Chapter 19
Sleep Bruxism

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Bruxism: Definition

The term 'la bruxomanie' was first introduced by Marie Pietkiewicz in 1907. It was later adopted as 'bruxism' to describe gnashing and grinding of the teeth occurring without a functional purpose [1]. Bruxism is an involuntary, nonfunctional repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Teeth grinding may have phasic (rhythmic), tonic (sustained), or mixed (both types) jaw muscle contractions. Bruxism is also defined as 'the parafunctional grinding of teeth'. Parafunctional activities are nonfunctional oromandibular and/or lingual activities that usually include jaw clenching, bruxism, tooth grinding, tooth tapping, cheek biting, lip biting, and object biting.

Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism) [2]. The ICSD-2 lists sleep bruxism among the sleep related movement disorders (which in previous version was listed as a parasomnia) [3]. The condition is defined as 'an oral activity characterized by grinding or clenching of the teeth during sleep, usually associated with electro-cortical (EEG) arousals'. Sleep bruxism (SB) differs in terms of etiology from daytime bruxism and hence should be distinguished from teeth clenching, bracing, or gnashing while being awake which may be purely behavioral in some patients. Some authors have suggested a clinical diagnostic grading system for sleep bruxism such as "possible", "probable", and "definite" [2].

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Epidemiology

Most studies have reported a prevalence from the self-reported symptoms of tooth grinding during sleep rather than any diagnostic testing. SB is common in the general population and represents the third most frequent sleep-related movement disorder. Large number of adults with bruxism also had childhood history of bruxism.

Bruxism is common during infancy and typically follows eruption of deciduous incisors, however infantile bruxism is considered clinically not significant. The prevalence of sleep bruxism during infancy and preadolescent age group is about 14–20 %, in adolescents and adults is about 8 %, and in older persons is around 3.6 % [4]. SB occurs equally between men and women. In comparison to sleep bruxism, thumb sucking is noted in 21 %, while snoring is reported in 14 % of the children. However concomitant oral activity such as nail biting is also noted in 9–28 % of those reporting SB [5].

Etiology

The etiology of sleep bruxism is multifactorial and multiple causes can coexist. The various risk factors of sleep bruxism are listed in Table 19.1. Common sleep disorders such as obstructive sleep apnea (OSA), restless leg syndrome (RLS), and periodic limb movement of sleep (PLMS) are thought to be associated with SB. Sometimes, bruxism may be incidentally noted during sleep studies done for evaluation of other sleep disorders and the patient may not be aware of these symptoms. SB can be idiopathic and may not have a clearly identifiable cause. Cigarette smoking, excessive caffeine intake, and use of some recreational drugs is associated with increase in SB. It can also be induced iatrogenically as a side effect of various medications (see Table 19.1) used in treatment of neurological and psychiatric disorders. Whenever SB is suspected or diagnosed, screening for the common risk factors needs to be undertaken because these factors should be treated for effective management of SB. SB is commonly associated with nocturnal gastroesophageal reflux disease (GERD) in adults.

In a large population study by Guilleminault et al., subjects with obstructive sleep apnea syndrome (odds ratio [OR], 1.8), loud snorers (OR, 1.4), subjects with moderate daytime sleepiness (OR, 1.3), heavy alcohol drinkers (OR, 1.8), caffeine drinkers (OR, 1.4), smokers (OR, 1.3), subjects with a highly stressful life (OR, 1.3), and those with anxiety (OR, 1.3) were at higher risk of reporting sleep bruxism [6].

Table 19.1 Risk factors for bruxism and medications causing bruxism. (Source: Modified from Kyger MH [15])

Sleep disorders	Sleep disordered breathing
	Periodic limb movement of sleep
	Restless leg syndrome
	REM behavior disorder
	Nocturnal seizure disorder
	Night terrors
	Nocturnal gastroesophageal reflux
	Confusional arousals
	Fragmentary sleep myoclonus
Neurological disorders	Tardive dyskinesia
	Cerebral palsy
	Developmental delay
	Tics disorder
	Parkinsonism
	Shy–Drager syndrome
Psychiatric disorders	Dementia
	Anxiety disorder
	Stress
Medications	Serotonin reuptake inhibitors
	Haloperidol
	Lithium
	Methylphenidate
	Nicotine
	Cocaine
	Alcohol
	Caffeine
	Methylphenidate
	Flunarizine

Familial Pattern

SB tends to occur in families and approximately 20–50 % of patients have at least 1 relative with history of tooth grinding. Bruxism is more common among first-degree relatives and monozygotic twins [7].

There are no genetic marker identified that are associated with sleep bruxism. The pattern of inheritance is also unknown.

Pathophysiology

There are several theories postulated to explain bruxism and these are a matter of controversy in the current literature; no single mechanism is able to fully explain this phenomenon. The pathophysiological disturbances causing SB may depend on the underlying etiology. The common explanation described in literature is based on three possible mechanisms which include central causes, peripheral causes, and psychosocial factors.

The central theory is loosely based on imbalance in neuronal plasticity in the basal ganglia. A neurotransmitter such as dopamine is implicated, and it is hypothesized that the direct and indirect pathways of the basal ganglia nuclei involved in the coordination of movements are disturbed in these patients [8]. Nicotine stimulates central dopaminergic action and this may explain why bruxism is more common in smokers than nonsmokers [8]. Serotonin reuptake inhibitors (SSRIs), which exert an indirect influence on the dopaminergic system may cause SB after long term use [9].

As bruxism commonly occurs during sleep, the physiology of sleep has been extensively studied to identify any pathophysiological explanation. The common sleep disorders such OSA, RLS, and PLMS are associated with SB. Hence some authors have termed sleep bruxism as a "parasomnia" triggered by sleep fragmentation. SB is also frequently associated with common childhood sleep disorders such as night terrors, nightmares, sleep walking, and nocturnal enuresis. The mechanism which might trigger these parasomnias may also be involved in inducing SB, and hence this may potentially explain high prevalence of SB in young children with disorders of arousals [10].

The second theory which is commonly discussed in the literature is the role of peripheral factors. It has been postulated that tooth malocclusion and other occlusal inferences have been associated with jaw grinding and teeth clenching. The lack of occlusal equilibrium can stimulate masticatory muscle activity through periodontal nerve receptors and may lead to frequent teeth grinding [4].

The last theory which is implicated in bruxism is psychosocial factors and coping mechanisms. A stressful life is considered as a big risk factor for SB [6]. Individuals with poor coping mechanisms, anxiety, and depression are particularly at higher risk for SB.

Effect of Bruxism on Sleep

As mentioned above, the American Academy of Sleep Medicine (AASM) (International Classification of Sleep Disorders, Second Edition [ISCD II]), classifies sleep bruxism as a sleep related movement disorder [3]. The characteristic electromyographic (EMG) feature of sleep bruxism is repetitive and recurrent episodes of masticatory muscle activity (RMMA) of temporalis and masseter muscles and is usually associated with electrocortical (EEG) arousals [3]. RMMA has a frequency of 1 Hz and occurs during sleep in association with many other orofacial motor activities such swallowing, lip smacking, coughing, smiling, and jaw movements. The RMMA is associated with tooth grinding sounds in approximately half of all cases as reported by their bed partners, parents, and/or self-reports. Compared to healthy individuals, patients with SB have normal sleep stage architecture, distribution, and cycling, and their sleep efficiency and macro sleep architecture is usually normal [11]. Most of these patients are good sleepers. Like OSA, SB is more common in supine position [12]. Recurrent SB is known to cause sleep disturbances in the patient and/or their bed partner due to sound of tooth grinding. It may also cause headaches

and jaw pain in the morning and rarely SB may trigger another parasomnia such as sleep terror, sleep talking or confusional arousal especially in children. In a study by Kothare et al. in children with sleep bruxism, the investigators did not find any difference of sleep architecture between patients and controls, except for a higher arousal index for the bruxism group (36.7 vs. 20.7, $p < .007$). Sleep bruxism occurred more frequently in stage 2 and rapid eye movement sleep, with arousals in 66 % of the cases. They also did not find any relationship of bruxism to gastroesophageal reflux or intelligence [13].

Effect of Sleep on Bruxism

The similar mechanism which causes periodic limb movement of sleep (PLMS), which is another type of sleep-related movement disorder may be involved in SB. Sleep bruxism can also occur after a physiologic event during sleep such as change in body position or yawning. About 80–90 % of RMMA episodes may result in EEG arousal during sleep. SB may be secondary to exaggerated transient motor and autonomic nervous system activation in relation to EEG arousals. Investigators have postulated that rather than triggering EEG arousals per se, SB may represent a series of an event occurring along the sequence of physiologic activations associated with micro-arousals during sleep [14]. There is a change in autonomic activity in moments preceding bruxism followed by increase in EEG alpha activity, increase in tidal volume of breathing and jaw-opening followed by jaw contraction [15]. This may be part of the central pattern generator (CPG) activity which may trigger these events. Frequency of RMMA activity is modulated by cyclical alternating pattern (CAP) during NREM sleep which is characterized by alternating periods of stable and unstable sleep [16, 17]. Most of the RMMA activities are seen in lighter stages of sleep (stage N1, N2) and during sleep stage transition from NREM to REM but rarely during REM sleep.

Clinical Presentation

The diagnosis of sleep bruxism should be based on criteria proposed by the AASM (see Table 19.2). The tooth grinding sounds during sleep is the pathognomonic sign of SB. Most of RMMA activities are associated with tooth grinding sounds during sleep but the patient may not be aware of this. Some patients may present to the dentist with complaints of tooth, jaw, and temporal mandibular pain which may be worse on awakening in the morning. Sometimes, these patients may have symptoms of sleep disturbances such as frequent nighttime awakenings and excessive daytime sleepiness, behavioral problems, and inattention or hyperactivity, thus mimicking ADHD. In a study to assess daytime cognitive performance and behavior in children with bruxism, Kothare et al. reported that 40 % of their patients had elevated scores

Table 19.2 Sleep bruxism: AASM clinical diagnostic criteria [3]

Diagnostic criteria
Reports of tooth-grinding sounds during sleep reported by patient, parent or bed partner for at least 3–5 nights per week in last 3–6 months
One or more of the following:
Abnormal wear of the teeth
Hypertrophy of masseter muscle on voluntary forceful clenching
Discomfort, fatigue, pain in the jaw (and transient morning headaches and or jaw pain)
Jaw muscle activity cannot be explained by any other current sleep, medical, neurological or psychiatric disorders and or use of medication or substance usage

Table 19.3 Questionnaire for detecting if a patient has bruxism [1]

Has anyone heard you grinding your teeth at night?
Is your jaw ever fatigue or sore on awakening in the morning?
Are you teeth or gums ever sore on awakening in the morning?
Do you ever experience temporal headache 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?

on the Achenbach Child Behavior Checklist, indicating significant attention and behavior problems, and there were moderate correlations between the arousal index and several of the behavior-problem scales from the Achenbach Child Behavior Checklist (0.5 to 0.6) [13].

All patients should be screened for nocturnal GE reflux symptoms as these are commonly associated. Symptoms of restless leg syndrome and use of any recreational drugs and/or medications should also be questioned. Table 19.3 lists a set of commonly asked questionnaires to patient for evaluation of bruxism.

Examination

On physical examination, there may be signs of tooth grinding activity such as tooth wear, tooth tenderness, tongue indentation, tooth fractures, hypertrophy of masseter and temporalis muscles, and jaw tenderness on digital palpation [18]. Although these signs are highly suggestive of bruxism, none are a direct evidence of concurrent bruxism. There may also be signs of increased upper airway resistance like Malampatti score 3 and 4, adenotonisllar hypertrophy, micrognathia, and nasal allergies, all of which increase the risk for OSA which in turn is a risk factor for SB. A neurological examination and temperomandibular joint examination is essential to evaluate secondary causes. Newly erupted permanent teeth may not show the wear and tear signs of bruxism.

Table 19.4 Diagnosis of bruxism

Patient questionnaires
Clinical diagnosis
Clinical history
Assess for risk factors (noted in Table 19.1)
Tooth examination for wear
Diagnostic criteria for sleep bruxism [3]
Tests
Measurement of pressure exerted on occlusal splints
Electromyography of masticatory muscles
Polysomnography

Table 19.5 Polysomnographic diagnostic criteria for sleep bruxism. (Source: Modified from references [3] and [15])

Mean SB EMG potentials:>10 or 20 % of the maximal clench while awake
Masseter muscles SB event episodes types are scored on PSG
Phasic (rhythmic): > 3 EMG bursts, separated by 2 interburst pauses, in masseter or temporalis muscle, each burst lasting > 0.25 and < 2.0 s
Tonic (sustained): 1 EMG burst lasting > 2.0 s
Mixed: both phasic and tonic types
Data are expressed in index as
Number of SB events per hour of sleep
Number of SB bursts (contractions) per hour of sleep
Number of SB episodes per night
Duration of SB EMG activity per hour of sleep

Diagnosis

Sleep bruxism diagnosis may be confirmed by using single ambulatory EMG recording to monitor master muscle activity during sleep or multichannel recording for EEG, EMG, ECG, movements and respiration [19]. Table 19.4 lists the diagnostic approach for SB. Although these testing are cheap and can be used in home setting, they are limited by multiple artifacts and lack of audio-video recording. Alternatively polysomnogram (PSG) may be considered for diagnosing sleep bruxism, which will also help to assess associated sleep disorder like OSA, PLMS and/or confusional arousals. As chin EMG alone may not be sufficient to diagnose SB, additional EMG leads on both masseter muscles may also be employed. Figure 19.1 shows characteristics PSG findings of sleep bruxism. The AASM diagnostic criteria for sleep bruxism on a sleep study have been listed in Table 19.5.

Long Term Consequences

The potential long term adverse effect of untreated bruxism includes dental erosion, tooth damage, tooth fracture, tooth sensitivity, headaches, orofacial pain, and temperomandibular disorders (TMD). More than 2/3rd of patients with SB report orofacial pain [20]. Children with SB have higher instances of behavioral and attentional problem [13].

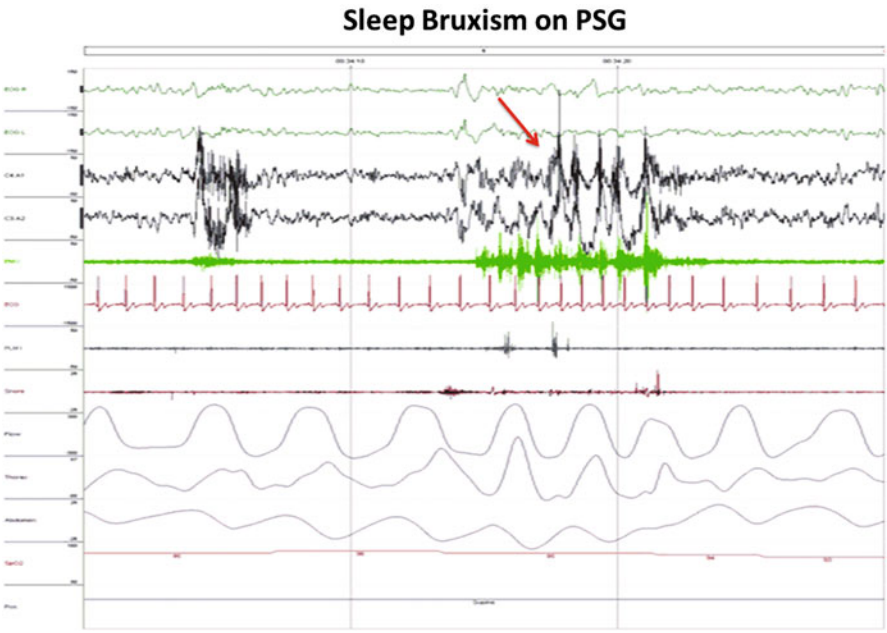

Fig. 19.1 Sleep bruxism on PSG

Table 19.6 Management of sleep bruxism. (Source: Modified from [15, 18])

Behavioral strategies
Avoid cigarette smoking, excessive caffeine, recreational drugs
Good sleep hygiene
Relaxation techniques, better coping skills
Hypnotherapy and biofeedback
Cognitive behavioral therapy
Oral appliances
Occlusal splints
Mandibular advancement devices
Pharmacotherapy
Clonazepam
Clonidine
Gabapentin
Botulinium toxin injection

Management

There is currently no cure for bruxism. The treatment approach aims at evaluation and treatment of risk factors and helps prevent harmful consequences of sleep bruxism. Table 19.6 lists all the treatment strategies for SB. In adults, identifying and avoiding risk factors is an important step toward treating SB. These include avoiding cigarette smoking, excessive caffeine intake, and recreational drugs. Management of stress and

poor coping skills with relaxation techniques, biofeedback, and cognitive behavioral therapy may also be helpful [18].

If bruxism is suspected or diagnosed, referral to dentist should be considered. Treatment with oral appliances such as oral and stabilization splints have been extensively used in dental clinical practice. The main purpose to use these modalities includes protecting the dental surfaces, preventing dental erosion, and relaxing the masseter muscle. By using occlusal splints, bruxism actually may not decrease as shown by RMMA activity, but the wear and tear and dental damage due to SB will be prevented. The teeth grinding noise with SB will also decrease with oral devices and hence may not cause sleep disturbances to the patients' bed partner. Patient with TMD will also find relief with occlusal splints [21]. For sleep bruxism, it is important to limit their use as a habit management aid and to prevent dental damage. Physiotherapy targeting the masseter muscle may also help relax the muscle and decrease SB. In patients with OSA and SB, the mandibular advancement appliances (MAA) may be helpful in increasing the upper airway patency, decreasing apnea–hypopnea index (AHI) and preventing dental damage due to SB. MAA is typically used to treat snoring and mild-to-moderate OSA; short term use of the adjustable MAA has also been demonstrated to be effective in treatment of SB in one crossover sleep laboratory study [22]. Therefore, MAA be an option in a subpopulation of patients with concomitant mild-to-moderate OSA and significant SB.

Pharmacotherapy should be considered if SB persists or oral appliances are not tolerated and associated with adverse consequences such as significant sleep fragmentation, excessive daytime sleepiness, recurrent parasomnias, behavioral and cognitive problems, dental damage, headaches, orofacial pain, and temperomandibular disorder. There are several medications that have been shown to reduce the RMMA and severity of SB which in turn help in supporting the central theory of its pathogenesis. Clonazepam, a benzodiazepine, hypnotic, anxiolytic, and myorelaxant drug is commonly used for treatment of sleep disorders such as parasomnias and sleep related movement disorders, and has been shown to be effective in reducing SB in a recent placebo-controlled study [23]. In addition, clonidine, an alpha-2 adrenergic agonist, a medication commonly used for treatment of insomnia in children is shown to reduce SB [24], however it has the potential side effect of drowsiness and hypotension and is not routinely used.

Botulinum toxin (Botox) injections can reduce the frequency of bruxism events, along with reducing bruxism-induced pain levels. In comparison with oral splints, botulinum toxins are equally effective on bruxism. Use of botulinum toxin injections at a dosage of < 100 U are safe in otherwise healthy patients and is a safe alternative in these patients [25].

Summary

Sleep bruxism is a common sleep related movement disorder which is prevalent in all age groups. It can be asymptomatic and is suspected on dental evaluation due to dental erosion or fractures and/or may be highly distressing to patients due to

tooth erosion, orofacial pain, headaches, sleep disturbances, and TMD. In children, it may be the cause of secondary parasomnia as well as behavioral and cognitive deficits. If SB is suspected or diagnosed, the patient needs to be screened for other risk factors such as nocturnal reflux, OSA, anxiety disorders, neurological disorders, use of medications such as SSRI and or recreational drugs. These patients should be referred to a dentist for further evaluation and treatment. When oral appliances fail or are not tolerated, pharmacotherapy should be considered in carefully selected patient populations. Alternatively, recent data suggest that botulinium toxin injections may be a safe option in otherwise healthy adult patients.

Practical Points

- Sleep bruxism is classified as a sleep related movement disorder
- It is common and seen across all age groups (infants–old age) and prevalence decreases with age
- There are multiple risk factors that are associated with sleep bruxism
- More than 2/3rd of the sleep bruxism episodes are associated with EEG arousal leading to sleep fragmentation
- Usually diagnosis is based on history and clinical examination finding of tooth damage, and jaw pain
- Children with sleep bruxism have higher incidence of daytime behavioral and cognitive problems
- Polysomnogram can be used to confirm diagnosis and a special masseter muscle electrode may need to be used
- Treatment involves referral to dentist, oral splint and mandibular advancement devices
- Pharmacotherapy is effective in treatment of SB in selected population

Case Example

A 28-year-old male presents to a dentist's office with tooth pain and headaches in the morning. This has been happening for several months and he recently fractured a tooth while biting on a walnut. On examination, he has significant erosion of this tooth and loss of molar cusps. There were several cracks visible and tooth tenderness with masseter muscle hypertrophy. Based on this, he was diagnosed with sleep bruxism. On further questioning, he had history of anxiety, heartburn, and was on Zantac as needed. He also has history of loud snoring and excessive daytime sleepiness. He underwent a sleep study which revealed moderate OSA with RDI of 15/h and had several instances of sleep bruxism noted during sleep stage transition and had an EEG arousal index of 30/h. He was recommended CPAP but had difficulty tolerating it. He was referred back to the dentist and was fitted with mandibular advancement

device. He had a sleep study with this oral device and his OSA was well controlled with residual RDI of 2/h and the EEG arousal index was 9/h. On 3-month follow up, his compliance was good and his symptoms had resolved. This oral appliance had helped him control his OSA and help prevent further tooth damage from sleep bruxism.

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