biomechanical properties of the articular tissues (141,142).

Experimental bruxism studies done in healthy subjects have shown that bruxism may be able to induce acute pain in temporal regions, cheeks, supraorbital regions, TMJ s, and teeth (143,144). However, it is still unclear why some patients with sleep bruxism develop chronic pain while others do not.

Some bruxing patients without any facial pain reported morning fatigue or pain, but did not develop chronic pain (18). Persistent and chronic pain conditions are associated with prolonged functional changes in the nervous system, commonly referred

to as "central sensitization" (145). In chronic pain patients, factors such as central sensitization, neuroplasticity, dysfunction of the inhibitory neural descending system, and psychosocial abnormalities may be present. Diffusion and amplification of persistent deep pain, such as TMD, may also be the result of an increase in endogenous descending facilitation (146).

2.10. Effects of bruxism on prosthetic treatment

Some studies reported that bruxism might not be a primary factor, but it contributed to the wear of restorative materials (147), tooth survival in periodontitis (148), cracks in posterior teeth (149), implant failure (150) and complications with fixed partial dentures on implants (151).

2.10.1. Effects of bruxism on prosthetic restorations on natural teeth

The most common failures reported in Fixed dental prostheses 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, several reports have noted the possible association between bruxism and survival of fixed dental prostheses (151,152).

In restoring missing teeth with an opposing occlusion of normal teeth, most clinicians and researchers agree that a high noble content metal occlusal surface, is preferred in order to minimize wear of the natural dentition (18). Unpolished ceramics can produce more wear to opposing natural teeth than high noble content metal. In cases of heavy occlusal load such as 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. 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 (153). 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 (154). All things considered, metal or metal ceramic restorations seem to be the safest choice in cases of high load conditions (155). Because of the risk of chipping of ceramic veneers in metal–ceramic restorations, many clinicians prefer gold acrylic fixed dental prostheses 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 (155,156). 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 (157,158).

2.10.2. Effects of bruxism on implant restorations

Bruxism is often considered as one of the risk factors for implant treatment (159,160). Some early papers on survival of fixed prostheses on osseointegrated implants often referred to bruxism and heavy occlusal loading as the cause of implant failures (161). On the contrary, in a prospective 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 effect (162). 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 (163).

Systematic reviews have concluded that the relationship between occlusal forces and loss of osseointegration has never been demonstrated (164,165). Although bruxism was included among risk factors, and was associated with increased mechanical and/or technical complications, it had no impact on implant survival (166). 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 (150,151,167).

Although scientific reviews on this topic state that there is insufficient evidence to support or refute the possible causal relationship between bruxism and implant failure (168), a careful approach should be recommended (169).

2.10.3. Effects of bruxism on removable dentures

Boucher (170) mention that clinical experience indicates that bruxism is a frequent cause of complaint of soreness of the denture-bearing mucosa, and parafunctions is included as a possible factor related to the magnitude of ridge reduction.

A paper described the management of four patients with severe sleep bruxism, and who were using conventional removable partial dentures. Each patient was provided with a splint-like removable partial denture, called a night denture, and followed-up for

2–6 years using the night denture. The authors concluded that the night denture appeared to be effective in managing problems related to sleep bruxism in patients with removable partial dentures (171).

2.11. Diagnosis of Sleep Bruxism

Early diagnosis of bruxism is extremely important for both the clinician and the patient. In patients with sleep bruxism, awareness or reports of current tooth grinding are essential elements for the diagnosis of this condition (11). The appearance of tooth wear and the patient's reports of jaw muscle tightness, discomfort, and pain are less reliable (4,13). For research purposes, sleep bruxism are frequently monitored with polygraphic and audiovisual recording systems in a laboratory (polysomnography) (21). An alternative is the use of a portable system at home, in the natural sleep environment (77). The tools for identifying and assessing bruxism are summered in table 2.

Table 2: Methods for assessing SB

Method		Note
Clinical diagnosis	Patient history	Patients, bed partner, parents, or siblings report current tooth grinding sounds during sleep.
	Clinical	By assessing the clinical signs and symptoms of SB.
	Questionnaires	To investigate patients' general and oral health, sleep quality, sleep habits, oral parafunctions, presence and characteristics of pain, headache, fatigue, depression, anxiety and stress.
Complementary methods	Portable EMG record	Recording EMG activity during sleep from the temporalis or masseter muscles. There is low specificity and sensitivity in distinguishing actual RMMA episodes from the many other oro-facial and motor activities that occur during sleep. There is no monitoring on awakening from sleep, arousal, sleep staging, or other sleep variables. This tool could be valuable in the clinical assessment of SB and in large sample studies.
	PSG recording	It is the gold standard for the diagnosis of SB and the sleep bruxism can be discriminated from other sleep disorders. full night monitoring of electrocardiogram (ECG), electroencephalogram (EEG), EMG, electroencephalogram (EOG), leg movements, respiratory effort, airflow.

2.11.1. Clinical diagnosis

a) ***Symptoms*:** questionnaires and personal interview.

b) ***Signs:*** observation, and mouth examination, model, and photographic examination to quantify tooth wear.

The clinical diagnosis of SB can be based on the international diagnostic criteria proposed by the American Academy of Sleep Medicine (AASM) (Table 3) (11).

Table 3: Clinical diagnostic criteria for SB (American Academy of Sleep Medicine)
Recent patient, parent, or sibling report of tooth grinding sounds occurring during sleep for at least 3 to 5 nights per week in the last 3 to 6 months. One or more of the following is present: • Abnormal tooth wear • Hypertrophy of the masseter muscles on voluntary forceful clenching • Discomfort, fatigue, or pain in the jaw muscles upon awakening. Jaw muscle activity cannot be better explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder.

2.11.1.1. Symptoms

Questionnaire is a rapid way of collecting information from patients. It is the most widely available method to be used in everyday practice, it can be applied to a large population, and they are useful for obtaining a great amount of information in a short time, although the information on bruxism is subjective in nature. Several researchers proposed questionnaires for detecting bruxers (172–174). Pintado et al. (174) reported that subjects who were classified as bruxers based on a history and clinical examination gave a positive response to at least two of the six Questionnaires presented in Table 4. However, bruxism episodes such as clenching are not accompanied by noise (173); consequently, a percentage of patients are considered to be unaware of their bruxism activity and thus will be unable to identify themselves as bruxers. Moreover, self-reports of bruxism related signs ⁄ symptoms and awareness of bruxism have been found to show substantial fluctuation over time (175,176) and under or overestimation of the prevalence of sleep bruxism has been reported (130,177).

Table 4:Questionnaire for detecting bruxer
Has anyone heard you grinding your teeth at night?
Is your jaw ever fatigued or sore on awakening in the morning?
Are your teeth or gums ever sore on awakening in the morning?
Do you ever experience temporal headaches on awakening in the morning?
Are you ever aware of grinding your teeth during the day?
Are you ever aware of clenching your teeth during the day?

2.11.1.2. Sign

In the clinical examination, some signs may be present to suggest bruxism. The character of these signs (and symptoms) is "relative", since they alone are not sufficient evidence of bruxism, and they have not yet been validated by scientific method. So far, only the clinical validity of tooth-grinding sounds during sleep has been studied. When checked against polysomnography, they showed a sensitivity of 78% and a specificity of 98% (21).

1. *Tooth wear:* Grinding causes an attrition of the teeth, Several studies have demonstrated a positive relationship between tooth wear and bruxism (178,179) but others have not (99,180). Although this has a strong association with bruxism, it is not a specific sign since there are many causes of tooth wear. Tooth wear can be studied by direct visual inspection of the mouth, by examining plaster models of the patient's teeth, and by taking a look at intraoral pictures.
2. *Masticatory muscle hypertrophy:* The presence of excessively developed masseter and temporalis muscles during voluntary contraction can be a sign of constant clenching (18).
3. *Bilateral linea alba:* A hyper keratinized white line in the cheek mucosa is another sign that has been ascribed to bruxism (86). This line is located inside the cheek, parallel to the occlusal plane and matching the occlusal plane. Whenever present, it is always bilateral (83). Some authors claim that the formation of linea alba is not related to bruxism in any way (181). Others ascribe it to the cheek pressure exerted during deglutition (83).

4. *Tongue indentation:* This refers to the impressions made by the teeth on the circumferential edge of the tongue. They result from the force that the tongue exerts against the dental arches simultaneously with clenching. Some authors (85) consider them to be a clinical sign of bruxism, but others disagree (181).
5. *Fracture:* There may be fractures in natural teeth, prostheses, dental implants, and dental restorations (18).
6. *Tooth mobility:* This can be due to widening of periodontal ligament as a result of trauma with no periodontal disease involved (81).
7. *Pulp necrosis:* There may be death of pulp tissue caused by the continuous occlusal trauma (18).

2.11.2. Complementary methods for diagnosis:

Complementary methods by Use of intraoral appliance, Masticatory muscle electromyographic recording (EMG) with portable devices, and polysomnography.

Clinical signs and symptoms can be used to diagnose bruxism in clinics, while in the research field these clinical signs and symptoms are not sufficient because they do not enable accurate quantification of the bruxism activity. Accurate quantification of the bruxism activity is important to study etiology causes, effect of bruxism on stomatognathic system and assess response to treatments. Complementary methods for bruxism research include intraoral devices, portable EMG devices, and polysomnography.

2.11.2.1. Intraoral appliance

Some researchers have proposed the use of intraoral appliance to measure bruxism in an objective way. There are two approaches: observation of wear facets of the intra-oral appliance, and those that measure the bite force through sensors embedded in the intraoral appliance.

a) Wear of intra-oral appliance

Some Clinicians reported a repetitive wear pattern on the occlusal appliance (182,183). This has led to research based on the interpretation of occlusal appliance wear caused by bruxism.

I. *Bruxofacets in appliances*

In study to investigate the effects of the occlusal splint on grinding and clenching during sleep in patients with bruxism and craniomandibular disorders, the author measure the bruxism activity by interpretation of splint wear caused by bruxism (182). They found that all of them bruxed over the splints. 61% of the patients showed repetition of the wear pattern in the control visits every 2 weeks; the remaining 39% also showed wear repetition but after longer periods. They found that the wear facets always occurred in the same place and with the same direction and movement patterns.

Another study quantitatively assessed wear facets after 3 month on the occlusal surfaces of maxillary stabilization splints by digitized and analyzed through specialized software (183). They reported that parafunctional nocturnal dental activity on full-arch occlusal stabilization splints resulted in wear, which was both asymmetric and uneven.

Unfortunately, no studies seem to have evaluated the accuracy of these methods for measuring bruxism (131,183).

II. *Bruxcore plate*

An intra-oral appliance introduced as a device for measuring sleep bruxism activity objectively (184) by counting the number of abraded microdots on its surface and by scoring the volumetric magnitude of abrasion.

It consists of a 0.51 mm thick polyvinyl chloride sheet with four colored layers (two red and two white layers arranged alternately) and a medium-shaded grid with 0.14 mm diameter microdots printed on its surface. The plate contains 2,228 microdots per square centimeter.

The device is fabricated using a plate, which is heated and pressed over a maxillary dental cast and is put on the maxillary dental arch of the subject. The number of missing microdots is counted to assess the abraded area and the number of layers uncovered represents the depth parameter.

One of the technical problems with Bruxcore is that the thickness of the device becomes uneven in the press forming process and the adjustment of the surface, and this can influence the accuracy of assessment (81). Another disadvantage of this method is that it might be difficult to count a large number of missing dots with good precision. a computerized system for wear analysis has been presented which improves objectivity by resorting to an algorithm for measuring the abrasion area in pixels (185).

In a 6 month clinical study to evaluate the Bruxcore Plate as a dependent measure of nocturnal bruxing activity (186), both Bruxcore plates and portable EMG recordings were employed in 40 subjects for 14 nights. The event or duration of bruxism analyzed with the EMG data did not correlate significantly with Bruxcore plate scores.

The validity of this system is questionable, since Bruxcore alone can alter the oral environment and therefore modify the parafunctional activity (186).

b) Detection of bite force

Teams of researchers have created systems for detecting the forces generated by sleep bruxism (102,187,188).

I. Intra-appliance force detector

This system uses a 100 μm thick deformation sensitive piezoelectric film that is embedded 1 mm below the splint occlusal surface and is extremely sensitive to splint deformation caused by bruxism. A threshold is set, and every time it is exceeded the information is sent through a wire to an amplifier detector and then to a computer programmed to record and store data (187,188).

It was confirmed that the duration of bruxism events during simulated bruxism (clenching, grinding, tapping and rhythmic clenching) evaluated with the intra-splint force detector was correlated with that of the masseter EMG (187,188). It was noted, the intra-splint force detector was not suitable for detecting the magnitude of force during sustained clenching behaviour because of the characteristic of the piezoelectric film, i.e. this transducer is best at detecting rapid changes in force, not static forces (187,188).

Nevertheless, the use of occlusal appliances alters the oral environment and, consequently, bruxism values, and this may be confusing (186).

II. Force measurement by intraoral appliances

Nishigawa and co-workers developed a system that measures bruxism by means of strain gauge sensors embedded in acrylic splints (fig 5) (102). Two sensors are placed in the upper splint, one to the right and the other to the left, in the molar area. In the lower splint, two transducers are placed as antagonists. Every time the patient clenches his or her teeth the transducers record vertical bite force.

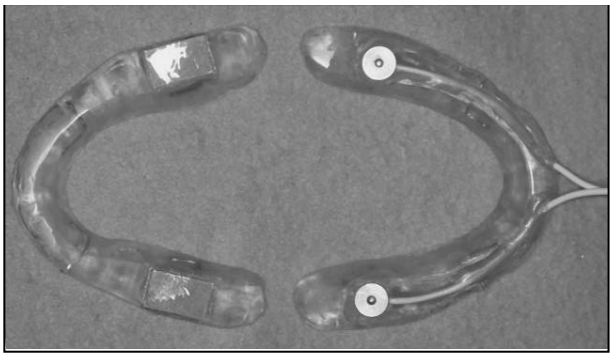


Fig 5: Upper and lower dental appliances containing miniature strain gauge transducers

2.11.2.2. Masticatory muscle EMG Records with portable devices

The study of muscular function by EMG is defined by the analysis of electric signal produced during the muscular contraction, and allows interpretations in pathological and normal conditions (189).

The (EMG) has been widely used as an auxiliary method on temporomandibular disorders diagnosis; EMG has been used to study muscle hyperactivity (24), muscle fatigue (190), the relationships between masticatory muscle activity and malocclusion, craniomandibular disorders, and associations between the nocturnal level of masticatory muscle activity and facial pain (191). It's also allows evaluating the functional and biomechanical responses of masticatory muscles in rest, chewing and grinding (192).

Bruxism can be assessed in an ambulatory way through EMG recording of the masticatory muscle activity. The possibility of recording bruxism without affecting the nature of the oral environment allows more accurate assessment.

In comparison to Polysomnography (PSG) the portable EMG devices have many advantages include their relatively low cost and the possibility of studying subjects in their own homes (the portable system can be easily operated by the subject), without the need for them to go to a research center every day. This makes it easier to study a large number of subjects over long periods.

The detection of sleep bruxism with portable EMG devices is generally considered inferior to that in a sleep laboratory (PSG) because other activities of the masticatory muscles (e.g., Swallowing, coughing, talking, and jaw movements without tooth contact) cannot be discriminated from sleep bruxism, and may be misrecorded as bruxism episodes (60,77,98,193,194). Moreover with portable EMG devices, other sleep disorders (e.g. sleep apnea and insomnia) cannot be ruled out or other

physiological changes related to sleep bruxism (e.g. microarousal, tachycardia and sleep-stage shift) cannot be monitored (60,98).

Muscle activities without tooth contact have been reported in both non-bruxers and bruxers (36,72,84). A study by Lavigne and co-workers (64), conducted in a sleep laboratory, found that 60% of normal individuals will show rhythmic muscle activities (of the masticatory muscles) without tooth contact, the frequency of these activities is three times more in bruxers subjects than control subjects, These non bruxing activities may be included in the records which may mislead the researchers (61). The authors recommend the use in sleep studies of a microphone connected to a recorder, so that real tooth grinding episodes can be distinguished from rhythmic movements without tooth contact.

Another limitation with portable EMG devices, it is less strict control over the studies, in terms of electrode position reproducibility, cleaning of the skin prior to electrode placement, potential electrical artifacts generated by badly adhered electrodes, etc.

Surface EMG is sometimes controversy as being an accurate method for muscle activity investigation because the results could be influenced by neighboring muscles and fatty tissues. Visser and colleagues have demonstrated in a study with masseter and temporalis muscles that there is no significant influence from electrode placement on their producibility of EMG recordings (195). Also the relocation of electrodes as a source of error seems to be less important than often estimated (196).

2.11.2.3. Polysomnography

Polysomnography (PSG) conducted in a sleep laboratory is the most accurate method for studying bruxism. It records biologic signals such as electromyography (EMG), electroencephalogram (EEG), electrooculogram (EOG), and electrocardiogram (ECG), as well as audio and video recordings. Trials carried out in sleep labs are constantly monitored by specialized staff and they allow recording of multiple aspects of body physiology (sleep, breathing, deglutition, pulse rate, etc.).

Because the sleep laboratory setting offers a highly controlled recording environment, other sleep disorders (e.g. sleep apnea and insomnia) can be ruled out and sleep bruxism can be distinguished from other oro-facial activities (e.g. swallowing and coughing) that occur during sleep (98,197,198). Physiological changes related to sleep bruxism (e.g. microarousal, tachycardia and sleep stage shift) can also be monitored.

Interactions between bruxism and sleep physiology are currently known by means of studies based on Polysomnography. In 1961, Takahama was the first to report on a bruxism study conducted in a sleep lab (199). Correlation with EEG findings revealed that bruxism occurred predominantly during the light sleep stage and was preceded by pulse, breathing, and cortical activity alterations. This suggested a relationship between sleep bruxism and activity of the autonomic nervous system.

Although polysomnography is considered as the gold standard, there are some limitations. One of limitation is the requirement for subjects to sleep in a specialized sleep laboratory. This might result in more stress than sleeping in their normal environment (home); thus change in the environment for sleep may influence the actual behaviour of bruxism. Also patients are usually less willing to participate due to disruption of lifestyle. Another limitation with polysomnography is that the recording is very expensive and not easy to conduct the multiple night recording, these drawbacks make it difficult to carry out studies on a great number of subjects and over long periods, and because the occurrence of sleep bruxism varies over a number of nights (177,191), multiple night recording is necessary to understand the substance of sleep bruxism (176).

2.12. Management of bruxism

Up to now, there is no specific cure for bruxism. The clinicians manage sleep bruxism, with the primary goals of preventing damage to the stomatognathic system and reducing patient's complaint.

Current managements are oriented toward:

1) Behavioral strategies.
2) Occlusal strategies.
3) Pharmacologic strategies.

2.12.1. Occlusal strategies

Two categories of occlusal management strategies for bruxism can be distinguished: true occlusal interventions and occlusal appliances.

2.12.1.1. True occlusal interventions

This strategy, which includes occlusal equilibration, occlusal rehabilitation and orthodontic treatment, is aimed at 'achieving harmonious relationships between occluding surfaces', and still controversy among dental clinicians and researchers.

Butler (200) described an occlusal adjustment procedure for the treatment of bruxism. Frumker (201) formulated a set of principles for a successful occlusal treatment, on the basis of an idea that the better the occlusal anatomy and function, the easier the bruxers 'relieve tension in the masticatory and associated musculature'.

Many letters and case reports published with description of the efficacy of occlusal interventions in the management of bruxism, by means of either occlusal equilibration (202) or occlusal rehabilitation with composite resin materials (203).

In contrast, in study to evaluate the effectiveness of an orthodontic technique (buccal separators) in relieving bruxism activity as monitored through masseter muscle EMG activity (204), no statistical differences between pretreatment and post-treatment evaluations or between the active treatment and control group were observed. The developer of the buccal separator technique failed to provide convincing evidence in favor of his technique (205).

There is no support in the literature for the use of true occlusal interventions like equilibration, rehabilitation and orthodontic alignment in the management of bruxism. Moreover, in view of the current insights into the etiology of bruxism, that the disorder is mainly regulated centrally not peripherally (168,206).

2.12.1.2. Occlusal appliances

The second category of occlusal management strategies for bruxism is the most frequently used strategies in bruxism management "occlusal appliances".

According to the Glossary of Prosthodontic Terms [8th ed.], "occlusal splint is defined as any removable artificial occlusal surface used for diagnosis or therapy affecting the relationship of the mandible to the maxilla. Occlusal appliances is considered an effective and non-invasive treatment for many sleep bruxism patients and those with temporomandibular disorders (207). These splints have different names [e.g.

occlusal bite guard, bruxism appliance, bite plate, night guard, and occlusal device, splints for muscle relaxation, stabilizing splints] and slightly different appearances and properties, most of them are made from hard acrylic-resin (208–211), another material for fabrication of appliances is soft-resin (212,213). Hard splints are generally preferred over soft splints because hard splints are suggested to be more effective in reducing bruxism activity than soft splints (214), and soft splints are more difficult to adjust than hard ones (practical reasons).

Occlusal appliance therapy with deferent design can be recommended for the following purposes:

- To protect oral tissues (teeth, cheek, and tongue), muscle of mastication, and the TMJ in patients with oral parafunctions.
- To eliminate the effects of occlusal interferences.
- To stabilize unstable occlusion.
- Masticatory muscle relaxation (treat muscle hyperactivity).
- Reducing musculoskeletal pain (myalgia, myofascial Pain).
- Restoring the occlusal vertical dimension of occlusion in patients with severely worn dentitions.
- Test the effect of changes in occlusion and vertical dimension on the TMJ and jaw muscle function before extensive restorative treatment.
- Disc interference disorders
- Retrodiscitis.

2.12.1.2.1. Mechanism of occlusal appliances work

There is no general agreement if the occlusal appliance treatments have a beneficial effect or the mechanism of how occlusal appliances work. Following theory (which is overlapped and not mutually exclusive) may explain how occlusal appliances can work:

Preventing the patient to close in maximal intercuspal position:

The occlusal appliance guides the patient to place his mandible in a new position, resulting in a new muscular and articular balance. The patient, disturbed in his habits will not clench his teeth like before (215).

Distribution of forces:

The forces generated during bruxism can be as much as six times the maximal force generated by normal chewing (216). The occlusal appliances distribute these forces across all the existing teeth with the same intensity of force in the direction of the longitudinal axis of each tooth and consequently, better dissipation towards the periodontal tissue. These appliances can decrease the frequency of bruxing episodes but not the intensity of bruxing episodes (182).

Normalizing periodontal ligament proprioception:

Proprioceptive fibers contained in the periodontal ligament of each tooth send message to central nervous system, triggering muscle patterns that protect them from overload. An occlusal appliance with even distributed contacts on all of the teeth prevent individual teeth overload. Thus an occlusal appliance balances the load and allows for muscle symmetry (217).

Relaxing the muscles:

Tooth interferences to the centric relation arc of closure hyper activate the lateral pterygoid muscles (25) and posterior tooth interferences during excursive mandibular movements cause hyperactivity of the closing muscles (218). The muscle hyperactivity can cause muscle fatigue and pain. If the hyperactivity is stopped, the pain caused by it will usually disappear. These interferences can be eliminated by means of occlusal appliances with even distributed contacts on all of the teeth, with anterior guidance (immediate disclusion of all posterior teeth) in all excursive movements; this can relax the elevator and positioning muscles (219).

Allowing the condyles to seat in centric relation:

Some studies (220,221) found that the percentages of the superior head of lateral pterygoid muscle inserted into the mandibular disc complex are about 30%, therefore it was suggested that the spasm of lateral pterygoid muscle was associated with anteromedially dislocation of the disc toward the origin of muscle (222,223). The occlusal appliances lead to relaxing of the lateral pterygoid muscle and the superior belly of lateral pterygoid obtain it's fully extension, allowing the articulator disc to obtain its anterosuperior position over the condylar head (217). However other study (224) indicate that only 10% of these fibers reach the disc, and 24% reach the joint

capsule. Moreover a study (225) found there was no statistically significant correlation between the lateral pterygoid muscle attachment types and TMJ abnormalities.

Increase in the vertical dimension of occlusion:

Temporary use of occlusal appliances with a vertical height exceeding the physiologic rest position cause isometric contraction of the elevators of mandible due to muscle fibers stretching, this does not cause neuromuscular relaxation (226,227). Although an immediate decrease in the electromyographic activity of the jaw elevator muscles has been observed at various vertical dimension of occlusion obtained with occlusal appliances there are no data pointing to the persistence of this effect in long term users of occlusal appliances.

Cognitive awareness:

The presence of the occlusal appliance as a foreign object in the mouth cause change in the oral tactile stimuli, decrease the oral volume and space for the tongue and make the patient conscious about the bruxism and the patient response by changing their behavior. As cognitive awareness is increased, factors that contribute to the disorder are decreased (217).

Placebo effect:

The favorable doctor patient relationship, and the explanation of the problem and reassurance that the appliance will be effective, often leads to a decrease in patient's emotional stress, which may be the significant factor responsible for the placebo effect (217).

Increased peripheral input to the central nervous system:

Nocturnal muscle hyperactivity appears to have its source in the CNS. When an occlusal appliances is placed between the teeth, it provides a change in peripheral input and thus decreases CNS-induced bruxism (217).

Overloading of the periodontal ligament of the anterior teeth

The proposed mechanism of action is that overloading of the periodontal ligament of the incisors teeth will cause activation of nociceptive afferent fibers, which by reflex pathways will inhibit the jaw-closing muscles and subsequently reduce the muscle tension (228).

Eliminates occlusal interferences

Occlusal appliance are eliminate the occlusal interferences, alter the periodontal Proprioceptive input to the central nervous system during night, and provide the patient with an "interference free" or "ideal occlusal scheme" That way, there is a breakdown and redirecting of reflex mechanisms leading to relaxation of overactive muscles (229–231). Therefore, whether oral appliance provide full or partial coverage of the teeth, they would "prevent disturbing influences to the neuromuscular system from occlusal contacts on mandibular closure and movements" (25,231).

However, the efficacy of the Occlusal appliance to reduce masticatory muscle activity and overall response to oral occlusal appliance treatment remains questionable (6), and there is not enough scientific evidence regarding the effects of appliance on sleep bruxism to date, mainly because of the difficulty in recording sleep bruxism activity in multiple nights and for a longer observation period (193).

2.12.1.2.2. Types of Occlusal appliances:

According to Okeson (232):

1) Stabilization appliance
2) Anterior repositioning appliances
3) Other types:
    - Anterior bite plane
    - Posterior bite plane
    - Pivoting appliance
    - Soft/resilient appliance

According to Dawson (233):

a) Permissive occlusal appliances / muscle deprogrammer
b) Directive occlusal appliances / non-permissive occlusal appliances

Permissive occlusal appliances: A flat plane appliance that is designed to unlock the occlusion to remove deviating teeth inclines from contact so that teeth do not interfere with complete seating of the condyles. This eliminates the cause and effect of muscle in co-ordination. The condyles are then allowed to return to their correct seated position in centric relation if the condition of the articular components permits.

Permissive occlusal appliances are often referred to as muscle deprogrammers. The two designs of permissive occlusal appliances are anterior midpoint contact splints

(nociceptive trigeminal inhibition (NTI) splint, Lucia jig and the B splint) and full contact occlusal appliance (centric relation occlusal appliance).

Directive occlusal appliances: (Anterior repositioning splint) are designed to guide the mandible in a specific relationship to the maxilla. The purpose of a directive occlusal appliance is to guide the condyles away from the fully seated joint position.

The directive occlusal appliances should be used only when a specifically directed position of the condyles is required (painful joint and TMJ clicking).

2.12.2. Behavioral strategies

A wide variety of behavioral strategies have been tried in the management of bruxism. Behavioral strategies should begin with a short and comprehensive explanation of bruxism to the patient, including its definition, causes, and consequences.

A) Biofeedback

Biofeedback uses the paradigm that bruxers can forget their behaviour when a stimulus makes them aware of their adverse jaw muscle activities (aversive conditioning). This technique has been applied for bruxism during wakefulness (234) as well as for sleep bruxism. While awake, patients can be trained to control their jaw muscle activities through auditory or visual feedback from a surface EMG. For sleep bruxism, auditory, electrical, vibratory and even taste stimuli can be used for feedback.

For the use of biofeedback in the management of sleep bruxism, Cherasia and Parks (235) used contingent arousal from sleep with actual awakenings. Nissani (236) used a taste stimulus to awaken the patient. This stimulus was caused by the bruxism related rupture of capsules, filled with an aversive substance (agreed upon with the patient, e.g. mustard, ginger, garlic, etc.) and embedded in a simple dental appliance.

A sound blast was applied as the aversive stimulus (237,238), the sound stimulus is supposed to actually wake up the patient, who is then supposed to switch off the sound and resume his sleep. The awakenings are a major disadvantage of such approaches, because sleep disruption may lead to serious side effects like excessive daytime sleepiness (239). Another techniques, the bruxism contingent vibratory feedback system of Watanabe et al. (240) and the jaw opening reflex feedback system that was recently developed by Jadidi et al. (91) are not inducing substantial sleep disturbance.

B) Other behavioral strategies

Other behavioral approaches that have been described in the literature for the management of bruxism include psychoanalysis/autosuggestion (241), hypnosis (242), progressive relaxation including meditation (243), habit reversal ⁄ habit retraining (244,245) and massed practice (246).

The value of the above described behavioral approaches is questionable, because there is no support in the literature for the use of these behavioral approaches; most studies are case reports, prescriptions and comparative studies. More well designed research is thus needed on the use of these approaches in the management of bruxism (247).

2.12.3. Pharmacologic strategies

Pharmacologic management is indicated on a short term basis only for acute or Severe Condition (248). Clinicians report that centrally acting drugs in the benzodiazepine group and muscle relaxants reduce bruxism related motor activity. Diazepam and methocarbamol have been tested in open study design (191). Clonazepam was reported to reduce sleep bruxism in a wide age range of patients (249). However, addiction risk needs to be considered. These medications are usually prescribed at bedtime, and patients must be informed of possible side effects (e.g., not driving in the morning due to potential drowsiness) (3).

Antidepressants such as tricyclics have also been used for managing sleep bruxism, but two controlled studies using ambulatory EMG failed to support the efficacy of a small dose of amitriptyline in management of sleep bruxism (248). The use of selective serotonin reuptake inhibitors such as fluoxetine, sertraline, and citalopram has been reported to induce clenching or grinding (248). The use of l-tryptophan (a serotonin precursor) in sleep bruxism management has been reported to have no effect. The use of a weight-control medication related to serotonin, fenfluramine, has been noted to exacerbate grinding. Therefore, caution is suggested before using serotonin-related medication in patients with sleep bruxism.

Patients with chronic antidopaminergic drug exposure (e.g., haloperidol, a dopaminergic antagonist) exhibited iatrogenic grinding similar to that associated with oral tardive dyskinesia, and a similar effect was seen with l-dopa (a dopaminergic precursor) in a patient already suffering from Parkinson’s disease (250). A randomized experimental trial demonstrated that acutely administered l-dopa modestly reduced

bruxism activity in otherwise healthy patients with sleep bruxism, whereas bromocriptine had no effect (38,43). So far, too few studies have been performed on dopaminergic regimens (e.g., l-dopa) to consider these in the long-term management of sleep bruxism. It also remains to be demonstrated whether, as in the case of periodic leg movement syndrome, a pharmacologic rebound will induce a resurgence of sleep bruxism activity later in the night or during the next day (3).

Another pharmacologic avenue for sleep bruxism management is the use of a beta-adrenergic antagonist, such as propranolol, or clonidine, an alpha agonist. A controlled experimental trial performed in laboratory with young patients with sleep bruxism, using placebo or long-action propranolol, resulted in no net reduction of sleep bruxism (251). Clonidine reduced sleep bruxism by 60%,but severe hypotension was experienced in the morning by 20% of subjects, which limits clinical use (251).

Botulinum toxin is reported to reduce the occurrence of RMMA and sleep bruxism (252).However, at this time, no controlled studies with polygraphic recordings have demonstrated that Botulinum toxin has long-term efficacy and safety for sleep bruxism. Moreover, it has been reported that the Botulinum toxin is carried to the central nervous system, and it is not known whether this explains its effects or whether it signifies a potential risk for the patient (253).

3. MATERIALS AND METHODS

3.1. Population sample

This study performed on 37 patients Over 18 years of age and Accept to volunteer who had sleep bruxism; Subjects varied in age between 21 and 37 years with a mean age of 28.29±5.16 years, 12 (38.7 %) females and 19 (61.3 %) male. However, 6 subjects were excluded from the statistical analysis because there is loss of electrode skin contact. The final statistical sample of subjects was reduced to 31. Patients were divided into two groups; Treatment group 20 patients (64.5 %), and untreated control group 11 patients (35.5 %)

Ethical approval for the study was obtained from the institutional ethics committee (Yeditepe Üniversitesi Etik Kurulu) prior to the study. All participants were informed about the objectives and methodology of the study and written informed consent was obtained. Each patient could at any time take the decision to stop his/her participation in the study, whatever is the reason.

The presence of bruxism was clinically established by means of an inspection of the soft and hard intraoral tissues.

3.2. Inclusive criteria:

The Patients selected on the basis of the following criteria: A history of tooth grinding or clenching in combination with at least one of the following conditions:

1. Abnormal tooth wear (Smith and Knight Tooth wear index) (Table 5) (254).
2. Bilateral Linea Alba, and/or tongue indentation (62,81).
3. Frequent reports of headache, stiffness, fatigue, or discomfort in the jaw muscles upon awakening by using questionnaire in table 6 (62,81).

3.3. Exclusion criteria

1. More than two missing posterior teeth excluding third molars.
2. Use of removable prosthesis.
3. Periodontal problem.
4. Dental pathology.
5. Use of medication with possible effects on sleep or motor behaviour.
6. Ongoing dental therapy including orthodontic treatment.

7. Neurological disorder.
8. Psychiatric disorder.
9. Medical disorder.
10. Hart disorder or patient use cardiac pacemaker.
11. Sleep disorder.
12. Pregnant.
13. History of sleep breathing disorders.
14. Refuse to participate in the research.

Score	Surface	Criteria
0	B/L/O/I	No loss of enamel surface characteristics.
	C	No loss of contour.
1	B/L/O/I	Loss of enamel surface characteristics.
	C	Minimal loss of contour.
2	B/L/O	Loss of enamel exposing dentine for less than one third of surface.
	I	Loss of enamel just exposing dentine.
	C	Defect less than 1 mm deep.
3	B/L/O	Loss of enamel exposing dentine for more than one third of surface.
	I	Loss of enamel and substantial loss of dentine.
	C	Defect less than 1-2 mm deep.
4	B/L/O	Complete enamel loss - pulp exposure - secondary dentin exposure.
	I	Pulp exposure or exposure of secondary dentine.
	C	Defect more than 2mm deep - pulp exposure - secondary dentine exposure.
B: buccal; L: lingual; O: occlusal; I: incisal; C: cervical.		

Table 5. Smith and Knight tooth wear index.

Questionnaire	Yes	No	Don’t now
Do you do grind your teeth when you sleep?			
Has anybody heard you grind your teeth while you sleep?			
On awaking up, do you usually find that you are clenching your teeth			
When you wake up, do you usually have jaw pain or jaw fatigue?			
When you wake up, do you usually have the feeling that your teeth are lose?			
When you wake up, do you usually have a headache in temples?			
When you wake up, do you usually have a jaw lock?			
Have you ever found that you were clenching your teeth in the daytime?			
Have you ever found that you were grinding your teeth in the daytime?			

Table 6. Bruxism Questionnaire

3.4. Final diagnosis for active-passive bruxism:

Active and passive bruxer determined by using acid aching technique (255). In order to do final decision are they active bruxer or not, 37% phosphoric acid etching is applied to the surface of the enamel of the teeth (*wear facets)* which has attritions for 15 second and then rinsed thoroughly and the tooth surface is checked to confirm the chalky appearance is formed.

After one night of sleep the chalky appearance is checked again (not more than 24 hours - Time for enamel completely return to normal appearance ranged from 96 h to 2 weeks; after 48 h, the enamel had an almost normal texture) (256). If the teeth facet lose the white chalky appearance indicates the patient is active bruxer (Fig 6) (255).

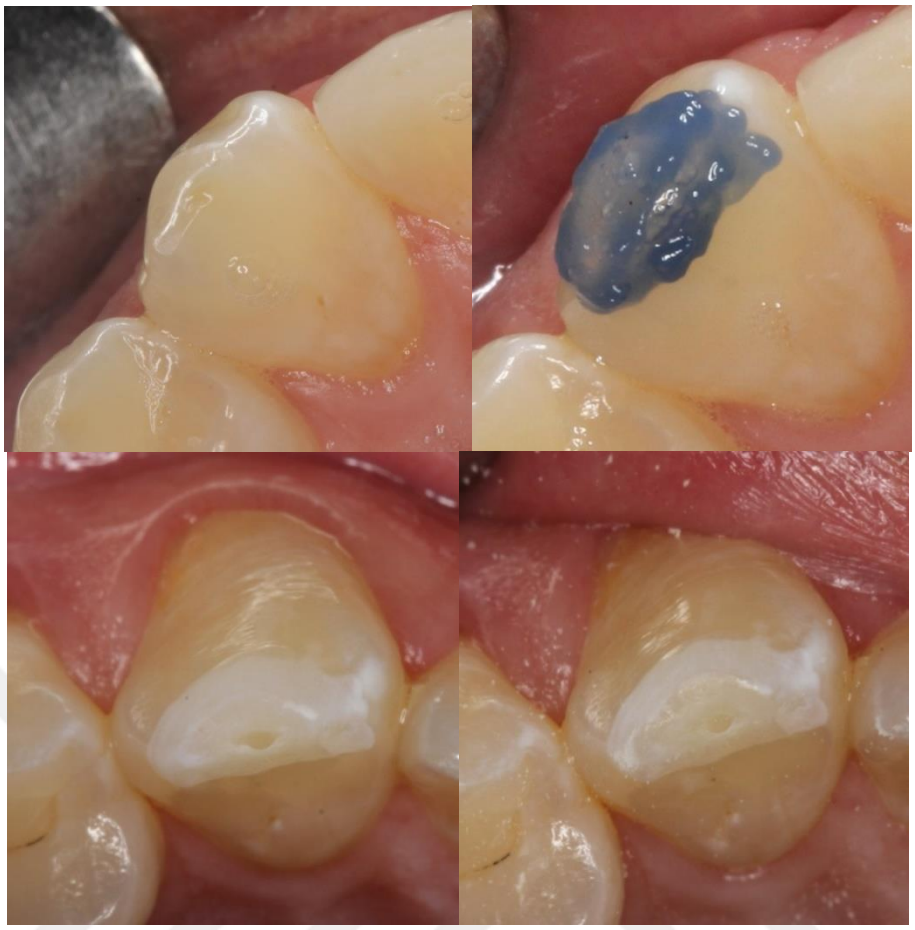


Fig 6: acid aching technique

3.5. Intraoral appliance fabrication

For all subjects, an impression of both the maxillary and mandibular arches were taken with irreversible hydrocolloid impression material (Kromopan alginate / Lascod) in suitable size stock trays. After the impression trays is removed from the mouth the impresion is inspected under good lighting to make sure the impression matrial are not separeted fom the trays, free of voids and tears, and all anatomical details is clearly recorded, if necessary, the impression were repeated. A centric relation record and face bow registration were carried out for each patient.

The impressions was poured in dental stone (Amberok) on vibretor to make free of voids dental casts, the casts is mounted on a semi-adjustable articulator (Artex, Girrbach Dental). The facebow record helps in mounting of the maxillary cast in the articulator and the centric relation bite record help in mounting of the mandibular cast in relation to the maxillary cast in the articulator. This relates exactly to that of the patient's existing jaw occlusion relation.

For all subjects, a full arch flat plane clear heat cure acrylic resin interocclusal appliance covering all of the mandibular teeth were fabricated on the mandibular casts with 3 mm thickness of acrylic between the maxillary and mandibular posterior teeth, the desired vertical dimension (3 mm) controlled by the incisal guide pin of the articulator (Fig 7).

Fig 7: Elevate the incisal guide pin of the articulator (vertical dimension 3 mm)

A milling machine is used to carefully remove any interference areas when placing the appliance in the mouth, the appliance must be fully seated and comfortly seat on the mandibular teeth, without rocking or shifting and with good retention and stabilization.

After checking the base of the appliance the occlusal surfece is adjusted intraorally to ensure bilateral anterior - posterior simultaneous contacts in centric relation with anterior and canine guidance (immediate posterior disclusion in lateral and protrusive excursions), the occlusal contacts are checked by articulating paper (Hanel)

during closing of the mouth in centric occlusin. The premature contacts are grinded until the working cusps of the posterior teeth and incisal ridges of the anterior teeth of the maxilla create simultaneous and even contacts with the appliance surface (light contact in anterior teeth) (Fig 8). The centric occlusal contacts adjusted to have freedom in the centric of around 0.5 mm forward, backward and laterally.

After centric relation occlusion is established an anterior guidance was developed by giving immediate posterior disclusion during any lateral excursion and protrusive movement.

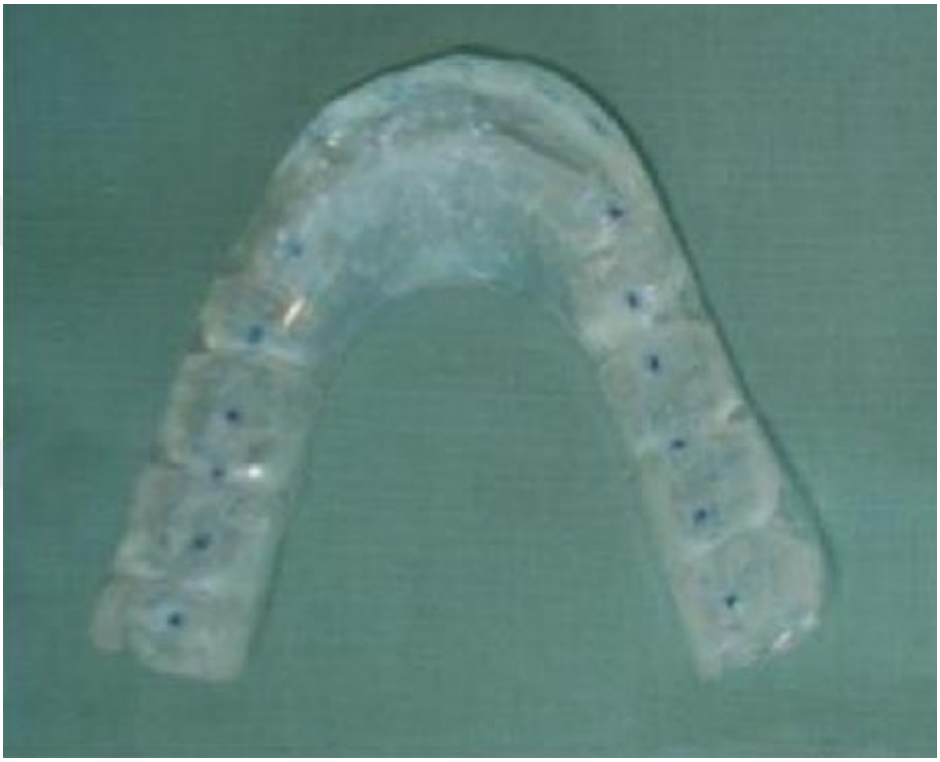


Fig 8: Occlusal appliance

3.6. EMG Event Monitoring

EMG activity during sleep was recorded from the right side of anterior temporalis muscle with portable EMG recording device (Grindcare®, MedoTech, Herlev, Denmark) (Fig 7).

Fig 7: GRINDCARE

GRINDCARE is a diagnostic tool for assessing and managing sleep bruxism, the basic principle of the device is a portable EMG apparatus (hang around the neck using a safety cord supplied) with three integrated electrodes placed on the skin overlying the temporalis muscle. The signals are conducted from electrodes over a cable to a Stimulator unit that digitizes, analyzes, and stores extracted event data in an internal microchip, this is done by filtering the EMG signals between 250 and 600 Hz, sampling the data (10-bit, 2-kHz sampling rate), performing Fast Fourier Transformation (FFT), and finally comparing the root mean square (RMS) outcome to a threshold value that is set to 20% of the maximum EMG during a clench at 60% of the maximum voluntary contraction (MVC), this to distinguish between jaw muscle activity associated with sleep bruxism events and other jaw or facial muscle activity.

Setup of the threshold level is done every time the device is mounted before sleep. When the amplitude of the EMG signal has been above the threshold for more than 100 ms, an event is recorded in the log file. An event can represent a voltage that exceeds threshold for up to 1 second; longer continuing events are counted as additional events. The total number of EMG events is then used to characterize each individual on

each night. Through its calibration procedure, the Grindcare device is intended to detect bruxism related EMG events, i.e., those occurring when the patient actually grinds his/her teeth and there is activity in the jaw closing musculature. Methodological studies have demonstrated that the signal recognition algorithm in the device differentiates among bruxism/clenching, relaxing, and grimacing activity (257).

Apparatus also automatically monitor the conductance between the electrodes and the skin which assured that the patients had placed the electrodes with adequate contact and monitor the conduction over the sleeping.

The device handles the following tasks: (a) recording of EMG activity; (b) processing of EMG signals in order to detect a particular activity (tooth-grinding/tooth-clenching); and (c) providing a battery powered contingent electrical stimulation based on individual parameters (biofeedback). These individual parameters are used as reference values and to determine threshold values and criteria for triggering the biofeedback signals to the anterior part of the temporalis muscle. The patients were able to adjust and set the intensity of the electrical biofeedback stimulus to a level that was suitable to the user, e. g. a level that was not uncomfortable to the patient but which clearly could be perceived.

The contingent electrical stimulation circuit was controlled by microprocessor, a controlled low voltage electrical impulse, which was adjusted to a clear, but non-painful intensity was applied through the EMG electrodes. These impulses interrupt the muscular activity and bring about local muscle relaxation without waking the user.

The device has no adverse effect (258), the contingent electrical stimulation was not associated with any significant perturbation of polysomnography or self-reported data on sleep and sleep quality (259), and there was no change in the overall amount of time the patients slept during using the device, which indicates that the patients were not disturbed by the contingent electrical stimulation or by wearing the device during sleep (260).

In this study the device is used for measuring EMG activity without feedback, so the intensity of the electrical biofeedback stimulus is adjusted to zero (0= no electrical biofeedback).

The device mainly consist of three parts; GrindDock, Stimulator, and Electrode.

GrindDock: The GrindDock (Fig 8) is the control of system. It is used for configuring and programming the stimulator, displaying the recorded data, battery charging of stimulator and for transferring of patient data to a PC. The communication between the GrindDock and stimulator is wireless (radiofrequency). The GrindDock is inactive during nightly use.

Fig 8: The GrindDock

Stimulator: The stimulator (Fig 9) continuously measures and records the EMG signal during sleep and analyzes the recorded signal using a proprietary algorithm.

Fig 9: The Stimulator

Electrode: The electrode is the connection between the stimulator and the skin which is placed around the forehead (above the eyes). It reads the EMG signal from the skin overlying the temporalis muscle and transmits it to the stimulator. The electrode is attached to the skin with a self-adhesive disposable gel pad (Fig 10).

Fig 10: The electrode and the self-adhesive disposable gel pad

3.7. Experimental procedure:

The patients were informed of the purpose of wearing the appliance, the expected effect of the appliance on the bruxism and the need for maintain the hygiene of the appliance. Grindcare device and an instruction brochure in which the step by step procedure was visually indicated are delivered to the patients and all patients received comprehensive training in the use of the device.

The patients were instructed to wear it every night over the 12 days of the study. During the first 6 consecutive nights, all patients used the device during sleep without the occlusal appliance to record the pre-treatment baseline data (number of EMG events/hour). During the next 6 consecutive nights, all patients in treatment group used the device during sleep with the occlusal appliance in, to record the treatment data (number of EMG events/hour).

They were instructed to place electrodes in the same sites with the same manner. The patients instructed to insert the occlusal appliance and activate the recording system just before going to sleep and stop it immediately after wake up.

After 12 days of using the device the patients returned the Grindcare devices back. The Grindcare device connected to the computer and the Patient data transferred to a computer using the software Grindcare Manager (Medotech A/S, DK) and the quality of the data was examined to make sure that the patient had used the device

correctly. The poor data due to lost electrode connection or misplaced electrode discarded.

3.8. Instruction of the Grindcare device use:

Just before going to sleep, wash and dry the skin on the temple and clean the skin with alcohol 70 %, Switch on both the GrindDock and Stimulator and wait for few second until the Stimulator is wireless communicated with GrindDock, then insert the electrode firmly into the Stimulator, after that place the gel pad over the electrode.

Place the electrode on the skin over the temporalis muscle on the temple (Fig 11), temporalis muscle determined by placing a couple of fingers on the flat part of the temple (above the eyes) and biting down, the temporalis muscle bulging out will be feel, the electrode must stick to the skin along its entire length so that all three terminals have good contact with the skin (there should be a minimum of three bars visible in the electrode contact icon).

Fig 11: Electrode over the temporalis muscle

With the electrode correctly placed and the Stimulator powered on, select “Measurement” from the main menu. Use the left arrow button to set the biofeedback level to zero (Measuring bruxism without biofeedback) Press OK to confirm your zero setting, and the next steps are to calibrate GRINDCARE to suit grinding pattern.

Start by relaxing your facial muscles, an arrow on the right of the GrindDock display will appear, then Clench (do not grind) the teeth in a good, firm bite (around 60% of maximum bite strength). Repeat a couple of times until the arrow disappear.

Measuring has now started and there is no longer wireless communication between the Stimulator and GrindDock.

Next morning immediately after wake up, carefully remove the electrode from temple without pulling on the cable, Measurement will stop automatically. Press the Stimulator on/off button to establish wireless communication, select “Data” from the main menu then press the middle button to enter the value and next “Synchronizing” is displayed to indicate upload of data from Stimulator to GrindDock, after Synchronizing finished turn of the device.

3.9. Statistical analysis

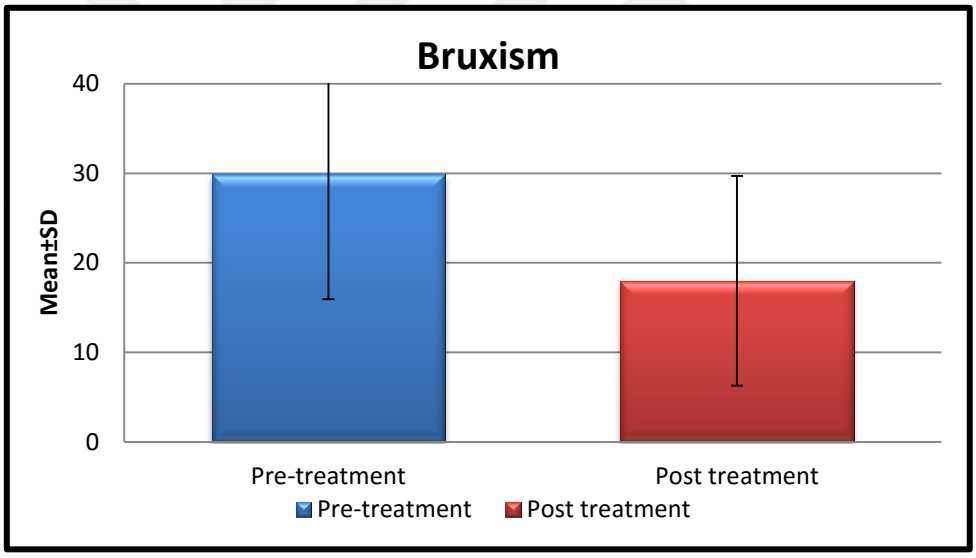
During the assessment of the data obtained in the study, IBM SPSS 22 (İstanbul, Turkey) was used for statistical analysis. During the assessment of the study data, conformity of the parameters to the normal distribution was assessed by the Shapiro-Wilks test. Parameters with normal distribution for the comparison of quantitative data were evaluated using Student’s t-test. Parameters with non-normal distribution for the comparison of quantitative data were evaluated using Wilcoxon Signed Ranks test. Significance was accepted at $p<0.05$ level.

4. RESULTS

Table 7. evaluation of pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour) and post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) in treatment group.

Pooled Data	pre-treatment	post-treatment	p
	Ort±SS	Ort±SS	
	29.89±13.95	17.99±11.72	**0.001***

Wilcoxon Signed Ranks Test ***p<0.01*

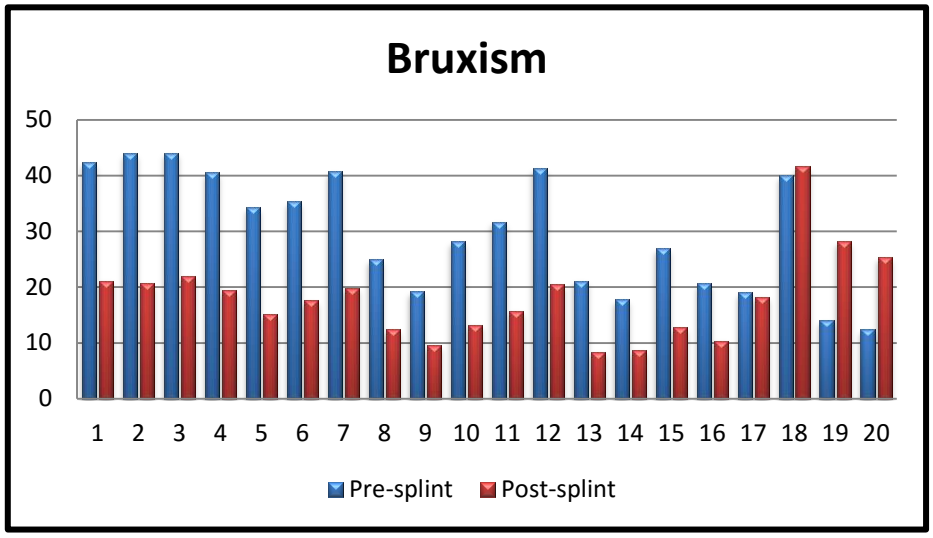


As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) ($p<0.05$).

Table 8. Individual level evaluation of pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour) and post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) in treatment group.

Patients (6 Night) Bruxism	Pre-splint Mean±SD (Median)	Post-splint Mean±SD (Median)	p
1	42.38±6.74 (42.35)	21.10±6.62 (20.20)	**0.028***
2	43.92±10.80 (45.65)	20.67±13.66 (18.60)	**0.028***
3	44.02±11.50 (46.00)	21.95±14.72 (19.15)	**0.046***
4	40.53±17.59 (44.95)	19.43±11.34 (17.20)	**0.116**
5	34.28±9.36 (32.90)	15.03±10.29 (11.65)	**0.028***
6	35.25±12.60 (38.25)	17.57±15.79 (10.10)	**0.046***
7	40.65±14.46 (45.15)	19.80±13.54 (15.90)	**0.046***
8	24.98±9.38 (29.45)	12.33±7.10 (11.30)	**0.028***
9	19.12±5.40 (20.75)	9.52±7.80 (8.00)	**0.046***
10	28.08±12.66 (28.75)	13.17±7.39 (10.35)	**0.028***
11	31.68±11.33 (32.65)	15.67±8.34 (16.45)	**0.028***
12	41.23±14.42 (40.95)	20.43±12.46 (17.50)	**0.075**
13	21.07±8.21 (20.45)	8.32±5.80 (7.00)	**0.046***
14	17.73±4.49 (19.70)	8.58±9.57 (5.20)	**0.075**
15	26.90±10.22 (27.65)	12.70±9.46 (9.85)	**0.028***
16	20.63±3.95 (21.65)	10.30±5.78 (8.45)	**0.028***
17	18.97±4.92 (21.60)	18.22±3.79 (18.95)	**0.600**
18	39.95±7.68 (40.00)	41.57±8.97 (44.05)	**0.600**
19	13.97±4.44 (12.75)	28.13±4.01 (28.25)	**0.028***
20	12.40±1.03 (12.80)	25.28±1.70 (25.20)	**0.028***

Wilcoxon Signed Ranks Test *$p<0.05$



In Treatment group;

1. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).
2. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).
3. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.046; p<0.05).
4. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significant (p>0.05).
5. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).
6. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.046; p<0.05).
7. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.046; p<0.05).
8. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the

post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).

9. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) tir (p:0.046; p<0.05).

10. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).

11. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).

12. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significant (p>0.05).

13. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.046; p<0.05).

14. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significant (p>0.05).

15. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).

16. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant reduction in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).

17. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is no statistically significant change in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) ($p>0.05$).

18. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is no statistically significant change in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) ($p>0.05$).

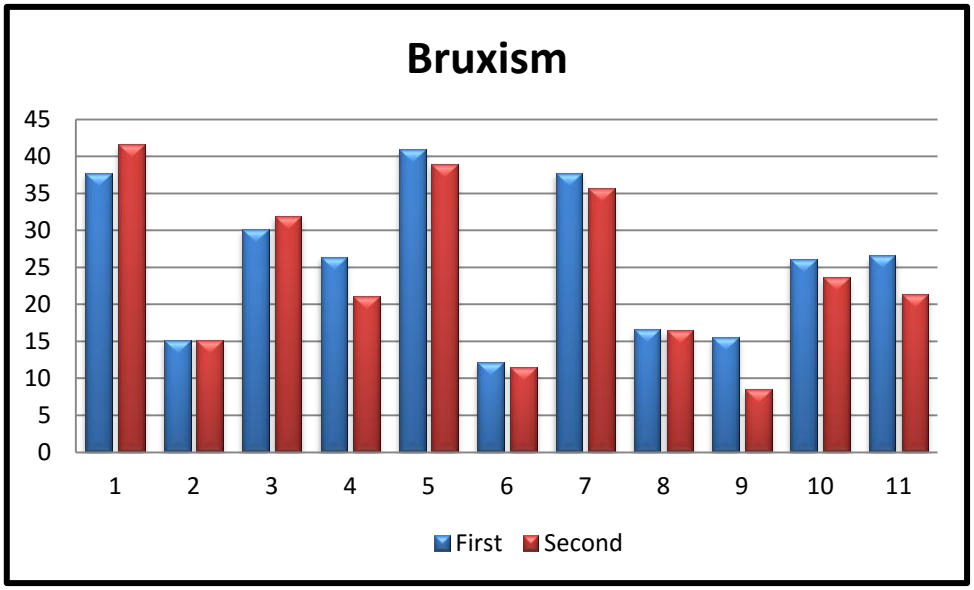
19. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant increase in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) ($p:0.028$; $p<0.05$).

20. As compared to the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significant increase in the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) ($p:0.028$; $p<0.05$).

Table 9. Evaluation of first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour) and second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour) in control group.

Patients (6 Night) Bruxism	First	Second	p
	Mean±SD (Median)	Mean±SD (Median)	
1	37.75±4.65 (37.95)	41.62±8.45 (44.20)	**0.173**
2	15.10±4.10 (16.00)	15.15±4.50 (15.55)	**0.753**
3	30.15±12.58 (35.80)	31.82±10.10 (30.40)	**0.917**
4	26.37±15.27 (18.80)	21.07±10.38 (18.35)	**0.345**
5	41.00±2.28 (40.85)	38.97±4.16 (39.05)	**0.249**
6	12.15±1.07 (12.00)	11.37±2.25 (11.50)	**0.686**
7	37.75±14.65 (38.25)	35.60±19.37 (34.90)	**0.917**
8	16.63±3.45 (15.80)	16.45±3.01 (16.95)	**0.917**
9	15.52±3.51 (15.30)	8.52±5.56 (6.35)	**0.028***
10	26.08±8.59 (29.35)	23.60±9.70 (27.30)	**0.600**
11	26.58±6.68 (27.50)	21.40±10.55 (22.25)	**0.345**

Wilcoxon Signed Ranks Test **p<0.05*



In control group;

1. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is increase in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this increase was not statistically significan ($p>0.05$).

2. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is increase in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this increase was not statistically significan ($p>0.05$).

3. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is increase in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this increase was not statistically significan ($p>0.05$).

4. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan ($p>0.05$).

5. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan ($p>0.05$).

6. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan ($p>0.05$).

7. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan ($p>0.05$).

8. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan ($p>0.05$).

9. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is statistically significan reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour) (p:0.028; p<0.05).

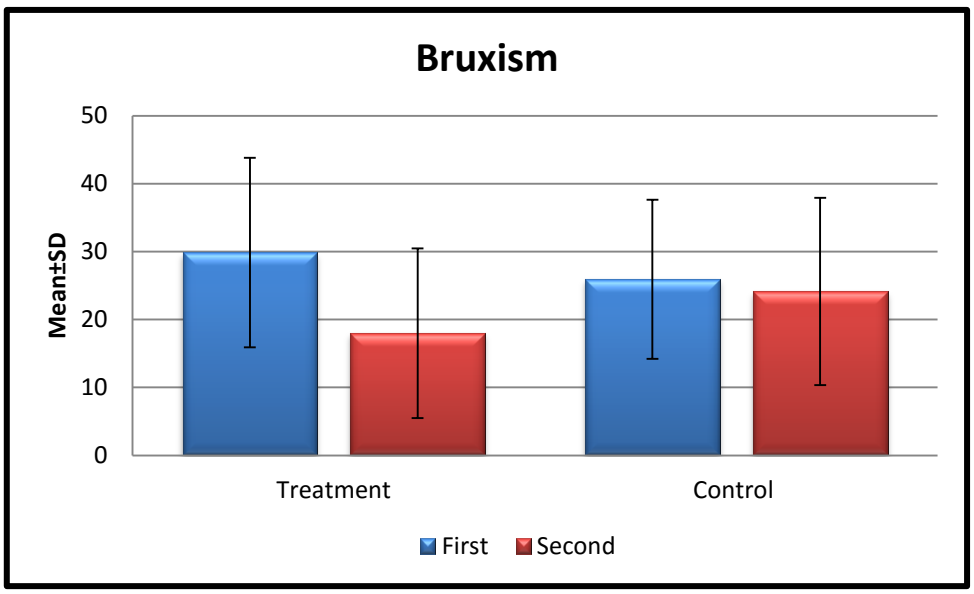
10. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan (p>0.05).

11. As compared to the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), there is reduction in the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour), however this reduction was not statistically significan (p>0.05).

Table 10. evaluation of bruxism levels of post-treatment and control groups

Pooled Data		**Treatment (n=120)**	**Control (n=66)**	**p**
		Ort±SS	**Ort±SS**	
Bruxism	**First**	29.89±13.95	25.92±12.48	**0.056**
	Second	17.99±11.72	24.14±13.81	**0.003****
	Difference	-11.90±15.71	-1.78±11.06	**0.001****

Student t Test ***p<0.01*



There is no statistically significant difference between the pre-treatment baseline temporalis muscle nocturnal bruxism activity (number of EMG events/hour) of treatment group and the first 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour) of control group ($p>0.05$).

There is statistically significant difference between the post-treatment temporalis muscle nocturnal bruxism activity (number of EMG events/hour) of treatment group and the second 6 continous nights temporalis muscle nocturnal bruxism activity (number of EMG events/hour) of control group ($p:0.003$; $p<0.01$).

There is statistically significant difference between the decrase in temporalis muscle nocturnal bruxism activity (number of EMG events/hour) of treatment group and decrease in temporalis muscle nocturnal bruxism activity (number of EMG events/hour) of control group ($p:0.001$; $p<0.01$).

5. DISCUSSION

Occlusal appliances have been commonly used in clinical practice to remove occlusal interferences, protect teeth and relax masticatory muscles. However, the effect of occlusal appliances on sleep bruxism is controversial; there is no common understanding regarding the effect of an occlusal appliances on nocturnal masticatory muscle activities among previous studies (8,261,262). Although positive effects of occlusal appliances have been shown in some studies (261,263,264), their exact mechanisms of action are still not clear, and no evidence supports their role in stopping sleep bruxism.

The aim of our study was to investigate the effect of the short term occlusal appliances therapy on the nocturnal electromyographic activity of the temporalis muscle in during Sleep bruxism.

In our research, a comparable EMG results obtained from 31 patients who had sleep bruxism; Subjects varied in age between 21 and 37 years with a mean age of 28.29±5.16 years, 12 (38.7 %) females and 19 (61.3 %) male. Patients were divided into two groups; Treatment group 20 patients (64.5 %), and untreated control group 11 patients (35.5).

5.1. Study Methodology

In most of the studies, bruxer subjects inclusion criteria and sleep bruxism activity are monitored with one of two methods: a) clinical signs and symptoms, and b) and/or recording of EMG baseline. Without monitoring baseline EMG, it is difficult to ascertain whether the populations studied were actually active sleep bruxers or not.

5.1.1. Baseline EMG studies

In our studies, the subjects are selected on the basis of questionnaire and clinical signs and the activity of bruxism was confirmed with acid etching technique and by using portable EMG recording device. The sleep bruxism activity was evaluated during the sleep with EMG recording device for 6 days before treatment and 6 days with occlusal appliances. We feel our data was used for statistical analyses, would reflect more dependable results than single night measurement studies.

At treatment group level, our results showed statistical significant reduction in nocturnal temporalis muscle activity while using occlusal splint in short term therapy (p:

0.001; p<0.01**)**. Moreover the treatment group shows significant improvement when compared to control group (no treatment) (p: 0.003; p<0.01). The finding of our study is in agreement with several studies (261,263,264) which showed a significant reduction in the EMG activity of masticatory muscles during sleep with occlusal appliances in situ. However, some other studies (262,265,266) found no such reduction in the EMG activity of masticatory muscles during sleep with occlusal appliances.

In 1975 the first published study evaluating bruxism patients undergoing occlusal appliances therapy, objectively measured the nocturnal masseter muscle activity, through electromyography by Solberg et al (261). The study monitored the effect of rigid full occlusal coverage maxillary stabilization splint on the muscle activities in 8 bruxer patients during sleep, Nightly recordings were made for three consecutive periods of approximately 10 days; before, during and following splint therapy. The results show that the nocturnal masseter muscle activity was reduced immediately following the insertion of the splint, and it remained low until the splints were removed. The EMGs done after the subjects stopped wearing the splints showed return of masseter activity to pretreatment levels in seven of the eight patients.

Pierce and Gale (263) evaluated the bruxing activity before, during, and after treatment with occlusal splints for two weeks in 20 bruxers by means of portable electromyography (EMG). A six month post treatment follow up of bruxing activity was obtained. Both EMG measured frequency of bruxing episodes and duration of bruxing activity decreased significantly for splint therapy. The two week treatment effects were transient, and bruxing activity generally returned to baseline levels when treatment was withdrawn.

Hiyama et al. (264) examine the effect of an occlusal appliance on nocturnal masticatory muscle activities in Six participate. Using a portable EMG recording unit; they recorded the electromyographic (EMG) activities of the right anterior temporalis and masseter muscles at night both with and without an occlusal appliance. The recorded night consisted of two parts; in the first half of the night, EMG activities were recorded without the occlusal appliance, then the subject was awakened to fit the occlusal appliance, and then sleep again, and the EMG activities were recorded. The results show that, in both muscles, the maximal EMG activity and the number of bruxing events decreased significantly by wearing the appliance. They suggest that nocturnal masticatory muscle activity is significantly reduced by wearing an occlusal

appliance, and that the use of such an appliance at night could help to relax masticatory muscles.

In sleep laboratory study (267) the participants evaluated for sleep quality and sleep bruxism activity at baseline, and after 1 and 3 months of using mandibular advancement device and mandibular occlusal splint. The results of this study showed that, after 1 month both the mandibular advancement device and mandibular occlusal splint reduce the sleep bruxism episodes but not significantly, while after 3 months both the MAD and mandibular occlusal splint provided significant decrease in the mean number of episodes per hour.

Some other study (262,265,266) reported the contrary for our results and previous study. Kydd and Daly (262) compared the electromyographic activity of the masseter muscle before and after use of occlusal appliance in ten bruxer subjects. EMG was recorded for three nights before the splint was used. After 2 weeks of splint using, EMG was recorded for three nights while using the splint. Authors found that the occlusal splints worn at night did not significantly reduce bruxism activity.

Baad-Hansen et al. (265) evaluate the effect of the occlusal splint and nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep. In randomized order, the 10 patients wore the splint during sleep for 1 week without EMG recording followed by 1 week with the splint and EMG recording (NTI + EMG/OS + EMG). After 1–2 weeks of washout period (to eliminating the carryover effect), another baseline EMG recording without splint was done followed by 2 weeks with the other type of splint, one without and one with EMG recordings. The occlusal splint showed no significant change, while the nociceptive trigeminal inhibitory splint showed significant decrease in bruxism activity. It is important to emphasize that the study sample size is relatively small (10 subjects).

Moreover van der Zaag et al. (266) assess the efficacy of occlusal stabilization splints in the management of sleep bruxism in relatively small sample (11 subjects). Two polysomnographic recordings that included bilateral masseter electromyographic activity were made: one prior to treatment, the other after 4 weeks of wearing the splint for 24 hours a day, except during eating. The number of bruxism episodes per hour of sleep (Epi/h), the number of bursts per hour (Bur/h) was established as outcome variable. Results show that occlusal stabilization splints not significantly reduce the sleep bruxism outcome variables measured on a group level. In this study, the results were based on a single night's recordings of each period; knowing that sleep bruxism

can itself fluctuate from night to night (63), one can argue that this variability may have modified the values of the oromotor outcomes. Moreover, fulltime use of occlusal splints could be a potential confounding factor due to its habituation effect. Another noteworthy element is that the study sample size is relatively small (11 subjects).

Some EMG studies investigate the effect of occlusal appliances on the masticatory muscles while the study subjects were awake (voluntary daytime recordings); did not have a sleep study, and they do not evaluate the changes of sleep bruxism during the sleep. A study (268) was performed to 9 temporomandibular joint dysfunction patients. In this research, the electromyographic activity of masseter and temporal muscles measurement was performed during maximum intercuspation (isometric contraction) before and after wearing occlusal splint for 2 weeks. The test results show that there is a statistically significant decrease in the contraction activities of the masticatory muscles after 2 week of wearing occlusal splint. In addition, there is also a correlation between the decrease in the electromyogram descriptive value in the masticatory muscles and the decrease in the temporomandibular joint dysfunction symptoms.

Landulpho et al. (269) studied the effect of occlusal splint therapy on TMD and EMG activities of masseter and temporal muscles in 22 patients with signs and symptoms of temporomandibular disorder which evaluated with the computerized electromyography. Electromyographic evaluations of masseter and anterior temporalis muscles were performed during the mandibular isometric clenching before inserting and after 90, 120 and 150 days of using the occlusal splints. The results showed that there were significant reductions in the electromyographic activity along the entire period of treatment during the mandibular isometric clenching for masseter and anterior temporalis muscles. The authors showed that the longer the therapy with splints, the more reduced was the EMG activity of masticatory muscles.

Daytime electromyographic study of the masseter and anterior temporal muscles was performed on fifteen bruxism patients with clinical symptoms such as muscle pain fatigue and tenderness, Hamada et al. (270) observed significant reductions in the masticatory muscle activity of bruxers. The post-treatment values were similar to those of healthy control subjects.

In a different study, Savabi et al. (271) investigate the effectiveness of occlusal splints on the electromyographic activity of the masseter and temporal muscles in 25 healthy individuals. Surface electromyographic recordings were done during maximum

clenching without the splint and immediately after application of the splint. On the contrary to previous voluntary daytime record studies, the result showed no significant difference between the electromyographic activities of both muscles before and after insertion of the splints. Investigators concluded that immediate application of occlusal splints has no significant effect on the activity of masseter and temporal muscles.

Visser et al. (272) assessed the effect of the use of a stabilization splint in a group of 35 myogenous craniomandibular disorder patients for three to six weeks. The patients were clinically examined and surface EMG recordings of the temporal and masseter muscles were made during clenching in the intercuspal position, immediately after the insertion of the splint, and after at least three weeks of splint treatment. The results show 3 groups; one group showed a decrease in temporal muscle activity during splint treatment, while another did not show any significant change, and the third group showed an increase of temporal muscle activity. In general, significant reductions in the amount of static pain were found. In the group with a significant reduction of temporal muscle activity (n = 15) there was a greater decrease in the amount of static pain than in the group (n = 4) with a significant increase of temporal muscle activity.

The postural activity (mandible at rest) of the temporal and masseter muscles in 31 patients with signs and symptoms of functional disorders and nocturnal bruxism was studied by Sheikholeslam et al. (273): before, during, and after three to six months of occlusal splint therapy. The fluctuating signs and symptoms, as well as the postural activity of the temporal and masseter muscles, were significantly reduced after treatment. Further, the coefficients of correlation within pairs of postural activity of the right and left muscles increased significantly. In about 80% of the patients, the signs and symptoms return to the pretreatment level within one to four weeks after splint therapy was stopped. They conclude that an occlusal splint can eliminate or diminish signs and symptoms of functional disorders and re-establish symmetric and reduced postural activity in the temporal and masseter muscles.

In contrast to previous study, Nascimento et al. (274) evaluates the effect of occlusal splint therapy on TMD and EMG activities of masseter and temporal muscles in patients with sleep bruxism. The authors followed 15 patients with signs and symptoms of TMD during 60 days of splints wearing; they using surface electromyography records of masseter and temporalis muscles in a mandibular rest position for 10 s after a maximum biting, as well as the Helkimo Index before and after 60 days of occlusal splints use. The authors showed that the occlusal splint presented a

significant reduction in the clinical signs and symptoms of TMD after 60 days of occlusal splints therapy, and seem to benefit patients with more severe symptoms of sleep bruxism. However, the EMG evaluation of the masseter and temporal muscles did not indicate a significant decrease in mean EMG levels over the therapy in the muscles.

Amorim et al. (275) examined the effects of an occlusal splint on the electric activity of the masseter and anterior temporalis muscle in 15 women who presented with sleep bruxism and temporomandibular disorders related to occupational stress. The EMG signals compared between pre and post splint records, at both resting and maximal clenching effort in two situations. The first was after a workday without using the occlusal splint; and the second, after a sleeping night using occlusal splints. Author observed significant difference between pre and post splint, in the maximal clench condition for right and left masseter muscles and right anterior temporalis muscle, and in rest position condition between pre and post splint for right and left masseter muscles. No significant difference was found between pre-splint and post-splint for and left anterior temporalis muscles in rest position and in the left anterior temporalis muscle in maximal clinching effort. The author suggests that the use of occlusal splint reduces EMG activity in the masseter and anterior temporalis muscles, in patients who presented with sleep bruxism related to occupational stress.

Although our results at group level showed significant reduction in nocturnal temporalis muscle activity during short occlusal appliances therapy, an individual difference in changes of sleep bruxism activities by wearing occlusal appliances is observed in this study. According to the individual data, 13 of 20 subjects showed statistical significant reduction in the sleep bruxism activity, 3 of 20 subjects showed reduction in the sleep bruxism activity however this reduction was not statistical significant, and 2 of 20 subject showed no difference between pre-treatment and post treatment sleep bruxism activity. On the contrary, 2 subjects showed increase in sleep bruxism activities while using occlusal appliance as compared to baseline data.

The individual difference in changes of sleep bruxism activities by wearing occlusal appliances was observed in many previous studies. Clark et al (8) monitored the effect of full arch maxillary occlusal splint therapy on Nocturnal electromyographic activity of the masseter muscle in 25 patients with symptoms of myofascial pain and abnormal jaw function. The Nocturnal EMG recorded for 10 nights before treatment with occlusal splints, after wearing the splints all night and as much of the day as possible for at least 14 days, another 10 night Nocturnal EMG levels were recorded. In

this studies Different results were obtained, a decreased nocturnal EMG level during treatment was noted for only 52% of the patients (13 patients), in 28% (7 patients) no change was shown and in 20% (7 patients), an increase was shown in nocturnal EMG levels. A return to pretreatment EMG levels after removal of the splint was noticed in 92% (12 of 13 patents) of the patients.

Rugh et al (276) evaluate the effect of the canine and first molar guidance splint in eight chronic bruxer patients for 10 to 14 nights. Results showed significant variations at the individual level. For both types of splints decreased EMG activity in three of the eight study subjects; another three subjects showed increased EMG activity; and no changes were observed in another subject. The remaining subject showed increased EMG activity while wearing the splint with canine guidance and no variation while wearing the molar disoclusion splint.

The individual difference in changes of sleep bruxism activities by wearing occlusal appliances was also observed by Harada et al. (193) in 6 weeks study. His results showed significant reduction in sleep bruxism immediately after the insertion of both occlusal splint and palatal splint devices. However, no reduction was observed in 2, 4 or 6 weeks. According to the individual data on, constant reduction in sleep bruxism activities was observed in seven subjects for occlusal splint and six for palatal splint throughout the 6-week recording period. Among these subjects, five for occlusal splint and two for palatal splint showed distinct reduction (>40% reduction from the baseline). On the contrary, only one subject showed constant increase in sleep bruxism activities for both splints throughout the recording period. The other subjects showed mixed changes, i.e. both increase and decrease, during the recording period.

Sjoholm et al. (277) monitor the EMG effects of occlusal stabilization splints. Recordings were taken before participants started wearing occlusal splints and after 2 months of wearing them. The results show that the bruxism levels decreased in only 36% of participants; 43% of participants showed increased activity, and no changes in the remainder 21%.

Although on the group level, Van der Zaag (266) found that both occlusal stabilization splints and palatal splints had no influence on the number of bruxism episodes per hour of sleep, or the number of bursts per hour, in individual level he found variable outcomes; Some patients show an increase of greater than 50% in their individual bruxism outcome variables, or a decrease in sleep bruxism of less than 50% in their individual bruxism outcome variables, while others showed no change.

Our findings related to increased muscle activity with occlusal appliances in situ, are in accordance with many previous studies; Clark et al. find 5 of 25 patients (8), Rugh et al. find 4 of 8 patients (276), Dube et al. find 1 of 9 patients (6), van der Zaag et al. find 10 of 21 patients (266), Harada et al. find 1 of 16 patients (193) and Landry et al (278) find 1 of 13 patients have increased muscle activity during splint therapy. Holmgren et al. (279) suggested that in extremely hypertonic muscles, the increase in vertical dimension can stretch the spindles of the mandibular elevators muscle to an extent that the afferent feedback from these causes additional increase of tonic activity. They also suggested that thickness of splint keeping the mouth opening more than the clinical rest position can stimulate pain and other receptors in muscles, tendons and/or joints, which can influence and even increase muscle contraction. Moreover, pressure from the splint to already periodontally traumatised teeth due to bruxism can also stimulate periodontal pain receptors, which may result in increased muscle tension. However, studies using palatal splint where there is no increase of vertical dimension or excessive pressure placed on teeth, still subjects showed increased sleep bruxism activity (266).

The effect of occlusal appliances on the nocturnal masticatory muscles activity could be transient and not strong enough to reduce the sleep bruxism activity for a long time due to the adaptation of the stomatognathic system to the occlusal appliances (193); Dube et al. (6) compared the efficacy of a hard occlusal splint versus a palatal control splint for two weeks; he concluded that both devices reduce muscle activity associated with bruxism. On the contrary, Van der Zaag et al. (266) did not observe significant effects for either hard occlusal splint or palatal control after four weeks of usage, and observed large differences between individual sleep bruxism patients. The combination of the results of both studies corroborates the finding by Harada et al. (193) that wearing either hard occlusal splint or palatal splint for short durations significantly decreased sleep bruxism EMG compared to pre-treatment. However for treatment provided longer than 2 weeks, neither splints produced any significant decrease in sleep bruxism EMG.

Moreover Matsumoto et al (280) compared the effect of intermittent use of occlusal splints on sleep bruxism with that of continuous use by measuring masseter muscle electromyographic activity using a portable electromyographic recording system in 20 bruxers. 10 Subjects in the continuous group wore occlusal splints during sleep for 29 nights continuously; the intermittent group wore splints during sleep every other

week (1st–7th, 15th–21st and 29th nights). Electromyographic activity of the masseter muscle during sleep was recorded for six time points: before, immediately after, and 1, 2, 3 and 4 weeks after the insertion of the occlusal splint. The results shows that, in the continuous group, nocturnal masseter electromyographic events were significantly reduced immediately and 1 week after the insertion of the splint, however no reduction was observed at 2, 3 and 4 weeks after insertion. In the intermittent group, nocturnal masseter electromyographic events and duration were significantly reduced immediately after and also 4 weeks after insertion of the splint. The authors suggest that the intermittent use of splints may reduce sleep bruxism activity for a longer period compared with that of continuous use.

5.1.2. Clinical signs and symptoms studies

With subject selection in these studies, sleep bruxers were selected on the basis of self-report and clinical signs and symptoms instead of objective EMG evaluation. Without monitoring baseline EMG, it is difficult to ascertain whether the populations studied were actually sleep bruxers.

Mejias and Mehta (281) assessed the individual responses of bruxers to splint therapy for one month. They use a special bruxism monitoring device ‘Bruxcore’ to check the pretreatment and post treatment nocturnal bruxism. They found that their all five participants reacted favorably to the treatment. They suggested that the short term splint therapy is effective in eliminating mild to moderate facial pain and dysfunction while reducing bruxism behavior. In this study the bruxism was assessed by wearing of ‘Bruxcore’, which itself may interfere with the bruxism behaviour as it an occlusal splint.

Ommerborn et al. (282) evaluate the efficacy of an occlusal splint treatment in 29 sleep bruxism patients over a period of 12 weeks, with 6 months follow up. Patients were examined pretreatment, post-treatment, and at 6 months of follow up for sleep bruxism activity with Bruxcore device, self-assessment of sleep bruxism activity. The analyses demonstrated a significant reduction in sleep bruxism activity, self-assessment of sleep bruxism activity, after 12 weeks of occlusal splint therapy that continued into a 6 months follow up.

Hachmann et al. (283) compared two groups of children with bruxism, one group were not submitted to treatment (control), the other group use nocturnal bite plate. Cast models were made for both groups, to evaluate the progression of wear

facets, during 8 months. The 4 children of the control group displayed increased wear facets during the study period, while the 5 children that used nocturnal bite plate, showed no increase of wear facets, even after the removal of the device. The authors conclude that the use of nocturnal bite plate is efficient against bruxism in 3 to 5 year old children.

In a study (284) to determine the relief time for temporomandibular disorders signs and symptoms in 30 patients with sleep bruxism treated with occlusal splints for a period of 180 days; the result for all variables was statistically significant. As to the TMD symptoms, in most patients the relief of pain in masseter, temporalis, cervical and TMDs occurred in the 3rd month. 20 % of the patients were aware of clenching teeth while awake and reported that this parafunction decreased by the end of 6 months, and 90% reported an improvement in sleep quality as well. The authors conclude that the occlusal splints were effective to treat temporomandibular disorders in patients with sleep bruxism, as well improved subjective symptoms.

A study (285) compared the success of 24 Hour occlusal splint therapy versus nocturnal occlusal splint therapy in reducing craniomandibular disorders (myogenous pain) in 64 patients. Patients were divided into two equal groups. One group received 24-hour splint therapy and the other received nocturnal splint therapy. Over the 8-week assessment period, it was found that nocturnal splint use was more successful in patients suffering from myogenous pain whereas patients with more arthrogenous sources of pain bene fitted from continuous splint use. Authors suggested that the cycling effect of the nocturnal splint constantly disrupted muscle habit patterns.

In 6 weeks of active treatment, patients diagnosed with myofascial pain who received full coverage hard maxillary stabilization splints, Glaros et al. (286) tested the hypothesis that pain reduction produced by splints is associated with reduction in parafunctional activity. The studies included 2 groups; one group instructed to maintain or contact with the splint. The other group instructed to avoid contact with the splint. Patients were reminded approximately every 2 h by pagers to maintain/avoid contact with the splint. In the results; Patients who reduced tooth contact intensity the most reported greater relief from pain. Authors suggest that the splints may produce therapeutic effects by reducing parafunctional activities associated with TMD pain.

Sheikholeslam et al. (287) studied the long-term effects of a full arch maxillary plane occlusal splint on chronic signs and symptoms of craniomandibular disorders in 31 patients with nocturnal bruxism. The splint was constructed as flat as possible,

without canine guidance ramps. The treatment with the occlusal splint continued until total elimination of signs and symptoms was attained or no further improvement could be observed during a period up to 6 months. With use of the occlusal splint the chronic signs and symptoms of craniomandibular disorders (except Joint sounds) diminished successively in 87% of the patients. The results showed that the score and intensity of signs and symptoms in this type of patient fluctuate from day to day and even within a single day. Although the symptoms of craniomandibular disorders were cured or improved with the long term use of the occlusal splint, the nocturnal bruxism continued. However, in general, the symptoms recurred to pretreatment level in less than 4 weeks after discontinuation of splint therapy.

Yap (288) investigate the short term effects of stabilization appliances on bruxing and clenching during sleep in 21 patients with and without signs of temporomandibular disorders. Patients were reexamined at 1 week and monthly intervals up to 3 months. For both patients with and without temporomandibular disorders, active shiny facets caused by nocturnal clenching were consistently present on the occlusal surfaces of appliances at every visit. Although stabilization appliance therapy is effective in eliminating the signs of temporomandibular disorders evaluated (except of TMJ clicks), the results revealed that stabilization appliances do not stop nocturnal parafunctional activities in both groups of patients.

Sheikholeslam et al. (287) and Yap (288) used awareness of bruxism behaviour and wear facets on the occlusal splint, in combination with indirect clinical measures as outcome variables, which can renders these studies inconclusive.

Holmgren et al. (182) studies the effects of the occlusal splint on grinding and clenching during sleep in 31 patients with bruxism and craniomandibular disorders for 6 months. A great reduction in symptoms related to TMD, like pain on temporal and cervical regions, headaches, TMJ pain, and noises. Despite considerable relief, the results revealed that the splint does not stop nocturnal bruxism. In 61% of the patients, wear facets on the splint were observed at every visit (2-week) and in 39%, from time to time. The wear facets reappeared in the same location with the same pattern and were caused mainly by grinding.

5.2. Effect of occlusal appliances Designs on bruxism

The reduced bruxing frequency produced by occlusal appliances use can be explained in several ways, depending on the preferred etiological theory and occlusal appliance design; Occlusion, vertical dimension, canine guidance, freedom of movement of the mandible.

5.2.1. Occlusion

As occlusal interferences was believed to be precursors to bruxing (24), then occlusal appliances effect due to removal of interferences by providing an occlusal balanced acrylic surface between the teeth (229–231). Riise and Sheikholeslam (289) investigate the effects of an intercuspal occlusal interference on the pattern of postural activity of the anterior temporal and masseter muscles in eleven patients with complete, natural dentitions by the insertion of a single occlusal interference (high amalgam filling). The results indicate that there is postural activity in the anterior temporal and sometimes in the masseter muscles. The EMG pattern of postural activity is influenced by the occurrence of an experimental occlusal interference, sometimes as early as 1 h after the insertion. After 48 h there was a significant increase of the activity in the anterior temporal muscles. This increased activity persisted until the interference was removed 1 week later and had almost disappeared 1 week after the removal.

In contrast with the findings of previous study, Rugh et al. (26) studies the influences of artificial occlusal interferences, incorporated in crowns in the molar region, on masticatory muscle activity during sleep. The crowns remained in place for 10 to 21 nights, after which the discrepancy was eliminated through occlusal adjustments. Nocturnal bruxism was monitored before, during, and after the deflective occlusal contact was placed the masticatory muscle activity was quantified by means of EMG recordings from the sleeping patient. The results show that the artificial interferences caused a significant decrease of sleep related masticatory muscle activity in 9 of 10 subjects (90% of the cases).

In other studies, the elimination of interferences in occlusion and articulation did not influence the bruxism activities (27,28). Moreover, not every bruxer has occlusal interferences and not every person with such interferences is a bruxer (29).

Dube et al (6) studies the effect of occlusal splint and palatal splint (no change in the occlusion) on the bruxism. This study extends for 5 weeks and Patients were given 2 weeks to get used to the splint. The authors found that both palatal splint and occlusal

splint significantly reduced bruxing frequency compared to no splint, and there was no significant difference in effectiveness between the two splint designs**.** The reduced sleep bruxism EMG produced by the palatal splint can't be explained as removal of the interferences because the appliance did not directly alter tooth contacts.

Harada et al. (193) also compared the efficacy of occlusal splint and a palatal device in 16 patients during 6 weeks with each device and used an interval of 2 month to a washout. The authors found no statistically significant difference between the two types of device, but the findings showed that there was statistically significant reduction in the masseter EMG activity immediately after the insertion of splints. However, on group level, they were no significant changes in 2, 4, and 6 weeks after the insertion of either splint.

Moreover, Baad-Hansen et al. (265) in study for 2 weeks splint treatments found no significant reduction in EMG activity in the masseter during sleep when patients used a standard flat occlusal stabilization splints with evenly distributed occlusal contacts and group contact on laterotrusion and protrusion.

According to these findings, it was considered that the change in occlusal contacts between maxillary and mandibular dental arches with occlusal appliances might not be a primary factor of the reduction of sleep bruxism activities.

5.2.2. Vertical dimension

The vertical thickness of occlusal appliances is an important consideration in the treatment of patients and may directly influence the clinical effect. Manns et al (226) and Christensen (290) reported that the vertical thickness of occlusal appliances should be set up beyond the mandibular rest position; however, Ramfjord and Ash reported that it should be as thin as possible.

Some authors (291,292) reported that increasing the occlusal vertical dimension beyond the clinical rest position could cause a reduction of elevator muscle EMG activity. Garnick (292) and Rugh (291) suggests that the decrease in muscle activity as a result of an increase in vertical dimension due to the presence of reciprocal activation of the motor neurons of the jaw depressor muscles and simultaneous inhibition of the elevators.

Manns et al (226) study the influence of occlusal splints constructed at three different vertical dimensions in the etiology of bruxism and myofascial pain dysfunction syndrome. The vertical dimension of least EMG activity was determined

for each of 75 patients who were randomly divided into three groups according to the vertical dimension at which the occlusal splint was constructed. Results showed a faster and more complete reduction in clinical symptoms for groups the occlusal splints were constructed at 4.42 mm, and 8.15 mm from the occlusal vertical dimension than for group occlusal splints were constructed at 1 mm from the occlusal vertical dimension. The temporary use of occlusal splints with a vertical height exceeding the physiologic rest position did not encourage a greater muscular tonus or hyperactivity of jaw muscles. They concluded that elongation of elevator muscles to or near the vertical dimension of least EMG activity by means of occlusal splints is more effective in producing neuromuscular relaxation.

Christensen (290) studies the effect of occlusal splint on maximal voluntary tooth clenching in six subjects. The electrical activity in the masseter muscle was recorded during maximal voluntary tooth clenching until masseter muscle fatigue. An occlusal splint was inserted and the clenching exercises were repeated, the fatigue threshold and the pain tolerance of the muscle were determined in seconds. The result shows that the use of splint did not significantly changes the subjective sensations of onset of fatigue. The use of the splint caused a significant decrease in the electrical activity of the pain tolerance test. They conclude that the mode of action of the splint in reducing the muscle activity may be due to stretching the elevator jaw muscles beyond their resting length.

In converge to our finding using 3 mm vertical thickness of occlusal appliances, the initial effects of the vertical thickness of occlusal splints on the electromyographic (EMG) activities of temporal and masseter muscles during sleep in subjects with a nocturnal bruxism habit were investigated (293). Two types of splint were made for 12 patients: a splint with a 3 mm vertical thickness at the central incisors (S3) and a splint with a 6 mm vertical thickness (S6). The muscle activities of the left anterior temporal muscles and masseter muscles were recorded without occlusal splints (NS), with the S3 splint, and with the S6 splint by a portable EMG recorder during sleep at night. In contrast to the previous studies, the results shows that the EMG values of masseter and temporal muscles decreased following insertion of the S3 splint but were not significantly affected by the S6 splint. Six subjects in masseter EMG and 7 subjects in temporal EMG got worse with the S6 splint compared to NS. They conclude that, in bruxism managements, the splint with 3 mm vertical thickness was superior to the splint with a 6-mm vertical thickness.

However, three studies (219,294,295) compared the effects of occlusal splints with canine guidance and occlusal splints with group function guidance (which made at equal vertical dimension) on EMG activity. The results show that the canine guidance significantly reduced temporalis muscle and masseter muscle EMG activity. Such muscle activity decrease can't be ascribed to increased vertical dimension, since both types of splint compared in these studies were ware made at equally increase vertical dimension.

In other study, Okeson (214) compared the effects of hard and soft occlusal splints on nocturnal bruxism. The vertical dimension as well as tissue coverage for the two splint designs used was very similar. Significant decrease in muscle acivity has been found in subjects using a hard splint, while increased sleep bruxism activity has been found in subjects using a Soft splint. Since the vertical dimension for the two splints was very similar, that can eliminates the vertical dimension as potential factors.

Moreover, Holmgren (279) suggested that the opening of the mouth beyond the clinical rest position can stimulate pain and other receptors in the muscles, tendons and/or joints, which in turn can influence and even increase muscle contraction.

5.2.3. Canine guidance

The occlusal appliances constructed with canine guidance was suggested by Ramfjord and Ash (25) to eliminate dental interference on the non-working side during the lateral movement of the jaw. D' amico's suggested that the periodontal feedback from canines could reduce masticatory muscular forces (296). Occlusal appliances constructed with molar guidance is based on Lous' use of a "pivot splint" that has contacts only in the second molar which suggested to relieve temporomandibular joint clicking and other symptoms (297).

Williamson and Lundquist (219), compared the EMG effects of splints with canine guidance and splints with group function in 5 subjects sample. The results show that the canine guidance significantly reduced temporalis muscle and masseter muscle EMG activity. The authors suggested that this result due to the effect produced by the canine eliminating interferences at the level of posterior teeth.

Shupe et al. (295) compared splints with canine guidance and splints with group function in 9 subjects sample, the authors obtained similar results; the use of canine guidance caused muscle activity to decrease during the lateral tooth grinding.

Manns et al. (294) also compared the effects of group function splints with canine guidance splints in eight healthy young adult subjects; the results show that the canine guidance splints was significantly reduce elevator muscle activity during lateral movements than with the intermediate and posterior group function.

Moreover, other studies (298–300) reported a significant reduction in the muscle activity developed through tooth clenching during the maximum dental inter cuspation when such activity was performed laterally over canine guidance.

However, Graham and Rugh (301) compared canine guidance splints with first molars disclusion splints in 10 subjects sample. In contrast to the finding of the above studies, both types of splint were found to reduce muscle activity in a similar way during lateral movements under tooth clenching in the lateral jaw position. The authors conclude that the reduced EMG activity resulted from the contact with one tooth only (canine or first molar), thus no longer ascribing the results exclusively to canines.

Moreover, Rugh et al (276) compared the effect of the canine guidance splint with first molar splint in eight chronic bruxer patients. The appliances were used for 10 to 14 nights. Both types of splint produced nearly similar effects while worn during sleep on nocturnal bruxism in seven of eight subjects; there is no significant difference in treatment effectiveness between occlusal splints with canine guidance vs. occlusal splints with first molar guidance. However, results showed significant variations at the individual level. Reduced EMG activity was reported in three of the eight study subjects; another three subjects showed increased EMG activity; and no changes were observed in another subject. These results were similar for both types of splints. The remaining subject showed increased EMG activity while wearing the splint with canine guidance and no variation while wearing the molar disclusion splint. Clinical examination and subjective pain ratings did not differ with the two guidance patterns. They conclude that these results question the common assumption that canine guidance is a critical design feature for the management of nocturnal bruxism and associated craniomandibular symptoms. The assumption that the therapeutic mechanism of splint therapy is via a reflexive inhibition of muscle activity through canine guidance is not supported by this study, it could be something other than canine guidance is responsible for the positive clinical effects.

The nociceptive trigerninal inhibition (NTI) splint is a splint that contacts only the incisors and not contacts the canine. Baad-Hansen et al. (265) found a significant reduction in EMG activity in the masseter during sleep when patients used nociceptive

trigeminal inhibitory (NTI) splint which not contacts the canine, in contrast, found no significant reduction in EMG activity in the masseter during sleep when patients used an occlusal splint with group contact on laterotrusion.

5.2.4. Freedom of movement of the mandible

According to the design of intraoral devices; some of them provide free mandible movement while the others restrict these movements. Anterior bite plane (Jeanmonod's bite plane) (302) is a flat anterior splint which allows contact of the six anterior teeth only, premolars and molars being in complete disclusion, and gives a posterior occlusal clearance of approximately 0.4mm. This allows freedom of movement of the mandible and removes any potential posterior intercuspation contacts.

Jeanmonod (302) evaluate the effect of the anterior bite plane on nocturnal parafunctional muscle activity in 10 bruxers in a sleep lab. The first record were taken before the use of the anterior bite plane, the second record were taken after the use of the anterior bite plane. All the patients showed decreased bruxism while wearing Jeanmonod's bite plane.

Moreover, Okkerse et al. (303) evaluate the effect of the anterior bite plane on the muscular activity of Masseter and Anterior Temporalis muscle in 21 bruxer patients during sleep. The results show a significant decrease in nocturnal parafunctional muscle activity with the use of bite plane.

In 30 subjects study, Becker et al. (304) compare the electromyographic activity of the masticatory muscles during clenching and grinding with and without the anterior bite plane. The results show significant reduction in electromyographic muscle activity of masticatory muscles during both clenching and grinding with anterior bite plane, except the anterior digastric muscle.

Lars et al. (305) compared the effect of the anterior bite plates and occlusal splints on the electromyographic activity in the masseter and temporal muscles during rest position, and gentle and maximum biting after use of the splint for 1 week. While both devices provide free mandibular movements, the author found that the EMG activity was significantly lower in the rest position in the temporal muscle after use of the occlusal splint than after use of the anterior bite plate.

The nociceptive trigerninal inhibition splint (a variation of Jeanmonod's splint, the difference being that nociceptive trigeminal inhibition splints do not extend beyond the incisors) is also allows freedom movement of the mandible. Baad-Hansen et al.

(265) found a significant reduction in EMG activity in the masseter during sleep when patients used nociceptive trigeminal inhibitory splint. Another study (306) found that the NTI splint reduced the intensity of the masseter and temporal muscle activity, whereas the stabilization splint showed no effect on this parameter.

On the contrary to the occlusal appliances that provide free movement of the mandible, Landry et al (278) compare the effect of a double arch mandibular advancement device and a maxillary occlusal splint on sleep bruxism and tooth grinding activity. In that study, it was found that both the maxillary occlusal splint and a non-protruding mandibular advancement device (allow full freedom of mandibular movements while sleeping) moderate reduced the frequency of sleep bruxism. However, a greater reduction (almost double) was founded with activation (minimum/intermediate protrusion) of the mandibular advancement device. The authors hypothesized to explain the reduction of sleep bruxism with the mandibular advancement device is restriction of mandible movement.

Moses (307) used daytime EMG measures to assess the restraining effect of his Passivator appliance which is an inter-arch acrylic appliance constructed in an edge to edge trajectory and separates the arches by 1.5 mm with acrylic in 18 bruxer patients; movement of the mandible is limited to opening and closing in an anterior trajectory which prevent significant lateral movement of the mandible. He measure electrical activity in on anterior temporalis and masseter muscles in maximal clench in both centric occlusion and in the corrected jaw position with the Passivator in place on each patient. The results of this experiment seem to indicate that the activity of muscles can be reduced by use of this restraining appliance. This study used indirect clinical measures of bruxism only (e.g. dental and musculoskeletal pain complaints) and not confirmed with EMG, moreover, this study did not have a sleep study, and they do not evaluate the changes of sleep bruxism during the sleep consequently, this study is difficult to interpret.

5.3. Comparison of different intraoral devices

There are many types of intraoral devices, they may be full or partial or no occlusal coverage, repositioning or stabilizing, made from hard or soft material.

5.3.1. Soft Splints

Pettengil et al. (308) compared the efficacy of hard and soft stabilizing appliances in the reduction of masticatory muscle pain in patients with temporomandibular disorders. With a sample of 7 patients used hard appliance and 11 patients used soft appliance for 10 to 15 week period, he found soft and hard appliances performed the same in reduction of masticatory muscle pain. He suggests that soft and hard stabilizing appliances may be equally useful in reducing masticatory muscle pain in short term appliance therapy. This study used indirect clinical measures of bruxism and did not measure the muscle activity.

In study (309) to compare the self-care treatment without any intraoral splint appliance, a conventional flat plane hard acrylic splint, and a soft splint; Subjects completed questionnaires and clinical examinations at 3, 6 and 12 months. There were no differences among groups for occlusal changes, either by self-report or by clinical evaluation using full arch articulating paper. The authors conclude that all patients improved over time, and traditional splint therapy offered no benefit over the soft splint therapy. Neither splint therapy provided a greater benefit than did self-care treatment without splint therapy.

Quran and Lyons (310) did a study to compare the effects of hard and soft splints on the activity of the anterior temporalis and masseter muscles in ten healthy young adults. Surface EMG recordings were made during clenching at 10% of maximum, 50% of maximum and at maximum clench, both before and after insertion of hard splint. This sequence was then repeated with a soft splint. The relative level of activity in the anterior temporalis and masseter muscles at all three activity levels were quantified by means of an activity index, which provides a measure of the balance of activity in the masseter relative to the activity in the anterior temporalis muscle. They were found that the hard splints led to a decrease in EMG activity in both muscles at maximum clench and particularly the anterior temporalis. Soft splints produced a slight increase in activity of both muscles, but particularly the masseter muscle. They suggest that the decrease in the activity of temporalis muscles relative to the masseter muscles is the therapeutic effect of both a hard and soft splint. Authors conclude that the hard

splint is likely to be more effective than a soft splint in reducing the activity of the jaw closing muscles, especially the anterior temporalis muscle.

In study (311) to compare the effectiveness of hard and soft occlusal splint in the management of myofascial pain, 30 patients were randomly assigned for the splint group and soft splint group. Patient was evaluated on baseline, 7, 30, 60 and 90 days with objective pain analysis using muscular palpation score. The results showed that both hard and soft splints are effective but the hard splint is more effective compared to soft splint.

Electromyographic study done by Cruz Reyes et al. (312) assessing the influence of hard occlusal stabilization splints and soft occlusal splints on the electromyographic activity of temporal and masseter muscles during voluntary muscular contraction of patients with bruxism. In this study, 2 groups of 8 patients were evaluated, first group used occlusal stabilization splint, second group used soft occlusal splint for 46 to 60 days, and they were instructed to wear the occlusal splints 24 hours a day, except when they ate. In the occlusal splint group, the results shows significantly increase in muscle electrical activity in 5 patients and decreased slightly in 3. In the soft splint group, there was considerable reduction of electrical activity in 6 patients and a slight increase in 2. There is a statistically significant difference between the muscle electrical activity generated in the occlusal splint group and in the soft splint group. The authors suggested that the increase in muscle electrical activity in the hard splint group may have been due to a neuromuscular recovery process; while the decrease in the soft splint group might have been due to a negative or decremental process of muscular organization to prevent the recruitment of new motor units. They conclude that the occlusal stabilization splints are considered better than soft occlusal splints.

It is important to emphasize that the soft splint may produce unwanted occlusal changes (313). Shore (314) suggested that soft splints may become perforated due to the action of sleep bruxism; this can lead to orthodontic action. Moreover, there an occlusal changes in the form of dental intrusion occurred after use of these splints (315). Also one publication reported mild premolar and molar intrusions in 67% of the participants who wore soft splints (316).

On the contrary, in study to investigate the intraoral soft splints treatment for masticatory muscle pain, the author suggested that the soft splint is an effective short term treatment for reducing the signs and symptoms of masticatory muscle pain in patients, and the soft splint does not cause occlusal changes (317).

Ramfjord and Ash (25) suggested that these splints are unlikely to inhibit bruxism, since clenching on a resilient surface is stimulating for patients. Okeson confirm this hypothesis (214) through ambulatory EMG, he compared the effects of wearing a hard versus a soft occlusal splint on nocturnal bruxism in the same person, the hard maxillary stabilization splint and the soft splint were carefully constructed to the same vertical dimension. Compared with the baseline periods, he reported that 5 of 10 participants (50% in study population) showed a statistically significant increase in nocturnal muscle activity when wearing a soft occlusal splint. One patient showed significant decrease and four showed no change. In comparison, 8 of 10 participants showed a statistically significant decrease in nocturnal muscle activity when wearing a hard occlusal splint. Two patients showed no significant change.

5.3.2. Jeanmonod's bite plane

Jeanmonod (302) evaluate a group of 10 bruxers in a sleep lab over two nights. The first night record were taken before the use of the anterior bite plane, the second night record were taken after the use of the anterior bite plane for 3 to 4 weeks in 24 hours a day. The results showed decreased bruxism while wearing “Jeanmonod's bite plane”.

Okkerse et al. (303) evaluate the influence of the anterior bite plane on bruxism activity during sleep; The nocturnal muscular activity of Masseter and Anterior Temporalis muscle has been registered in 21 patients suffering from bruxism, prior and after 3 to 4 weeks treatment with a 24 h bite plane in place. The data shows a significant decrease in nocturnal parafunctional muscle activity with the use of bite plane. The author postulated that the reduction is due to a reflex inhibition of jaw muscle activity as a result of the sensory information gathered from the periodontal ligament of anterior teeth during contact with the splint.

Becker et al. (304) measured the effect of a prefabricated anterior bite stop on the activity of the anterior temporalis, posterior temporalis, masseter, and anterior digastric during clenching and grinding in 30 selected subjects. Electromyographic activity was measured during clenching and grinding both with and without the anterior bite stop. The results show that the anterior bite stop significant reduced electromyographic muscle activity for the anterior and posterior temporalis and the masseter muscles during both clenching and grinding, except the anterior digastric muscle. Author concludes that the anterior bite stop reduced electromyographic muscle

activity for the anterior and posterior temporalis and the masseter muscles during both clenching and grinding.

Lars et al. (305) study the influence of the anterior bite plates and stabilization splints on the electromyographic activity of the masseter and temporal muscles. Seventeen healthy subjects wore the anterior bite plate and the full coverage stabilization splint at night, each for 1 week. The EMG activity was recorded without appliances in situ, in the rest position, and during gentle and maximum biting before and after use of the different appliances. After use of the stabilization splint, the activity in the rest position was significantly lower in the anterior and posterior parts of the temporal muscles. The EMG activity was significantly lower in the rest position in both parts of the temporal muscle after use of the stabilization splint than after use of the anterior bite plate. In a control group of eight subjects in whom no appliances were used, the EMG activity did not change significantly between the initial and 1 or 5 week recordings. Authors suggest that the occlusal design of the appliances seems to be of importance for the influence on the EMG activity in the masticatory muscles, at least in healthy subjects.

However, due to its design, it is not advisable to wear it for more than 3-4 weeks, since prolonged use may result in the eruption of the posterior teeth (18).

5.3.3. Nociceptive trigeminal inhibition splint

The nociceptive trigerninal inhibition splint is compared to conventional occlusal splints (265), in 10 patients with a self-report of tooth grinding during sleep. Patients were examined at baseline and after each treatment period with the use of the Research Diagnostic Criteria for TMD. A portable EMG-device was used to record EMG activity from the masseter muscle during sleep. A baseline EMG recording without splint was done then the patients wore the splint during sleep for 1 week without EMG recording followed by 1 week with the splint and EMG recording (NTI + EMG or OS + EMG). After 1–2 weeks of washout period, another baseline EMG recording without splint was done followed by 2 weeks with the other type of splint, one without and one with EMG recordings. A total of 4 weeks EMG data were obtained for each patient. EMG sleep evaluation showed significant muscle activity inhibition in patients who wore nociceptive trigerninal inhibition splint, in contrast with those patients who wore conventional splints which showed no significant differences. This short term study indicated a strong inhibitory effect on EMG activity in jaw closing

muscles during sleep of the nociceptive trigerninal inhibition splint, but not the occlusal splint. However, the EMG activity was not directly related to clinical outcome. In this study the effects were evaluated over a short period and in a small sample size 10 subjects.

In study (306) investigating the effect of stabilization splint with canine guidance and nociceptive trigerninal inhibition splint on sleep bruxism by using polysomnography; 20 sleep bruxism patients that were confirmed to have sleep bruxism by polysomnography electromyography were randomly divided into a stabilization splint group (9 patients) and a NTI splint group (11 patients). The participants were instructed to wear the splint during sleep for 4 months. Masticatory muscle activity was measured during sleep. The first 2 nights were base nights, and the third study night was conducted 4 months after regular use of the splints. The number of bruxism episodes and the duration and intensity of sleep bruxism were analyzed. No statistically significant reduction in the frequency and duration of sleep bruxism was observed with either type of splint. However, the nociceptive trigerninal inhibition splint reduced the intensity of the masseter and temporal muscle activity, whereas the stabilization splint showed no effect on this parameter. Author concludes that the changes in the feedback to the peripheral oral receptors (alterations in the occlusal contact relation and increased vertical dimension) did not change the frequency and duration of sleep bruxism, but that the nociceptive trigerninal inhibition splint reduced the intensity of the sleep bruxism. Therefore, it seems likely that the therapeutic mechanism of the nociceptive trigerninal inhibition splint must be related to factors that reduce excessive masticatory muscle activity and redistribute the overloading from the masticatory system rather than preventing sleep bruxism. The findings of this study demonstrate that the nociceptive trigerninal inhibition splint is a viable alternative for the management of bruxism.

Dalewski et al. (318) compared occlusal splint with nociceptive trigerninal inhibition splint in bruxism therapy using surface electromyography. EMG activity levels during postural activity and maximum voluntary contraction of the superficial temporal and masseter muscles were compared before and after 30 days of treatment in two groups of bruxers (15 patients each). The patients in the first group used occlusal splints, while those in the second used nociceptive trigeminal inhibition splints. The patients were instructed to wear the splint overnight and not to exceed 12 hours a day. The use of neither device affects the asymmetry index or postural activity/maximum voluntary contraction ratio after one month of treatment. Author concludes that neither

the occlusal nor the nociceptive trigeminal inhibition splint showed any significant influence on the examined muscles.

In a study (228) to compare between stabilization splint and nociceptive trigerninal inhibition splint an occlusal changes in a patient who wore nociceptive trigerninal inhibition splint for a long period was observed. In this study; a clinical examination was performed and subjective symptoms were registered before start of treatment and after 3 and 6 months. Participants were offered to change to the other type of splint at the 3 month follow-up in case of no improvement or impairment of their symptoms. At the 3 month follow-up, 4 patients that had received nociceptive trigerninal inhibition splints accepted the offer to change to stabilization splints due to no improvement or impairment of their symptoms. These treatments were judged as failures. No one in the stabilization splint group utilized the offer to change treatment. At the 6 month follow-up, 7 of the remaining 10 subjects with nociceptive trigerninal inhibition splints reported some (n = 1) or significant (n = 6) improvement, 2 reported no change and one reported impairment. All 14 who had been treated with a stabilization splint reported some (n = 2) or significant (n = 12) improvement. For all variables registered, the results were in favor for the stabilization splint. One subject treated with a nociceptive trigerninal inhibition splint exhibited an impaired occlusion at the 6 month follow-up.

The nociceptive trigerninal inhibition splint, due to its design of covering only some of the anterior teeth, has the potential for developing an anterior open bite because of the over eruption of the posterior teeth or intrusion of the anterior teeth (319).

5.3.4. Palatal splint

Palatal splint is the splint that covers only the hard palate without modifying dental occlusion. The beneficial effects of the palatal splint have been noted by Greene and Laskin (320) and were explained as a stomatognathic alteration (an indirect occlusal separation effect). Young (321) has shown that placement of a 2.5mm thick acrylic palatal appliance increased interocclusal distance (freeway space) even when the appliance did not cover any teeth. The author postulated that mechanical displacement of the tongue inferiorly by 2.5mm posture the mandible inferiorly and produced an interocclusal separation.

A group of scientists (322) compared the effects of full occlusal coverage splints and palatal splints in a female bruxer with portable electromyography. These authors

showed that the number of EMG bruxism events per hour of sleep reduced when both hard occlusal splint and a palatal splint was worn as compared with a no-splint condition. When the use of either type was interrupted, EMG activity returned to its initial values. This study was conducted on just one bruxer, so its results have limited scientific.

The effects and safety of occlusal coverage splints and palatal coverage splints were compared through PSG in Nine subjects with sleep bruxism (6). First night of sleep laboratory recording was for habituation. The second night was used to establish baseline data. Patients are randomly assigned which of the two oral devices was to be worn first by each patient. Patients were given two weeks to get used to the splint. The subjects then spent a third night at the laboratory; the polygraphic data recoded while wearing their first splint. Next morning the treatments were swapped and the second splint was worn by the subject for two weeks. Further laboratory recordings were made on the fourth night, with subjects wearing their second splint. A statistically significant reduction in the number of sleep bruxism episodes per hour (decrease of 41%) and sleep bruxism bursts per hour (decrease of 40%) was observed with the two devices. Both oral devices also showed 50% fewer episodes with grinding noise. No difference was observed between the devices. Only one patient showed an exacerbation of sleep bruxism with the occlusal coverage splints. Moreover, no changes in respiratory variables were observed. Both devices reduced muscle activity associated with sleep bruxism.

Harada et al. (193) also compared the effects of full occlusal coverage splints and palatal in the management of sleep bruxism, using a portable electromyographic (EMG) recording system. The EMG activities of the right masseter muscle during sleep were recorded for three nights each in five recording periods: before, immediately after, and 2, 4 and 6 weeks after the insertion of the splint. The sample consisted of 16 patients with bruxism who were divided randomly into two groups (n = 8) according to the splint used, One group used occlusal splint for the first 6 weeks, and then after an interval of 2 months for a washout period, they wore palatal splint for the second 6 weeks. The other group used palatal splint first, and then wore occlusal splint with a washout period for 2 months. The authors found no statistically significant difference between the two types of device. The findings showed that both splints significantly reduced sleep bruxism immediately after the insertion of devices, but there were no statistically significant changes at 2, 4 and 6 weeks after the insertion. Author

Conclusion: Both splints reduced the masseter EMG activities associated with sleep bruxism; however, the effect was transient. . The authors ascribed this to an adaptation of the stomatognathic system to the splint.

Van der Zaag et al. (266) studies the efficacy of occlusal stabilization splints and palatal splint in the management of sleep bruxism. 11 participants were used an occlusal splint and 11 participants were used a palatal splint group. Bilateral masseter electromyographic activity recorded two times with polysomnography; one prior to treatment, the other after a treatment period of 4 weeks. On group level, the author found that, both occlusal stabilization splints and palatal splints had no influence on the number of bruxism episodes per hour of sleep, or the number of bursts per hour. In individual level, author found variable outcomes: Some patients show an increase of greater than 50% in their individual bruxism outcome variables, or a decrease in sleep bruxism of less than 50% in their individual bruxism outcome variables, while others showed no change. Author Conclusion**:** The absence of significant group effects of splints in the management of sleep bruxism indicates that caution is required when splints are indicated, apart from their role in the protection against dental wear. The application of splints should therefore be considered at the individual patient level. The results of this study were based on a single night's recordings of each period. Moreover, the participants wearing their splint 24 hours a day, except during eating; Fulltime use of occlusal splints could be a potential confounding factor due to its habituation effect.

Another group of researchers (323) carried out study on the relationship between masseter muscle activity during sleep and variant palatal coverage splint thickness. The masseter muscle electromyography (EMG) was recorded in eight healthy participants during sleep, four types of appliances (horse shoe, thin, thick and medium thick) was worn by each participant during sleep for one week with a one week interval without appliance during sleep. The number of bursts per hour, episodes per hour, and bursts per episode with the thick type appliance was significantly lower than the baseline condition without appliance. They suggested that a thick type appliance has an active effect on suppression of masseter muscle activity.

5.3.5. Intraoral mandibular advancement devices (MAD)

Intraoral mandibular advancement devices (Mandibular anterior repositioning) are a double arch custom fit appliance designed to protrude the mandible, which increase airway space, the device is commonly prescribed to Patients suffering from

sleep apnea (324). A group of scientists compared the effects of mandibular advancement devices and maxillary occlusal splints in the management of sleep bruxism (278). In their study of 13 heavy bruxers, PSG recordings were taken before and during the alternate use of both types of splint; also recordings were obtained for different degrees of mandibular protrusion: (i) without the retention pin between the arches (full freedom of movement), (ii) with the retention pin in a slightly advanced position, and (iii) with the retention pin in a more advanced position of the lower arch. Both splints decreased the bruxism significantly. Maxillary occlusal splits caused the number of bruxism episodes per hour to decrease by 42%. Mandibular advancement splints performed better; at mandibular advancement devices free (full freedom of movement) bruxism decreased by 44%, at minimum advancement, bruxism decreased by 77%, while at maximum protrusion it decreased by 83%. At individual level, one of the study subjects showed bruxism exacerbation, with tooth grinding episodes while wearing the splints. Sleep parameters remained stable; no variations were observed with any of the devices used. 8 of 13 subjects reported pain, mainly to the mandibular anterior teeth and gums, with mandibular advancement devices min and the mandibular advancement devices max. When requested to choose between the occlusal stabilization splint and the mandibular advancement device, none of the patients showed a preference for the mandibular advancement devices, because they were cumbersome, uncomfortable, and unaesthetic compared to the occlusal splint. The authors hypothesized to explain the reduction of sleep bruxism with the mandibular advancement devices, oriented toward one or more of the following: dimension' and configuration of the appliance, presence of pain, restriction of mandible movement, or change in upper airway patency. After all, it has been reported that the presence of pain may considerably reduce the bruxism activity (138,325,326) . In the authors' opinion, the occlusal splint still remains the oral device of choice, since it is reversible, protects the teeth against damage, and more importantly, is well tolerated by the patient. In this study there were no washout periods between appliances, and the EMG levels were not reassessed during the periods in between appliances to control the sleep bruxism activity level. Moreover, they record a single night for every device, and the variability of sleep bruxism from night to night (63) might have modified the value of the sleep bruxism oromotor outcomes.

Another study (327) compared the effect of mandibular advancement device and mandibular occlusal splint on Sleep bruxism. Polygraphic and audio/video recordings

made over 5 nights in sleep laboratory for 12 patients with frequent sleep bruxism. Subjects were randomized to tow sequences, first sequence (7 subjects) use mandibular occlusal splint then MAD (25% advancement), then MAD (75% advancement). Second sequence (5subjects) use MAD (25% advancement), then MAD (75% advancement), then mandibular occlusal splint. The first night was for habituation and was not included in the statistical analysis. The second night used to establish the baseline for sleep bruxism. Subjects were given 2 weeks to adapt to each appliance before third, fourth and fifth experimental nights. The result shows that the mean number of sleep bruxism episodes per hour was significantly reduced by 39% from baseline with the MAD at a protrusion of 25% and 47% at a protrusion of 75%. The mandibular occlusal splint slightly reduced the number of sleep bruxism episodes per hour without statistical significance. Individual variations have been observed; three of the 12 subjects showed increased sleep bruxism motor activity during the night spent wearing the mandibular occlusal splint. All 12 subjects preferred the mandibular occlusal splint to the MAD, the complaints about the MAD size and difficulty in closing the lips (too bulky, uncomfortable). When asked about their perception of the devices' efficiency, five of the 12 subjects thought that the mandibular occlusal splint was more effective in reducing sleep bruxism. One subject rated the MAD at 25% as most efficient, and three subjects rated the MAD at 75% as being the most efficient. The results of this study based on single night record for every device, there is no washout period was allowed between conditions, and no reassessment of oromotor activity was made to ensure a return to baseline level, this could have influenced the results.

Other study evaluate the effect of a MAD and a maxillary occlusal splint on the sleep quality and sleep bruxism activity in patients with sleep bruxism (267). The sample consisted of 28 patients who used either a MAD or maxillary occlusal splint; the electromyographic activity of the masseter muscle was evaluated with polysomnography. The patients spent 2 nights in a sleep laboratory, first night to acclimatize to the sleep recording environment and excluded from statistical analysis. In second night the polysomnography records and the sleep quality of the patients are used as baseline data. The devices were worn for a period of 2 weeks for habituation, then the participants evaluated for sleep quality and sleep bruxism activity at 1 and 3 months. The results of this study showed that, after 1 month both the MAD and maxillary occlusal splint improve the sleep quality and reduce the sleep bruxism episodes but not significantly, while after 3 months both the MAD and maxillary occlusal splint provided

significant improvement in sleep quality and significant decrease in the mean number of episodes per hour. The participants with the MAD showed a greater reduction in episodes per hour, bursts per hour compared to participants with the maxillary occlusal splint. Although the MAD showed a greater reduction, it was associated with discomfort, including tooth sensitivity and drooling. While most participants using the maxillary occlusal splint were satisfied at the end of the study, those using the MAD preferred to use a lighter and more comfortable appliance if possible. The author suggested that the mechanism behind the positive effect of the MAD may be explained by the forward movement of the mandible, which increases the airway space and reduces microarousals, allowing deeper stages of sleep, as most bruxism episodes (80%) occur in the light sleep stage (328).

However, Mandibular anterior repositioning appliances are used in the treatment of intermittent joint locking occurring on waking. After some weeks, the effects of bruxism will usually be clearly visible on these appliances; this clearly proves that such devices didn't stop the bruxism (18). Moreover, certain studies have also reported some oro-dento-skeletal modifications with the use of a MAD (324,329–332), this modifications should be considered when using this device as a long term management therapy.

According to the results of these studies, such a device may be appropriate only for the treatment of bruxers patients suffering from sleep apnea. However, these studies monitored the effects for only a short period; thus, there are doubts regarding potential long term effects.

6. CONCLUSIONS

In this study the following conclusions were drawn:

1) There is no specific treatment available at this time to completely stop bruxism, so the management of the bruxism focus on reduces the adverse effects of the bruxism.

2) Minimally invasive and reversible therapies should be first choice in treatment protocols of seep bruxism. There is no scientific evidence that irreversible treatments, such as occlusal adjustment, treats or prevents Sleep bruxism and TMD.

3) In this study the observed decrease in the temporalis muscles activity with short term occlusal appliance therapy, suggested that the occlusal appliance should be regarded as a management for rapidly relaxing masticatory muscles.

4) Since some individual variations have been observe, the splint is not always effective in all bruxism patients, and the clinicians should always monitor the signs and symptoms reported by the patient when using an oral device for the management of sleep bruxism.

5) Although in some individual there is no reduction in sleep bruxism activity, the occlusal appliance still provide tooth protection, as previously suggested.

6) The effect of occlusal appliance could be transient and not strong enough to stop or reduce the sleep bruxism activity for a long time, this observation suggest that the splint therapy in most cases should be regarded as a symptomatic treatment rather than the etiotropic treatment.

7) Amongst the various types of oral appliances, the occlusal appliance is the oral device of choice, since it is safe, reversible, protects the teeth, and well tolerated by the patient.

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Ethical approval

T.C. YEDİTEPE ÜNİVERSİTESİ

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Konu: Etik kurul Başvurusu hk.

22 / 04 / 2015

İlgili Makama (Sayın Salih Aboghrara)

Yeditepe Üniversitesi Diş Hekimliği Fakültesi Protetik Diş Tedavisi Dt.Salih Aboghrara'nın sorumlu olduğu "**Uyku Bruksizmi Sırasında ,Oklüzal Splint Kullanımının Temporal Kas Aktivitesi Üzerine Kısa Dönem Etkisi**" isimli araştırma projesine ait KAEK Başvuru Dosyası (1047kayıt sayılı KAEK Başvuru Dosyası), Yeditepe Üniversitesi Klinik Araştırmalar Etik Kurulu tarafından 22-04 2015 tarihli toplantıda incelenmiştir.

Kurul tarafından yapılan inceleme sonucu, çalışmanın yapılmasında etik ve bilimsel açıdan uygun olduğuna karar verilmiştir (Karar No: **57/480**).

Bilginizi ve gereğini saygılarımla arz ederim.

Prof. Dr. Turgay ÇELİK
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