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Sleep Bruxism in Respiratory Medicine Practice

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Sleep bruxism (SB) consists of involuntary episodic and repetitive jaw muscle activity characterized by occasional tooth grinding or jaw clenching during sleep. Prevalence decreases from 20% to 14% in childhood to 8% to 3% in adulthood. Although the causes and mechanisms of idiopathic primary SB are unknown, putative candidates include psychologic risk factors (eg, anxiety, stress due to life events, hypervigilance) and sleep physiologic reactivity (eg, sleep arousals with autonomic activity, breathing events). Although certain neurotransmitters (serotonin, dopamine, noradrenalin, histamine) have been proposed to play an indirect role in SB, their exact contribution to rhythmic masticatory muscle activity (RMMA) (the electromyography marker of SB) genesis remains undetermined. No specific gene is associated with SB; familial environmental influence plays a significant role. To date, no single explanation can account for the SB mechanism. Secondary SB with sleep comorbidities that should be clinically assessed are insomnia, periodic limb movements during sleep, sleep-disordered breathing (eg, apnea-hypopnea), gastroesophageal reflux disease, and neurologic disorders (eg, sleep epilepsy, rapid eye movement behavior disorder). SB is currently quantified by scoring RMMA recordings in parallel with brain, respiratory, and heart activity recordings in a sleep laboratory or home setting. RMMA confirmation with audio-video recordings is recommended for better diagnostic accuracy in the presence of neurologic conditions. Management strategies (diagnostic tests, treatment) should be tailored to the patient's phenotype and comorbidities. In the presence of sleep-disordered breathing, a mandibular advancement appliance or CPAP treatment is preferred over single occlusal splint therapy on the upper jaw.

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Sleep bruxism (SB) is an involuntary trigeminal motor activity characterized by episodic and repetitive jaw muscle activity with occasional tooth grinding or jaw clenching during sleep. By consensus, SB has been defined as repetitive jaw muscle activity characterized by clenching or grinding of the teeth and bracing or thrusting of the

ABBREVIATIONS: EMG = electromyography; GERD = gastroesophageal reflux disease; PSG = polysomnography; RBD = rapid eye movement behavior disorder; REM = rapid eye movement; RERA = respiratory effort-related arousal; RMMA = rhythmic masticatory muscle activity; SB = sleep bruxism; SDB = sleep-disordered breathing; TMD = temporomandibular disorder

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mandible.[1] During sleep, SB usually is quantified by scoring rhythmic masticatory muscle activity (RMMA) on electromyography (EMG).[2,3] RMMA episodes consist of jaw muscle contractions that are phasic (more than three EMG bursts lasting 0.25-2.0 s each), tonic (sustained for $>$ 2 s), or a mixture of both. However, audio-video recordings are more specific in the presence of other orofacial motor activity or comorbidity.[3] Note that when all subjects with SB have RMMA, some will present no or little tooth grinding, whereas others will have more-frequent RMMA with more tooth grinding, as described later.[4]

The estimated prevalence of sleep-related bruxism awareness is based on reports by parents or a sleep partner. Prevalence is the highest in childhood, at approximately 14% to 20%. It stabilizes at around 8% to 12% in teenagers and adults and decreases thereafter with aging to 3% (Fig 1).[5-7] Parents of children aged $<$ 12 years report more complaints of tooth grinding, whereas older teenagers self-report more awareness of wake-time clenching.[8] Tooth grinding awareness is based mainly on complaints by parents or a sleep partner, which are not always reliable or accurate.[9,10]

Bruxism has two distinct circadian manifestations: during wakefulness (wake bruxism) and during sleep (SB) with a dominance of RMMA episodes in non-rapid eye movement (REM) sleep.[3,11] The two types can overlap.[12] Tooth grinding is dominant during sleep, and clenching is dominant during wake.[4,8,12]

Etiology and Putative Mechanisms of SB-Related RMMA

The etiology of idiopathic primary SB is broad and nonspecific. Included risk factors are anxiety and stress, a goal-oriented personality, sleep-wake autonomic cardiac and respiratory reactivity, motor excitability, and circadian/ultradian non-REM-REM rhythm. The putative SB pathophysiology is described in Figures 2 and 3.[13-17] There is no single definitive explanation.

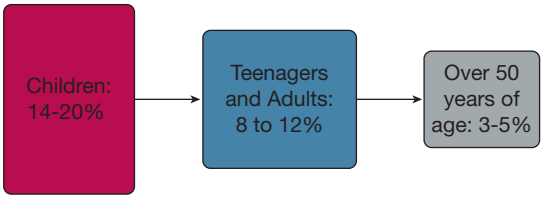

Figure 1 – *Decrease in prevalence of tooth grinding with aging based on reports by parents or sleep partners.*

SB is highly variable over time: In most subjects, it occurs as a short-term fluctuation, with alternating periods of silence over the life span.[18] If SB is present in childhood, it may persist into adulthood.[7,11] Furthermore, some wake-time moods or types of activity may persist into sleep, known as the carryover effect. For example, in otherwise healthy subjects, an anxiety trait was associated with masticatory muscle activity in the first hour of sleep.[19] The typical individual with SB is prone to anxiety, has maladaptive coping skills, and exhibits competitive and performance-oriented behaviors.[20-22] Many habits, tics, or atypical types of oral activity may be present during wake, some of which may persist into sleep.[2,3] A general psychobiologic state of hyperarousal may also contribute to RMMA onset in some subjects with SB, as seen for insomnia, but this remains to be confirmed.[23] Tonsillar hypertrophy and retrognathia are also reported as SB risk factors in pediatric and teenager populations, but this association is based on questionnaires and clinical examinations, and awaits confirmation by PSG studies.[8,24]

The list of neurotransmitters (Fig 2) potentially associated with RMMA is long and nonspecific because most are also involved in many behavior, mood, wake, or sleep vigilance disorders.[25-27] The presumed roles of many as-yet unproven neurotransmitter-related genes are also under study (eg, serotonin, dopamine, noradrenalin, histamine). The suggested association between bruxism and a gene related to a serotonin receptor (HTR2A C-allele carrier) should be replicated using more rigorous methods to quantify wake bruxism and SB.[28] SB is reported to run in families: one-third to one-half of subjects with SB have a direct relative with a history of tooth grinding. However, the environment is also strongly influential, and the SB phenotype is probably large, which may explain why it is difficult to identify a specific gene for RMMA.[11,29]

Technological innovations, including fine time-scale analysis and multiple polysomnography (PSG) monitoring, have advanced the understanding of putative mechanisms of sleep and movement disorders. The early suggestion that the autonomic nervous system and CNS are connected is now supported by stronger evidence.[30,31] We and others have observed that RMMA can be present in association with sleep arousal (ie, transient 3-15 s heart rate, brain, and muscle activity).[15,32] This association was time related for 50% to almost 80% of RMMA in otherwise healthy children and adults.[2,3,33,34] This association is not the only cause, nor does it fully explain the genesis of all