

An overview of sleep bruxism and management, a review

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The concept of sleep bruxism (SB) has evolved over the past several decades. Many theories and hypotheses have been proposed as to the definition, pathophysiology, and management of SB. An international consensus was attained on the definition of SB as masticatory muscle activities that occur during sleep (characterized as rhythmic or non-rhythmic). The etiology of bruxism is not well known, but it is believed to be multifactorial. Assessment tools of bruxism may be made by patient report, clinical interview, clinical examination, and instrumental assessment, leading to diagnostic criteria that challenge to provision of scientifically proven information regarding SB management. Several investigations have been carried out to confirm the safety and efficacy of various kinds of treatment aimed at solving bruxism. In addition, the methodological quality, the different criteria of SB diagnosis, and the lack of homogeneity in terms of study design can result in poor meta-analysis data, mislead readers, and induce malpractice. These issues are also discussed.

Keywords: assessment, etiology, management, sleep bruxism

How to cite: Leeraphongnan J, Wichienroj P. An overview of sleep bruxism and management, a review. M Dent J 2025;45(3): 214-228.

Introduction

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 bruxism (AB) and sleep bruxism (SB), based on when the activity occurs [1-3]. A recent international consensus was attained on the definition of bruxism as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible [3]. SB is a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder, while AB is a masticatory muscle activity during

wakefulness that is characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder AB [3]. There is increasing evidence of the role of the autonomic nervous system, genetics, and comorbidities in the genesis of SB. The scientific literature seems to refute the role of dental occlusion in the causation of bruxing movements. As per the literature, there has been a paradigm shift in the definition and genesis of SB and its possible dental implications and management, which also highlights the need for succinct scientific studies in this regard [4]. A recent study showed SB may enhance masticatory performance in healthy dentate adults without temporomandibular disorder pain or bruxism-related jaw symptoms by increasing occlusal force and enlargement of the occlusal contact area [5]. By the way, focus is given to

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Received: 11 September 2025

Revised: 12 October 2025

Accepted: 18 October 2025

the consequences with a negative impact on the individual, mostly due to overloading by the repetitive masticatory muscle activities that characterize bruxism [6]. Therefore, proper management approaches have been applied to relieve harmful effects of SB.

This article aims to provide an overview of the understanding and current practice in etiology, assessment tools, diagnosis, and management of SB.

Etiology

SB is no longer considered a parasomnia nor is its etiology believed to be based on purely mechanical factors or psychological issues [4]. The etiology of bruxism is not well known, but it is agreed that the etiology is multifactorial [4, 7].

Initially, the occlusion concept was popularized in a classical article that believed that occlusal disharmony or premature tooth contacts as the principal etiological factor of bruxism [8]. Occlusal adjustment was reported to diminish or stop this sleep activity. However, several studies show that SB activity is not reduced by occlusal therapy [9]. The evaluated occlusal and functional parameters do not differ between SB subjects and non-sleep bruxism subjects [10]. Also, there is no reliable evidence that demonstrates that occlusal interferences can cause SB or stop it [11].

Psychological stress factors were once considered major factors in the etiology of SB. Research seems to find such an association when bruxism is diagnosed by self-report [12-14]. However, in polysomnographic studies, there are no statistically significant correlations between the intensity of SB and psychological stress [15-16]. While systematic reviews and meta-analysis articles show a significant association between bruxism and stress symptoms, caution should be

exercised due to the moderate risk of bias and low quality of evidence included in these reviews to support the association [17-19]. Stronger methodological and standardized prospective studies (i.e., participant selection from the general population, standardized clinical and test protocols with long-term follow-up) are needed to increase the quality of evidence in order to confirm this possible association and to help clarify the direction of the association between these variables [17-19]. In terms of anxiety, current literature shows controversy regarding an association between SB and generic symptoms of anxiety in adults. It seems that some specific symptoms of the anxiety disorders spectrum might be associated with probable SB [20].

B Kuang *et al.* [21] presented the systematic review identified sleep-related disorders that were possibly associated with SB, including obstructive sleep apnea (OSA), restless legs syndrome/periodic limb movements during sleep (RLS/PLMS), sleep-related gastroesophageal reflux disease (GERD), insomnia, Parkinson's Disease (PD), rapid eye movement behavior disorders (RBD), and sleep-related epilepsy. Within the main limitation of this review (i.e., large methodological differences between the included studies in the assessment of SB and of other sleep disorders), the prevalence of SB in patients with OSA, RLS/PLMS, sleep-related GERD, RBD, and sleep-related epilepsy was higher than that in the general population, which brought more light on the importance of routine SB screening in patients with the aforementioned sleep-related disorders. Even though the specific mechanisms behind the associations between SB and other sleep-related disorders have not been identified yet, considering all the available evidence, sleep arousals could be a common factor with which all the identified disorders are associated, except RBD and PD [21].

There is a notable prevalence of bruxism in patients with OSA, suggesting possible shared pathophysiological mechanisms, although the exact nature of this relationship remains uncertain [22]. Deshui Li *et al.* [23] conducted a large-scale polysomnographic study, and they found that SB is highly prevalent, about 50%, in adults with obstructive sleep apnea. Similarly, Dadphan *et al.* [24] studied patients with OSA who underwent split-night polysomnography. They found that almost half were observed to have SB. The bruxism episode index is positively associated with the apnea-hypopnea index in patients with mild to moderate obstructive sleep apnea, defined as an apnea-hypopnea index of <30 events/h [25]. Some researchers propose that SB may play a protective role during respiratory-related arousal [25].

Rintakoski *et al.* [26] examined the role of genetic and environmental factors in the phenotypic variance of bruxism in a large population-based cohort of young adult twins in Finland. They found that genetic factors account for a substantial proportion of the phenotypic variation of the liability to sleep-related bruxism, with no gender difference in its genetic architecture [26]. There are some genes related to bruxism in a study exploring the contribution of genetic variants in dopaminergic pathways to bruxism development [27]. The genome-wide association study (GWAS) reveals a significant correlation between the rs10193179 variant of the Myosin IIIB gene (MYO3B) and sleep bruxism [28]. A Pecori *et al.* [29] studied the association between genetics and environmental risk factors in a deeply characterized Italian cohort of 769 individuals (aged 6–89 years) coming from Northern Italy's genetically isolated populations. Their statistical analyses determined anxiety as a bruxism risk factor, and GWAS highlighted three novel genes potentially associated with bruxism,

namely NLGN1, RIMBP2, and LHFP genes. These findings significantly support the CNS-mediated theory of bruxism [29]. However, even though the genetic factor has been found to relate to SB, the entire mechanism behind it remains unknown [26].

The potential association between the use of psychotropic medications with SB has been investigated. Duloxetine, paroxetine, venlafaxine, barbiturates, and methylphenidate may be associated with increased odds regarding SB, while no evidence of an association between the use of benzodiazepine, carbamazepine, citalopram, escitalopram, fluoxetine, mirtazapine, sertraline, and valproate with SB [30]. However, the quality of evidence is considered very low, and caution should be exercised when interpreting these findings. There is still insufficient evidence-based data to draw definite conclusions concerning medications and addictive substances inducing or aggravating SB [31].

Heavy alcohol use, excessive caffeine consumption, and tobacco use also contribute to bruxism; however, there is still a need for stronger evidence based on studies with greater methodological rigor [32].

Assessment tools

Assessment tools of bruxism may be made by the following:

- Patient report and clinical interview
- Clinical examination
- Instrumental assessment
 - Intraoral appliances
 - Electromyography (EMG)
 - Polysomnography (PSG).

Patient report and clinical interview

The patient or their family may report teeth grinding or strange tooth grinding sounds.

The interview should include awareness, frequency, and symptoms from SB [33]. However, the diagnostic validity of self-reported measures of SB was low to modest. Using only self-reported measures for the assessment of SB may not have high validity [34-36].

Clinical examination

Clinical examination may reveal masseteric hypertrophy, muscle tenderness on palpation, wear facets on occlusal surfaces either at centric and eccentric jaw positions, shiny spots on restorations, restoration or tooth fracture, temporomandibular disorder, and an inspection of the cheek and tongue mucosa (e.g., linea alba, tongue scalloping, traumatic lesions) [37]. However, tooth wear is a multifactorial condition, leading to the loss of dental hard tissues [38]. It can be associated with bruxism, erosion from dietary or gastric sources, abrasion, and abfraction. Tooth surface loss may be historical and cannot be used to indicate static clenching activity. The extent of tooth wear would be influenced by factors such as dietary and gastric acids, enamel quality and quantity, and lack of posterior tooth support [33]. This multifactorial etiology means that the presence of tooth wear alone is not necessarily a sign of bruxism (and cannot be used as an indicator of static clenching). Some clinical studies have failed to demonstrate an association between tooth surface loss and bruxism. As such, tooth surface loss is described as a weak indicator of bruxism [39-41]. By the way, 'bruxofacets', that is, tooth wear in an eccentric position of closure, would seem to provide more convincing evidence [37]. In addition, it should be noted that some sign such as the presence of masseter hypertrophy, indentations on the tongue or lip, the presence of mucosal ridges on the inner cheek, the validated evaluation criteria do still not exist [42].

Instrumental assessment

- Intraoral appliances

The use of intraoral appliances in diagnosis relies on patient tolerance and on the assumption that the insertion of the device will not affect bruxist activity. The observation of wear facets on intraoral splints has been observed in the literature and anecdotally, although this has not been validated in the detection of bruxism [33,43]. Holmgren *et al.* [44] reported a repetitive wear pattern on the occlusal splint. They observed wear facets on full-arch acrylic resin splints, which reappeared in the same location with a similar pattern and direction, even after adjustment of the splints. While Koriath *et al.* [45] reported that parafunctional nocturnal dental activity on full-arch occlusal stabilization splints resulted in wear, which was both asymmetric and uneven. Unfortunately, no confirmation of the reliability of these methods has been reported. The relationship between wear and bruxism activity is still questionable [43]. "BruxChecker" was introduced in 2006. It is an inexpensive instrument using the pressure molding technique to fabricate for the individual patient to visualize patterns of teeth grinding by marking areas where teeth contact occurs during SB without affecting the stomatognathic system, possible to use in routine diagnostics and follow-ups of SB [46]. The recent study indicated that the BruxChecker system provided excellent reliability in the quantitative assessment of sleep bruxism at the dental level by measuring the peeled area in the studied population [47].

- Electromyography (EMG)

EMG records the electrical activity of muscles generated during movement and will provide information on the extent, duration, and force of muscle activity. EMG uses sensors attached to the skin overlying the masseter or temporalis muscles. Recordings can be made

using EMG ambulatory devices, and are suitable for the detection of AB as well as SB [33]. This single-channel home SB recording is easy and inexpensive to use and has high patient acceptability for multiple recordings, and has been a great technological improvement in dental sleep medicine, so it is predominantly used in research and in clinical settings [48-49]. However, due to the absence of audio-video, the over-scoring of EMG events is possible from the occurrence of orofacial activities such as snoring, swallowing, vocalization, tooth tapping, sucking, and grunting. Furthermore, EMG cannot detect grinding noises, lacks EMG specificity, and wake/sleep recognition. Therefore, diagnostic accuracy and a clear assessment of its predictive value on oral health outcomes are the challenges in the future [48-49]. Home SB testing may not be needed for all individuals with SB; we need to set criteria for such a decision based on the personal and social impact of SB, and the presence of comorbidities [48].

- **Polysomnography (PSG).**

PSG with audio and video recordings is the gold standard for bruxism diagnosis, which is available on many channels, involving electroencephalography, electro-oculography, EMG, electrocardiography, thoracoabdominal movements, oronasal airflow, and oxygen saturation. Masticatory muscle activity is measured by means of grading the information from EMG channels of masticatory muscles [4]. PSG is considered to be the gold standard for diagnosing sleep-related breathing disorders, which include OSA, central sleep apnea, and sleep-related hypoventilation/hypoxia. PSG can also be utilized to evaluate for other sleep disorders, including nocturnal seizures, narcolepsy, periodic limb movement disorder, and rapid eye movement sleep behavior disorder. A routine PSG requires a comprehensive

monitoring system to record sleep stages, limb movements, airflow, respiratory effort, heart rate and rhythm, oxygen saturation, and body position. This type of study, also known as a Type I (Level I) sleep study, is done in a sleep lab with a trained sleep technician present throughout the duration of the study [50]. However, due to the disadvantages relating to its complexity and requiring specialist equipment can only be performed in a sleep laboratory, which cannot be applied chairside in the dental clinic. This investigation is clearly out from the scope of dental practice, expensive for patients, and is preferred to be used in a research study [33].

Self-reporting reflects the patient's experiences and beliefs. It provides that the patient is aware of the behavior and allows the assessment of the perceived time course of bruxism [51]. In addition, clinical examination does not measure bruxism itself but rather clinical signs of the motor behavior that are possibly present independently from the patients' beliefs (e.g., tongue impressions) and may also be historical (e.g., mechanical tooth wear) [51]. Finally, device-based tools are used to actually measure jaw-muscle activities, as to provide insight into, for example, the pathophysiological mechanisms or physiological correlates of those activities [51]. Hence, the selection of one or the other assessment mode depends on the actual clinical need or specific research question [3].

Moreover, reassessing and upgrading SB metrics (both qualitative and quantitative), with evidence-based data, will improve the accuracy and relevance of diagnosis and treatment decisions. Current advances in technologies and methodologies offer unique opportunities to estimate masticatory muscle activity (or other equipotent or better metric or metrics)

accuracy and its predictive value. To achieve this aim, the establishment of a collaborative task force on SB metrics that includes dental and sleep researchers, technology developers, and the participation of clinicians and patients would be required [48].

Diagnostic protocols of sleep bruxism

The most widely accepted diagnostic and assessment criteria are those proposed according to the International Classification of Sleep Disorders, 3rd edition (ICSD-3) in 2014 [52] and the grading system from the International Consensus in 2018 [2], as shown in Table 1.

According to ICSD-3, SB requires self-report of regular or frequent tooth grinding sounds during sleep and clinical findings of abnormal tooth wear with or without jaw muscle pain, temporal headache,

and jaw locking upon waking [52]. The International Consensus in 2018 proposes that SB patients be categorized into possible, probable, and definite bruxism based on positive self-reports, clinical findings, and instrumental evaluation, respectively [2]. Recently, An International Consensus in 2024 revised the grading system (i.e., possible, probable, and definite) and replaced it with the terms subject-based, clinically based, and device-based [3]. M Palinkas *et al.* [53] evaluated the diagnostic capability of signs and symptoms of SB as per ICSD-3 criteria and a diagnostic grading system proposed by International Consensus Experts for assessing SB. They found that muscle fatigue, temporal headaches, and ICSD-3 criteria were associated with the highest sensitivity (78%, 67%, 58%, respectively) and with the highest diagnostic odds ratio (OR = 9.63, 9.25, 6.33, respectively).

Table 1 Proposed diagnostic and assessment criteria of SB according to the International Classification of Sleep Disorders, 3rd edition in 2014, and grading system from the International Consensus in 2018.

DIAGNOSTIC CONSIDERATIONS	DESCRIPTION
International Classification of Sleep Disorders, 3rd Edition	
A	Positive regular or frequent tooth grinding sounds during sleep
B	Presence of 1 of these clinical findings: Excessive tooth wear (consistent with positive reports of nocturnal tooth grinding) Momentary morning jaw muscle pain or fatigue, headache located on the temporal area, or jaw locking on awakening (consistent with positive reports of nocturnal tooth grinding)
International Consensus	
Possible	Based on a positive self-report
Probable	Based on a positive clinical evaluation, with or without a positive self-report
Definite	Based on positive polysomnography (possibly combined with audio or video recordings), with or without a positive self-report or a positive clinical evaluation

Jaw locking, muscle pain, and the criterion of “probable SB” were associated with the worst sensitivity (16%, 18%, 22%, respectively). They concluded that the presence of muscle fatigue and temporal headaches is considered to be a good tool for screening SB patients. Absence of jaw muscle pain, sounds during sleep, muscle fatigue, and abnormal tooth wear could be a good screening tool to diagnose patients without SB. None of the diagnostic criteria evaluated was able to accurately identify patients with SB. However, the ICSD-3 criteria had the best diagnostic accuracy of the evaluated tests; even it did not attain diagnostic values high enough to replace the current gold standard (PSG), it should be used as a screening tool to identify SB [53]. There is an acknowledged gap in the literature regarding the diagnosis of SB, which is multifactorial in etiology and poses clinical challenges. Although a single set of simple criteria cannot capture the complexity of such a sleep movement disorder, it may be reasonable to combine the criteria evaluated in this study into a clinical algorithm that could form the basis for a new diagnostic system combining intelligent methodologies and clinical insights [53].

In recent years, researchers have worked to develop a system of the Standardized Tool for the Assessment of Bruxism (STAB). This instrument comprises patient-based, clinical-based, and instrument-based evaluation methods, including PSG and ambulatory devices to assess for sleep and awake bruxism, to provide a multidimensional evaluation of bruxism status, comorbid conditions, etiology, and consequences. It is divided into two axes. Axis A includes the self-reported information on bruxism status and potential consequences (subject-based report) together with the clinical (examiner report) and instrumental assessment (technology report). Axis B includes the self-reported information

(subject-based report) on factors and conditions that may have an etiological or comorbid role for bruxism, which are psychological assessments of anxiety and depression, concurrent sleep-related conditions assessment, concurrent non-sleep conditions, report of taking medications and drugs, and additional factors. This comprehensive multidimensional assessment system will allow building a predictive model for clinical and research purposes [6]. The recent study compared non-instrumental and instrumental tools in the bruxism diagnostics system proposed by the STAB. They found that non-instrumental evaluation of bruxism through questionnaire and clinical exam had its value, by identifying 67% of SB cases and 89% without SB. This report indicated that the possible and probable diagnosis of bruxism was a starting point for bruxism management [54]. For an accurate and certain diagnosis of bruxism, instrumental evaluation as a gold standard can complete the clinical evaluation. The STAB criteria prove to be useful in bruxism diagnosis, but they should be tested on larger groups. Portable devices are useful both in research and clinical settings, and their properties should be validated through extensive research [54].

Management of sleep bruxism

Due to the multifactorial etiology of bruxism, clinical management strategies are a challenge. There are many approaches to the treatment of bruxism. However, the consideration of therapy must be based on individualized and applied with caution for the process to be successful [55]. Actually, not all patients with SB will need treatment; management is only indicated where problems arise as a result of SB [33]. The risks to health (including oral health) and the quality of life of individuals with SB (and probably sleep partners if a grinding noise is a major complaint)

should form the basis for this decision, using a personalized approach focused on the individual when it is time to select treatments [48].

- **Occlusal splint (OS)**

An OS is a removable appliance worn in the upper jaw (maxilla) or the lower jaw (mandible), with coverage of the dental surfaces. There are many designs of OS, such as stabilization splint, mandibular anterior repositioning splint, and anterior splint. Indication of its use is questionable about sleep outcomes, but it may provide some benefit concerning tooth wear [56]. In a systematic review, Minakuchi *et al.* [57] examined 11 RCT articles which eligible for study. In these articles, five articles applied the only palatal coverage splint. Four articles applied a mandibular anterior repositioning splint, and the other two applied the anterior splint. Several types of splints were used as controls, such as maxillary occlusal splint, mandibular occlusal splint, with flat plane or canine raise design. They found the oral appliances tended to reduce the number of SB events, although there was no significant difference compared to other types of splints. Philip Riley *et al.* [58] reported no clear evidence to support the provision of splints for the various subtypes of TMD or bruxism due to differences in diagnoses, splint type, and outcome measurement/reporting. There was insufficient evidence to recommend OS therapy over no treatment or other treatment modalities [59]. This controversy also continues the debate about the exact mechanism of action of OS. Mechanisms include muscle relaxation/habit-breaking for patients with increased parafunctional or muscle-tightening habits; protection of teeth and jaws, particularly where teeth clenching and grinding may lead to damage of teeth; normalizing periodontal ligament proprioception, by utilizing a splint to spread the forces placed on individual teeth; and repositioning of the jaws and condyles into centric relation [58].

However, it seems that occlusal guard/appliance is the most often approached to bruxism management by dental practitioners, even though the goal is to prevent further damage, yet it does not address the bruxism behavior [60].

- **Mandibular advancement device (MAD)**

MAD is commonly used in the management of OSA as a good alternative if the patient does not accept CPAP. The study showed a short-term improvement in sleep bruxism scores, sleep quality, and reduction in occlusal force in sleep bruxism participants after using MAD [61]. When compared to OS, the studies found MAD and OS provided significantly improved sleep quality and a decrease in SB episodes at 3 months. By the way, using MAD reported more discomfort than using OS; patients preferred the comfort of an OS [62-63]. Therefore, the suggestion to manage patients with SB who need an OS and present with a suspicion of OSA, should be referred to a medical sleep physician to first receive the proper diagnosis and then, based on test results. In a positive OSA case, the options include continuous positive airway pressure (CPAP) use with an occlusal splint or mandibular advancement device (MAD) [64]. SB alone is not an indication for using MAD.

- **Biofeedback therapy (BFT)**

Biofeedback aims to provide immediate information to the patient about their behavior, enabling its reduction [33]. It has been used for AB and SB. The device for biofeedback purposes has various designs. Briefly, we can detect excessive occlusal force via the pressure sensor that is inserted into the fabricated intraoral splint or using surface EMGs. When excessive force is detected or EMG activity exceeds the determined amplitude, the signal will occur and induce stimuli action via electric stimulation, vibration, or sound stimulation on subjects. In systematic reviews, it can be concluded that BFT would certainly

have a reduction effect on SB-related EMG activities [55,57,65]. Many studies show SB reduction without substantial interference in sleep, as evidenced by questionnaire-based assessment and the EMG/PSG study. These results may indicate that contingent electrical stimulation (CES) at non-painful intensities does not cause major arousal responses in sleep parameters or interfere with self-reported sleep quality. By the way, there have not been deeply elucidated the learning effect, long-term effect, and adverse event for the sleep quality, teeth, and orofacial muscle. In the future, research is expected to examine the negative components of BFT in addition to the SB inhibitory effect in a well-designed study [57]. In terms of pain-related symptoms effect, it seems to be controversial; some studies report a decrease in pain-related symptoms [66-67] while some found no statistically significant changes [68-69]. At this point, whether the reduction in EMG activity through CES could also alleviate the pain variables, thus revealing a link between oromotor activity and pain, remains to be addressed. The result of these studies and systematic reviews indicates that changes in pain could not be entirely attributed to changes in nocturnal EMG events [57].

- **Physical therapy**

Treatment methods used in physical therapy include electrotherapy, therapeutic exercises, muscle relaxation, postural awareness, acupuncture, and manual therapy. Cognitive behavioral therapy (CBT) is a psychological intervention that is also frequently used with physiotherapy. The aim of this treatment is the improvement of muscle pain and activity, mouth opening, oral health, anxiety, stress, depression, temporomandibular disorder, or head posture in individuals with bruxism. However, what represents the most effective treatment is still unclear, and there is no conclusive evidence of effectiveness. Systematic reviews show there is very low-quality

evidence that diverse and distinct methods of physical therapy interventions. The low value of this evidence is mainly because of the poor methodological quality of most of the studies included in the review [57,70].

- **Sleep hygiene**

Sleep hygiene measures include avoidance of caffeine, tea, and alcohol close to bedtime; keeping the bedroom well-ventilated and quiet; relaxing close to bedtime; and relaxation techniques before sleep [71]. These measures aim to reduce any influence of psychological stress on SB. However, a randomized controlled trial with 16 participants found that sleep hygiene and relaxation had no effect on SB [71].

- **Botulinum toxin (BTX)**

BTX, a purified exotoxin of the gram-positive anaerobic bacteria *Clostridium botulinum*, is a protease that inhibits neuromuscular transmission. Injection of BTX into the muscle can cause flaccid paralysis by inhibiting the release of acetylcholine from nerve terminals. Its effects are transient, nondestructive, and largely limited to the area, which justifies its clinical application in the treatment of bruxism [72-73]. BTX exists as eight serotypes; however, only BTX type A (BTX-A) is available for clinical use in the United States, under the tradename Botox (Allergan, Inc., Irvine, CA) [72]. BTX has been used in aesthetic and general medicine for several years. Nowadays, the use of BTX is being deepened to address the problem of bruxism [74]. Fernandez-Nunez *et al.* [73] examined 4 RCTs from 68 studies and stated that BTX-A injections can reduce the frequency of bruxism episodes, decrease pain levels, and the maximum occlusal force, and offer more satisfactory clinical results in the treatment of bruxism compared to control groups that were treated with placebo or traditional methods. They concluded that the use of BTX-A was an effective alternative in the treatment of SB [73].

While Agren *et al.* [75] examined 4 articles from 333 articles and concluded that the available research was inconclusive and did not show enough evidence that bruxism can be treated with BTA injections [75]. This dilemma suggests that more studies are needed to evaluate the effects of botulinum toxin. In the recent systematic review by Sinda Yacoub *et al.* [76], there were 12 RCTs in this review. Six studies reported a reduction in muscle activity after the administration of BTX-A, recorded by rhythmic masticatory muscle activity (RMMA) and EMG. Three studies of them indicated a significant reduction in muscle pain intensity after receiving BTX-A, measured by using the visual analogue scale. Finally, a study highlighted improved sleep quality in patients with bruxism who were rehabilitated with a single-arch implant overdenture and received either BTX-A or occlusal appliances. They concluded that BTX-A can effectively reduce symptoms of bruxism. However, the included studies exhibited heterogeneity and methodological differences. Long-term follow-up studies with large sample sizes and the incorporation of repeated injections are necessary to further validate the findings [76]. The local adverse effects of BTX-A included pain and mild cutaneous reaction at the site of injection. While the systemic adverse effects may be headache and nervous atrophy, and some reversible specific effects such as dysphonia, dysphagia, and dry mouth [73]. The complications resolved independently within a relatively short time frame (1 to 4 weeks), leaving almost no patients dissatisfied with the treatment [74]. In addition, some animal studies found that long-term use of Botox may be associated with changes in bone density, but this has not been confirmed in human studies [77-79].

- **Medication**

While some classes of medication are known to be causally related to the occurrence

and/or worsening of bruxism, several drugs have been suggested for its management (rabeprazole, L-tryptophan, levodopa, bromocriptine, amitriptyline, clonazepam, propranolol, clonidine, gabapentin, pramipexole, and botulinum toxin type A) [31]. Clinicians should be cautious in using medication to manage SB since most evidence is derived from small sample size studies, only a few drugs have been tested with a solid study design, that is, randomized control trial (RCT), and very few studies have been replicated [31,57,64]. Therefore, these drugs are not recommended for use in everyday clinical practice. In addition, when these drugs are prescribed, the clinician should know their indications, contraindications, and side effects [64].

Finally, the difficulty involved in treating SB is that it does not always need to be controlled, as in most cases, it should not be considered a pathology according to the current literature. It can be a risk factor or a protective factor; therefore, treatment should not be focused on SB as such, but on investigating the pathologies, comorbidities, or associated factors that lead to its onset [80].

Conclusions

The definition and concept of bruxism have evolved over the past several decades. Regarding the etiology of bruxism, in the past, it was associated with occlusal discrepancies, but nowadays, it is no longer considered as such, since several studies mention that SB is centrally regulated and agree that the etiology is multifactorial. This has resulted in a major transformation in our understanding of SB. A multidisciplinary approach for the management of sleep bruxism may involve a general dentist, neurologist, physical therapist, pain management

specialist, and psychologist. It is time to improve the accuracy and refinement of SB assessment by developing more homogenized SB metrics which relevant to clinical outcomes and benefit patients who suffer from one or more possible negative consequences of SB.

Acknowledgement

None.

Funding sources

None.

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