Sleep Bruxism: A Sleep-Related Movement Disorder

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KEYWORDS

• Sleep bruxism • Sleep related movement disorders
• Teeth grinding • Sleep architecture

Sleep bruxism (SB) with concomitant tooth grinding was recently reclassified as a sleep-related oromotor movement disorder falling within sleep medicine. Over several decades, however, the clinical relevance and pathophysiology of SB has been discussed by dental professionals rather than by sleep physicians, because SB has been associated with orodental consequences such as tooth wear, masticatory muscle and temporomandibular joint problems, and dental work fractures, rather than severe sleep disturbance and daytime sleepiness (rare in patients with SB). In this article, the authors review the current knowledge of SB in terms of prevalence, risk factors, diagnosis, pathophysiology, and management.

THE DEFINITION OF SLEEP BRUXISM

SB is defined as a stereotyped oromandibular activity during sleep characterized by teeth grinding and clenching. In 1990 it was classified as parasomnia in the first version of the International Classification of Sleep Disorders (ICSD-1).[1] However, in the revised version (ICSD-2) in 2005, SB was reclassified into the new category, ''sleep-related movement disorders.''[2] Sleep-related movement disorders are classified under simple, stereotypic, repetitive, and localized movements during sleep[3,4]; they also include periodic limb movement disorder and rhythmic movement disorder (eg, head banging).[2] On the other hand, parasomnias also include movement disorders characterized by complicated, seemingly purposeful behaviors during sleep (eg, somnambulism and rapid eye movement sleep behavior disorder [RBD]).

Sleep bruxism is regarded as primary when no clear causes are present.[2,5] SB associated with sleep disorders and neurologic diseases, drug use and medications, and can be regarded as secondary or iatrogenic. The comorbidity with SB, other medical conditions, and drugs/substances are discussed later in this article.

In dentistry, the word bruxism has been used for the diagnosis of oromandibular parafunctional activity occurring during sleeping and waking time.[6–8] This definition can include a broad spectrum of ''nonfunctional'' oromandibular behaviors such as clenching, bracing, tooth gnashing and grinding, nail biting, and even tongue/lip habits. Although some SB patients may be aware of bruxism during wakefulness (eg, tooth clenching), the question of whether bruxism during sleep and wakefulness shares a common physiologic alteration in oromotor controls awaits further investigation.[5,8,9]

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When making a clinical diagnosis of SB followed by a management plan, clinicians need to be aware that both SB and oral parafunctions during wake time may present similar orodental problems such as orofacial pain. To avoid the confusion of the term "bruxism" and to better understand the information in the literature on bruxism, a scheme for the classification of bruxism is presented in **Fig. 1**. This article is devoted to sleep bruxism, that is, the occurrence during the sleep period of rhythmic masticatory muscle activity (RMMA) associated with occasional tooth grinding. The use of the word rhythmic refers to the fact that SB episodes tend to occur recurrently in clusters during sleep. Recording activities of the masticatory muscles with an electroencephalogram allows one to quantify data for diagnosis and to study outcomes related to various management strategies.

PREVALENCE AND RISK FACTORS

The prevalence of SB is estimated by subjective reports of tooth-grinding noise during sleep. An awareness of SB, based on sleep partner reports of tooth grinding, is reported by 5% to 8% of the adult population.[10,11] The prevalence of SB decreases from childhood (10%–20%) to old age (3%).[10–13] No gender difference is noted. The prevalence seems to decrease within a similar range in North American and European countries but might be higher in the Asian population.[10,11,14] It remains to be seen whether this difference is due to the type of questionnaire used, to a cultural awareness in relation to the sleeping environment that may increase the likelihood of tooth-grinding awareness, or to ethnicity/genetic specific factors.

Several risk factors have been reported for SB, although the causal associations between these factors and SB have not been established. SB is 1.9 times more frequent in smokers.[15,16] Caffeine and alcohol intake are reported as a risk for self-reported SB.[11,17] Drugs or substances acting on the central nervous system have been reported to increase the risk of having SB.[5,18] Adult and pediatric patients with anxiety, stress, or certain personality traits may report self-awareness or be the subject of family reports of SB more frequently than those without.[5,11,12,19–22] Familial predisposition has also been reported (see Pathophysiology section).[5]

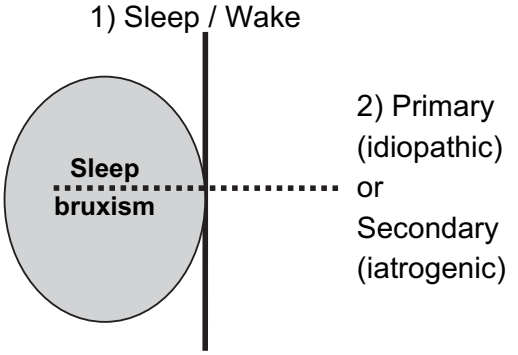

Fig. 1. Classification of bruxism. Bruxism can be classified by 2 axes: (1) sleep or waking occurrence, and (2) primary (idiopathic) or secondary (iatrogenic) type.

Sleep disorders that have been reported to be concomitant with SB include obstructive sleep apnea, parasomnias, restless legs syndrome, oromandibular myoclonus, and rapid eye movement behavior disorders (RBD).[5,23,24] SB is also observed in patients with neurologic, psychiatric, and sleep disorders or following the administration or withdrawal of medication, or any combination of both.[5,8] The secondary form of SB is discussed later.

Sleep bruxism can be accompanied by a risk of secondary orodental consequences (see next section) that may include tooth destruction, dental work failure, temporomandibular joint and jaw muscle pain or jaw movement limitation, and temporal headache.[8,25–27] When tooth grinding and the habit of daytime clenching are concomitant, the odds ratio of reporting temporomandibular disorders or chronic myofascial pain in the masticatory muscles is 4 to 8, although the causal relationship remains to be proved.[5,8,28–31] A recent study reports that 43% of patients with temporomandibular disorder have 2 or more sleep disorders (eg, insomnia, sleep apnea, bruxism).[32] Most children and adult patients with SB (up to 65%) complain of headaches.[13,33–36]

CLINICAL FEATURES

Patients with SB can present the following clinical features (**Box 1**). Young SB patients do not usually complain of sleep disturbance and daytime sleepiness. However, sleep disturbance and daytime sleepiness can be reported in older SB patients or in those who have chronic head-neck pain and sleep disorders.[11,34,37] Aging and pain conditions are important factors influencing sleep organization, and the prevalence of some sleep disorders (eg, sleep apnea, periodic limb movement syndrome) is higher in the older population.[38,39]

Nonetheless, the symptom most common in SB patients is the production of an unpleasant and embarrassing noise during sleep. The noise is created by friction of the teeth related to the frequent and intense rhythmic contractions of the jaw-closing

Box 1
Clinical features related to sleep bruxism

Self-report from patient or sleep partner

– Sleep

  Sleep partner complains of grinding noise (occasionally tapping noise with oromandibular myoclonus)

– Waking in the morning

  Patient reports jaw muscle discomfort/fatigue

  Temporal headache of short duration

  Difficulty in jaw opening, jaw stiffness, temporomandibular joint noise

  Tooth hypersensitivity to cold stimuli (eg, food, beverage or air)

Clinical observations

– Visual inspection

  Tooth wear, fracture, and cervical defects

  Tongue indentation

– Digital palpation

  Masseter muscle hypertrophy during voluntary clenching (bilateral)

  Jaw muscle tenderness (masseter, temporalis) and temporomandibular joint pain

Miscellaneous

Dental restoration failure or fracture (eg, crown, denture, inlay, implant)

Occlusal trauma

Tongue biting (observed in oromandibular myoclonus)

muscles (eg, masseter and temporalis). Although the patient is usually unaware of it, a loud tooth-grinding noise often disturbs the sleep of the patient's bed partner or persons nearby.

On waking in the morning, SB patients report jaw muscle discomfort, stiffness, and fatigue.[40,41] These symptoms can be associated with a high number of jaw muscle events during sleep of the previous night. Jaw muscle symptoms can appear in the temporal regions of head (temporalis muscle area); patients may display temporal headache.

Frequent tooth grinding can be associated with secondary tooth destruction (eg, wear, noncarious cervical lesions and cracks). Tooth wear can be evident on the flat edges of anterior incisor teeth or on the flat occlusal surfaces of molar teeth.[42,43] Although tooth wear is frequent in patients with SB,[44] it cannot reliably determine the current presence of SB; wear could have happened months or years before the time of clinical observation, and approximately 40% of normal persons can exhibit tooth wear.[45,46] Many factors contribute to tooth wear (eg, aging, diet and daytime clenching). Noncarious cervical lesions (a defect in the cervical region of the tooth) are usually associated with tooth brushing and erosion but, for reasons so far unidentified, they are more often observed in patients who are aware of tooth grinding than in those who are unaware.[47,48] The clinician needs to recognize that more cracks and failure lines may be present in the restored teeth of SB patients.[49,50] Tooth damage can be related to an unpleasant tooth sensation or pain. The morning after sleep with intense or frequent teeth grinding or clenching, patients report tooth hypersensitivity to cold liquids or air (eg, when brushing teeth). Patients may also complain of a history of acute tooth pain on chewing hard objects if they have a cracked tooth.[49]

Masseter hypertrophy can be seen in the cheek/face area between the zygomatic bone and mandibular angle when patients clench their teeth, but it does not confirm the diagnosis of SB because a habit of wake-time clenching produces the same results.[46] Tooth ridging and indentations on the buccal oral mucosa or margins of the tongue, respectively, can be observed in SB patients. Again, both masseter hypertrophy and tooth indentation can be also associated with daytime-wake time oral parafunctions such as teeth clenching, tongue pushing, and excessive swallowing.[5,51] Temporomandibular joint problems such as the limitations in jaw opening and clicking noises can be reported by SB patients.

Other conditions secondary to SB include the fracture of dental prostheses and their restoration, occlusal trauma (eg, localized bone loss around the teeth), and complaints of a metallic taste.[52]

RECOGNITION AND DIAGNOSIS

Clinical Evaluation

In ICSD-2, the following items are listed for the clinical diagnosis of SB.[2,5]

– Tooth grinding reports by parents or sleep partner (so far the most reliable)

  Plus:

– Tooth wear (again care must be taken because it may not be time related and may have other causes)
– Jaw muscle discomfort, fatigue pain, and locked jaw on waking
– Masseter muscle hypertrophy on voluntary forceful clenching

SB can be clinically recognized by interview and orofacial examination, and confirmed by electrophysiological recordings (eg, polysomnography) in the sleep laboratory or at home.[5,46,52,53] Gathering anamnestic information and clinical signs and symptoms is a starting point for a diagnosis of sleep bruxism: the information is further confirmed by sleep recording. Moreover, the information gathered from interviews about sleep habits (eg, sleep-wake pattern), sleep-related complaints (eg, daytime sleepiness and fatigue, difficulty in falling asleep, frequent awakening in night, unrefreshing sleep), signs and symptoms of sleep disorders (eg, snoring, respiratory pauses/apnea, excessive movements in sleep/periodic limb movement), and items associated with risk factors (eg, smoking, alcohol intake, medication, stress) help in managing SB and accompanying orodental problems or concomitant sleep disorders.[54] Readers can also consult the another article in this issue (by Bailey) for a better understanding of oral examination techniques and procedures.

Tooth-grinding noise

A history of tooth-grinding noise is the primary feature of SB. The grinding noise should be distinguished from other oral sounds emitted by the mouth and throat during sleep (eg, snoring, grunting, groaning, vocalization, tongue clicking, or temporomandibular joint noise) and from any squeak or clattering sounds made by the bedstead in association with body movements/sleeping position changes.[5,52] A tooth-grinding history cannot be collected in patients who sleep alone or who are edentulous. In some patients, fluctuation in grinding history can be associated with jaw muscle symptoms and with the presence of risk factors for SB (eg, stress, medication).[15,55] Because the occurrence of SB episodes and grinding noise can vary greatly over time, it is helpful to collect information about the frequency, intensity, and any temporal patterns or fluctuations in tooth grinding.[55–57]

Tooth wear

Tooth wear can be observed visually under the light after using air or cotton rolls to dry the teeth. Tooth wear does not necessarily reflect current bruxing activity.[46] The edges of worn teeth on upper and lower dentition fit together when patients slide the lower jaw laterally at an eccentric position. The severity of tooth wear can be assessed according to the previously published criteria.[42,43] Severity ranges from shiny spots on enamel, to dark yellow dentin exposure, to the reduction of crown height in a localized tooth or in a whole dentition. Severity can increase with age.[58] Attrition by dental work (eg, crown, bridge, denture) and erosion by chemicals (gastroesophageal reflux, bulimia, acid foods/beverages) should be ruled out. Wear can be very severe in SB patients in the presence of concomitant dry mouth and hyposalivation.[59] Models made from dental casts can be used to record a pattern of tooth wear and to assess time-course change. Intraoral appliances (eg, Bruxocore) are an alternative technique for indirectly assessing the mechanical impact of SB on teeth.[60,61] Patients use the appliance, which covers upper dentition, during sleep for a few weeks, and the surface area and volume of the attrition on the appliance are evaluated. When this technique is used, it is noted that jaw muscle activities during sleep are not always correlated with the degree of attrition, and that intraoral appliances can have an unpredictable influence on jaw muscle activity during sleep (see Management section).

Jaw muscle symptoms

Muscle symptoms in the face and head related to SB are distinguished from those related to other concomitant disorders. SB patients most frequently report masticatory muscle pain/discomfort on awakening in the morning, whereas myofascial pain in the jaw muscles is most likely to be reported in the evening.[62,63] Temporal headaches (mostly the tension type) on waking in the morning or in the night should be differentiated from mild generalized headache related to sleep breathing disorders (hypoventilation to hypopnea and apnea; see article in this issue by Graff-Radford).[23,64] Other orofacial symptoms related to temporomandibular disorders (eg, limited jaw opening, temporomandibular joint noise, and jaw muscle and joint pain) can be concomitant.[53] Detailed procedures for these assessments can be found in other textbooks.[53] Although several studies have suggested an association between self-reported SB and orofacial pain such as temporomandibular disorders (TMD), causation has not been established.[29,63] Polysomnographic studies were not able to prove such a link.[32,41,65] SB patients with orofacial pain have been found to exhibit significantly lower jaw motor activity than pain-free patients.[66,67] Pain sensitivity in some patients with TMD might be due to the disturbance of sleep continuity (eg, insomnia, sleep duration <6 hours or >9 hours), concomitant sleep disorders (eg, disordered breathing or limb movement), or medication, emotional disorder, persistent pain, and pain in the previous day.[32] The association between orofacial pain symptoms and SB is probably not independent of the

interaction between pain and poor sleep (see the article in this issue by Merrill).[63]

Muscle hypertrophy

Masseter muscle hypertrophy can be palpated on both sides of the face. If hypertrophic, the volume of the masseter muscle increases about 2 times while the patient clenches his or her teeth, compared with the patient in a relaxed state.[5] Patients should be questioned about the presence of habitual concomitant tooth clenching during wakefulness because it can be associated with masseter muscle hypertrophy.[9] Masseter muscle hypertrophy needs to be differentiated from any swelling of parotid glands caused by tumor, inflammation, or blockage (eg, parotid-masseter syndrome).[5]

Daytime clenching

Awake bruxism, mainly characterized by tooth clenching, is thought to be a different entity from SB.[9] Awake bruxism is mainly reactive, and is induced or exaggerated by stress or anxiety.[8] It is reported by 20% of the population, more frequently among females.[9] Awake bruxism can be assessed by conscious awareness, although persons with awake bruxism are often unaware of the habit. Thus, awareness will improve after a doctor informs the patient about the habit and subsequently asks for the patient's report.[68,69] Patients with SB often report awake bruxism: mild SB patients are more frequently aware of daytime clenching and daytime stress than severe patients.[40] Awake bruxism has been reported to be associated with temporomandibular disorders (eg, jaw muscle tension/pain, joint noise, limited jaw opening capacity), tooth wear, and tongue indentation.[70,71] In addition, the coexistence of bruxism in sleep and waking may exacerbate temporomandibular disorders.[41,70,72]

Physiologic Evaluation

Jaw motor activity related to SB can be monitored at home or in sleep clinics using electrophysiological techniques. The techniques are demanding, and so far no simple system has provided a reliable proxy for valid SB diagnosis. To confirm the presence of SB in the ambulatory home setting or sleep laboratory, jaw masseter muscle electromyographic (EMG) recording of the usual polysomnographic montage with audio-video signal is strongly recommended.[4,5,23] For routine clinical purposes, the addition of one masseter EMG with audio-video will allow the frequency of RMMA episodes to be scored as described later; for research purposes burst counts and the exclusion of nonspecific oromandibular activities is mandatory.

Video monitoring

Audio-video monitoring at home can estimate jaw movements and grinding noise.[5] This technique can be useful for children or patients who refuse to sleep at the sleep laboratory with electrodes and sensors. The video camera focuses on the head/neck or upper body regions, but observation becomes difficult when the patient moves out of view. In addition, the lack of physiologic information recorded by electrodes and sensors makes it difficult to identify observed movements and sounds.[73]

Ambulatory monitoring

Ambulatory EMG recordings permit the objective measurement of jaw-closing muscle (eg, masseter) contractions during sleep. Their use in the natural sleep milieu is a major advantage. Recorded data from a single-surface EMG signal usually are stored in a portable battery-operated device. The addition of a heart rate measurement can improve the identification of SB events related to sleep arousals.[74] **Table 1** lists suggested criteria for detecting SB events using an ambulatory system.[75] However, in the absence of audio-video recording, SB episodes cannot be distinguished from oromotor events associated with swallowing, snoring, grunting, coughing, sighs, and other nonspecific jaw motor activity related to body movements, RBD, or Parkinson-related movements during sleep.[5,73] A recent study showed that 85% of body and head/neck movements were accompanied by non-SB activities in normal subjects, and that in SB patients 30% to 40% of oral and mandibular movements were not SB-related. It should be noted that in the absence of audio-video recording, confounding orofacial activities may not be properly discriminated, which can result in the overestimation of SB scoring in normals and SB patients or the misidentification of abnormal motor activities.[3,4,76]

An ambulatory EMG system can detect jaw-closing muscle bursts exceeding an EMG threshold predefined before sleep, and can quantify EMG events during sleep. Several ambulatory EMG recording systems have recently been developed to improve the reliability of recording[77] or to simplify cumbersome recording setups (Bitestrip, GrindCare).[78,79] Based on the authors' experience in a comparative sleep laboratory study, the algorithm of one of these devices does not allow the specific recognition of SB; in the other one the collection of temporalis EMG activity is a reasonable proxy that needs to be further validated in an independent sleep laboratory. Another type of ambulatory recording system has been developed to measure bite force.[80] In this system, piezoelectric film is embedded in the occlusal appliance

Table 1
Criteria for diagnosis of sleep bruxism

Ambulatory recording	
Acquisition EMG bursts EMG events	Sampling rate: 16.7–20 Hz (minimum) Amplitude: >10% voluntary maximum voluntary contraction (MVC) Duration: >3 s Interval: <5 s Heart rate: >5% increase
Laboratory polysomnography (audio-video plus EMG from masseter or temporalis)	
Acquisition EMG bursts	128 Hz (minimum) with audio-video recordings – Amplitude: >10%–20% of MVC – Duration: Phasic: 0.25–2 s Tonic: > 2 s – Interval: <3 s
Episode types	– Phasic (rhythmic): >2 phasic bursts – Tonic (sustained): tonic burst – Mixed: both phasic and tonic bursts
Polysomnographic diagnostic criteria (mild to moderate/severe case based on episode frequency estimated by EMGs)	
A: ≥4 episodes per hour of sleep for moderate/severe case Or <4 episodes per hour of sleep for low case *(mild case 2–4 episodes/h)* B: ≥25 EMG bursts per hour of sleep for moderate/severe case C: At least 2 episodes with tooth-grinding sounds at night (for both low and moderate/severe cases) Cut-off criteria: (A or B) and C	

fabricated for the patient. The diagnostic power of ambulatory systems for SB has not yet been validated in comparison with polysomnographic evaluation. With this limitation in mind, the ambulatory system can still be useful for recording jaw muscle activity in a daily life environment (eg, at home) for multiple nights in a large sample population at low cost.[5,46,81]

Polysomnographic evaluation

Compared with an ambulatory system, polysomnographic recordings are made in a controlled environment for a limited number of nights.[5] Some patients cannot sleep during the first night in unfamiliar laboratory conditions (first-night effects). Thus, in the research setting the first night is used for habituation and the data from the second night are scored for diagnosis.[5] The following biosignals are recorded in this system: a usual montage for the diagnosis of sleep disorders (eg, electroencephalograms EEGs), electrooculograms, EMGs from submental/suprahyoid and anterior tibialis muscles, nasal air flow/pressure, thoracoabdominal movements, pulse oximetry and heart rate, and EMGs of the jaw-closing muscles (eg, masseter and temporalis) and an audio-video monitor.[73] The biosignals allow concomitant sleep disorders to be identified and permit the specific recognition of SB episodes.[5,73] Powerful ambulatory polysomnographic systems are now available for home recording; again, simultaneous audio-video recording is recommended.[57,73,76,82]

Oromotor tasks such as swallowing, coughing, jaw opening, tooth tapping, and tooth clenching need to be recorded before sleep for further signal discrimination. In addition, other sleep disorders need to be distinguished from usual respiratory activities (exclude apnea-hypopnea and Cheyne Stokes breathing, a marker of a cardiac problem) and usual body movements (exclude periodic limb movement in sleep and RDB, a precursor of neurodegenerative disease).[4,8,52]

Scoring sleep bruxism

To begin scoring SB, jaw motor EMG episodes (single or repetitive = rhythmic) are identified in order to score bursts from at least one masseter muscle recording or, ideally, bilateral masseter plus temporalis. The EMG activity should be at least 10% to 20% of the maximum voluntary teeth clenching before sleep. All oromandibular EMG activities are scored in parallel with audio-video signals.[40] As described earlier, SB episodes should be discriminated from oromotor events associated with swallowing, snoring, grunting, coughing, sighing, and other nonspecific jaw motor contractions.

Next, EMG episodes related to SB are identified as RMMA because episodes are repeated across the sleep period. Each episode is further classified into a type: phasic (rhythmic), tonic (sustained), or mixed (a mixture of both), according to the criteria outlined in **Table 1** and **Fig. 2**.[5,83] SB episodes occurring with grinding noise are also documented. Very brief EMG bursts (duration: <0.25 second) with a brief jaw jerk or tooth-tapping movements are scored separately as myoclonic events.[84]

Diagnosis of sleep bruxism

For scoring, technicians count the number of total EMG episodes, the total number of bursts, and episodes with grinding noise. Then the frequency of bursts and episodes per hour of sleep is calculated.[84] The duration of SB episodes per hour of sleep (total duration of episodes divided by total sleep time) is a surrogate outcome variable that is also of interest.[57] Based on the diagnostic criteria, moderate to severe SB can be predicted in 83.3% of patients with SB and asymptomatic subjects can be confirmed in 81.3% of controls (sensitivity: 72%; specificity: 94%; **Table 1**) if 4 RMMA episodes per hour of sleep are scored.[83] This criterion remains constant over several years, although night-to-night variation in SB episodes (25%) and for SB episodes with tooth-grinding noise (50%) has been reported in patients with moderate to severe SB.[56] When these criteria were first proposed in 1996, patients with moderate to severe SB were clinically selected by the presence of frequent grinding noise during sleep at least 5 nights per week in the previous 6 months.[83]

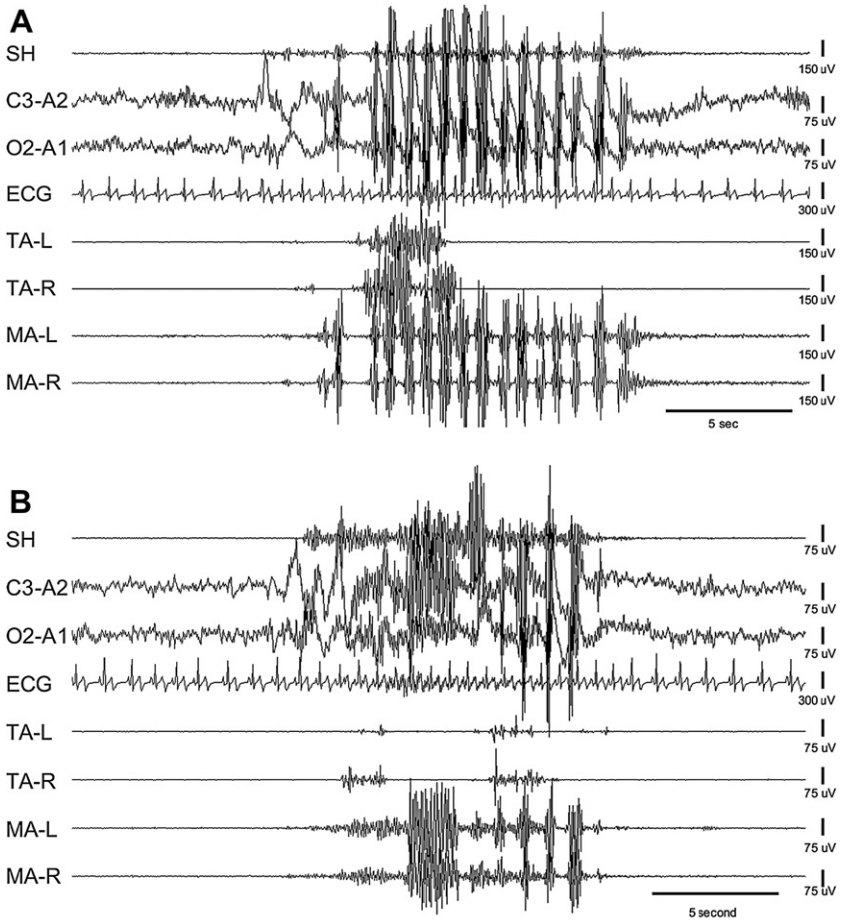

Fig. 2. Polysomnographic examples of sleep bruxism episodes. (*A*) A rhythmic type of SB episode. Phasic masseter (MA) bursts occurred rhythmically in left (L) and right (R) MA muscles. This episode is associated with grinding noise. (*B*) A mixed type of SB episode. This episode is characterized by a tonic MA burst (>2 seconds duration) followed by rhythmic MA bursts. Both episodes are associated with tachycardia on ECG and a change in brain activity on EEG (C3-A2 and O2-A1). During these episodes, EEG changes are obscured by muscle burst artifacts. SH, suprahyoid muscles; TA, anterior tibialis muscle.

When patients with a lower frequency of grinding sounds during sleep ("at least 3 nights per week") were included recently for reevaluation for RDC/SB in a study of 100 SB patients and 43 controls, a cluster of 46 SB patients did not fulfill the criteria mentioned for RDC/SB. Although they had a home history of tooth grinding, in the sleep laboratory setting they presented less than 4 episodes per hour of sleep with occasional tooth grinding.[40] These results suggest that a lower cut-off value of 2.5 RMMA episodes per hour, instead of 4.0, is clinically relevant. However, the diagnostic sensitivity and specificity of this cluster of patients with a lower frequency of SB-RMMA episodes was around 70%.[40] Moreover, the cluster of low (ie, according to frequency-based criteria rather than clinical complaint or tooth damage) SB patients formed a subgroup of patients who were different from the moderate to severe patients (again based on the frequency of EMG episodes, ie, those with more than 4.0 RMMA episodes per hour of sleep). The SB patient group with a lower EMG frequency of SB-RMMA episodes had a higher likelihood of reporting orofacial pain in morning.[40]

Supplemental sleep variables

The following sleep variables are also required for SB diagnosis: total sleep time, sleep latency, sleep stage distribution, and the frequency of arousals and awakenings. In addition, to exclude other concomitant sleep disorders and to understand the patient's sleep profile, the following also need to be documented: variables for diagnosing periodic leg movements in sleep (PLM index), sleep apnea-hypopnea index (AHI), or respiratory disturbance index (RDI), and other observations on polysomnography traces (eg, epilepsy, myoclonus) and video (eg, motor behaviors such as RBD).

PATHOPHYSIOLOGY

Although a specific cause for SB remains to be determined, studies have suggested that the occurrence of SB is subject to multifactorial influences: sleep homeostasis and arousal activity, oromotor excitability, psychological and personality traits, genetics, neurochemical activities, and oropharyngeal functions.[5,8,85,86]

Polysomnographic Findings

Sleep macrostructure

Young adult SB patients (20 to 40 years old) without concomitant medical problems (eg, no chronic pain, no sleep apnea) exhibit normal sleep architecture (eg, sleep latency, total sleep time, sleep stage distribution, sleep efficiency, number of awakenings).[8] Approximately 60% of normal subjects can exhibit RMMA in the absence of tooth grinding, at a frequency of 1.8 times per hour of sleep.[87] On the other hand, moderate to severe SB patients exhibit SB episodes 5.8 times per hour of sleep, more than 90% of which contain RMMA, occasionally associated with tooth grinding.[87,88] In SB patients, the amplitude of masseter EMG bursts is frequently as high as 30% to 40% in comparison with controls.[87] These observations suggest that RMMA in SB patients represents an extreme manifestation of a natural, physiologic oromotor activity.[8,89] As described later, 74% of RMMA episodes can be scored in a supine position and 60% of episodes are concomitant with swallowing in SB patients as well as in normal subjects.[90] Isolated or repetitive myoclonic masseter bursts can be concomitantly observed in SB patients and normal subjects.[84]

Up to 85% of SB episodes are found to occur in light non–rapid eye movement (NREM) sleep (stages 1 and 2).[27,34,83,91–96] Fewer SB-RMMA episodes are observed during rapid eye movement (REM) sleep (approximately 10%) and in deep NREM sleep (approximately 5%–10%) in young adults, in contrast to previous results.[97,98] Regarding the relationship to sleep cycles during the night, the occurrence of SB episodes is higher in the second and third NREM to REM sleep cycles compared with the first and fourth cycles (each cycle lasts between 90 and 110 minutes).[92] In addition, SB episodes are most frequently found in the ascending period within a sleep cycle (eg, the period shifting from deep NREM toward REM sleep).[92] It is known that the ascending period is correlated with an increase in sympathetic tone and in arousal activity.[99,100] Thus, the heterogenic distribution of SB episodes within the sleep cycle and across the night suggests that a normal sleep process related to endogenous ultradian (NREM and REM) or semi-circadian rhythm is an underlying physiologic condition for the genesis of SB-RMMA.

Sleep microstructures

Other observable findings in the sleep microstructure of SB patients include the association between RMMA and phasic EEG and motor events during sleep. Fewer K-complexes were scored during the 10 seconds preceding SB-RMMA episodes in SB patients (12.1%) than in normals (21.2%).[101] Unlike patients with periodic leg movements during sleep, SB patients have a smaller number of total K-complexes and K-alphas during sleep compared with normal

subjects (42.7% and 61.5% lower for K-complexes and K-alphas, respectively).[101] Sleep spindles were not associated with RMMA, and their frequency does not differ between SB patients and normal subjects.[101] Another phasic event related to SB is the microarousal (EEG arousal), characterized by a brief (more than 10 or 15 seconds) cortical, autonomic-cardiac, and motor activation.[99,100,102] Observational studies report that the changes in EEG frequency or alpha EEG waves are scored in association with SB episodes. Tachycardia (increasing up to 25% of baseline heart rate), leg jerk (>80% of episodes), and body movements (up to 24% of episodes) have also been observed in relation to SB episodes.[74,93–95,103–105] Most SB episodes result in sleep stage shifts.[92,95]

Compared with normal subjects, the frequency of microarousals (3–10 second periods of increased activity on EEG, electrocardiographic (ECG), and EMG recordings; note that in the United States the more generic but less precise word arousal is frequently used instead of microarousal) is within an upper range of the normal limit in SB patients (10 to 15 times per hour of sleep).[101,106] The association between microarousals and SB episodes is correlated with the occurrence of the cyclic alternating pattern (CAP) that is repeated every 20 to 60 seconds in clusters during NREM sleep.[93] The CAP reflects cyclic physiologic and behavioral changes in response to endogenous and environmental influences during sleep. More than 80% of SB episodes happen during CAP phase A3 (the high arousal pressure period) and more than half of SB episodes occur in a cluster within 100 seconds.[92] The frequency and duration of CAP has been shown to be similar between SB patients and normal subjects.[93] Microarousal and CAP phase A3 predominantly occur in the ascending phase of the sleep cycle in association with an increase in sympathetic balance.[100,107] The occurrence of SB episodes is thus more likely to be associated with a periodic arousal fluctuation under the influence of a subtle change in the balance of the autonomic nervous system activity during sleep.[8,85,89] However, what predisposes SB patients to be vulnerable to such powerful arousals is unknown.

Physiologic sequence

Recent studies have examined temporal relationships between SB and changes in EEG and autonomic nervous system activity to address the question, ‘‘does microarousal cause SB or does SB cause microarousal?’’ When sympathetic tone was assessed using heart rate frequency analysis, an increase in sympathetic tone was found to present around 4 minutes before the SB-RMMA episodes.[92] Mean heart rate subsequently starts to increase around 10 seconds before the episodes.[103] Next, a significant increase in brain alpha (fast waves) and delta (slow waves) EEG activity and an augmentation in respiratory activity occurs approximately 4 seconds before the onset of an RMMA episode, and a significant increase in heart rate occurs 1 cardiac cycle before an RMMA episode.[34,94] At the onset of RMMA, an increase in suprahyoid muscle activity and a major breathing effort precedes rhythmic jaw-closing muscle activation by 0.8 second (**Fig. 3**).[87,108] A clear sequence was found in 80%–90% of RMMA episodes in both SB patients and normal subjects.[87,103] These results delineate a definite physiologic sequence of autonomic/cardiac, cortical brain, and jaw motor activation in the genesis of SB-RMMA, and further demonstrate that the SB-RMMA episode is the final event during a microarousal (**Fig. 4**).[8,85,89]

Motor Excitability

In general, muscle tone in the limbs, upper airway, and jaw muscles decreases from wakefulness to sleep. The changes in muscle tone are attributed to the ascending or descending neural inputs, neurotransmitter release, and motoneuron excitability.[109,110] The sleep stage dependent changes in muscle tone have been reported to differ between jaw and leg muscles in humans.[111] In masticatory muscles (eg, masseter, suprahyoid), decrease in muscle tone does not differ between NREM sleep stages.[111,112] During REM sleep, masticatory muscle tone becomes minimal but does not disappear completely (eg, hypotonia). In the quiet sleep period without motor activity, masseter and suprahyoid muscle tone in SB patients does not differ from that of normal subjects during NREM and REM sleep.[112] This finding suggests that SB patients have a normal tonic motor excitability in the masticatory muscles. When microarousals were induced experimentally by sensory stimuli (auditory, vibrotactile), arousal responsiveness to stimuli did not differ between SB patients and normal subjects.[112] Nonetheless, RMMA is triggered by arousal response 7 times more frequently in SB patients than normals, and 86% of induced RMMAs involved teeth grinding.[112] SB patients may have an increased transient responsiveness of rhythmic jaw motor excitation in response to microarousal.

Most SB episodes are found to occur with leg and body movements. SB patients have been reported to exhibit increased motor activity in the body during

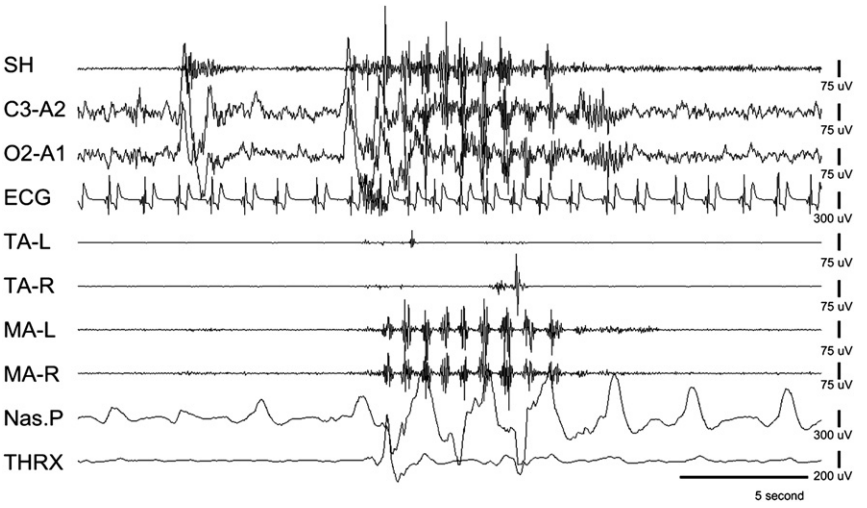

Fig. 3. An increase in respiration in association with a SB episode. A rhythmic SB episode is associated with an augmentation of nasal airway pressure (Nas.P) measured by nasal cannula (Canule). After the episode, an amplitude of respiration gradually decreased to a baseline level.

sleep.[113,114] Several studies have suggested that the degree of motor suppression or activation during sleep might differ between muscles (eg, jaw and limb muscles).[111,115,116] In addition, body movements are most likely to occur in response to the higher level of arousal response (eg, awakening).[110,117] Because arousal responsiveness is associated with an intrinsic difference in the recruitment patterns of autonomic, cortical, and motor activations,[99,110,118,119] a clarification of the thresholds (or excitability) for motor activation in jaw and body muscles would assist the understanding of increased motor activity in SB patients.[120]

Neurochemicals

The influence of neurochemicals on SB activity has been written up in case reports and in the results of clinical trials (see the Management section for more details).[18] It has been suggested that catecholamines such as dopamine, noradrenaline, and serotonin are involved in SB pathophysiology.[8,86]

One pilot imaging study has suggested that dopamine, a catecholamine, is involved in SB. In this study, researchers observed an asymmetric distribution of striatal dopamine binding sites in SB patients.[121] However, the overall density of the striatal dopamine receptors was found to be

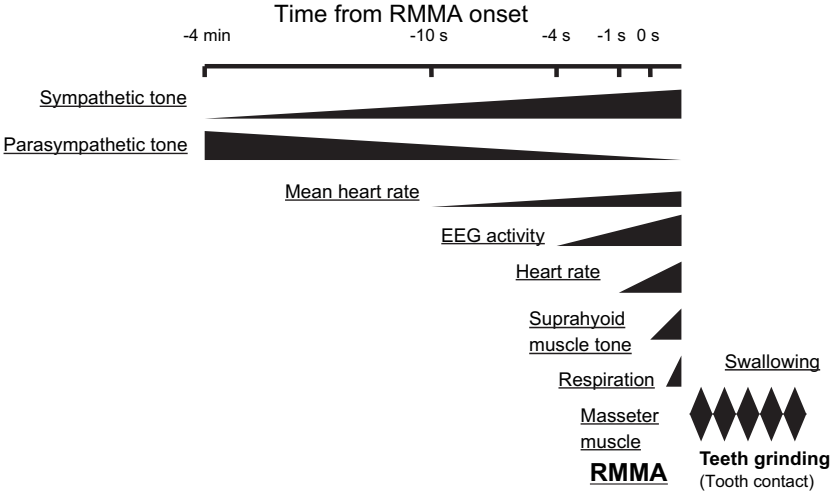

Fig. 4. Proposed sequence of physiologic changes associated to the onset or occurrence of RMMA/teeth-grinding episodes with sleep arousal.

within normal range in young adults with SB tooth grinding. A randomized experimental controlled trial using L-dopa, a catecholamine precursor, had a mild but significant suppressive effect on SB; by contrast, a moderate dopamine receptor agonist, bromocriptine, had no effect on SB episodes, and this medication failed to restore the imbalance of the striatal dopamine binding sites.[122,123] Although a case report suggested that a catecholamine-related noradrenaline β-blocker, propranolol, may reduce SB,[124] a randomized experimental controlled study failed to reproduce the results.[125] The Scandinavian group's initial supposition was of great interest, however, because an α-receptor agonist, clonidine, has been shown to decrease SB in relation to a decrease in sympathetic tone.[125]

Based on clinical observation and questionnaires, the role of serotonin is more difficult to understand: some selective serotonin reuptake inhibitors (SSRI) can exacerbate or initiate sleep bruxism (eg, fluoxetine, sertraline, citalopram), whereas a low-power study found that iatrogenic SB was suppressed by a different type of SSRI drug (eg, buspirone).[18,126,127] Other drugs related to γ-aminobutyric acid (GABA) (eg, clonazepam, diazepam, tiagabine) have suppressing effects on SB activity, but only clonazepam has been tested under a powerful methodological paradigm.[18]

This information suggests that various neurochemicals have a modulating influence on SB. Neurochemicals are known to be involved in sleep-wake regulation, autonomic functions, motor controls, and anxiety/stress conditions. In addition, these neurochemicals may interact with each other and with various endocrine functions (eg, growth hormone, corticotropin-releasing hormone, ghrelin, leptin) that regulate endogenous sleep regulations related to ultradian and circadian rhythms.[128,129] The specific roles of neurochemicals and endocrine systems during sleep and SB activity need to be investigated in a future study.

Stress and Psychological Influences

There is a common belief that psychological stress contributes to SB pathophysiology. Studies have suggested that children and adults reporting self-awareness of tooth grinding are more anxious, aggressive, and hyperactive.[11,12,19,21,22,130–134] However, the evidence is not strong.[135] Several of these studies listed had methodological limitations for interpreting the association between psychosocial factors and bruxism: some made no distinction between a daytime clenching habit and SB, and others did not perform objective physiologic recordings to validate SB diagnosis.[135] Of note, SB patients diagnosed by polysomnography showed similar reaction times and vigilance to normal controls under an attention-motor test condition.[136] However, they scored higher than normals on anxiety regarding successful test performance. A few studies suggest that SB patients are more likely to deny the impact of life events due to their coping styles or personality.[137,138] In addition, in a few case studies masseter EMG activity increased during sleep following days with emotional or physical stress,[139,140] whereas other studies did not replicate the finding.[141–143] In a study of 100 SB patients, a correlation between self-reported daytime stress and masseter muscle EMG activity during sleep was found in 8% of patients.[142] Thus, there might be a subgroup of SB patients whose psychosocial response to life events, in the form of jaw motor activity, differs from that of normal controls.

Subjective SB studies were reported to be associated with increased concentration of urinary dopamine, adrenaline, and noradrenaline during the daytime.[144,145] These results were consistent with those from a study using ambulatory EMG recording.[146] Although high urinary catecholamine is considered to be a response to sympathetic nervous activity and psychological stress, severe SB patients did not have disturbed autonomic functions and perceived less stress than mild SB patients.[92] Catecholamine concentration and sympathetic tone can be associated with other factors such as concomitant chronic orofacial pain, sleep fragmentation, and sleep-related body movements.[147–149] The significance of high catecholamine levels in SB patients clearly remains to be investigated in combination with sleep endocrinology.

To understand the relationship between SB and psychological factors, further studies are needed to clarify the roles of individual susceptibility (eg, genetic or personality traits) and the interaction of sleep and psychophysiological functions (eg, autonomic and endocrine systems) in jaw motor systems.

Genetic Factors

Some studies made using questionnaires or tooth wear examinations have suggested that there is a genetic or familial predisposition for SB. Twenty percent to 50% of SB patients may have a family member who also reports tooth grinding during childhood.[150–152] In twin studies, the report of tooth grinding is more concordant in monozygotic than in dizygotic twins.[153–155] In addition, the

presence of SB in childhood persists in 86% of adults.[154] Nonetheless, a cohort study has found that self-reported tooth grinding can fluctuate over 20 years from childhood to adulthood.[156] Thus, environmental factors are also likely to be involved in the genesis of SB in addition to genetic factors. In addition, sleep parasomnias and SB have been suggested to share genetic influences.[12,153,157] Genetic influences may explain individual differences in the genesis of SB and in SB activity in response to medication, drugs, and psychological stress. The electrophysiological assessment of SB in studies conducted over several generations will be needed to determine genetic factors contributing to SB; cost is a main limiting factor for such studies.

Oropharyngeal Functions

Oropharyngeal structures play several important physiologic roles for functional and tissue integrity (eg, swallowing, respiration) during sleep.[52,59] Swallowing is a physiologic oropharyngeal motor activity that occurs 5 to 10 times per hour during sleep: a much lower rate than wakefulness (up to 60 times per hour during noneating periods).[158] The decrease in swallowing rate may be related to decreased salivary secretion and reflex sensitivity during sleep. Pharyngeal swallowing and subsequent secondary esophageal peristalsis may prevent the invasion of acid reflux to the oral cavity, pharynx, and lung in patients with gastroesophageal reflux.[159] Swallowing, therefore, plays a protective function during sleep, probably in association with saliva.[59] Swallowing occurs predominantly in light NREM sleep in relation to arousals.[89,158] Swallowing was found to occur with approximately 60% of RMMA episodes in SB patients and normal subjects.[90] Masseter EMG bursts associated with RMMA were found to occur when esophageal pH decreased in SB patients who did not suffer from sleep-related gastroesophageal reflux.[160] In children, no correlation was found between SB episodes and esophageal pH.[106] The interaction between factors such as swallowing, esophageal pH, microarousals, and salivation needs to be studied in association with autonomic and gastroenteric systems that are linked to sleep and visceral functions.[8,161,162]

During sleep, the jaw is usually open for 90% of total sleeping time because oropharyngeal muscle tone decreases.[163] The mandible and tongue collapse into the pharynx, which results in a narrowing of the upper airway during sleep.[164] The reduction in upper airway space is worst in a supine position, where obstructive sleep apnea can occur most frequently in obstructive sleep apnea syndrome (OSAS) patients.[164] Of note, 75% of RMMA episodes were found to occur in a supine position.[90] A recent study has shown that respiratory activity shows a simultaneous and significant increase on the activation of the suprahyoid muscles when RMMA episodes occur (see **Fig. 3**).[108] However, an increase in respiratory amplitude preceding RMMA episodes is more likely to be associated with an autonomic drive during the arousal response, rather than with the upper airway opening found after an obstructive apnea event. RMMA episodes rarely occur after apneic events[23] and the role of limited airway flow or upper airway resistance remains to be demonstrated, as suggested by Simmons and colleagues at the last Sleep2009 meeting in Seattle[165] (see also section on Secondary SB). Nonetheless, another study has reported that the use of an oral appliance that opens the airway reduces the frequency of RMMA episodes in SB patients who do not have sleep-disordered breathing.[166,167] It remains to be demonstrated whether arousal levels related to subclinical airflow limitation might be one of various intrinsic factors contributing to the genesis of RMMA.

Peripheral Occlusal Factors

Contrary to a common belief in dentistry, current knowledge does not support the idea that occlusal factors such as premature tooth contacts trigger SB. [8,85,86,168–170] In healthy people, an average time for tooth contacts, including meals, is 17.5 minutes per day.[171] Tooth contact is usually absent during sleep without motor activity, whereas it can occur in association with arousal, swallowing, and motor activity.[163,172] In addition, tooth contacts are found to occur in clusters approximately every 90 to 120 minutes during the night, suggesting that tooth contacts during sleep are a consequence of jaw-closing muscle activation within a sequence following microarousal.[80,172,173] In addition, edentulous patients exhibit RMMA when they sleep without their dentures.[174,175] Moreover, no correlation between dental morphology (eg, dental arch, occlusion) and SB episodes has been found in adult SB patients assessed by polysomnography.[176]

IATROGENIC AND SECONDARY BRUXISM

Various drugs and chemical substances have been reported to exacerbate SB (**Box 2**). Orofacial movements during sleep, including secondary SB, have been reported in several movement and neurologic disorders (**Box 3**).[24,177] Evidence of iatrogenic and secondary SB is scarce because

Box 2
Drug and chemical substances associated with sleep bruxism

Chemical substances: habitual or recreational use[5,11,15–18]

- Alcohol, caffeine, nicotine (smoking)
- Cocaine, 3,4-methylenedioxymethamphetamine (MDMA; ecstasy) (mainly for bruxism during wakefulness)

Medications[a]

- Antipsychotic drugs: haloperidol, lithium, chlorpromazine
- Antidepressive drugs: SSRI (eg, floxetine, sertraline, paroxetine, venlafaxine)
- Cardioactive drugs: Calcium blocker (eg, flunarizine)
- Psychostimulants: methylphenidate[178,179]
- Nonpsychostimulants: atomoxetine[180]

SSRI: selective serotonin reuptake inhibitors

[a] For details, see Lobbezoo et al, 2001,[181] Kato et al, 2001,[52] Winocur et al, 2003,[18] Kato et al, 2003,[24] Lavigne et al, 2005,[5] Lobbezoo et al, 2006.[181]

Box 3
Secondary sleep bruxism (eg, tooth grinding reported to be concomitant with the following medical conditions)

Movement disorders

- Hyperkinetic movement disorders: Oromandibular dystonia, Tics (Tourette syndrome), Huntington disease, Hemifacial spasms
- Hypokinetic movement disorders: Parkinson disease
- Neurologic/psychiatric disorders and other medical conditions
- Neurologic: Cerebellar hemorrhage, Olivopontocerebeller atrophy, Whipple disease, Shy-Drager syndrome, Coma, Mental retardation[a]
- Psychiatric: Anxiety disorder, Depression, Attention deficit hyperactivity disorder[a]
- Other medical conditions: Angelman syndrome[a],[182] allergy[a],[183]

Sleep disorders

- Insomnia
- Snoring[a], obstructive sleep apnea[a]
- NREM parasomnias: Sleep walking, night terrors, confusional awakening
- REM parasomnias: Rapid eye movement sleep behavior disorders, also named RBD
- Oromandibular myoclonus
- Sleep groaning[184]
- Sleep epilepsy[185,186]
- Enuresis[a],[187,188]
- Restless legs syndrome, periodic limb movement disorders

[a] Sleep bruxism is reported in pediatric patients. For details of secondary sleep bruxism, please see Huynh and Guilleminault 2009,[189] Lavigne et al, 2005[5] and Kato et al, 2003.[24]

most data are derived from case reports without electrophysiological assessment of SB.

In several case reports, tooth grinding and clenching during sleep have been reported in movement disorders such as oromandibular dystonia, Parkinson disease, Huntington disease, hemifacial spasms, tic, epilepsy, and neuroleptic-induced abnormal involuntary movements. Patients with olivopontocerebellar atrophy, Whipple disease, and Shy-Drager syndrome have been reported to show SB. SB is often reported in pediatric and adult patients with psychiatric and cognitive problems.[133,190–196]

Sleep bruxism has been reported to occur in several sleep disorders.[24] Whether concomitant occurrence of SB in sleep disorders is associated with secondary influence of sleep disruption (eg, increased microarousals) or with a presence of common mechanisms for oromotor activation remains to be investigated.

In a cross-sectional epidemiologic study, snoring or OSAS was reported in more than 30% of adult subjects with signs and symptoms of SB (eg, grinding history and morning jaw muscle discomfort).[11] The odds ratio of having SB was 1.4 for snoring and 1.8 for sleep apnea. In a few polysomnographic studies, tooth grinding/RMMA events were observed in 40% to 60% of a small group of adult patients (10–20 patients) with OSAS.[197,198] However, these studies failed to show a temporal association between apneic events and EMG episodes of RMMA in patients with OSAS, suggesting that postapneic respiratory activation might be a different form of physiologic response from respiratory activation preceding RMMA.[108] Instead, tonic masseter muscle activity was frequently found at the end of apneic events, as a nonspecific oromotor activation in response to apnea-induced arousals.[177,197–201] Sensory impairment of the pharynx has been found in OSAS and snoring patients, but the influence of such changes on motor activity in response to arousals is not known (see the article in this issue by Guilleminault).[23] Further studies are needed to determine whether the concomitant occurrence of SB is associated with a degree of sleep fragmentation (eg, severity of apnea) rather than an increase in postapneic arousal responses in patients with OSAS.

The concomitant occurrence of sleep bruxism and sleep apnea or snoring has been reported in pediatric patients.[13,22,202] It has also been suggested that upper airway and face morphology contribute to the SB seen in pediatric patients.[203–206] Because upper airway morphology is a significant risk for snoring and sleep apnea in children, the occurrence of SB in children with abnormal upper airway morphology provides a future challenge to be considered in the pathophysiology and management strategies in pediatric SB patients.[189]

Parents more frequently report tooth grinding in children with common pediatric parasomnias (eg, sleep talking, sleepwalking, enuresis, night terrors) than in children without.[189] Familial predisposition and correlation with anxiety and stress have also been reported for these parasomnias.[153,207] In adults, the prevalence of SB is 1.5 to 3 times higher in patients with violent parasomnias such as REM sleep behavior disorder, sleepwalking, or night terrors.[193] Most SB episodes are associated with leg and body movements whereas periodic limb movement disorder is found in few SB patients.[24,34] Tooth grinding was reported in only 10% of patients with restless legs syndrome.[10] Oromandibular myoclonus (OMM) is characterized by repetitive or isolated tappinglike jaw movements.[84] Familial patterns can be traced for OMM.[208] Approximately 10% of SB patients can exhibit OMM, although OMM is a different entity from SB.[84,209] Patients with oromandibular myoclonus may complain of nocturnal tongue biting.

MANAGEMENT

Because researchers have yet to determine the specific causes of SB, current suggestions concentrate on managing the consequences of SB tooth grinding rather than proposing a curative treatment. The approaches proposed for managing SB range from behavioral modification and orodental appliances and splints to pharmacologic strategies (**Table 2**). It is relevant to note that not all approaches have been found to be effective, and some risks or side effects may prevent their use in some patients.[210] The clinician's choice of management option is driven by the need to protect orofacial structures from damage, to relieve any accompanying pain-related sensory complaints, and to reduce the putative risks for exacerbation, while taking into account the patient's medical history, age, and benefit-efficacy over side effect or risk ratio.[5,211–213]

Behavioral Strategies

Two major behavioral strategies for managing SB are psychological relaxation and lifestyle instruction, and approaches that include sleep hygiene and the use of biofeedback techniques.

Sleep hygiene instructions are used to guide patients toward good-quality sleep and the avoidance of several risk factors for SB (eg, stress, alcohol, smoking, and irregular life habits).[5,214,215] First, doctors or dentists explain current concepts of SB risks and pathophysiology. Then the following instructions are given: (1) avoid intense mental and physical activities during the late evening and relax before sleep; (2) avoid large meals and beverages such as coffee, tea, and alcohol, and avoid smoking in the evening; (3) install a comfortable sleep environment (eg, containing elements like a quiet room, a moderate temperature, a comfortable bed set); and (4) maintain a regular bedtime hour (if patients are engaged in shift work, the work schedule would be balanced with recovery rest periods).

For relaxation, patients learn a relaxation or meditation technique such as abdominal breathing or biofeedback practice. The patient can then practice the technique in daily life whenever he or she becomes aware of stress and tension, or before sleep. Psychologists can help patients to master these procedures. Although these instructions seem a reasonable approach to managing SB, their therapeutic effect on SB has been rarely tested. One study tested the effects of cognitive-behavioral therapy (CBT) in which patients attended a combination of stress management and nocturnal biofeedback sessions for over 12 weeks.[216] CBT reduced SB activity, as measured by abrasion on an oral device, associated symptoms, and psychological impairment. However, the effects of CBT did not differ from those observed with the use of an occlusal splint and did not last for 6 months. The approaches outlined would be appealing to patients with complaints of concomitant insomnia or sleep disturbance or whose sleep is instable. Psychological management and other strategies would also be considered for SB patients exhibiting a tendency toward maladaptive coping.[138] Because primary SB patients exhibit normal sleep structure, the efficacy of sleep hygiene, relaxation techniques, CBT, and hypnotherapy for sleep stability and SB remains to be demonstrated in a controlled study. In an open study, hypnosis reduced EMG activity and tooth grinding.[217] This result needs to be confirmed in a controlled study.

Biofeedback paradigms activated by masticatory EMG activity (eg, sound stimuli) were reported

to reduce SB activity. However, the effect does not seem to persist after treatment ceases.[218,219] Because loud sound stimulation awakens patients from sleep, it is a potential cause for daytime sleepiness. In addition, sound stimuli may disturb the sleep of the patient's bed partner. Alternative stimulus modalities, such as vibration on the teeth and electrical shocks to the skin of the lip and forehead, have been tested in several studies. In a few case studies, a vibratory stimulus or an unpleasant taste stimulus applied in the mouth reduced SB activity, and the effects lasted over several months.[220,221] Non-noxious electrical stimulation to the lip at the time of tooth contact decreased the duration of SB episodes rather than the number of episodes.[222] Another study used non-noxious electrical stimulation on the skin of forehead.[78] The number of jaw motor events was decreased during a biofeedback treatment period while the signs and symptoms of temporomandibular disorders did not change. In addition, subjective sleep quality and total sleep time did not differ between the periods with and without treatment.[78] Complete polysomnographic recordings, to assess influence on sleep continuity, were rarely used in the studies employing the biofeedback paradigm.

This paradigm has transient effects during the treatment period. Thus, the efficacy of long-term use needs to be evaluated in terms of the patient's habituation to the stimulation and the accumulated influence of subtle sleep disturbance. Another question is whether sensory stimuli used in the biofeedback system suppress jaw-closing muscle activity directly by exteroceptive suppressive influence or indirectly by sleep modification and cognitive influence.

Orodental Strategies

Oral appliances such as occlusal splints and mouth guards have been used for managing SB and temporomandibular disorders in dentistry for years. However, the physiologic mechanisms underlying the action of the devices remain to be demonstrated.[223,224] Oral appliances can be fabricated in hard (acrylic resin or thermosensitive material) or soft (vinyl silicone on the occlusal surface with a hard body, or a full appliance in soft material) materials in a dental laboratory or in the clinic using special systems currently available on the market. A dentist needs to adjust such appliances to the patient's dentition.[53] Both hard acrylic occlusal splints and soft vinyl mouth guards usually cover maxillary or mandibular dentition to control the mechanical load on the teeth or dental restorations.[53,223] Based on clinical experience, a hard occlusal splint is mainly recommended for long-term full-night use. A soft mouth guard is principally suggested for short-term use in adults, because it is less expensive. However, a soft mouth guard is the appliance of choice for pediatric SB patients with mixed dentitions because the oral device needs to be remade as teeth are replaced and grow. However, caution is needed when recommending mouth guards: they may increase SB activity in 50% of patients.[225]

Like a soft mouth guard, a hard occlusal splint does not always reduce SB activity in all subjects, and its effect on muscle activity seems to last for only a few weeks. The effect of an occlusal splint on the frequency, intensity, and duration of SB episodes has varied between or within studies (eg, decreasing or increasing effects, and no effects).[104,219,225–230] Even though an occlusal splint does not affect sleep architecture and is an effective technique to protect teeth from damage in patients with SB, the splint may disturb some physiologic orofacial activity during sleep (eg, swallowing).[227]

The choice of the best oral appliance design remains open to debate; studies comparing the effects of different designs of occlusal splint (eg, pattern of contacts between splint and dentition) on SB have failed to show any significant difference between the various models.[231] Using a cross-over design, a few studies demonstrated no difference in the effects of an occlusal splint and a palatal splint that did not cover maxillary teeth.[227,228,230] More importantly, in a study of subjects using an occlusal splint for 6 weeks, the decreasing effects, if any, lasted for only 2 weeks and did not continue after withdrawal.[219,228,232,233] These findings suggest that the occlusal splint should be used for protecting teeth from the force generated by jaw muscle contractions, rather than for controlling SB activity.[223,224,234] This concept is appropriate because the genesis of SB is regulated by the central nervous system.[8,85,86]

Although dentists have generally considered the occlusal splint to be a conservative and safe management option for SB management, a recent study has raised the possibility that it might have a harmful influence on breathing during sleep. It was found that the use of the occlusal splint in patients with obstructive sleep apnea could aggravate abnormal breathing.[235] This preliminary open study suggests that it is important for clinicians to assess a history of sleepiness and snoring in patients at risk of OSAS. It is recommended that the clinician assess sleepiness with an Epworth scale and estimate the risk of sleep breathing disorders (reports of the cessation of breathing

Table 2
Management strategies for SB

Strategies	Effects (evidence): comments
Orodental strategies	
Occlusal splint	+ or – (B): Protects tooth from grinding-related damage; short-term reduction of EMG activity but after 2–4 wk levels seems to return to baseline values; possible risk for exacerbating snoring and apnea
Mouth guard	+ or –: Short term; may increase EMG activity
NTI splint	+ (B): Short term data only; may change occlusion if used for a long term
Mandibular advancement appliance (MAA)	++ (B): Short term data only; teeth or jaw pain if not well titrated
Occlusal therapy	Questionable/Low level of evidence as a universal therapy; not reversible
Behavioral strategies	
Management of life style, stress, and sleep hygiene	+ or – (B): Lack of strong evidence/Expected to reduce SB: may help if combined with other strategies. Coping style of SB patients is considered
Biofeedback	+ (B): Reduction of EMG activity; No influences on subjective sleep quality if short-term use. Unknown influence on sleep for long-term use (may increase sleep arousal frequency and intensity)
Pharmacologic strategies	
Anxiolytic	Empiric data only
Diazepam	+ or – (B):
Clonazepam	+ (A): Risk of dependence; not for regular use—short term (1–3 nights per week)
Muscle relaxant	
Methocarbamol	+ (B) Risk of dizziness and sleepiness
Dopaminergic	
L-Dopa	+ (A)
Bromocriptine	– (A)
Pergolide	+ (one case so far)

Cardioactive	
Clonidine	++ (A): Risk of severe hypotension in the morning if given to normotensive subjects
Propranolol	− (A)
Antidepressant	
L-Tryptophan	− (A)
Amitriptyline	− (A)
Buspirone	+: reduction of SSRI-induced sleep bruxism (few cases only)
Proton-pump inhibitor	
Rabeprazole	+ (A): Reduction of SB and low esophageal pH events
Botulinum toxin	Little evidence available, small sample size controlled report

(A) and (B) correspond to the grade of evidence. Grade (A): randomized controlled trials and meta-analyses; Grade (B): other level of evidence such as well-designed controlled experimental trial and uncontrolled studies. No grade was added for case reports. For details, see text.

during sleep; hypertension, retrognathia, deep palate, large tongue, narrow dental arch) when prescribing an occlusal splint. Moreover, during the follow-up period, signs and symptoms related to sleep apnea should be reassessed. Patients need to be informed that oral splints may change the way they feel their bite during the hours following awakening, but that such effects are usually transient. It is recommended that dentists make follow-up appointments to assess oral appliance stability and oral hygiene (eg, caries or gum disease) every 6 months. Little information is available on the management of SB with occlusal splints in children. A few descriptive studies have reported that an occlusal splint prevents tooth wear in 3- to 5-year-old children.[236,237]

Apart from the occlusal splint, different types of oral devices have been tested for their efficacy in reducing SB. One oral device, the NTI (standing for Nociceptive Trigeminal Inhibitory), only covers the upper incisors, creating a one-point contact with the lower incisors. The NTI significantly reduced the frequency and intensity of SB.[238] Compared with this device, the occlusal splint has more therapeutic effects on the signs and symptoms of temporomandibular disorders, but the risk of changes in occlusion needs to be disclosed to patients if the NTI splint is used long term.[239,240] Researchers also tested the effect of another type of oral appliance on SB patients, the mandibular advancement appliance (MAA), which covers the dental arch and is made for sleep-related snoring and sleep apnea. The MAA allows a few degrees of mandibular advancement in comparison with a single arch occlusal splint.[166,167] When the jaw was placed either in an edge-to-edge tooth position or in a slightly advanced position using an MAA, the index of masticatory muscle activity was reduced significantly in comparison with an upper-maxillary or lower-mandibular occlusal splint.[167] In the first study, a thermo-molded appliance was fitted to the patient's dentition. More than 60% of patients reported discomfort or pain in the jaw and teeth when they used the oral appliances.[167] In the second study a laboratory custom-made hard appliance was used.[166] Although the mechanism for reducing SB by means of an oral appliance remains to be understood (eg, preventing airway collapse during sleep or jaw retrusion that may occlude the airway passage or the reduction of free mandibular movement due to the mechanism that advances the lower jaw), this result suggests that as long as the titration is appropriate, an MAA can be useful for managing SB in patients with sleep-disordered breathing. This possibility needs further investigation.

The belief that fine tuning the upper and lower jaw tooth contacts (ie, equilibrating tooth contacts by trimming "premature" tooth contacts on natural teeth or dental restorations) cures or relieves bruxism is not supported by controlled and bias-protected protocol.[168,241] In theory, this type of "occlusal adjustment therapy" stabilizes the forces at the temporomandibular joint or between the teeth. As described in the earlier section on Pathophysiology, current knowledge of SB does not support the concept that teeth contacts generate SB; the efficacy of occlusal adjustment is yet to be demonstrated in a controlled study.[86,170] Thus, whereas occlusal therapy is indicated for the restoration of orodental comfort when there are major prosthodontic (eg, crown, denture, bridge), restorative (eg, inlay) or orthodontic treatments, firm evidence is awaited as to its efficacy in SB management due to its irreversible nature in patients with natural dentition.[27,168,242,243]

Pharmacologic Strategies

Several drugs acting on the central nervous system have been suggested to reduce SB. However, it is unclear whether they act directly on the motor system related to SB or indirectly on the sleep arousal system. In addition, long-term efficacy has not been assessed for the drugs presented here.

In an open study, central muscle relaxants (eg, methocarbamol; 1–2 g/night) have been reported to have the effect of reducing SB.[244] Benzodiazepines (diazepam; 5 or 10 mg/night) at bedtime have been reported to reduce SB.[245] Compared with placebo, triazolam improved sleep but did not alter jaw-closing muscle activity during sleep in patients with orofacial pain.[246] In a single-blind, nonrandomized study, the acute effects of clonazepam on SB were investigated in SB patients with insomnia, restless legs syndrome, and periodic leg movements in sleep.[247,248] Clonazepam (1 mg/night) at bedtime decreased SB by approximately 30%, improved sleep quality, and reduced concomitant sleep-related movement disorders. Thus, low to modest effects can be expected when these drugs are used for a short period (eg, 1 or 2 nights). Although a long-term efficacy of clonazepam (approximately 1 mg/night for up to 3.5–8 years) has been reported for parasomnias (eg, sleepwalking, RBD) with low adverse effects,[249] further controlled trials for long-term usage are needed in SB patients not presenting other medical disorders. Patients should be informed that these drugs carry significant risks of dizziness, sleepiness, and cognitive impairment. Moreover, the risk of addiction or dependence needs to be assessed.

In a placebo-controlled study, small doses of amitriptyline (25 mg/night) or the serotonin precursor L-tryptophan failed to reduce SB activity and associated discomforts.[250–252] SSRI antidepressants should be avoided for SB management because several case reports have indicated that they may induce secondary SB.[253–258] Nonetheless, some cases with SSRI-induced SB may be resolved by a different type of SSRI drug (buspirone).[127] The influence of serotonergic drugs on SB remains unknown, and the interaction between SSRI drugs needs to be improved for an understanding of secondary SB.

In a few case reports, anticonvulsant drugs (eg, gabapentin or tiagabine) have been reported to reduce both primary and secondary self-reported bruxism.[255,259] The efficacy, role, and active mechanism of these drugs in relation to SB remains to be demonstrated.

A placebo-controlled study has reported that a catecholamine-related medication (dopamine, serotonin, adrenaline), with a major action on dopamine, L-dopa (2 doses of 100 mg/night) modestly reduced SB activity by 30%.[123] In a case study with 2 patients, the administration of the dopamine agonist bromocriptine (7.5 mg/night) resulted in a significant reduction of SB activity.[260] However, in a placebo-controlled study, bromocriptine (7.5 mg/night), in combination with domperidone (20 mg/night) for reducing nausea, failed to decrease SB.[122] A recent report presented a case in which a strong dopaminergic agonist, pergolide (0.3–0.5 mg/night) with domperidone, also reduced SB.[261] Subjects were given low doses of these medications to prevent excessive side effects such as nausea, emesis, and dizziness. The efficacy and long-term safety of dopaminergic medications requires further clarification because the side effect ratio prevents their use in most SB patients.

In 2 case reports (see earlier discussion), a reduction in SB activity in an SB patient was noted in response to the administration of the β-adrenergic receptor antagonist propranolol (2 doses of 60 mg/night). The same effect was noted in 2 secondary SB patients with antipsychotic drug exposure (up to 240 mg/d or 20 mg 3 times a day).[124,262] In a randomized experimental trial to further understand SB pathophysiology, propranolol (120 mg/night) failed to reduce SB whereas the α-receptor agonist clonidine (0.3 mg/night) decreased SB by 60%.[125] It is worth noting that clonidine acts mainly at the level of the central nervous and autonomic systems. The use of clonidine in primary SB patients is not

indicated because severe hypotension in the morning was observed in 20% of patients following the administration of an intermediate dose.[125]

Compared with placebo, a proton-pump inhibitor has been reported to decrease RMMA episodes and events with decreased esophageal pH in SB patients (not patients with gastroesophageal reflux) and in controls.[160] Further study is required to assess the efficacy of this drug. The influence of visceral functions in association with autonomic nervous system activity is an area worthy of examination as regards the management of SB.

Botulinum toxin type A (BTX-A) is known to be effective for controlling involuntary orofacial movements and secondary bruxism in patients with movement disorders (eg, cranial dystonia).[263,264] One study reported a decrease in jaw muscle EMG activity during sleep after BTX–A injection.[265] However, the treatment effects of BTX-A have not yet been fully evaluated in a large sample of patients with primary SB using sleep and EMG recordings.

SUMMARY

SB is not as simple a jaw movement as chewing; it is a rhythmic movement with an intense jaw muscle contraction that can damage teeth and trigger pain or headache. When SB is clinically reported by tooth grinding, the final diagnosis is only possible with polygraphic and audio-video recordings in a home or sleep laboratory environment. The occurrence of SB is associated in some subjects with homeostatic sleep regulation (ie, biologic need for sleep over circadian rhythm) and sleep instability (eg, CAP and microarousals). Other modulating factors that need to be recognized are neurochemicals, psychological stress, and oro-esopharyngeal functions (mucosal dryness, breathing). The contribution of child development (associated with high prevalence of tooth grinding) and aging (associated with low prevalence of tooth grinding) remains to be investigated. Concomitant sleep disorders and the use of some medication or drugs should not be overlooked. Although the complex influences of these factors can involve the genesis or exacerbation of SB, there are still discrepancies in the understanding of the relationships between sleep physiology, SB pathophysiology, and orodental consequences. Therefore, a single ideal treatment for SB has yet to be recognized. The clinician's main objective remains the prevention of damage to orofacial structures and associated orofacial sensory complaints. Thus, in managing cases of SB and related consequences, such as tooth damage or pain, and even more so if SB is secondary to medication use or a medical condition, it is necessary for the clinician to plan a multidisciplinary approach based on the best scientific evidence available.

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REFERENCES

1. Thorpy MJ. International classification of sleep disorders: diagnostic and coding manual. Rochester (NY): Minnesota: American Sleep Disorders Association; 1990.
2. International classification of sleep disorders: diagnostic and coding manual. 2nd edition. Westchester (IL): American Academy of Sleep Medicine; 2005.
3. Walters AS. Clinical identification of the simple sleep-related movement disorders. Chest 2007; 131:1260.
4. Walters AS, Lavigne G, Hening W, et al. The scoring of movements in sleep. J Clin Sleep Med 2007;3:155.
5. Lavigne GJ, Manzini C, Kato T. Sleep bruxism. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 946.
6. The glossary of prosthodontic terms. J Prosthet Dent 2005;94:10.
7. De Leeuw R, editor. Orofacial pain: guidelines for assessment, classification, and management. 4th edition. Chicago: Quintessence; 2008.
8. Lavigne GJ, Khoury S, Abe S, et al. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil 2008;35:476.
9. Kato T, Dal-Fabbro C, Lavigne GJ. Current knowledge on awake and sleep bruxism: overview. Alpha Omegan 2003;96:24.
10. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep 1994;17:739.
11. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest 2001;119:53.
12. Laberge L, Tremblay RE, Vitaro F, et al. Development of parasomnias from childhood to early adolescence. Pediatrics 2000;106:67.
13. Ng DK, Kwok KL, Cheung JM, et al. Prevalence of sleep problems in Hong Kong primary school children: a community-based telephone survey. Chest 2005;128:1315.

14. Hicks RA, Lucero-Gorman K, Bautista J, et al. Ethnicity and bruxism. Percept Mot Skills 1999; 88:240.
15. Ahlberg J, Savolainen A, Rantala M, et al. Reported bruxism and biopsychosocial symptoms: a longitudinal study. Community Dent Oral Epidemiol 2004;32:307.
16. Lavigne GL, Lobbezoo F, Rompre PH, et al. Cigarette smoking as a risk factor or an exacerbating factor for restless legs syndrome and sleep bruxism. Sleep 1997;20:290.
17. Hojo A, Haketa T, Baba K, et al. Association between the amount of alcohol intake and masseter muscle activity levels recorded during sleep in healthy young women. Int J Prosthodont 2007;20:251.
18. Winocur E, Gavish A, Voikovitch M, et al. Drugs and bruxism: a critical review. J Orofac Pain 2003;17:99.
19. Manfredini D, Landi N, Fantoni F, et al. Anxiety symptoms in clinically diagnosed bruxers. J Oral Rehabil 2005;32:584.
20. Petit D, Touchette E, Tremblay RE, et al. Dyssomnias and parasomnias in early childhood. Pediatrics 2007;119:e1016.
21. Pingitore G, Chrobak V, Petrie J. The social and psychologic factors of bruxism. J Prosthet Dent 1991;65:443.
22. Suwa S, Takahara M, Shirakawa S, et al. Sleep bruxism and its relationship to sleep habits and lifestyle of elementary school children in Japan. Sleep Biol Rhythms 2009;7:93.
23. Kato T. Sleep bruxism and its relation to obstructive sleep apnea-hypopnea syndrome. Sleep Biol Rhythms 2004;2:1.
24. Kato T, Blanchet PJ, Montplaisir JY, et al. Sleep bruxism and other disorders with orofacial activity during sleep. In: Chokroverty S, Hening W, Walters A, editors. Sleep and movement disorders. Philadelphia: Butterworth Heinemann; 2003. p. 273.
25. Baba K, Haketa T, Sasaki Y, et al. Association between masseter muscle activity levels recorded during sleep and signs and symptoms of temporomandibular disorders in healthy young adults. J Orofac Pain 2005;19:226.
26. Lobbezoo F, Brouwers JE, Cune MS, et al. Dental implants in patients with bruxing habits. J Oral Rehabil 2006;33:152.
27. Tosun T, Karabuda C, Cuhadaroglu C. Evaluation of sleep bruxism by polysomnographic analysis in patients with dental implants. Int J Oral Maxillofac Implants 2003;18:286.
28. Casanova-Rosado JF, Medina-Solis CE, Vallejos-Sanchez AA, et al. Prevalence and associated factors for temporomandibular disorders in a group of Mexican adolescents and youth adults. Clin Oral Investig 2006;10:42.
29. Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain 1997;11:15.
30. Manfredini D, Cantini E, Romagnoli M, et al. Prevalence of bruxism in patients with different research diagnostic criteria for temporomandibular disorders (RDC/TMD) diagnoses. Cranio 2003;21:279.
31. Raphael KG, Marbach JJ, Klausner JJ, et al. Is bruxism severity a predictor of oral splint efficacy in patients with myofascial face pain? J Oral Rehabil 2003;30:17.
32. Smith MT, Wichwire EM, Grace EG, et al. Sleep disorders and their association with laboratory pain sensitivity in temporomandibular disorders. Sleep 2009;32:779.
33. Aromaa M, Sillanpaa ML, Rautava P, et al. Childhood headache at school entry: a controlled clinical study. Neurology 1998;50:1729.
34. Bader GG, Kampe T, Tagdae T, et al. Descriptive physiological data on a sleep bruxism population. Sleep 1997;20:982.
35. Camparis CM, Siqueira JT. Sleep bruxism: clinical aspects and characteristics in patients with and without chronic orofacial pain. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:188.
36. Vendrame M, Kaleyias J, Valencia I, et al. Polysomnographic findings in children with headaches. Pediatr Neurol 2008;39:6.
37. Boutros NN, Montgomery MT, Nishioka G, et al. The effects of severe bruxism on sleep architecture: a preliminary report. Clin Electroencephalogr 1993;24:59.
38. Bliwise DL. Norma aging. In: Kryger MH, Roth T, Dement WC, editors. Principles and practices of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 24.
39. Lavigne GJ, McMillan D, Zucconi M. Pain and sleep. In: Kryger MH, Roth T, Dement WC, editors. Principles and practices of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 1246.
40. Rompre PH, Daigle-Landry D, Guitard F, et al. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res 2007;86:837.
41. Rossetti LM, Pereira de Araujo Cdos R, Rossetti PH, et al. Association between rhythmic masticatory muscle activity during sleep and masticatory myofascial pain: a polysomnographic study. J Orofac Pain 2008;22:190.
42. Johansson A, Haraldson T, Omar R, et al. A system for assessing the severity and progression of occlusal tooth wear. J Oral Rehabil 1993;20:125.
43. Lobbezoo F, Naeije M. A reliability study of clinical tooth wear measurements. J Prosthet Dent 2001; 86:597.
44. Abe S, Yamaguchi T, Rompré PH, et al. Tooth wear in young subjects: a discriminator between

sleep bruxers and controls? Int J Prosthodont 2009;22:342.
45. Baba K, Haketa T, Clark GT, et al. Does tooth wear status predict ongoing sleep bruxism in 30-year-old Japanese subjects? Int J Prosthodont 2004; 17:39.
46. Koyano K, Tsukiyama Y, Ichiki R, et al. Assessment of bruxism in the clinic. J Oral Rehabil 2008;35:495.
47. Ommerborn MA, Schneider C, Giraki M, et al. In vivo evaluation of noncarious cervical lesions in sleep bruxism subjects. J Prosthet Dent 2007;98:150.
48. Rees JS, Jagger DC. Abfraction lesions: myth or reality? J Esthet Restor Dent 2003;15:263.
49. Lynch CD, McConnell RJ. The cracked tooth syndrome. J Can Dent Assoc 2002;68:470.
50. Ratcliff S, Becker IM, Quinn L. Type and incidence of cracks in posterior teeth. J Prosthet Dent 2001; 86:168.
51. Takagi I, Sakurai K. Investigation of the factors related to the formation of the buccal mucosa ridging. J Oral Rehabil 2003;30:565.
52. Kato T, Thie NM, Montplaisir JY, et al. Bruxism and orofacial movements during sleep. Dent Clin North Am 2001;45:657.
53. Okeson JP. Management of temporomandibular disorders and occlusion. 5th edition. St Louis (MO): Mosby; 2003.
54. Chervin RD. Use of clinical tools and tests in sleep medicine. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 602.
55. Egermark I, Carlsson GE, Magnusson T. A 20-year longitudinal study of subjective symptoms of temporomandibular disorders from childhood to adulthood. Acta Odontol Scand 2001;59:40.
56. Lavigne GJ, Guitard F, Rompre PH, et al. Variability in sleep bruxism activity over time. J Sleep Res 2001;10:237.
57. Van Der Zaag J, Lobbezoo F, Visscher CM, et al. Time-variant nature of sleep bruxism outcome variables using ambulatory polysomnography: implications for recognition and therapy evaluation. J Oral Rehabil 2008;35:577.
58. Van't Spijker A, Rodriguez JM, Kreulen CM, et al. Prevalence of tooth wear in adults. Int J Prosthodont 2009;22:35.
59. Thie NM, Kato T, Bader G, et al. The significance of saliva during sleep and the relevance of oromotor movements. Sleep Med Rev 2002;6:213.
60. Ommerborn MA, Giraki M, Schneider C, et al. A new analyzing method for quantification of abrasion on the Bruxcore device for sleep bruxism diagnosis. J Orofac Pain 2005;19:232.
61. Pierce CJ, Gale EN. Methodological considerations concerning the use of Bruxcore Plates to evaluate nocturnal bruxism. J Dent Res 1989;68: 1110.
62. Dao TT, Lund JP, Lavigne GJ. Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. J Orofac Pain 1994;8:350.
63. Svensson P, Jadidi F, Arima T, et al. Relationships between craniofacial pain and bruxism. J Oral Rehabil 2008;35:524.
64. Biondi DM. Headaches and their relationship to sleep. Dent Clin North Am 2001;45:685.
65. Camparis CM, Formigoni G, Teixeira MJ, et al. Sleep bruxism and temporomandibular disorder: clinical and polysomnographic evaluation. Arch Oral Biol 2006;51:721.
66. Arima T, Arendt-Nielsen L, Svensson P. Effect of jaw muscle pain and soreness evoked by capsaicin before sleep on orofacial motor activity during sleep. J Orofac Pain 2001;15:245.
67. Lavigne GJ, Rompre PH, Montplaisir JY, et al. Motor activity in sleep bruxism with concomitant jaw muscle pain. A retrospective pilot study. Eur J Oral Sci 1997;105:92.
68. Glaros AG, Waghela R. Psychophysiological definitions of clenching. Cranio 2006;24:252.
69. Katase-Akiyama S, Kato T, Yamashita S, et al. Specific increase in non-functional masseter bursts in subjects aware of tooth-clenching during wakefulness. J Oral Rehabil 2009;36:93.
70. Carlsson GE, Egermark I, Magnusson T. Predictors of signs and symptoms of temporomandibular disorders: a 20-year follow-up study from childhood to adulthood. Acta Odontol Scand 2002;60: 180.
71. van Selms MK, Lobbezoo F, Visscher CM, et al. Myofascial temporomandibular disorder pain, parafunctions and psychological stress. J Oral Rehabil 2008;35:45.
72. Velly AM, Gornitsky M, Philippe P. A case-control study of temporomandibular disorders: symptomatic disc displacement. J Oral Rehabil 2002;29: 408.
73. Malow BA, Aldrich MS. Polysomnography. In: Chokroverty S, Hening W, Walters A, editors. Sleep and movement disorders. Philadelphia: Butterworth Heinemann; 2003. p. 125.
74. Ikeda T, Nishigawa K, Kondo K, et al. Criteria for the detection of sleep-associated bruxism in humans. J Orofac Pain 1996;10:270.
75. Gallo LM, Lavigne G, Rompré P, et al. Reliability of scoring EMG orofacial events: polysomnography compared with ambulatory recordings. J Sleep Res 1997;6:259.
76. Dutra KM, Pereira FJ Jr, Rompre PH, et al. Orofacial activities in sleep bruxism patients and in normal subjects: a controlled polygraphic and audio-video study. J Oral Rehabil 2008;36:86.
77. Yamaguchi T, Mikami S, Okada K. Validity of a newly developed ultraminiature cordless EMG

measurement system. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:e22.
78. Jadidi F, Castrillon E, Svensson P. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. J Oral Rehabil 2008;35:171.
79. Shochat T, Gavish A, Arons E, et al. Validation of the BiteStrip screener for sleep bruxism. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:e32.
80. Baba K, Clark GT, Watanabe T, et al. Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. J Orofac Pain 2003;17:58.
81. Nagamatsu-Sakaguchi C, Minakuchi H, Clark GT, et al. Relationship between the frequency of sleep bruxism and the prevalence of signs and symptoms of temporomandibular disorders in an adolescent population. Int J Prosthodont 2008;21:292.
82. Doering S, Boeckmann JA, Hugger S, et al. Ambulatory polysomnography for the assessment of sleep bruxism. J Oral Rehabil 2008;35:572.
83. Lavigne GJ, Rompre PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 1996;75:546.
84. Kato T, Montplaisir JY, Blanchet PJ, et al. Idiopathic myoclonus in the oromandibular region during sleep: a possible source of confusion in sleep bruxism diagnosis. Mov Disord 1999;14:865.
85. Kato T, Thie NM, Huynh N, et al. Topical review: sleep bruxism and the role of peripheral sensory influences. J Orofac Pain 2003;17:191.
86. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 2001;28: 1085.
87. Lavigne GJ, Rompre PH, Poirier G, et al. Rhythmic masticatory muscle activity during sleep in humans. J Dent Res 2001;80:443.
88. Sjoholm T, Lehtinen II, Helenius H. Masseter muscle activity in diagnosed sleep bruxists compared with non-symptomatic controls. J Sleep Res 1995;4:48.
89. Lavigne GJ, Huynh N, Kato T, et al. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. Arch Oral Biol 2007;52:381.
90. Miyawaki S, Lavigne GJ, Pierre M, et al. Association between sleep bruxism, swallowing-related laryngeal movement, and sleep positions. Sleep 2003;26:461.
91. Dettmar DM, Shaw RM, Tilley AJ. Tooth wear and bruxism: a sleep laboratory investigation. Aust Dent J 1987;32:421.
92. Huynh N, Kato T, Rompre PH, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. J Sleep Res 2006;15:339.
93. Macaluso GM, Guerra P, Di Giovanni G, et al. Sleep bruxism is a disorder related to periodic arousals during sleep. J Dent Res 1998;77:565.
94. Reding GR, Zepelin H, Robinson JE, et al. Nocturnal teeth-grinding: all-night psychophysiologic studies. J Dent Res 1968;47:786.
95. Satoh T, Harada Y. Electrophysiological study on tooth-grinding during sleep. Electroencephalogr Clin Neurophysiol 1973;35:267.
96. Wieselmann G, Permann R, Korner E, et al. Distribution of muscle activity during sleep in bruxism. Eur Neurol 1986;25(Suppl 2):111.
97. Reding GR, Rubright WC, Rechtschaffen A, et al. Sleep pattern of tooth-grinding: its relationship to dreaming. Science 1964;145:725.
98. Reding GR, Zepelin H, Robinson JEJ, et al. Sleep pattern of bruxism: a revision. In: APSS Meeting, vol. 4, p. 396, 1967.
99. Halasz P, Terzano M, Parrino L, et al. The nature of arousal in sleep. J Sleep Res 2004;13:1.
100. Terzano MG, Parrino L, Boselli M, et al. CAP components and EEG synchronization in the first 3 sleep cycles. Clin Neurophysiol 2000;111:283.
101. Lavigne GJ, Rompre PH, Guitard F, et al. Lower number of K-complexes and K-alphas in sleep bruxism: a controlled quantitative study. Clin Neurophysiol 2002;113:686.
102. EEG arousals: scoring rules and examples: a preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. Sleep 1992;15:173.
103. Kato T, Rompre P, Montplaisir JY, et al. Sleep bruxism: an oromotor activity secondary to microarousal. J Dent Res 1940;80:2001.
104. Kydd WL, Daly C. Duration of nocturnal tooth contacts during bruxing. J Prosthet Dent 1985;53:717.
105. Okeson JP, Phillips BA, Berry DT, et al. Nocturnal bruxing events: a report of normative data and cardiovascular response. J Oral Rehabil 1994;21: 623.
106. Herrera M, Valencia I, Grant M, et al. Bruxism in children: effect on sleep architecture and daytime cognitive performance and behavior. Sleep 2006; 29:1143.
107. Ferri R, Parrino L, Smerieri A, et al. Cyclic alternating pattern and spectral analysis of heart rate variability during normal sleep. J Sleep Res 2000;9:13.
108. Khoury S, Rouleau GA, Rompre PH, et al. A significant increase in breathing amplitude precedes sleep bruxism. Chest 2008;134:332.
109. Chase MH, Morales FR. Control of motoneurons during sleep. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 154.

110. Kato T, Montplaisir JY, Lavigne GJ. Experimentally induced arousals during sleep: a cross-modality matching paradigm. J Sleep Res 2004;13:229.
111. Okura K, Kato T, Montplaisir JY, et al. Quantitative analysis of surface EMG activity of cranial and leg muscles across sleep stages in human. Clin Neurophysiol 2006;117:269.
112. Kato T, Montplaisir JY, Guitard F, et al. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res 2003;82:284.
113. Bader G, Kampe T, Tagdae T. Body movement during sleep in subjects with long-standing bruxing behavior. Int J Prosthodont 2000;13:327.
114. Sjoholm TT, Polo OJ, Alihanka JM. Sleep movements in teethgrinders. J Craniomandib Disord 1992;6:184.
115. Frauscher B, Iranzo A, Hogl B, et al. Quantification of electromyographic activity during REM sleep in multiple muscles in REM sleep behavior disorder. Sleep 2008;31:724.
116. Kato T, Masuda Y, Kanayama H, et al. Muscle activities are differently modulated between masseter and neck muscle during sleep-wake cycles in guinea pigs. Neurosci Res 2007;58:265.
117. Akerstedt T, Billiard M, Bonnet M, et al. Awakening from sleep. Sleep Med Rev 2002;6:267.
118. Grosse P, Khatami R, Salih F, et al. Corticospinal excitability in human sleep as assessed by transcranial magnetic stimulation. Neurology 1988;59: 2002.
119. Terzano MG, Parrino L, Rosa A, et al. CAP and arousals in the structural development of sleep: an integrative perspective. Sleep Med 2002;3:221.
120. Gastaldo E, Quatrale R, Graziani A, et al. The excitability of the trigeminal motor system in sleep bruxism: a transcranial magnetic stimulation and brainstem reflex study. J Orofac Pain 2006;20:145.
121. Lobbezoo F, Soucy JP, Montplaisir JY, et al. Striatal D2 receptor binding in sleep bruxism: a controlled study with iodine-123-iodobenzamide and single-photon-emission computed tomography. J Dent Res 1804;75:1996.
122. Lavigne GJ, Soucy JP, Lobbezoo F, et al. Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. Clin Neuropharmacol 2001;24:145.
123. Lobbezoo F, Lavigne GJ, Tanguay R, et al. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Mov Disord 1997;12:73.
124. Sjoholm TT, Lehtinen I, Piha SJ. The effect of propranolol on sleep bruxism: hypothetical considerations based on a case study. Clin Auton Res 1996;6:37.
125. Huynh N, Lavigne GJ, Lanfranchi PA, et al. The effect of 2 sympatholytic medications—propranolol and clonidine—on sleep bruxism: experimental randomized controlled studies. Sleep 2006;29:307.
126. Lobbezoo F, van Denderen RJ, Verheij JG, et al. Reports of SSRI-associated bruxism in the family physician's office. J Orofac Pain 2001;15:340.
127. Ranjan S, S Chandra P, Prabhu S. Antidepressant-induced bruxism: need for buspirone? Int J Neuropsychopharmacol 2006;9:485.
128. Jones BE. Basic mechanisms of sleep-wake states. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 136.
129. Van cauter E. Endorine physiology. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 266.
130. Hicks RA, Conti PA, Bragg HR. Increases in nocturnal bruxism among college students implicate stress. Med Hypotheses 1990;33:239.
131. Kampe T, Edman G, Bader G, et al. Personality traits in a group of subjects with long-standing bruxing behaviour. J Oral Rehabil 1997;24:588.
132. Kampe T, Tagdae T, Bader G, et al. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. J Oral Rehabil 1997;24:581.
133. Manfredini D, Ciapparelli A, Dell'Osso L, et al. Mood disorders in subjects with bruxing behavior. J Dent 2005;33:485.
134. Restrepo CC, Vasquez LM, Alvarez M, et al. Personality traits and temporomandibular disorders in a group of children with bruxing behaviour. J Oral Rehabil 2008;35:585.
135. Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain 2009;23:153.
136. Major M, Rompre PH, Guitard F, et al. A controlled daytime challenge of motor performance and vigilance in sleep bruxers. J Dent Res 1999;78: 1754.
137. Ahlberg K, Ahlberg J, Kononen M, et al. Reported bruxism and stress experience in media personnel with or without irregular shift work. Acta Odontol Scand 2003;61:315.
138. Schneider C, Schaefer R, Ommerborn MA, et al. Maladaptive coping strategies in patients with bruxism compared to non-bruxing controls. Int J Behav Med 2007;14:257.
139. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. J Behav Med 1980;3:385.
140. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. Adv Neurol 1988;49:329.
141. Makino M, Masaki C, Tomoeda K, et al. The relationship between sleep bruxism behavior and salivary stress biomarker level. Int J Prosthodont 2009; 22:43.

142. Pierce CJ, Chrisman K, Bennett ME, et al. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. J Orofac Pain 1995;9:51.
143. Watanabe T, Ichikawa K, Clark GT. Bruxism levels and daily behaviors: 3 weeks of measurement and correlation. J Orofac Pain 2003;17:65.
144. Seraidarian P, Seraidarian PI, das Neves Cavalcanti B, et al. Urinary levels of catecholamines among individuals with and without sleep bruxism. Sleep Breath 2009;13:85.
145. Vanderas AP, Menenakou M, Kouimtzis T, et al. Urinary catecholamine levels and bruxism in children. J Oral Rehabil 1999;26:103.
146. Clark GT, Rugh JD, Handelman SL. Nocturnal masseter muscle activity and urinary catecholamine levels in bruxers. J Dent Res 1980;59:1571.
147. Ahlberg K, Jahkola A, Savolainen A, et al. Associations of reported bruxism with insomnia and insufficient sleep symptoms among media personnel with or without irregular shift work. Head Face Med 2008;4:4.
148. Okura K, Lavigne GJ, Huynh N, et al. Comparison of sleep variables between chronic widespread musculoskeletal pain, insomnia, periodic leg movements syndrome and control subjects in a clinical sleep medicine practice. Sleep Med 2008;9:352.
149. Yoshihara T, Shigeta K, Hasegawa H, et al. Neuroendocrine responses to psychological stress in patients with myofascial pain. J Orofac Pain 2005; 19:202.
150. Abe K, Shimakawa M. Genetic and developmental aspects of sleeptalking and teeth-grinding. Acta Paedopsychiatr 1966;33:339.
151. Kuch EV, Till MJ, Messer LB. Bruxing and nonbruxing children: a comparison of their personality traits. Pediatr Dent 1979;1:182.
152. Reding GR, Rubright WC, Zimmerman SO. Incidence of bruxism. J Dent Res 1966;45:1198.
153. Hori A. Twin studies on parasomnias. In: Meier-Ewert K, Okawa M, editors. Sleep-wake disorders. New York: Plenum Press; 1998. p. 115.
154. Hublin C, Kaprio J, Partinen M, et al. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998;7:61.
155. Lindqvist B. Bruxism in twins. Acta Odontol Scand 1974;32:177.
156. Carlsson GE, Egermark I, Magnusson T. Predictors of bruxism, other oral parafunctions, and tooth wear over a 20-year follow-up period. J Orofac Pain 2003;17:50.
157. Hublin C, Kaprio J, Partinen M, et al. Parasomnias: co-occurrence and genetics. Psychiatr Genet 2001;11:65.
158. Lichter I, Muir RC. The pattern of swallowing during sleep. Electroencephalogr Clin Neurophysiol 1975; 38:427.
159. Orr WC, Johnson LF, Robinson MG. Effect of sleep on swallowing, esophageal peristalsis, and acid clearance. Gastroenterology 1984;86:814.
160. Miyawaki S, Tanimoto Y, Araki Y, et al. Association between nocturnal bruxism and gastroesophageal reflux. Sleep 2003;26:888.
161. Chen CL, Orr WC. Analysis of 24-hour esophageal pH monitoring: the effect of state of consciousness. Curr Gastroenterol Rep 2008;10:258.
162. Orr WC. Gastrointestinal physiology. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 283.
163. Miyamoto K, Ozbek MM, Lowe AA, et al. Mandibular posture during sleep in healthy adults. Arch Oral Biol 1998;43:269.
164. Schwab RJ, Kuna ST, Remmers JE. Anatomy and physiology of upper airway obstruction. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 983.
165. Simmons J, Prehn R. Airway protection: the missing link between nocturnal bruxism and obstructive sleep apnea. Sleep 2009;32 (abstract supplement): A218, #0668.
166. Landry-Schonbeck A, de Grandmont P, Rompre PH, et al. Effect of an adjustable mandibular advancement appliance on sleep bruxism: a crossover sleep laboratory study. Int J Prosthodont 2009;22:251.
167. Landry ML, Rompre PH, Manzini C, et al. Reduction of sleep bruxism using a mandibular advancement device: an experimental controlled study. Int J Prosthodont 2006;19:549.
168. Ash MM. Paradigmatic shifts in occlusion and temporomandibular disorders. J Oral Rehabil 2001;28:1.
169. Manfredini D, Landi N, Romagnoli M, et al. Psychic and occlusal factors in bruxers. Aust Dent J 2004; 49:84.
170. Tsukiyama Y, Baba K, Clark GT. An evidence-based assessment of occlusal adjustment as a treatment for temporomandibular disorders. J Prosthet Dent 2001;86:57.
171. Graf H. Bruxism. Dent Clin North Am 1969;13:659.
172. Powell RN. Tooth contact during sleep: association with other events. J Dent Res 1965;44:959.
173. Powell RN, Zander HA. The frequency and distribution of tooth contact during sleep. J Dent Res 1965; 44:713.
174. Okeson JP, Phillips BA, Berry DT, et al. Nocturnal bruxing events in healthy geriatric subjects. J Oral Rehabil 1990;17:411.
175. von Gonten AS, Palik JF, Oberlander BA, et al. Nocturnal electromyographic evaluation of masseter muscle activity in the complete denture patients. J Prosthet Dent 1986;56:624.

176. Lobbezoo F, Rompre PH, Soucy JP, et al. Lack of associations between occlusal and cephalometric measures, side imbalance in striatal D2 receptor binding, and sleep-related oromotor activities. J Orofac Pain 2001;15:64.
177. Kato T, Blanchet PJ. Orofacial movement disorders in sleep. In: Lavigne GJ, Cistulli PA, Smith MT, editors. Sleep medicine for dentists: a practical overview. Hanover Park (IL): Quintessence; 2009. p. 101.
178. Gara L, Roberts W. Adverse response to methylphenidate in combination with valproic acid. J Child Adolesc Psychopharmacol 2000;10:39.
179. Mendhekar DN, Andrade C. Bruxism arising during monotherapy with methylphenidate. J Child Adolesc Psychopharmacol 2008;18:537.
180. Mendhekar D, Lohia D. Worsening of bruxism with atomoxetine: a case report. World J Biol Psychiatry 2009. DOI:10.1080/15622970802576488.
181. Lobbezoo F, Van Der Zaag J, Naeije M. Bruxism: its multiple causes and its effects on dental implants—an updated review. J Oral Rehabil 2006;33:293.
182. Bruni O, Ferri R, D'Agostino G, et al. Sleep disturbances in Angelman syndrome: a questionnaire study. Brain Dev 2004;26:233.
183. Olson RE, Laskin DM. Relationship between allergy and bruxism in patients with myofascial pain-dysfunction syndrome. J Am Dent Assoc 1980; 100:209.
184. Manconi M, Zucconi M, Carrot B, et al. Association between bruxism and nocturnal groaning. Mov Disord 2008;23:737.
185. Khatami R, Zutter D, Siegel A, et al. Sleep-wake habits and disorders in a series of 100 adult epilepsy patients—a prospective study. Seizure 2006;15:299.
186. Meletti S, Cantalupo G, Volpi L, et al. Rhythmic teeth grinding induced by temporal lobe seizures. Neurology 2004;62:2306.
187. Ghanizadeh A. Comorbidity of enuresis in children with attention-deficit/hyperactivity disorder. J Atten Disord 2009. DOI:10.1177/1087054709332411.
188. Tani K, Yoshii N, Yoshino I, et al. Electroencephalographic study of parasomnia: sleep-talking, enuresis and bruxism. Physiol Behav 1966;1:241.
189. Huynh N, Guilleminault C. Sleep bruxism in children. In: Lavigne GJ, Cistulli PA, Smith MT, editors. Sleep medicine for dentists: a practical overview. Chicago: Quintessence; 2009.
190. Bracha HS, Ralston TC, Williams AE, et al. The clenching-grinding spectrum and fear circuitry disorders: clinical insights from the neuroscience/paleoanthropology interface. CNS Spectr 2005; 10:311.
191. Ghanizadeh A. ADHD, bruxism and psychiatric disorders: does bruxism increase the chance of a comorbid psychiatric disorder in children with ADHD and their parents? Sleep Breath 2008;12: 375.
192. Lindqvist B, Heijbel J. Bruxism in children with brain damage. Acta Odontol Scand 1974;32:313.
193. Ohayon MM, Caulet M, Priest RG. Violent behavior during sleep. J Clin Psychiatry 1997;58:369.
194. Richmond G, Rugh JD, Dolfi R, et al. Survey of bruxism in an institutionalized mentally retarded population. Am J Ment Defic 1984;88:418.
195. Shur-Fen Gau S. Prevalence of sleep problems and their association with inattention/hyperactivity among children aged 6-15 in Taiwan. J Sleep Res 2006;15:403.
196. Winocur E, Hermesh H, Littner D, et al. Signs of bruxism and temporomandibular disorders among psychiatric patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:60.
197. Okeson JP, Phillips BA, Berry DT, et al. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. J Craniomandib Disord 1991;5:258.
198. Sjoholm TT, Lowe AA, Miyamoto K, et al. Sleep bruxism in patients with sleep-disordered breathing. Arch Oral Biol 2000;45:889.
199. Inoko Y, Shimizu K, Morita O, et al. Relationship between masseter muscle activity and sleep-disordered breathing. Sleep Biol Rhythms 2004;2:67.
200. Montagna P. Physiologic body jerks and movements at sleep onset and during sleep. In: Chokroverty S, Hening W, Walters A, editors. Sleep and movement disorders. Philadelphia: Butterworth Heinemann; 2003. p. 247.
201. Phillips BA, Okeson J, Paesani D, et al. Effect of sleep position on sleep apnea and parafunctional activity. Chest 1986;90:424.
202. Ng DK, Kwok KL, Poon G, et al. Habitual snoring and sleep bruxism in a paediatric outpatient population in Hong Kong. Singapore Med J 2002;43: 554.
203. DiFrancesco RC, Junqueira PA, Trezza PM, et al. Improvement of bruxism after T & A surgery. Int J Pediatr Otorhinolaryngol 2004;68:441.
204. Eftekharian A, Raad N, Gholami-Ghasri N. Bruxism and adenotonsillectomy. Int J Pediatr Otorhinolaryngol 2008;72:509.
205. Grechi TH, Trawitzki LV, de Felicio CM, et al. Bruxism in children with nasal obstruction. Int J Pediatr Otorhinolaryngol 2008;72:391.
206. Restrepo CC, Sforza C, Colombo A, et al. Palate morphology of bruxist children with mixed dentition. A pilot study. J Oral Rehabil 2008;35:353.
207. Hublin C, Kaprio J. Genetic aspects and genetic epidemiology of parasomnias. Sleep Med Rev 2003;7:413.
208. Vetrugno R, Provini F, Plazzi G, et al. Familial nocturnal facio-mandibular myoclonus mimicking sleep bruxism. Neurology 2002;58:644.

209. Loi D, Provini F, Vetrugno R, et al. Sleep-related faciomandibular myoclonus: a sleep-related movement disorder different from bruxism. Mov Disord 1819;22:2007.
210. Huynh NT, Rompre PH, Montplaisir JY, et al. Comparison of various treatments for sleep bruxism using determinants of number needed to treat and effect size. Int J Prosthodont 2006;19: 435.
211. Lobbezoo F, Blanchet PJ, Lavigne GJ. Management of movement disorders related to orofacial pain. In: Sessle B, Lavigne GJ, Lund JP, et al, editors. Orofacial pain: from basic science to clinical management. 2nd edition. Illinois: Quintessence; 2008. p. 211.
212. Lobbezoo F, van der Zaag J, van Selms MK, et al. Principles for the management of bruxism. J Oral Rehabil 2008;35:509.
213. Winocur E. Management of sleep bruxism. In: Lavigne GJ, Cistulli PA, Smith MT, editors. Sleep medicine for dentists: a practical overview. Hanover Park (IL): Quintessence; 2009.
214. Morin CM. Psychological and behavioral treatments for primary insomnia. In: Kryger MH, Roth T, Dement C, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 726.
215. Restrepo CC, Alvarez E, Jaramillo C, et al. Effects of psychological techniques on bruxism in children with primary teeth. J Oral Rehabil 2001; 28:354.
216. Ommerborn MA, Schneider C, Giraki M, et al. Effects of an occlusal splint compared with cognitive-behavioral treatment on sleep bruxism activity. Eur J Oral Sci 2007;115:7.
217. Clarke JH, Reynolds PJ. Suggestive hypnotherapy for nocturnal bruxism: a pilot study. Am J Clin Hypn 1991;33:248.
218. Cassisi JE, McGlynn FD, Belles DR. EMG-activated feedback alarms for the treatment of nocturnal bruxism: current status and future directions. Biofeedback Self Regul 1987;12:13.
219. Pierce CJ, Gale EN. A comparison of different treatments for nocturnal bruxism. J Dent Res 1988;67: 597.
220. Nissani M. Can taste aversion prevent bruxism? Appl Psychophysiol Biofeedback 2000;25:43.
221. Watanabe T, Baba K, Yamagata K, et al. A vibratory stimulation-based inhibition system for nocturnal bruxism: a clinical report. J Prosthet Dent 2001; 85:233.
222. Nishigawa K, Kondo K, Takeuchi H, et al. Contingent electrical lip stimulation for sleep bruxism: a pilot study. J Prosthet Dent 2003;89:412.
223. Dao TT, Lavigne GJ. Oral splints: the crutches for temporomandibular disorders and bruxism? Crit Rev Oral Biol Med 1998;9:345.
224. Kato T. Peripheral sensory influences in sleep bruxism: a physiological interpretation for clinicians. In: Daniel P, editor. Bruxism: theory and practices. Hanover Park (IL): Quintessence; in press.
225. Okeson JP. The effects of hard and soft occlusal splints on nocturnal bruxism. J Am Dent Assoc 1987;114:788.
226. Clark GT, Beemsterboer PL, Solberg WK, et al. Nocturnal electromyographic evaluation of myofascial pain dysfunction in patients undergoing occlusal splint therapy. J Am Dent Assoc 1979;99:607.
227. Dube C, Rompre PH, Manzini C, et al. Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. J Dent Res 2004;83:398.
228. Harada T, Ichiki R, Tsukiyama Y, et al. The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. J Oral Rehabil 2006;33:482.
229. Okkerse W, Brebels A, De Deyn PP, et al. Influence of a bite-plane according to Jeanmonod, on bruxism activity during sleep. J Oral Rehabil 2002;29:980.
230. van der Zaag J, Lobbezoo F, Wicks DJ, et al. Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. J Orofac Pain 2005;19:151.
231. Rugh JD, Graham GS, Smith JC, et al. Effects of canine versus molar occlusal splint guidance on nocturnal bruxism and craniomandibular symptomatology. J Craniomandib Disord 1989;3:203.
232. Rugh JD, Solberg WK. Electromyographic studies of bruxist behavior before and during treatment. J Calif Dent Assoc 1975;3:56.
233. Solberg WK, Clark GT, Rugh JD. Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. J Oral Rehabil 1975;2:215.
234. Macedo CR, Silva AB, Machado MA, et al. Occlusal splints for treating sleep bruxism (tooth grinding). Cochrane Database Syst Rev 2007;4: CD005514.
235. Gagnon Y, Mayer P, Morisson F, et al. Aggravation of respiratory disturbances by the use of an occlusal splint in apneic patients: a pilot study. Int J Prosthodont 2004;17:447.
236. Hachmann A, Martins EA, Araujo FB, et al. Efficacy of the nocturnal bite plate in the control of bruxism for 3 to 5 year old children. J Clin Pediatr Dent 1999;24:9.
237. Jones CM. Chronic headache and nocturnal bruxism in a 5-year-old child treated with an occlusal splint. Int J Paediatr Dent 1993;3:95.
238. Baad-Hansen L, Jadidi F, Castrillon E, et al. Effect of a nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep. J Oral Rehabil 2007;34:105.

239. Magnusson T, Adiels AM, Nilsson HL, et al. Treatment effect on signs and symptoms of temporomandibular disorders—comparison between stabilisation splint and a new type of splint (NTI). A pilot study. Swed Dent J 2004;28:11.
240. Stapelmann H, Türp JC. The NTI-tss device for the therapy of bruxism, temporomandibular disorders, and headache - where do we stand? A qualitative systematic review of the literature. BMC Oral Health 2008;8:22. DOI:10.1186/1472-6831-8-22.
241. Yustin D, Neff P, Rieger MR, et al. Characterization of 86 bruxing patients with long-term study of their management with occlusal devices and other forms of therapy. J Orofac Pain 1993;7:54.
242. Ash MMJ. Philosophy of occlusion: past and present. Dent Clin North Am 1995;39:233.
243. De Boever JA, Carlsson GE, Klineberg IJ. Need for occlusal therapy and prosthodontic treatment in the management of temporomandibular disorders. Part II: tooth loss and prosthodontic treatment. J Oral Rehabil 2000;27:647.
244. Chasins AI. Methocarbamol (Robaxin) as an adjunct in the treatment of bruxism. J Dent Med 1959;14:166.
245. Montgomery MT, Nishioka GJ, Rugh JD, et al. Effect of diazepam on nocturnal masticatory muscle activity (abstract). J Dent Res 1986;65:96.
246. DeNucci DJ, Sobiski C, Dionne RA. Triazolam improves sleep but fails to alter pain in TMD patients. J Orofac Pain 1998;12:116.
247. Saletu A, Parapatics S, Anderer P, et al. Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. Eur Arch Psychiatry Clin Neurosci 2009. DOI:10.1007/s00406-009-0034-0.
248. Saletu A, Parapatics S, Saletu B, et al. On the pharmacotherapy of sleep bruxism: placebo-controlled polysomnographic and psychometric studies with clonazepam. Neuropsychobiology 2005;51:214.
249. Schenck CH, Mahowald MW. Long-term, nightly benzodiazepine treatment of injurious parasomnias and other disorders of disrupted nocturnal sleep in 170 adults. Am J Med 1996;100:333.
250. Etzel KR, Stockstill JW, Rugh JD, et al. Tryptophan supplementation for nocturnal bruxism: report of negative results. J Craniomandib Disord 1991;5:115.
251. Mohamed SE, Christensen LV, Penchas J. A randomized double-blind clinical trial of the effect of amitriptyline on nocturnal masseteric motor activity (sleep bruxism). Cranio 1997;15:326.
252. Raigrodski AJ, Mohamed SE, Gardiner DM. The effect of amitriptyline on pain intensity and perception of stress in bruxers. J Prosthodont 2001;10:73.
253. Alonso-Navarro H, Martin-Prieto M, Ruiz-Ezquerro JJ, et al. Bruxism possibly induced by venlafaxine. Clin Neuropharmacol 2009;32:111.
254. Bostwick JM, Jaffee MS. Buspirone as an antidote to SSRI-induced bruxism in 4 cases. J Clin Psychiatry 1999;60:857.
255. Brown ES, Hong SC. Antidepressant-induced bruxism successfully treated with gabapentin. J Am Dent Assoc 1999;130:1467.
256. Ellison JM, Stanziani P. SSRI-associated nocturnal bruxism in four patients. J Clin Psychiatry 1993; 54:432.
257. Romanelli F, Adler DA, Bungay KM. Possible paroxetine-induced bruxism. Ann Pharmacother 1996; 30:1246.
258. Stein DJ, Van Greunen G, Niehaus D. Can bruxism respond to serotonin reuptake inhibitors? J Clin Psychiatry 1998;59:133.
259. Kast RE. Tiagabine may reduce bruxism and associated temporomandibular joint pain. Anesth Prog 2005;52:102.
260. Lobbezoo F, Soucy JP, Hartman NG, et al. Effects of the D2 receptor agonist bromocriptine on sleep bruxism: report of two single-patient clinical trials. J Dent Res 1997;76:1610.
261. Van der Zaag J, Lobbezoo F, Van der Avoort PG, et al. Effects of pergolide on severe sleep bruxism in a patient experiencing oral implant failure. J Oral Rehabil 2007;34:317.
262. Amir I, Hermesh H, Gavish A. Bruxism secondary to antipsychotic drug exposure: a positive response to propranolol. Clin Neuropharmacol 1997;20:86.
263. Tan EK, Jankovic J. Treating severe bruxism with botulinum toxin. J Am Dent Assoc 2000;131: 211.
264. Tan EK, Jankovic J, Ondo W. Bruxism in Huntington's disease. Mov Disord 2000;15:171.
265. Lee SJ, McCall WD Jr, Kim YK, et al. Effect of botulinum toxin injection on nocturnal bruxism: a randomized controlled trial. Am J Phys Med Rehabil 2009. DOI:10.1097/PHM.0b013e3181bc0c78.