Topical Review: Sleep Bruxism and the Role of Peripheral Sensory Influences

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Sleep bruxism (SB) is an unusual orofacial movement described as a parafunction in dentistry and as a parasomnia in sleep medicine. Since several peripheral influences could be involved in sleep-wake regulation and the genesis of rhythmic jaw movements, the authors have reviewed the relevant literature to facilitate understanding of mechanisms possibly involved in SB genesis. Various animal and human studies indicate that during either wakefulness or anesthesia, orofacial sensory inputs (eg, from periodontium, mucosa, and muscle) could influence jaw muscle activity. However, the role of these sensory inputs in jaw motor activity during sleep is unclear. Interestingly, during sleep, the jaw is usually open due to motor suppression; tooth contact most likely occurs in association with sleep arousal. Recent physiologic evidence supports an association between sleep arousal and SB; a sequential change from autonomic (cardiac) and brain cortical activities precede SB-related jaw motor activity. This suggests that the central and/or autonomic nervous systems, rather than peripheral sensory factors, have a dominant role in SB genesis. However, some peripheral sensory factors may exert an influence on SB through their interaction with sleep-wake mechanisms. The intent of this review is to integrate various physiologic concepts in order to better understand the mechanisms underlying the genesis of SB.
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Introduction and Overview of Sleep Bruxism

"Bruxism" has been defined as an oral parafunctional activity regardless of the time of its occurrence.[1] This broad definition includes not only tooth grinding and clenching, but also other oral habits such as nail biting, tongue pushing, and jaw bracing. More recently, however, bruxism has been classified into primary (idiopathic) and secondary (iatrogenic) forms (Fig 1).[2–5] According to this latter proposal, the primary form includes daytime clenching and sleep bruxism (SB) in the absence of a medical cause, while the secondary form is associated with either neurologic, psychiatric, sleep disorders, use of medication, and/or possibly any combination of these.

Bruxism occurring during wakefulness needs to be differentiated from bruxism occurring during sleep, since wakefulness and sleep are different physiologic states with different influences on oromotor excitability.[6–8] The American Academy of Sleep Medicine (formerly the American Sleep Disorders Association) has classified SB

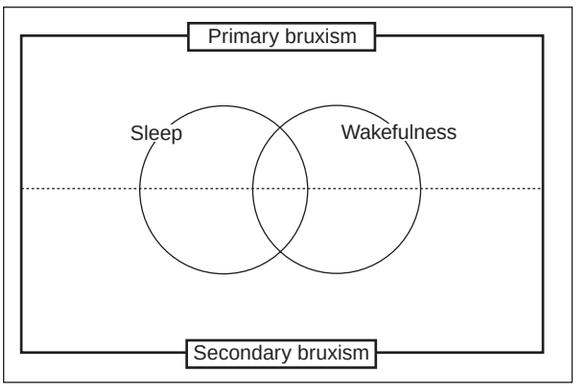

Fig 1 Proposed bruxism classification. Bruxism can be classified based on the basis of: *(1)* occurrence during sleep or wakefulness and *(2)* primary or secondary causes. Some subjects may report both sleep and awake bruxism, hence the overlap of the circles indicated in the figure. Secondary bruxism is associated with medical conditions (eg, disease, medication) that may exaggerate primary bruxism or may cause bruxism.

as a sleep parasomnia: "a sleep disorder which is not an abnormality of the processes responsible for sleep and awake states per se but an undesirable physical phenomenon that occurs during sleep."[9] Accordingly, we propose the following definition of SB: a parasomnia and an oral parafunctional activity that is characterized during sleep by either jaw clenching (tonic activity) and/or repetitive, phasic jaw muscle activity that produce tooth grinding.

Sleep bruxism episodes are further subclassified into either phasic, tonic, or both (mixed) types according to the duration of jaw-closing muscle bursts.[10] It has been found that approximately 90% of SB episodes are of the phasic and mixed types involving repetitive phasic muscle bursts occasionally associated with tooth grinding.[10–13] Recently, our group introduced the term "rhythmic masticatory muscle activity (RMMA),"[14,15] since repetitive phasic muscle activity, in the absence of tooth grinding, is observed during sleep in several sleep disorders (eg, somnambulism, rapid eye movement sleep disorders) and is observed in approximately 60% of normal control subjects.[11,16–18]

The mechanisms involved in the genesis of SB are not yet fully understood. It has been suggested that several neurochemicals are involved, as has also been reported in other sleep movement disorders.[2,4,19–21] When L-dopa (a precursor of dopamine, adrenaline, and noradrenaline) was tested in young healthy SB patients in a controlled trial, it produced a modest (≈ 30%) but significant reduction in SB activity.[22] The specificity of dopamine in the genesis of SB remains to be determined because a recent controlled study using a modest dopamine agonist (eg, bromocriptine) did not reveal any effect in SB patients.[23] Furthermore, the finding of a subtle side-to-side asymmetry but normal striatal dopamine binding in young SB patients suggests no early nigrostriatal degeneration as seen in patients with Parkinson's disease.[24] Propranolol, a catecholamine beta-adrenergic receptor blocker, has been reported to reduce SB. However, this finding was based on a comparison of treatment effects made several months after the baseline measurement in only 1 SB patient.[25] In addition, the administration of selective serotonin reuptake inhibitors (eg, fluoxetine, sertraline, fluvoxamine, and paroxetine) has been associated with reports of tooth clenching or tooth grinding during sleep in the absence of quantitative recordings.[26] In the absence of controlled double-blind studies, it is difficult to draw conclusions about the specific roles of propranolol or serotonin inhibitors in SB pathophysiology. It should be noted that the above neurochemical substances have a broad spectrum of influence on mechanisms related to sleep-wake regulation, orofacial sensory/motor activity, and autonomic functions.[19,20,27,28]

The role of the autonomic nervous system in SB pathophysiology is not clearly understood. In an open study, 64% of SB patients were reported to have abnormal responses to autonomic function tests during wakefulness but other researchers have not been able to reproduce this observation.[5,29–31] However, in response to a simple reaction task during wakefulness, polygraphically diagnosed SB patients showed similar changes in heart rate compared to normal subjects.[31] Although SB is associated with an arousal-related heart rate increase, several controlled polygraphic studies failed to show an abnormality in heart rate measures during sleep.[7,32,33] Overall, SB is not likely to be a primary disorder of the autonomic nervous function, and more detailed analyses (eg, heart rate spectral analysis) are needed to clarify the influence of the balance between the sympathetic and parasympathetic components of the autonomic nervous system in relation to SB.[33.34]

A psychologic response to stress has been thought to facilitate SB genesis but little physiologic information is available. Several case studies have suggested a positive association between stress and masseter electromyographic (EMG) activity during sleep[35]; however, when masseter muscle activities were recorded during sleep for 15 nights, self-reported daily stress was positively correlated with these activities in only 8 of 100

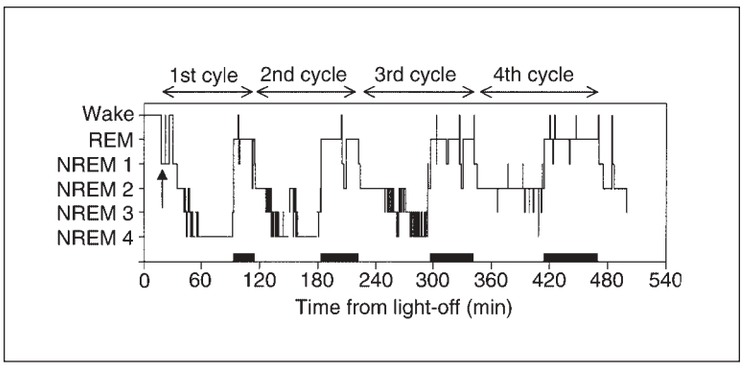

Fig 2 Sleep histogram (hypnogram) of a 23-year-old female normal subject. This hypnogram shows the time course of sleep (eg, sleep stages/sleep cycles) across the night. Sleep started *(arrowhead)* several minutes after the light was turned off. In this subject, 4 sleep cycles were repeated across nights. The duration of non-rapid eye movement (NREM) sleep stages 3 and 4 (deep sleep) decreases from first cycle and that of rapid eye movement (REM) sleep (thick bars) increases toward the fourth one.

subjects.[36] In a psychophysiologic reaction task study performed during wakefulness, SB patients reported higher levels of anxiety concerning test performance than normal subjects.[31] Clearly, further studies are needed to demonstrate the roles of psychologic aspects in SB pathophysiology.

Several studies have reported SB in patients with chronic pain, sleep apnea, and insomnia related to anxiety-related stress. It is well known that these medical conditions alter sleep microstructure and macrostructure and sensory perception. In studies where EMG activity was recorded during sleep in SB patients with orofacial pain complaints, in comparison to those without pain, the number of SB episodes and/or the amplitude of masseter EMG activity were significantly lower in the former group.[37,38] Sleep apnea is another condition characterized by numerous sleep arousals (eg, micro-arousals, awakenings).[39] Concomitant SB is reported in some patients with obstructive sleep apnea (OSA),[40,41] but time sequence analysis between RMMA-tooth grinding episodes and apnea events fails to reveal a clear association between the two.[41,42] This suggests that in sleep apnea patients, sleep arousal related to SB motor events is not time-related to either the instability of respiratory control that occurs in central sleep apnea or with the decrease in upper airway muscle tone that occurs in OSA.[43–46] Sleep bruxism has also been reported in patients with insomnia,[47] but the specificity of the association is questionable since insomnia is a condition often associated with anxiety, depression, and stress.[48–50]

In view of the emphasis in the SB literature on occlusal factors, psychologic influences as well as factors related to motor and sleep neurophysiology, and oropharyngeal obstruction, the focus of this review is on the sensory influences that may occur during sleep and that could be involved in the genesis of SB.

Sleep Processes

Sleep is a very different physiologic condition compared to wakefulness. In the dental community, however, this is often overlooked in discussions of the pathophysiology of SB. The authors therefore provide a summary of most relevant information on sleep physiology.

Sleep Macrostructure

Sleep usually lasts 7 to 9 hours in adults and about 30 minutes less in the elderly.[51,52] We usually sleep to recover energy loss from waking activity; it is probable that sleep also contributes to our mental health. It is widely recognized that lack of sleep, or frequently interrupted sleep (eg, fragmentation), triggers low daytime performance, poor vigilance, loss of memory, etc.[51–53]

Sleep is divided into non-rapid eye movement (NREM) and rapid eye movement (REM) cycles (Fig 2).[51,54] NREM sleep is further classified into 4 stages, from light (stages 1 and 2) to deep (stages 3 and 4) sleep. A normal subject usually falls asleep within 10 to 20 minutes to progress into NREM sleep stage 1, then to stage 2, and next to deeper stages 3 and 4, and returns into a brief period of light sleep before reaching REM sleep. NREM and REM sleep alternate cyclically with a period of about 90 minutes across the night (sleep cycles; see Fig 2). These cycles are repeated 3 to 5 times across a typical night. In the first third of total sleep, the duration of stages 3 and 4 is the longest and decreases or disappears in the last third. REM sleep duration is completely opposite: it increases toward the last third of sleep (Fig 2).[51,54]

Stage 1 NREM sleep is usually observed in the minutes following sleep onset and can reappear through the night in relation to transitional stage periods or body movements (eg, SB); this stage is

Table 1 Sleep Microstructure Variables in NREM and REM Sleep

Phasic events	Sleep stages	Characteristics
K-complex	NREM stage 2 (sometimes in stages 3 and 4)	Bi-triphasic EEG complex, consisting of an initial negative and successive slow waves (duration: > 0.5 s; amplitude: > 75 μV; density: 1–5/min)
Sleep spindle	NREM stage 2 (sometimes in stages 3 and 4)	Sequences of sinusoidal and fusiform waves at 12 to 14 Hz (duration: 0.5 to 2.0 s; amplitude: > 5 μV; density: 3–10/min)
Delta burst	Prevalent in NREM stages 3 and 4 (but clearly observed in stage 2)	Sequence of 2 or more slow waves (0.5 to 4 Hz) with a voltage > 100 μV or of at least 1/3 greater than that of background activity
Saw-tooth waves	A few seconds before or during REM sleep	Trains of 3 or more angular EEG waves (2 to 5 Hz); amplitude: 20 to 100 μV
Sleep arousals	NREM and REM	An abrupt EEG desynchronization • with alpha (8 to 12 Hz)/beta (12 to 14 Hz) activity and/or frequencies more than 16 Hz in NREM • with duration of > 3s; can be accompanied by increase in muscle tone and/or body movement
Micro-arousal		An arousal lasting more than 3 to 10 or 15 s (the half of the scoring epoch)
Awakenings		An arousal lasting more 10 or 15 s (half the scoring epoch); scored as wake
Sleep stage shift	NREM and REM	Rapid changes from deeper to lighter sleep stage
Cyclic alternating pattern (CAP)	NREM stages 1 to 4	Sequences of EEG activity that repeat cyclically; prevalent in NREM sleep; CAP cycle may last from 2 to 60 s
Phase A (A1-A3)		Associated with micro-arousals, either isolated or associated with slow waves: • A1: synchronized EEG pattern • A2: desynchronized EEG pattern preceded by high voltage slow waves • A3: desynchronized EEG pattern exceeding by at least 2/3 of the length of the entire phase A
Phase B		Quiet background EEG activity

characterized by low-voltage cortical electroencephalographic (EEG) activity. Stage 2 occupies more than 50% of the total sleep time; several phasic EEG patterns (eg, K-complex, sleep spindles) characterize this stage.[52,55] Sleep stages 3 and 4 occur for 15% to 20% of the total sleep time and are associated with the sleep "recovery" effect; stages 3 and 4 are characterized by the presence of high-voltage slow wave activity (75 μV, < 4 Hz). REM sleep occupies 20% to 25% of total sleep time and is the sleep period with the most vivid dreams; it is characterized by low-voltage cortical EEG activity with bursts of rapid eye movement, an increase in heart rate, sexual organ tumescence, and muscle hypotonia (muscle tone is reduced to a nearly paralyzed state). Interestingly, autonomic nervous system activity (eg, cardiac, respiratory activation) is also altered during sleep. As sleep becomes deeper from stages 1 to 4 of NREM sleep, sympathetic activity decreases and parasympathetic activity increases (heart rate becomes less variable).[34] During REM sleep, sympathetic activity is sometimes even higher than during wakefulness. Since REM sleep is dominant during the early morning hours (in the last third of the total sleep period), it has been proposed that there is a higher risk of cardiac arrest occurring during sleep in this period.[34]

Sleep Microstructure and Arousal

The microstructure of sleep consists of a series of physiologic events that are present within a given sleep stage (Table 1).[52,55] For example, in sleep stage 2, EEG events (eg, K-alphas, K-complexes, or single K) are observed 1 to 3 times per minute and help to preserve sleep continuity under exogenous (eg, noise) or endogenous (eg, fluctuation in blood pressure) influences.

Sleep arousals are classified as micro-arousals or awakenings. Sleep micro-arousal is defined as a transient and brief cortical, autonomic (cardiac), and motor activation in the absence of consciousness.[52,56,57] Micro-arousals are thought to be a physiologic adjustment to environmental and endogenous influences; they are more frequent at the end of a sleep cycle when sleep becomes lighter.[46,52,58] Awakening is an arousal activity lasting more than half of a scoring epoch (10 or 15 seconds) and could be associated with a sleep stage shift (eg, from deeper to a lighter stage).[46,59]

Table 2 The Role of Orofacial Sensory Inputs on Jaw Motor Activity During Wakefulness and Sleep

	During wakefulness	During sleep	
		NREM	REM
Afferent activity			
Skin, mucosa	Mechanoreception, thermal, nociception: ↑ by stimuli during mastication	↓	↑ or ↓
Tooth pulp	Nociception: ↑ by noxious heat stimuli	↓	↓↓
Periodontal ligament	Mechanoreception: ↑ by tooth (food) contacts during mastication ↑ by stimulus force or direction	?	?
Muscle			
Muscle spindles	Proprioception (Groups Ia and II): ↑ in closing phase during mastication ↑ by passive jaw muscle stretch	? (↓ in limb muscles)	?
Golgi organs	Proprioception (Group Ib): ↑ by passive jaw movement	?	?
Groups III and IV	Mechanoreception or nociception ↑ by passive jaw movement	?	?
Temporomandibular joint	Proprioception and mechanoreception ↑ by passive muscle movement	?	?
Reflex activity			
Jaw-opening reflex	Induced by stimuli applied to skin, mucosa, tooth pulp, and periodontal ligament		
	Low-threshold type: ↓ during closing in mastication → during opening in mastication	↓ or →	↓
	High-threshold type: ↑ during closing in mastication ↓ during opening in mastication	?	?
Jaw-closing reflex (H-reflex, stretch reflex)	Induced by stimuli related to muscle spindle or periodontal afferent responses: ↑ during closing in mastication ↓ during opening in mastication	↓ or →	↓

Increase = ↑; decrease = ↓; no change = →; unknown = ?.

The cyclic alternating pattern (CAP) represents a condition of sleep instability that protects and regulates the macrostructure of sleep. It is dominant in non-REM sleep in which clusters of cortical, autonomic, and motor activities are repeated every 20 to 60 seconds.[46,60]

Conclusion

The macrostructure and microstructure of sleep represent the substrate for sleep, and the sensory inputs that can influence sleep arousal operate within the context of the homeostatic balance between wakefulness and sleep. The following sections consider those sensory influences that originate from orofacial structures.

The Orofacial Sensory System, Oromotor Activity, and Sleep

Although oral health professionals have for decades associated peripheral morphologic factors (eg, occlusal interferences) with the genesis of SB (eg, initiation of SB), very little attention has been given to the physiologic aspects of sensory inputs on SB or RMMA during sleep. The role of orofacial sensory inputs in jaw motor activity has been extensively investigated in numerous animal and human studies. However, there are little data showing how these afferent inputs influence the oromotor system in sleep. The focus of this section is to review orofacial sensory factors and their potential physiologic influence on SB genesis.

Facial Perioral Skin, Oral Mucosa, and Dental Afferents

Skin and Mucosal Afferents. Primary afferents that innervate facial/perioral skin and oral mucosa (eg, lip, tongue, palate, and gingiva) convey tactile, thermal, and nociceptive sensory information (Table 2). In humans, most facial and lip afferents are of the slowly adapting type whereas those of the tongue are mainly rapidly adapting.[61,62] The latter afferents discharge during jaw movements

(usually jaw closure), in response to either pressure from a food bolus and/or deformation of their receptive fields, and to contacts between 2 oral parts (eg, tongue and lip, upper and lower lips).[61–64]

The influence of sensory inputs from facial and perioral skin and from oral mucosa on the jaw motor system has been studied in humans and animals. The excitatory and inhibitory influences on the jaw motor system are thought to depend on the stimulus modality, stimulus intensity, background motor excitability, and location of the applied stimuli.[65–73] In general, low-intensity (non-noxious) stimulation that activates low-threshold afferents produces excitatory effects on jaw-closing muscles whereas activation of high-threshold afferents by high-intensity (noxious) stimulation inhibits jaw-closing muscles (also see sections on jaw-opening reflex).[64] Stimulating skin and mucosal afferents in humans and animals also modulates rhythmic jaw movements.[74–77] Thus, cutaneous and mucosal afferents from the orofacial region provide sensory information for coordinating movements of facial and oral structures during mastication, swallowing, and speaking.

When conscious tactile stimulus detection and spatial resolution of the lower lip was recently compared between patients, with or without an awareness of SB, no difference was found between groups. This result suggests that perioral sensory acuity (eg, 2-point discrimination) is normal during wakefulness in subjects with an awareness of tooth grinding during sleep.[78] During wakefulness, patients with OSA showed an impaired sensory function (eg, vibratory detection) of the mucous membrane in the upper airway and a reduction in genioglossus muscle tone following anesthesia of nasopharynx topical receptors, indicating the importance of the suprahyoid musculature in maintaining airway patency.[79,80] This emphasizes the importance of receptors in the upper airway which can respond to chemical, thermal, and mechanical stimuli and influence jaw and tongue position and thereby airway patency.[81–83] To our knowledge, intraoral and upper airway mucosal sensory influences on jaw and upper airway muscles have not been tested in SB patients.

In the case of the influences of these various afferents during sleep, cutaneous (ie, skin) sensory inputs from the infraorbital region (division II of the trigeminal nerve) to the trigeminal sensory nucleus complex (TSNC) of the brainstem in animals are facilitated during quiet sleep (equivalent to NREM sleep in humans) but are suppressed during active sleep (equivalent to REM sleep in humans) (Table 2).[84] When compared to wakefulness, the response of TSNC neurons to electrical stimulation of the inferior alveolar nerve (trigeminal nerve division III containing periodontal, tooth pulp, and lip cutaneous afferents) is also suppressed during active REM sleep but suppression is not obvious during quiet NREM sleep.[85,86] These observations support the idea that synaptic transmission is likely suppressed during REM sleep.[87]

Interestingly, sensory neurons projecting to the thalamus from the TSNC discharge differently during REM sleep: neurons innervating hair mechanoreceptors around the face show facilitated responses to air-puff stimulation but those that respond to tooth pulp stimuli are suppressed.[88] Since in animals this type of stimulus and response may be more relevant for bodily protection and survival, different types of sensory information may be processed differently based on their biologic relevance during sleep.[89,90] This was also observed in human experiments using non-trigeminal sensory stimulation during sleep. Non-noxious vibrotactile stimulation applied to the arm was found to induce more awakening in light NREM sleep only. Following experimental muscular pain, however, the awakening response was preserved in all sleep stages.[91] In another study, cutaneous heat pain induced more frequent sleep arousals than non-noxious warm and cold stimulation.[92]

Periodontal Afferents. In the periodontal ligament, afferents convey nociceptive and tactile sensory information. The latter is especially subserved by rapidly and slowly adapting mechanoreceptors that respond to the mechanical stimulation (eg, displacement, pressure) of the teeth (Table 2). The rapidly adapting receptors fire abruptly after tooth contact associated with food chewing, while the firing frequency of the slowly adapting receptors increases as the forces applied to the teeth increase.[64,93,94] In addition, the afferent fibers from the rapidly adapting receptors have higher thresholds compared to those of slowly adapting receptors.[93]

The excitatory and inhibitory influences of periodontal afferents on jaw muscle activity have been studied in both humans and animals.[64,95–98] When activated by the insertion of chewing objects (eg, steel ball, plastic strips)[99,100] or an unexpected mechanical load,[101] low-threshold–type afferents are thought to facilitate jaw-closing muscle activity through a positive feedback loop. The activation of high-threshold afferents, on the other hand, suppresses jaw-closing muscles and activates jaw-opening muscles in humans and animals; this has been considered as a protective mechanism to guarantee the integrity of the oral tissues.[99,102–105]

There has been only the 1 sleep study noted above that investigated the response of TSNC neurons to electrical stimulation of the inferior alveolar nerve, which contains periodontal nerve afferents.[85] Here the response of TSNC neurons was found to be lower in REM sleep when compared to NREM sleep and wakefulness. From this indirect information, however, it is difficult to extract definitive information on the influence of periodontal afferents on jaw muscle activity during sleep in relation to SB in humans.

Afferents from Muscles

Muscle sensory organs inform the brain about muscle length, displacement velocity, and nociception (Table 2). Their associated afferents are classified into several groups (Ia, Ib, II, III, IV) according to the afferent fiber diameter.[64,97,106] Within the jaw musculature, muscle spindles are distributed mainly in the jaw-closing muscles (eg, masseter, temporalis, and medial pterygoid muscles) while none or very few are distributed in the jaw-opening (eg, digastric) muscles.[107]

The larger afferents, groups Ia and II, innervate the muscle spindles. Group Ia afferents have primary endings that are mainly responsive to the velocity and acceleration of muscle stretch whereas group II afferents mainly respond to the length of muscle stretch.[108,109] The sensitivity of these muscle spindle afferents to muscle stretch is controlled by the activity of the fusimotor (gamma motor) system.[106] The muscle spindle afferents are most active during jaw movement and voluntary jaw-closing muscle contraction.[110–113] They have also been reported to facilitate jaw-closing muscle activity during experimental chewing in animals.[100,114–116]

The group Ib afferents are also muscle afferent fibers but their role in the masticatory muscles has not been clearly documented.[64,117,118] These types of afferents convey information about muscle tension that is sensed by Golgi tendon organs. They are reported to be excited by muscle stretch and by jaw muscle contraction during mastication. The last groups, III and IV muscle afferents, have been reported to carry sensory information from mechanosensitive and nociceptive receptors. The group III muscle afferents are reported to show a response to jaw muscle stretch similar to that of muscle spindle afferents. Interestingly, some recent studies have revealed that activation of muscle nociceptive afferents suppresses agonistic muscle activity but facilitates antagonistic muscle activity when muscle pain is induced although others have indicated that both muscle groups (jaw opener and closer) may be activated simultaneously in certain conditions.[119–122] A decrease in movement speed and amplitude is observed both in humans and animals. Experimental muscle pain also reduces the jaw-closing muscle activity during chewing in humans and the frequency of rhythmic muscle bursts in anesthetized animals.[123–125]

There have been no studies of jaw muscle afferents during sleep. However, in the limb muscles of cats, the discharge of muscle spindle afferents is tonically suppressed during NREM sleep and substantially decreased in REM sleep.[126] A transient facilitation of discharge during REM sleep can occur spontaneously, however, with or without simultaneous muscle twitch, probably due to transient activation of gamma motoneurons.[126,127] When transient sleep arousals were induced by experimental pinch stimulation, some of the gamma motor fibers exhibited a phasic increase in their firing frequency.[127]

Afferents of the Temporomandibular Joint (TMJ)

The temporomandibular joint afferents that innervate the joint capsule are sensitive to small displacements and movements (eg, opening, laterotrusive, and protrusive jaw movements)[117,129] and mechanical or chemical stimuli (Table 2).[122,130,131] However, these afferents do not discharge at extreme jaw positions.[117] This suggests that these receptors are important for proprioceptive jaw movement control. It has also been reported that masseter motor nerve activity is suppressed when the TMJ condyle is pressed against the joint capsule in cats.[129] Only a few studies have investigated the role of TMJ afferents on jaw motor control[64,97,121,122,132] and, to our knowledge, no study has been made during sleep.

Trigeminal Reflexes

Jaw-opening Reflex (JOR). The JOR is a very rapid di-synaptic motor response associated with the protection of oral tissues (Table 2). In animals, it can be evoked by stimulation of orofacial low-threshold or high-threshold mechanoreceptors (eg, lip and oral mucosa), electrical stimulation of the trigeminal nerve (eg, inferior alveolar nerve), and mechanical impact on teeth.[64,93,97,99,104,133] The inhibition of jaw-closing motoneurons and the facilitation of jaw-opening motoneurons has been extensively studied in animals but its role in humans is less clear.[64,76,93,134,135] In animals, when the JOR is triggered during chewing by the stimulation of low-threshold mechanoreceptors (eg, non-painful

stimuli), the reflex is inhibited during the closing phase but is preserved during the opening phase.[64,133,136] In contrast, when the JOR is induced by stimulation of high-threshold mechanoreceptors (eg, using high intensity of electrical stimulation), it is facilitated during the closing phase and inhibited during the opening phase.[64,105] The latter reflex system may also play an important role during chewing by protecting oral structures from damage (eg, tooth fracture, oral mucosa biting).[64,105] Its role in reducing jaw-closing muscle activity during jaw closure is more evident if a person bites either their tongue or an unexpected hard object in food.

Although in cats the amplitude of the JOR has been reported to be facilitated during NREM sleep when compared to wakefulness,[137] the opposite finding was reported recently in rabbits (Table 2).[138] However, in REM sleep, the JOR was strongly reduced in both studies.[137,138] Although observations in human experiments using electrical stimulation to trigger a leg flexion reflex have shown that the reflex threshold was increased in NREM sleep compared to wakefulness and was maximal in REM sleep, no direct information is available for the JOR in humans.[137]

Lateral Jaw Reflex. In anesthetized animals, the lateral jaw reflex occurs in response to tonic mechanical stimuli of periodontal mechanoreceptors, which enhances lateral pterygoid muscle activity, leading to the large lateral jaw excursions observed during the slow closing phases of chewing.[99,114,139,140] Although its relevance in humans is not as clearly defined, it has been observed that larger lateral jaw movements, related to an increased duration of the jaw-closing muscle bursts and chewing cycle, are present when both humans and animals chew hard foods.[100,114,141–144]

Jaw-closing Reflex. In awake humans, a monosynaptic reflex of the jaw-closing muscles is elicited by a mechanical chin tap that stretches muscle receptors (stretch reflex) or by direct electrical stimulation of muscle spindle afferents in the trigeminal nerve (H-reflex), or by mechanical or electrical activation of periodontal receptors.[64,81,97,128,145–147] The amplitude of these reflexes depends on the excitability of the trigeminal alpha and gamma motoneurons and the amount of muscle spindle afferent input to the alpha motoneurons. These monosynaptic reflexes are increased during the jaw-closing phase of rhythmic jaw movements and during tooth-clenching tasks (Table 2).[148–151] In animals, the monosynaptic masseteric reflex (MMR) is evoked by electrical stimulation of neurons in the trigeminal mesencephalic nucleus where the primary afferent cell bodies of jaw muscle spindle afferents are located. This reflex activity is increased during the jaw-closing phase and inhibited during the jaw-opening phase of mastication.[136]

To our knowledge, there have been no studies on trigeminal monosynaptic reflex modulation during sleep in humans, perhaps due to the invasive technique required for nerve stimulation and problems associated with stability of the stimulation set-up (eg, electrode displacement in response to body movements during sleep). In animals, the amplitude of the MMR did not significantly decrease from wakefulness to NREM sleep, and was markedly suppressed during REM sleep.[129,138] However, there are controversial findings that the amplitude of the MMR is facilitated or suppressed in relation to rapid eye movement episodes during REM sleep.[129,138]

Orofacial Sensory Inputs Triggering Rhythmic Jaw Muscle Activity

Several animal studies have reported that rhythmic jaw muscle activity is observed following peripheral sensory stimulation applied to oral mucosal mechanoreceptors.[64,152–154] It was also reported that under light anesthesia, both tonic mechanical stimulation of the palate and specific directional pressure applied to the incisor teeth elicited rhythmic jaw muscle activity in rats.[155,156] To the authors' knowledge, these types of experimental responses do not occur in humans, and so their relevance to SB is difficult to determine.

Conclusion

Several animal studies have shown either facilitation or inhibition of the trigeminal peripheral orofacial sensory factors and/or related reflexes in relation to sleep but their relevance to SB genesis has not yet been elucidated in humans. Clearly, further studies on orofacial sensory feedback mechanisms in relation to sleep are needed.

Jaw Muscle Tone, Mandibular Posture, and Tooth Contact During Sleep

When discussing the relevance of peripheral sensory factors in SB genesis (including tooth contact), the classic question arises: "Which occurs first, tooth contact or jaw muscle activation?" The following section is focused on the putative role of muscle tone and mandibular posture during sleep in SB.

Muscle Tone and Mandibular Posture

Generally, during sleep, skeletal muscles are relaxed and muscle tone is lower when compared to wakefulness.[6] Similarly, masticatory muscle tone decreases from wakefulness to NREM sleep to a similar level between sleep stages 1 to 4.[7,8] During REM sleep, muscle tone is normally minimal due to the powerful motor suppression, also termed "muscle atonia" to reflect the complete loss of muscle tone.[6,7,20,157] However, masticatory muscle activity does not disappear completely during REM sleep; very low residual muscle tone may persist, suggesting that the term "hypotonia" would more accurately describe the state of masticatory muscle tone during REM sleep.[7,157]

With the aid of intraoral devices that measure the vertical distance between the maxillary and mandibular incisors, it has been demonstrated that during sleep the jaw is usually open 1 to 5 mm for approximately 90% of the total sleep time.[158] This distance can be significantly greater during REM sleep when compared to the lighter non-REM sleep stages (eg, stage 1). Interestingly, a sleep arousal reaction precedes an increase in jaw muscle tone (likely associated with jaw closure) during the sleep of normal subjects, sleep apnea, and SB patients.[7,31,158,159] These observations suggest that tooth contact is most likely to be a final outcome in jaw closure secondary to the jaw muscle activation during sleep.

The use of ambulatory systems to record masseter EMG activity has revealed that edentulous subjects also show RMMA during sleep even in the absence of dentures in the mouth.[160] This finding further supports the idea that jaw motor activation during sleep can occur in the absence of tooth contact and feedback from periodontal receptors.

Jaw Motor Events and Tooth Contacts

There are several studies reporting data on the duration and frequency of jaw motor events in normal and SB patients (Table 3).[11,12,161–170] The jaw motor events, scored as SB, were observed more frequently in SB patients (3.6 to 6.8 episodes per hour of sleep) than in normal subjects (0.5 to 5.6 episodes), and the total duration of these events per hour of sleep was longer in SB patients (0.6 to 1.65 minutes) compared to normal subjects (0.12 to 0.89 minutes). On the other hand, only a few studies have directly measured tooth contact during sleep (Table 3).[171–176] These studies used intraoral sensors to assess tooth contact but no recordings were made of quantitative sleep variables. One study reported that SB patients with a temporomandibular disorder (TMD) showed significantly higher frequency and duration of tooth contacts compared to normal subjects and patients with TMD.[176] Several techniques allow indirect assessment of the number of tooth contacts in relation to SB-related tooth grinding (Table 3).[10,11,170,177] The results from the polygraphic studies using an audio monitor reveal that the incidence of tooth grinding-related noise is more frequently observed in SB patients. From these results, we can summarize that SB patients have approximately a 3 to 4 times higher number and duration of SB episodes and tooth contacts compared to normal subjects. In addition, the presence of a tooth grinding noise seems to be characteristic of SB patients.

When interpreting these data, however, the reader should consider a number of limitations that may account for discrepancies between study results: *(1)* The different methods used to record SB and tooth contact (eg, polygraphic versus ambulatory recording systems, EMG of masticatory muscles versus mechanical impact on sensors to score motor events). *(2)* Standardized cut-off criteria for normal subjects and SB patients were not used; this creates heterogeneity in the study sample. *(3)* Different scoring criteria for SB episodes (eg, EMG threshold, muscle burst patterns) were used by the research groups, with no clear consensus on the definition of an SB episode. As an aside, our group has recently carried out a post-hoc analysis for EMG threshold, which we previously proposed as more than 20% of maximum voluntary contraction (MVC) of masseter muscle activity during wakefulness. A retrospective analysis of the 20% criteria used has shown, in reality, that some SB events were also scored in the range between 10% and 20% MVC. Thus, in our recent studies, we used 10% to 20% of MVC as a minimum EMG threshold, similar to another study.[11,162] *(4)* Finally, several studies have reported that the patterns of jaw muscle contractions (eg, in closer and opener muscles) and jaw movements related to SB are not the same as those found in awake chewing in humans.[7,14,20,32,166,178,179] This suggests that tooth contact related to SB tooth grinding may take a different form from that occurring during wakefulness.[180–184]

Conclusion

During sleep, the lower jaw tends to be open due to jaw-closer muscle relaxation. Following a sleep arousal, it is frequently observed that an increase

Table 3 SB and Related Variables: EMG Events, Tooth Contacts, Tooth Grinding Sound

Author	Sample (age/range or mean)	Frequency†	Duration†	Recording system	Thresholds for SB episode	Criteria for SB/ normals‡
SB-related jaw motor events						
Gallo et al[164]*	21 normals (31;22–37)	10.2	0.8 min	Ambulatory EMG recording	Algorithms	1,2,3
Okeson et al[161]*	20 normals (42.5;21–58)	5.6	0.6 min	PSG	> 40% MVC	—
Okeson et al[165]*	30 normals (70.2;60–87)	3.0	0.3 min	PSG	"	—
Clarke et al[168]*	10 adults	4.9 (0.3–11.6)	0.7 min (0.1–2.3)	Ambulatory EMG recording	> 2-second duration	—
Amemori et al[166]*	2 SB patients and 1 normal (26–32)	8.2 (4.5–10.9)	1.8 min (0.8–2.9)	PSG + jaw movement recording	> 5% MVC	1,2
Ikeda et al[162]*	9 SB patients (26.2)	3.6	0.6 min	Ambulatory EMG recording	> 10% MVC	2,3
Pierce and Gale[169]	100 SB patients (18–72)	16.7 (1.4–67.6)	0.03–2.6 min	Ambulatory EMG recording	> 20 μV	1,2,5
Nishigawa et al[167]*	5 normals (29.6;26–36) 5 SB patients (27.6;23–33)	3.5 (0.3–11.5) 3.5 (0.5–7.5)	0.4 min (0.02–1.4) 0.4 min (0.03–0.9)	Ambulatory bite force recording with full arch oral appliance	> 5 kgf	1,2,3
Kydd and Daly[163]*	10 normals (21–43) 10 SB patients (21–43)	— —	0.4 min (0.4–0.6) 1.4 min (0.4–2.0)	Ambulatory EMG recording	Unknown	1,2
Sjöholm et al[12]	6 normals (26.3;17–36)	1.9	0.9 min (0.3–1.4)	PSG	> 20% MVC	1,2,3
Lavigne et al[11]	31 normals (27.6;15–40) 33 SB patients (26.8;20–50)	1.8 (0.1–12.6) 5.8 (1.2–15.2)	— —	PSG with audio-video	> 10/20% MVC‡	1,2,4
Miyawaki et al[170]	7 normals (25.3) 9 SB patients (23.2)	0.5 6.8	0.12 min (0.05–0.25) 1.15 min (0.68–1.9)	PSG with audio-video	> 10/20% MVC‡	1,2,4
Tooth contact						
Powell and Zander[171]	7 normals (34–56)	29.8 (6–140)	—	A sensor in fixed bridge	/	1,2,3
Graf[172]	1 normal	—	0.16 min	A strain gauge on the molar	/	—
Brewer[173]	Dental patients	—	(0.38–18.8 min)	Radio transmitter in denture	/	—
Yamashita et al[174]*	3 normals (26–31)	40.7 (25.7–65.1)	0.8 min (0.3–1.5)	Micro-photo sensor in metal attachment on dentitions	/	—
Akamatsu et al[175]*	2 SB patients (26 and 27)	52.4 (10.3–94.5)	0.9 min (0.5–1.3)	Magnetic sensor in metal attachment on upper/lower dentitions	/	1
Trenouth[176]*	10 normals (21.4;20–28) 9 SB patients with TMD (21.7;16–36) 6 TMD patients (21.5;15–35)	45 165 125	0.68 min 4.8 min 1.4 min	An electric circuit consist with wires placed on the incisors	/	1,2,3
Tooth grinding noise incidents						
Reding et al[177]*	18 normals (24;17–56) 40 SB patients (20.6;7–50)	0 5.8	0 0.34 min	PSG with microphone	> 40 μV	1
SB episodes with grinding noise						
Lavigne et al[10]*	18 normals 18 SB patients	0 0.97	— —	PSG with audio-video	> 10/20% MVC§	1,2,4
Lavigne et al[11]*	31 normals (27.6;15–40) 33 SB patients (26.8;20–50)	0 1.9	— —	PSG with audio-video	> 10/20% MVC§	1,2,4
Miyawaki et al[170]	7 normals (25.3) 9 SB patients (23.2)	0.1 (0–0.3) 3.4 (0.1–10)	0.03 min (0–0.07) 0.62 min (0.01–1.54)	PSG with audio-video	> 10/20% MVC§	1,2,4

*The numbers were estimated from the data presented in the article.
†Frequency and duration [mean (range)] were presented as times per hour of sleep and minutes per hour of sleep, respectively.
‡Selection criteria: 1 = a history/awareness of tooth grinding; 2 = clinical signs of SB including tooth wear and morning jaw muscle discomfort and masseter muscle hypertrophy; 3 = clinical signs of temporomandibular disorders; 4 = polysomnographic criteria; 5 = ambulatory criteria.
§Ten percent of SB episodes were between 10% and 20% of MVC range (see text).
PSG = polysomnography; MVC = maximum voluntary clenching during wakefulness; TMD = temporomandibular disorders.
/ = unavailable or unknown.

in jaw muscle activity may lead to the jaw closure. Thus, tooth contact seems to be the consequence of jaw motor activation during sleep rather than a cause. It has also been clearly shown that jaw muscle EMG events and tooth contact occur more frequently in SB patients than normal subjects. This observation is supported by polysomnographic studies that demonstrate more frequent tooth grinding episodes in SB patients in comparison to normal subjects.

Sleep Processes and SB

This section aims to clarify the association of SB with sleep processes.

Sleep Macrostructure and SB

Interestingly, most young and healthy SB patients exhibit a normal sleep structure and usually do not complain of sleep disturbance unless they have either concomitant chronic pain and/or other sleep influencing disorders (eg, sleep breathing disorders).[2–5,185] In contrast to an original report that

was later refuted,[186,187] and to studies describing cases of secondary SB,[46,188–190] most of the oromotor activity related to SB occurs in stages 1 and 2 of NREM sleep (60% to 85%). Very few SB episodes occur in sleep stages 3 and 4 of NREM sleep (< 5%), while on occasion SB is observed in REM sleep (< 10%).[10,13,177,191–194] The occurrence of SB episodes in REM sleep is an interesting paradox since this sleep stage is usually characterized by muscle hypotonia. So far, the only explanation given for the occurrence of SB in REM sleep in patients without sleep disorders is the presence of brief and transient sleep arousal activity that can occur during this sleep stage.[6,46,56]

Sleep Microstructure, Micro-arousal, and SB

Previously, SB tooth-grinding episodes were reported to be associated with K-complexes in SB patients.[176,193] However, in a recent controlled study, K-complexes were found to be associated with only 12% of RMMA episodes in SB patients and with 21% in normal subjects.[185] Moreover, SB patients were found to present 42.7% fewer K-complexes during sleep compared to normal subjects.[184] Although the number of sleep spindles did not differ between SB patients and normal subjects, SB episodes were not associated with sleep spindles.[175,185]

Since an episode of SB lasts anywhere from 5 to 15 seconds, it can be considered a transient motor event occurring during sleep (Table 4).[11,12,32,161–163,177,190,195] As previously described, SB patients show approximately 3 times more RMMA episodes, twice as many muscle bursts, and 40% higher EMG burst amplitudes during sleep than normal control subjects.[11,12] These findings suggest that SB is an exaggerated transient motor (muscle) activity occurring during sleep in an otherwise "normal" sleeper.

In most studies, young healthy SB patients display a normal incidence of micro-arousals, but the incidence lies in the upper range of the normal limit.[11,13,32,185] However, polysomnographic studies from as early as the 1960s have reported that SB episodes are associated with physiologic changes (eg, micro-arousals) that include transient EEG activity, increases in heart rate, and frequent sleep stage shifts (Table 5).[177,190,193,196,197] Furthermore, recent studies now support the association between SB and a transient EEG/autonomic activation. The first few of these studies have shown that nearly 80% of SB episodes are scored in association with a phase A3 of the cyclic alternating pattern (CAP).[13,60,198,199] Additional recent evidence supporting an association between SB and sleep arousals is derived from the observation that in both SB patients and normal subjects most of the RMMA episodes are preceded by suprahyoid muscle activation, an EMG sign of sleep micro-arousal.[11,32] Interestingly, several polysomnographic studies have also reported other specific physiologic changes (eg, cortical EEG change and heart rate acceleration) before the occurrence of an SB episode (Table 4). For example, changes in EEG frequency are observed before SB episodes and alpha EEG intrusion precedes 50% of SB episodes.[177,190,195–197] In addition, an increase in heart rate is followed by jaw-closer muscle activity.[177,195–197] When these changes in EEG activity and heart rate are measured in relation to the onset of RMMA episodes in SB and normal subjects, a transient sequence is found: *(1)* Sleep bruxism patients have a significant increase in EEG activity (alpha and delta EEG amplitude) approximately 4 seconds before the onset of an RMMA episode. *(2)* A significant increase in heart rate occurs 1 cardiac cycle before an RMMA episode in SB patients, but the overall time course from EEG activity and increases in heart rate related to RMMA onset are statistically similar in both SB patients and normal control subjects.[32] Thus, a sequence that starts with cortical EEG activation followed by heart rate acceleration precedes jaw-closer muscle activation, with the RMMA episode in SB being the final event in this sleep micro-arousal reaction (Fig 3). This finding has been supported by our experimental study in which RMMA occurred following experimentally induced arousals.[7] Furthermore, several studies have revealed that small and subtle changes in autonomic-cardiac heart rate variability can precede sleep arousals (> 10 seconds).[34,200] After applying frequency analysis on heart rate variability, our preliminary observations suggested that in the 3 minutes before RMMA episodes, SB patients showed a higher sympathetic and a lower parasympathetic activity compared to normal subjects.[33] The changes in EEG and EMG activity are probably under the influence of the autonomic nervous system fluctuation during sleep.

Several issues must be considered when interpreting the literature mentioned above:

1. In most studies, patients were monitored in a sleep laboratory setting, which is not a natural sleep environment, even though environmental habituation is done prior to recording.[9]
2. The analysis of autonomic (cardiac) activity was carried out in a time domain (beats per minute), which does not allow for the detection of subtle changes in sympathetic and parasympathetic components that could have preceded the EEG,

Table 4 Summary of Evidence that Micro-Arousal is Related to SB

Author	Sample (age/range or mean)	SB episode duration	Heart rate acceleration % increase	EEG change	Motor activation
Takahama[196]	7 SB patients	/	Increase during episode	Change before episode	Wrist/masticatory muscles activity before episode
Tani et al[197]	3 SB patients (13–25)	/	Increase before episode	Theta, alpha, beta before episode	/
Reding et al[177]	40 SB patients (20.6; 7–50) 18 normals: not described (24.0;17–56)	9.0 s (2.7–66.5)	4 beats before episode (26.8%; 2.0–68)	Low amplitude and high frequency EEG before episode	24% with body movement
Satoh and Harada[193]	15 SB patients (19–48)	/	Increased	Associated with alpha activity and K complex	/
Kydd and Daly[163]	10 SB patients (21–43)	/	Increased 18 beats/min (2%–30%)	/	Never
	10 normals (21–43)	/	Increased 18 beats/min (2%–30%)	/	Never
Okeson et al[161]	20 normals (42.5; 21–58)	6.7 s	(16.6%; 6.1–40.2)		57.7% with leg jerk
Ikeda et al[162]	9 SB patients (26.2)	10 s	(10%–14.9%)	/	/
Okura et al[195]	5 SB patients (33.8; 25–48)	9.1 s	4 to 8 s before episode	Beta and delta activity before episode	/
Bader et al[190]	24 SB patients (35; 27–67)	6.3–11.1 s	At the onset of masseter burst (17.6%)	EEG alpha activity before episode	/
Macaluso et al[13]	6 SB patients (34) 6 normals (34): not described	/	(19.3%)	87.3% during CAP	79.6% with leg jerk
Lavigne et al[11]	8 SB patients (27.6)	9.4 s	/	/	78.6% preceded by jaw-opener activation
	8 normals (26.8)	7.8 s	/	/	69.1% preceded by jaw-opener activation
Kato et al[32]	10 SB patients (26.5; 20–36)	14.9 s	1 heart beat before episode (26.7%)	4 s before episode	87.5% with neck and leg jerk
	10 normals (24.2; 18–41)	11.9 s	Statistically similar to SB patients (28.1%)	Statistically similar to SB patients	85% with neck and leg jerk

CAP = cyclic alternating pattern.
/ = unavailable or unknown.

electrocardiogram (EKG), and EMG activation.[34,201,202] This issue is currently being investigated in our laboratory.[33]
3. Tonic clenching activity (< 10% of all SB episodes) was not analyzed.
4. Sleep bruxism occurring in REM (< 10% of all SB episodes) is not well understood.
5. Most recent studies used only severe SB patients. It is not known whether the association between EEG, EKG, and jaw EMG activity is related to the severity (light to severe levels) of SB patients.
6. Studies were not population-based (ie, SB patients were mostly in their twenties and the influence of age on sleep architecture is already known, especially after the fourth decade[203]).

Conclusion

Although SB episodes occur frequently in young healthy SB patients, gross sleep architecture is undisturbed. Based on polysomnographic studies, SB seems to occur in relation to subtle changes in both sleep macrostructure (eg, in light non-REM sleep stages 1 and 2) and microstructure (eg, EEG-cardiac activation). These studies suggest that SB may be a masticatory muscle activation related to the exaggerated responses often found associated with sleep arousals. The observation that with sleep arousals a rise in EEG and cardiac activities precedes RMMA in SB patients further suggests a primary role for autonomic and central nervous systems in the genesis of SB.

Interaction Between Sensory Inputs, Sleep, and Oromotor Activity

Sleep architecture (eg, macrostructure and microstructure including arousal) can be altered by various sensory influences.[52,53,89] Therefore, interactions between sensory input, autonomic, and central nervous systems are important in sleep and wake regulation. In this section, the roles of various sensory influences in SB genesis will be reviewed.

Table 5 Studies of SB-Tooth Grinding and Occlusal Factors

Author	Sample*	Intervention or design	a: Selection criteria† b: Assessment of SB†	Outcomes and results	Critical comments
Epidemiologic or observational studies					
Nilner[209]	440 children (7–14; 222M, 218 F)	Oral examination, interview	a,b: 1	Correlation with OI	Cross-sectional analysis, subjective assessment
Nilner[210]	309 adolescents (15–18; 147 M, 162 F)	Oral examination, interview	a,b: 1	No correlation with OI	Cross-sectional analysis, subjective assessment
Brandt[211]	1,342 children (6–17; 667 M, 675 F)	Oral examination, interview	a,b: 1	Association with overjet, overbite, and mesiodistal molar relationships	Cross-sectional analysis, subjective assessment
Gunn et al[212]	151 children (6–18; 67 M, 84 F)	Oral examination, interview	a,b: 1	No correlation with occlusal measures	Subjective assessment, small sample
Lindqvist[213]	79 teenagers (14)	A: SB (n = 34); B: controls (n 45) oral examination	a,b: 1	No. of subjects with OI: A (55%) > B (29%)	Subjective assessment, small sample
Henrikson et al[214]	183 girls (11–15)	A: Class II malocclusion (n = 123); B: normals (n= 60) oral examination	a,b: 1	No. of grinding reports: 23/123 (A) > 9/60 (B)	Subjective assessment
Kampe et al[215]	225 children (13–15)	A: with restorations (n = 129) B: without restorations (n = 96)	a,b: 1	No. of grinding reports: 9/129 (A) > 4/96 (B)	Subjective assessment
Polygraphic studies					
Lobbezoo et al[216]	10 SB patients and 10 normals	A: 10 SB patients; B: 10 normals; cephalometry, cast model, oral examination	a: 1,2,4 b: 4	No group differences for any occlusal and morphological variables	Objective assessment but cross-sectional analysis
Clinical studies					
Forssell et al[217]	91 TMD patients with headache (16–52; 11 M, 80 F)	Double-blind placebo controlled A: Occlusal adjustments (n = 48) 5- to 20-month follow-up B: Placebo adjustments (n = 43) 2- to 6-month follow-up	a,b: 1	Before treatment A: 16/48; B: 14/43 After treatment A: 9/48; B: 8/43	No statistical analysis; subjective assessment; orofacial pain
Kirveskari et al[218]	65 healthy university students (19 M, 46 F)	Double-blind placebo controlled A: Occlusal treatment (n = 33) B: Placebo treatment (n = 32) 2-year follow-up	a,b: 1	3 drop-outs in A A: improved in 1 and impaired in 5 subjects B: impaired in 7 subjects	No statistical analysis, subjective assessment no description of SB population
Henrikson and Nilner[219]	183 girls (11–15)	A: Class II-treatment (n = 65) B: Class II-no treatment (n = 58) C: Normals (n = 60) 2-year follow-up	a,b: 1	Before treatment A: 23%; B: 22%; C: 9% 2 years after treatment A: 11%; B: 15%; C: 7%	Subjective assessment
Kerstein and Farrell[220]	32 SB patients with jaw muscle pain (15–51; n = 32)	Descriptive open trial occlusal equilibration	a: 1 b: 6	Marked decrease or complete absence for 7 days after treatment	Poor description for orofacial pain
Yustin et al[221]	86 TMD patients (36.4; 18 M, 68 F)	The use of anterior bite splint during sleep (15.5 months) followed by the continuation of splint, occlusal adjustment, orthodontic/prosthetic treatment or orthognathic surgery	a: 6 b: 6	Most patients relieved from bruxism	Poor description for orofacial pain
Bailey and Rugh[222]	9 SB patients	Occlusal adjustment baseline-intervention-postbaseline	a: 1 b: 5	No change in 6, increase in 2 and decrease in 1	Abstract, unclear diagnosis
Kardachi et al[223]	16 SB patients 4 normals (18–39; 8 M, 12 F)	Randomized trials A: Occlusal adjustment (n = 4) B: Placebo adjustment (n = 4) C: Biofeedback (n = 4) D: Control feedback (n = 4) E: Normals (no intervention; n = 4) Comparison: baseline-intervention	a: 6 b: 5	No difference between groups	Unclear diagnostic criteria for SB, randomized but small sample, unclear for specific SB activity
Experimental studies					
Shiau and Syu[224]	13 SB patients 14 normals (18–30; 15 M, 12 F)	Open trial: occlusal interferences created by metal overlay (1.5 mm thickness) 1-month follow-up	a,b: 1	A: 4 dropouts; no increase in 5; decrease or absence in 4 B: 2 dropouts, no change	Subjective assessment
Rugh et al[225]	8 SB patients (23–46; 2 M, 6 F)	Cross-over design splint: canine guidance (A) and molar guidance (B) 1-week recording	a: 1,2,5 b: 3,5	No difference between the 2 splints	Objective assessment, influence of splint insertion, unclear for specific SB activity
Rugh et al[226]	10 normal subjects (26–41; 5 M, 5 F)	Open design, occlusal interference created by crown (0.5–1.0 mm); 1-week recording	a: 1,2 b: 3,5	Increase in 1, no changes in 4, decrease in 5	Objective assessment, unclear for specific SB activity
Takeda et al[227]	7 normal subjects	Open design, occlusal interference created by class I inlay (0.1 mm); 1-week recording	a: 6 b: 6	Increased masseter EMG activity after intervention	Abstract, objective assessment, unclear for specific SB activity

*Age (range or average); sex (F = female; M = male).
†Criteria to select SB patients and assess SB activity: 1 = a history/awareness of tooth grinding; 2 = clinical signs of SB including tooth wear and morning jaw muscle discomfort and masseter muscle hypertrophy; 3 = clinical signs of temporomandibular disorders (TMD); 4 = polysomnographic criteria; 5 = sleep masseter muscle activity with ambulatory system; and 6 = unclear.
OI = occlusal interferences.

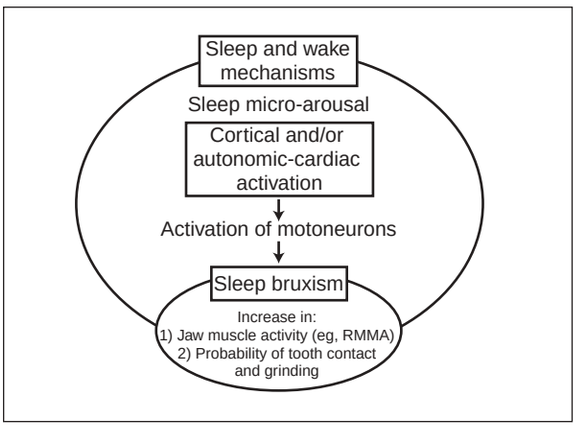

Fig 3 Sleep arousal sequence in SB genesis. Sleep micro-arousal is associated with cortical and autonomic-cardiac activation and, occasionally, with masticatory muscle activation. A sequence of cortical and/or autonomic-cardiac activation precedes masticatory muscle bursts of rhythmic masticatory muscle activity (RMMA) associated with SB (≈ 80%). In SB patients, masticatory muscle activation is more pronounced and associated with more frequent tooth grindings (or tooth contact).

Peripheral Occlusal Influences on SB

Local or peripheral factors were long thought to be an important cause of bruxism during wakefulness and sleep. However, the validity of occlusal disharmony (or malocclusion) as a primary cause is not strongly supported by the literature.[21,180,181,204–208] In reviewing occlusal influences on SB, the authors distinguished between SB (eg, tooth grinding) and daytime parafunctional habits (eg, clenching).

Epidemiologic or observational studies have reported negative and positive correlations between reports of tooth grinding and the presence of occlusal interferences (Table 5).[209–215] The negative correlation was supported by a controlled study using polygraphic recordings (Table 5).[216] Moreover, contrary to the case reports, clinical studies using a control group have not demonstrated a positive effect of either occlusal adjustments or orthodontic treatment on SB (Table 5).[217–221] This is also made apparent in studies in which sleep masseter EMG activity was monitored as the outcome.[222,223] The influence of artificially created occlusal interferences on both reports of SB tooth grinding and masseter EMG activity during sleep is debatable and controversial (Table 5).[224–227]

Several limitations should be noted when interpreting these data. The diagnostic criteria for SB tooth grinding were based mainly on a clinical interpretation of tooth wear and on subjective reports (family member, bed partner, self-report). Observations of tooth wear and subjective reports of grinding are not highly reliable since tooth wear is observed in normal subjects who do not report tooth grinding, and a report/awareness of tooth grinding during sleep needs an observer (eg, sleep partner) who also recognizes and/or frequently complains of this nighttime activity.[2,3,15,228] Other recognizable limitations that need to be controlled for include factors that influence oromotor function during sleep, such as orofacial pain and TMD. Several studies used unblinded and uncontrolled designs with a small sample size. Moreover, there have been very few studies using objective recordings to assess the physiologic influences of occlusal factors on SB activity. Owing to these limitations, causal relationships between occlusal factors and SB remain to be clarified. In addition, it is also difficult to link physiologic mechanisms to the clinical data on the role of occlusion in SB.

Intraoral and Perioral Stimulation

Several studies have used intraoral stimulation in an attempt to prevent SB genesis. In these studies, stimuli were given to intraoral structures after SB had been initiated. Studies with small sample sizes have reported that stimulation of oral sensory afferents in humans decreased SB motor events. One study used a 4-channel ambulatory system (EMG, ECG) and found that non-noxious electrical stimulation to the lip, triggered by tooth contact on a sensor within an oral appliance, reduced overall sleep masticatory muscle EMG activity in 7 SB patients.[229] In a single SB patient, a general vibratory stimulus was applied to either teeth or oral mucosa with an oral appliance fabricated with a vibrator (vibratory stimuli were triggered when tooth contact force exceeded a pre-determined non-sleep threshold). The number and duration of vibratory events lasting more than 1 second were calculated as an indirect measure of SB episodes. The number of vibratory events was reduced by 25% and the duration by 44% at the 4-month follow-up recording.[230] Another study using disagreeable taste stimulation also reported a reduction of SB in a single patient over an 8-month period.[231] In this latter study, a capsule placed on an oral appliance ruptured when clenching or tooth grinding occurred and delivered an unpleasant taste (eg, quinine) stimulus.

It is obvious that the efficacy of all these interesting clinical tools needs to be further assessed in controlled trials. Moreover, to understand the mechanisms behind these paradigms, further investigation is required to determine the precise roles

of sensory stimulations on sleep architectures (eg, sleep stages, micro-arousal) and behavioral conditioning (eg, biofeedback paradigms) in relation to trigeminal nerve excitability.[20,89,90,98,232–234]

Visceral Sensory Stimuli, Salivation, and Swallowing

During wakefulness, chemical or mechanical stimuli applied to the pharynx and esophagus (eg, by either experimental stimuli or refluxed stomach content) induces swallowing, which prevents aspiration of undesirable substances and damage to esophageal structures.[98,235,236] The stimulation of visceral vagal afferents (tenth cranial nerve) in these structures activates the swallowing centers located in the brainstem.[237,238]

Interestingly, visceral vagal afferents also project to central nervous system structures (eg, the nucleus tractus solitarius) involved in both arousal and swallowing.[238–242] During sleep, human swallowing can be induced by stimulation of the pharyngeal and esophageal tissues[243–247] and sleep arousals have been found to concomitantly increase the number of swallowing episodes.[244,247] This suggests that sensory input from vagal visceral afferents could also trigger sleep arousals, which may in turn facilitate orofacial activity during sleep. In fact, gastroesophageal reflux (GR) events resulting in decreasing esophageal pH were reported to be associated with both increased sleep arousals and swallowing during sleep.[235,248,249] Although an increase of swallowing in relation to GR events is characteristic in patients with GR, healthy subjects showed swallowing during sleep in the absence of reflux events.[250,251]

Salivary flow is known to decrease significantly during sleep.[252,253] However, even during sleep, saliva still plays important physiologic roles such as the lubrication and protection of oroesophageal structures, teeth, and mucosa.[236] During sleep, dryness, and/or acidification of oroesophageal tissues may generate sensory signals, possibly as a "warning" signal, which induces sleep micro-arousal with subsequent salivation secondary to sleep RMMA.[235,236,254,255] Interestingly, salivary flow is associated with the stimulation of intraoral mechanoreceptors and gustatory receptors.[93,256–259] Salivation is high on the chewing side of the mouth and depends on chewing frequency and bite force.[256,260] Thus, it has been hypothesized that RMMA may be associated with salivary secretion and lubrication of the oral cavity and the upper alimentary tract during sleep.[236]

SB and swallowing occur more frequently during light NREM sleep and they are associated with sleep arousal. Recently, we found that close to 60% of RMMA episodes in SB patients and normal subjects occurred concomitantly with swallowing-related laryngeal movements.[170] Moreover, swallowing occurred more frequently in the last third of SB episodes. It remains possible that RMMA associated with SB is also triggered by oral dryness, which in turn can cause more dental damage due to the absence of lubricant.[236] The relationships between sleep micro-arousals, sleep RMMA/swallowing, and salivation during sleep need further investigation before the above hypotheses are confirmed.

Other Sensory Stimuli that Influence Sleep Bruxism Activity

In a biofeedback experiment, high-intensity auditory stimuli (> 65dB) were applied at the initiation of an SB episode in order to arouse patients from sleep. Each auditory stimulus was triggered when masticatory EMG activity and/or biting force exceeded a predefined non-sleep threshold.[234] When auditory arousing stimuli were used, the duration (but not frequency) of SB episodes was found to decrease,[234,261,262] but the effect did not persist over time.[3,234] On the other hand, when SB patients were awakened from sleep by auditory stimuli and then forced to do behavioral tasks, both the frequency and duration of SB episodes were reduced and the effects persisted over long periods of time.[234,263]

In contrast to these previous findings, a few studies reported that SB might be induced by applying sensory stimulation during sleep. In normal subjects with no orofacial pain problems, a sudden increase of masseter EMG activity was scored in sleep following loud auditory noise (> 60dB).[264] In an open study, tooth-grinding episodes during sleep were found to follow 7.8% of experimental arousals (tactile, auditory, and photic stimuli) in patients who reported a history of tooth grinding.[193] In a recent controlled study, experimental micro-arousals were induced by auditory (< 45dB) and arm vibrotactile stimuli (without full awakening) in 8 SB patients diagnosed by polysomnography and in 8 normal control subjects.[7] The probability of experimentally induced tooth-grinding episodes (7.9% of experimental arousal) was found to be similar to the previous study.[7,193] Moreover, RMMA and/or tooth-grinding episodes occurred in all 8 SB patients but only 1 control subject showed RMMA (without tooth grinding). Therefore, in patients diagnosed with SB, 7 times more RMMA episodes were induced secondarily by experimental micro-arousal.[7]

Conclusion

Several sensory stimuli can be associated with arousal and oromotor systems during sleep. Orofacial stimulations, given after SB initiation, seem to interrupt any ongoing SB episode. Similarly, SB can be interrupted by high-intensity auditory stimuli if they are strong enough to arouse sleeping subjects. The mechanisms involving such influences on SB require further study. The influence of visceral sensory stimuli on sleep arousal and oromotor activity has been documented in the literature. The concomitant occurrence of SB and swallowing in relation to visceral sensory stimuli and salivation still needs to be investigated. Interestingly, SB episodes can also result from sensory-induced micro-arousal in SB patients, supporting a strong association between sleep arousal and SB genesis. These findings suggest that there is a possible interaction between sensory input and sleep-wake mechanisms in SB genesis.

Concluding Remarks

To date, polysomnographic studies have revealed a relationship between sleep micro-arousal (ie, cortical and autonomic activation preceding jaw-closer muscle motor activation) and SB episodes, suggesting that autonomic/central nervous system activation are primary factors responsible for initiating SB. Further studies are needed to confirm the role of the autonomic nervous system as well as the central nervous system in the pathophysiology of SB. From the above evidence, it appears that peripheral sensory influences are more likely to be secondary rather than primary contributing factors in the genesis of SB. Since at present there is insufficient information to adequately define the role of the periodontal-occlusal influences on orofacial sensory- motor system(s) during sleep, it is probably wise to be extremely cautious in interpreting data. Moreover, it should be reiterated that periodontal factors are one of the numerous sensory inputs that can influence trigeminal sensorimotor function, homeostasis, and the sleep-wake system. The age of describing a disease or disorder with only 1 neurotransmitter or 1 gene or 1 factor is past. An integrated approach is mandatory when the mechanism of a condition, such as SB, is under investigation.

With these concepts in mind, and in terms of the management of SB in clinical practice, occlusal splints, for example, could be used in order to prevent the undesirable consequences of SB (eg, tooth wear, tooth-grinding sounds, pain) rather than being advocated as a means of preventing the initiation of SB episodes.[181] Other interventions that may in fact influence either the central oromotor system and/or the sleep process (eg, medication, behavioral-cognitive strategies) could then be tested to see whether they reduce the probability of SB genesis or decrease oromotor responsiveness to micro-arousal. The efficacy of SB management interventions also needs to be assessed in patients with concomitant clinical problems frequently associated with increased sleep micro-arousals, such as chronic pain, sleep apnea, and insomnia.

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