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Review

Sleep less and bite more: Sleep disorders associated with occlusal loads during sleep

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Abstract

Occlusal overload during sleep is a significant clinical issue that has negative impacts on the maintenance of teeth and the longevity of dental prostheses. Sleep is usually viewed as an 'out-of-functional' mode for masticatory muscles. However, orodental structures and prostheses are not free from occlusal loads during sleep since masticatory muscles can be activated at a low level within normal sleep continuity. Thus, an increase in masticatory muscle contractions, by whatever the cause, can be associated with a risk of increased occlusal loads during sleep. Among such conditions, *sleep bruxism* (SB) is a type of sleep-related movement disorders with potential load challenge to the tooth and orofacial structures. Patients with SB usually report frequent tooth grinding noises during sleep and there is a consecutive increase in number and strength of rhythmic masticatory muscle activity (RMMA). Other types of masticatory muscle contractions can be non-specifically activated during sleep, such as brief contractions with tooth tapping, sleep talking, non-rhythmic contractions related to non-specific body movements, etc.; these occur more frequently in sleep disorders. Studies have shown that clinical signs and symptoms of SB can be found in patients with sleep disorders. In addition, sleep becomes compromised with aging process, and a prevalence of most sleep disorders is high in the elderly populations, in which prosthodontic rehabilitations are more required. Therefore, the recognition and understanding of the role of sleep disorders can provide a comprehensive vision for prosthodontic rehabilitations when prosthodontists manage complex orodental cases needing interdisciplinary collaborations between dentistry and sleep medicine.

Keywords: Sleep bruxism; Masticatory muscle contractions; Sleep disorders; Occlusal loads; Dental sleep medicine

Contents

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1. Indroduction

Main purposes of prosthetic rehabilitations are reconstruction of orodental structures by dental prosthesis/implants and rehabilitation of oral motor functions and aesthetic issues in daily life (e.g., chewing, talking, and smiling). Since teeth and dental prostheses are exposed to atypical occlusal loads (i.e., not only force but timing within the fine sequence of closing movement toward tooth contacts) in relation to varying intensity and modality of masticatory muscle contractions, prosthodontists are aware of the significance of occlusal loads operating on teeth and/or dental prostheses when planning of prosthetic rehabilitations. In fact, occlusal 'overloads', a concept of which are revised here since they may not only be excessive in term of strength but can be eccentric in terms of direction relative to tooth physiological axis, are associated with treatment failures or technical complications [1–5]. To prevent these complications, many clinical strategies have been proposed for the choice of materials and the designs ofprostheses [1–4], as well as for their biological integrities with intraoral tissues (e.g., periodontal tissues, mucus membrane) and their functions (e.g., saliva) [6–9].

Masticatory muscles can produce a wide range of biting forces (up to 400–1100 N) [10]. However, they are controlled to produce adequate biting force while functioning (e.g., chewing and other oral functions) [10,11]. These 'functional' activations of masticatory muscles disappear during sleep, a state usually described as an unconscious state. Nonetheless, sleep is not a totally quiet period for masticatory muscles [11]. Increased masticatory muscle contractions during sleep have been thought to potentially cause occlusal overloads and thereby cause clinical complications for teeth and prostheses used in prosthodontic rehabilitation. Interestingly, masticatory muscles can occasionally exert a strong masticatory force during sleep [12,13]. Thus, masticatory muscle contractions during sleep, such as *sleep bruxism*, have been receiving attention from dental professionals as a potentially harmful factor in prosthodontic rehabilitation [2]. Moreover, since disordered sleep is related to increased motor activity and movements, masticatory muscle contractions can be found in various types of sleep disorders. Nonetheless, *sleep* and its problems are rarely discussed as a factor influencing masticatory muscle activity during sleep.

Today, clinical dentistry is collaborating with the field of *sleep medicine* much more than a few decades ago. Sleep problems have emerged as significant medical and social concerns; approximately 20% of the general population in Japan complains of sleep problems [14–16]. It should be noted that sleep problems are more prevalent in middle aged and elderly people than in young people [15,17]: prosthodontic rehabilitations are needed in older than middle aged people due to an increase of the risk for tooth loss [18–20]. Since obstructive sleep apnea syndrome (OSAS) and primary snoring are now significant sleep disorders for which dentists are involved in the clinical management with oral appliances, a field of 'dental sleep medicine' is being developed [21,22]. It is not rare that sleep problems are reported in patients with chronic orofacial pain and headache [23,24]. In addition, SB has been recognized by the sleep medicine society as one of the common sleep disorders [25].

This review aims to propose the potential importance of sleep problems as putative underlying factors for occlusal overloads (e.g., increased strength and/or eccentric directions of occlusal load) during sleep that can potentially cause clinical complications on teeth, dental prostheses and related structures. For this purpose, a brief overview of how masticatory muscle contractions can interact with sleep is presented, and the comorbid sleep disorders with SB are described.

2. Definition and diagnosis of sleep bruxism

The term 'sleep bruxism' and related words used must be clarified to avoid a misunderstanding of the content of this review. In dentistry, 'bruxism' has been defined as oromandibular parafunctional activities including tooth grinding and clenching when an individual is asleep and awake [26,27]. To share the common interpretations of bruxism, a recent consensus proposed that bruxism has distinct circadian manifestations such as sleep and awake states [28]; the two states physiologically differ in terms of masticatory motor controls [29,30]. With this clarification, we will use the term '*sleep bruxism*' according to the International Classification of Sleep Disorders version 2 [25]: it is categorized as a sleep-related movement disorder (e.g., simple, stereotypic, repetitive and localized movements during sleep) and defined as stereotyped oromandibular activity characterized by tooth grinding and clenching during sleep.

In addition to the distinction of behavioral states, a diagnostic grading system for SB has been proposed for clinical and research purposes [28]. A diagnosis of *possible* SB

Table 1
Clinical signs and symptoms for the chairside diagnosis of sleep bruxism.

Items	Limitations
1. Reports of tooth grinding or tapping sounds by a sleep partner or family members	Cannot be reported by edentulous patients and patients who sleep alone Confounding oral noises during sleep
2. Presence of tooth wear seen at an eccentric position	Not specific to ongoing SB; may represent past SB and daytime clenching
3. Presence of masseter muscle hypertrophy on voluntary contraction	Not specific to SB; may represent daytime clenching habit
4. Complaint of masticatory muscle discomfort, fatigue, or stiffness in the morning (reported as transient headache in the temporal muscle region)	Concomitant sleep breathing disorders and sleep disturbance Resolved in the morning
5. Hypersensitive teeth in the morning	Usually in the morning
6. Clicking or locking of the temporomandibular joint	May not be directly associated with SB
7. Tongue indentation	May represent daytime clenching habit

Note: The diagnosis based on #1 can be regarded as *possible* sleep bruxism, and that with #1–7 can be regarded as *probable* sleep bruxism.

can be made on the basis of self-report of tooth grinding noise during sleep; a diagnosis of *probable* SB can be made by a self-report and findings of clinical examinations such as tooth wear, morning masticatory muscle discomfort/pain and masseter hypertrophy upon voluntary tooth clenching; and *definite* SB can be diagnosed when the former two grades (e.g., possible and probable SB) are electrophysiologically confirmed. At the chairside, gathering a history of tooth grinding and clinical signs and symptoms is mandatory for a diagnosis of SB (Table 1) [31]. However, limitations of clinical information obtained at the chairside should be acknowledged for a better understanding of the diagnostic process of '*sleep bruxism*' and for better communication of this issue among clinicians and researchers [31,32] (Table 1). For example, although a history of tooth grinding during sleep is a primary feature of SB, self-reported (*possible*) SB can be biased by the common belief on the putative associations with TMD and stress [24,33]. Therefore, the probability would be improved if a history of tooth grinding noise is witnessed by the family and bed partners (Table 1). Tooth wear is commonly used for the clinical diagnosis of SB. However, the presence of tooth wear can indicate high probability of the past and current SB. Tooth wear should be used with the self-report of frequent tooth grinding noise when estimating the presence of current SB [34].

Another point that is usually overlooked in the clinical diagnosis of SB is whether or not patients have concomitant medical problems [32,35,36]. Thus, SB is regarded as *primary* type when no clear medical complications are present. As described later in this review, SB associated with sleep disorders can be regarded as *secondary* type [36,37]

3. Masticatory electromyographic activity and sleep bruxism

Confirmation of the clinical diagnosis of *possible* and *probable* SB can be done by electrophysiological recordings. However, the interpretation for occlusal overloads during sleep would differ between distinct electrophysiological assessments made in clinical and research settings, i.e., how masticatory EMG events are *identified* and how SB is *diagnosed* based on the identified motor events. Due to the availability and convenience for obtaining data in home environments, electromyographic (EMG) activity of the masticatory muscles has been recorded with a portable recording system in many studies for assessing the masticatory EMG activity related to '*sleep bruxism*' during sleep. In these systems, EMG events or bursts are automatically detected from digitized data of EMG activity during presumable sleep periods based on mathematical algorithms (e.g., detection threshold level) [38–43] (Fig. 1A). Motor systems are greatly influenced by changes in the sleep process: 'background' motor activities exist in the jaw as well as in the body during normal sleep [11]. These include several physiological oromotor activities related to sleep talking, swallowing (physiological events), and undefinable activities related to body movements (e.g., non-specific events) [44] (Fig. 2). Thus, a portable EMG system, probably with an automatic detection method, is able to identify overall masticatory muscle contractions during a sleep period as long as the EMG bursts fulfill the detection criteria (Fig. 1A).

In sleep medicine, to diagnose sleep-related movement disorders, polysomnographic (PSG) recordings with audio-video (i.e., video-polysomnography) are used to visually distinguish *specific* motor events from a variety of 'background' motor activities occurring within the target body regions during sleep [45,46]. This process is very important to avoid an overestimation of motor events by scoring the confounding motor activities. Then the frequency of *specific* motor events during sleep is calculated for determining whether its score fulfills the diagnostic criteria for the respective disorders (Fig. 1B) [47]. In patients who are diagnosed as having SB based on a self-report and clinical examination, jaw motor events are characterized by phasic and/or tonic masticatory muscle contractions such as rhythmic masticatory muscle activity (RMMA) and a small number of isolated tonic contractions: SB patients showed significantly higher frequency of RMMA than that in subjects without a clinical diagnosis [48,49]. Normal subjects showed no RMMA or a small number of RMMA, while background motor activities were scored for both normal subjects and SB patients during sleep (Fig. 2) [44,50]. Thus, among various masticatory EMG

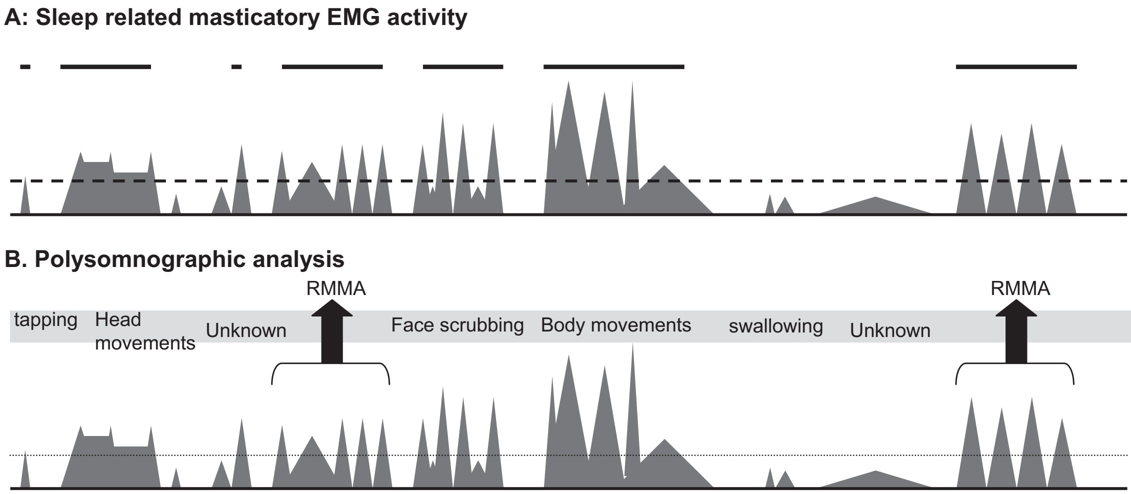

Fig. 1. Schematic illustrations for the assessments of jaw motor events during sleep. (A) In portable EMG recording, EMG events are detected based on the algorithms. An schematic example for burst detection using amplitude criteria is shown. Detection algorithms differ between the laboratories. (B) In PSG recording, EMG events can be identified by the audio-video observation (shaded). Then, specific motor activities are selected among non-specific ones.

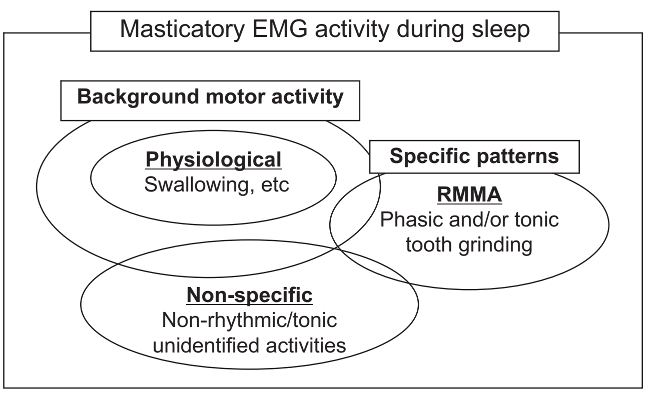

Fig. 2. Classification of masticatory EMG activity during sleep.

activities during sleep, RMMA can be used as an 'EMG marker' for diagnosing SB [36].

Even if electrophysiological assessments are made, physiological interpretations of 'sleep bruxism' would not be the same: 'sleep-related masticatory EMG activity' based on portable EMG recording in combination with an automatic detection algorithm and 'RMMA' based on video-polysomnographic evaluations. However, integrated interpretations of the data from the two different electrophysiological assessments will provide the physiological basis of occlusal overload during sleep as well as the pathophysiology of SB. Ambulatory polysomnography can be another promising tool since sleep variables and masticatory EMG activity can be recorded without audio-video in home environment [42,51]. In the next section, physiological backgrounds of increased masticatory motor activity and SB will be briefly discussed.

4. Increased masticatory motor activity during sleep

Normal sleep consists of a few cycles of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep for 7–9 h. Transition from wakefulness to sleep is related to a reduction of postural muscle tone in the jaw as well as in the body. Thus, an upright posture cannot be maintained during sleep. Decreased muscle tone does not remain at a stable level throughout the night because the two sleep states differ in tonic/sustained motor inhibition [11,52]. In NREM sleep, muscles are less active and excitable in association with a decrease in descending excitatory inputs (e.g., noradrenaline, serotonin and glutamate) to maintain muscle tone and posture [53]. In REM sleep, motoneurons are actively inhibited by the REM sleep generator and thereby jaw muscles are least active and excitable [53]. As a result, REM sleep is characterized by a lower muscle strength in mastiactory muscles (e.g., masseter and temporalis contraction of lower amplitude) and suprahyoid muscles [54]. Since a decrease in muscle strength in the face, jaw, tongue and pharynx results in a narrowing upper airway route, we are sleeping in a disadvantageous situation for breathing. If there are additional factors such as obesity, enlarged tongue, micrognathia, and narrow palate, breathing during sleep would be disturbed as seen in patients with OSAS [55,56]. On the other hand, when the REM inhibitory system is disturbed, phasic and tonic masticatory muscle activities can emerge in relation to dream-enacting movements and behaviors as seen in patients with REM sleep behavior disorders [57,58].

Inhibitory effects on masticatory motor activity during NREM and REM sleep are periodically not constant, there is pauses in the sleep inhibitory activity observed by sudden cortical and cardiac activations, named arousals [59,60]. In normal sleep, microarousals (lasting 3–15 s) occur at a frequency of 10–15 times per hour of sleep and tend to occur at the end of an NREM period [61,62]. A more intense arousal activity, awakening (lasting more than 15 s), is less frequent (3–4 times per hour of sleep) but is often associated with body position changes and sleep stage shift (e.g., from a deeper to a lighter stage). These arousal activities repeatedly occur every 20–60 s and are called cyclic alternating patterns (CAP) [62]. It is normal for these transient interruptions to occur during sleep. The frequency of these arousal events are found to increase with age, especially from young adults to elderly people [61]. However, when they occur very frequently and sleep becomes fragmented, physiologic (e.g., elevated sympathetic activity) and psychologic (e.g., mood changes) alterations can be induced as seen in patients with sleep problems.

The occurrence of transient arousals is associated with not only merely an increase of muscle tone but also contraction(s) of single or multiple muscles or gross body movements [60,63]: a larger motor activity often involves muscle contractions in the jaw and body. Tonic and non-rhythmic sustained masseter contractions are frequently associated with awakenings related to body movement in normal subjects [64,65]. These non-specific oromotor activities consist a part of the 'background' EMG activities and represent EMG bursts that are not definable on the bases of patterns of signals from sensors (e.g., EMG electrodes, airflow) or on the basis of the audio-video observation (e.g., voice, jaw/face movements) (Fig. 1B) [45–47]. When sleep is fragmented by frequent arousals, the chances of masticatory muscle contractions will be elevated as indicate by the results of the studies showing that sleep becomes 'noisy' with body movements and behaviors in patients with sleep disorders [47]. In other words, elevated sleep-related masticatory EMG activity can increase the risk of occlusal loads during sleep as well as reflect the possibility that patients have disturbed sleep with sleep disorders. In most studies, sleep-related masticatory EMG activity was investigated in normal subjects. Thus, portable EMG techniques can be used for large populations in association with sleep-related questionnaires and measures of biobehavioral variables (e.g., body movements assessed by actigraphy) [64].

5. RMMA and tooth grinding

Among the various background masticatory EMG activities during sleep, RMMA is characterized by repetitive phasic and/or tonic contractions of masticatory muscles, which can be identified by EMG signals and audio-video. Approximately 60% of young normal subjects (e.g., without tooth-grinding history or clinical complaints) exhibited a small number of RMMA (1.8 episodes/hour) in the absence of tooth grinding during sleep. Among young subjects investigated, patients with moderate to severe SB showed a three-time larger number of RMMA (5.8 episodes/hour) with two-times larger number of muscle bursts and 30% higher EMG amplitude [66]. In SB patients, most episodes of RMMA occur in NREM sleep stages I and II (now named N1 and N2 by the American Academy of Sleep Medicine [67]) during the period shifting from deep NREM sleep (stages III and IV, named N3) to REM sleep [68]. RMMA episodes are associated with physiologic changes related to microarousals such as transient EEG change, a transient increase in heart rate, and frequent shifts in sleep stages and CAP as well as augmentation of respiratory activity, increase of blood pressure and concomitant swallowing [69–74]. However, these transient arousals are not directly coupled with RMMA, since studies have shown that an experimental increase in transient arousals failed to increase the occurrence of RMMA in healthy controls (e.g., very small number or no RMMA) and SB patients (large number of RMMA) [75–77]. In OSAS patients whose sleep is fragmented by arousals, arousals induced by apnea are rarely associated with RMMA [56,78]. In addition, some OSAS patients do not exhibit RMMA in spite of frequent arousals [63]. These findings suggest that transient arousals are not a direct triggering factor for RMMA and further indicate the presence of predisposed conditions (i.e., a physiological window of increase probability of motor events being present) for SB patients to exhibit a heightened responsiveness of RMMA to arousals [59,76]. Further investigations are needed what are the triggering factors for RMMA; based on above and current evidences, not a single factor for all patients will be identified [30].

6. Tooth contacts and masticatory force measurements

Several studies have directly measured tooth contacts during sleep using intra-oral sensors [79,80]. In normal subjects, tooth contacts of incisors were found to be associated with swallowing and body movements during sleep when jaw closing muscles are activated following a transient arousal [79–81]. This supports the idea that 'background' masticatory muscle contractions are associated with occlusal loads during sleep in normal subjects. Tooth contacts occurred in clusters during night [79,80,82,83] as reported in recent polysomnographic studies in SB patients [68,73]. Interestingly, tooth contacts occurred more frequently in patients with a witnessed tooth grinding than normal subjects without it [83]; the results are consistent with those of PSG studies in which the incidence of tooth grinding-related noise is higher in SB patients than in normal subjects [29].

A few studies challenged to measure bite force during sleep by using occlusal splints equipped with force detecting sensors [13,83,84]. In 10 subjects with or without a self-report of SB, an average peak bite force during sleep was approximately 30% of that during maximal voluntary clenching (MVC) and maximal bite force over night in each subject ranged from 17 to 111.6% of MVC with a mean of 53.1% [13,83,84]; these values overlapped between subjects with and without a self-report of SB. The above results should be interpreted with caution since actual occlusal loads are not simply reflect the number of tooth contacts and the EMG activity of masticatory muscle contractions; bite force measurements are sensitive to the design of device, the place of the measurement on dental arch (e.g., incisor vs. molar), positional relationships between mandibular and maxillary bones, inter-individual difference of muscle strength, etc. In fact, other studies have shown that the various patterns of masticatory muscle contractions and jaw movements during sleep were found to be unique compared to awake chewing [11,13,85–87]. Currently, physiological significance of masticatory muscle contractions on actual occlusal loads in relation to clinical complications remains to be clarified. Further studies are needed to investigate the interrelationships among masticatory EMG activity, mandibular movements/positions, and other factors for developing techniques useful in the assessment of occlusal loads during sleep in clinical settings.

7. Sleep bruxism and sleep disorders

7.1. Prevalence of sleep bruxism

Based on subjective reports of tooth grinding noise during sleep (e.g., *possible* SB), the prevalence of SB decreases throughout life time. In children, SB is reported by parents in 10–30% of cases [36,88]. Although there might be fluctuation over generations [89,90], cross-sectional studies have shown a decreasing trend in the prevalence from young adults to elderly people [91–93]. It has often been suggested that a low prevalence of SB in elderly population is due to the methodological limitations of an epidemiological study; tooth grinding noise cannot be generated in subjects with severe tooth loss or edentulous subjects. However, a recent cross-sectional study challenged this suggestion and showed that age is an independent factor for a lower prevalence of self-reported SB in the elderly [93]. The decrease in the prevalence of SB is interesting when compared to the age-related changes in sleep architectures and in the prevalence of other sleep disorders in adults (see the next section and Table 2). The amount and quality of sleep are decreased in elderly people without medical complications; sleep in elderly people is characterized by less deep sleep and higher sympathetic tone, with more frequent arousals [61,62,94,95]. As discussed below, in contrast to SB, most sleep disorders (e.g., insomnia, OSAS, restless legs syndrome [RLS]/periodic leg movements in sleep [PLMS]) are more prevalent in the elderly population [25].

7.2. Concomitant occurrence of sleep bruxism and sleep disorders

Sleep disorders cover a wide spectrum of clinical conditions and pathophysiology. In ICSD-2, sleep disorders are categorized into eight sections with other additional conditions [25,96]. It should be noted that sleep disorders can concomitantly occur in a single patient. Moreover, SB has been reported to occur in several sleep disorders (Table 2). Thus, clinical signs and symptoms for the clinical diagnosis of SB (e.g., frequent tooth grinding history, tooth wear, and morning masticatory muscle symptoms) can be found in patients with sleep disorders. Although sleep is disturbed and motor activity is often increased due to the distinct pathophysiological background in sleep disorders, evidence regarding co-morbidity with SB and increase of masticatory EMG activity has been gathered from case reports and subjective assessments; few electrophysiological assessments have been made [35]. Whether a concomitant presence of SB in sleep disorders is associated with secondary influence of sleep disruption (e.g., increased microarousals) or with common mechanisms for rhythmic oromotor activation remains to be investigated [97]. Since increased sleep-related masticatory EMG activities have been found in patients with OSAS by using polysomnography, the association between the two conditions will be discussed in another section. Detailed information on clinical symptoms and prevalence is presented in Table 2.

7.2.1. Insomnias

The general definition of *insomnias* includes a complaints of difficulty in initiating and/or maintaining sleep, waking up too early, nonrestorative sleep and poor sleep. Today, these sleep complaints were found in approximately 20% of the Japanese population [14–16]. Patients with insomnia often complain of muscle stiffness, headache, gastrointestinal discomfort, fatigue and mood changes in relation to sleep problems (Table 1). Since insomnia-like symptoms are common in patients with sleep disorders and inadequate sleep hygiene, additional interviews would be needed to rule out the possibility of complaints of other signs of sleep disorders (e.g., snoring, leg movements, sleep-related behaviors) (Table 1). Insomnia-like symptoms are often found in patients with temporomandibular disorders (TMD) [24]; there are interactions between pain and sleep loss [98]. In addition, a recent study with a large sample suggest the direct link between TMD with myofacial pain and SB diagnosed by PSG is not obvious [33].

Transient insomnia is characterized by the above complaints lasting less than 3 months and associated temporally with stressors in the daily life events. A classical portable EMG study performed for a period of several months showed that there were a few patients whose masticatory EMG activity at night was elevated in response to stressful life events [99]. The result more likely suggests an increase of masticatory EMG activity in relation to short-term sleep disturbance secondary to stress. In a later study, 8% of 100 subjects showed a correlation between daily stress level and sleep-related masticatory EMG events [100]. How transient insomnia can contribute to the night-to-night variability of masticatory EMG activity and SB remains to be elucidated.

Patients with p*sychological insomnia*, so-called *chronic insomnia*, have complaints of difficulty in sleeping lasting at least 4 weeks. They usually exhibit heightened arousals and overconcerns about health. Sleep for patients with insomnia is characterized by delayed sleep latency, shortend sleep duration, and increased number of awakenings. Psychological stress, emotion and personality traits are associated with the development of chronic insomnia and changes in sleep architectures (e.g., duration, fragmentation). A recent study showed that sleep-related masticatory EMG activity was higher in subjects with a trait of anxiety than in those without such a trait [101]. However, there have been few studies on the associations between SB, anxiety/stress and insomnias [24].

Symptoms of insomnia can be reported by people who are engaged in *shift work* and people who have *inadequate sleep hygiene*. It has been reported that irregular shift workers reported stress and insomnia symptoms more frequently than did workers on a regular daytime schedule and that self-reported SB was indirectly related to insomnia symptoms [102,103]. Symptoms of insomnia were found in patients with mental disorders (e.g., depression) with medications: tooth grinding and other masticatory muscle contractions during sleep were observed by PSG recordings [104,105]. Medications can exacerbate the occurrence of RMMA, trigger it, or increase of the risk of having clinical signs and symptoms of SB without a clear increase of tooth grinding. In fact, various types of drugs

Table 2
Characteristics of sleep disorders associated with sleep bruxism [15–17,25,96,126,150–153].

	Clinical symptoms	Prevalence/age
Sleep breathing disorders		
Obstructive sleep apnea syndrome	- Loud and frequent snoring, witnessed apneas, daytime sleepiness; physical symptoms: hypertension, obesity, large neck circumstance; orodental findings: narrow palate, enlarged tonsils/adenoid and tongue, micrognathia, oral dryness	- 2–4% of middle-aged adult - Male:female = 2:1 - More common after middle age
Insomnias	- Difficulty in initiating sleep, and maintaining sleep, waking up too early, nonrestorative sleep, poor quality of sleep; symptoms lasting for <4 weeks (transient) or for >4 weeks (chronic insomnia) - Muscle tension/pain, fatigue, attention impairment, daytime sleepiness, mood disturbance, chronic orofacial pain - Note that the symptoms are common for other sleep disorders	- 6–33% of the general population - 20% in the Japanese population - Male < female - Increase with aging
Hypersomnias		
Narcolepsy	- Excessive daytime sleepiness, sleep attack - With or without cataplexy (sudden loss of muscle tone triggered by strong emotions: laughter, pride, elation, surprise)	- 0.02–0.18% in US and European populations - 0.16–0.18% in the Japanese population - Slight preponderance of males - Very rare before 5 years of age - More common in teenagers (peak age: 14 years)
Circadian rhythm sleep disorders	- Insomnia, excessive daytime sleepiness - Delayed sleep phase type (habitual sleep–wake time delayed usually more than 2 h) - Advanced sleep phase type (advance of habitual sleep onset and wake up time) - Shift work type (work times scheduled during usual sleep time) - Irregular sleep–wake type - Jet lag type	- 7–16%, more common in adolescents and young adults (delayed sleep phase type) - 1% in middle-aged and older adults, increases with age (advanced sleep phase type) - 2–6% estimated (shift work type)
Parasomnias		
NREM parasomnias		
Sleep walking	- Ambulation during sleep - Persistence of sleep, altered state of consciousness, impaired judgment during ambulation	- 17% in children (peak age = 8–12 years) - Up to 4% in adults
Sleep terror	- Sudden episode of terror during sleep: cry or loud scream - Accompanied by tachycardia, tachypnea, diaphoresis, increased muscle tone	- 1–6.5% in children - 2.3–2.6% in 15–64-year-old people - 1% in people over 65 years of age
REM parasomnia		
REM sleep behavior disorders (RBD)	- Sleep-related injurious and disruptive behavior: talking, laughing, shouting, grabbing, arm flailing, kicking, punching, sitting up	- 0.38% in the general population - 0.5% in the elderly population - Male:female = 9:1 - Predominant after the age of 50 years
Sleep-related movement disorders		
Restless legs syndrome	- Urge to move legs, in the evening or night, worse at rest - Sensory distress (uncomfortable sensation in legs)	- 5–10.6% in adults in US and European populations - 1.4% in the Japanese elderly population - Male:female = 1:1.5–2
Periodic leg (limb) movements in sleep (PLMS)	- Periodic episodes of repetitive stereotyped limb movements at an average interval of 5–90 s during sleep - Most frequent in lower extremities, typically involving extension of the big toe, often in combination with partial flexion of the ankle or knee - PLMS with symptomatic sleep disruption is called periodic limb movement disorder (PLMD)	- Quite common, increasing with advancing age, up to - 34% in the population over 60 years of age - PLMD is rare (exact prevalence not known)
Other disorders		
Sleep-related gastroesophageal reflux	- Recurrent awakening with shortness of breath or heartburn - Sour bitter taste in the mouth, sleep-related coughing or choking	- 7–10% of the population during waking hours - Sleep-related prevalence unknown
Sleep-related epilepsy	- Abrupt awakening from sleep, generalized tonic–clonic movements of limbs, focal limb movements, automatism (lips macking, orofacial movements), urinary incontinence, tongue biting, postictal confusion and lethargy	- Estimated 10–45%

acting on the central nervous systems can be associated with SB [36,106]

7.2.2. Hypersomnia

One common type of hypersomnia is *narcolepsy.* Patients with narcolepsy complain of severe daytime sleepiness or inability of staying awake and some of them report sudden and transient episodes of loss of muscle tone triggered by emotions (e.g., laughing), so-called *cataplexy* (Table 2). Although SB in patients with narcolepsy has not been reported, sleep of narcoleptic patients seems less fragmented [107]. It should be noted that these patients usually take medications for stimulating the central nervous system to maintain vigilance (Litalin®, Modafinil®); these drugs may increase tooth grinding and clenching during sleep and wakefulness [108].

7.2.3. Circadian rhythm disorders

In circadian rhythm disorders, a delayed or an advanced shift of sleep–wake timing causes misalignment with engogeneous circadian rhythms (e.g., hormones, body temperature) and is associated with sleep disturbance, daytime sleepiness and insomnia. Currently, little information is available on sleep-related masticatory EMG activity and SB is in patients with circadian rhythm disorders. The occurrence of RMMA seems affected by hemi-circadian rhythm during sleep; it occurs more frequently in the middle of night [68]. It may be important to remind readers that chewing or eating behavior is regulated by circadian rhythm in relation to endocrine functions for feeding and metabolism (e.g., grehlin and leptin) [109]. Whether or not the occurrence of RMMA is associated with such behavioral cues remains to be demonstrated.

7.2.4. Parasomnias

Parasomnias are undesirable physical events or experiences that occur during sleep. Usually, complex, purposeful and goal-directed behaviors occur outside conscious awareness (Table 2). These patients may have a history of abnormal behaviors and frequent sleep talking during sleep. Tooth grinding during sleep is more frequently reported in adult patients with violent behaviors than in those without such behaviors [110].

Most parasomnias, such as sleep talking, enuresis, sleep-walking, confusional arousals, and sleep terrors, are more prevalent in children than in adults. Correlations with anxiety and stress as well as familial predisposition have also been reported for these parasomnias in children. Studies have been shown that children with these parasomnias reported tooth grinding during sleep more frequently than did those without these parasomnias [111] Moreover, several studies have indicated that these parasomnias and SB may share common genetic predispositions [112,113].

REM sleep behavior disorder (RBD) is characterized by excessive muscle activity and/or abnormal behaviors and talking related to the dream content during REM sleep (Table 2). *RBD* is more prevalent in the elderly, and the majority of patients develop Parkinson's disease. One patient with subclinical *RBD* exhibited tooth grinding and clenching during sleep [57]. In a controlled study with a small sample size, phasic masticatory EMG activities, such as RMMA and oromandibular myoclonus (e.g., tooth tapping), were scored more frequently during REM sleep in RBD patients than in age-matched controls [58,114]. In addition, due to the presence of oromotor activities during sleep, one-fourth of RBD patients would fulfill the PSG criteria for diagnosis of SB. Thus, RBD should be suspected in the elderly patients who complain of tooth grinding, sleep talking and dream-related behaviors during sleep. Then the diagnosis can be confirmed by polysomnographic evaluation if RMMA is found to occur in REM sleep [58].

In other parasomnias, sleep-related groaning (*catathrenia*) is characterized by monotonus vocalization during sleep. The sound should be distinguished from other noises such as snoring and talking. Catathrenia has been reported to be associated with masticatory muscle contractions associated with SB [115].

7.2.5. Sleep-related movement disorders association to SB

Restless legs syndrome (RLS) is characterized by intense unpleasant feelings in the legs and an urge to move the legs for relief (Table 2). Sensory complaints are often reported in the elderly populations [150–153]. The symptoms become worse in the evening especially when patients are at rest. Tooth grinding during sleep was reported in only 10% of patients with restless legs syndrome [91,116]. Periodic leg movements in sleep (PLMS) is characterized by periodically reccurring leg motor events with dorsiflexion of the ankles at an average interval of 5–90 s during sleep. The majority of RLS patients exhibit PLMS in PSG evaluations [117]. In addition, PLMS can occur more frequently in normal subjects older than 60 years of age [118]. In one PSG study, PLMS was found in few SB patients, although RMMA can occasionally be associated with leg and body movements that cannot be scored as PLMS [119]. Thus, the occurrence of SB and RLS/PLMS can be overlapped without a common cause and pathophysiology in a single patient. Dopaminergic drugs are usually prescribed for patients with RLS/PLMS: One case report showed that such a drug reduced SB in patients with implant failure [120] while another study using a different type of dopaminergic drug did not demonstrated the same results in the randomized design [154].

7.2.6. Other sleep problems

Oromandibular myoclonus (OMM) is characterized by repetitive or isolated tapping-like jaw movements. Patients with oromandibular myoclonus may complain of nocturnal tongue biting and insomnia. OMM is a different entity from SB, although approximately 10% of SB patients exhibit OMM [121,122]. OMM can be found in patients with RBD and epilepsy. In patients *with sleep-related epilepsy*, nocturnal seizures are associated with a variety of movements in the limbs (e.g., bicycling legs) and the mouth (e.g., chewing automatism). Among these motor activities, RMMA with tooth grinding has been observed in one case [123]. *Sleep-related gastroesophageal reflex* (sleep-related GER) can be associated with SB [124], while co-morbidity between the two remains unknown. Patients with sleep-related GER report the heartburn sensation in the morning. Recurrent reflux events can be associated with transient arousals and swallowing. Tooth wear and erosion can become more severe when sleep-related GER is co-morbid with SB [6].

8. Obstructive sleep apnea syndrome and SB

Obstructive sleep apnea syndrome (OSAS) is a common sleep-related disorder that affects 2–4% of the adult population [125]. This disorder is predominantly reported in males who are past middle age and are obese. Obstructive sleep apnea syndrome is characterized by recurrent episodes of complete or partial upper airway obstruction, occasionally associated with loud snoring during sleep [55,126]. Frequent occurrence of upper airway obstructions induces arousals from sleep and leads to fragmentation of the sleep process. Patients with OSAS often show excessive daytime sleepiness, fatigue and cognitive impairment during wakefulness. In addition, many medical complications have been reported to be associated with OSAS (e.g., hypertension, cardiac arrhythmia, ischemic heart disease, and cardiovascular disease). It has been reported that Asian patients with OSAS are less obese but that symptoms are more severe due to craniofacial structures (e.g., cranial base) that increase the risks of upper airway narrowing [127]. It should be noted that the sleep medicine society emphasizes the roles of dental professionals since craniofacial risks for OSAS can be screened by dental professionals in usual oral examinations [128,129].

In the adult population, snoring or OSAS was reported in more than 30% of adult subjects with *possible* SB (e.g., grinding history and/or morning jaw muscle discomfort) [92,93]. The odds ratios of having *possible* SB were 1.4–2.58 for snoring and 1.8 for sleep apnea [92,93]. Approximately 10% of Japanese patients with OSAS have subjective reports of tooth grinding and clenching during sleep [130]. In a few polysomnographic studies, increased masticatory EMG activities including RMMA were observed in 40–60% of a small group of adult patients (10–20 patients) with OSAS [78]. It should be noted that the association between apnea/hypopnea and arousals is opposite to the association between SB and arousals: apneic events trigger arousals while RMMA is triggered during arousals [56]. Nonetheless, the studies failed to show a temporal association between apneic events and RMMA. Instead, tonic masseter muscle activity was frequently found at the end of apneic events [78,131,132]. In OSAS patients, such non-specific masticatory EMG activity can occur in response to arousals related and unrelated to apneic events, suggesting that non-specific oromotor activation can occur in relation to frequent arousals [47,63]. These findings indicate that SB, characterized by the occurrence of RMMA, does not necessarily occur in OSAS patients with sleep fragmentation. Moreover, even though patients do not have a history of tooth grinding during sleep or other clinical signs and symptoms of SB, they may exhibit an increase in masticatory muscle contractions during sleep due to sleep fragmentation. Whether or not the concomitant occurrence of RMMA and sleep-related mastiactory EMG activity is associated with the degrees of sleep fragmentation (e.g., high frequency of apnea events) is currently being investigating in a large group of OSAS patients.

Over the past few decades, bi-maxillary oral appliances (OAs) have become a management option for patients with primary snoring or mild to moderate OSAS [133]. OAs increase the upper airway space by pulling the mandible or tongue in an advanced position [134]. As a result, OAs reduce the number of obstructive events during sleep and restore fragmented sleep in association with improvement of clinical symptoms [133,135]. Recently, OAs have been tested in patients with SB. OAs were effective for reducing the occurrence of RMMA than were the conventional uni-maxillary appliances (e.g., occlusal splint) [136,137]. In addition, the reduction of RMMA in SB patients by OAs was associated with a decrease in orofacial pain complaints [138], although the study has been done only for a short-term period. On the other hand, one study has shown that an occlusal splint can exacerbate apnea and hypopnea in patients with OSAS [139].

It has also been suggested that upper airway and face morphology contribute to the SB seen in OSAS pediatric patients, while morphological influences on concomitant OSAS and SB are not known [140]. Two studies have reported the opposite results on whether a use of dentures at night reduces or increases apnea events in edentulous OSAS patients [141,142]. Interestingly, edentulous subjects can exhibit either rhythmic or tonic masseter contractions even when they sleep without wearing dentures [143,144]. It would be important for prosthodontists to ask about the presence of past SB for the edentulous patients undergoing occlusal rehabilitation; masticatory muscle contractions may affect occlusal loads after establishing a new occlusion [145,146].

The above clinical findings suggest the intra-oral devices or prostheses can have either positive or negative effects on sleep-related breathing, motor activity and sleep, probably in association with individual morphological and pathophysiological backgrounds (e.g., OSAS). Further studies are needed to clarify pathophysiology and to develop management strategies in patients with concomitant SB and OSAS.

9. Recognition of sleep disorders at the chairside

No definitive treatment strategies for eliminating SB have yet been established. Thus, prosthodontists' main strategy is to reduce the effects of occlusal overloads on the teeth and prostheses in prosthodontic rehabilitation [2,147]. Prosthodontists usually ask about the awareness of tooth grinding during sleep when they find intraoral signs and symptoms related to SB (Table 1). However, they may not ask about sleep. Although objective measurement of masticatory EMG activity during sleep is ideal, for clinical perspectives, sleep disorders can be screened by clinical interviews and oral examinations [32,35]. Patients with sleep disorder(s) often complain of common sleep problems (e.g., poor sleep, daytime sleepiness, fatigue, etc.) (Table 2). Thus, interviews for specific features of sleep problems can be carried out for detecting possible sleep disorders in patients undergoing prosthodontic rehabilitation. It is also important to ask about the presence of risk factors proposed for SB; most are also known as risk factors for sleep disorders. For example, smoking is a risk factor for SB [148], but it disturbs sleep architectures and induces insomnia-like symptoms [149]. When patients report a history of sleep-related breathing events (e.g., snoring), OSAS can be screened by intraoral examination in addition to the interview [96]. When sleep disorders have been clinically diagnosed, the patient can be referred to a sleep specialist. Thus, sleep specialists will play a major role in the diagnosis and treatment of sleep disorders, and prosthodontists can manage orodental complications.

10. Conclusion

This review shows the role of masticatory motor activities as a contributing factor to occlusal overloads (eccessive loads or loads in eccentric axis) during sleep, such as in presence of SB. Atypical occlusal load during sleep is probably not independent of mechanism related to motor control and/or to wake–sleep mechanisms and their relationship to the occurrence of sleep disorders. Dentist has to be aware that SB can be isolated or associated with other sleep disorders in a given single patient. Thus, controlling concomitant sleep disorders can be effective in reducing the risks for occlusal overloads with a decrease in masticatory motor activity and SB as well as improvement of the patient's quality of life. Neither a single mechanism, nor a simple clinical response can be given to complex cases with comorbid sleep or medical disorders. Dentists have to understand the role of personalized medicine when they try to find explanation to patients with complex clinical complications, as many biological and psychosocial factors may interplay in causing various disorders and diseases. The recognition of sleep problems can provide a new clinical dimension for prosthodontists in understanding and managing orodental complications related to occlusal loads on tooth and orofacial structures as well as in broadening the field of SB as another interdisciplinary area linking dentistry to sleep medicine.

Conflict of interest

Authors have no conflict of interest concerning the present manuscript.

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