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.\(^5,8,9\)
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).

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
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. 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. Although tooth wear is frequent in patients with SB, 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. 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. The clinician needs to recognize that more cracks and failure lines may be present in the restored teeth of SB patients. 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.

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. 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. 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.

### RECOGNITION AND DIAGNOSIS

#### Clinical Evaluation

**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**
- 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)

**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)
SB can be clinically recognized by interview and orofacial examination, and confirmed by electrophysiological recordings (eg, polysomnography) in the sleep laboratory or at home.54,52,53 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 orofacial 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, fluctuations 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.52 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.
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

Table 1
Criteria for diagnosis of sleep bruxism

<table>
<thead>
<tr>
<th>Ambulatory recording</th>
<th>Laboratory polysomnography (audio-video plus EMG from masseter or temporalis)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acquisition</strong></td>
<td>128 Hz (minimum) with audio-video recordings</td>
</tr>
<tr>
<td><strong>EMG bursts</strong></td>
<td>- Amplitude: &gt;10%–20% of MVC</td>
</tr>
<tr>
<td></td>
<td>- Duration: Phasic: 0.25–2 s Tonic: &gt; 2 s</td>
</tr>
<tr>
<td></td>
<td>- Interval: &lt;3 s</td>
</tr>
<tr>
<td></td>
<td>Phasic (rhythmic): &gt;2 phasic bursts</td>
</tr>
<tr>
<td></td>
<td>Tonic (sustained): tonic burst</td>
</tr>
<tr>
<td></td>
<td>Mixed: both phasic and tonic bursts</td>
</tr>
</tbody>
</table>

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

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. 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.

**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. 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. 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. 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. 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.

![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.](image)
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. 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%. 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.

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. 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). 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. 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. In SB patients, the amplitude of masseter EMG bursts is frequently as high as 30% to 40% in comparison with controls. These observations suggest that RMMA in SB patients represents an extreme manifestation of a natural, physiologic oromotor activity. 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. Isolated or repetitive myoclonic masseter bursts can be concomitantly observed in SB patients and normal subjects.

Up to 85% of SB episodes are found to occur in light non–rapid eye movement (NREM) sleep (stages 1 and 2). 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. 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). 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). It is known that the ascending period is correlated with an increase in sympathetic tone and in arousal activity. 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%). 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-alsphas, respectively). Sleep spindles were not associated with RMMA, and their frequency does not differ between SB patients and normal subjects. 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. 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. Most SB episodes result in sleep stage shifts. Microarousal and CAP phase A3 predominantly occur in the ascending phase of the sleep cycle in association with an increase in 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. Mean heart rate subsequently starts to increase around 10 seconds before the episodes. Next, a significant increase in heart rate occurs 1 cardiac cycle before an RMMA episode. At the onset of RMMA, an increase in suprahypoid muscle activity and a major breathing effort precedes rhythmic jaw-closing muscle activation by 0.8 second. A clear sequence was found in 80%-90% of RMMA episodes in both SB patients and normal subjects. 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.

**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. The sleep stage dependent changes in muscle tone have been reported to differ between jaw and leg muscles in humans. In masticatory muscles (e.g., masseter, suprahyoid), decrease in muscle tone does not differ between NREM sleep stages. During REM sleep, masticatory muscle tone becomes minimal but does not disappear completely (e.g., 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. 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. Nonetheless, RMMA is triggered by arousal response 7 times more frequently in SB patients than normals, and 86% of induced RMMAs involved teeth grinding. 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
Several studies have suggested that the degree of motor suppression or activation during sleep might differ between muscles (e.g., jaw and limb muscles). In addition, body movements are most likely to occur in response to the higher level of arousal response (e.g., awakening). Because arousal responsiveness is associated with an intrinsic difference in the recruitment patterns of autonomic, cortical, and motor activations, 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.

**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). It has been suggested that catecholamines such as dopamine, noradrenaline, and serotonin are involved in SB pathophysiology.

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. However, the overall density of the striatal dopamine receptors was found to be...
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

However, the evidence is not strong.135 Several aggressive, and hyperactive.11,12,19,21,22,130–134 awareness of tooth grinding are more anxious, some 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 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.

**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 neurochemicals and endocrine systems during sleep and psychophysiological 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.

**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. Nonetheless, a cohort study has found that self-reported tooth grinding can fluctuate over 20 years from childhood to adulthood. 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. 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. 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). 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. Swallowing, therefore, plays a protective function during sleep, probably in association with saliva. Swallowing occurs predominantly in light NREM sleep in relation to arousals. Swallowing was found to occur with approximately 60% of RMMA episodes in SB patients and normal subjects. 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. In children, no correlation was found between SB episodes and esophageal pH. The interaction between factors such as swallowing, esophageal pH, microarousals, and saliva needs to be studied in association with autonomic and gastroenteric systems that are linked to sleep and visceral functions.

During sleep, the jaw is usually open for 90% of total sleeping time because oropharyngeal muscle tone decreases. The mandible and tongue collapse into the pharynx, which results in a narrowing of the upper airway during sleep. 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. Of note, 75% of RMMA episodes were found to occur in a supine position. A recent study has shown that respiratory activity shows a simultaneous and significant increase on the activation of the suprahypophysial muscles when RMMA episodes occur (see Fig. 3). 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 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 (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. 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. In healthy people, an average time for tooth contacts, including meals, is 17.5 minutes per day. Tooth contact is usually absent during sleep without motor activity, whereas it can occur in association with arousal, swallowing, and motor activity. 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. In addition, edentulous patients exhibit RMMA when they sleep without their dentures. Moreover, no correlation between dental morphology (eg, dental arch, occlusion) and SB episodes has been found in adult SB patients assessed by polysomnography.

**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). Evidence of iatrogenic and secondary SB is scarce because
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.\textsuperscript{133,190–196}

Sleep bruxism has been reported to occur in several sleep disorders.\textsuperscript{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).\textsuperscript{17} 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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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).\textsuperscript{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. It has also been suggested that upper airway and face morphology contribute to the SB seen in pediatric patients. 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.

Parents more frequently report tooth grinding in children with common pediatric parasomnias (eg, sleep talking, sleepwalking, enuresis, night terrors) than in children without. Familial predisposition and correlation with anxiety and stress have also been reported for these parasomnias. 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. Most SB episodes are associated with leg and body movements whereas periodic limb movement disorder is found in few SB patients.

Tooth grinding was reported in only 10% of patients with restless legs syndrome. Oromandibular myoclonus (OMM) is characterized by repetitive or isolated tappinglike jaw movements. Familial patterns can be traced for OMM. Approximately 10% of SB patients can exhibit OMM, although OMM is a different entity from SB. 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 orodontal 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. 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.

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). 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. 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. 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. 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.\textsuperscript{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.\textsuperscript{78} 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.\textsuperscript{222} Another study used non-noxious electrical stimulation on the skin of forehead.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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).\textsuperscript{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).\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{223,224,234} This concept is appropriate because the genesis of SB is regulated by the central nervous system.\textsuperscript{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.\textsuperscript{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>
<thead>
<tr>
<th>Strategies</th>
<th>Effects (evidence): comments</th>
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<tbody>
<tr>
<td><strong>Orodental strategies</strong></td>
<td></td>
</tr>
<tr>
<td>Occlusal splint</td>
<td>+ 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</td>
</tr>
<tr>
<td>Mouth guard</td>
<td>+ or –: Short term; may increase EMG activity</td>
</tr>
<tr>
<td>NTI splint</td>
<td>+ (B): Short term data only; may change occlusion if used for a long term</td>
</tr>
<tr>
<td>Mandibular advancement appliance (MAA)</td>
<td>++ (B): Short term data only; teeth or jaw pain if not well titrated</td>
</tr>
<tr>
<td>Occlusal therapy</td>
<td>Questionable/Low level of evidence as a universal therapy; not reversible</td>
</tr>
<tr>
<td><strong>Behavioral strategies</strong></td>
<td></td>
</tr>
<tr>
<td>Management of life style, stress, and sleep hygiene</td>
<td>+ or – (B): Lack of strong evidence/Expected to reduce SB: may help if combined with other strategies. Coping style of SB patients is considered</td>
</tr>
<tr>
<td>Biofeedback</td>
<td>+ (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)</td>
</tr>
<tr>
<td><strong>Pharmacologic strategies</strong></td>
<td></td>
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<tr>
<td>Anxiolytic</td>
<td>Empiric data only</td>
</tr>
<tr>
<td>Diazepam</td>
<td>+ or – (B):</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>+ (A): Risk of dependence; not for regular use—short term (1–3 nights per week)</td>
</tr>
<tr>
<td>Muscle relaxant</td>
<td></td>
</tr>
<tr>
<td>Methocarbamol</td>
<td>+ (B) Risk of dizziness and sleepiness</td>
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<tr>
<td><strong>Dopaminergic</strong></td>
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<tr>
<td>L-Dopa</td>
<td>+ (A)</td>
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<tr>
<td>Bromocriptine</td>
<td>– (A)</td>
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<tr>
<td>Pergolide</td>
<td>+ (one case so far)</td>
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</table>
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.

<table>
<thead>
<tr>
<th>Cardioactive</th>
<th>Clonidine</th>
<th>Propranolol</th>
<th>Amitriptyline</th>
<th>Buspirone</th>
<th>Rabeprazole</th>
<th>Botulinum toxin</th>
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<tbody>
<tr>
<td>(A): Risk of severe hypotension in the morning if given to normotensive subjects</td>
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<td>(A): Risk of severe hypotension in the morning if given to normotensive subjects</td>
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<tr>
<td>Little evidence available, small sample size, controlled report</td>
<td>Little evidence available, small sample size, controlled report</td>
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<td>Little evidence available, small sample size, controlled report</td>
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</table>

(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.
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. 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. Thus, whereas occlusal therapy is indicated for the restoration of orofacial 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.

**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. Benzodiazepines (diazepam; 5 or 10 mg/night) at bedtime have been reported to reduce SB. Compared with placebo, triazolam improved sleep but did not alter jaw-closing muscle activity during sleep in patients with orofacial pain. In a single-blind, nonrandomized study, the acute effects 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. 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. SSRI antidepressants should be avoided for SB management because several case reports have indicated that they may induce secondary SB. Nonetheless, some cases with SSRI-induced SB may be resolved by a different type of SSRI drug (buspirone). 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. Several drugs acting on the central nervous system have been suggested to reduce SB. Benzodiazepines (diazepam; 5 or 10 mg/night) at bedtime decreased SB by approximately 30%. 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. 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. A recent report presented a case in which a strong dopaminergic agonist, pergolide (0.3–0.5 mg/night) with domperidone, also reduced SB. 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). 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%. 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.\textsuperscript{125}

Compared with placebo, a proton-pump inhibitor has been reported to decrease RMMA episodes and with decreased esophageal pH in SB patients (not patients with gastroesophageal reflux) and in controls.\textsuperscript{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).\textsuperscript{263,264} One study reported a decrease in jaw muscle EMG activity during sleep after BTX–A injection.\textsuperscript{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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