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Sleep Bruxism Etiology: The Evolution of a Changing Paradigm

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ABSTRACT

Various definitions, classifications and theories been ascribed to bruxism. Knowledge gained through expanding research initiatives have transformed some of the concepts that were once held as truths. Sleep bruxism is no longer considered a parasomnia nor is its etiology believed to be based on purely mechanical factors or psychological issues. It is now considered to be primarily a sleep-related movement disorder with a yet to be determined multifactorial etiology involving complex multisystem physiological processes. Dental practitioners should recognize the transformation occurring in the study of sleep bruxism, understand the evolution in both definitions and classification of this phenomenon and embrace and consider new concepts related to its etiology. This paradigm shift will certainly affect the daily practice of dentistry.

Over the years, various definitions, classifications and theories regarding the etiology of bruxism have been presented, reflecting the evolution and growth of knowledge of this subject. Currently, bruxism is no longer accepted as a single entity, but is divided into two distinct entities — awake and sleep bruxism — based on when the activity occurs. Furthermore, contemporary research methods have enabled the study of a myriad of physiologic systems, including brain activity, muscle activity, cardiac function and breathing, resulting in a major transformation in our understanding of sleep bruxism.[1,2] Thus, sleep bruxism is no longer considered to be simply related to mechanistic factors, such as occlusal discrepancies, or a result of psychological issues, such as stress, anxiety or depression or a combination thereof.[3,4] Instead, most authorities now consider sleep bruxism to be primarily a sleep-related movement disorder with a yet to be discerned multifactorial etiology and complex multisystem physiological processes.

The aim of this article is to provide the dental practitioner with a review of the transformation that has occurred in the study of sleep bruxism, highlighting the evolution of its definition and classification and providing a detailed discussion on the change in thinking regarding its etiology. We conclude with a discussion of how this paradigm shift affects the daily practice of dentistry.

Definitions

To date, three definitions of sleep bruxism have been provided by the American Academy of Sleep Medicine (AASM). In 1990, the International Classification of Sleep Disorders (ICSD) defined sleep bruxism within the category of parasomnias (or disorders that intrude on sleep, but are not associated with complaints of insomnia or sleepiness) as a stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep.[5] In the second edition of the ICSD in 2005, sleep bruxism was categorized as a sleep-related movement disorder and defined as an oral parafunctional activity characterized by tooth grinding or jaw clenching during sleep, usually associated with sleep arousals.[6] Recently, an updated definition of general bruxism was adopted for the third edition of the ICSD: a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.[7]

Classification

Several classification schemes have been proposed for sleep bruxism based on different criteria. If sleep bruxism is classified according to etiology, then it has two distinct categories: primary or idiopathic sleep bruxism, which is without an identifiable cause

or any associated socio-psychological or medical problem; and secondary sleep bruxism, which is related to a socio-psychological or medical condition (e.g., movement or sleep disorder including periodic limb movement disorder and rhythmic movement disorders, such as head banging, sleep disordered breathing due to upper airway resistance or apnea–hypopnea events, neurologic or psychiatric condition, drug/chemical related).[8,9]

Another classification system, recently developed by consensus among an international group of experts, employs a novel diagnostic grading system for both clinical and research purposes using the terms possible, probable and definite to categorize sleep or awake bruxism (Table 1).[10] The third edition of the ICSD employs a different set of criteria for the diagnostic classification of the new term "sleep-related bruxism" (Table 2).[7]

Table 1 Diagnostic grading system for sleep and awake bruxism provided by an international group of experts[10]

Possible	Based on self-report using a questionnaire and/or the anamnestic part of the clinical examination
Probable	Based on self-report plus the inspection report of the clinical examination
Definite	Based on self-report, a clinical examination and a polysomnographic recording preferably containing audio/visual recordings

Table 2 Diagnostic criteria for sleep-related bruxism based on the International Classification of Sleep Disorders (third edition)[7]

Presence of regular or frequent tooth grinding sounds occurring during sleep
Presence of one or more of the following clinical signs: i. abnormal tooth wear consistent with above reports of tooth grinding during sleep ii. transient morning jaw muscle pain or fatigue; and/or temporal headache; and/or jaw locking on awakening consistent with above reports of tooth grinding during sleep

The evolution of the definition and classification of sleep bruxism has allowed this entity to become more distinct and homogenous. This increased focus facilitates research activities, hopefully resulting in improved understanding of etiological factors and physiological processes associated with sleep bruxism.

Etiology: the Historical Perspective

Peripheral Factors and Influences

Initially, the dental profession was quite convinced that there was a mechanistic and singular etiology for sleep bruxism directly related to peripheral factors or influences, such as occlusal factors, with tooth wear confirming a clinical diagnosis. The occlusion concept was popularized in a classical article by Ramfjord in 1961,[3] and later studies supported this concept, as occlusal corrections were reported to diminish or stop this sleep activity.[11,12] However, closer scrutiny showed that Ramfjord's original article was methodologically flawed.[13,14] Several studies challenged occlusal disharmony or premature tooth contacts as a principal etiological factor and reported that sleep bruxism activity was not reduced by occlusal therapy.[15-18] Other studies reported a similar prevalence of sleep bruxism in people with and without occlusal interferences,[19-21] and multiple logistic regression models were unable to clearly differentiate sleep bruxers from non-bruxers.[22] Furthermore, studies reported a lack of correlation between dental morphology (dental arch, occlusion) and sleep bruxism events among adult patients with sleep bruxism,[23] and differences between sleep bruxism patients and non-sleep bruxism control groups in terms of various occlusal and functional variables could not be found.[24]These studies negate the effect of peripheral anatomical–structural factors and influences as reliable etiological factors for

sleep bruxism.

From a clinical perspective, it was believed that a diagnosis of sleep bruxism could be confidently confirmed by the observation of tooth wear.[25-28]However, this is questionable as tooth wear may be produced by other etiologic factors, such as oral habits, food consistency and acid reflux.[29,30] Moreover, occlusal attrition does not reliably confirm sleep bruxism, especially in the absence of a report of tooth grinding during sleep as witnessed by a sleep partner.[25,31] In addition, Menapace et al.[32] reported that although tooth wear was present in 100% of sleep bruxism patients, it also occurred in 40% of asymptomatic individuals. Although sleep bruxism patients (young adults) presented with greater tooth wear than those with no history of tooth grinding or sleep laboratory evidence of sleep bruxism, tooth wear could not discriminate between patients with moderate–high versus low levels of sleep bruxism.[33]

To date, the etiology of sleep bruxism remains rather elusive as it does not appear to be related to mechanistic peripheral factors, nor can it be attributed to a singular cause. Certainly, the once strongly held belief that occlusion and the presentation of tooth wear were demonstrative of this entity is rather doubtful.

Stress and Psychological Factors and Influences

Stress and psychological factors were once considered major factors in the etiology of sleep bruxism. Early studies observed that masticatory muscle activity and periodic pain during sleep increased during stressful periods among those reporting sleep bruxism.[34-36] However, other studies found this association to be true in only a small percentage of people.[37-39] Studies suggest that children and adults reporting awareness of tooth grinding are more anxious, aggressive and hyperactive.[40-47]

However, the methods used in these studies were found to have significant limitations resulting in relatively weak evidence.[4,48] Some studies posit that sleep bruxism patients are more likely to deny the impact of life events because of their coping style or personality.[49,50] In some case studies, masseter electromyographic (EMG) activity increased during sleep following days with emotional or physical stressors,[51,52] but these findings were not consistent in all studies.[37,39,53]In a recent epidemiologic study using polysomnography, the authors reported no association between sleep bruxism and anxiety or depression, but did find a significant association between sleep bruxism and complaints of insomnia.[54] Overall, a subgroup of sleep bruxism patients might exist whose psychosocial response to life stressors or experimental stressors is manifested by jaw motor activity during sleep, but this reaction differs from that of normal individuals.[4,50,55,56]

Etiology: Current Hypotheses

The most recent hypotheses on the etiology of sleep bruxism support the roles of the central and autonomic nervous systems in the genesis of oromandibular activity during sleep. More specifically, sleep-related mechanisms under the influence of brain chemicals and maintenance of airway patency during sleep may increase motor activity underlying the genesis of sleep bruxism and rhythmic masticatory muscle activity (RMMA), the motor manifestation of sleep bruxism preceding tooth grinding during sleep.

The Role of Neurochemicals

The first evidence suggesting that tooth grinding might be linked to a chemical substance in the brain came from a case report in which a patient suffering from Parkinsonism was treated for tooth grinding with l-3,4-dihydroxyphenylalanine (l-DOPA), a catecholamine precursor.[57] In a series of controlled trials in young, healthy sleep bruxism patients, l-DOPA has been reported to produce a modest but significant reduction in sleep bruxism-RMMA frequency compared with placebo, while bromocriptine (a more direct dopamine agonist) had no obvious influence on RMMA genesis.[58,59]

Given the putative role of noradrenaline in bruxism, experimental trials with propanolol and clonidine have also been carried out.[60,61] Propranolol, a non-selective beta blocker, did not cause a significant reduction in sleep bruxism-RMMA; however, clonidine, an alpha agonist acting on the central nervous system, significantly reduced the sleep bruxism-RMMA index compared with placebo (this experiment was reproduced with a lower dose, 0.1 mg, in Dr. K. Baba's laboratory, Japan, unpublished information). This effect was partly associated with a concomitant reduction in the cardiac-autonomic sympathetic dominance that precedes RMMA, as described below.[60,61] It should be noted that clonidine is associated with severe hypotension in the morning, and its use for sleep bruxism therapy is, thus, cautionary.

Sleep Arousal and Motor Activity of Sleep Bruxism

Studies have shown that most sleep bruxism-RMMA episodes occur during transient (3–10 s) arousal associated with brain and cardiac activity as shown by a rapid increase in heart rate, i.e., tachycardia, at the onset of RMMA during recurrent sleep micro-arousal episodes.[62,63] Micro-arousals are natural activities during sleep that consist of a repetitive rise in heart rate, muscle tone and brain activity 8–15 times/h of sleep.[14]

Sleep is divided into three to five non-rapid-eye-movement (REM) and REM periods of 90–110 minutes. Non-REM sleep is further

divided into light sleep (stages 1 and 2) and deep sleep (stages 3 and 4). Most sleep bruxism episodes are observed during light non-REM sleep (stage 2), whereas about 10% occur during REM sleep in association with sleep arousal. Sleep bruxism tends to occur in relation to recurrent micro-arousal within the so-called cyclic alternating pattern, which repeats every 20–60 s during non-REM sleep.[64,65] A summary of the genesis of most sleep bruxism episodes is presented in Table 3.

Table 3 Stages in the genesis of most sleep bruxism episodes[66]

1. Rise in sympathetic cardiac activity at minus 8–4 minutes
2. Rise in the frequency of electroencephalography activity at minus 4 s
3. Heart rate tachycardia starting at minus 1 heartbeat
4. Increase in jaw opener muscle activity, probably responsible for mandible protrusion and airway opening
5. An associated major increase in the amplitude of respiratory ventilation
6. Observable electromyography incidents scored as sleep bruxism rhythmic masticatory muscle activity, with or without tooth grinding

The role of respiration in the genesis of sleep bruxism-RMMA is not fully understood but recent evidence suggests that it may be relevant in some patients. RMMA tends to occur with large breaths, and oral appliances used to improve airway patency help to reduce sleep bruxism-RMMA frequency.[67-69] However, before dental practitioners assume a direct role of respiration or a cause-and-effect relation between breathing disorders and sleep bruxism, more robust evidence is required.

Treatment

Currently, no therapy has been proven effective in treating sleep bruxism. The available treatment approaches report various levels of efficacy in managing the potentially harmful consequences of sleep bruxism.[70] Sleep bruxism can be managed by behavioural strategies, which include avoidance of risk factors and triggers (e.g., smoking, alcohol, caffeine, drug assumption), patient education (e.g., control of wake-time oral parafunctions such as clenching activity), relaxation techniques, sleep hygiene, hypnotherapy, biofeedback and cognitive behavioural therapy (CBT).[71-74] Most of these strategies are not supported by evidence from controlled trials. However, a recent study showed that a new biofeedback device that applies electrical pulses to inhibit EMG activity in the temporalis muscles was effective in reducing EMG activity during sleep without disrupting sleep quality.[73] Likewise, 12 weeks of CBT for sleep bruxism subjects was found to reduce sleep bruxism activity. However, CBT was not significantly different from occlusal appliance therapy in reducing sleep bruxism.[74]

Occlusal appliances, either on the maxillary or mandibular arch, to remove occlusal interferences, protect dental surfaces and relax masticatory muscles, have been extensively used in clinical practice. However, their exact mechanisms of action are still under debate, and no evidence supports their role in stopping sleep bruxism. Moreover, the lack of well-designed randomized controlled clinical trials and long-term studies in the literature complicates evaluation of their effectiveness. Most studies reported a decrease in sleep bruxism activity in the first 2 weeks of treatment independent of appliance design.[64,74-79] However, their effect seems to be transitory and highly variable among patients. Moreover, approximately 20% of patients display an increase in EMG activity during sleep when they wear an occlusal appliance, especially the soft mouth guard type.[80]

Several medications have been associated with both a decrease and an increase in sleep bruxism activity, supporting the probability of involvement of central mechanisms in the genesis of sleep bruxism.[57] In particular, the dopaminergic, serotoninergic and adrenergic systems are considered to be involved in the orofacial motor activity. However, there is still lack of evidence of both the efficacy and safety of medications for sleep bruxism patients, and pharmacological treatments should be considered only in symptomatic severely affected patients and only as short-term therapy. Recently, a placebo-controlled study demonstrated a 40% reduction in sleep bruxism activity with a dose of clonazepam (1 mg).[81] However, no data are available on long-term treatment and potential side effects, such as sleepiness, tolerance and dependence. It is worth noting that patients with sleep bruxism must be screened for other comorbid medical conditions before being considered for pharmacotherapy. Underlying disorders and medications may interfere with motor activities during sleep and must be assessed before other treatments are recommended.

Conclusion

Dental practitioners must embrace the concept that outdated views of sleep bruxism should be abandoned. New knowledge is being gained through scientific exploration and challenging what was once thought of as fact. Such is the case with the definitions, classifications and etiology of sleep bruxism. Therefore, it is important for dental practitioners to recognize and understand the new paradigms, as this will lead to improved interventions for patients who deserve the best evidence-based care available.

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