A Comprehensive View of Bruxism: From Etiology to Evidence-Based Management

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Abstract: Bruxism is increasingly understood as a centrally regulated behavior rather than a consequence of occlusal discrepancies. Current evidence distinguishes Sleep Bruxism and Awake Bruxism as separate entities influenced by neurochemical mechanisms, sleep-related arousal patterns, and psychosocial factors. Sleep Bruxism is strongly associated with micro-arousals and autonomic activation and may act as a compensatory response in individuals experiencing airway obstruction, particularly those with obstructive sleep apnea. Awake Bruxism, conversely, is more closely linked to emotional tension, stress, and cognitive focus. Epidemiological findings vary widely across populations due to differing diagnostic standards, while pediatric bruxism often reflects developmental and airway-related influences and tends to diminish with age. Neurochemical and genetic research highlights the roles of dopaminergic and serotonergic pathways and demonstrates familial clustering. Despite advances in understanding, management remains primarily protective, as occlusal splints can safeguard oral structures but do not suppress the underlying central neural activity. Adjunctive strategies such as botulinum toxin, pharmacologic agents, behavioral interventions, and multidisciplinary care are beneficial in selected cases, especially when bruxism is medication-induced or clinically severe. Diagnostic accuracy continues to rely on the graded system of possible, probable, and definite bruxism, with polysomnography reserved for complex presentations.

In conclusion, bruxism represents a multifactorial behavioral phenomenon that requires a comprehensive, individualized approach. Effective management integrates psychological, respiratory, pharmacologic, and behavioral considerations to address both its manifestations and the underlying drivers that contribute to its persistence.

Keywords: *Bruxism, Sleep Wake Disorders, Temporomandibular Joint Disorder, Occlusal Splints, Botulinum Toxins.*

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I. INTRODUCTION

The medical and dental understanding of bruxism has traversed a complex historical trajectory over the past century. In the early 20th century, the phenomenon was scientifically labeled "la bruxomanie," a term heavily laden with psychological stigma that implied a neurotic or manic compulsion.[1] For decades, the dental community operated under a binary view, struggling to differentiate between diurnal habits and nocturnal events, often conflating them into a singular pathological entity. It was not until the mid-to-late 20th century that the term "bruxism" was standardized to describe the grinding of teeth, regardless of temporal occurrence. This evolution eventually crystallized into the contemporary circadian classification of two distinct nosological entities: Sleep Bruxism (SB) and Awake Bruxism (AB).[2]

In the modern era, the definition has been refined through rigorous international consensus. Bruxism is no longer viewed merely as a mechanical wear-and-tear phenomenon but is defined as a repetitive jaw-muscle activity

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characterized by the clenching or grinding of teeth, and/or the bracing or thrusting of the mandible.[3] While both Sleep and Awake Bruxism utilize the same anatomical machinery specifically the masticatory musculature including the masseter, temporalis, and medial pterygoids they are increasingly recognized as separate entities with distinct etiologies and physiological drivers.[4] Awake bruxism is predominantly a static, sustained clenching behavior often associated with psychosocial tension, concentration, and emotional reactivity. In contrast, sleep bruxism is a dynamic, sleep-related movement behavior characterized by rhythmic (phasic) or mixed (tonic-phasic) muscle contractions.[5]

A pivotal paradigm shift occurred with the release of recent international consensus papers, which argued that bruxism in otherwise healthy individuals should not be inherently classified as a disorder.[3] Instead, it is categorized as a behavior that acts as a risk factor. This distinction is crucial for clinical decision-making; the behavior only transitions into a pathological condition when it exceeds the physiological tolerance of the stomatognathic system, resulting in harmful clinical consequences.[2] These consequences may range from severe dental attrition, abfractions, and fracture of restorations to masticatory muscle hypertrophy and temporomandibular disorders (TMD).[4] Conversely, emerging physiological models propose that sleep bruxism may serve a protective or homeostatic function in certain individuals. Theories suggest that the rhythmic muscle activity may stimulate salivary flow to lubricate the oropharynx during sleep, or more critically, re-establish airway patency in the presence of respiratory obstruction.[6] This "protective hypothesis" challenges the traditional view that all grinding must be eliminated, suggesting instead that in some patients, it is a compensatory mechanism for a distinct underlying pathology.

A. *Comprehensive Epidemiology*

Determining the precise prevalence of bruxism is a significant challenge in epidemiological research. The heterogeneity in diagnostic criteria ranging from subjective self-reports and questionnaires to the gold standard of polysomnography (PSG) creates a wide dispersion in reported data.[4,7] However, recent systematic reviews and meta-analyses provide a clearer picture, suggesting that when all forms are combined, the global prevalence of bruxism sits at approximately 22.22%. [7]

➢ *Epidemiology in the Adult Population:*

In the general adult population, the prevalence of Sleep Bruxism is estimated to range between 8% and 13%, making it a common but not ubiquitous condition.[8] Awake Bruxism appears to be significantly more frequent, affecting approximately 22% to 31% of adults.[7] A robust and consistent finding across multiple studies is the linear decline of sleep bruxism with age. The condition is highly prevalent in young adults but drops significantly in the geriatric population, persisting in only about 3% of individuals over the age of 60.[8] This age-related decline suggests a potential link to the maturation of the central nervous system, the reduction in dopaminergic tone, or changes in sleep architecture as one ages. Regarding gender distribution, sleep bruxism generally shows no significant predilection for males or females, suggesting a balanced biological drive. However, Awake Bruxism is reportedly more common in women.[7] This gender disparity in awake clenching may be attributable to sociocultural differences in stress reporting, hormonal variances, or distinct psychosocial coping mechanisms.[13]

➢ *Epidemiology in the Pediatric Population:*

The prevalence of bruxism in children is notably higher and more variable than in adults, with reported rates ranging wildly from 3.5% to 40.6% depending on the methodology and population studied.[10] In specific cohorts, such as a study involving school children in Brazil, prevalence rates as high as 35.3% have been recorded.[11] Pediatric bruxism presents a unique clinical trajectory; it is often a self-limiting condition. Parental reports and longitudinal studies indicate a steady decline in tooth grinding as children age into adolescence.[12] This natural resolution supports the "wait-and-see" approach often adopted by clinicians for pediatric cases, provided there are no severe comorbidities such as obstructive sleep apnea.[10] The high prevalence in childhood may reflect the developing nervous system's response to growth, dental eruption, or airway maturation.

➢ *Geographic and Cultural Disparities:*

Recent large-scale meta-analyses have highlighted interesting geographic variations in bruxism prevalence. North American populations report the highest prevalence rates of Sleep Bruxism (approximately 31%), followed by South America and Europe.[7] Conversely, South American studies report the highest prevalence of Awake Bruxism (30%).[7] These variations are likely multifactorial, reflecting a combination of genetic diversity, cultural differences in lifestyle and stress levels, and inconsistencies in diagnostic reporting and research focus across different regions.[9] For example, cultural acceptance of expressing stress or differences in sleep environments could influence self-reporting rates in questionnaire-based studies.

B. *Psychosocial Determinants and the Stress-Coping Model*

One of the most extensively researched aspects of bruxism is its relationship with the human psyche. The historical "peripheral" theory, which blamed bruxism on dental occlusal discrepancies (such as high fillings, malocclusion, or premature contacts), has largely been dismantled and replaced by a "centrally regulated" theory.[5] In this central model, psychosocial factors play a dominant role, particularly in the genesis of Awake Bruxism.

➢ *The Role of Stress, Anxiety, and Depression*

There is a robust and well-documented association between self-reported bruxism and psychosocial distress. Individuals who report high levels of perceived stress, anxiety, and depression are consistently found to have a higher incidence of grinding or clenching.[13] This relationship is particularly evident in high-performance environments; for example, studies of university students have found a significant correlation between high perceived stress scores and the incidence of awake bruxism.[14] In this context, the clenching behavior may serve as a somatic outlet for emotional tension a physical manifestation of

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psychological strain where the masticatory muscles act as an end-organ for limbic system activation.[14]

Furthermore, the concept of "trait anxiety" a stable personality characteristic referring to a tendency to perceive situations as threatening has been linked to bruxism.[15] Instrumental studies utilizing electromyography (EMG) have shown that high trait anxiety is a predictor of increased masticatory muscle activity duration, particularly during the first hour of sleep.[15] This suggests that individuals with anxious personalities may carry their physiological hyperarousal into the initial stages of sleep, manifesting as bruxism before deep sleep is achieved.

- *The Paradox of Perception vs. Reality*

A critical nuance in the literature is the divergence between self-reported data and instrumental data. Studies utilizing self-reports almost always find a strong link between stress and bruxism.[13] However, studies utilizing instrumental diagnosis (PSG or EMG) often find a weaker association or, in some cases, none at all.[15,16] This paradox suggests that psychosocial stress may not necessarily increase the sheer volume or intensity of grinding events. Instead, stress likely lowers the patient's pain threshold and increases their interoceptive awareness of muscle tension.[16] Therefore, a stressed individual is more likely to feel the effects of their bruxism and report it, whereas a non-stressed individual with the same level of motor activity may remain asymptomatic and unaware. This "perceptual sensitization" is a key factor in why stress management is a valid treatment modality, even if it does not strictly stop the muscle contractions.

- *Psychosocial Factors in Children:*

The link between stress and bruxism extends to the pediatric population. Children with behavioral difficulties, high levels of responsibility, or neuroticism traits are more likely to exhibit sleep bruxism [17]. It has been hypothesized that children, lacking the verbal or cognitive tools to process complex emotions, may release accumulated daily tension through chronic bruxism during sleep, acting as a primitive stress-relieving mechanism [12].

C. Etiology:

The etiology of bruxism is currently understood as multifactorial, involving a complex interplay of biological, psychological, and exogenous factors. The consensus has shifted decisively away from morphological factors such as dental occlusion or skeletal anatomy as primary triggers.[5] Occlusal equilibration, once the gold standard treatment, is no longer supported by evidence as a curative measure for bruxism.

- *Neurochemistry and Genetics:*

Central factors involving brain neurotransmitters are heavily implicated in the genesis of bruxism. Disturbances in the dopaminergic system are considered significant.[5] This is evidenced by the induction of bruxism in patients taking dopamine antagonists and the reduction of bruxism in some cases with dopamine precursors like L-Dopa. The imbalance in the direct and indirect pathways of the basal ganglia likely leads to the motor disinhibition observed in bruxers. Serotonin also plays a critical, albeit complex, role, particularly in secondary bruxism induced by medications.[18]

Genetic predisposition is another key factor that cannot be overlooked. Studies indicate that sleep bruxism runs in families, with a high concordance rate among twins.[8] Childhood bruxism persists into adulthood in a significant percentage of cases, suggesting a hereditary phenotype related to sleep architecture or central nervous system excitability.[12] While specific genetic markers are still under investigation, the familial trend is strong enough to be a clinical predictor.

- *Drug-Induced (Secondary) Bruxism:*

A specific and clinically relevant etiology is secondary bruxism caused by pharmacotherapy. The use of serotonergic antidepressants, including Selective Serotonin Reuptake Inhibitors (SSRIs) such as fluoxetine, sertraline, and citalopram, as well as SNRIs like venlafaxine, is a well-documented trigger.[18] Symptoms typically manifest within 3 to 4 weeks of treatment initiation or dose titration. The proposed mechanism involves the inhibition of dopaminergic pathways in the basal ganglia by increased serotonin levels, leading to motor disinhibition and involuntary jaw clenching.[18] Case reports involving drugs like escitalopram further validate this mechanism, highlighting the need for vigilance when prescribing these common medications.[19] This form of bruxism is often reversible upon discontinuation of the drug or the addition of counter-agents like buspirone. Additionally, melatonin receptor agonists like ramelteon have been discussed in the context of sleep disturbance management, though the primary focus remains on the dopaminergic-serotonergic balance.[20]

- *Exogenous Lifestyle Factors:*

Beyond endogenous neurochemistry, external lifestyle choices play a significant role. Social habits such as smoking, heavy alcohol consumption, and high caffeine intake are independent risk factors that elevate the odds of developing sleep bruxism.[8] Nicotine promotes dopaminergic activity, while alcohol fragments sleep architecture, potentially increasing the frequency of arousals where bruxism occurs.[8]

D. Pathophysiology: Mechanisms of Action

Understanding the pathophysiology of bruxism requires looking beyond the teeth to the brain's arousal systems, the respiratory tract, and the biomechanics of the jaw joint.

- *Sleep Arousal Physiology:*

Sleep bruxism is intimately linked to the concept of "micro-arousals." These are brief, transient shifts in sleep depth that do not necessarily result in full awakening but represent a surge in central nervous system activity.[4] Polysomnographic studies have meticulously mapped the sequence of events: a Rhythmic Masticatory Muscle Activity (RMMA) episode is often preceded by a rise in heart rate (tachycardia), increased muscle tone, and cortical EEG activation.[21] This sequence suggests that SB is a sleep-

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related movement behavior triggered by the brain's arousal mechanism.[4] It occurs predominantly during light sleep (stages N1 and N2) and during transitions between sleep stages, supporting the theory that it is a manifestation of central nervous system state-switching.[15] This classifies SB closer to other sleep-related motor disorders, like periodic limb movement, rather than a dental pathology.

- *The Respiratory or Airway Patency:*

One of the most significant pathophysiological discoveries in recent years is the association between sleep bruxism and Obstructive Sleep Apnea (OSA).[6] There is a strong correlation between these two conditions, leading to the hypothesis that bruxism may serve a protective physiological role. In patients with partial airway obstruction or high upper airway resistance, the jaw muscles may activate reflexively to protrude the mandible.[6] This protrusion pulls the tongue forward, reopening the airway and preventing hypoxia. In this model, the grinding sound is merely a byproduct of the body's attempt to breathe.

This link is particularly evident in pediatric populations. Children with tonsillar hyperplasia (enlarged tonsils) often exhibit sleep-disordered breathing and high rates of concomitant bruxism.[22] A pivotal study demonstrated that adenotonsillectomy (surgical removal of tonsils and adenoids) resulted in a dramatic reduction in bruxism prevalence, dropping from 45.6% pre-surgery to 11.8% post-surgery.[23] This strongly supports the theory that, in many children, bruxism is a physiological response to airway obstruction rather than a primary neurological disorder.

- *Biomechanical Stress on the TMJ:*

While the origin of bruxism is central, its consequences are peripheral and biomechanical. Finite element analysis of the temporomandibular joint (TMJ) has revealed that the type of muscle activity dictates the damage pattern.[24] Sustained clenching typical of Awake Bruxism generates significantly higher shear stresses and compressive loads on the articular disc compared to the rhythmic grinding of Sleep Bruxism.[24] This sustained loading is considered highly detrimental to the TMJ tissues, potentially leading to disc degeneration, hypoxia of the retrodiscal tissues due to capillary compression, and the development of TMD symptoms.[24] However, the relationship between bruxism and pain is not linear; some patients with severe grinding on PSG report no pain (likely due to central neural adaptation), while others with minimal activity but high central sensitivity report significant myofascial pain.[16]

E. Diagnostic Approaches

Accurate diagnosis is the cornerstone of effective management, yet it remains a challenge due to the unconscious nature of the condition. Up to 80% of sleep bruxers are unaware of their habit until a partner hears the noise or a dentist identifies tooth wear.[4] To standardize research and clinical practice, an international grading system has been proposed.[3]

- *The Graded Diagnostic System:*

- *Possible Bruxism:*

Diagnosis is based solely on self-report (questionnaires) or the report of a sleep partner. While useful for large epidemiological screenings, this method has low reliability and sensitivity because it relies on the patient's awareness, which is notoriously poor for sleep behaviors.[3]

- *Probable Bruxism:*

Diagnosis is based on self-report plus clinical inspection. Clinical signs include abnormal tooth wear (attrition) that matches between arches, hypertrophy of the masseter muscles (boxy jaw appearance), and indentations on the tongue or cheek (linea alba/morsicatio buccarum).[2] This is the standard for most clinical dental settings.

- *Definite Bruxism:*

This requires confirmation via instrumental methods, specifically polysomnography (PSG) or electromyography (EMG).[4] PSG is considered the "gold standard" because it can differentiate true sleep bruxism from other sleep-related motor activities such as epilepsy, oromandibular myoclonus, or REM behavior disorder.[21]

- *Clinical Utility and Differential Diagnosis:*

While PSG is the most accurate tool, its high cost, labor intensity, and the need for a sleep laboratory limit its use primarily to research and complex clinical cases.[4] For general dental and medical practice, a "Probable" diagnosis based on a thorough clinical examination and history is usually sufficient to initiate management.[2] It is vital to differentiate bruxism from other conditions; for instance, tooth wear can also be caused by acid erosion (GERD) or abrasion, and face pain can arise from neuropathic sources.

F. Clinical Management Strategies and Therapeutics

Currently, there is no "cure" that permanently stops bruxism activity derived from the central nervous system.[25] Management is therefore palliative and symptom-oriented, focusing on three goals: protecting the dentition from mechanical damage, reducing muscle activity to alleviate pain, and managing associated comorbidities.[4]

- *Occlusal Splint Therapy:*

Occlusal splints (night guards) are the most common first-line therapy. These are hard or soft appliances, typically made of acrylic resin, worn over the dental arch. They are highly effective at protecting teeth from mechanical attrition and reducing the noise of grinding, which is often the primary complaint of sleep partners.[4] However, their ability to stop the behavior itself is limited. While splints may transiently reduce muscle activity by altering sensory feedback (breaking the pattern), EMG levels often return to baseline as the patient adapts to the device.[26] Furthermore, splints must be used with caution in patients with OSA. Standard maxillary splints can sometimes aggravate respiratory events by reducing intraoral space or allowing the mandible to fall back; in such cases, mandibular advancement devices (MADs) are the preferred alternative.[6]

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➢ *Pharmacological Interventions:*

When bruxism is severe, painful, or induced by necessary medication, pharmacological strategies may be employed as an adjunct to mechanical therapy.[25]

- *Botulinum Toxin (Botox)*

Injections of Botulinum toxin type A into the masseter and temporalis muscles have gained significant popularity. The toxin induces temporary paralysis by blocking acetylcholine release at the neuromuscular junction.[27] A randomized trial comparing Botox to splints found that Botox was significantly more effective at reducing myofascial pain scores in bruxers.[26] It is particularly indicated for severe, refractory cases or those involving neurological movement disorders. While it reduces the force of contraction (thereby mitigating damage and pain), it does not necessarily stop the central neural drive to brux; the brain still sends the signal, but the muscle cannot respond as forcefully.[27]

- *Managing Drug-Induced Bruxism*

For patients suffering from antidepressant-induced bruxism, the addition of Buspirone (5–10 mg) has been shown to effectively alleviate symptoms.[18] Buspirone is a partial serotonin agonist that may modulate the serotonergic inhibition of dopamine. Other strategies include reducing the antidepressant dose or switching to an agent with a different profile, such as bupropion.[18]

- *Other Agents*

Clonidine (an alpha-2 agonist) has been shown to reduce sleep bruxism activity by approximately 60% in acute settings, likely by dampening autonomic sympathetic tone, though risks of hypotension limit its use.[25] Clonazepam may improve sleep quality and reduce leg and jaw movements, but its high risk of dependency and tolerance limits its long-term application.[25]

- *Behavioral and Cognitive Therapies:*

Behavioral approaches aim to alter the habit through awareness, relaxation, and habit reversal.[25] Cognitive-behavioral therapy (CBT) can be particularly effective for Awake Bruxism by helping patients recognize triggers and practice "jaw relaxation" techniques (e.g., the "lips together, teeth apart" mantra). Biofeedback, involving auditory or vibratory signals triggered by clenching, can reduce EMG episodes during use.[25] However, these methods often lack long-term efficacy once the feedback mechanism is removed. General sleep hygiene measures (reducing caffeine, maintaining a consistent sleep schedule, reducing blue light exposure) are universally recommended but rarely sufficient as a monotherapy.[4]

- *Pediatric Management:*

Management in children differs significantly from adults. Given the self-limiting nature of pediatric bruxism and the potential for devices to interfere with craniofacial growth, irreversible occlusal adjustments or rigid splints are generally avoided.[17] The primary management strategy is "observation and reassurance".[10] However, screening for airway obstruction is critical. If tonsillar hypertrophy or adenoid issues are identified, referral to an otolaryngologist for Adenotonsillectomy (T&A surgery) is the treatment of choice.[23] Studies show this surgery often resolves the bruxism alongside the respiratory issue, further confirming the airway-bruxism link in this demographic.[23]

II. DISCUSSION

- The literature reviewed herein illustrates a fundamental transition in the clinical understanding of bruxism, moving from a dental-centric model of "occlusal disharmony" to a complex, centrally regulated model involving neurochemistry and sleep physiology.[1,5] This shift has profound implications for the Oral and Maxillofacial Surgeon and the general practitioner. The acknowledgment that bruxism is primarily an arousal-related movement disorder[6] rather than a dental pathology requires a change in diagnostic mindset. Clinicians can no longer rely solely on intraoral signs like wear facets to gauge the severity of the condition, as "perceptual sensitization" often leads to a mismatch between clinical signs and patient-reported pain.[16]
- A critical theme emerging from recent data is the "Protective Hypothesis," particularly regarding the relationship between Sleep Bruxism (SB) and Obstructive Sleep Apnea (OSA).[6] The physiological evidence suggesting that Rhythmic Masticatory Muscle Activity (RMMA) events may serve to reopen the airway during sleep challenges the traditional therapeutic reflex of prescribing a standard occlusal splint for every grinder. If bruxism is indeed a compensatory mechanism for hypoxia, suppressing it or introducing a bulky maxillary appliance that invades the tongue space could theoretically exacerbate the respiratory obstruction.[6] This necessitates a comprehensive screening protocol where the clinician must rule out OSA before initiating appliance therapy.
- Furthermore, the management of bruxism remains a challenge of palliation rather than cure. While occlusal splints remain the gold standard for tooth protection, the evidence suggests they do little to arrest the central neural drive.[26] This limitation highlights the importance of adjunctive therapies, particularly in refractory cases. The use of Botulinum toxin offers a promising avenue for reducing the mechanical load on the TMJ and alleviating myofascial pain, yet it is not without cost and transience.[27] Similarly, the management of antidepressant-induced bruxism requires a collaborative approach between the dentist and the prescribing psychiatrist to balance mental health needs with stomatognathic integrity.[18]

Ultimately, the distinction between "Probable" and "Definite" bruxism remains a significant hurdle in both research and practice.[3] Until portable, cost-effective EMG/PSG devices become common place in dental settings, the "probable" diagnosis will likely remain the clinical standard. However, by integrating the psychosocial, respiratory, and pharmacological profiles of the patient, clinicians can move beyond simple mechanical protection toward a more holistic management strategy that addresses the specific drivers of the behavior in each individual.

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III. CONCLUSION

Bruxism represents a multifactorial behavior rooted in complex interactions between neurochemical regulation, sleep physiology, psychosocial influences, and, in some cases, airway dynamics. The shift from an occlusal-based theory to a centrally mediated model has redefined both diagnosis and management, emphasizing the need to evaluate patients beyond dental findings alone. The relationship between Sleep Bruxism and obstructive sleep apnea highlights the importance of airway assessment before introducing occlusal appliances, as splints may worsen respiratory compromise in susceptible individuals. Management remains primarily protective, with occlusal splints offering structural safeguarding rather than altering central neural activity. Adjunctive options such as botulinum toxin, behavioral interventions, and pharmacologic strategies provide symptom relief in selected cases. Ultimately, effective care requires a holistic approach that integrates psychosocial, respiratory, and pharmacological considerations, enabling clinicians to address both the manifestations of bruxism and the underlying factors that sustain the behavior in each individual.

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