

# An Operational Clinical Approach in the Diagnosis and Management of Sleep Bruxism: A First Step Towards Validation

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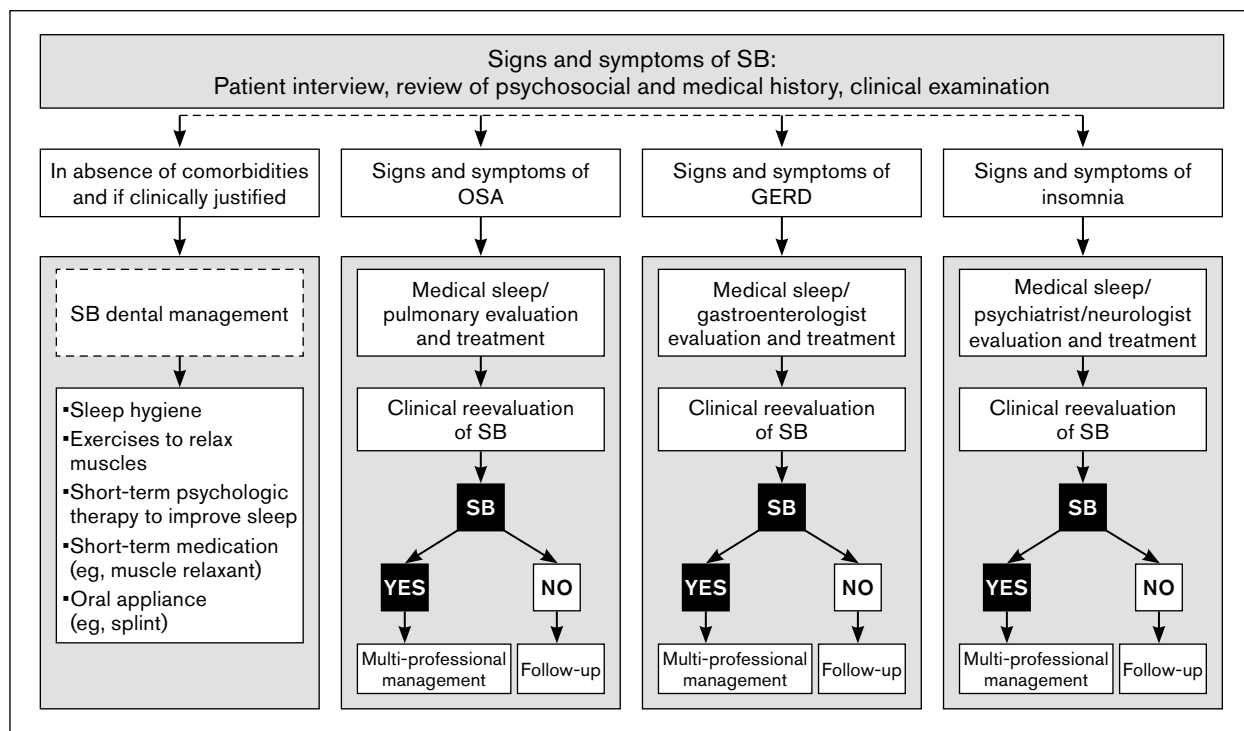
Exacerbation of nighttime sleep-related oromotor activity is often recognized as a relevant clinical entity commonly known as sleep bruxism (SB). Many pragmatic issues about SB diagnosis and management remain controversial. Therefore, within a critical review of the literature, this article proposes an operational clinical approach for SB diagnosis and management, with a focus on three comorbidities frequently occurring in relation to sleep: obstructive sleep apnea (OSA), gastroesophageal reflux disease (GERD), and insomnia. In the absence of any comorbidities, and if clinically justified, short-term medication and/or splints may be considered. If a comorbid condition is suspected, then the patient should be screened for OSA, GERD, and insomnia. For OSA screening, the Epworth Sleepiness Scale, STOP-Bang, and NoSAS questionnaires are available validated tools. For GERD screening, a positive patient report, whether associated or not with clinical signs and symptoms of heartburn and/or regurgitation, can be tested. For insomnia screening, report of difficulties initiating or maintaining sleep or of early morning awakening more than three times a week may be useful for diagnosis clarification. An adequate clinical approach for comorbid SB requires that both SB and the related comorbid condition be properly assessed and managed. Very often, improvement of SB with treatment of the associated condition will confirm the relationship and establish a more precise diagnosis (ie, secondary SB). Clinicians intending to manage SB should be able to identify these possible clinical interactions, and, if needed, perform an integrative multidimensional approach. Some approaches will benefit from a multidisciplinary approach for achieving therapeutic success. *J Oral Facial Pain Headache* 2020;34:236–239. doi: 10.11607/ofph.2616

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The occurrence of rhythmic masticatory muscle activity (RMMA) during sleep is a physiologic phenomenon that is present in most individuals at a low frequency per hour without consequences. RMMA during sleep may function to lubricate the oroesophageal tube.<sup>1</sup> However, when increased in both frequency and amplitude, this activity is most likely identified as sleep bruxism (SB). SB can be associated with some clinical consequences for the stomatognathic system (eg, tooth damage, pain) and may compromise quality of life (QoL).

Thus, improved operational diagnosis and management approaches are required. It is noteworthy that not all patients with SB need a full sleep polysomnographic evaluation or extensive treatment. What leads RMMA to progress from a usual physiologic activity to a sleep disorder in certain individuals is not yet established.<sup>2</sup> The interrelations among risk, vulnerability, psychopathologic factors, and comorbidities must be better understood.<sup>3</sup>

The overlap of SB with different clinical conditions, such as mood, pain, or other sleep disorders, leads to a multifactorial conceptualization. The current scientific evidence points mainly to overlaps with obstructive sleep apnea (OSA), insomnia, and gastroesophageal reflux disease (GERD).<sup>3–6</sup> Some aggravating or triggering factors bring these three conditions together in a possible unidentified triad in the exacerbation of RMMA that can lead to SB and tooth grinding. Use or abuse



**Fig 1** Flowchart of operational clinical approach for the diagnosis and management of sleep bruxism (SB). OSA = obstructive sleep apnea; GERD = gastroesophageal reflux disease.

of certain substances, such as anti-depressants (ie, selective serotonin reuptake inhibitors) or cocaine, can also trigger or aggravate bruxism, but it is less clear whether they also influence SB. The intent of this paper is to focus on the three most frequent comorbidities occurring in relation to sleep: OSA, GERD, and insomnia.

In the era of precision medicine, better identification of SB phenotypes is mandatory to improve its diagnosis and management in the absence or presence of comorbidities. Among the risk factors and phenotypes are the following: lifestyle; dominant supine sleep position; association of RMMA with sleep arousal; caffeine, nicotine, or alcohol use; hypertension; oropharyngeal/craniofacial characteristics known to be dominant in OSA; obesity; and respiratory effort that can be linked to OSA and/or GERD. Rare comorbid neurologic conditions are sleep epilepsy and rapid eye movement (REM) behavior disorders, which require adequate vigilance from the clinician.

Based on the evidence that SB may overlap with other sleep-related conditions, individualized diagnosis and management protocols are mandatory. The clinician who aims to manage SB should be able to identify and adequately assess these possible interactions and refer the patient to different specialties when necessary.

**Table 1 Sleep/Awake Bruxism Grading System**

Possible	Based on a positive self-report only.
Probable	Based on a positive clinical inspection with or without a positive self-report.
Definite	Based on a positive instrumental assessment with or without a positive self-report and/or positive clinical inspection.

Adapted from Lobbezoo et al<sup>2</sup> (2018).

In this context, it is understood that the clinical assessment of SB patients must be comprehensive and individualized. The main goal of this paper is to provide a pragmatic route to aid the clinician in SB diagnosis and management. Therefore, the following operational clinical approach is proposed to improve SB diagnosis and management (Fig 1). Such a challenging exercise is an assumed work in progress that will need further validation.

## Operational Clinical Approach

### In the Absence of Comorbidities

A patient interview, review of psychosocial and medical histories, and clinical examination of SB complaints

can reveal whether SB is possible or probable (Table 1).<sup>2</sup> Common indicators of SB include patient awareness of clenching and/or grinding and sleep partner complaints of tooth grinding. Because self-reports are not always precise and may not be strongly related to SB consequences or comorbidities, whether these complaints show an association with clinical signs and symptoms—such as tooth wear or fracture of teeth and/or restorations—can be assessed, but it should be noted that clinical signs and symptoms are not an absolute criterion. Complaints of morning masticatory muscle fatigue or pain/headache may also be an indicator of SB. In the presence of nonrestorative sleep and/or morning headache, the clinician should suspect insomnia, periodic limb movement, or sleep disorder-related breathing, such as OSA.

If SB management is to be initiated in the absence of comorbidities, it may be clinically justified to progress from less invasive management protocols (eg, sleep hygiene, exercises to relax muscles, short-term psychologic therapy to improve sleep) to short-term medication (eg, muscle relaxants) and/or oral appliances (eg, splint).<sup>3,7</sup> It should be noted that the level of evidence of such management strategies is modest.<sup>8</sup> Only one medication, clonidine, is currently supported by the evidence reproduced by independent investigators.<sup>9,10</sup> Clonidine is for short-term use in SB patients with obvious cardiac sympathetic overactivity during sleep. Clonidine has a major risk of side effects (eg, hypotension), is off label, and needs to be prescribed by a physician. Botulinum toxin is another alternative for reducing the strength (but not the onset or frequency) of RMMA activity, but stronger evidence is needed for its use.<sup>11</sup>

### **Comorbid SB with OSA, GERD, or Insomnia**

When examination of SB complaints suggests the presence of comorbidities, the same approaches as above can be used in addition to assessing whether the SB is associated with consequences such as worse QoL/wellbeing or with another comorbidity (eg, mood, hypertension, diabetes mellitus). Then, the patient should be screened for OSA, GERD, or insomnia using some of the available tools. For OSA, the following validated tools/measures can be valuable aids: a score of > 10 points on the Epworth Sleepiness Scale<sup>12</sup>; positive answers to more than 3 items on the STOP-Bang<sup>13</sup> questionnaire; and/or a score of > 8 points on the NoSAS (neck, obesity, snoring, age, sex) questionnaire.<sup>14</sup> Fatigue can be noted from self-reports or using the item in the STOP-Bang. For GERD, screening is based on patient complaints and a clinical interview (ie, positive patient report, associated or not with signs and symptoms of heartburn or regurgitation). For insomnia, screening is based on complaints and a patient

interview related to difficulties initiating or maintaining sleep or of early morning awakening (acute or chronic) more than three times a week. Screening may be performed using the validated Insomnia Severity Index.<sup>15</sup> Self-report questionnaires to assess sleepiness or fatigue and sleep partners complaining of snoring or cessation of sleeping help to improve screening reliability.

In the presence of positive evidence and/or any doubts, evaluations from a sleep physician should be requested for OSA (including breathing parameters and recording of at least the masseter muscle [possible at home]), GERD (the patient should be referred to a gastroenterologist for a sleep test with an esophageal sensor probe [possible at home]), and insomnia (no sleep recording test is needed unless comorbidities are present or in the case of diurnal hypersomnia, and then multiple sleep latency tests are done in the laboratory).

If SB management is to be initiated in the presence of comorbidities, it is suggested to follow the above list of recommended treatment protocols with the exception of a maxillary/palatal splint in OSA cases, as this could exacerbate the respiratory disturbance index (apnea/hypopnea events per hour of sleep).<sup>16,17</sup>

It is of significant importance to explain to the patient that their condition will probably require multi-professional clinical management for their concomitant SB and each specific comorbidity.

For OSA and SB, this management may progress from:

- Sleep hygiene; otorhinolaryngologic inspection in order to exclude nasal or pharyngeal obstruction; diet and exercise to control weight; oropharyngeal exercises; or, if OSA is dominant when in the supine position, a sleep-positioning device (eg, cushion or electronic trainer)
- Continuous positive airway pressure devices or an oral appliance such as a mandibular advancement device
- Other more invasive treatments, such as tongue/nose or bariatric surgeries and hypoglossal nerve stimulation

For GERD and SB, the treatments may progress from:

- Dietary advice and nutritional support to avoid acidic foods, eat smaller portions of food with shorter intervals, etc
- Behavioral measures to avoid the supine position (eg, sleep-positioning device) and/or elevate the bed

- Medication such as prokinetics or appropriate antacid medication
- Gastroenterologic surgical procedures, etc

For insomnia and SB, the treatments may progress from:

- Sleep hygiene; exercise during the day; sleep-restriction stimulus control therapy; cognitive behavioral therapy; acupuncture
- Pharmacologic approaches (eg, hypnotics, anti-depressants, melatonin)

If patient complaints include an aggravation of SB in relation to some medication, attention should be given to this possible interaction. Despite no absolute evidence, changing the medication may sometimes improve SB.

It is noteworthy that, although rare, SB, OSA, GERD, and insomnia can be found together in some patients. Use of medication for depression or a neurologic disorder may exacerbate the condition, and, likewise, opioids may increase the risk of central sleep apnea.<sup>18,19</sup> It is therefore wise to review the medication list, as well as implement other individually oriented approaches.

## Conclusions

A clinician treating an SB patient with comorbidities must adopt a transdisciplinary approach. Lonesome management on the part of a physician or dentist is not the current standard of care. Management of the above concomitant conditions may constitute an example of interprofessional collaboration for the well-being of patients. The proposed operational clinical approach for advancement in SB diagnosis and management is obviously a work in progress that needs further validation in a collegial collaboration.

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## References

1. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med* 2003; 14:30–46.
2. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil* 2018;45:837–844.
3. Mayer P, Heinzer R, Lavigne G. Sleep bruxism in respiratory medicine practice. *Chest* 2016;149:262–271.
4. Lavigne G, Palla S. Transient morning headache: Recognizing the role of sleep bruxism and sleep-disordered breathing. *J Am Dent Assoc* 2010;141:297–299.
5. Maluly M, Andersen ML, Dal-Fabbro C, et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. *J Dent Res* 2013;92(7 suppl):s97–s103.
6. Ohmure H, Takada H, Nagayama K, Sakiyama T, Tsubouchi H, Miyawaki S. Mastication suppresses initial gastric emptying by modulating gastric activity. *J Dent Res* 2012;91:293–298.
7. Manfredini D, Serra-Negra J, Carboncini F, Lobbezoo F. Current concepts of bruxism. *Int J Prosthodont* 2017;30:437–438.
8. Manfredini D, Ahlberg J, Winocur E, Lobbezoo F. Management of sleep bruxism in adults: A qualitative systematic literature review. *J Oral Rehabil* 2015;42:862–874.
9. Huynh N, Lavigne GJ, Lanfranchi P, Montplaisir JY, de Champlain J. The effect of 2 sympatholytic medications—propranolol and clonidine—on sleep bruxism: Experimental randomized controlled trials. *Sleep* 2006;29:307–316.
10. Sakai T, Kato T, Yoshizawa S, et al. Effect of clonazepam and clonidine on primary sleep bruxism: A double-blind, crossover, placebo-controlled trial. *J Sleep Res* 2017;26:73–83.
11. Shim YJ, Lee MK, Kato T, Park HU, Heo K, Kim ST. Effects of botulinum toxin on jaw motor events during sleep in sleep bruxism patients: A polysomnographic evaluation. *J Clin Sleep Med* 2014;10:291–298.
12. Johns MW. A new method of measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep* 1991;14:540–545.
13. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: A tool to screen patients for obstructive sleep apnea. *Anesthesiology* 2008;108:812–821.
14. Marti-Soler H, Hirotsu C, Marques-Vidal P, et al. The NoSAS score for screening of sleep-disordered breathing: A derivation and validation study. *Lancet Respir Med* 2016;4:742–748.
15. Morin CM, Belleville G, Bélanger L, Ivers H. The Insomnia Severity Index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep* 2011;34:601–608.
16. Gagnon Y, Mayer P, Morisson F, Rompré, PH, Lavigne GJ. Aggravation of respiratory disturbances by the use of an occlusal splint in apneic patients: A pilot study. *Int J Prosthodont* 2004;17:447–453.
17. Nikolopoulou M, Ahlberg J, Visscher CM, Hamburger HL, Naeije M, Lobbezoo F. Effects of occlusal stabilization splints on obstructive sleep apnea: A randomized controlled trial. *J Orofac Pain* 2013;27:199–205.
18. Marshansky S, Mayer P, Rizzo D, Baltzan M, Denis R, Lavigne GJ. Sleep, chronic pain, and opioid risk for apnea. *Prog Neuropsychopharmacol Bio Psychiatry* 2018;87:234–244.
19. Winocur E, Gavish A, Voikovitch M, Emodi-Perlman A, Eli I. Drugs and bruxism: A critical review. *J Orofac Pain* 2003; 17:99–111.