Review Article

Diagnosis and Management of the Bruxism: A Conceptual Review

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Abstract:
Bruxism is a common parafunctional habit, occurring both during sleep and wakefulness. Awake bruxism is found more in females as compared to males while sleep bruxism shows no such gender prevalence. Etiology of the bruxism is multifactorial. Treatment modalities involves occlusal correction, behavioural changes and pharmological approaches in cases of both awake and sleep bruxisim. Association of behavioral strategies and dental protection appliances seem to be the most adequate therapeutic measures for mid- and long-term bruxism treatment.
Key words**:** Bruxism, Night grinding, Parafunctional habit.

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Introduction

Bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and by bracing or thrusting of the mandible.[1] It is the commonest of the many parafunctional activities of the masticatory system. 'Bruxism' originates from the Greek word brychein, meaning to 'gnash the teeth'. An early and common definition of bruxism was thus ''gnashing and grinding of the teeth for non-functional purposes''.[2] Later definitions have been more specific, for example, ''involuntary, non-functional, rhythmic or spasmodic gnashing, grinding, and clenching of teeth, usually during sleep''.[3]

Tooth grinding is an activity particularly important to the dentist because of breakage of dental restorations, tooth damage, induction of temporal headache and temporomandibular disorders.[4] Bruxism is considered to have a multifactorial aetiology that includes currently poorly defined aspects of central nervous system function, genetic and behavioural factors.[2,3,4] Broadly bruxism may be either primary (idiopathic) or secondary.[4,5] Primary bruxism is divided into two types that are thought to be clinically unrelated: sleep and awake bruxism. Sleep bruxism (SB) is involuntary and is classified as a sleep-related movement disorder.[1] Awake

bruxism (AB), by contrast, is defined as the awareness of jaw clenching, and appears to be semi-voluntary.[4,5,6] In adults under 65, the prevalence of SB is around 10% and declines gradually with age.[4,5,7] AB prevalence is probably higher and, unlike SB, is more frequently observed in females.[6,7] Secondary bruxism has been observed as a side effect of medication use and in some neurological and developmental disorders. In the future, it may be possible to further differentiate forms of bruxism according to the underlying cause and clinical manifestations.[8] A number of other conditions may coexist with bruxism, including temporomandibular joint disorder (TMD), orofacial pain, headaches and sleep-disordered breathing. The causal relationships between these conditions and bruxism remains unclear.[9] Some authors view bruxism as an underlying cause of TMD, while others consider both bruxism and TMD as related consequences of abnormal muscle activation. The management of bruxism is not affected by coexisting TMD.[10]

Diagnosis of the bruxism: Episodes of bruxism consist of masticatory muscle activity during wakefulness or sleep and manifest as arange of signs and symptoms in the orofacial region.[11] The symptoms of AB and SB are similar and include tooth grinding, jaw-muscle discomfort with or without frank pain,[12] temporomandibular joint tenderness,Vfacial pain and headache.[13] SB symptoms are usually worst in the morning on waking and improve during the day, while patients with AB may develop symptoms only after waking.[6] The signs include abnormal tooth wear, tongue indentation, increase in jaw muscle activity (measured by electromyograph (EMG) or polysomnograph (PSG)), masseter muscle hypertrophy, reduction in salivary flow, lip or cheek biting, gum recession, limitation of ability to open the mouth, and burning tongue.[2,12,14] The negative consequences of bruxism include subjective impacts like stress, anxiety, tiredness and poor sleep quality, as well as damage to teeth, which may ultimately result in premature loss of dentition.[13] The diagnosis of the bruxism is based particularly on history, tooth mobility, tooth wear and other clinical findings.[5] Because of the variety of symptoms and overlap with other conditions, diagnosis of bruxism requires a careful process of assessment that incorporates questionnaires, history taking and examination. Few question regarding to grinding of teeth during sleep has anyone noticed, jaw fatigue, soreness of the jaw, gums or temporal headache on awakening and are you ever aware of grinding or clenching your teeth during day, should be asked. [6]

Objective testing includes EMG recording of the activity of the masticatory muscles and PSG recording of the sleeping patient. While full audio-video PSG recording remains the gold standard for diagnosis of SB,[1,14,15] standardized clinical diagnostic criteria have also been proposed.[1,15,16] There are no validated objective tests for the diagnosis of AB, which relies instead on direct questions and visual observation of patient behaviour.[5]

A clinical diagnosis and/or ambulatory EMG/ electrocardiograph/PSG diagnosis or full PSG diagnosis of SB; or a diagnosis of AB, identified by means of direct questions and visual observation of patient behavior.[5] For SB, we will accept any diagnosis based on clinical or research PSG criteria that are consistent with the International Classification of Sleep Disorders Second Edition criteria (Table 1).[15] Broadly bruxism can be evaluated by clinical examination of tooth wear, by using intraoral appliances and electromyographic recording of masticatory muscles.

Examination of tooth wear: Bruxism was for long considered a major cause of tooth wear. Several studies showed a positive relationship between tooth wear and bruxism[17] but others have not.[18] Tooth wear is a cumulative record

Table 1: Clinical and Polysmnographic criteria for diagnosis of sleep bruxism

Clinical criteria	Polysmnographic criteria
The participant reports or is aware of tooth grinding or clenching during sleep (3-5 nights per week over past 6 months) and one or more of following: • Abnormal tooth wear • Jaw muscle discomfort, fatigue or pain and jaw lock upon awakening • Masseter muscle hypertrophy evident on voluntary forceful clenching	Polysomnographic monitoring demonstrates both of the following jaw muscle activity during the sleep period and absence of associated epileptic activity Polysomnographic diagnostic cut off criteria: • More than four bruxism episode per hour • More than six bruxism bursts per episode and/or 25 bruxism bursts per hour of sleep and • At least two episodes with grinding sounds

of both functional and parafunctional activities and various factors such as age, gender, diet and bruxism are associated with tooth wear. Erosion by acidic drink is considered to be major contributing factor to tooth wear.[19] All mechanisms of tooth wear rarely act alone and usually interacts with each other to cause wear. So the evaluation of tooth wear to for predicting actual bruxism is controversial and is difficult to estimate the degree of contribution of bruxism to tooth wear alone.

Intraoral appliances: Bruxism activity can also be evaluated either by observation of wear facets of the intra-oral appliance[20,21] or measurement of bite force loaded on the intra-oral appliance.[22] Holmgren et al and Korioth et al reported a repetitive wear pattern and parafunctional nocturnal dental activity on full-arch occlusal splints.[20,21] They observed wear facets on full-arch acrylic resin splints, which reappeared in the same location with a similar pattern and direction, even after adjustment of the splints while in parafunctional activity it was both asymmetric and uneven. Unfortunately, no confirmation of the reliability of these methods has been reported. The another intra oral device is Bruxcore Bruxism-Monitoring Device (BBMD) and it was used as a device for measuring sleep bruxism activity objectively.[23]

The BBMD is a 0.51-mm-thick polyvinyl chloride plate that consists of four layers with two alternating colors and a halftone dot screen on the topmost surface. It evaluates bruxism activity by counting the number of abraded microdots on its surface and by scoring the volumetric magnitude of abrasion. The number of missing microdots is counted to assess the abraded area and the number of layers uncovered represents the depth parameter. Both parameters are combined to obtain an index for the amount of bruxism activity. The major disadvantage with this method is that it is difficult to count the number of missing dots with good precision. Pieree and Gale[24] did not find any significant correlation between bruxcore plate and bruxism analyzed with the EMG data

Takeuchi et al.[25] developed a intra oral recording device for sleep bruxism, an intra-splint force detector (ISFD), which uses an intra-oral appliance to measure the force being produced by tooth contact onto the appliance. The force is detected using a thin, deformation-sensitive piezoelectric film. ISFD did not correctly capture force magnitudes during sustained clenching because of the characteristic of the piezoelectric film, because it is best at detecting rapid changes in force, not static forces. ISFD was not suitable for

detecting the magnitude of force during steady-state clenching behaviour. It is obvious, however, that the major problem of these methods is that subjects have to wear the intra-oral device and this may change the original bruxism activity.

Electromyographic recording of masticatory muscles: The principal advantage of electromyographic recording is, that the occurrence of bruxism can be assessed generally without intra-oral devices, which may change natural bruxism activity. Among the various methods for the assessment of bruxism. In this the electromyograpgic (EMG) recording has been commonly used to measure actual sleep bruxism activity directly. Starting in the 1970s, sleep bruxism episodes were measured over an extended period in patients homes with the use of battery-operated EMG recording devices.[26] The portable EMG recording system has become easy for subjects to operate and can measure masticatory muscle activity more minutely, i.e. the number, duration and magnitude of bruxism events can be evaluated with fair accuracy. The detection power of sleep bruxism is generally considered inferior to that in a sleep laboratory because other confounding oro-facial activities (e.g. sight, coughing and talking) cannot be discriminated from sleep bruxism. Other sleep disorders cannot be ruled out or other physiological changes related to sleep bruxism (e.g. microarousal, tachycardia and sleep-stage shift) cannot be monitored.[4,27,28] The implement for recording the heart rate was recommended as one of the compensatory measures for improving the accuracy of sleep bruxism recognition. Later on, a surface EMG electrode with a built-in buffer-amplifier and a cordless type of EMG measurement system was developed to improve the reliability of recordings.[29]Another device known as self-contained EMG detector–analyser (Bite- Strip) was developed as a screening test for moderate to high level bruxers. This device is comprised of EMG electrodes, an amplifier, a central processing unit (CPU) with software, a display which presents the outcome in the morning, a light emitting diode and a lithium battery records the number of masseter muscle activities above a preset threshold. The special feature of this device is that the number of bruxism events can be objectively estimated by simply attaching it to the skin over the masseter muscle. Minakuchi and Clark[30] compared the sensitivity and the sensitivity and specificity of the BiteStrip recording versus masseter EMG recordings during a polysomnogram in five suspected bruxers. The result was good specificity for all subjects but fair sensitivity for subjects that exhibit moderate to high levels of EMG determined bruxism. An another device, Telemetry EMG recording device (TEL-EMG) is used. It is a single-channel telemetry electromyographic (TEL-EMG) system for recording masseter muscle activity. This device consists of an ultraminiature transmitter unit and a receiver unit. The transmitter unit contains bipolar electrodes, a reference electrode, a amplifier, bandpass filters, a digital (A ⁄ D) converter, a central processing unit (CPU) a battery and a transmitter. Amplified EMG digital data are transmitted to the receiver unit. The obtained EMG data are stored in a compact flash memory card (CF card), and the card is then transferred to a personal computer for analysis. The transmitter unit was attached over the masseter muscle by three adhesive gel pads on electrodes to conduct electrical signals. The receiver antenna was placed beside a pillow. The polygraphic recordings, using surface electrodes, included electroencephalograms (EEG), bilateral electrooculograms (EOG), electrocardiograms (ECG) and electromyograms (EMG) from the chin, masseter, bilateral temporalis and tibialis muscles. Audiovisual recordings were performed simultaneously.[31]

Polysomnography (PSG) is used for the diagnosis and study of different sleep disorders based on the recording of physiological events

throughout an entire night of sleep using electrodes and sensors in a laboratory setting. it include electroencephalogram, EMG, electrocardiogram and thermally sensitive resistor (monitoring air flow) signals along with simultaneous audio– video recordings. The EMG measures the electrical activity of the submental/ suprahyoid regions, thoracic-abdominal movements, oximetry, heart rate, nasal flow pressure, as well as the tibialis anterior, mentalis, masseter, and temporal muscles. Audio visual recording is also involved.[32] polysomnographic study allows for multidimensional analyses of sleep-related physiological behaviours and studies on sleep laboratory EMG-based assessments are reported to be very reliable because the sleep laboratory setting offers a highly controlled recording environment and other sleep disorders (e.g. sleep apnoea and insomnia) can be ruled out and sleep bruxism can be discriminated from other orofacial activities (e.g. myclonus, swallowing and coughing) that occur during sleep related to sleep bruxism (e.g. microarousal, tachycardia and sleep-stage shift) can also be monitored. The limitation of this device is that a change in the environment for sleep may influence the actual behavior of bruxism. Another limitation is the expense as multiple night recording is to be taken for the occurrence of sleep bruxism varies over a number of nights.[6]

Management of Bruxism

Treatment Approach: No therapy till date has been shown to effectively and permanently cure bruxism. The most accepted approaches focus mainly on symptom management and prevention of complications.[10]

Awake bruxism: The initial management of awake bruxism should be directed to receive proper information from the individual and counseling about awake bruxism and the potential negative consequences.[1] Counseling can be directed toward stress management and lifestyle modifications (reduction of nicotine, caffeine, and alcohol). Physical therapy can also be useful in the management of jaw-muscle pain and fatigue.

Occlusal splints or appliances are commonly used in the management of the awake bruxism. They are of different types like occlusal bite guard, bruxisim appliances, bite plate, night guard, occlusal devices but scientific evidence shows that occlusal splints only reduce bruxism activity with no long-term effects.[1,33] Twenty-four-hour use of oral splints is contraindicated due to the risk of creating iatrogenic changes in occlusal contact patterns.[1,4] Generally, pharmacologic management of awake bruxism is not indicated and should be avoided in most patients. However, in patients with significant jaw-muscle pain that does not respond to other treatments, short-term use of mild analgesics may help alleviate the pain.

Sleep bruxism: There is currently no specific, definitive treatment for sleep bruxism, although various preventive measures (e.g., occlusal splint, stress management) and certain drugs (benzodiazepines, antidepressants) can be used for acute cases, particularly those involving pain.[34]

SB treatment utilizes a combination of behavioral treatment, dental and pharmacological treatment according to the carrier's profile.[1,4,5,35] Secondary SB treatment should focus on the specific cause. One of the most important therapeutic tools is to give the patient information and a detailed, simple explanation of the clinical picture. Self-relaxation measures are very important elements for helping to reduce the frequency and intensity of masticatory muscle activity during wakefulness, which favours muscle relaxation and the reduction of bruxing episodes during sleep. If the patient does not assume responsibility for this important aspect of self-management in therapy and apply it, any other measures will be of only very limited usefulness. There are some pharmacological compounds (botulinum toxin

type A, benzodiazepines and other muscle relaxants, anticonvulsants, beta blockers, dopamine and other dopaminergic drugs, antidepressants, clonidine, etc. that can help control SB, although their use must be restricted to nonrecurrent situations, such as the start of treatment or periods of exacerbation due to a rise in emotional tension, and always as part of a comprehensive, interdisciplinary approach.[36]

Several types of intraoral appliances (dental plates) have been developed for the purpose of relieving local pain, preventing lesions in orofacial structures, and preventing temporomandibular articulation dysfunction.[37] Four simple methods are described in the literature for the fabrication of occlusal splints. First and second one is hard acrylic resin and composite resins, third one is 'Nociceptive Trigeminal Inhibition (NTI) Clenching Suppression System'—a small anterior splint that is supposed to be effective amongst others in the management of bruxism and finally, the fourth on is pre-fabricated and chairside adjustable ''Bruxism 'S' Splint'' that can be used in combination with active orthodontic treatment. More research is needed to assess the efficacy and safety of such unconventional, chair-side solutions before their application in dental practice can be recommended. In an study by Clark et al. it was shown that occlusal splint treatment resulted in a decrease in nocturnal EMG activities in about half of the patients, while in another half of the patients, no change or even increase in EMG activity was observed.[6] The mechanisms of action of intraoral appliances and their effectiveness at reducing neuromuscular activity during sleep have not yet been well established.[38]

Summary

Bruxism is a common parafunctional habit, occurring both during sleep and wakefulness, and sleep bruxism and awake bruxism should be differentiated. It usually has no serious effects, but may, in some patients, have pathological consequences. It is of multifactorial origin and The etiology of bruxism is not well known There is no specific treatment available at this time to stop Bruxism. The prognosis for bruxism is variable depending on the severity and duration of the factors producing arousals during sleep. Intraoral appliances may protect dental structures from wear; however, a long lasting reduction in nocturnal masticatory muscle activity and awakening pain complaints may not be possible without identifying and addressing the etiology of the underlying sleep disturbance.

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