Sleep Bruxism: What Orthodontists Need to Know?

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5.1 Definition of Sleep Bruxism

The American Academy of Sleep Medicine defines general bruxism in the International Classification of Sleep Disorders (ICSD-3 available only on website at http://www.aasmnet.org/library/default.aspx?id=9) as the following: A repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Furthermore, bruxism has been divided into two distinct categories based upon a 24 h circadian cycle as to when this activity occurs: sleep bruxism (SB – occurring during sleep) and awake bruxism (AB – occurring during wakefulness) [1].

5.2 Classification of Sleep Bruxism

According to the ICSD-3, the clinical criteria for classification as SB include the following: (A) presence of regular or frequent tooth grinding sounds occurring during sleep; (B) presence of one or more of the following clinical signs: (1) abnormal tooth wear consistent with above reports of tooth grinding during sleep; (2) transient morning jaw muscle pain or fatigue; and/or temporal headache; and/or jaw locking upon awakening consistent with above reports of tooth grinding during sleep. It should be noted that although polysomnography (PSG) is not required for the diagnosis of SB, it is ideally recorded with masseter and/or temporalis muscle activity along with audio-video signal to increase diagnostic reliability [1].

SB may be classified according to etiology into two distinct categories: (A) primary or idiopathic/essential SB which is without an identifiable cause or any associated medical problem and (B) secondary SB which is related to a medical condition (e.g., movement or sleep disorder, sleep disordered breathing, neurologic or psychiatric condition, drug/chemical related). Orthodontists should be aware that SB may be concomitant with many other sleep disorders such as sleep epilepsy, REM (rapid eye movement) behavior disorder, and sleep breathing disorders due to upper airway resistance or apnea-hypopnea events [2, 3].

SB motor events may also be classified according to motor activity based upon stringent criteria (Table 5.1). Using PSG and audio-video recordings (either ambulatory or from the sleep laboratory), motor activity pattern types based on

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Table 5.1 Criteria for classification of bruxism according to motor activity pattern types as recorded by electromyography (EMG)

Phasic (rhythmic) – more than three EMG bursts (masseter or temporalis muscles) at a frequency of 1 Hz, separated by two inter-burst pauses with each burst lasting between 0.25 and 2.0 s
Tonic (sustained) – one EMG burst lasting >2.0 s
Mixed events – combination of phasic/tonic

EMG electromyographic
Note: For each burst, EMG is 10–20 % or more of the voluntary contraction and each burst must last for at least 0.25 s

Table 5.2 Diagnostic grading system of bruxism, for clinical and research purposes [1]

Possible – based upon self-report using a questionnaire and/or the anamnestic part of the clinical examination
Probable – based upon self-report *plus* the inspection report of the clinical examination
Definite – based upon self-report, a clinical examination and a polysomnographic recording preferably containing audio/visual recordings

electromyographic (EMG) signals of masseter and/or temporalis muscles referred to as rhythmic masticatory muscle activity (RMMA) can be subdivided into phasic (rhythmic), tonic (sustained), and mixed events [4, 5]. The majority of these EMG events (88 %) are of the phasic or mixed variety while rarely do we observe the tonic type that characterizes clenching; these EMG events occur at a mean frequency of 5.4 to 5.8 episodes per hour of sleep [4–6].

Another classification system for SB recently developed by consensus among an international group of experts employs a novel diagnostic grading system for both clinical and research purposes using the terms possible, probable, and definite (Table 5.2) [1].

5.3 Epidemiology

The prevalence of SB is difficult to establish as most of the studies are based on self-report of bruxism and do not distinguish between SB and AB. It has been found that SB peaks during childhood and decreases with age without gender differences [7]. Based on self-report of tooth grinding awareness, SB affects about 8 % of the adult population [7–9]. In children and adolescents, however, there is high variability reported (4–46 %) due to the different age groups under investigation [10–15].

5.4 Risk Factors

There are a number of risk factors for SB including cigarette smoking (Odds Ratio, OR = 1.3), caffeine (OR = 1.4), alcohol (OR = 1.8), and recreational drugs such as ecstasy, cocaine, or amphetamines; medications such as selective serotonin reuptake inhibitors or haloperidol; and sleep disordered breathing (SDB) problems such as snoring (OR = 1.4) and obstructive sleep apnea (OSA; OR = 1.8) [16–22].

On the other hand, SB is a risk factor for tooth wear, damage and fracture, muscle fatigue and pain (primarily in the morning), headache, and temporomandibular disorders (TMD). Of interest, there is an increased risk for tooth wear, jaw muscle fatigue and difficulty with wide mouth opening among children with SB [16].

Orofacial pain has been reported in 66–84 % of SB patients [23, 24]. Contrary to popular belief, increased frequency of SB events is not associated with greater presence or intensity of pain [25, 26]. Rather, a low level of SB activity (between 2 and 4 episodes/h of sleep) increases the risk for orofacial pain and headache complaints among SB patients compared to those with a high level of SB activity (>4 episodes/h of sleep) [26].

5.5 Comorbidities

There are some medical disorders that may be comorbid with SB. Among these are certain sleep disorders including parasomnias such as sleep walking and sleep talking; enuresis; restless leg syndrome; and SDB [8, 22, 27–32]. Also, other medical disorders such as attention deficit hyperactivity disorder (ADHD) [33, 34], Parkinson's disease [35], epilepsy [36–38], and gastroesophageal reflux [39] may be comorbidities of SB.

5.6 Pathophysiology

5.6.1 Sleep Architecture

Normal sleep comprises two distinct states: NREM (non-rapid eye movement), which, based upon electroencephalography (EEG), is subdivided into three distinct stages (N1-3) and REM (rapid eye movement). A typical normal sleep pattern is where individuals progress from wakefulness to the NREM state, followed by the REM state and then cyclically alternating between REM and NREM stages. Overall, a night of sleep consists of approximately 75–80 % of NREM sleep and 20–25 % of REM sleep. Humans typically cycle through NREM/REM sleep stages at a rate of four to six times per sleep period with duration of each cycle being 90 to 110 min. NREM allows for physiological restoration and REM accommodates psychological restoration.

Young adult SB patients (20 to 40 years of age) without coexisting medical problems such as chronic pain or those experiencing OSA exhibit a normal sleep architecture [40]. When investigating the occurrence of SB during the sleep cycles at night, it has been found that SB events are higher in the second and third transition from NREM to REM sleep cycles as compared to the first and fourth cycles [41]. SB events are most frequently identified in the ascending period within a sleep cycle where there is a shift from deep NREM toward REM sleep associated with arousal activity and increase in sympathetic tone [42, 43]. Furthermore, it is important to appreciate that the manifestation of tooth grinding is preceded by a cascade of complex and well timed physiologic events (Table 5.3). Evidence regarding the pathophysiology of rhythmic masticatory muscle activity (RMMA) supports the hypothesis that this activity is associated with autonomic sympathetic cardiac activity and sleep arousals [6, 41, 44, 45]. Arousals are the response of the sleeping brain to external (environmental) and internal (physiological or pathological) stimuli [46]. The purpose of these arousals or active periods are that they are "windows" whereby the sleeping individual can readjust his/her body position, reset body temperature, and if any harmful event is perceived, can become fully awake, i.e., a fight or flight reaction could be triggered [47]. In normal healthy adults, sleep arousals occur between 6 and 14 times per hour of sleep and tend to occur at the end of a NREM period [48]. Approximately 80 % of SB events, i.e., repetitive jaw muscle contractions with or without tooth grinding, are observed during such recurrent arousal periods while the source of the genesis of the other 20 % is under investigation [49]. Evidence that SB and RMMA are associated with sleep arousal is supported by the observation that tooth grinding and RMMA can be

Table 5.3 Sequence of physiological events preceding the oromotor activity of rhythmic masticatory muscle activity/sleep bruxism (RMMA/SB) [44, 169]

Time (prior to RMMA or tooth grinding episode)	Physiologic event
−8 to −4 min	Increase in sympathetic cardiac activity Reduction in parasympathetic activity
−4 s	Increase in cortical – brain activity (sleep arousal) Presence of alpha and delta waves recorded on the EEG
−1 s	Increase in suprahyoid muscle (jaw opening muscles) tone (possibly involved in mandibular protrusion or airway patency) Increase in respiratory and cardiac frequency (tachycardia)
−0.8 s	Initiation of two large inspirations Modest but significant rise in blood pressure
Onset of RMMA	Initiation of phasic or tonic contraction of masseter and temporal muscles (jaw closing muscles), with or without tooth grinding. This is followed in about 60 % of SB episodes by swallowing activity
Note of importance	Approximately 80 % of RMMA events are associated with sleep arousals with or without accompanying leg or body movements
Note of importance	Over 90 % of RMMA/SB events could be predicted by an increasing heart rate of 110 %

RMMA rhythmic masticatory muscle activity, *EEG* electroencephalography, *SB* sleep bruxism

evoked experimentally through manipulations that trigger arousal [2, 6, 22, 23, 50]. Interestingly, there does not appear to be any presence of arousals during RMMA events in normal adult volunteers who do not experience SB [45].

5.6.2 Catecholamines and Neurochemistry

Catecholamines such as dopamine, norepinepherine, and serotonin have been suggested as being involved in SB pathophysiology [20, 40, 51]. Studies have reported that SB patients have elevated levels of catecholamines in their urine compared to controls, thus suggesting a link between stress and SB [52, 53]. In a pilot imaging study [54] involving dopamine, it was found that there was an asymmetric distribution of striatal dopamine binding sites in the brains of SB patients. However, the overall density of the striatal dopamine receptors was found to be within normal range in young adults with SB. In a clinical trial using L-dopa (a dopamine precursor), the results indicated an inhibitory effect on SB; however, when bromocriptine (a dopamine receptor agonist) was administered it did not result in any effect on SB events, and it failed to restore the imbalance of the striatal dopamine binding sites [55, 56].

The observation that smoking exacerbates tooth grinding provides indirect evidence for the role of the cholinergic system mediated through the nicotinic receptors as a mechanism for SB [57–59]. However, it remains to be determined if this occurrence is indeed due to the effect of nicotine receptor activation (increased vigilance and brain arousal), or if it increases the risk of SB as an oral habitual behavior.

5.6.3 Stress and Psychosocial influences

There is a common belief that stress and psychosocial variables contribute to SB. Studies suggest that children and adults reporting self-awareness of tooth grinding are more anxious, aggressive, and hyperactive [13, 17, 60–65]. However, the majority of these studies had methodological limitations resulting in rather weak evidence [66]. SB patients diagnosed by PSG displayed similar reaction times to vigilance as normal controls under an attention motor test condition [67]. Interestingly, the SB patients scored higher than the normal controls on anxiety regarding successful test performance. There is a suggestion among some studies that SB patients are more likely to deny the impact of life events due to their coping styles or personality [68, 69]. Additionally, in some case studies, masseter EMG activity increased during sleep following days with emotional or physical stressors; [70, 71] however, these findings were not consistent in all studies [72–74]. From these studies it can be concluded that there might exist a subgroup of SB patients whose response to life stressors includes excessive jaw motor activity and this reaction differs from that of normal individuals [66, 69, 75].

5.6.4 Genetic and Familial Predisposition

A genetic or familial predisposition for SB has been suggested by studies utilizing a questionnaire format or tooth wear examinations [76]. Twenty to 50 % of SB patients may have a family member who also reports tooth grinding during childhood [77–79]. Analyzing twin studies, it has been revealed that tooth grinding has greater concordance among monozygotic than dizygotic twins [80, 81]. Furthermore, the presence of SB in childhood persists in 86 % of adults [80]. In a large population-based cohort of young adult twins, it was reported that genetic factors accounted for 52 % of the total phenotypic variance [82]. In contrast, Michalowicz et al. [83], on the basis of a combined questionnaire and clinical study with almost 250 pairs of twins, concluded there was a lack of genetic correlation with SB. To date, no genetic variants or genetic inheritance patterns have been associated with SB. Yet, in a recent case-control study involving a Japanese population (non-related participants) it was found that the C allele carrier of the serotonin receptor 2A single nucleotide polymorphism

(rs6313) was associated with an (OR = 4.25) increased risk of SB [84]. This finding is the first to identify a specific genetic component contributing to the etiology of SB. Despite this finding, it must be understood that SB is a multi-factorial disorder in which many other factors including other candidate genes are most likely involved in the etiology of this oral motor behavior or activity.

5.6.5 Local Factors Including Dental Occlusion

Historically, the dental profession was quite convinced that SB was directly related to occlusal factors, and early studies seemed to indicate that occlusal corrections diminished or stopped this activity [85–87]. However, later studies challenged the concept that occlusal factors such as occlusal disharmony or premature tooth contacts could be considered as principal initiating factors, while other studies showed that SB activity was not reduced by occlusal therapy [88–91]. There has also been a lack of correlation between dental morphology (dental arch, occlusion) and SB events among SB adult patients assessed by PSG [92]. Furthermore, the average tooth contact time, including meals, in healthy individuals is approximately 17.5 min/day [93]. Usually tooth contact is absent during sleep without motor activity, whereas it does occur in association with arousal, swallowing, and motor activity [94, 95]. Tooth contacts seem to occur in clusters approximately every 90 to 120 min during the night, suggesting that tooth contact is a consequence of jaw closing muscle activation within a sequence following arousal rather than a cause [95–97]. Interestingly, patients who are edentulous exhibit RMMA when they sleep while not wearing their dentures [98, 99]. In a study by Manfredini et al. [100], it was concluded that the role of various occlusal features such as interferences and centric slides, bite relationships, horizontal overlap, and midline discrepancies in the pathogenesis of SB is very minor and the contribution of occlusion to the differentiation between bruxers and non-bruxers is negligible.

5.6.6 Salivary Flow, Airway Patency, and Jaw Motor Activity During Sleep

Swallowing is a normal physiologic oropharyngeal motor activity occurring five to ten times/hour during sleep, which is a much lower rate as compared to wakefulness (60 times/hour during non-eating periods) [101]. This decreased rate of swallowing during sleep may be related to a decrease in salivary secretion and/or reflex sensitivity. Swallowing seems to occur predominantly in light NREM sleep in relation to arousals [44, 101]. Swallowing has also been found to occur with approximately 60 % of RMMA events in both SB patients and normal adult individuals [102]. Masseter bursts associated with RMMA occur when esophageal pH decreased in SB patients who did not experience sleep-related gastroesophageal reflux [39]. The relationship between swallowing, esophageal pH, microarousals, and salivation requires further investigation as it relates to sleep.

There appears to be an interaction between airway patency and jaw motor activity during sleep. During sleep, due to a decrease in oropharyngeal muscle tonicity, the jaw is open for 90 % of the total sleep time [94]. Narrowing of the upper airway during sleep occurs as the mandible and the tongue collapse into the pharynx [103]. The reduction in this space is exacerbated when sleeping in the supine position as a result of gravitational forces. Intriguingly, 75 % of RMMA events also occur in the supine position [102]. Khoury et al. [104] reported that an increase in the amplitude of respiration was observed with a simultaneous and significant increase in the activation of the suprahyoid (jaw opening) muscles when RMMA events occur. This increase in respiratory amplitude preceding RMMA, however, seems more likely to be associated with an autonomic drive during arousals rather than to function as an opening of the upper airway after an apneic event. Furthermore, studies have shown that RMMA events rarely present after apneic events [105]. Therefore, it remains to be demonstrated whether or not SB is a reactive-protective mechanism of the upper airway to overcome upper airway collapse.

5.7 Clinical Features of Sleep Bruxism

5.7.1 Tooth Grinding Reports

A primary feature of SB is tooth grinding noise. When clinically assessing the presence of SB it is imperative to differentiate tooth grinding noise due to SB from that of other oral sounds emitted from the mouth and throat during sleep such as snoring, grunting, groaning, vocalization, tongue clicking, lip smacking, or temporomandibular joint noise [106]. Additionally, sounds made from the bed itself due to movements and sleeping position changes also must be taken into account. Clearly, it is very difficult for a tooth grinding history to be reliably elicited from the patients who do not have a sleep partner or who are edentulous. In certain individuals, fluctuation in grinding history may be associated with jaw muscle symptoms or other risk factors such as stressors and medication use [58, 107, 108]. Therefore, tooth grinding noise should not be used as the sole determinant of SB activity.

5.7.2 Tooth Wear

The severity of tooth wear can be assessed according to published criteria [109, 110]. However, it is not possible to separate patients with SB from those without by observing tooth wear factors [111], as tooth wear may be produced by other etiologic factors (oral habits, food consistency, acid reflux, alimentary disorders, etc.); therefore, occlusal attrition cannot be considered an accurate indicator of this habit being currently performed [112]. Menapace et al. [113] reported that tooth wear was present in 100 % of SB patients but also in 40 % of asymptomatic individuals. Abe et al. [114] determined that SB patients (young adults) present with greater tooth wear as compared to controls (no report of any history of tooth grinding or sleep laboratory evidence of SB) but tooth wear was not able to discriminate between different sub-groups (moderate/high versus low) of SB patients. Furthermore, SB cannot be assumed to exist if there is no current report of tooth grinding as witnessed by a sleep partner, since the tooth wear may have occurred years before the SB activity.

5.7.3 Jaw Muscle Symptoms

Muscle pain (myalgia) and dysfunction symptoms related to SB may be quite different than those related to concomitant disorders. SB patients most frequently report myalgia on awakening in the morning, whereas masticatory myofascial pain intensifies as the day progresses [115, 116]. Other orofacial symptoms associated with TMD such as limitation in opening, TMJ noise, and arthralgia can be present concomitantly [117]. Although studies have suggested an association between self-reported SB and TMD, causation has not been clearly established [116, 118]. Furthermore, PSG studies have been unable to confirm such a link [119–121]. Raphael et al. [122] in a case-control study (124 vs. 46; all females) investigating the association between SB and myofascial TMD, using two-night laboratory PSG monitoring, found no statistically significant differences in SB rates among cases (9.7 %) compared to controls (10.9 %). They concluded there was no relationship between SB and myofascial TMD, but their study did not address the possibility that SB could be involved in the initial onset or triggering of myofascial TMD. Their findings merely emphasized that treatment aimed at reducing SB among those who already have chronic myofascial TMD may be inappropriate, since myofascial TMD patients do not brux at excessive rates while asleep. Other studies, using PSG and masseter EMG recordings, have reported that SB patients with orofacial pain report significantly less bruxism episodes per hour of sleep and less EMG activity in the masticatory muscles during sleep than pain free controls [123, 124]. It appears the association between orofacial pain symptoms and SB may be somewhat dependent on poor sleep, as pain and sleep have a bidirectional association [116, 125, 126].

5.7.4 Muscle Hypertrophy

Masseter muscle hypertrophy may be bilaterally manually palpated. If these muscles are hypertrophic, the volume of muscle tissue increases approximately two times while the teeth are clenched in comparison to a relaxed state [2]. However, masseter muscle hypertrophy does not strictly imply sleep muscle activity as it can also occur as a result of awake clenching [127].

5.7.5 Awake Clenching

As previously discussed, awake bruxism or AB is considered a distinct nosologic entity from SB. AB, based upon self-report studies, tends to be mainly a reactive process and is induced or exaggerated by stressors and/or anxiety or hyperactivity [107, 128]. SB patients often report an awareness of AB, with patients who have mild SB more often being cognizant of AB and stress than those with severe SB [26]. Physiologic recordings in subjects with and without orofacial pain while experiencing natural stress (before an examination) or during experimental stress (mental calculations) revealed increases in muscle tone, heart rate and/or voluntary chewing/clenching [129–131]. The clinical consequences associated with AB may deleteriously impact dental structures (natural dentition and prosthetic devices) and/or involve pain and dysfunction of the jaw musculature and joints [120, 132–134].

5.7.6 Headaches

Headache is a common finding in the general adult population with a lifetime prevalence of 85–95 % [135]. Headache is also a problem in children, with as many as 70 % of children being affected at least once in childhood [136, 137]. The prevalence of reported headache-related complaints among SB patients is also high (60–90 % of SB patients) [138–140]. Children who have migraine headaches have been shown to have a high prevalence of sleep disturbances, including snoring and SB [141]. Furthermore, it has been reported that 30–50 % of SB adult patients complain of headache either in the morning (most frequently) or during the day [142]. In a descriptive PSG study, it was reported that within a SB patient population spanning from 23 to 67 years of age, 65 % reported morning headaches [143]. The exact mechanisms underlying the possible interactions between SB and headache requires further investigation, but this is a difficult challenge due to the high prevalence of headaches in general.

SB may be a possible cause of tension-type headaches if patients wake with facial and/or temporal skull area pain, with pain typically subsiding as the day progresses [24, 71, 121]. These morning headaches may be explained as a post-exercise soreness in the temporalis muscles [144]. SB patients may report waking up in the middle of the night with pain and tension in facial and cranial areas following sustained SB events. In a study by Kampe et al. [62], 14 % of SB patients reported pain at night, while 31 % reported pain during both at night and daytime. It is important to recognize that nocturnal pain and headaches that may be induced by SB can be confused with similar symptoms experienced by fibromyalgia patients, which include muscle tenderness areas and morning stiffness, fatigue, and poor sleep [145, 146].

5.7.7 Sleep Disordered Breathing (SDB)

A cause and effect relationship between SB and SDB, which is a combination of upper airway resistance syndrome and OSA, has yet to be established despite frequent claims of an association among these entities [17]. However, other studies have shown a correlation between habitual snoring and SB [147]. In a PSG study, increased masticatory EMG activity including RMMA was detected in approximately 50 % (10/21) of adult patients) with OSA [22]. In another PSG study investigating sleep disorders among a group of 53 myofascial pain patients

(75 % met self-report criteria for SB, but only 17 % met PSG criteria for active SB), two or more sleep disorders were diagnosed in 43 % of those patients; insomnia disorder (36 %) and OSA (28.4 %) demonstrated the highest frequencies [119]. In another PSG study involving 119 patients between the ages of 2–16 years referred to a pediatric sleep center for snoring, SB was identified in 70 patients [148]. There have been clinical observations and some studies that have provided indirect evidence of a relationship between SB and SDB by reporting a decrease in SB after the patients have undergone treatments (adenotonsillectomy and continuous positive airway pressure) for the underlying sleep disorder [149–151]. These findings support the hypothesis that RMMA may be a sleep oromotor activity that assists in reinstating airway patency following a respiratory obstruction [104, 152]. It is important to note that the association between apnea/hypopnea and arousals is opposite to the association between SB and arousals; apneic events trigger arousals, while RMMA is triggered during arousals [105]. Nonetheless, several studies failed to show a temporal association between apneic events and RMMA; instead, tonic masseter muscle activity is frequently found at the termination of apneic events [22, 29, 153]. Overall, the factors responsible for the induction of increased RMMA frequency in patients with SB require further investigation.

5.7.8 Gastroesophageal Reflux

In a study of healthy young adults, it was reported that a significant relationship between decreased esophageal pH and RMMA, short EMG bursts and tooth clenching seems to occur when the person is sleeping mainly in a supine position. Of note, only about 10 % of the episodes of decreased esophageal pH (defined as a rapidly decreasing intraesophageal pH with a decrease of more than 0.4 per 2 s) included clenching episodes and the number of clenching episodes was independent of various sleep positions [154]. More specifically, it was found that RMMA is a secondary event to gastroesophageal reflux occurring via sleep arousal and often associated with swallowing [39]. Furthermore, RMMA events including SB were induced by esophageal acidification [155]. It has been proposed that preventing gastroesophageal reflux and avoiding sleeping in a supine position might be effective in decreasing the frequency of SB [154]. Overall, the physiologic link between SB, the increase in salivation and the association with gastroesophageal reflux requires further investigation.

5.8 Diagnostic Considerations

5.8.1 Clinical Assessment

SB is frequently reported to dentists or physicians by the patient and/or bed partner and parents. Given a positive report about tooth grinding, the diagnosis of SB is usually clinical, based on the observation of the following signs and symptoms: abnormal tooth wear, hypertrophy of masseter muscles, fatigue, discomfort or pain of jaw muscles [156]. However, none of these clinical findings is a direct proof of current SB activity. Tooth wear for example, although widely reported as the distinctive dental sign of bruxism in general may be related to many other factors that can influence the presence of attrition and erosion on dental surfaces.

There is an intraoral appliance (Bruxocore™) that indirectly assesses the mechanical impact of SB on the dentition [157, 158]. This appliance covers the upper dentition and is worn for a few weeks while the patient is sleeping, and the surface area and volume of attrition on the appliance are evaluated. When this technique is employed, it has been found that jaw muscle activities during sleep are not always correlated with the degree of wear. Therefore, to reliably and accurately diagnose SB, electronic recording and documenting devices are utilized with strict criteria to detect and classify SB activity. It is also important that the presence of other conditions such as orofacial pain, headache, and SDB be assessed in patients with SB by questionnaire at the time of initial examination.

5.8.2 Ambulatory Monitoring

Attempts have been made to monitor SB activity in natural home settings using ambulatory monitoring. Despite the obvious benefits of these devices such as lower cost and being used in the natural environment, the specificity of SB motor activity assessment remains a limitation [2]. In the absence of simultaneous audio-visual recording, it is difficult to exclude the presence of non-SB-specific orofacial movements during sleep such as swallowing and scratching [159]. A novel portable EMG device (Grindcare®) has been designed to provide online recording of EMG activity, online processing of EMG signals to detect a particular oromotor activity (tooth grinding/tooth clenching), and also for use as a biofeedback device. Encouraging results have been reported from several studies where this device has been utilized due its ability to detect EMG events associated with SB, and to exclude orofacial movements unrelated to SB (grimaces, swallowing, etc.) [160, 161]. In a systematic review assessing the diagnostic accuracy of ambulatory monitoring devices compared to PSG in the measurement of SB, it was concluded that the validity of portable instrumental diagnostic approaches is not sufficient to support any non-PSG techniques employed as a stand-alone diagnostic method in the research setting, with the possible exception of the Bruxoff® device which needs to be further confirmed with future investigations [162].

5.8.3 Sleep Laboratory Recording

Although a variety of tools have been developed to assess jaw muscle activity during sleep, the gold standard for SB diagnosis remains a full night PSG audio-video recording (highly controlled but in an unnatural environment). This is the only protocol, which allows the simultaneous monitoring of sleep electroencephalographic, electrocardiographic, electromyographic, and respiratory signals during sleep. However, PSG recordings are not routinely performed for clinical SB diagnosis, as they are both costly and time consuming. A PSG investigation may be indicated in cases of SB associated with other signs and symptoms suggestive of other sleep disorders, especially SDB. In these cases, the patient should be referred to a sleep physician for further investigations and diagnosis.

5.9 Management of Sleep Bruxism

Treatment of SB is primarily based on managing the harmful consequences of SB. Currently there are three strategies available for the management of SB, namely: (1) behavioral measures; (2) occlusal therapies; and (3) pharmacologic therapies (Table 5.4). Prior to treatment, SB patients need to be questioned about other comorbid medical conditions (e.g., SDB, insomnia, ADHD, depression, mood disorders, gastroesophageal reflux), especially when considering a pharmacotherapeutic approach. This provides an opportunity for management of SB and associated comorbidities, but it should be recognized that some management strategies may aggravate associated comorbidities.

There are many behavioral measures such as cognitive behavioral therapy and biofeedback available for the management of SB with only weak to moderate evidence. However, these strategies are typically cost effective and safe.

Similarly, there are occlusal therapies which are mostly reversible and with good short-term evidence for the management of SB [163]. As these therapies are without significant side effects, they also may be used in the long term. However, there are now studies, which have reported aggravation of snoring and OSA with the use of a stabilization-type maxillary occlusal splint for the management of SB. Therefore, clinicians considering oral appliance therapy for SB should screen patients for snoring and OSA. The effect of the mandibular occlusal splint on snoring and OSA is yet to be investigated [164, 165].

There are several drugs with probable centrally-acting mechanisms involving the dopaminergic, serotoninergic, and adrenergic systems for the management of SB [20]. The evidence on

Table 5.4 Management strategies for sleep bruxism

	Strategy	Comment
Behavioral [160, 170–173]	Avoidance of risk factors: smoking, alcohol, caffeine, drug use	Weak evidence
	Relaxation techniques	Weak evidence
	Good sleep hygiene	Weak evidence
	Hypnotherapy	Weak evidence
	Biofeedback	Moderate evidence in short term
	Cognitive behavioral therapy	Moderate evidence in short term
Occlusal therapies	Occlusal adjustments/removal of occlusal interference	No evidence
	Occlusal appliance [6, 173–178]	Decrease SB activity for 2 weeks only, but able to protect dentition from wear
	Anterior appliance (e.g., Hawley anterior platform or mini-anterior type) [179–185]	No better than full coverage occlusal appliance No evidence of long-term efficacy or safety
	Mandibular advancement appliance [186]	Decrease SB activity (up to 70 % reduction) during sleep, especially when worn in advanced positions (50–75 % of the maximal protrusion of the patients). No evidence of long-term efficacy or safety
Pharmacologic	Clonazepam [187]	40 % decrease in SB activity in the short term with risk for tolerance and dependency.
	Buspirone [188]	Weak evidence
	Clonidine [189]	Reduced SB by 60 %; however associated with severe hypotension in the morning
	Gabapentin [190]	Decrease in jaw muscle EMG and improved sleep. Need larger studies to reproduce this finding
	Botulinum toxin [191, 192]	Decrease in jaw muscle EMG activity during sleep. Its effect is short term

their efficacy and safety is quite minimal, so they should only be considered in severe symptomatic patients and only as a short-term therapy [166].

5.10 The Effects of Sleep Bruxism on Orthodontic Procedures

Currently there are no available data on the prevalence of SB during orthodontics. Also, the effect of orthodontic treatment on SB is unknown. Similarly, the effect of SB on orthodontic treatments or outcomes is unknown. Theories proposing that the attainment of an "ideal occlusion" after orthodontics may negate SB and TMD have largely been debunked. One study reported a decrease in anterior teeth wear by patient report alone after orthodontic treatment was performed on 296 children and adolescent patients [167], suggesting that orthodontic treatment may have a similar effect as oral appliance therapy. However, this study could not exclude AB activity, nor did it study SB utilizing PSG. Hence, the suggestion that orthodontic treatment may temporarily interrupt or permanently reduce parafunctional activities is unsubstantiated. In another study, it was reported that previous orthodontic treatment did not alter the presence of current bruxism (i.e., no better or worse) [168].

Based on a rational approach and clinical experience, SB is not a contraindication for orthodontic treatment. However, if a patient has clinically significant TMD symptoms related to SB, it is prudent that the TMD should be managed prior to embarking on orthodontic treatment to minimize the likelihood of interruption or alteration of the orthodontic treatment plan. Similarly, if TMD related to SB occurs during active

orthodontic treatment, it will be necessary to interrupt that process and treat the pain and dysfunction prior to continuation of orthodontic treatment (see Chap. 3).

Once orthodontic treatment has been completed in a patient with SB, the fabrication of an occlusal splint to protect the dentition and provide retention may be appropriate. The utilization of standard removable or lingual bonded orthodontic retainers is unlikely to withstand the forces of SB and probably will require frequent replacement, so other retention strategies should be considered.

Take Home Messages

- Sleep bruxism is mainly regulated by the central nervous system rather than being initiated peripherally.
- Sleep bruxism is not a simple jaw movement like chewing, but rather there are rhythmic movements with intense jaw muscle contractions.
- Management of sleep bruxism should focus on protecting the stomatognathic system from its harmful consequences, and interventions to do so should be conservative and reversible.
- Orthodontists should screen for sleep bruxism and sleep disordered breathing prior to initiating orthodontic treatment.
- Orthodontists should realize that establishing an "ideal" occlusion is not a preventive or curative measure for sleep bruxism.

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