International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
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ARTICLE TITLE	MULTIDISCIPLINARY MANAGEMENT OF AWAKE AND SLEEP BRUXISM: A LITERATURE REVIEW
DOI	https://doi.org/10.31435/ijitss.1(49).2026.5135
RECEIVED	12 December 2025
ACCEPTED	25 February 2026
PUBLISHED	09 March 2026
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MULTIDISCIPLINARY MANAGEMENT OF AWAKE AND SLEEP BRUXISM: A LITERATURE REVIEW

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ABSTRACT

Introduction: Bruxism is currently understood as a range of jaw muscle activities and teeth contact that occur during sleep and/or wakefulness. Awake bruxism can be distinguished from sleep bruxism based on differences in etiology, comorbidities, and the spectrum of muscle activity and clinical manifestations. Although the exact cause of bruxism remains unclear, it is considered to be multifactorial. This article aims to highlight both awake and bruxism and explore various approaches to their management.
Aim of the Study: The aim of this article is to review current knowledge on awake and sleep bruxism and increase awareness of its multidisciplinary management.
Materials and Methods: A literature review was conducted using the PubMed and Google Scholar databases. The following keywords were used in various combinations: "bruxism," "awake," "sleep" "management," "treatment," and "oral health." Fourty four relevant articles published between 2001 and 2025 were selected for analysis.
Results: The most common current approaches to managing awake and sleep bruxism include occlusal splints, mandibular advancement devices, cognitive-behavioral therapy (CBT), biofeedback therapy, mindfulness techniques, self-awareness and self-regulation strategies, physical therapy (such as physiotherapy, exercise, and dry needling), botulinum toxin injections, and non-steroidal anti-inflammatory drugs (NSAIDs) for pain exacerbation.
Conclusion: Bruxism and its associated symptoms can significantly affect patients' quality of life. Early identification and appropriate management can help reduce facial muscle soreness, headaches, and general fatigue and pathological tooth wear.

KEYWORDS

Awake, Sleep, Bruxism, Treatment, Symptoms, Oral Health, Management

CITATION

Aleksandra Karolak, Jakub Michał Lichoń, Mateusz Józef Goldyn, Ewa Wietrzykowska, Piotr Trojan, Andrzej Wydrych, Patryk Pindlowski, Agata Karasiewicz, Maja Kuklo, Marta Kras. (2026) Multidisciplinary Management of Awake and Sleep Bruxism: A Literature Review. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.5135

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I. Introduction

In recent years, the clinical conceptualization of bruxism has evolved from a strictly pathological condition to a spectrum of masticatory muscle activities with potential physiological or protective significance (Lal et al., 2024; Manfredini et al., 2022). In otherwise healthy individuals, bruxism is increasingly recognized as a muscular behavior rather than a primary disorder (Manfredini et al., 2020). Nonetheless, when the intensity and frequency of this activity exceed the individual's adaptive capacity, it can manifest in clinical complications, including myalgia, temporomandibular joint (TMJ) disorders, prosthodontic failures, and advanced tooth wear (Lal et al., 2024; Manfredini et al., 2020, 2022; Manfredini & Lobbezoo, 2010; Svensson et al., 2008; Wetselaar et al., 2016).

Modern nomenclature distinguishes two primary circadian phenotypes: Awake Bruxism (AB) and Sleep Bruxism (SB) (de Baat et al., 2021). While they share certain muscular commonalities, they differ significantly in etiology, pathophysiology, and clinical manifestations. According to the current international consensus, AB is defined as masticatory muscle activity during wakefulness characterized by repetitive or sustained tooth contact and/or bracing and thrusting of the mandible (Manfredini et al., 2020). Conversely, SB is characterized as a sleep-related rhythmic (or non-rhythmic) masticatory activity, typically associated with micro-arousals during non-REM sleep (Manfredini et al., 2020).

The etiology of both phenotypes is now understood to be centrally, rather than peripherally, mediated. SB is strongly associated with autonomic nervous system fluctuations, neurotransmitter imbalances (specifically dopaminergic and serotonergic systems), and psychosocial factors such as high-state anxiety. Furthermore, a significant correlation exists between SB and Obstructive Sleep Apnea (OSA), where grinding may function as a subconscious compensatory mechanism to maintain airway patency by protruding the mandible (Martynowicz et al., 2019). AB, in contrast, is more intimately linked to psychosocial distress, environmental stress, and emotional regulation. Both forms can be classified as primary (idiopathic) or

secondary, the latter often resulting from neurological disorders or as an adverse effect of medications, such as Selective Serotonin Reuptake Inhibitors (SSRIs) (de Baat et al., 2021; Manfredini et al., 2020; Manfredini & Lobbezoo, 2009).

Given this etiological complexity, a singular treatment approach is often insufficient. This review article delineates the current multidisciplinary management strategies for AB and SB. For AB, management focuses on cognitive-behavioral interventions, mindfulness-based stress reduction, and biofeedback-assisted habit reversal to address the psychogenic drivers. For SB, therapy prioritizes mechanical protection and the stabilization of the stomatognathic system through occlusal stabilization splints or Mandibular Advancement Devices (MAD) when respiratory comorbidities are present (Carra et al., 2012; Khoury et al., 2016; Martynowicz et al., 2019). Finally, adjunctive interventions—including Botulinum Toxin Type A (BoNT-A) injections, dry needling, and targeted pharmacotherapy—are evaluated as cross-phenotype solutions for managing muscular hypertonicity and acute pain (Ågren et al., 2020; Blasco-Bonora & Martín-Pintado-Zugasti, 2017).

II. Materials and Methods

A comprehensive literature review was conducted to synthesize current evidence regarding the etiology, symptomatology, and multidisciplinary management of both awake bruxism (AB) and sleep bruxism (SB).

1.Search Strategy and Data Sources

The literature search was performed across the PubMed/MEDLINE and Google Scholar databases, covering publications from January 2001 to December 2025. The search employed various combinations of Medical Subject Headings (MeSH) terms and keywords, including: *"bruxism," "awake bruxism," "sleep bruxism," "etiology," "therapeutic management," "psychosocial factors," "occlusal splints," and "botulinum toxin."* To ensure a high level of clinical relevance, the search was restricted to articles published in English and peer-reviewed journals.

2. Inclusion and Exclusion Criteria

Studies were eligible for inclusion if they met the following criteria:

-primary focus on the diagnosis, symptoms or management of AB and/or SB;

-clinical trials, systematic reviews, meta-analyses, and high-impact consensus papers;

-studies involving human subjects (adults and children).

Exclusion criteria involved:

-articles focusing solely on non-masticatory movement disorders;

-papers with insufficient statistical data or those published in predatory journals;

-abstracts from conferences without full-text availability.

3. Selection Process

The initial search using the broad term 'bruxism' yielded a vast number of results (approximately 89,000 on Google Scholar); therefore, a stringent filtering strategy was applied to ensure the selection of high-quality, peer-reviewed evidence. The search was narrowed using specific Boolean operators (AND, OR) and refined keywords, including: *'awake bruxism' AND 'management'*, *'sleep bruxism' AND 'etiology'*, and *'multidisciplinary treatment'*. After removing duplicates and screening titles and abstracts, 52 articles underwent full-text assessment. Ultimately, 44 articles were selected based on their methodological rigor and contribution to the current understanding of multidisciplinary bruxism management.

III. Etiological Determinants of Awake and Sleep Bruxism

The shift in the international consensus (Lobbezoo et al., 2018) fundamentally redefined bruxism as a muscle behavior rather than a simple movement disorder. However, the underlying mechanisms driving this behavior differ significantly between the two circadian phenotypes.

III.A. Pathophysiology and Etiology of Sleep Bruxism (SB)

Sleep bruxism is currently understood as a **centrally mediated phenomenon**, primarily regulated by the autonomic nervous system and sleep architecture, rather than peripheral factors like dental occlusion.

1. The Role of Sleep Architecture and Micro-arousals

The most widely accepted theory for SB is the "Arousal Theory." Research using polysomnography (PSG) has demonstrated that over 80% of SB episodes occur during Non-REM (NREM) sleep, particularly in the transitions between sleep stages N1 and N2. These episodes are almost always preceded by a micro-arousal—a transient shift in sleep depth characterized by a sudden increase in heart rate, respiratory effort, and sympathetic dominance. This "autonomic surge" occurs approximately 4–8 seconds before the actual rhythmic masticatory muscle activity (RMMA). Therefore, SB is viewed as part of an exaggerated arousal response,

where the central nervous system "wakes up" the masticatory muscles (Carra et al., 2012; Khoury et al., 2016; Martynowicz et al., 2019).

2. Neurotransmitter Imbalances and the Basal Ganglia

The central drive of SB is also linked to the dopaminergic and serotonergic systems within the basal ganglia. Dopamine plays a crucial role in regulating motor control; fluctuations in dopamine levels (or receptor sensitivity) can lead to involuntary muscle movements. This is evidenced by the "Secondary Bruxism" seen in patients taking SSRIs or dopamine antagonists, where the altered neurochemical environment triggers RMMA as a side effect (de Baat et al., 2021; Khoury et al., 2016; Singh et al., 2019).

3. Genetic Predisposition

Emerging evidence suggests a strong hereditary component. Longitudinal studies have shown that individuals with a first-degree relative who exhibits SB are up to four times more likely to brux themselves. This suggests that certain genetic polymorphisms, particularly those affecting serotonin receptors (5-HT2A), may predispose individuals to heightened nocturnal muscle activity (Khoury et al., 2016; Ohayon et al., 2001).

III.B. Psychosocial and Behavioral Drivers of Awake Bruxism (AB)

In contrast to the sleep-related autonomic triggers of SB, **Awake Bruxism** is intimately tied to the patient's emotional state, environmental stressors, and cognitive processing.

1. The Psychosomatic Link: Stress and Anxiety

AB is predominantly considered a physical manifestation of psychosocial distress. Individuals with high levels of **state-anxiety** or those who score high on neuroticism scales are significantly more prone to daytime clenching and bracing. Unlike the rhythmic grinding of SB, AB often manifests as a "tonic" (sustained) contraction, which serves as a subconscious coping mechanism for emotional regulation. The "stiff upper lip" or "clenching one's teeth" are not just metaphors but physiological realities for patients under chronic work-related or domestic stress (J.-D. Orthlieb et al., 2013).

2. Stress-Coping Styles and Personality Traits

Research by Marcolino-Cruz et al. (2022) highlights that it is not the *amount* of stress, but the *type of coping* that determines bruxism severity. "Emotion-oriented" coping styles—characterized by internalization, rumination, and avoidance—are more strongly correlated with AB than "task-oriented" styles. These individuals tend to maintain elevated muscle tone throughout the day, leading to what is known as **"jaw bracing"** or "tooth contact habit," which eventually results in myofascial fatigue.

3. Cognitive Awareness and Habitual Patterns

AB is also maintained through maladaptive habit loops. For many, clenching becomes a learned motor response to concentration (e.g., while working at a computer) or physical exertion. This is often referred to as a "para-functional habit." Because it occurs during wakefulness, it is theoretically under the control of the prefrontal cortex, which is why behavioral interventions like Ecological Momentary Assessment (EMA) are so effective—they interrupt the subconscious habit loop and bring the muscle activity into the patient's conscious awareness (Bracci et al.,2022; Manfredini et al. 2016).

III.C. Secondary Bruxism: Clinical and Pharmacological Etiologies

While primary bruxism is considered idiopathic and arises without an evident underlying medical cause, **Secondary Bruxism** is a symptomatic manifestation of another clinical condition or a side effect of exogenous substances and medications. Understanding these secondary drivers is crucial for a multidisciplinary differential diagnosis (Singh et al., 2019).

1. Pharmacological Induction: The "SSRI Paradox" One of the most complex etiological factors in secondary bruxism is the administration of **Selective Serotonin Reuptake Inhibitors (SSRIs)** and **Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs)** (de Baat et al., 2021; Singh et al., 2019). Medications such as fluoxetine, sertraline, and venlafaxine are frequently prescribed to patients with anxiety and depression—both of which are primary risk factors for Awake Bruxism. Paradoxically, these agents can induce or significantly exacerbate both AB and SB.

The proposed mechanism is the **serotonin-dopamine interaction** within the extrapyramidal system. High synaptic levels of serotonin can lead to an inhibition of dopaminergic transmission in the basal ganglia, specifically within the nigrostriatal pathway. This "hypodopaminergic" environment can trigger involuntary orofacial movements, including clenching and rhythmic grinding. This condition, often termed "medication-induced bruxism," usually manifests within the first few weeks of therapy or following a dose increase (de Baat et al., 2021; Singh et al., 2019).

2. Neurological and Systemic Comorbidities Secondary bruxism is frequently observed in patients with neurological disorders, where the central regulation of motor activity is compromised. Conditions such as **Parkinson's disease**, **Huntington's disease**, and various forms of **orofacial dystonia** often present with bruxism-like muscle activity. Furthermore, there is a strong etiological link between SB and **Gastroesophageal Reflux Disease (GERD)**. Current research suggests that nocturnal grinding may be a secondary protective reflex; the chemical stimulus of gastric acid in the esophagus triggers a micro-arousal and subsequent RMMA to stimulate bicarbonate-rich salivary flow, thereby neutralizing the acid(Carra et al., 2012).

3. The Role of Exogenous Stimulants and Lifestyle Factors Lifestyle choices significantly influence the neurochemical and physiological triggers of bruxism. **Nicotine** is a potent etiological factor; its agonistic effect on nicotinic acetylcholine receptors increases central dopaminergic activity and sympathetic tone, leading to a higher frequency of RMMA events in sleep bruxers (Carra et al., 2012; Khoury et al., 2016; Minakuchi et al., 2022; Ohayon et al., 2001).

Similarly, **alcohol consumption** disrupts the normal progression of sleep stages. The rebound effect of alcohol during the second half of the night leads to increased sleep fragmentation and sympathetic surges, which are the primary catalysts for nocturnal bruxism episodes (de Baat et al., 2021; Singh et al., 2019).

4. Sleep-Disordered Breathing: The SB-OSA Link A critical secondary etiology for SB is its association with **Obstructive Sleep Apnea (OSA)**. In these cases, bruxism is often viewed as a subconscious compensatory mechanism (Martynowicz et al., 2019; Minakuchi et al., 2022). The rhythmic contraction of the masseter and suprahyoid muscles during a respiratory event may serve to stabilize the upper airway or facilitate mandibular protrusion, helping to restore airway patency. In such patients, treating the underlying respiratory obstruction (e.g., with CPAP or MAD) often leads to a significant reduction in secondary sleep bruxism activity (Martynowicz et al., 2019).

IV. Epidemiology and diagnosis

The diagnostic landscape for bruxism has undergone a paradigm shift, transitioning from a symptom-based approach to a comprehensive evaluation of "possible," "probable," and "definite" status. While Awake Bruxism (AB) and Sleep Bruxism (SB) share certain musculoskeletal outcomes, their epidemiological profiles and diagnostic requirements differ significantly.

1. Epidemiology: Age-Dependence and Prevalence

Current data suggest that the prevalence of bruxism in the Western adult population ranges from 8% to 31.4% for AB and 12.8% ± 3.1% for SB [1, 28]. However, the epidemiology of SB is uniquely characterized by a progressive age-dependent decline. Prevalence is highest in children (approximately 14–20%), dropping to 8–13% in young adults, and reaching its lowest levels in the elderly (approximately 3%) (Manfredini et al., 2020). This decline is hypothesized to be linked to the maturation of the central nervous system and age-related changes in sleep architecture.

Recent findings by Stanisic et al. highlight that in specific populations, such as those with temporomandibular disorders (TMD), the prevalence of "possible" AB can reach 50.0%, while systemic conditions increase the risk to 40.1% (Stanisic et al., 2025). In the context of SB, gender distribution appears to be relatively equal, unlike AB, which occasionally shows a slight female predominance in clinical samples due to its psychosocial links.

2. Etiology and Pathophysiology of Sleep Bruxism

Unlike AB, which is primarily driven by psychosocial factors and stress-processing styles (Marcolino-Cruz et al., 2020), SB is recognized as a centrally mediated, neuromotor event. SB is associated with imbalances in the dopaminergic and serotonergic systems. The rhythmic masticatory muscle activity (RMMA) observed in SB is believed to be triggered by a rise in sympathetic nervous system activity, often occurring 4–8 seconds before the actual grinding event (Khoury et al., 2016). Moreover SB is usually linked with sleep architecture, its episodes predominantly occur during Stage N1 and N2 (light sleep) and are rarely seen in REM sleep (Carra et al., 2012). They are almost always preceded by a micro-arousal—a sudden shift in sleep depth accompanied by increased heart rate and respiratory effort (Carra et al., 2012; Khoury et al., 2016; Martynowicz et al., 2019). There is also a genetic factor – research show strong evidence for a hereditary component in SB. Individuals with a first-degree relative who bruxes are up to four times more likely to develop the condition, suggesting a genetic predisposition in primary SB(Khoury et al., 2016; Manfredini et al., 2020).

3. Comorbidities and Global Posture

A critical clinical correlation exists between SB and Obstructive Sleep Apnea (OSA) (Martynowicz et al., 2019; Ohayon et al., 2001). Research suggests that SB may act as a compensatory mechanism; the rhythmic contraction of the masseter muscles may facilitate the stabilization of the upper airway or trigger the protrusion of the mandible to prevent collapse. Furthermore, other sleep-related disorders, such as periodic limb movement disorder (PLMD) and gastroesophageal reflux disease (GERD), frequently co-occur with SB (Carra et al., 2012; Martynowicz et al., 2019).

The systemic nature of the condition is further evidenced by its impact on body posture. Motta et al. (2011) identified that children with bruxism often exhibit a significantly larger cranio-cervical angle (98.99° vs. 89.58°; $p < 0.005$), indicative of forward head posture (FHP) (Saito et al., 2016). This suggests that SB is not merely an isolated dental issue but part of a broader musculoskeletal and physiological complex.

4. Diagnostic "Gold Standards" and Criteria

According to the International Classification of Sleep Disorders (ICSD-3), the diagnosis of bruxism relies on specific clinical and instrumental criteria:

A) Sleep Bruxism (SB): The definitive diagnosis ("Definite SB") requires Polysomnography (PSG) with synchronized audio-video and electromyographic (EMG) leads placed on the masseter and temporal muscles. This allows clinicians to distinguish between RMMA, tooth grinding, and other orofacial activities (e.g., swallowing or coughing) (Carra et al., 2012; Khoury et al., 2016; Martynowicz et al., 2019).

B) Awake Bruxism (AB): The focus remains on real-time monitoring through Ecological Momentary Assessment (EMA) using smartphone applications like BruxApp© (Bracci et al., 2022; Manfredini et al., 2016). EMA is currently the most reliable method for capturing the semi-voluntary clenching or "bracing" characteristic of AB, providing a "probable" or "definite" diagnosis when combined with ambulatory EMG (Bracci et al., 2022).

The high prevalence and age-related variations of bruxism necessitate a clear understanding of its downstream clinical effects on the stomatognathic system, as discussed in the following section.

V. Pathogenesis of Bruxism-Related Clinical Complications

The chronic and repetitive nature of both awake and sleep bruxism exerts significant pathological strain on the components of the stomatognathic system. Understanding the pathogenesis of these complications is essential for establishing a timely multidisciplinary intervention.

1. Temporomandibular Joint (TMJ) Remodeling and Dysfunction. The TMJ is particularly vulnerable to the sustained or rhythmic loading characteristic of bruxism. Unlike functional movements such as mastication or speech, bruxing events involve high-magnitude forces without the interposition of a food bolus, leading to direct intra-articular pressure. Chronic overloading can result in the **micro-traumatization of the bilaminar zone** and the subsequent thinning of the articular disc (Wetselaar et al., 2019).

Pathologically, this may progress to **internal derangement**, most commonly manifested as disc displacement with reduction (DDwR). In severe, long-term cases, the adaptive capacity of the joint is exceeded, leading to degenerative changes such as **osteoarthritis**, characterized by the flattening of the condylar head and the formation of osteophytes. These structural alterations are the primary cause of the restricted mandibular range of motion and joint "clicking" or "crepitus" reported by patients (Smardz et al., 2021; Wetselaar et al., 2019).

2. Myofascial Pain and Muscle Hypertrophy. The muscular response to bruxism involves both structural and biochemical changes. Sustained clenching (AB) and repetitive grinding (SB) lead to **adaptive muscle hypertrophy**, particularly of the masseter and temporalis muscles, which often results in a characteristic "square-jaw" facial appearance. On a cellular level, chronic overwork induces localized ischemia and the accumulation of metabolic byproducts (such as lactic acid), which trigger the formation of **myofascial trigger points** (Smardz et al., 2021).

These hyperirritable spots are responsible for the "tension-type" headaches and referred pain patterns frequently associated with AB. The constant state of muscular hypertonicity can also lead to a "pain-clenching-pain" cycle, where the discomfort from the muscles leads to further protective guarding and increased clenching (Smardz et al., 2021).

3. Structural Degradation of the Dentition and Periodontium The most visible consequence of bruxism is the mechanical degradation of the hard dental tissues. **Attrition**, characterized by the loss of occlusal and incisal enamel, leads to the formation of "wear facets." If left unchecked, this progresses to dentin exposure, resulting in dentinal hypersensitivity and a loss of vertical dimension (Smardz et al., 2019; Wieckiewicz et al., 2014).

Furthermore, the lateral forces exerted during grinding are particularly destructive to the cervical regions of the teeth, contributing to the development of **non-carious cervical lesions (abfractions)**. The periodontium also reacts to these occlusal traumas; in the presence of pre-existing periodontal disease, bruxism can accelerate bone loss and increase tooth mobility due to the widening of the periodontal ligament space (Khoury et al., 2016; Smardz et al., 2019; Wieckiewicz et al., 2014).

VI. Results: Multimodal Management of Awake and Sleep Bruxism

The therapeutic management of bruxism requires a nuanced, bifurcated strategy that addresses the distinct triggers of both awake bruxism (AB) and sleep bruxism (SB). While AB management is centered on psychophysiological regulation and habit reversal, SB therapy primarily focuses on mechanical protection, airway patency, and the mitigation of involuntary nocturnal muscle activity (Manfredini et al., 2020; Minakuchi et al., 2022).

VIA. Mechanical Interventions and Occlusal Splint Therapy

The use of dental appliances remains a cornerstone of bruxism management, particularly for the nocturnal phenotype. **Occlusal stabilization splints** (e.g., Michigan-type hard acrylic guards) are the gold standard for managing **Sleep Bruxism**. Unlike behavioral therapies, these appliances are not intended to eliminate the central drive of SB; rather, they serve a critical protective function by redistributing occlusal forces, preventing pathological tooth wear (attrition), and reducing intra-articular pressure within the temporomandibular joints (TMJ) during rhythmic masticatory muscle activity (RMMA) (Carra et al., 2012; Wetselaar et al., 2019; Wieckiewicz et al., 2014). Moreover, it is crucial to distinguish between hard stabilization splints (Michigan type) and soft resilient appliances. While hard splints facilitate muscle relaxation and joint stabilization, soft appliances may inadvertently increase EMG activity in some patients by triggering a 'chewing reflex,' potentially exacerbating the condition instead of mitigating it (Carra et al., 2012; Ohayon et al., 2001; Saito et al., 2016; Yap & Chua, 2016).

Furthermore, in patients where SB is secondary to respiratory disturbances, **Mandibular Advancement Devices (MAD)** are increasingly utilized (Minakuchi et al., 2022). These devices serve a dual purpose: they protect the dentition while simultaneously protruding the mandible to maintain upper airway patency, thereby reducing the micro-arousals that often trigger grinding episodes. In the context of **Awake Bruxism**, dental appliances may also be employed as biofeedback tools; the physical presence of an orthotic can increase the patient's awareness of their jaw position, discouraging daytime clenching and bracing (Yap & Chua, 2016).

VIB. Behavioral Therapy and Awareness Training

Behavioral interventions aim to decouple the stress response from the masticatory system. **Cognitive-behavioral therapy (CBT)** is highly effective for AB, focusing on the reprogramming of mandibular resting posture and stress self-management. Interestingly, interdisciplinary studies, such as those by Trindade et al., have shown that combining CBT with **occlusal splint therapy** for SB patients yields significantly better muscle relaxation than dental monotherapy alone (Yap & Chua, 2016). **Habit Reversal Training (HRT)** and **Biofeedback**—utilizing EMG-linked devices or mobile applications like BruxApp—allow patients to gain conscious control over involuntary muscle activity, providing real-time data that is essential for long-term habit modification (Lobbezoo et al., 2013; Manfredini et al., 2016).

VIC. Stress Management

Since psychosocial distress is a primary driver of AB and a contributing factor to the severity of SB, stress-reduction techniques are vital. **Mindfulness, meditation, and yoga** assist in autonomic regulation, helping patients achieve a state of physical and mental well-being that reduces the frequency of clenching episodes (Guaita & Quera-Salva, 2013; Huynh et al., 2006). While **hypnotherapy** is often viewed as a complementary treatment for AB, it has demonstrated specific efficacy in reducing the intensity of nocturnal grinding in SB cases, making it a versatile tool in a multidisciplinary protocol (Polmann et al., 2019).

VID. Physical Therapy and Manual Interventions

Physiotherapy serves as an essential modality for managing the myofascial fatigue and pain associated with both circadian phenotypes. Common approaches include manual therapy, postural training, and **dry needling**. Manual techniques—such as myofascial release and joint mobilization—aim to restore functional balance within the stomatognathic and cervical systems.

Research by Motta et al. (2011) suggests a profound mechanical link between **forward head posture** and bruxism-related muscle tension. Consequently, manual therapy targeting the cervical and upper thoracic spine is crucial to alleviate nociceptive input and facilitate global neuromuscular relaxation. (Saito et al., 2016). **Dry needling** has emerged as a particularly effective invasive technique for deactivating myofascial trigger points in the masseter and temporalis, providing rapid pain relief for patients suffering from both daytime clenching and nighttime grinding (Ahn et al., 2023).

VIE. Pharmacotherapy and Botulinum Toxin

Pharmacological intervention is typically considered an adjunctive or second-line treatment. In acute cases, **Non-steroidal anti-inflammatory drugs (NSAIDs)** are utilized to manage localized inflammation and pain (Alhudaithi et al., 2023). However, clinicians must remain vigilant regarding the "SSRI paradox," where certain antidepressants may induce or exacerbate bruxism as a side effect (Przystańska et al., 2019).

Botulinum toxin type A (BoNT-A) injections represent a significant advancement in treating moderate-to-severe cases of both AB and SB. By inhibiting acetylcholine release at the neuromuscular junction, BoNT-A attenuates the maximum force of muscle contraction. In **Sleep Bruxism**, this effectively prevents the severe mechanical degradation of the teeth and TMJ during involuntary RMMA episodes. In **Awake Bruxism**, it reduces the physical capacity for clenching, providing a therapeutic "window" during which behavioral and physical therapies can be more effectively implemented (Colonna et al., 2020; Guo et al., 2017; Sendra et al., 2023).

VII. Discussion

The findings of this comprehensive review underscore that bruxism is a multifaceted phenomenon that transcends the traditional boundaries of clinical dentistry. While Sleep Bruxism (SB) has historically been more thoroughly investigated, our analysis highlights a critical need for a deeper understanding of Awake Bruxism (AB), particularly given its distinct psychogenic drivers and its tendency to be underdiagnosed in routine clinical practice (Lobbezoo et al., 2018).

A central theme emerging from the literature is the stark contrast in the therapeutic "target" for each phenotype. Sleep bruxism is increasingly viewed through the lens of sleep physiology and autonomic regulation. Consequently, the reliance on occlusal splints remains a clinical mainstay. However, the discussion within the scientific community is shifting from seeing splints as a "cure" to recognizing them as a vital "damage control" strategy. These appliances protect the integrity of the stomatognathic system from the involuntary, high-magnitude forces generated during nocturnal RMMA events (Carra et al., 2012). Moreover, the integration of Mandibular Advancement Devices (MAD) into the treatment protocol for SB patients with comorbid respiratory issues represents a significant leap toward a multidisciplinary approach that bridges dental sleep medicine with pulmonology.

In contrast, Awake Bruxism management is anchored in the biopsychosocial model. Because AB is so intimately linked to stress-coping mechanisms, emotional internalization, and environmental stressors, the literature increasingly supports psychological interventions such as Cognitive-Behavioral Therapy (CBT) and Mindfulness-Based Stress Reduction (MBSR)(Huynh et al., 2006; Polmann et al., 2021). Our review suggests that for the awake bruxer, the goal is not mechanical protection but neuromuscular reprogramming. This involves the transition from subconscious clenching to a conscious "physiological rest position" through the use of biofeedback and interoceptive awareness (Huynh et al., 2006).

The role of physiotherapy serves as a vital bridge between both circadian phenotypes. Whether the muscle hyperactivity occurs during the day or night, the resulting myofascial fatigue, trigger points, and cervical imbalances require manual intervention. The correlation identified by Motta et al. (2011) between forward head posture and bruxism emphasizes that clinicians must look beyond the oral cavity. Addressing the cranio-cervical complex is essential to break the cycle of chronic pain and postural compensation that can perpetuate both AB and SB symptoms.

Furthermore, the emergence of Botulinum Toxin Type A (BoNT-A) provides a cross-phenotype pharmaceutical tool that manages the peripheral "exhaust" of a centrally-driven problem (Colonna et al., 2020; Guo et al., 2017; Sendra et al., 2023). While BoNT-A does not address the psychological triggers of AB or the micro-arousals of SB, it effectively mitigates the destructive forces of both, offering a secondary line of defense for treatment-resistant patients (Sendra et al., 2023).

Clinical Implications and Future Directions

Clinicians should adopt a proactive screening protocol for all patients presenting with jaw pain, masseteric hypertrophy, or TMD symptoms. This review suggests that a multidisciplinary team—comprising dentists, physical therapists, psychologists, and sleep specialists—is no longer a luxury but a necessity for optimal outcomes.

Future research should focus on the long-term efficacy of combined therapies, such as the synergistic effect of splint therapy and CBT, and the development of more accessible EMA (Ecological Momentary Assessment) tools (Manfredini et al., 2013; Manfredini et al., 2016). There is also a pressing need for longitudinal studies to determine if treating SB can effectively prevent the progression of TMD or if the relationship is purely correlative. Ultimately, the goal of modern bruxism management is to move away from "monotherapy" toward a personalized, multimodal framework that addresses the unique physiological and psychological profile of each patient.

VIII. Conclusions

The evolution of bruxism from a perceived dental pathology to a recognized motor activity necessitates a fundamental shift in clinical management. This review underscores that while **Sleep Bruxism (SB)** and **Awake Bruxism (AB)** share a common nomenclature and certain musculoskeletal consequences, they represent distinct physiological events requiring tailored, multidisciplinary interventions.

A significant conclusion drawn from the current literature is the necessity of a **dual-track therapeutic approach**. For **Sleep Bruxism**, the evidence confirms that management must prioritize the protection of the stomatognathic system and the stabilization of sleep physiology. The use of **occlusal stabilization splints** remains a critical defensive strategy to prevent irreversible dental attrition and joint degradation (Mainieri et al., 2012; Yap & Chua, 2016). Furthermore, the strong correlation between SB and respiratory micro-arousals suggests that screening for sleep-disordered breathing is no longer optional but a vital component of the dental diagnostic protocol(Lobbezoo et al., 2020; Minakuchi et al., 2022).

In contrast, the management of **Awake Bruxism** demands a transition from passive mechanical protection toward active **behavioral and psychotherapeutic engagement** (Huynh et al., 2006; Polmann et al., 2021; Smardz et al., 2019; Yap & Chua, 2016). Since AB is primarily driven by psychosocial distress, anxiety, and maladaptive stress-coping mechanisms, long-term success is predicated on habit reversal, biofeedback, and cognitive-behavioral interventions. The "gold standard" for AB is increasingly defined by the patient's ability to achieve autonomous neuromuscular control over their masticatory activity during wakefulness (Polmann et al., 2021).

Furthermore, this review highlights **physiotherapy** as the essential connective tissue in bruxism therapy. Regardless of the circadian timing of the muscle activity, the resulting myofascial pain, trigger points, and cranio-cervical imbalances require manual intervention and postural correction to prevent the chronicity of symptoms. The integration of adjunctive therapies, such as **Botulinum Toxin Type A**, offers a potent peripheral solution for severe cases, though it must be viewed as a temporary "window of relief" rather than a definitive cure for the underlying central drive(Sendra et al., 2023).

In summary, effective therapy for both awake and sleep bruxism must transcend conventional dental monotherapy. A comprehensive, multidisciplinary strategy—incorporating patient education, sleep hygiene optimization, habit retraining, and specialized physical therapy—offers the most robust framework for mitigating the clinical impact of bruxism. Future research should prioritize longitudinal studies that evaluate the long-term efficacy of these combined modalities, aiming to establish a standardized, evidence-based protocol for the holistic management of the bruxing patient.

Disclosures:

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All authors have read and agreed with the published version of the manuscript.

Funding Statement: The study did not receive special funding.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.
Conflict of interest: No conflict of interest.
Declaration of the use of generative AI and AI-assisted technologies in the writing process: In preparing this work, the authors used ChatGPT for the purpose of grammar checking and improving the readability of the text. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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