Sleep Bruxism: A Comprehensive Overview for the Dental Clinician Interested in Sleep Medicine

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KEYWORDS

• Sleep bruxism • Tooth grinding • Tooth clenching
• Sleep arousal • Sleep-disordered breathing • Headache
• Temporomandibular disorders

DEFINITION AND CLASSIFICATION OF SLEEP BRUXISM

In dentistry, bruxism is traditionally considered an oral parafunction characterized by involuntary grinding and clenching of the teeth.[1,2] Although this definition describes the main characteristics of the disorder, it lacks a substantial and important distinction between the wake and sleep states in which this oral parafunction may occur. In fact, a wake-time habit of clenching, grinding, or gnashing the teeth seems to be a different nosologic entity, probably with a different cause and pathophysiology, and it should be distinguished from bruxism during sleep.

According to the American Academy of Sleep Medicine (AASM) (*International Classification of Sleep Disorders, Second Edition* [ICSD-II]),[3] sleep bruxism (SB) is

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classified as a sleep-related movement disorder. The characteristic electromyography (EMG) pattern of SB is found in repetitive and recurrent episodes of rhythmic masticatory muscle activity (RMMA) of the masseter and temporalis muscles that are usually associated with sleep arousals.[3,4] RMMA shows a frequency of 1 Hz and typically occurs cyclically during sleep (**Fig. 1**).[4] RMMA episodes are observed in 60% of the general adult population as physiologic activity of the jaw muscles during sleep.[5,6] Many other forms of masticatory and facial muscle activity are also observed during sleep, such as swallowing, coughing, sleep talking, smiling, lip sucking, jaw movements, and myoclonus.[4,7] These orofacial activities account for approximately 85% of EMG events scored on the masseter and temporalis muscles in control subjects and 30% in patients with SB.[5,8–10] In fact, RMMA frequency is 3 times higher in patients with SB than in controls and is typically associated with tooth-grinding sounds (in 45% of cases), as reported by patients, bed partners, parents, or siblings.

SB may be an extreme manifestation of a physiologic orofacial motor behavior during sleep (RMMA and chewinglike activity) whereby certain factors increase its occurrence until it falls into the pathologic range of jaw-muscle activity.[11,12] Therefore, SB refers to the sleep motor disorder, whereas RMMA is the characteristic EMG pattern that is scored during sleep to make a polysomnographic diagnosis of SB.

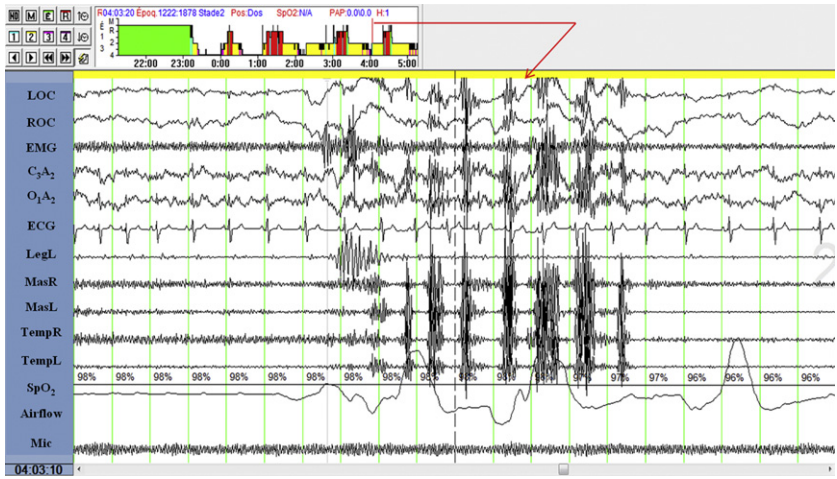

Fig. 1. Hypnogram and polysomnographic tracing showing an episode of RMMA during sleep. The full-night hypnogram (graph in the upper left) represents sleep stage distribution in non-REM sleep 1, 2, 3, 4, and REM sleep, whereas the 20-second polysomnographic page shows a clear example of RMMA during sleep. The patient is in non-REM sleep stage 2. RMMA is defined when at least 3 consecutive EMG bursts (frequency 1 Hz) lasting greater than or equal to 0.25 seconds are scored on the masseter and temporalis channels. Corresponding with the RMMA episode, note the increased frequency in cortical activity (EEG central [C_3A_2] and occipital [O_1A_2] derivations), increased heart rate (on the ECG channel), and increased amplitude of respiratory airflow (naso-cannula). Immediately before the RMMA onset, a leg movement event is also seen on the EMG channel of the tibialis muscle. Airflow, naso-cannula airflow; C_3A_2, the central derivation of the electroencephalogram; ECG, electrocardiogram; EMG, electromyographic activity of the suprahyoid muscle; LOC, left electrooculogram; LegL, EMG of the left tibialis muscle; MasR and MasL, EMG of the right and left masseter muscles; Mic, microphone; O_1A_2, the occipital derivation of the EEG; ROC, right electrooculogram; SpO_2, oxygen saturation level (expressed as %); TempR and TempL, EMG of the right and left temporalis muscles.

ASSESSMENT AND DIAGNOSIS OF SB

The assessment and diagnosis of SB are often challenging. Generally, the assessment is based on reports of tooth-grinding sounds during sleep and the presence of clinical signs and symptoms.[3] However, only an EMG recording of the masticatory muscles can confirm the SB diagnosis. Several portable diagnostic tools have been developed to record masseter or temporalis EMG activity during sleep to avoid using the more sophisticated but highly cost- and time-consuming polysomnography (PSG). However, the reliability of most portable devices has not yet been validated, and their use may be considered only as support in a clinical assessment of SB. In fact, the SB diagnosis is usually clinical, although the gold standard remains a full-night PSG with audio-video recording (**Table 1**). The future direction for SB assessment would be to develop a handy tool that can directly, reliably, and rapidly measure ongoing bruxism activity and that can be used in both clinical (for diagnosis, treatment outcome evaluation, and follow-up) and research settings.

Clinical Diagnosis of SB

The clinical diagnosis of SB should be based on the international diagnostic criteria proposed by the AASM (**Box 1**).[3,13] Grinding sounds caused by tooth contacts are the pathognomonic sign of SB and they are usually reported by patients, bed partners, siblings, or parents. However, not all RMMA episodes are accompanied by tooth grinding and many patients or family members may not be aware of this.

A clinical examination of the oral cavity allows identifying signs and symptoms that are markers of tooth-grinding activity and a clenching habit. These signs and symptoms include hypertrophy of the masseter and temporalis muscles, tongue indentation, tooth wear, jaw muscle tenderness or pain on digital palpation, and reports of morning headache.[4,14] However, none of these signs and symptoms constitutes direct proof of current SB activity. For example, although tooth wear is widely reported in the literature as the classic dental sign of bruxism (both awake and during sleep), it may be related to many other factors that can induce attrition and erosion on dental surfaces (eg, age, occlusal conditions, enamel characteristics, diet, carbonated drinks, medications, gastroesophageal reflux, and alimentary disorders).[4,14–18] Moreover, it was recently demonstrated that tooth wear cannot be used as an absolute criterion to assess SB severity: no difference in tooth wear grade was found between low and high frequency of muscle contractions in young adults with SB.[16]

During the clinical examination, dental clinicians can also identify early risk factors for SB and other sleep or medical disorders (eg, sleep-disordered breathing) and promote further investigations when necessary. In particular, the risk of having or developing sleep-disordered breathing (SDB) increases with retrognathia, micrognathia, macroglossia, adenotonsillar hypertrophy, and a Mallampati score of III and IV.[19] The Mallampati score qualifies oropharyngeal obstruction, with I standing for no obstruction (tonsils, pillars, and soft palate are clearly visible) and IV for high obstruction (only the hard palate is visible).[20] In addition, clinicians can directly observe breathing habits (mouth breathing vs nasal breathing), behavioral attitudes (agitation, anxiety), and a tendency to fall asleep. Although it remains under investigation, some of these factors have been associated with an increased risk for both SB and SDB.[21]

Appropriate questionnaires can also be used to investigate general health, quality of life, pain, headache, sleep quality, and sleepiness. Some questionnaires have been validated for both clinical and research purposes (eg, the Pittsburg Sleep Quality Index and the Epworth Sleepiness Scale). Questionnaire assessments may give the clinician

Table 1
Methods for assessing SB (order of increasing reliability)

Method	Notes
Patient history	Many patients may not be aware of their tooth-grinding habit during sleep. It is more reliable if the bed partner, parents, or siblings report current tooth-grinding sounds during sleep
Clinical assessment	It is used to assess the clinical signs and symptoms that suggest SB (eg, tooth wear; see **Box 1**) and the presence of potential risk factors for other comorbidities (eg, enlarged tonsils, skeletal class II, and Mallampati score III or IV for the risk of concomitant SDB)
Questionnaires	It is used to investigate patients' general and oral health, sleep quality, sleep habits, oral parafunctions, presence and characteristics of pain, headache, fatigue, depression, anxiety and stress, and comorbidities.
Ambulatory EMG monitoring	It allows recording EMG activity during sleep from the temporalis or masseter muscles, depending on the device used. However, there is very low specificity and sensitivity in distinguishing actual RMMA episodes from the many other orofacial and motor activities that occur during sleep. Furthermore, there is no monitoring on awakening from sleep, arousal, sleep staging, or other sleep variables. This tool could be valuable in the clinical assessment of SB and in large-sample studies (eg, general population epidemiologic studies)
Ambulatory PSG recording (type II, III, and IV)	It is usually performed at patients' homes. Normally, there is no audio-video monitoring; specificity and sensitivity in detecting RMMA depends on the device used, and more particularly, on the number of variables monitored (EEG, EOG, ECG, EMG, and respiratory channels). This method may be used for scoring sleep stages, sleep arousals, leg movements, and EMG activity, and for monitoring breathing
Full audio-video PSG recording (type I)	It remains the gold standard for the diagnosis of SB and the assessment of comorbidity with other sleep disorders (eg, SDB, PLMS, RLS, RBD, parasomnias). Normally, it allows full-night monitoring of EEG, EOG, EMG, ECG, leg movements, respiratory effort, airflow, and oxygen saturation. Concomitant audio-video recording increases the specificity and sensitivity in RMMA detection and scoring by distinguishing between RMMA episodes and orofacial (eg, swallowing, coughing, sleep talking) and other muscular activities (eg, head movements, eye blinking) that occur during sleep

Abbreviations: ECG, electrocardiogram; EEG, electroencephalogram; EOG, elecrooculogram; PLMS, periodic limb movement during sleep; RBD, REM sleep behavior disorder; RLS, restless leg syndrome; SDB, sleep-disordered breathing.

Data from Lavigne G, Manzini C, Huynh NT. Sleep Bruxism. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 5th edition. St Louis (MO): Elsevier Saunders; 2011. p. 1129–39; and Hirshkowitz M, Kryger MH. Monitoring techniques for evaluating suspected sleep-disordered breathing. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 5th edition. St Louis (MO): Elsevier Saunders; 2011. p. 1610–23.

an indication of the risk of comorbidity between SB and other, more severe sleep disorders, such as SDB or restless leg syndrome (RLS) (see **Table 1**).

Ambulatory Assessment of SB

Several portable EMG monitoring systems have been developed to assess SB activity. They differ in degree of complexity, ranging from miniature self-contained EMG detectors to ambulatory PSG systems (type II, III, and IV),[22] which allow monitoring only a limited number of channels (see **Table 1**). These devices enable multiple-night

Box 1
AASM clinical diagnostic criteria for SB

Patient history

- Recent patient, parent, or sibling report of tooth-grinding sounds occurring during sleep for at least 3 to 5 nights per week in the last 3 to 6 months

Clinical evaluation[a]

- Abnormal tooth wear
- Hypertrophy of the masseter muscles on voluntary forceful clenching
- Discomfort, fatigue, or pain in the jaw muscles (and transient, morning jaw-muscle pain and headache)

Jaw-muscle activity cannot be better explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder.

[a] None of these signs and symptoms constitutes direct proof of current SB activity. Full-night PSG with audio-video recording remains the gold standard for SB diagnosis.

Data from International classification of sleep disorders, 2nd ed.: Diagnosis and coding manual. (ICSD-2). Westchester, Illinois.: American Academy of Sleep Medicine (AASM) eds.; 2005. Section on Sleep Related Bruxism. p.189–92; and Lavigne G, Manzini C, Huynh NT. Sleep Bruxism. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 5th edition. St Louis (MO): Elsevier Saunders; 2011. p. 1129–39.

recordings in patients' homes at minimal expense and could be useful research tools in large sample studies. However, the lack of standardized scoring criteria and evidence-based validity limit their application to both clinical and research settings.

Because automatic EMG detectors and analyzers usually use a unique algorithm for RMMA activity scoring, their validity remains to be demonstrated. Conversely, ambulatory PSG recordings provide very good quality EMG signals and, depending on their complexity, they can usually assess other sleep parameters, such as sleep electroencephalogram (EEG) (essential for sleep staging) or respiratory variables. In addition, on the masseter or temporalis EMG channels, RMMA episodes can be distinguished as phasic, tonic, or mixed. Furthermore, episode and burst frequency and muscular strength can be calculated (**Box 2**).[4] However, ambulatory PSG is usually performed in the patients' homes without audio-video monitoring. This situation may lead to the overestimation of RMMA episodes because of confounding and non-SB–specific motor activities during sleep. The authors are currently validating RMMA scoring criteria on ambulatory PSG recordings and have observed a modest concordance rate between RMMA scored with and without video on the same night (Carra, unpublished data, 2012). Although preliminary, this finding suggests that, in the absence of audio-video recording, more rigorous criteria should be applied to the clinical assessment and EMG scoring of SB-related activity.

Polysomnographic Diagnosis of SB

PSG for SB is mainly used for research purposes (see **Table 1**). The research diagnostic criteria have been developed from PSG with audio-video recordings performed in a hospital setting with a sleep technician attending full-night monitoring.[9,13] This PSG (referred to as type I)[22] allows assessing several sleep physiologic parameters (eg, EEG, electrooculogram, electromyogram, electrocardiogram, airflow, respiratory effort, oxygen saturation), whereas audio-video recording enables documenting tooth-grinding sounds and distinguishing between RMMA and orofacial (eg,

Box 2
Polysomnographic research diagnostic criteria for SB for scoring RMMA episodes

Mean EMG amplitude: at least 10% of maximum voluntary clenching activity

Types of RMMA episodes

- PHASIC: at least 3 EMG bursts lasting ≥0.25 seconds and <2 seconds
- TONIC: 1 EMG burst lasting >2 seconds
- MIXED: phasic and tonic bursts
- EMG bursts must be separated by <2 seconds to be considered part of the same episode.

SB diagnosis can be made based on[a]

- The RMMA INDEX: number of RMMA episodes per hour of sleep
- The BURST INDEX: number of EMG bursts per hour of sleep
- The BRUXISM TIME INDEX (%): total time spent bruxing/total sleep time × 100
- TOOTH-GRINDING SOUNDS: at least 1 RMMA episode with tooth-grinding sounds

Positive SB diagnosis (based on the frequency of EMG episodes with positive tooth-grinding history or confirmation in a sleep laboratory)[a]

- LOW FREQUENCY: when the RMMA index ≥2 and <4
- HIGH FREQUENCY: when the RMMA index is ≥4 or the burst index ≥25

[a] Best level of reliability when performing audio-video PSG recordings and the presence of at least 2 RMMA episodes associated with tooth-grinding sounds.
Data from Refs.[3,4,9,23–26]

swallowing) and other muscular activity (eg, head movements) during sleep. The validated criteria for a sleep laboratory diagnosis of SB showed 72% sensitivity and 94% specificity.[9] Based on the RMMA index (number of episodes per hour of sleep), SB is diagnosed when RMMA episodes are greater than or equal to 2 (low-frequency SB, mild bruxism) or RMMA episodes are greater than or equal to 4 (high-frequency SB, severe bruxism) (see **Box 2**).[4,9,27]

PSG recordings are not usually indicated for patients who report SB only. However, the clinician should refer patients to a sleep physician for further investigation and diagnosis if other sleep disorders are suspected (eg, sleep apnea, sleep-related epilepsy, rapid eye movement [REM] sleep behavior disorder, periodic limb movement, or other neurologic disorder).

EPIDEMIOLOGY OF SB

In large population-based studies, it is difficult to assess SB by objective measures, such as PSG recordings. The epidemiology of SB is, therefore, largely determined by questionnaires, self-reports, or clinical findings (eg, tooth wear).

SB is reported by 8% of the general adult population.[28,29] It typically peaks during childhood (with prevalence approaching 40% in children aged less than 11 years)[30–36] and tends to decrease after adulthood. No gender difference has been observed.[28,29,34,37] SB is a common sleep disorder. However, the wide prevalence range reported in the literature is most probably because many studies failed to distinguish between wake-time and sleep-related bruxism or to assess the presence of medical comorbidities that may influence its occurrence. Indeed, SB is frequently concomitant (approximately one-third of patients) with wake-time bruxism, which is

characterized mainly by a tooth-clenching habit.[38] Wake-time bruxism tends to increase with age, with an estimated prevalence of 12% in children[21,32] and more than 20% in adults.[32,39–41]

CAUSE AND PATHOGENESIS OF SB

The exact cause and pathophysiology of SB are still unknown.[42] The putative etiologic mechanisms for SB genesis include sleep arousal, autonomic sympathetic-cardiac activation, genetic predisposition, neurochemicals, psychosocial components, exogenous factors, and comorbidities (**Table 2**).

Masticatory muscle movements during sleep (RMMA) are probably different from chewing activity while awake. In fact, SB is characterized by rhythmic motor activity

Table 2
Cause and pathophysiology of SB

Putative Etiologic Factors and Mechanisms	Evidence[a]
Sleep arousal More than 80% of RMMA episodes occur in association with sleep arousal. However, sleep arousal is considered the permissive window that facilitates RMMA occurrence during sleep rather than a trigger or cause of SB	+++
Autonomic sympathetic cardiac activity An increase in sympathetic cardiac activity precedes the onset of most RMMA episodes. This increase is also followed by an increase in heart rate and blood pressure immediately before the muscular activity of the jaw opening and closing muscles	+++
Neurochemicals The potential role of catecholamines (adrenaline, noradrenalin, and dopamine); Patients with SB seem to have higher urinary levels of catecholamines. The putative role of other neurochemicals includes: gamma-aminobutyric acid, orexin, serotonin, and acetylcholine (all involved in the genesis and maintenance of wake and sleep; as yet unknown roles)	+
Genetic and familial predisposition In more than 80% of cases, SB persists from childhood to adulthood. Higher concordance in monozygotic than dizygotic twins. Approximately one-third of patients with SB have a direct family member with a positive tooth grinding history	+
Psychosocial factors Anxiety and stress are risk factors for SB. Patients with SB seem to have maladaptive coping strategies and a more task-oriented personality than patients without SB.	++
Exogenous factors Alcohol, caffeine, cigarette smoking, illicit drug use (eg, cocaine, ecstasy), and medication intake (eg, SSRI) can trigger or increase wake-time bruxism and SB activity	++
Comorbidity A common underlying pathogenetic mechanism is suspected (eg, SB and SDB: does SB play a role in reinstating airway patency following an apnea event, or is SB an apnea-related arousal reaction?)	++

Abbreviation: SSRI, selective serotonin reuptake inhibitor.
+, weak evidence; ++, moderate evidence; +++, strong evidence.
[a] Strength of available scientific evidence.
Data from Refs.[4,15,42,44,48,52,58,70,72,76,78,99,114]

that occurs without any food triturating purpose and is associated with co-contraction of both the jaw-closing and jaw-opening muscles. Moreover, although SB occurs without apparent cortical involvement, unlike chewing, which is initiated at the cortical level, it is strongly influenced by autonomic nervous system activity and arousals during sleep.[15,43] Nevertheless, many scientific studies suggest that SB is centrally regulated, probably in the brainstem, and its genesis is more likely multifactorial.[4,44–46] Conversely, there is little scientific evidence to support a predominant role for peripheral factors, such as occlusal interferences, in the cause of SB.[47]

Sleep Arousal

According to the ICSD-II definition, most RMMA episodes (75%–88%) occur in association with sleep arousals.[3,13,48] This association was first observed by Reding and colleagues[49] in 1968 and by Satoh and Harada[50] in 1971, who described tooth-grinding activity as an arousal reaction. Since then, many studies have used polysomnography and electrophysiology to investigate the complex relationship between SB and sleep arousal.[11,12,42,48,51–53] Sleep arousal is defined as a brief awakening from sleep (for at least 3 seconds) characterized by increased EEG, autonomic, cardiac, and muscular activities without a complete return to consciousness.[13] Arousals normally reoccur from 6 to 14 times per hour of sleep as the response of the sleeping brain to external (environmental) and internal (physiologic or pathologic) stimuli. Recent evidence on the pathophysiology of SB supports the hypothesis that the frequency of RMMA episodes is modulated by the cyclic occurrence of sleep arousals, called the cyclic alternating pattern (CAP).[12,42,48,49,52] CAP is scored on non-REM sleep EEG to identify periods of stable sleep (phase B) that alternate with periods of active and unstable sleep (phase A, arousal).[54,55]

RMMA episodes are observed more frequently in non-REM sleep stages 1 and 2 (light sleep), in sleep stage shifts, and especially in the transition period from non-REM to REM sleep.[4,48,52,56] More than 80% of RMMA episodes are time correlated with CAP phase A, and they recur in rhythmic clusters, with a periodicity of 20 to 30 seconds, which is similar to the physiologic arousal rhythm of CAP.[12,48,52] Notwithstanding this strong association between sleep arousal and SB, sleep arousals (and CAP phase A) are neither the cause nor the trigger of SB. Instead, they constitute the permissive window that facilitates RMMA during sleep.[12,56]

Autonomic Sympathetic-Cardiac Activity

Recent evidence on SB pathophysiology highlights the role of the autonomic nervous system.[42,46,57] It has been well demonstrated that RMMA onset is associated with a sequence of physiologic events that occur within a sleep arousal. Briefly, the genesis of most RMMA episodes is preceded by the following cascade of events[4]:

- An increase in the autonomic sympathetic-cardiac activity with a concomitant withdrawal of parasympathetic influences (from 8 to 4 minutes before RMMA onset)[52]
- The appearance of rapid-frequency EEG cortical activity (sleep arousal; approximately 4 seconds before RMMA onset)[42]
- An approximately 25% increase of heart rate (beginning 1 second before RMMA onset), concomitant with
- An increase in EMG activity of the jaw opener muscle (eg, the suprahyoid muscle, probably responsible for mandible protrusion and airway opening), concomitant with
- An increase in the airflow amplitude visible as two big breaths preceding or concomitant with[58]

- An increase in diastolic and systolic blood pressure[59]
- An observable EMG incident in the jaw-closing muscles (masseter and temporalis), scored as RMMA with or without tooth-grinding sounds[4]; Almost 60% of RMMA episodes are followed in the 5 to 15 seconds after onset by swallowing (**Fig. 2**).[60]

Neurochemicals

Many neurochemicals and neurotransmitters may be involved in the genesis and modulation of jaw movements during sleep, especially those that participate in controlling motoneuron activity and regulating sleep and wake states (acetylcholine, noradrenalin, dopamine, orexin).[44,61] The dopaminergic system was first investigated after the early observation of tooth-grinding activity in a patient with Parkinson disease treated with L-dopa.[62] However, further studies using the dopamine precursor, L-dopa, and the dopaminergic agonist, bromocriptine, demonstrated only a modest effect of dopamine-related medications on SB.[63–65] Dopamine is not usually very active during sleep but it may be linked to sleep arousal reactivation.[66] Conversely, clonidine, an adrenergic agonist, reduced RMMA episodes by 60%, supporting the role of sympathetic-cardiac activation, adrenaline, and noradrenalin in SB genesis.[67] Because noradrenergic action is critical during non-REM sleep in the minutes preceding REM sleep onset, it may participate in the transition from non-REM to REM sleep, a state associated with muscle hypotonia.[68]

Other neurotransmitters, such as serotonin, gamma-aminobutyric acid, cholecystokinin, and orexin, may have a role in modulating RMMA during sleep. Ionic channels, receptors, and their cellular expression may also be involved in SB genesis. However,

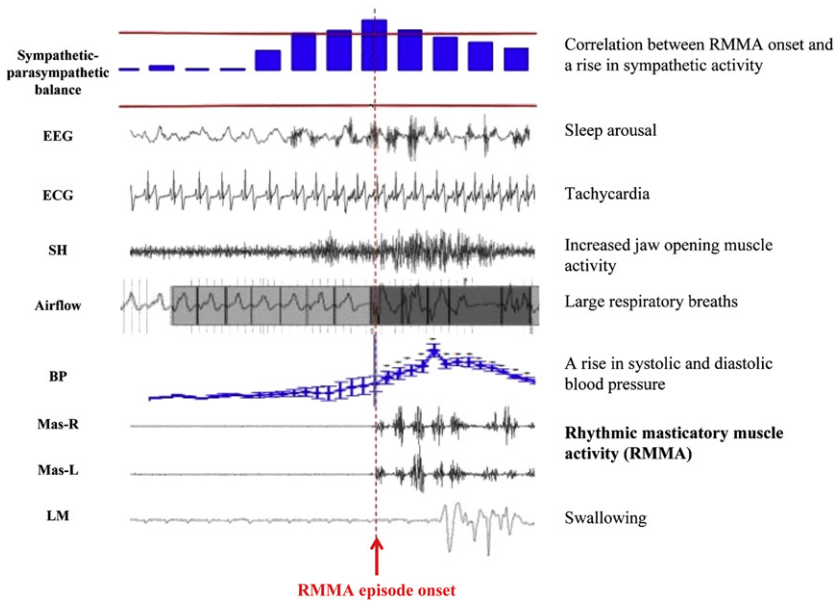

Fig. 2. Genesis of an RMMA episode. The cascade of physiologic events that precedes RMMA onset is shown (schematic representation). A detailed explanation is provided in the text. BP, blood pressure; ECG, electrocardiogram; LM, laryngeal movements; SH, EMG of the suprahyoid muscle; Mas-R and Mas-L, EMG of the right and left masseter muscles. (*Data from* Refs.[11,42,52,53,58,59])

either data are not yet available or the findings are supported by indirect evidence only, derived from case reports on drug and medication use. Prospective and randomized control experimental trials are needed before firm conclusions can be drawn on neurochemical participation in SB genesis.

Genetic Factors

There is little evidence for a genetic predisposition for SB. Children of patients with SB are more likely to be affected than children of individuals who never had SB or who suffer from wake-time bruxism only.[69] From 20% to 50% of patients with SB have a direct family member who ground his or her teeth in childhood, and childhood SB persists in adulthood in 87% of patients.[70,71] In a Finnish twin cohort study, higher concordance was found among monozygotic than dizygotic twins.[71,72]

Despite this early evidence of a genetic basis for SB, the inheritance pattern remains unknown, and no genetic marker has been identified to date. Further research on population-based samples is needed to explore and delineate the probable genetic component in SB genesis. It would be more likely related to genetic polymorphism than a single gene mechanism. Moreover, links to other wake and sleep behaviors would probably emerge.[73,74] It is worth noting that SB assessment tools in large populations, frequently based on a positive history of tooth grinding alone, have yet to be validated for acceptable sensitivity and specificity, especially in the general population. A clinical diagnosis of SB supported with portable systems or single-channel EMG recording is feasible and promising but still lacking in specificity.

Psychosocial Factors: Stress, Anxiety, and Behavior

Aside from a probable genetic predisposition, many other causal or risk factors may play a role in the genesis of SB activity. Psychosocial components in particular, such as anxiety and stress, have frequently been associated with SB.[29,75–79] Both child and adult patients reporting SB were found to have higher levels of urinary catecholamines (adrenaline, noradrenaline, dopamine) than controls.[80–82] These results were attributed to stress factors that activate the hypothalamic-adrenal axis, which controls the catecholamine release. Other studies, mainly questionnaire based, suggest that patients with SB may have maladaptive coping strategies: they seem to be more anxious, stressed, and task oriented as a result of their personality and coping style (eg, type A personality).[29,75,77,78,83,84] Especially in children, SB has been associated with behavioral habits and complaints. These complaints include neuroticism, perfectionism, aggressiveness, lack of concentration and attention (eg, at school), thought disorders, antisocial behaviors, and conduct disorders.[21,85–87] Moreover, all these psychosocial factors have been related to wake-time bruxism.[21] In fact, tooth clenching may be an adaptive or reactive learning behavior (to cope with stress, anxiety, and social life) that may also occur during sleep. However, the overlapping and interactions between wake-time and sleep-time bruxism are still matters of debate.

Alternatively, SB has been considered a tic, an automatism, a movement fragment, or tardive dyskinesia, which may manifest during wake time and persist during sleep.[88] In any case, the many and contrasting findings in the literature indicate that further research is needed to better understand the role of psychosocial factors in SB pathophysiology.[74,76]

Exogenous Factors and Comorbidities

Several exogenous factors and medical conditions have been associated with SB or bruxismlike activities during either sleep or wake time. The exogenous risk factors for

SB include alcohol consumption, cigarette smoking, caffeine intake, medication use (eg, selective serotonin reuptake inhibitor [SSRI]), and drug use (eg, ecstasy).[29,89–99] SB may also be observed in comorbidity with medical disorders, such as attention-deficit/hyperactivity disorder (ADHD)[100,101]; movement disorders (eg, Parkinson disease and Huntington disease)[102,103]; dementia[104–106]; epilepsy[107–109]; gastro-esophageal reflux[110]; and other sleep disorders, such as parasomnias (eg, sleep walking, sleep talking, enuresis, REM sleep behavior disorder [RBD]), periodic limb movements (PLM), RLS, and SDB (**Box 3**).[111–115] It remains to be assessed, however, whether these are cases of intersecting prevalence between 2 parallel disorders or if one condition causes or exacerbates the other.[116]

When SB is associated with medication or drug intake or with medical diseases, it is defined as secondary or iatrogenic SB. Conversely, in the absence of medical causes, SB is considered to be primary, or idiopathic, and it can in turn lead to several clinical consequences on the stomatognathic system, such as tooth wear, tooth damage, tooth fractures, muscle fatigue, orofacial pain, temporomandibular disorders (TMD), and headache.[4]

Box 3
SB and comorbidities

- Parasomnias
  - Enuresis
  - Sleep talking
  - Sleep walking
  - RBD
- Other sleep-related disorders
  - Sleep-disordered breathing (snoring, obstructive sleep apnea)
  - Sleep-related epilepsy
  - PLM and RLS
  - Sleep-related gastroesophageal reflux
- Medical and psychological conditions
  - Hypertrophic tonsils or adenoids
  - Allergies
  - ADHD
  - Headaches
  - Orofacial pain and temporomandibular disorders
  - Stress and anxiety
  - Neurologic and psychiatric disorders (eg, dementia, depression)
  - Movement disorders (eg, Parkinson disease, oromandibular dystonia, tics)
- Oral habits and parafunctions
  - Tics
  - Nail biting, pen biting, and so forth
  - Wake-time tooth clenching

Data from Refs.[23,100–102,107,108,110,115,117–120]

SB, Orofacial Pain, and TMD

Orofacial pain is reported by from 66% to 84% of patients with SB.[117,118] However, the presence or intensity of pain does not seem to be directly correlated with the frequency of RMMA episodes.[23,119,121] In fact, patients with SB with a low frequency of RMMA (2–4 episodes per hour of sleep) seem to have higher risks for orofacial pain and headache than patients with SB with a high frequency of RMMA (>4 episodes per hour of sleep).[23,122] Furthermore, note that SB may coexist with wake-time tooth clenching and other oral parafunctions (eg, lip, cheek, or nail biting), which can also cause or contribute to the development and persistence of orofacial pain.[123–128]

SB has been largely considered a sign or cause of TMD in both adult and pediatric populations.[129–134] Several studies suggest that SB may play a role in TMD genesis, especially the myogenous component, because of muscle hyperactivity during sleep. Nevertheless, TMD pain and morning jaw-muscle pain may be different entities. Most patients with TMD report a pain intensity peak in the late afternoon, whereas patients with SB report transient masseter and temporalis muscle pain or soreness mainly in the morning.[23,135,136]

SB and Headaches

SB has been frequently associated with headaches.[21,113,120,137–141] In a questionnaire-based study, children with SB reported approximately 3 times more headaches than control subjects, with an odds ratio of 4.3.[21] From 30% to 50% of adult patients with SB complain of headache either in the morning (most frequently) or during the day.[138] However, the exact mechanism underlying the possible interactions between SB and headaches remains unknown. It can be hypothesized that SB, which is characterized by repetitive rhythmic and sustained contractions of the masticatory muscles during sleep, may cause tension-type headaches during the daytime. In fact, this comorbidity is controversial because of the overlap with forms of TMD pain and TMD-related headaches.[142,143] Furthermore, the presence of an underlying sleep disorder, such as SDB, has often been associated with both SB and headache. In this latter case, the role of intermittent hypoxia and hypercapnia and sleep fragmentation (after obstructive respiratory events) may be the actual cause of the headaches (**Fig. 3**). Alternatively, SB, headache, and SDB may share common risk factors or pathophysiological substrates without a specific cause-and-effect relationship. For example, it has been shown that children with headaches frequently have concomitant sleep problems, such as SB and SDB, and a higher incidence of TMD.[144,145]

SB and SDB

Although SB and SDB (eg, upper airway resistance, obstructive sleep apnea [OSA], and central sleep apnea) have frequently been associated, the possible cause-and-effect relationship has not yet been elucidated.[29,146–148] Two open clinical studies and one case report have provided indirect evidence for this relationship by showing

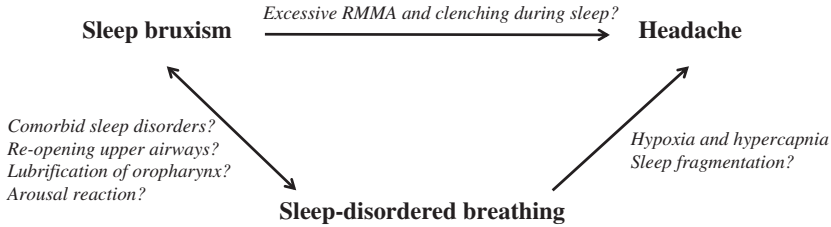

Fig. 3. Comorbid SB, headache, and SDB: putative mechanisms.

a decrease in SB after different SDB treatments (eg, adenotonsillectomy and continuous positive airway pressure).[149–151] These findings support the hypothesis that RMMA may be an oromotor activity that helps reinstate airway patency following an obstructive respiratory event during sleep.[58] An alternative hypothesis considers RMMA a physiologic motor event that is required to lubricate the oropharyngeal structures during sleep, a period when salivary flow and swallowing rate are normally reduced.[60,152] The factors that induce RMMA to reach abnormal frequency in patients with SB remain to be elucidated.

MANAGEMENT OF SB

No therapy to date has been proven effective to cure SB. The available treatment approaches aim at managing and preventing the harmful consequences of SB to the orofacial structures (**Table 3**).[153]

Behavioral Strategies

SB can be managed by behavioral strategies, including the avoidance of SB risk factors and triggers (eg, consumption of tobacco, alcohol, caffeine, and drugs), patient education (eg, control of wake-time oral parafunctions), relaxation techniques, sleep hygiene, hypnotherapy, biofeedback, and cognitive behavioral therapy.[154–158] However, most of these strategies have not been adequately tested in controlled trials. Nevertheless, a recent study showed that a new biofeedback device that applies electrical pulses to inhibit EMG activity in the temporalis muscle was effective in the short term in reducing EMG activity during sleep, without disrupting sleep quality.[155] In addition, a 12-week cognitive behavioral therapy session with patients with SB was found to reduce SB but showed no significant benefits over occlusal splint therapy.[156] Although these behavioral techniques have not yet shown clear or persistent effects, they seem to improve patients' well being and should be considered the first-line management approach in patients with SB.

Oral Appliances

To protect dental surfaces and relax the masticatory muscles, occlusal splints, either on the maxillary or the mandibular arch, have been extensively used in clinical practice. However, the exact mechanism of action is still under debate and there is no evidence to support their role in halting SB. Moreover, the lack of well-designed randomized controlled clinical trials and long-term studies in the literature makes it difficult to assess their effectiveness.[159] Most studies show a decrease (40%–50%) in the RMMA index in the first period of treatment (2–6 weeks), regardless of the type of occlusal splint.[24,160–162] However, the effect seems to be transitory, with values returning to baseline after a short time and the outcomes are highly variable between patients. Moreover, it has been reported that approximately 20% of patients with SB show increased EMG activity during sleep when wearing an occlusal splint, especially the soft mouth guard type.[163]

Occlusal and anterior tooth appliances (eg, the nociceptive trigeminal inhibition system, NTI [http://www.nti-tss.com]) are also used in cases of SB comorbid with orofacial pain and TMD to relief muscle and joint pain.[164–168] Their effectiveness is still controversial because they rarely halt RMMA occurrence.[169] However, it has been hypothesized that these devices may make patients more conscious of their oral parafunctional habits by altering proprioceptive inputs, thus, helping them reduce clenching activity, albeit mainly during wake time.[170,171] Patients with TMD seem to find relief with occlusal splints compared with other or no treatment, especially the most severe

Table 3
Management of SB

Clinical Approach	Functions	Potential Side Effects
Behavioral strategies		
• Patient education, sleep hygiene, relaxation techniques, hypnotherapy, biofeedback, and cognitive behavioral therapy	• Avoid SB risk factors (eg, smoking, alcohol, caffeine, drugs) • Control wake-time oral parafunctions • Improve sleep habits and sleep environment • Control and reduce stress and anxiety (coping) • Relax muscles and reduce EMG activity during sleep	• None identified to date
Intraoral appliances		
• Occlusal or stabilization splint • NTI	• Protect tooth surfaces • Reduce EMG activity (?)	• Impaired occlusion[a] • Increased SB activity • Posterior dental overeruption or anterior dental intrusion (for NTI)[a]
• Mandibular advancement appliances (commonly used for snoring and mild to moderate OSA)	• Reposition and stabilize the lower jaw, tongue, and soft tissues • Open the upper airway space	• Excessive salivation or dry mouth • Tenderness in the teeth, TMJ, muscles • Perception of abnormal occlusion in the morning • Occlusal changes (eg, reduced overjet and overbite)[a]
Pharmacotherapy (recommended in the short term only)		
	• Reduce SB activity + extra effects related to the kind of medication used (eg, hypnotic, analgesic)	Depends on the medication used: • Clonazepam: tolerance, physiologic dependence, fatigue, somnolence • Clonidine: hypotension • Botulinum toxin: risk of retrograde transportation from the site of injection to CNS with systemic side effects

Abbreviations: CNS, central nervous system; NTI, nociceptive trigeminal inhibition system; TMJ, temporomandibular joint.

[a] Only in long-term treatment.

Data from Lavigne G, Manzini C, Huynh NT. Sleep Bruxism. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 5th edition. St Louis (MO): Elsevier Saunders; 2011. p. 1129–39.

cases with TMD pain.[172] Physiotherapy sessions targeting the masticatory muscles may also be useful in cases of SB associated with orofacial pain or TMD.[173,174]

It is worth mentioning that occlusal appliances and anterior tooth splints are not free of unwanted side effects, including changes in dental occlusion, single tooth

positioning, dental hypersensitiveness, and worsening of orofacial pain and SDB.[175] For example, in a pilot study of 10 patients with OSA, a maxillary occlusal splint was found to increase the hypopnea/apnea index in half of the patients, probably by reducing the intraoral space for the tongue, which changes the tongue position during sleep.[175] When SB is concomitant with OSA, or when SDB are suspected, a mandibular occlusal splint (custom made for the lower jaw) or a mandibular advancement appliance (MAA) would be preferable.

MAAs, which are currently used to treat snoring and mild to moderate forms of OSA, have also been tested in the short term to challenge the role of the airways in the genesis of RMMA episodes and to assess therapeutic benefits in patients with SB. An MAA was demonstrated effective in decreasing SB (up to 70%), especially when worn in advanced positions (50%–75% of the maximal protrusion).[176,177] They also seem to relieve daily morning headaches in patients with a low frequency of RMMA during sleep.[178] Although the use of an MAA for SB showed good effectiveness,[153] all these studies assessed the effect after short-term treatment only (2 weeks average). It remains to assess their effectiveness and side effects in long-term studies.[179,180]

Pharmacotherapy

Several medications and drugs have been associated with decreased or increased SB activity, supporting the probability of central mechanisms for SB genesis (**Box 4**).[89] In particular, the dopaminergic, serotoninergic, and adrenergic systems are thought to be involved in this orofacial motor activity. However, evidence is lacking on both the effectiveness and safety of using medications in patients with SB. Therefore, in symptomatic and most severe patients, pharmacologic treatments should be considered as a short-term therapy only.[4]

A recent placebo-controlled study demonstrated a 40% reduction in SB activity with an acute dose of clonazepam (1 mg).[181,182] Clonazepam is a benzodiazepine with hypnotic, anxiolytic, anticonvulsive, and myorelaxant effects. It acts at various levels of the central nervous system. The beneficial effect on SB genesis may result from actions on different systems linked to muscle activity, emotions, and behaviors. However, there are no available data on long-term treatment or potential side effects, such as sleepiness (risk of transportation or work-related accidents), pharmaco-behavioral tolerance, and dependence.

Antidepressant drugs have also been recommended for SB as well as for chronic orofacial pain. However, there is little evidence to support their use. Low doses of amitriptyline (a tricyclic antidepressant) were found to be ineffective against SB,[189,190] and SSRI medications (eg, fluoxetine, sertraline, paroxetine) actually increased tooth grinding and clenching.[95,183,191]

Adrenergic beta-blockers, such as propranolol, were shown to be ineffective on SB.[67] Conversely, an acute dose (0.3 mg) of the alpha$_2$-adrenergic agonist, clonidine, reduced SB by 60%, supporting the role of autonomic cardiac activation in the genesis of this sleep-related motor disorder. However, clonidine is associated with sleep structure changes (eg, less REM sleep) and severe morning hypotension.[56,67] Its use for SB therapy is highly controversial.

Anecdotal reports suggest a positive effect on SB of gabapentin,[184] tiagabine,[192] buspirone,[193] topiramate,[185] and botulinum toxin.[186,187] However, their effectiveness and safety need to be assessed in randomized controlled clinical trials. Potential candidates for more specific or more potent medications are substances that regulate the wake-sleep balance (eg, acetylcholine, noradrenaline, dopamine, orexin, histamine, serotonin), ionic channels, and cellular receptors (on neurons and glia).

Box 4
Effect of medications and chemical substances on SB or SB-like activity[a]

Increased SB activity

- SSRI (eg, paroxetine, fluoxetine, sertraline)
- Norepinephrine-selective reuptake inhibitors (eg, venlafaxine)
- Antipsychotic (eg, haloperidol)
- Flunarizine
- Amphetamines (eg, methylphenidate)
- 3,4-methylenedioxymethamphetamine (ecstasy)
- Cocaine
- Caffeine
- Nicotine
- Alcohol

Decreased SB activity

- Clonazepam
- Diazepam
- Methocarbamol
- Buspirone
- Levodopa
- Pergolide
- Clonidine
- Gabapentin
- Topiramate
- Botulinum toxin

No effect on SB activity

- Propranolol, bromocriptine, L-tryptophan

[a] The scientific evidence is based primarily on case reports (except for 2 randomized controlled clinical trials with PSG [Huynh 2006; Saletu 2010]). No long-term studies have assessed safety or benefits.

Data from Refs.[64,67,89–91,97,181–188]

SUMMARY

SB is a common sleep-related disorder that can be highly distressing because of several harmful consequences to the stomatognathic system, including tooth damage, headaches, muscle pain, and TMD. Dental clinicians are responsible for detecting and preventing these detrimental consequences to patients' oral health. However, SB is much more than tooth wear. Patients with SB need to be screened for other comorbid medical conditions (eg, SDB, insomnia, ADHD, depression, mood disorders, gastroesophageal reflux) before undertaking any treatment approach, especially pharmacotherapy. Because underlying disorders and medication intake may interfere with motor activities during sleep, they need to be assessed before other treatments are recommended. Furthermore, if a medical comorbidity is

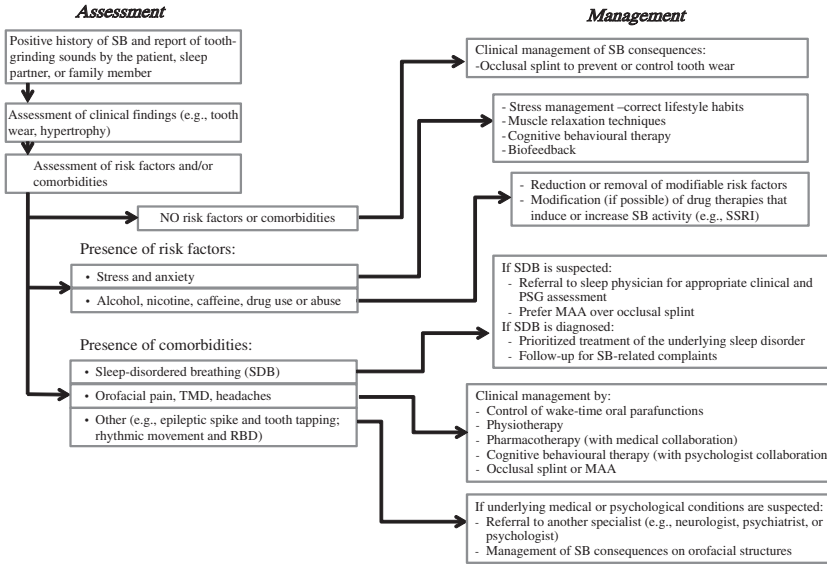

Fig. 4. Algorithm for the clinical assessment and management of SB.

diagnosed (eg, SDB), the therapeutic approach should primarily address the medical disorder while managing the consequences of SB (**Fig. 4**).

CASE STUDY

History and Clinical Examination

A 43-year-old woman complains about tooth grinding during sleep almost nightly. The patient has normal weight and no medical or neurologic diseases but smokes approximately 20 cigarettes a day. She judges herself as highly stressed at work. She reports occasional transient morning jaw-muscle pain, a sensation of jaw locking, and uncomfortable dental occlusion on awakening. However, these symptoms tend to disappear after 30 to 60 minutes and they do not particularly disturb the patient's quality of life. The patient reports good quality of sleep, but tooth-grinding noises and moderate snoring disturb her husband's sleep. The patient's chief complaint is the severe tooth damage she has observed on her dental surfaces, with a negative impact on her esthetic profile, to the point that she does not feel like smiling anymore.

Assessment and Diagnosis

At the clinical examination, severe tooth wear, masseter muscle hypertrophy, and mild pain on palpation at the lateral pterygoid muscles are observed. The patient presents a dental and skeletal class II, a narrow and deep palate, and a Mallampati score of III. The Epworth Sleepiness Scale score indicates a low likelihood of daytime sleepiness, and no other SDB-related signs or symptoms are charted. Based on the patient's and her husband's reports of tooth-grinding sounds and the presence of relevant signs and symptoms, the patient is clinically diagnosed with SB and anatomic predisposition for SDB (history of snoring, retrognathia, and narrow and deep palate, Mallampati III). A PSG evaluation is not mandatory at the moment because the patient does not present any SDB-related symptoms (except for snoring). However, a long-term follow-up is recommended because the risk of SDB increases in women after menopause.

Suggested Clinical Management

The patient should be informed on the characteristics and consequences of SB as well as the available management options. First, cigarette smoking and other possible triggers should be avoided or reduced. Behavioral strategies should be tried to decrease stress, improve coping strategies, and relax the masticatory muscles. To control and prevent tooth wear, an intraoral appliance is recommended. However, because the patient is already a snorer and presents anatomic risk factors for SDB, an MAA would be preferable to an occlusal or stabilization splint. To address esthetic concerns, conservative and prosthodontic treatments should be planned. Follow-up visits must be scheduled to customize and adjust the MAA, verify the patient's general and oral status (stress level), and prevent SB consequences (tooth wear, pain) from worsening.

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