Oral Appliances and the Management of Sleep Bruxism in Adults: A Century of Clinical Applications and Search for Mechanisms

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The phenomenon of sleep bruxism (SB) has been recognized and described for centuries, including literary references to the gnashing of teeth. Early etiologic explanations were generally focused on mechanistic factors, but later, attention was focused on psychologic issues such as stress and anxiety; by the end of the 20th century, most opinions combined these two ideas. However, recently, the study of the SB phenomena has occurred primarily in sleep laboratories in which patients could be observed and monitored over several nights. Various other physiologic systems were also studied in sleep laboratories, including brain activity, muscle activity, cardiac function, and breathing. As a result of these studies, most authorities now consider SB to be a primarily sleep-related movement disorder, and specific diagnostic criteria have been established for the formal diagnosis of that condition. All of these changes in the understanding of the SB phenomena have led to a corresponding change in thinking about how oral appliances (OAs) might be used in the management of SB. Originally, they were thought to be a temporary measure that could help dentists analyze improper dental relationships. Unfortunately, this often led to dental procedures to "improve" these relationships, including equilibrations, orthodontics, bite opening, or even major restorative dentistry. However, it is now understood that the proper role for OAs is to protect the teeth and hopefully to diminish muscle activity during sleep. This paper reviews these evolutionary changes in the understanding of SB and how this affects concepts of designing and using OAs. *Int J Prosthodont 2010;23:453–462.*

Most clinicians are familiar with the terms oral appliances (OAs) and sleep bruxism (SB), with the majority having used OAs in the management of SB. However, clinicians should be aware that these terms have recently undergone a major transformation as a result of an explosion in new appliance design and research findings on both topics. OAs, which used to be simple processed acrylic devices that covered all or most of the teeth in one arch, are now available in a variety of materials and designs with multiple claims as to their mechanism of action. SB, which used to be viewed as a simple nocturnal parafunctional activity, is now being studied as a sleep-related movement disorder. As the dental profession evolves and adopts a more comprehensive, integrative, and evidence-based model for understanding SB, that perspective will have a great influence on the appropriate strategies to be used by practitioners in the management of SB.

The aim of this paper is to provide a brief historical review of SB and the use of OAs in its management in the traditional dental model. In addition, an overview regarding the current knowledge on SB will be provided, followed by a discussion based on the current scientific evidence regarding how OAs should be used in the management of SB. The term "splints," which often appears in the dental literature as a synonym for OAs, will not be used here because it has several other definitions in dentistry (eg, splinting teeth together).

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Historical Aspects

Sleep Bruxism

The word "bruxism" originates from the Greek word "brychien," meaning "to gnash the teeth." The first description of this phenomenon in the scientific literature was by Marie and Pietkiewicz in 1907, when they used the French word "la bruxomanie" to describe bruxism.[1] In 1931, Frohman described the concept of "bruxomania" as a psychic state and further stated that "bruxism" is not necessarily audible.[2] This reference to "bruxism" may have been the first time this term was used in the scientific literature. Miller, in 1936, alluded to "bruxism" as a term to be used when describing nocturnal bruxism and "bruxomania" to denote habitual diurnal tooth grinding.[3] In a 1990 review by Faulkner,[4] several terms such as "occlusal habit neurosis," "neuralgia traumatic," "bruxomania," "teeth gnashing-grinding," and "parafunction" were listed as having been used to describe this phenomenon. Another term, applied to this phenomenon in 1992, is "brycose," which was suggested for the severe destructive form of bruxism: "brycho" for movement with teeth contact and "ose" for exaggerated activity.[5] Over the years, SB has also been classified according to its possible underlying pathophysiologic processes. These have ranged from being considered a sleep parasomnia, or a sleep-related movement disorder, to being an orofacial parafunction, a tic, or an automatism.[6] Furthermore, psychiatrists view this as a psychiatric disorder due to stress problems, neurologists relate SB to a central nervous system disease, pneumologists consider this a respiratory problem, and many dentists associate SB with parafunctional habits and occlusal disharmonies. Undoubtedly, the many descriptions and classifications applied to this activity, in addition to the diverse disciplines that have proposed a variety of etiologic factors, are merely a reflection of the confusion associated with this subject matter.

Oral Appliances

According to a search completed by the authors, it appears that the first publications regarding the use of OAs in the management of SB were reported by Karolyi in the German literature in the early 1900s; bite-raising gold "caps" and vulcanite "splints" covering the occlusal surfaces of all teeth were recommended to be used at night for patients with bruxism. Many prominent practitioners from the 1940s through the 1960s such as Sved,[7] Block,[8] Christensen,[9] and others wrote about using occlusion-changing procedures in the management of SB, as well as with temporomandibular disorders. Most of these procedures also included the use of OAs to control SB, relax the muscles, determine correct jaw positions, and so forth. This belief was widely accepted, as witnessed by the various designs and types of OAs used for patients with SB and as reported in the many articles published on this subject in the 1940s and 1950s.[10–15]

In 1961, Ramfjord[16] popularized the concept that the most important factor responsible for bruxism was a discrepancy between centric relation (the terminal jaw position) and centric occlusion (maximal occlusal contact position), with heavy balancing side occlusal contacts being a secondary significant trigger. He suggested that such a discrepancy will elicit asynchronous contractions or sustained strain in the masticatory muscles during swallowing, and therefore occlusal adjustment was the treatment of choice to alleviate this problem. This concept was later expanded to include the use of an OA (Michigan splint) as an initial treatment modality preceding the occlusal equilibration.[17] This mechanistic concept of the etiology and treatment of bruxism has persisted within the dental profession for many years, despite the fact that Ramfjord's original research was later shown to be flawed methodologically.[18,19] Rugh et al[20] demonstrated that removal of occlusal interferences did not influence bruxist behavior, and they also found that artificial interferences resulted in a decrease in electromyographic (EMG) activity in 90% of test subjects. Furthermore, SB prevalence seems to be similar in individuals with or without occlusal interferences.[21,22] A detailed debate over the role of dental occlusion in SB is important but beyond the scope of this paper; however, an extensive review of peripheral sensory involvement in SB can be found from other sources.[23]

Current Concepts

Sleep Bruxism

It is important to distinguish between awake and sleep bruxism. The use of the word "night" or "nocturnal" as it relates to bruxism is obsolete and will not be used, since many individuals sleep during the day due to work schedules. Awake bruxism is thought to be a semivoluntary mandibular activity characterized mainly by tooth clenching and rarely by tapping or certain types of jaw bracing without tooth contact; usually, it is not associated with audible sounds in healthy or nonmedicated individuals.[24,25] It has an estimated prevalence (based on self-reported awareness of tooth clenching during wakefulness) of between 5% and 25% of children and adults, being more common in females and decreasing with advancing age.[26–29]

There are two categories of SB: *(1)* primary or idiopathic SB, which is without an identifiable cause or any associated medical problem, and *(2)* secondary SB,

Table 1 Clinical Consequences Associated with SB

Dental	Temporomandibular disorders (myogenous and arthrogenous)	Other
1. Severe occlusal and incisal wear (chipping), tooth fracture, and attrition	1. Masticatory muscle hypertrophy* (secondary to clenching, awake bruxism, habit of tic)	1. Lateral border of the tongue indentations/scalloping*
2. Tooth mobility	2. Masticatory muscle discomfort due to fatigue (may be with or without pain)	2. Reduction in salivary flow or xerostomia
3. Hypersensitivity of teeth to air and cold or hot foods and beverages	3. Pericranial muscle tenderness or pain (considered a morning headache in absence of sleep disorder breathing or neurologic condition)	3. Lip, cheek, or tongue biting
4. "Cracked tooth syndrome" and frequent breakage of dental restorations	4. Stiff, tight mandible with reduced movement and difficulty with mastication of food upon awakening	4. Glossodynia due to parafunctional habits* (secondary to clenching, awake bruxism, habit of tic)
5. Exacerbation of periodontal disease (controversial)*	5. Temporomandibular joint discomfort or pain	5. Excessive concern or anxiety about tooth wear
6. Failure of dental implants due to excessive forces		

*These conditions are commonly associated with SB by clinicians, based on clinical experience, but there is little evidence of cause and effect relationships.[39]

which is related to a medical condition (eg, movement or sleep disorder, neurologic or psychiatric condition, drug or chemical related). Clinicians have to understand that SB may be concomitant with many sleep disorders such as sleep epilepsy, REM behavior disorder (RBD), and sleep breathing disorders. RBD is characterized by rapid and involuntary motor activity during REM sleep, a period usually characterized by muscle hypotonia; patients with RBD are at risk for neurodegenerative disorder. Sleep breathing disorders may be a result of upper airway resistance or apnea-hypopnea.[6]

The prevalence of primary SB (based on grinding sounds reported from family members or sleep partners) declines linearly from childhood (12% to 20%) to adult life (5% to 8%) and even more over 60 years of age (3%) without any gender differences.[30–33] This low figure reported in the elderly population may be explained by the presence of dentures or changes in sleeping behavior (ie, in isolation). The actual prevalence of SB is probably much higher, since individuals are often unaware of this sleep-related motor behavior (if the subject is sleeping alone, he or she may not be aware of current grinding) and also because the frequency of the activity is highly variable over time.[34] Additionally, epidemiologic studies have shown that SB manifests in the majority of the population at some time in their life (85% to 90%).[24,35–37]

The etiology and pathophysiology of awake bruxism appears to be quite different than that occurring during sleep. SB is an oral activity characterized by tooth grinding or jaw clenching during sleep, usually associated with sleep arousals (a rapid and transient rise in brain, heart, and muscle activity).[6,19,38]

There are clinical consequences associated with SB, which may impact dental structures (natural dentition and prosthetic devices) deleteriously or involve pain and dysfunction of the jaw musculature and joints, although the evidence for this latter relationship is somewhat controversial (Table 1).[39] Interestingly, it has been estimated that only 20% to 30% of SB patients report concomitant orofacial pain, usually occurring on awakening.[40,41] Studies conducted in sleep laboratories have suggested that SB episodes are most often observed secondary to a cascade of physiologic events (autonomic/cardiac/respiratory activities) related to sleep arousal (Fig 1).[42–44] Studies have also demonstrated the association of SB with brief, transient, and recurrent arousal (rise in autonomic, brain, and muscle activity for 3 to 10 seconds) during sleep. Yet, in young and healthy individuals with SB, sleep organization and macrostructure has been found to be generally normal.[44–46]

SB episodes can be subdivided into phasic (rhythmic), tonic (sustained), and mixed events based on polygraphic and audio-video recordings (ambulatory or sleep laboratory) monitoring EMG of the masseter and temporalis muscles and sleep activity.[45,47] The majority of these EMG events (88%) are of the phasic or mixed variety and rarely of the tonic type that characterize clenching, with between 60% and 80% presenting themselves in the lighter stages of the sleep cycle (stages 1 and 2).[44,45,47]

Clinically, the diagnosis of SB is often made from a self-reported history of tooth grinding, in addition to complaints of masticatory muscle tightness, fatigue, discomfort, morning headaches, or pain. Many clinicians also feel that the diagnosis of SB is confirmed

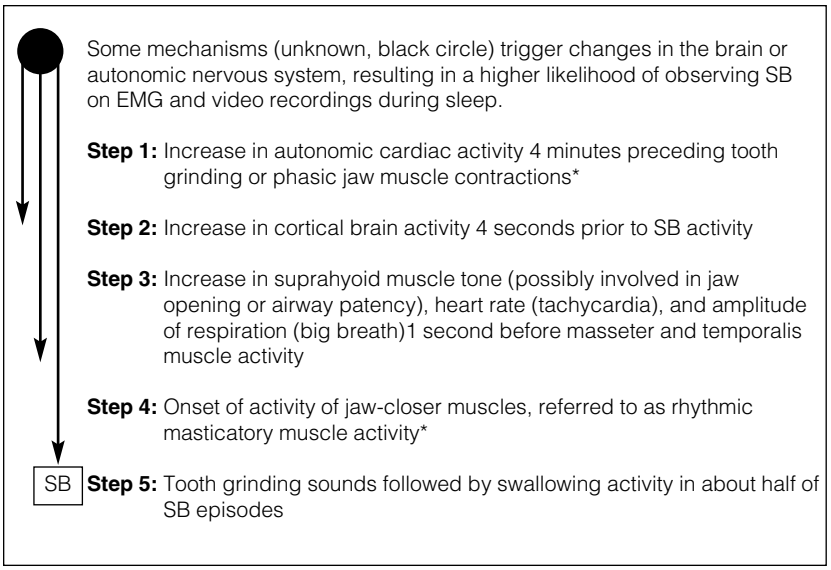

Fig 1 Physiologic sequence of events preceding SB as observed in most patients in approximately 85% of SB episodes recorded during sleep.[19] *These muscle phenomena are observed with EMG recordings and confirmed with audio-video recordings for specificity.

by the clinical findings of tooth wear.[48] However, self-reporting is not an accurate assessment of SB because it is impossible to differentiate SB motor activity from other jaw activities during sleep (swallowing, coughing, sleep talking, sighing, grunting, yawning, smiling, or myoclonus).[6,45,49] It also is not possible to separate patients with bruxism from those without by observing tooth wear factors,[50] since tooth wear may be produced by other etiologic factors (oral habits, food consistency, acid reflux, etc), and occlusal attrition does not confirm reliably that this habit is being performed currently.[48] However, Abe et al[51] determined that SB patients (young adults) present with greater tooth wear compared to controls (no report of any history of tooth grinding or sleep laboratory evidence of SB), but tooth wear was not able to discriminate between different subgroups (moderate-to-high versus low) of SB patients. Furthermore, SB cannot be assumed to exist if there is no current report of tooth grinding, as witnessed by a sleep partner, since the tooth wear may have occurred years before the SB activity began. Therefore, to diagnose SB reliably, electronic recording and documenting devices are used with strict criteria to detect and classify SB activity. Currently, the devices employed are either simple ambulatory (home portable) devices or complex electronic machines used only in sleep laboratory facilities (polysomnography). The latter system remains the gold standard, but it is time consuming and the patient is not able to sleep in his or her natural environment; consequently, several nights are needed to allow habituation of the patient to the recording conditions. Ambulatory polygraphy of single EMG channel (masseter or temporalis muscles) recording offers several advantages, but the absence of an audio-video signal to assess SB specificity reduces its validity; over 30% of EMG events recorded may not be SB.[52] However, validation for both the diagnosis and scoring criteria for either method is required for large population samples.[48]

Controversy surrounds the etiology of SB since it is probably the product of both biologic and psychosocial influences. Factors implicated range from peripheral mechanisms, such as occlusal discrepancies, to a variety of central factors, such as stress and psychosocial influences, alterations in catecholamine levels and other neurochemicals, and cardiac-autonomic interactions related to airway patency and salivary flow. In addition, there appears to be some genetic and familial predisposition for developing chronic SB.[19,53]

Oral Appliances

Since changes have occurred in our scientific understanding of SB, traditional ideas regarding the use of OAs to manage this condition must also be reconsidered. Okeson[54] provided several explanations as to the effects that OAs can have on both SB and TMDs, but most of them simply reflect the bias of certain clinical approaches (Table 2).

In a study by Clark et al,[56] it was shown that OA treatment resulted in a decrease in EMG activity during sleep in approximately 50% of patients; 25% experienced no change while the remaining 25% displayed an increase in activity. Hiyama et al[57] obtained similar findings in their six study participants, but nevertheless, they suggested that masticatory muscle activity during

Table 2 Explanations for the Efficacy of OAs[54]

Dental and oropharyngeal reasons for efficacy*	Nondental reasons for efficacy*
1. Alteration of the occlusal condition (EB: low)	1. Cognitive awareness (HD only)
2. Alteration of the condylar position (EB: low)	2. Placebo effect (EB)
3. Increase in the vertical dimension of occlusion (EB: low)	3. Increased peripheral sensory inputs to the central nervous system resulting in decreased motor activity (EB: low)
4. Improved breathing during sleep due to reduction of airflow and retracted tongue and jaw position (HD only)	4. Regression to the mean (eg, natural fluctuation of symptoms)

EB = evidence-based; HD = hypothesis-driven (no substantial evidence).
*EB grading was based on the authors' evaluations of available evidence, as presented in the references cited and the Oxford Centre for Evidence-Based Medicine Levels of Evidence criteria.[55]

sleep is significantly reduced by wearing an OA. Interestingly, the former study found that of those who experienced a significant decrease in EMG levels with OA use, almost all returned to pretreatment levels on OA discontinuation. This finding is in agreement with several other studies that found that OAs do not stop SB.[58–61] These observational studies suggest that OAs are effective in reducing sleep muscle activity, but only for a variable period of time and only in certain individuals.

OAs may be fabricated to fit either the maxilla or mandible; lack of strong evidence prevents firm recommendation for choosing one over the other. The main exception is that thick maxillary OAs are not indicated in patients with sleep breathing disorders such as apnea-hypopnea because they may produce a rise in the Respiratory Disturbance Index.[62] Most OAs are fabricated from one of two materials based on consistency: either a hard or soft occluding surface. Some OAs are fabricated with a thermal-sensitive material in the intaglio that needs to be heated in hot water prior to use, while others are made of a hard shell and smooth occlusal surface. The majority of OAs are made in a commercial dental laboratory and are pressure-cured, with newer and novel techniques being offered for chairside fabrication. Unfortunately, at this time there is little information available on the benefit of such design modifications over more traditional OAs, ie, hard and soft full-arch coverage.

Controversy exists as to the efficacy of the two traditional designs in the management of SB. In a study monitoring EMG activity in a group of 10 bruxers alternating between hard and soft appliance use, it was found that 8 subjects experienced significantly reduced sleep muscle activity with use of hard OAs. In comparison, the soft OAs reduced muscle activity significantly in only 1 participant, while causing a statistically significant increase in EMG activity in 5 others.[63] Despite being a small-sample single-cohort study, this research suggests that hard OAs are more effective in reducing SB than soft OAs. However, the use of soft OAs is still a popular choice in spite of the lack of scientific evidence supporting their efficacy and effectiveness, as witnessed in a survey among Swedish general dental practices.[64] In the authors' experience, soft OAs should be used for short periods of time or in children who still have ongoing dental bone growth.

Many practitioners advocate the incorporation of canine-protected articulation on OAs to disocclude the posterior teeth during eccentric movements. Some studies suggest the incorporation of this design in asymptomatic individuals is more effective in reducing muscle activity,[65,66] while other studies have shown no differences in muscle activity in healthy subjects[67] or in TMD symptom reduction[68] by using this feature. In a study comparing canine-to-molar guidance–fabricated OAs specifically in SB patients,[69] it was determined that the two appliances provided nearly equivalent effects (increased, decreased, or no effect on the same number of patients) according to EMG recordings during sleep.

Recently, studies have evaluated the efficacy of conventional OAs compared to a "placebo" appliance (nonoccluding palatal control) in the management of SB.[70–72] The cumulative results of these studies, once again, indicated a lack of a consistent group effect with either a brief reduction (at 2 weeks), an increase (in only a few subjects), or no change. There was a return to baseline pre-OA values for the index of sleep-related jaw muscle activity regardless of the type of appliance employed. This corroborates the findings from previously mentioned studies that OAs may have only a transient effect (a couple of days to 2 weeks), if any, on the reduction of EMG activities in SB patients on an individual basis. Since these nonoccluding OAs are not altering the occlusion of SB patients, their mechanism of action may be due to the behavioral intervention of a doctor placing it. Furthermore, the short-term and transient effect may be due to a physiologic adaptation of tongue position and airway space respiration or to

sensory feedback (from muscle spindles or periodontal sensory organs that may trigger a circular transient change in EMG) rather than a redistribution of mechanical forces, as produced by occluding in a conventional OA. In the absence of strong evidence, clinicians need to maintain a critical appraisal of such mechanistic explanations. This takes on greater importance if the potential influence of a placebo effect in relation to any treatment is considered. For a review of placebo effects in general, as well as specific placebo aspects of OA therapy, the reader is referred to this recent paper.[73]

A differently designed short-term study[74] compared a custom-made mandibular repositioning or advancement appliance (MRA or MAA; a double-arch custom-fit appliance designed to protrude the mandible, commonly used in the management of snoring and mild to moderate obstructive sleep apnea) to an OA fit to the maxillary arch. In that study, it was found that both the OA and a nonprotruding MAA (designed to allow full freedom of mandibular movements while sleeping) reduced the frequency of SB. However, a greater reduction (almost double) was established upon activation (minimum/intermediate protrusion) of the MAA. The authors hypothesized that the mechanism of action, which may explain the reduction of SB with the MAA, was one or more of the following: dimension and configuration of the appliance, presence of pain, restriction of movement, or change in airway patency. In another short-term, randomized crossover controlled study, it was once again reported that the number of SB episodes per hour was reduced significantly with two types of MAAs (protrusion at 25% and 75%), while an OA fit to the mandible only reduced the number of SB episodes per hour slightly; however, this did not reach statistical significance when compared to baseline.[75] Because these studies are only short-term, the use of an MAA is only recommended in patients experiencing snoring, airway resistance-hypoventilation, or apnea-hypopnea events.

In another study comparing a miniature anterior appliance (nociceptive trigeminal inhibitor [NTI]) to an OA in SB patients, it was reported that the NTI was associated with a significant reduction in EMG activity, whereas the OA had no effect. Intriguingly, neither the NTI nor OA had any effects on the clinical outcome measures (pain scores, pain intensity, number of painful muscles, or maximum unassisted jaw opening capacity).[76] Furthermore, the NTI, due to its design of covering only some of the anterior teeth, has the potential for developing an anterior open bite because of the overeruption of the posterior teeth or intrusion of the anterior teeth. There have also been reports on the risks of swallowing or aspirating this device.[77,78]

Given the results described previously, mostly based on short-term studies, it is apparent that the benefit, safety, and proper role of OAs in managing SB must be reconsidered. The practitioner must use good clinical judgment regarding the appropriate application of OAs in the management of SB, and the word "palliative" rather than "curative" is much more appropriate for discussing these matters. SB is a transient activity with limited damage in most individuals; therefore, irreversible and invasive dental procedures, such as full-mouth rehabilitation, occlusal equilibration, and orthodontics in subjects with a healthy natural dentition, are not only inappropriate, but they are completely ineffective as treatments for SB. Special attention needs to be given to patients with obstructive sleep apnea syndrome and retrognathia or deep palate, as these are complex cases that require medical cosupervision. Management of these complexities may require surgical intervention (maxillofacial or otolaryngologic), while use of only an MAA or a maxillary OA risks exacerbating the breathing disorder. OAs are analogous to crutches in orthopedics in that they will help a patient prevent damage to their dentition and restorations and reduce pain exacerbation.

Furthermore, other palliative approaches such as medication for short periods of time may be useful; however, prudence and good judgment must be a consideration in light of the limited available literature. In a short-term, single-blinded, placebo-controlled, nonrandomized, crossover study comparing SB patients to a sex- and age-matched control group, it was reported that a low dose (1 mg) of clonazepam given at bedtime decreased the SB index significantly in all patients. Additionally, there was an improvement in a number of objective and subjective sleep quality measures.[79] Caution should be employed when considering this medication since this is only a short-term study and clonazepam has been associated with several adverse side effects, along with the potential for physiologic dependence and tolerance after long-term use. Furthermore, in the presence of any sign or symptoms of sleep breathing disorders (eg, sleepiness, snoring with cessation of breathing, hypertension, report of transportation accident), clinicians should avoid prescribing central nervous system depressant medications that can exacerbate respiratory dysfunction. Clonidine, an alpha 2-adrenergic receptor agonist, was tested in a randomized controlled crossover mechanistic study with a placebo and active treatments in SB patients. The investigators concluded that clonidine decreased cardiac sympathetic tone in the minute preceding the onset of SB, thus reducing SB by preventing the sequence of autonomic sleep arousal that leads to the motor activation of SB. Unfortunately, it was found that approximately 20% of patients experienced morning

Table 3 Management Strategies Suggested or Currently Used for SB[6,83,84]

Behavioral*	Dental*	Pharmacologic*	Others*
1. Education and reassurance regarding SB (opinion)	1. Athletic protective mouth guard (EB: low; may increase oral movement during sleep)	1. Benzodiazepenes: clonazepam (EB: one short-term trial), diazepam, lorazepam (open trial)	1. Botulinum toxin (EB: low; small sample size, may have risk)
2. Lifestyle modification (cessation of smoking, caffeine, and alcohol intake) (opinion)	2. Hard acrylic flat plane stabilization OA (EB; reduction for 1 or 2 weeks in certain individuals, muscle activity tends to return to baseline level after a few weeks)	2. Muscle relaxants: methocarbamol, cyclobenzaprine (opinion)	2. Continuous positive airway pressure (case study only; no benefit in young subjects because most cannot sleep with mask)†
3. Relaxation or sleep hygiene entrainment (opinion)	3. OA incorporating vibratory mechanism or with an electrical lip stimulation device (EB: one short-term trial)	3. Tri- or tetra-cyclic antidepressants: amitriptyline (EB: no benefit), trazodone (opinion)	3. Gamma-hydroxybutyrate (case study)
4. Hypnosis (case study)	4. Traditional anterior bite plane or mini-anterior OA (EB for NTI: short-term trials only; risk of swallowing or aspiration)	4. Serotonin-related medications: venlafaxine (case study), tryptophan (EB: no benefit)	
5. Biofeedback (EB: low; may cause sleep fragmentation, more arousal during sleep)	5. Mandibular repositioning or advancement appliance (appliance for snoring and/or obstructive sleep apnea) (EB: short-term trials only)	5. Dopamine-related medications: L-dopa (EB: modest benefit), bromocriptine (EB: no effect), pergolide (case study), pramipexole (opinion)	
6. Psychologic counseling (EB)		6. Cardiac-related medications: propanolol (EB: no effect), clonidine (EB: benefit but side effect, severe hypotension)	
		7. Anticonvulsants: gabapentin, tiagabine (case studies)	

OA = oral appliance; EB = evidence-based; NTI = nociceptive trigeminal inhibitor.
*The majority of these strategies were trialed for only short periods of time and only a few are evidence-based.
To date, none have been found to be curative of SB.
†GJL laboratory (unpublished data, 2009.)
EB grading was based on the authors' evaluations of available evidence, as presented in the references cited and the Oxford Centre for Evidence-Based Medicine Levels of Evidence criteria.[55]

hypotension; therefore, further dose-dependent research is required to assess the safety of clonidine in the management of SB.[80] The authors cautioned that clonidine should not be used to manage SB in the absence of close medical supervision.

Cognitive and behavioral therapies (CBTs) may be another approach indicated for long-term management in the absence of secondary or medical SB. Ommerborn et al,[81] in a randomized study evaluating the efficacy of an OA with CBTs (problem solving, progressive muscle relaxation, nocturnal biofeedback, and training of recreation and enjoyment) in SB patients, reported a significant reduction in SB activity, self-assessment of SB activity, and psychologic impairment, as well as an increase in positive stress-coping strategies for both groups. However, the effects were small, and no group-specific differences were seen for any dependent variable. The authors concluded that this study was only an initial attempt to compare CBTs and OAs in SB patients and in no way confirms that CBTs are an effective therapeutic intervention in obtaining a long-term effect in SB patients. Lobbezoo et al,[82] in a review regarding the management principles of bruxism, concluded that the value of behavioral approaches is questionable due to the deficiencies in performing well-designed and sound scientific studies.

To date, there are no specific management strategies for SB that can predictably reduce or eliminate the activity (Table 3). Unfortunately, no definitive pharmacologic approach can be recommended for the long-term management of SB. Furthermore, the effectiveness of a behavioral approach in the management of SB remains undecided. Presently, it appears that OAs are the most prudent therapeutic approach to manage SB. However, in a recent Cochrane review, the authors concluded that there was insufficient evidence to state that OAs are effective in treating SB with regard to improved sleep outcomes, but they may be of some benefit with regard to preventing tooth wear.[85] Table 4 will assist the practitioner in the decision-making process regarding the use of OAs for SB patients.

Table 4 OA Uses and Limitations in SB Patients

What OAs can do*	What OAs cannot do (opinion-based)*
1. Decrease/alter loading on TMJ by reducing force intensity, frequency, and duration of oral parafunctional activities (EB: low)	1. Unload the TMJ by distracting the condyles
2. Briefly reduce muscle activity by introducing "foreign body" of occlusal platform in some individuals (EB)	2. Retrain muscles to be less active after splint is removed
3. Reduce headache intensity or frequency if it is triggered by SB-induced myalgia or arthralgia (EB: low)	3. Relieve headache conditions that are primarily neurovascular or vascular in origin
4. Improve internal derangement symptoms of locking/catching on awakening related to strong sleep muscle activity (clenching/grinding)	4. Recapture displaced disks, enhance retrodiskal tissue healing, prevent progression from ADD-R to ADD-NR
5. Protect occlusal surfaces of teeth and dental restorations from SB forces	5. Permanently reduce or eliminate (cure) SB activities

OA = oral appliance; SB = sleep bruxism; TMJ = temporomandibular joint; EB = evidence-based; ADD-R = disk displacement with reduction; ADD-NR = disk displacement without reduction.
*EB grading was based on the authors' evaluations of available evidence, as presented in the references cited and the Oxford Centre for Evidence-Based Medicine Levels of Evidence criteria.[55]

Conclusion

Oral appliances continue to be a mainstay in the management of SB, with an emphasis on their palliative, conservative, and reversible application. An extensive description of how OAs should be viewed is provided by Dao and Lavigne[86] in their review article on these devices. They conclude by suggesting that the term "oromandibular crutches" may be the most appropriate descriptor until their physical and behavioral mechanisms of action are better understood.

While it seems obvious that OAs may prevent or limit damage to the teeth and dental restorations, it should be highlighted that based on current clinical research literature, they do not stop people from performing parafunctional activities during sleep. Incidence of SB and tooth grinding events are variable from night to night[34] and may disappear for months (according to sleep partner complaints). Based on the current literature, OAs do not stop SB permanently[58–61]; rhythmic masticatory muscle activity and tooth grinding seem to diminish for short periods (1 to 2 weeks) after initial OA usage but resume over time.[70–72] There are several minor negative outcome possibilities with the use of OAs. One is the paradoxical development or continuation of morning muscular pain; another is the onset of snoring. In some cases, they can cause wake time sleepiness in a small number of patients, which requires a different approach (eg, assessment of sleep instability due to breathing- or movement-related sleep disorders). For the majority of SB patients, however, these devices can be beneficial to protect teeth and restorations and manage grinding sounds. Their use should be encouraged to manage (but not cure) SB whenever it is clear that the patient is experiencing a simple sleep movement disorder in the absence of the concomitant medical conditions previously described. If a sleep breathing disorder is suspected in a patient with SB, a sleep medical consultation is mandatory since some OA designs may exacerbate sleep apnea, while others help to manage the comorbidity.

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Erratum

In IJP issue 4, 2010, in the article by Ma et al, the legends of Figures 1e and 2e and the fifth row of Table 4 should refer to a 3.95-mm Southern ball patrix and 3.95-mm-diameter abutment. The online version of this paper has been corrected.