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A 7-year old male, Brad presented to his dentist with toothache in the left lower mandible area. Nothing was found after thorough examination and the dentist could not identify any particular triggers or relieving factors for Brad's on and off toothache. A course of NSAID was prescribed which did provide temporary relief. Brad usually sleeps alone in his own room but during recent home renovation different sleep arrangement was made. One night when he was co-rooming with his parents, mother could hear "strange" noise coming from Brad's bed, and it turned out Brad was grinding his teeth during sleep. Brad could not recall his teeth grinding. The family represented to the dentist who arranged polysomnography for Brad and his confirmed bruxism was treated accordingly. Parents wondered whether Brad's bruxism would recur!

17.1 Definition and Epidemiology

American Academy of Sleep Medicine (AASM) defined bruxism as: "repetitive jaw muscle activity characterized by the clenching or grinding of teeth and/or bracing or thrusting of the mandible [1]." Bruxism has two separate manifestations according to the circadian rhythm: it can occur during sleep (sleep bruxism) or during wakefulness (awake bruxism) [2]. For the purpose of this topic, we will discuss further the sleep bruxism. The definition of sleep bruxism according to International Classification of Sleep Disorders, second edition (ICSD-2) is "an oral parafunction characterized by grinding or clenching of the teeth during sleep that is associated with an excessive or intense sleep arousal activity" [3]. Sleep bruxism in adults was reported at 13%, while in children the prevalence varied from 6% to 50% [4, 5]. The varying prevalence in children was seen due to different diagnostic tools (self-report, electromyography—EMG, polysomnography—PSG) used in studies [5]. It is also difficult to gather the exact prevalence of sleep bruxism because most studies used self-reported questionnaires and yet most bruxers are unaware of their habit [1]. In a systematic review, no gender difference was reported for sleep bruxism [6]. It was implied that complaints of tooth-grinding during sleep decrease over time, as seen in 14% children to 8% adults to 3% patients over 60 years of age [7]. Specifically in children, it seems that

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with increased age (around 9–10 years old), the prevalence of sleep bruxism tends to decline, possibly as a result of the stabilization of occlusion [6].

17.2 Pathophysiology

Sleep can be divided into two parts, occurring in 3–6 cycles at an interval of 60–90 min: non-REM (Rapid Eye Movement) sleep and REM sleep. Non-REM sleep consists of light sleep (stages 1 and 2) and deep sleep [7]. Most bruxism episodes occur during the light sleep of non-REM and less than 10% during REM sleep. Bruxism during REM sleep is associated with sleep arousals, which is characterized by a brief (3–15 s) cortical brain activity along with or without an increase in heart rate and motor activity. Normally, during REM sleep, muscles are relaxed to the point of paralysis, but the brain activity is similar to when a person is awake. Bruxism during REM sleep can be counted as parasomnia [1].

There is also an involuntary mechanism called rhythmic masticatory muscle activities (RMMA) which occur during sleep. Rhythmic masticatory muscle activities are observed in up to 60% normal subjects and 80% patients with sleep bruxism. RMMA are slow (1 Hz) chewing-like movements in the absence of tooth grinding. When RMMA are frequent or associated with tooth-grinding, sleep bruxism is identified [1].

17.3 Etiology

Sleep bruxism probably is multifactorial, because the exact etiology is still unknown. In the past, morphological (peripheral) factors, like malocclusion and the anatomy of the orofacial bones, have been considered the main causative factors for bruxism [8]. However, Lobbezoo et al. [9] showed that orofacial morphology of sleep bruxers did not differ from non-bruxers. This finding indicates that there is no correlation between anatomical structure of the face and sleep bruxism.

Multiple studies suggest that sleep bruxism is regulated centrally and not peripherally [8, 10]. The central factors can be further divided into pathophysiological and psychosocial factors (Table 17.1). Sleep microarousals have been linked with sleep bruxism based on polysomnographic (PSG) studies. It is known that the bruxers' jaw motor activity is preceded by brain activation and increased heart rate, implying the central/autonomic nervous system involvement in sleep bruxism [7]. Several neurochemicals also play a role in sleep bruxism, namely clonidine (alpha agonist) and L-dopa (dopamine precursor), which both can reduce sleep bruxism [1]. The study of neurochemicals role in bruxism needs to be examined further.

Sleep bruxism was reported in about 50% family members of bruxers. Based on a review of family studies, twin studies, and one DNA analysis, it can be concluded that bruxism appears to be genetically determined [11]. Abe et al. [12] found that the C allele carrier of HTR2A single

Table 17.1 Possible sleep bruxism etiology [1]

| Potential factor | Central factor | |
|---|--|-----------------------------------|
| | Pathophysiological | Psychosocial |
| Facial shape & features | Sleep problems | Mental stress |
| Condylar asymmetry | Brain neurotransmitter imbalance | Emotional problems (e.g. anxiety) |
| Dental arch form | Genetic | |
| Malocclusion | Lifestyle (e.g., alcohol, caffeine, smoking) | |
| Centric relation—maximum intercuspation discrepancy | Allergies | |
| Balancing side contacts | Nutritional deficiencies (e.g. calcium, magnesium) | |
| Occlusal interferences | Drug/medicine consumption | |

Adapted from: Yap AU, Chua AP. Sleep bruxism: Current knowledge and contemporary management. *J Conserv Dent.* 2016;19(5):383–9

nucleotide polymorphism rs6313 was associated with an increased risk of sleep bruxism.

In the psychosocial part, stress is known to increase bruxism events during sleep. Karakoulaki et al. [13] showed that bruxers had higher levels of perceived stress than non-bruxers. Moreover, the salivary cortisol was higher in bruxers compared to non-bruxers [13]. This finding was similar to a meta-analysis by Chemelo et al. [14] which suggested that stressed individuals showed a higher chance of presenting bruxism when compared to healthy individuals, although the level of evidence was low due to different evaluation parameters in some studies and lack of established methodological criteria.

Souto-Souza et al. [15] composed a meta-analysis about attention-deficit/hyperactivity disorder (ADHD) and bruxism. They found that children and adolescents with a definitive diagnosis of ADHD are at greater chance of developing sleep and awake bruxism than those without this disorder. It should be stressed that the association between bruxism and ADHD was only significant in cases for which there was a diagnostic confirmation by a physician. These findings were limited by the cross-sectional study design, so the true causal relationship between ADHD and bruxism remained inconclusive [15].

17.4 Presentation and Diagnosis

Sleep bruxism is characterized by clenching and grinding of teeth. This causes heavy load to teeth and mastication muscles. Tooth wear is common in bruxers. Also, pain in the mastication muscles and temporomandibular joint (TMJ) area is frequently observed. Other clinical findings in patients with sleep bruxism are periodontal disease, hypertrophy of the masticatory muscles, and headaches [16].

Diagnosis can be made through clinical presentations according to the international diagnostic criteria proposed by the AASM. Patient history should be taken regarding “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”. Also, clinical evaluation should be made towards abnormal tooth wear, hypertrophy of the masseter muscles on voluntary forceful clenching, and discomfort, fatigue, or pain in the jaw muscles (and transient, morning jaw-muscle pain and headache). Keep in mind that the jaw-muscle activity cannot be better explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder [17]. Moreover, bite marks on the buccal mucosa can also serve as a complementary sign for clinical diagnosis of sleep bruxism [18].

Full-night audio-video PSG recording (type I) remains the gold standard for diagnosis of sleep bruxism and the assessment of comorbidity with other sleep disorders (sleep apnea, periodic limb movements, parasomnias) [1, 17]. Objective PSG recordings include brain activity (electroencephalogram), eye movements (electrooculogram), jaw/leg movements (electromyogram), heart rate/rhythm (electrocardiogram), thoracoabdominal movements, oronasal airflow and oxygen saturation [1]. The recordings of the audio-video PSG were checked to see how many RMMA events happened during sleep. The audio and video footages were helpful to distinguish orofacial activities (OFA) and other muscle activities (OMA) that could easily be mistaken for RMMA events [19]. Sleep bruxism was diagnosed if there were: (1) more than 4 episodes per hour, (2) more than 6 bursts per episode and/or 25 bursts per hour of sleep, and (3) at least 2 episodes with grinding sounds [20]. There are also several ambulatory devices for EMG or PSG monitoring (type II, III, and IV) to record RMMA, although the sensitivity and specificity depends on the device used [17].

17.5 Management

To current knowledge, there is no “cure” yet for sleep bruxism. The goal for management of sleep bruxism is directed toward tooth restoration/pro-

tection, reduction of bruxism activity, and pain relief [1]. Management in sleep bruxism can be distinguished in five: pharmacological, psychological, physical therapy, dental strategies, and alternative/homeopathic medicine [21].

In controlled clinical trials for adults, only clonidine and L-dopa managed to reduce sleep bruxism [1]. Clonazepam (a benzodiazepine drug) also showed promising effect to reduce sleep bruxism index in all adult patients included in the study. However, the risk of dependency ceased the drug for long-term use [22]. Botulinum toxin (BTX type A) can be injected locally to masseter muscles in patients with severe bruxism, which are refractory to conventional therapy. Although, this method has its portion of side effects, which can include: difficulty chewing or talking, muscle pain, and asymmetric changes in the facial features [1].

While in children, certain drugs had shown promising result in reducing parent-reported sleep bruxism. Flurazepam (15 mg/day), hydroxyzine (both 25–50 mg/day and 5–25 mg/day), imipramine (25 mg/day) all decreased the occurrence of sleep bruxism when given for 4 weeks. Trazodone (0.5 mg/kg/day) on the other hand, had a significant reduction of bruxism and morning pain after 2 and 4 weeks of intervention. But these drugs had various side effects, such as: drowsiness, nausea, vomiting, irritability, dry mouth, insomnia, confusion, aggression, headache, and diminished appetite. Moreover, it was not known whether the episode of sleep bruxism returned after the therapy had been stopped [21].

Psychological efforts to reduce sleep bruxism include biofeedback, hypnotherapy, cognitive therapy, behavioral therapy, stress, and relaxation management. Even though there is a link between psychological factors (e.g. stress) and sleep bruxism, there is limited evidence pointing out that these therapies have a good efficacy in treating sleep bruxism [1]. Targeted muscle relaxation and reaction competence were proved to be able to reduce parent-reported bruxism in children [21]. Also, it was found that sleep bruxism can decrease the children's quality of life, mainly because of functional limitation and pain. More approaches need to be explored further regarding the quality of life of sleep bruxers [3].

As for physiotherapy, one study of 26 children aged 3 to 6 years showed a 77% reduction of the occurrence of parent-reported tooth-grinding or tooth-clenching during sleep in the intervention group, as opposed to 15% reduction in the control group. The intervention involved 10 sessions of physical therapy done once a week per session [21].

Dental treatment for sleep bruxism consists of occlusal therapy and occlusal splints. There is no association between occlusion and sleep bruxism, although occlusal therapy is recommended if the dentition needs to be rebuilt after being noticeably worn down. Occlusal splints are removable dental devices that fit in between the maxillary and mandibular teeth and are composed of hard acrylic or soft vinyl. Its main function is to shield teeth and restorations from abrasion and harmful traumatic loading. Occlusal splints, depending on how they are made, can also unload, stabilize, and improve TMJ functioning as well as lessen painful muscle activity and aberrant muscle activity [1]. The use of occlusal splint correlate to a reduction in self-reported bruxism in 30 children aged 7 to 10 years old [21].

For alternative medicine, two studies were identified using *Melissa officinalis L* and *Phytolacca decandra* extracts to reduce bruxism in children. It turned out that both extracts could reduce the parent-reported bruxism, with one study showed in 2 years of follow-up, no recurrence of sleep bruxism was identified [21].

Another new therapy method, photobiomodulation, had reportedly successful in treating some muscle disorders [18]. Photobiomodulation and light-emitting diode (LED) therapy are treatment options for reducing pain and inflammatory processes as well as inducing the regeneration of the target tissue. Salgueiro et al. [18] conducted a study to see the effectiveness of photobiomodulation on sleep bruxism. Photobiomodulation over acupuncture points proved to be an alternative treatment for children with sleep bruxism, leading to fewer reports of headache and a reduction in bite strength [18].

Due to various side effects of the treatment, pharmacological therapy must be kept aside for persistent cases of bruxism. The first line inter-

vention should be a psychological therapy. This recommendation was made because the psychological interventions are generally considered not to be harmful, not interfering with maxillofacial growth/development and, therefore, not presenting contraindications. After that, the intervention can be followed by physiotherapy, although no study has investigated if this follow-up improves the results [21].

17.6 Summary

- Sleep bruxism can be regarded as one of the parasomnias, due to motor activities during REM sleep stage.
- Its etiology is multifactorial between peripheral and central mechanism.
- Gold standard for sleep bruxism diagnosis is by using a full-night audio-video polysomnography (PSG) recording, although diagnosis can also be made via clinical judgement or by using ambulatory devices.
- There is no “cure” yet for bruxism. Some studies suggest psychological support as the first line to help children with bruxism.

17.7 Research Gaps

- Difficulty in evaluating the real prevalence of sleep bruxism
- Brain chemistry pathophysiology in causing the bruxism
- Various approaches to reduce or cure bruxism: pharmacological, psychological, physical therapy, dental strategies, alternative medicine
- The association between the bruxers and their quality of life

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