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REVIEW

Understanding the clinical management of co-occurring sleep-related bruxism and obstructive sleep apnea in adults: A narrative and critical review

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Abstract

Sleep-related bruxism (SRB) is a motor oral behavior characterized by tooth grinding and jaw clenching activity, reported by 8%–12% of the adult general population and 3% of older individuals. The frequency of one of its biomarkers, rhythmic masticatory muscle activity (RMMA), remains elevated across ages. Obstructive sleep apnea (OSA) is associated with the brief and repetitive pause of breathing (apnea) and with transient reduction in oxygen (hypoxia). OSA is observed at all ages and in about 50% of older individuals with a male preponderance. SRB clinical assessment is based on self-reporting of tooth grinding sound, awareness of clenching, jaw pain or headache, and clinical observation of tooth damage. OSA clinical assessment is based on sleepiness and fatigue, snoring, sleep quality, and awareness of breathing cessation, plus clinical examination of anatomical factors (e.g., obesity, retrognathia, large tonsil, macroglossia), age, gender, and body mass. Although the literature does not support association or causality between these two conditions, the co-occurrence is reported in about 30%–50% of adults. To confirm a diagnosis of co-occurring SRB and OSA, home sleep testing (HST) may be indicated. A sleep test is performed using electromyography (EMG) of jaw muscle (masseter or temporalis) and cardio-respiratory variables (e.g., air flow, respiratory effort, oxygen level, heart rate). The management of co-occurring SRB and OSA for individuals with prosthodontic needs is challenging to prevent compromising the oro-pharyngeal space and breathing efficiency. OSA treatment in the presence of SRB includes continuous positive airway pressure (CPAP) use alone or with an occlusal splint or mandibular advancement device (MAD). In addition, the following may be considered: supine sleep correction device, myofuncional therapy, medications, and surgeries. All have limitations and risks. Individual variability suggests that phenotyping is mandatory to select the most efficient and personalized treatment.

KEYWORDS

co-occurrence, diagnosis, mandibular advancement appliance, obstructive sleep apnea, occlusal splint, oral appliance, sleep apnea, sleep bruxism, sleep, treatment

Oral rehabilitation by prosthodontists in patients presenting sleep-related bruxism (SRB) alone is already challenging due to the risk of restoration damage. Co-occurrence of SRB and obstructive sleep apnea (OSA), the latter being a life-threatening condition, increases the complexity. The diagnosis and treatment plan should be based on the severity of OSA-related health conditions (e.g., hypertension, diabetes, metabolic syndrome, cancer) and oral and dental anatomical

features (e.g., retrognathia, narrow and deep palate). The risk of increasing OSA or snoring might be avoided by an early screening or diagnosis of OSA and SRB. Even though evidence is lacking in the field of prosthodontics regarding teeth position and OSA, some research shows that the tongue position and uvula have prominent roles in the severity of OSA.[1]

The objective of this narrative and critical review is to summarize clinically relevant and evidence-based information to guide prosthodontists on how to manage individuals with co-occurring SRB and OSA. The paper is organized into four sections: (1) an overview of SRB; (2) an overview of OSA; (3) the strength of the association or causality of such co-occurrence; and (4) how to manage such complex cases.

This review is novel and important since it provides prosthodontist's a scientific and clinical overview of the association of the two conditions with a high prevalence in the clinic. The focus of this review is not on the management of one condition or the other, but rather on targeting situations in which both conditions are present at the same time during treatment planning in prosthodontics.

The literature cited was extracted from the usual tools (e.g., PubMed, Scopus, Web of Sciences, Cochrane) and the author's reading and research over the years. This narrative and critical review is not based on a hypothesis; it is a putative exploration of available knowledge. Furthermore, the quality of available literature and the low number of published randomized controlled trials prevented a systematic review and more powerful meta-analysis. The co-authors critically reviewed the paper during editing.

OVERVIEW OF SLEEP-RELATED BRUXISM

SRB is a motor oral behavior characterized by brief and repetitive tooth grinding and jaw clenching.[2] It is dominant in young and adult populations with a prevalence of about 12%.[3,4] In the general population, one of its biomarkers, rhythmic masticatory muscle activity (RMMA), specific repetitive electromyographic activity from masseter or temporalis, remains present in all ages.[5,6] Indeed, although the awareness of clenching and complaints of tooth grinding sounds drop with aging, oral motor activity remains high in older individuals with SRB.[5]

SRB dental and medical diagnosis is based on self-reporting of tooth grinding sound, awareness of clenching, transient jaw tension-pain, or headache. Poor quality sleep might be a clinical complaint in some individuals, although it is not always present. However, it was recently shown that individuals with more than four SRB episodes per hour present less slow wave sleep.[7,8] Clinical diagnosis based on self-reports is graded as *possible*. If upon clinical examination, tooth wear or damage are noted, it is graded as *probable* even though evidence is inconclusive about the association between SRB and tooth wear.[9] To confirm its presence, measurement tools can be used: these include recording of electromyography (EMG) of masseter or temporalis muscle, or quantification of the number of tooth contact on the oral device, or jaw movement sensor. EMG is one of the biomarkers used to quantify the frequency and duration of sleep-related RMMA, also expressed as a bruxism event index (BEI). Tooth contact count, using sensors implanted in oral appliance, or the quantitative analyses of chin movement (acceleration and direction) are available tools for SRB assessments.[10–12] It is critical to recognize that the occurrence of SRB motor activity is not stable; it is variable from night to night and across periods of life.[13,14] So far, standardization of SRB assessment, from self-reports and clinical examination, to device-assisted quantification, is a work in progress.[15–17] This is justified due to the estimation of the accuracy of reporting of tooth grinding, variables from time to time, and tooth wear observation is modest (47% to 62%).[13,14,18] Assessment of tooth wear alone is not precise for SRB diagnosis; the relation between the estimation of tooth wear or damage severity and frequency of EMG activity is somewhat weak.[19–22] The precision of tooth wear to establish an SRB diagnosis is influenced by other co-occurring conditions such as gastroesophageal reflux, oral dryness, and OSA.[23] It is important to clarify that the use of polysomnography (PSG) is not required for an SRB diagnosis in otherwise healthy individuals, that is, in the absence of co-occurring conditions such as sleep apnea or neurological conditions. However, some clinicians may value its use to assess treatment efficacy.[24,25] A home portable system, ideally, needs to be used for at least 3 nights to capture the SRB motor behavior with a cut-off of 4, 5, or 7 events per hour of sleep.[26–29] Standardization of the method for home recording of SRB is in progress.[15,16]

The cause or mechanism of RMMA onset explaining SRB is not a single possibility; SRB is a heterogeneous condition with specific psycho-physiological reactivity and genetic expression.[24,30–32] Indeed, data from the Finland genetic cohort with 12,297 individuals with SRB showed a significant association with the Myosin IIIB (MYO3B) gene.[30] In otherwise healthy and young individuals, most RMMA or SB episodes are associated with transitory arousals.[33–43] Furthermore, it was also shown that RMMA is associated with the cyclic alternating pattern (CAP), dominant in phase A3, a physiological rhythm of transient arousal.[33,44] Ongoing CAP acts as a permissive window facilitating the onset of RMMA-SRB in predisposed individuals.[6] Indeed, some studies have described a cascade of physiological events that precede the RMMA events. They include a rise in autonomic sympathetic activity (4–8 min before the event), a rise in brain activity (4 s before), tachycardia, and a rise in supra-hyoid muscle tone (1 s before). Finally, the RMMA onset happens with possible tooth grinding with a concomitant rise in blood pressure.[33–37] The presence of such a sequence is unknown when SRB is observed with co-occurring sleep disorders such as insomnia, OSA, REM Behavior Disorder, or sleep epilepsy. It is noteworthy that SRB is not associated with mortality risk conversely with OSA and insomnia.[45–47]

RMMA can be scored on the PSG traces as phasic, tonic, or mixed. Phasic episodes are characterized by three or

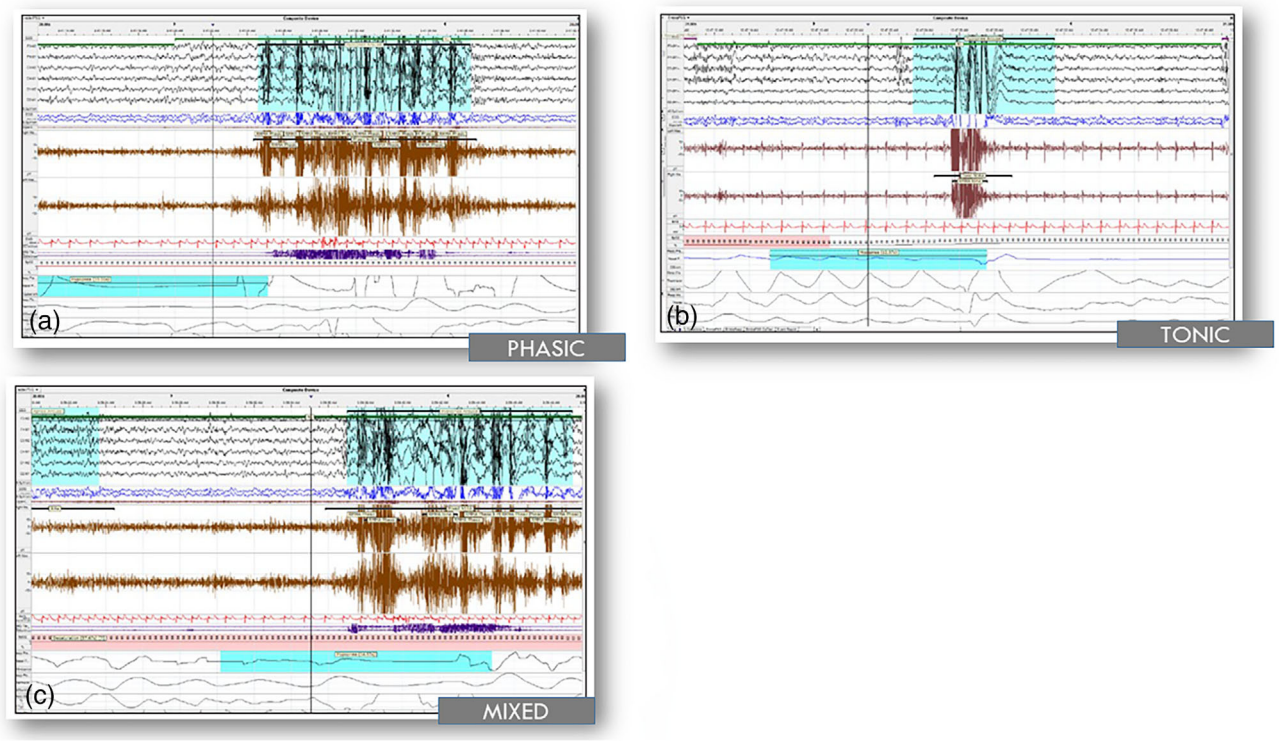

FIGURE 1 Examples of RMMA scored on the PSG traces as phasic, tonic, or mixed (Image credit: Dr. Maluly). RMMA, rhythmic masticatory muscle activity.

more bursts (short contractions) of 0.25 s and >2 s each, in a regular sequence. Tonic episodes are bursts with 2 s duration or more, and mixed is a combination of both (Figure 1).[48] Table 1 describes the parallel between these two possible concurrent conditions, in relation to diagnosis, the need for a PSG or HST, prevalence in adults, and consequences. SRB cannot be treated definitively since it is recurrent and variable in frequency and intensity over time. Instead, management of SRB is more appropriate since it includes the protective role of oral appliances such as occlusal splints (OS, for tooth damage) or mandibular advancement devices (MAD, for breathing), cognitive behavioral therapies, including sensory feedback devices, and, in some cases, medication (e.g., muscle relaxant, botulinum toxin, benzodiazepine, cardioactive drug-clonidine, pump proton inhibitors) (Table 2).[24,49,50]

TABLE 1 General overview of SRB and OSA.

	SRB	OSA
Diagnostic	Dental	Medical
Need of a PSG or HST for diagnosis	Only if other conditions are suspected	Yes
Prevalence in adults	8%–16%	15%–33%
Life threatening condition?	No	Yes

Abbreviations: OSA, obstructive sleep apnea; SRB, sleep-related bruxism.

OVERVIEW OF OSA

OSA is characterized by the presence of partial or complete events of airway obstruction (≥10 s, apnea/cessation, and hypopnoea/reduction in breathing amplitude) with arousals (transient and brief rise in cardiac and brain activity) and hypoxia (≥3% drop in oxygen). OSA is dominant in older male individuals and females after menopause.[51]

As mentioned above, while SRB is not a life-threatening condition, OSA poses a higher risk of morbidity and mortality if not well managed due to the cumulative impact of the chronic repetitive hypoxia burden and sleepiness.[51–55] The prevalence of OSA, estimated from PSG recordings, is 9%–38% of the adult population for apnea and hypopnea index (AHI) over 5. Using a criterion of AHI over 15 per hour or more, the prevalence reaches 49.7% for males and 23.4% for females.[56–60]

The diagnosis of OSA is medical since it is a systemic condition frequently associated with comorbidities and a risk of morbidity-mortality. The complaints of loud and chronic snoring, poor sleep quality, and breathing cessation are associated with sleepiness in males and fatigue in females, and possibly mood and cognitive alterations (e.g., memory, attention, concentration, irritability). The clinical examination of anatomical factors (e.g., obesity, retrognathia, large tonsil, macroglossia) is an essential step to establish a diagnosis.[51,61] The presence of concomitant conditions (e.g., sleepiness, unexplained fatigue, hypertension, diabetes, metabolic syn-

TABLE 2 Evidence for SRB management with evidence supported by RCT or SR.

SRB management	Evidence supported by RCT, SR	Risk or benefit for OSA
Oral appliances		
• OS	RCT+ and SR+	Possible risk
• MAD	RCT+	Benefit
Behavioral therapies		
• EMG Biofeedback	RCT+ and SR+	Unknown
• Biofeedback with splint	RCT+ and SR+	Unknown
Drugs		
• Clonazepam	RCT±	Risk of increase AHI and sleepiness
• Clonidine	RCT+ and SR+	Risk of sleepiness
• Gabapentin	RCT+	Risk of sleepiness
• Botulinum toxin	RCT+ and SR+	Risk of increase AHI in elderly
• Pump proton inhibitors (Rabeprazole)	RCT+ (1 study)	Unknown
• Cyclobenzaprine	Under trial	No risk, contradictory on benefit
• L-Tryptophan	RCT-	Possible risk, no data
• Bromocriptine	RCT-	Unknown
• Propanolol	RCT-	Unknown
• Pramipexole	RCT-	Unknown
• Amitriptyline	RCT-	Unknown Unknown

Abbreviations: AHI, apnea and hypopnea index; EMG, electromyography; MAD, mandibular advancement device; OS, occlusal splint; OSA, obstructive sleep apnea; RCT-, randomized controlled trials with negative results; RCT+, randomized controlled trials with positive results; SR, systematic review; SRB, sleep-related bruxism.

drome) or occupation (e.g., airplane pilot) contribute to treatment planning.

It is important to note that not all OSA patients present symptoms. According to an Iceland cohort study with 822 individuals presenting moderate or severe OSA, 24% were minimally symptomatic, 32% presented disturbed sleep, and 42% had excessive daytime sleepiness.[62]

PSG or home sleep testing (HST) is required to confirm an OSA diagnosis and to assess the severity of the AHI and oxygen saturation. Complaints of sleepiness, fatigue, insomnia, or other symptoms leading to impaired sleep-related quality of life should be present to consider OSA a disorder.[25,63] Recently, it has been demonstrated that AHI is not necessarily the strongest outcome to predict the risk of morbidity and mortality associated with OSA severity, although it remains one of the outcomes used in clinics. Other factors have been proposed to assess OSA burden in an individualized manner (e.g., hypoxic burden).[64–67] Conventionally, in the clinic, AHI is graded as mild OSA when the number of events per hour of sleep is between 5 and 15, moderate from 15 to 30, and severe over 30 events.[48,66,67]

TABLE 3 Evidence for OSA management with evidence supported by RCT or SR.

OSA clinical management	Evidence supported by RCT, SR	Risk or benefit for SRB
Devices		
• CPAP	RCT+ and SR+	Benefit
• MAD	RCT+ and SR+	Benefit
Behavioral therapies:		
• Positional therapy	RCT+ (check)	Under trial
Drugs		
• Atomoxetine	RCT+	Risk suggested[a]
• Reboxetine	RCT+	Risk suggested
• Pump proton inhibitors (Rabeprazole)(63)	SR±	Benefit

[a]One case report on a child and a letter to the editor.[25,63]

Abbreviations: AHI, apnea and hypopnea index; EMG, electromyography; MAD, mandibular advancement device; OS, occlusal splint; OSA, obstructive sleep apnea; RCT-, randomized controlled trials with negative results; RCT+, randomized controlled trials with positive results; SR, systematic review; SRB, sleep-related bruxism.

When OSA and insomnia co-exist in the same patient it is COMISA for comorbid OSA and insomnia.[68] SRB is not a morbid condition, so it is not surprising that a recent study showed no difference in SRB frequency between OSA and COMISA.[45,69]

OSA clinical management includes positive airway pressure devices (continuous positive airway pressure [CPAP], BIPAP), MAD, myofunctional therapy, and medications that are still emerging. Some management options, such as positional therapy and acupuncture may be indicated as adjunct therapies to the above treatments. All have limitations and/or risks.[51,70] Other treatments not reviewed here are nasal-tonsil, maxillary-mandibular advancement, and hypoglossal nerve stimulation surgeries (Table 3).[51]

STRENGTH OF THE ASSOCIATION OR CAUSALITY OF CO-OCCURRENCE

Many studies from different countries reported a possible association between SRB and OSA. Indeed, 33% to 49.7% of individuals with OSA from Canada, Japan, Poland, Singapore, The Netherlands, and Thailand presented SRB. This is based on EMG scoring of 2 or more RMMA/h, a debated topic.[38,40–42,71–74] Also, 31% of patients with severe OSA have severe SRB (≥ 4 RMMA/h).[73] Few findings have shown that individuals with OSA present a higher chance of having SRB compared to the control group (OR: 3.96; 95% CI: 1.03–15.20; $p < 0.05$).[38] Individuals with SRB seem to have more severe OSA (higher AHI) and higher oxygen desaturation index (ODI).[38] And even individuals with a high risk of OSA (high score on Stop Bang risk questionnaire assessment), but with no PSG diagnosis for OSA yet, presented a higher BEI (bruxism episode index).[75] Alternatively, there are findings of a low association between SRB and OSA. According to a

study from Poland, individuals with both disorders (OSA and SRB) present large body movements and higher levels of jaw motor activity, non-specific to RMMA.[76]

In a scoping review, the strength of such an association was rated as possibly being spurious, that is, a causal relationship may be coincidental or due to other factors.[72] Though a few systematic reviews (SR) also showed a positive, but rather weak association between SRB and OSA, caution is recommended in their interpretation or finding extrapolation since some are based on only a few studies.[77–80]

Indeed, many factors may contribute to the perception of an association or possible causality between SRB and OSA. Among these, age, gender, and different phenotypes, that is, observable characteristics of an individual influenced by their genetics and environment, may explain differences and discrepancies between findings.

Besides, it was proposed that RMMA contributes to reversing upper airway collapse. A recent case–control study from Spain showed a negative correlation between BEI and OSA. However, it should be noted that the analyses excluded sleep arousal which is frequently associated with the onset of SRB. Despite such limitations, the authors reiterate that SRB can be a protective factor for OSA with an odds ratio below 1 (OR: 0.15 [0.036–0.68; $p < 0.05$]).[81]

Furthermore, the prevalence of SRB complaints and the prevalence of OSA are in opposition. While SRB self-complaints decrease with age, OSA is increasing.[3,5,56,57] Few explanations may concur to give the impression of a strong overlap between self-reported SRB and the observed OSA prevalence in the age period between 35 to 50 years, in which both complaints may occur in some individuals. This may explain the clinical perception that SRB and OSA are concomitant.[82] Although the co-occurrence between SRB and OSA is frequent, the causality is not yet established.

DOES SRB REALLY HELP PROTECT THE AIRWAY?

As introduced previously and described in more detail below, it was hypothesized that SRB may be a protective mechanism to open the airway and help restore patency at the end of an apnea or hypopnea event. A series of studies have shown the temporal association between sleep bruxism events (SBe) and apnea and hypopnea events (AHe). To clarify, in summary, an AHe occurring before an SBe is scored as a T1 temporal sequence and as a T2 sequence when it is the opposite (Figure 2).

The first publication describing that jaw muscle reactivation during sleep may contribute to upper airway opening is likely Hollowell and Suratt from 1991. They showed that individuals with OSA have more masseter activation during sleep than healthy ones. They hypothesized that masseter activation occurs like it was in a T1 type to stabilize the jaw, allowing the submental muscles to exert traction on the hyoid bone, as a proactive or a protective mechanism to help open the airway.[83]

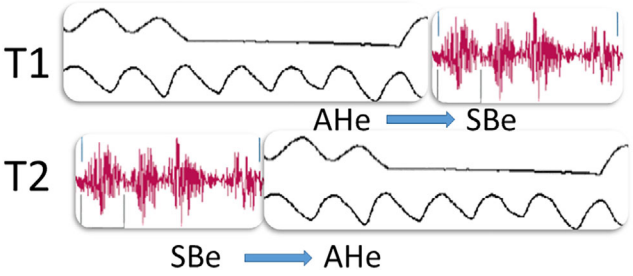

FIGURE 2 Example of the temporal association between SBe and AHe in patients with both OSA + SRB. AHe, apnea or hypopnea events; OSA, obstructive sleep apnea; SBe, sleep-related bruxism events; SRB, sleep-related bruxism.

A positive, but modest correlation between SBe and the breath amplitude ($R^2 = 0.26$; $p < 0.02$) was also shown in healthy young individuals with SRB (without OSA), which presented a big breath with the probable re-opening airway.[37] Not all SRB individuals presented such big breath with the onset of RMMA and it remains to be investigated if those presenting it will be more at risk of OSA with aging.

The investigation of the temporal association between the AHe and SBe was also studied in a sample of 10 individuals from Japan with co-occurring OSA (AHI>5/h) and SRB (SBe ≥4/h). It was observed that in a 5 min time window, before and after an AHe, 55% of events were T1 type (AHe before SBe), 25% were T2 type (SBe before AHe), and in 20% of episodes, there was no association. The two events occurred with less than 10 s in 86.8% for T1 and 65.8% for T2.[84] In a one-night PSG Thailand cohort study, using a split-night procedure (half of the night without and half with a CPAP device), SBe followed AHe in 73.5% of the 49 individuals with co-occurring OSA and SRB, a criteria of 5 s was used.[73] Only T1 was investigated in that study.

One major difference in the studies on the temporal association between SBe and AHe is the time window; again, there is no standardization since these studies were exploratory or pilot studies. The time window varied from 5 min in two studies and 5 s in two.[73,84–86] In a study from Japan, the 5-min time window was used to compensate for the possible sudden rise in autonomic sympathetic activities, known to occur 4–8 min before the onset of SBe.[84,86] Despite methodological discrepancies, overall the frequency of T1 was 3.7 to 10 times more frequent than T2 for obstructive events in the three studies.[84–86] Indeed, in the two other studies cited, T1 type was observed (AHe preceding the SBe) although the association was low.[85,86] It is noteworthy that in two studies, the T1 type dominated for OSA events while the T2 type was more frequent with central apnea.[39,85] In summary, it seems that the T1 type may be associated with a form of airway protection against airway obstruction when SRB co-occurs. The case–control study described above also supports that SRB, without concomitant sleep arousal, may also be a protective mechanism to reverse obstructive events (apnea and hypopnea). The OR was 0.15 (0.036–0.68; $p < 0.05$).[81] In the next

sections, we will explore a few possible phenotypes that may explain the variability in the findings described above.

PHENOTYPES OR CLINICAL FEATURES

OSA severity

Although some studies found more SRB associated with OSA in moderate to severe cases,[41,84–86] others reported much greater numbers in mild to moderate OSA, although with a modest correlation.[75,81] A 2019 study from Martynowicz presented a positive correlation ($r = 0.24$; $p < 0.05$) between AHI with BEI, which explains only 6% of the variability.[41] It also showed that a third of patients with severe OSA have a high frequency of SBe (≥ 4 RMMA/h).[73]

Other studies also showed that such increases are not exclusive to OSA. Indeed, a rise in SBe was also observed with primary snoring (PS) in individuals without OSA (AHI<5/h). In 129 individuals from Poland with PS, 76% presented SRB with sleep laboratory PSG with the use of an acoustic sensor and a nasal pressure transducer to assess snoring.[87] It was also reported that in individuals from Brazil, sleep snoring events were longer in SRB ($n = 45$) than in non-SRB ($n = 45$; 460 min vs. 401 min, respectively).[88] In contradiction, in a Netherlands study with 968 individuals reporting SRB (no PSG), no association was found between SRB and PS.[89]

SRB and hypoxia

Does hypoxia contribute to SRB? This is an open question recently discussed in a review on the role of intermittent hypoxia.[90] In non-OSA healthy, young individuals with SRB, mild hypoxia (i.e., drop in oxygen level by less than 2%) was observed in association with RMMA in about 20% of subjects with the onset of RMMA.[91] It remains to be demonstrated if these individuals will be more vulnerable or at risk of developing OSA with aging or after gaining weight. The evidence in individuals with OSA and SRB is divergent. In two descriptive studies, with an adequate sample size of OSA individuals, it was observed that sleep bruxers presented higher ODI than sleep non-bruxers.[38,40] In another study with 101 OSA participants, the association between hypoxia and SRB was mild (40% of women). Furthermore, the type of RMMA seems to matter, a mild positive correlation was observed between phasic bruxism (three EMG bursts per RMMA episode) and ODI ($r = 0.23$; $p < 0.05$), and there was a negative correlation between phasic bruxism and minimal saturation ($r = -0.26$; $p < 0.05$).[41]

Type of SRB episode

The type of SRB event is also relevant to the association with OSA since at least three different studies found that most of the SB events associated with OSA are phasic.[38,40,41] SRB phasic events were positively correlated with snore intensity despite the body position, in individuals with PS (snoring with AHI<5).[87]

Sleep position

Sleep position may also be considered as a risk factor for SRB genesis. Indeed, one study showed that SRB events are more frequent in the supine position in individuals with OSA[43] and a few descriptive studies showed that SRB is more frequent in supine OSA.[92–94] A similar observation was also made in individuals with PS (without OSA) and SRB, a higher frequency of both events in the supine position was found.[87]

OSA and sleep stage

Sleep is divided into non-REM and REM stages. Non-REM light sleep is labelled as stages N1 and N2 and the deep sleep stage is N3. In otherwise healthy individuals, most SBe are observed in non-REM light sleep.[95] In individuals with co-occurring OSA and SRB, there is a higher percentage of AHe also in non-REM light sleep stages N1 and N2.[39,42] Another study investigated whether SBe occurrence across sleep stages and specifically in REM sleep, was a specific phenotype in individuals with OSA. That hypothesis was rejected.[94] Further studies will provide more information on that topic.

Clinical parameters

Another important element is the fact that some studies found that SRB is more frequently associated with OSA in men than women,[41,42,73,82] those who are nonobese, and those with low body mass index (BMI)[42] or overweight.[3,82] Recently, a retrospective study of 100 individuals with OSA reported that being male is a strong risk factor for the high frequency of SBe-RMMA (SBe ≥ 4/h; OR: 4.01, 95% CI: 1.02–15.88).[73]

GENETICS

Many gene candidates, mainly related to serotonin and dopamine, have been associated with awake and sleep bruxism. These studies include populations with and without OSA. Although the topic is too large to be covered in this review, a recent publication noted above is noteworthy. Indeed, a genome-wide study in a population from Finland ($n = 12{,}997$ individuals, 3.26% of a larger sample) found an association of probable bruxism to the MYO3B gene. SRB was described as a heterogeneous condition since there was some correlation with co-occurring pain, sleep apnea, gastroesophageal reflux disease, upper respiratory diseases, psychiatric traits, plus the use of antidepressants

and sleep medication was observed.[30] Although no gene has been proven as responsible for the association between OSA and SRB, a study with 74 SRB individuals, 28 with OSA, and 125 healthy ones suggested the serotonin HTR2A rs2770304 polymorphism may affect this association.[96] In the absence of OSA, one dopaminergic variant was associated with the risk of SRB and serotonin-related one to its pathophysiology.[31,32,96] Indeed, lower serotonin levels were shown to be associated with higher ODI scores and an increase in the number of obstructive and central sleep apnea episodes, as well as with increased sleep bruxism episodes index.[97,98]

HOW TO EVALUATE AND MANAGE COMPLEX CASES

It is important to assess the possible co-occurrences of SRB and OSA to reduce health risks and improve quality of life. An operational and methodic clinical approach in the diagnosis and management of SRB is suggested when co-occurrent OSA, insomnia, or, to a certain extent, GERD (gastroesophageal reflux disease) is suspected. The use of an operational approach helps clinicians assess signs and symptoms (e.g., tooth wear, pain, Mallampati or Friedman scores), and improve referral prioritization in documenting history, clinical observations, and summary of screening tests assessing the risk of OSA. Interdisciplinary collaboration between dentists and sleep physicians or other sleep-related professionals such as speech or manual therapists, and otorhinolaryngologic or oral and maxillofacial surgeons is required to offer better health outcomes for the patient.[99]

SCREENING TOOLS

Some web-based screening questionnaires help clinicians to better evaluate patients and define the presence of OSA in individuals with SRB. Among them, the *STOP-Bang questionnaire* is clinically useful in evaluating the risk of OSA; it seems to be more accurate in detecting mild, moderate, and severe OSA in comparison to the *Berlin* questionnaire or the *Epworth Sleepiness Scale*. The *STOP-Bang questionnaire* has a higher sensitivity and diagnostic odds ratio for detecting OSA and it can be used in the first evaluation, before defining the need for a PSG or an HST.[100] The *Epworth Sleepiness Scale* is more sensitive to assess the consequences of OSA in men. The *STOP-Bang questionnaire* helps assess the risk of various co-occurring conditions to OSA and fatigue complaints, an important sign in women.[101] So far, the diagnosis of OSA is done by a qualified sleep physician.

To assess the presence of SRB, some questionnaires have been developed for research or clinical use. The Standardised Tool for the Assessment of Bruxism (STAB) is an extensive research tool, under validation, to evaluate awake bruxism and SRB, etiology, consequences, and comorbidities.[102,103] As recently suggested, we need "applicable, affordable, and accessible" questionnaires, such as the *BruxScreen* from Lobbezoo, and the brief one from Paesani, focusing on grinding and clenching to reach criteria for clinical practice.[104,105] The questionnaire from Paesani is simple to use in clinics to discriminate SRB and awake bruxism, although, like all tools, it has some limitations (Table 4).[105]

TABLE 4 Paesani questionnaire.

Answer yes or no to the following questions:[91]
1. Sleep grinding item: are you aware of the fact that you grind your teeth during sleep?
2. Sleep grinding referral item: did anyone tell you that you grind your teeth during sleep?
3. Sleep clenching item: on morning awakening or awakenings during the night, do you have your jaws thrust or braced?
4. Awake clenching item: do you clench your teeth while awake?
5. Awake grinding item: do you grind your teeth while awake?

CLINICAL EXAMINATION

The OSA variables to collect and grade include BMI (weight and height), a score of the shape of oropharyngeal pillars (Mallampati and Friedman), angle classification, palate (narrow and ogival), and maxillary/mandibular relation such as retrognathia (Figure 3).[106] For SRB, a method for grading tooth wear, pain, and to a certain extent tongue indentation or check linea alba is included in the comprehensive questionnaire and clinical examination, such as the BruxScreen.[104]

MANAGEMENT

As noted above, the usual management of SRB includes oral splints, a biofeedback device, and medication. In the absence of pain or mood disorders, physical therapy or psychology are not yet based on solid evidence.[107] For OSA, breathing devices, CPAP or MAD, are commonly used, as speech or manual therapy, oropharyngeal exercise and body weight therapies, and in some specific cases, hypoglossal nerve stimulation and surgeries (ENT, maxilla, bariatric) or orthodontics.[51,64]

It is possible that not all patients with SRB need therapy; indeed, there is evidence that suggests that the level of "harm" should drive the decision.[104,108] The clinical SRB management should be accessed in a personalized way, when signals or symptoms are present, such as pain, discomfort, tooth grinding noise disrupting sleep partner, tooth damage with pain, function, or esthetic complaints.

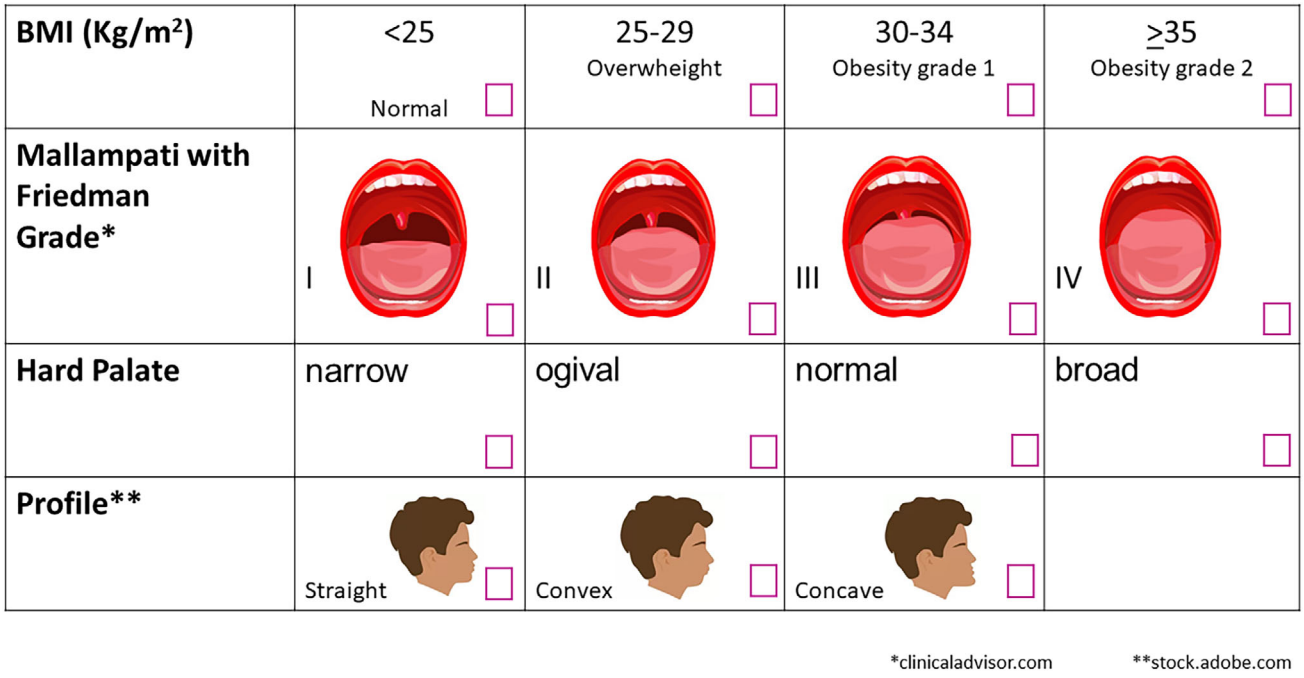

FIGURE 3 Guide for clinical assessment of OSA patients. OSA, obstructive sleep apnea.

Oral appliances: OS and MADs

Occlusal splints have been used for decades to control harm related to SRB (e.g., sound, tooth damage). It seems more probable that OSs are not able to cure or stop the oral behavior, or habit, of grinding or clenching teeth during sleep; most likely some patients may continue to "bite" on their device. However, there is a low to modest level of evidence showing that OSs are an effective approach to protecting the teeth and managing SRB sound since they may reduce (by about 30%–40%) the frequency and intensity of SRB activity in some patients.[107,109] Two observational studies reported that inserting an oral appliance in the mouth, a device without occlusal contact also reduces the index of RMMA.[110,111] It remains to be demonstrated if tongue position or change in airway patency or the augmentation on salivation may already be influenced by the insertion of a non-occluding device (e.g., similar to Hawley).

Meanwhile, it was observed that when an individual with OSA uses an occlusal splint for SRB, there is a risk of worsening the AHI or respiratory event index (REI).[112–114] In one of these studies, the REI increased in individuals receiving the following: standard occlusal splint, palatal appliance (no occlusal surface), and thick splint (vertical rise of 5–8 mm). The REI increased in 15 to 23% of young mild-OSA patients (15 events or more per hour) and in 10 to 40% of individuals without OSA (rise by 5 events or more per hour).[114] Such an increase, although it is low, may be an additive risk in some individuals with OSA. As is frequent in clinical science, one study on temporomandibular disorder (TMD) patients reported opposite findings: AHI or ODI was below or over 5 at follow-up. A significant reduction in AHI was noted at 2 weeks follow-up. Both maxillary and mandibular oral orthotics were used.

Thus we recommend the following: (1) use the above screening questionnaires; (2) assess the putative role of retrognathia, deep palate, size of the dental arch, macroglossia, high Mallampati or tonsil Friedman scores; (3) when in doubt ask for a sleep test; and (4) if SRB and OSA are confirmed, consider use of a MAD, with or without protrude titration position, instead of an occlusal splint.

In the usual management of OSA, it is well known that MADs can be a good alternative if the patient does not accept CPAP.[51] Studies with MADs found a reduction in RMMA and the other outcome measure, BEI, with different types of devices, such as the Silencer,[116] Narval,[117] and Twin block.[118] The MAD does not necessarily need to be in maximum protrusion to reduce the BEI. With a MAD with 50% of the maximum protrusion, a reduction in the BEI was 65% in relation to baseline values after 30 days of MAD use and the quality of sleep [measured with the Pittsburgh sleep quality questionnaire (PSQI)] was 60% better (both with $p < 0.001$).[118] Moreover, when the MAD was compared with an occlusal splint in a randomized controlled trial, it remained better in reducing BEI after 3 months of treatment.[119]

Considering these findings, it could be questionable why MADs are not used for all SRBs instead of an OS. A few explanations could be proposed: (1) the standard training for dentists has indicated OS for SRB for decades; (2) the cost–MADs are in general more expensive and may not be covered by public or private insurance plans; and (3) OSs are usually more comfortable and easier to accept than MADs.

Indeed, a recent paper showed that although MADs induce a greater reduction in SRB activity index, patients preferred the comfort of an OS.[120]

It is then suggested that patients with SRB 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, be offered a MAD or a CPAP.[99] If the patient's choice is the OS, it should be used with the CPAP if they are OSA positive.

Based on recent literature, SRB improves with CPAP[73] and the co-occurring SRB and OSA can be managed, with an equipotent efficacy, by CPAP and MAD.[121,122] Even though CPAP remains the most efficacious treatment for OSA,[123] the decision should be based on OSA severity and patient acceptability and tolerance to the type of treatment.[124,51] What matters most with OSA is to get treatment, as demonstrated in the Japanese population, with a lower mortality of all causes with CPAP.[53] A similar finding, although with a smaller sample, also showed lower mortality rates for MADs, similar to the one with CPAP.[125] Furthermore, since there is a high subject variability between the two comparative CPAP/MAD trials cited above, phenotyping characterization needs to be precise in future studies to offer patients the best personalized treatment.

In summary, the level of clinical usefulness evidence for OS in the presence of SRB alone is rather high, although the magnitude of the effect, that is, reduction of SRB motor activity, is modest, about 30–40. For the co-occurrence of SRB and OSA, the level of evidence is modest to high, due to the large response variability between subjects in the studies reported. Again, caution is recommended if an OS is used by patients with OSA, especially in women with chronic use of OS near menopause, since there is an increased risk of OSA. Phenotyping seems needed as a route of investigation to select treatment with more accuracy.

Medications

Many medications have been tested for SRB in the absence of OSA (e.g., anti-convulsant, anti-depressive, anti-hypertensive, anti-histaminic, anxiolytic, anti-psychotic, dopamine precursor or agonist, neurotoxin protein).[49,107] Clinicians should be cautious in using medication to manage SRB 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.[49,107] In addition, when these drugs are prescribed, the clinician should know their indications, contra-indications, and side effects. For instance, to our knowledge, there are no publications on the pharmacological approach for this specific co-occurrence of SRB with OSA.

Based on a recent systematic review, the following medications reduce SRB with a reasonable level of certainty: clonazepam, clonidine, gabapentin rabeprazole, and hydroxyzine.[107,126–130] However, they have side effects and risks such as sleepiness, hypotension, and addiction. They should be used with caution with OSA subjects who may also present various comorbidities (diabetes, blood hypertension, depression). Tables 2 and 3 show the evidence supported by RCTs or SRs for both SRB and OSA management and the risk or benefit for other conditions (SRB or OSA).

Botulinum toxin is known to reduce the intensity and the strength of SRB events, but not the frequency, an expected effect since SRB is most likely centrally driven.[107,130–134] There is no strong evidence for botulinum toxin's effect on OSA. A pilot study on snoring (with no OSA) reported a reduction in subjective snoring with botulinum toxin type A.[135] The level of clinical usefulness evidence is high. Many studies converge toward a positive beneficial effect although cost can be an issue and long-term side effects are unknown. There is no data yet on use for co-occurring SRB and OSA.

The effect of clonidine, a central-acting antihypertensive medication, was tested in two SRB RCTs. Clonidine also reduced SRB motor activity, in a dose–response fashion (30%–60%). The smaller dose was less powerful and did not trigger hypotension, a side effect observed in 20% of study participants with the higher dose.[126,127] Clonidine can also trigger awake-time sleepiness in some individuals, but it did not interfere with AHI.[136] The level of clinical usefulness evidence is modest to high due to side effects; use is recommended with medical supervision. There is no data yet on the use of co-occurring SRB and OSA.

Clonazepam, a benzodiazepine, and anti-convulsant, was tested for SRB in two RCTs and the findings were divergent. The first study showed that clonazepam reduces SRB motor activity from 9.3 to 6.3/h of sleep. That study was done with individuals in their mid-forties without OSA, but with insomnia, anxiety, depression, and sleep-related movement disorders. Clonazepam improved sleep quality and PSG parameters (total sleep period, total sleep time, sleep efficiency, sleep latency, and time awake during the total sleep period); however, it slightly increased the sleep respiratory AHI.[137] Another randomized controlled trial, from dental sleep investigators and done with individuals in their mid-twenties without comorbidities, concluded that clonazepam (1 mg) did not reduce SRB compared to a placebo.[127] The level of clinical usefulness evidence is questionable and contentious. Caution is recommended since clonazepam is known to have a risk of addiction and its putative effects on sleep breathing may restrict its use in individuals with OSA.[24]

In an open control study with another anticonvulsant, gabapentin (given at bedtime for 2 months) was tested face to face with an OS (used during sleep for 2 months). In those two mid-age samples, both were found to reduce SRB motor index by about 50% and 40%, respectively.[128] Sleep duration, sleep latency, and sleep efficiency (% time asleep in bed) were all improved with gabapentin and not with OS. Such findings are not surprising since gabapentin is known to induce awake time sleepiness and improve sleep quality. Again, caution is recommended since gabapentin, especially in older men, can exacerbate sleep breathing, that is, increase the AHI.[138] Due to the open nature of the evidence, the clinical usefulness of evidence is questionable and replication is needed. There is no data yet on the use of co-occurring SRB and OSA.

Pramipexole, a D-3 preferred receptor agonist, was also challenged in an open randomized crossover study. No significant effect was observed on SRB outcomes and only a small effect on OSA variables.[139] Use of other related dopaminergic medications also reveals low usefulness in managing SRB. Indeed, levodopa (L-dopa), a dopamine precursor, and bromocriptine, a dopaminergic preferential agonist with serotonin and adrenergic receptor affinity, also reduced SRB motor activity, but to a lower extent, about 20%–30%. The level of clinical usefulness evidence of dopaminergic agents is modest and it is not likely a robust candidate to manage SRB alone or for co-occurring conditions.[140]

The effect of hydroxyzine, an antihistaminic, on SRB was shown only in children. Two RCTs found a reduction with hydroxyzine for reported sleep bruxism estimated on the questionnaire by parents, that is, no EMG measure to assess motor activity.[130,141] It was also described as a possible option for children and adolescents with SRB in two systematic or scoping reviews.[142,143] Again, improvement in sleep quality could have positively influenced the perceived benefit, and the medication was well tolerated. The level of clinical usefulness evidence for adults is unknown. There is no data yet on the use of co-occurring SRB and OSA.

On the other hand, some drugs have been tested to treat OSA without SRB. It may be possible that these will have the opposite effect, with a rise in SRB. As an example, atomoxetine or reboxetine, two selective norepinephrine reuptake inhibitors, are pharmacological candidates that contribute to reducing AHI and even more, improve oxygenation.[144–147] Their mechanism works by increasing upper airway muscle tone, but it was not tested to see if SRB or sleep-related tension-type headaches could be aggravated.[148] There is no data yet on the use of co-occurring SRB and OSA.

Although cyclobenzaprine, a myorelaxant, is often prescribed by dentists and physicians for SRB and TMD,[133,149] there is no evidence for SRB or OSA alone or co-occurring. Caution is recommended since it can induce dizziness and falls. There is no data yet on the use of co-occurring SRB and OSA.

In addition, the use of a proton pump inhibitor, rapeprazole indicated for GERD control, was evaluated for SRB in an experimental RCT. A reduction in the number of SRB motor events was reported.[129] Another study used the same drug to manage OSA without SRB and, interestingly, found a reduction in AHI.[129,150] The level of clinical usefulness evidence is low and questionable due to the open nature of the case–control study design. There is no data yet on the use of co-occurring SRB and OSA.

Other approaches

Another option for OSA and snoring management is positional therapy for individuals with positional OSA.[151] Positional OSA is defined as more than 50% of AHI in supine sleep position compared to non-supine positions.[152] Individuals with SRB, alone or with snoring, who tend to sleep mostly in the supine position may present a higher risk of snoring and gastric reflux.[87,92,93] To assess the potential benefits of correcting supine sleep position on SRB occurrence, a pilot study using a sleep position trainer device, based on a biofeedback vibration algorithm located on the neck (Night Shift),[153] reduced RMMA onset by about 50% (24%–85%) in about half of the individuals tested. (Suzuki Y, Tokushima University, Japan, personal communication (2024)). Another recent pilot study showed that SRB events are significantly reduced by sleeping on the low Fowler's position (15°–30°), this is a mild effect attributed to a reduction in intracranial pressure in a comparative study with a medication.[154] There is no evidence about the positional therapy for patients with co-occurring SRB and OSA.

There is no direct data on the sleep hygiene benefit for co-occurring SRB and OSA. However, since both SRB and OSA can be associated with sleep quality complaints,[3,82,155] the importance of good sleep hygiene might help both conditions. Individuals with mild to moderate OSA may have symptom relief if they are trained in correct sleep hygiene and healthy lifestyle habits.[156] As health professionals, dentists should inform patients about the potential effects of excessive use of screens, such as anxiety, physical inactivity, stress disorders, and daytime dysfunction.[157] Psychology and hypnotherapy also have promising benefits for SRB, but more studies are needed to confirm their efficacy.[107,158,159]

In a recent systematic review, certainty of evidence based on literature tended to support the use of biofeedback to manage SRB.[107] Few sleep devices are available on the market based on contingent electric stimulation (GrindCare, Sunstar) and some vibration systems are embedded in OSs (bruXane, AesyBite).[10,26,160,161] So far, it seems these devices are well tolerated and do not trigger sleep disruption, and the level of clinical usefulness evidence for SRB alone is high although debatable.[162] The adaptation to such device use may be a concern.[163] The effect is modest on related motor activity (about 30%–40%). Furthermore, caution is recommended with OSs with vibration devices on the palatal area. Indeed, the size of the device occupying the palatal space (reduction of the oral and pharyngeal space) is a risk for individuals with sleep breathing disorders; exacerbation of AHI was observed in open studies in some patients for whom phenotype explaining such effect needs to be identified.[112,113] There is no evidence for use in co-occurring SRB and OSA.

For the other clinical approaches (e.g., cannabis, transcranial magnetic stimulation) or surgical management for OSA (e.g., nose, oropharynx, or maxillofacial, hypoglossal nerve stimulation), there is not yet evidence to recommend use for co-occurring SRB and OSA. Clinical guidance is suggested in Table 5, however not all recommendations are proven or evidence-based yet.

LIMITATIONS

This narrative review is not a systematic review and is not based on a hypothesis. Indeed, the debate on the putative

TABLE 5 Recommended clinical guidance for SRB and OSA.

SRB and low risk of sleep-related breathing issues		**SRB and high risk of sleep-related breathing issues:**
Screening based on interview: • Nocturnal symptoms: sleep time snoring, cessation of breathing, nocturnal enuresis, and wake time sleepiness; • Diurnal symptoms: fatigue, sleepiness, cognitive changes, headache; • Clinical examination: body mass/obesity, anatomical oral and pharyngeal characteristics listed above) • Questionnaires: Stop Bang, Berlin, or for sleepiness, Epworth Sleepiness Scale. • Decision for sleep test under medical supervision or not.		
SRB + mild to moderate OSA:	**Gray Zone****	**SRB + moderate to severe OSA, mainly severe hypoxia:**
• MAD or occlusal splint (lower seems safer, and it depends on how the OSA has been managed, CPAP, surgery)		• CPAP may be more indicated. If needed, combine with an occlusal splint, ideally a lower jaw one, based on preliminary evidence, although auto-CPAP may mitigate the negative effect of the palatal thickness of the maxillary splint).
• Sleep position corrector (cushion of intelligent devices) may be indicated for position OSA.		• MAD may be indicated in some cases, especially with non-adherence to CPAP.
		• Sleep position corrector (cushion of intelligent devices) may be indicated for position OSA.

Important notes:

(i) *We should inform patients not to use occlusal maxillary splint alone, without simultaneous use of CPAP. We should remind them that OSA is morbid, and SRB is not.
(ii) Emerging pharmacological approaches may contribute to alleviating the consequences of OSA (e.g., atomoxetine, Rx by physician).
(iii) Be cautious with peri and post-menopause women. Fatigue is dominant over sleepiness and apnea-hypopnea and hypoxia-related to OSA tend to occur mostly in the second part of the night, during REM stages. OSA severity assessment by a sleep physician is essential.
(iv) Unfortunately, it is not as simple as described, since some individuals with possible sleep breathing disorder may fall in between, in a gray zone. It is the moment to use clinical judgment based on evidence and sleep physician's report by patience understanding and collaboration.
(v) Our goal is to offer the best management in a personalized way.

**The gray zone is related to asymptomatic OSA patients who may search for SRB management. Prosthodontists should always evaluate the risk of OSA, since some patients are not aware of OSA and do not present symptoms.

role and causality link(s) for the interplay between SRB and OSA remains open. Phenotypes (clinical characteristics) for a specific endotype (mechanism(s) toward treatment) remain to be established for co-occurring SRB and OSA. Due to morbidity and mortality associated with OSA, it is highly recommended to manage the co-occurrence of SRB and OSA in collaboration with a physician trained in sleep medicine. Collaborative work is essential for patient health and well-being.

FUTURE DIRECTIONS

1. Identification of specific clinical at-risk characteristics (phenotype) of SRB-OSA co-occurrence.
2. Identification of risk factors (anatomical, medical history, versus selection of best restoration design to preserve and optimize airway function).
3. Identification of teeth position, as well as tongue position on the breathing function/OSA severity, and the role of teeth inclination on prosthodontic planning.
4. Endotype identification, selecting the best treatment based on mechanisms.
5. Recognition of the risk factors that a given individual with SRB may present that exacerbate OSA and vice versa.
6. Assessments of the relationship between SRB and OSA severity, anatomical features (e.g., retrognathia, palatal wideness and deepness, airway pillars, and tonsil obstruction/Mallampati and Friedman scores), and comorbid conditions (e.g., obesity, gender/age, hypertension, diabetes, medication).
7. Evidence-based algorithm to guide clinicians on how to manage SRB with a mild level of OSA without health risk versus a severe OSA case with comorbid risk. These scenarios require different approaches.
8. Role of Comisa in exacerbating the association SRB-OSA in patients with comorbid insomnia. The presence of insomnia may trigger a lack or difficulty in adherence to SRS and OSA clinical management strategies.
9. Finally, be ready for the use of monitoring tools related to SRB (muscle activity recording and bite sensors on oral devices, owing to their limitations). Be ready also

for OSA monitoring tools (apps and watch or ear sensors available for home oxygen measurements by patients over weeks and months). Recognize that such devices may trigger orthosomnia, i.e., over-awareness of sleep quality and physiological efficiency performance that may trigger anxiety and insomnia.

CONCLUSIONS

The association between SRB with OSA is not definitively demonstrated. Although there have been numerous publications on the topic, the evidence supporting a solid cause-and-effect relationship is lacking. OSA and SRB are probably associated with a subgroup of at-risk individuals, with risk to be identified as noted in the above sections. Collaborative work between prosthodontists and sleep physicians offers a higher likelihood of success and health protection for individuals with SRB and at-risk levels of OSA.

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CONFLICT OF INTEREST STATEMENT

There is no direct COI in relation to this narrative critical review. Cibele Dal Fabbro, Thomas Bornhardt-Suazo, Anaïs Landry Schönbeck, and Micheline de Meyer have no conflicts of interest to disclose in relation to this paper. Gilles Lavigne: no direct conflicts of interest in relation to this paper, although he is a consultant for Straumann, Switzerland, and he receives financial support for oropharyngeal cancer and sleep research from Panthera, Canada, at the CHUM-stomatology department.

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