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Is there an association between sleep bruxism and obstructive sleep apnea syndrome? A systematic review

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Abstract

Purpose To elucidate current knowledge on the potential association and causality between sleep bruxism (SB) and obstructive sleep apnea (OSA) using full-night polysomnography.

Methods Search strategies were developed for PubMed, Web of Science, Cochrane, LILACS, MEDLINE, and BBO-ODO and conducted until May 2019. The methodological quality was evaluated using the Qu-ATEBS tool.

Results Two hundred seventy articles were identified and after independent screening of abstracts by two authors, 17 articles underwent full-text reading. Ten articles were excluded for not meeting the inclusion criteria and 7 were included in qualitative synthesis. Four studies support the association between SB and OSA: (a) a subtype of OSA patients may have SB as a protective response to respiratory events, (b) most episodes of bruxism occur shortly after the end of apnea/hypopnea (AH) events, (c) bruxism episodes occur secondary to arousals arising from AH events, and (d) there is a correlation between the frequency of SB and AH events, and three studies did not support: (e) AH episodes are related to non-specific SB oromotor activities, (f) SB episodes are not directly associated with the end of AH events, and (g) patients with OSA did not experience more SB events than control group.

Conclusion There is no scientific evidence to support a conclusive relationship between SB and OSA. Further, well-designed and randomized studies with control groups are needed to investigate whether possible mechanisms common to SB and OSA exist and whether OSA treatment could improve SB negative oral health outcomes in patients with SB and comorbidity of OSA.

Keywords Bruxism · Obstructive sleep apnea · Sleep bruxism · Sleep-disordered breathing · Systematic review

Introduction

Sleep bruxism (SB) is a masticatory muscle activity characterized by repetitive clenching or grinding of the teeth and/or by bracing or thrusting of the mandible, usually associated with sleep arousal [1–3]. Despite being a sign of a disorder in some individuals, in healthy individuals, SB should be rather considered as a motor behavior with multifactorial etiology that can be a risk factor for negative health outcomes such as tooth wear, temporomandibular disorders, headaches, fatigue or pain in the masticatory muscles, and poor quality of sleep [4–7].

Like SB, obstructive sleep apnea (OSA) is a sleep-related condition and, thus, share a common physiologic pathway with SB. OSA is a respiratory disorder characterized by total (apnea) or partial (hypopnea) airway obstruction leading to arousals in response to respiratory effort [8–10]. It is important to notice that arousal is a period of sleep instability characterized by an abrupt

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variation of brain activity frequency without re-consciousness, following a sleep disruption [11–14]. That syndrome affects about 8 to 9% of the general adult population [15] and is associated with many comorbidities that turn it a public health problem [16, 17].

Concerning the methodology of diagnosis, the polysomnography (PSG) allows the quantitative recording of rhythmic masticatory muscular activity (RMMA), respiratory events, and many sleep parameters, and is therefore recommended to confirm and complement the diagnosis of both SB and OSA. Full-night PSG at a sleep clinic represents the gold standard of OSA diagnosis. The definitive diagnosis of SB should meet the following criteria: personal report, clinical signs and symptoms, and positive polysomnography findings [2, 8, 18, 19]. Although the absence of a diagnostic standard in studies compromised the analysis of the SB prevalence [20, 21], it is observed a decrease in the incidence with age, ranging from 2 to 30% in children [22–24] and 5 to 10% in adults [24–26] with no gender differences.

Considering that SB may be a motor reflex of the central nervous system in response to arousal and that OSA leads to sleep arousal, several studies have demonstrated a significant association or comorbidity between SB and OSA [24, 25, 27–29]. Besides the occurrence of masticatory muscle contraction after respiratory episodes associated with arousal may play a role in the reopening of compromised upper airways, thus bruxism may be a protective factor whereas is associated with a positive health outcome [4, 30–32]. Two reviews were performed on the same topic, Canto et al. [5] and Jokubauskas and Baltrušaitytė [33] concluded that there is not enough conclusive scientific evidence to define a clear link between OSA and SB. However, in our study, the Apnea–Hypopnea Index and the positive diagnosis of SB were not defined as eligibility criteria like in Canto et al. and Jokubauskas and Baltrušaitytė reviews, respectively. Thereby, we aim to reach data from more studies leading to more conclusive answers.

Regarding the high prevalence of SB in daily dental practice and the serious consequences of OSA, the aim of this study is to elucidate the potential association between these two. The clinical question in PECOS design was: "Is there an association between Sleep Bruxism and Obstructive Sleep Apnea in patients previously diagnosed with OSA via PSG?"

Materials and methods

Search strategy

This review was conducted according to the PRISMA STATEMENT (www.prismastatement.org) and registered in PROSPERO (CRD42016043324).

The PECOS used to guide the development of this review and the search in the literature was: population (P) = patients diagnosed with OSA; exposure (E) = severity of OSA and SB; comparator (C) = OSA × healthy; outcome (O) = presence of association between apnea/hypopnea events and bruxism events. Study design (S) = cross-sectional studies or cohort studies published in scientific journals.

Advanced searches were conducted in detailed and individualized strategies in the five databases: PubMed, The Cochrane Library, LILACS, MEDLINE, and BBO-ODO. An appropriate "MeSH terms" combination (Medical Subjects Headings) (www.nlm.nih.gov/mesh/meshhome.html) was used, initially, in the PubMed database and adapted for each of the databases. The references cited in the selected articles were screened and the gray literature search was taken using Google Scholar.

Eligibility criteria

The following inclusion criteria were employed for this systematic review: (1) studies with adults (over 18 years) of both genders, with no year or language restrictions, and published until May 2019; (2) observational studies with the clear purpose to assess the relationship between SB and OSA using full-night ambulatory PSG; (3) full-night ambulatory PSG as the "gold standard" to diagnose OSA; (4) SB events confirmed by full-night PSG recordings.

The following were the exclusion criteria: (1) those that were not research articles, such as reviews, meta-analysis, book chapters, personal opinions, and case report and (2) studies on patients with genetic syndromes or craniofacial anomalies.

Data extraction

The studies selection was completed in two phases. In the first phase, the title and abstract of all identified electronic database articles were independently reviewed by two reviewers (AJCL and MCMM); studies that did not fulfill the inclusion criteria were excluded. In phase 2, the articles were independently evaluated in full text by the same two reviewers. Disagreements at any stage were resolved by the third reviewer (TCAC).

The following data were independently collected from the articles by two reviewers (AJCL and MCMM): title, author, year of publication, sample size, inclusion criteria, method of SB assessment, and results.

Quality assessment

The methodological quality of selected studies was evaluated using the seven-item quality-assessment tool for experimental bruxism studies (Qu-ATEBS) [34]. Seven items are phrased as questions and rated on a five-point Likert scale. The

maximum attainable score was 70 points; a score between 0 and 50 was considered low quality and a score between 51 and 70 was considered high quality. The same two reviewers (AJCL and MCMM) independently scored each item and disagreements between the two were resolved by a third author (TCAC).

Confidence in cumulative evidence

A summary of the overall strength of evidence available was performed using "Grading of Recommendations Assessment, Development and Evaluation" (GRADE). A Summary of findings table was produced via GRADEpro software.

Results

Study selection

In phase one of the study selection, 270 articles were identified across the six electronic databases (162, PubMed; 4, LILACS; 78, MEDLINE; 1, BBO-ODO; 25, COCHRANE; and 0, Google Scholar). After removal of duplicates, 185 articles were analyzed. After the evaluation of titles and abstracts, 168 articles were excluded because they were not relevant to the subject of the study and 17 studies were selected for full-text reading. The references cited in these pre-selected articles were screened but any new article was included. Of the 17 pre-selected studies, 10 were excluded because they did not meet the inclusion criteria and 7 were included and selected for detailed analysis; assessing of the methodological quality was through the Qu-ATEBS form and data extraction. A flow diagram of the process of identification, selection, exclusion, and inclusion of studies is shown in Fig. 1.

Quality assessment

After the methodological evaluation of the articles through Qu-ATEBS [34], two articles reported low methodological quality (scores below 51 points). The methodological limitations of the included studies are mainly related to poor reporting or inadequate eligibility criteria, the absence of a control group and statistical methods and data considered inadequate or insufficiently described (Table 1).

Synthesis of results of included studies

Of the 270 articles extracted from the databases, 7 met the inclusion criteria and were screened for the risk of bias. The assessment of the methodological quality of the included articles are described in Table 1 and the data extracted from them in Table 2.

Philipps et al. [35] assessed the association between sleep apnea and SB activity in patients with apnea ($N = 14$; 2 females and 12 males; mean age 50.2 ± 16.4 years; mean weight (lbs) 250 ± 56.8) and in a control group ($N = 10$; 1 female and 9 males 1/9 F/M; mean age 52 ± 15.9 years; mean weight (lbs) 206.1 ± 29.7). Their findings suggest that there is a positive association between sleep apnea and SB activity by linear regression ($R = 0.49$ and $p < 0.05$). This relationship is probably due to the frequent arousals resulting from apnea–hypopnea events, which are responsible for an increase in SB activity.

Okeson et al. [36] investigated the occurrence of bruxism episodes in patients with ($N = 12$; 12 M; mean age 57 ± 11.5 years) and without ($N = 12$; 12 M, mean age 57 ± 11.7 years) sleep apnea. The authors hypothesized that sleep apnea patients experience more bruxism events than a non-apnea control group considering that bruxism events are secondary to arousals resulting from breathing complications. It was observed that although there was a strong association between bruxism and arousal events in both groups, t test did not reveal statistical difference regarding the number of bruxism events between the two groups (16.1 events per night of sleep for the OSA group and 26.2 for the control group; $p < 0.05$).

Sjoholm et al. [37] performed a study to test the hypothesis of a direct association between sleep-disordered breathing and bruxism. It was assessed the association between SB episodes and the end of apnea–hypopnea events in patients with sleep apnea ($N = 21$). The patients were divided into 2 groups, the mild OSA group (AHI < 15; $N = 11$; 1/10 F/M; mean age 39.4 ± 9.8 years; mean body mass index 29.1 ± 5.04 kg/m^2) and the moderate OSA group (AHI > 15; $N = 10$; 1/9 F/M; mean age 40.7 ± 8.7 years; mean BMI 30.6 ± 5.1 kg/m^2). The results show that only 3.5% of SB events in the moderate OSA group and 14.4% in the mild OSA group ($p < 0.05$) of SB events are directly associated with the AH events. Hence, the SB events appear to be more related to the disturbed and instable sleep pattern of OSA patients.

Hosoya et al. [28] carried out a study to assess the association between respiratory events and SB in patients with OSA ($N = 67$; 18/40 F/M; mean age 54.3 ± 13.2 years; mean BMI 28.1 ± 6.7 kg/m^2) and in healthy patients ($N = 16$; 8/8 F/M; mean age 23.9 ± 5.5 years; mean BMI 19.7 ± 5.1 kg/m^2). According to the authors, the sleep apnea group presents a higher risk of SB when compared to the control group (R = 3.96, 95% confidence interval, 1.03–15.20; $p < 0.05$) and SB episodes, particularly those of the phasic type, have a strong association with events of apnea/hypopnea ($R = 0.35$; $p < 0.01$). Besides, the findings suggest that episodes of bruxism in patients with OSA are secondary to arousals consequent on AH events (4.6 ± 10.5 SB events per hour of sleep during arousals ensued from an AH event versus 1.6 ± 1.5 SB

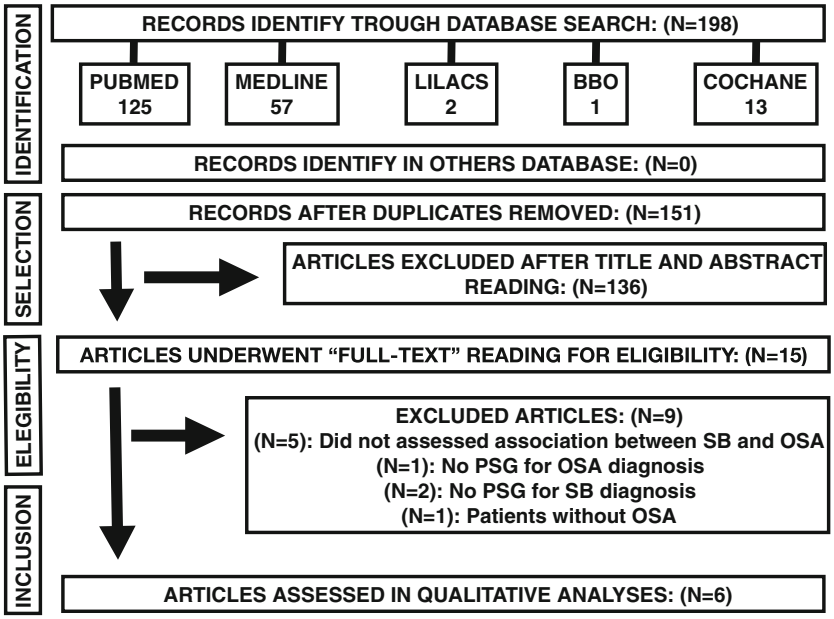

Fig. 1 Procedural flow of the literature search and selection process

events per hour during arousals that were not ensued from an AH event; $p < 0.01$).

Similarly, Saito et al. [38] selected 10 patients (10 males; mean age 46.7 ± 11.5 years; mean BMI 27.7 ± 3.9 kg/m^2) with OSA and SB in order to investigate the temporal association between respiratory events of apnea–hypopnea and rhythmic masticatory muscular activity, considering a 5-min time window. The authors report that most episodes of bruxism (55%; $p < 0.05$) occur soon after AH events (mean of 33.4 s; $p < 0.05$), constituting, thus, a secondary form of SB.

In a later study with a larger sample size ($N = 59$; 12/47 F/M; mean age 44.8 ± 10.8 years; mean BMI 25.8 ± 4.2 kg/m^2), Saito et al. [39] evaluated the association between respiratory apnea–hypopnea (AH) events and masseter contraction events in patients with suspected OSA and SB. A correlation coefficient analysis was performed between dependent and independent variables ($p < 0.05$), so it was observed that sleep arousals in patients with concomitant OSA and SB are not strongly associated with the onset of SB episodes ($p = 0.042$). In addition, authors suggested that AH events were related to a higher occurrence of sleep oromotor activity not specific to SB ($R = 0.37$; $p = 0.004$) that may be due to oral dryness-mouth breathing.

Tan et al. [40] selected 147 OSA patients (47/100 F/M; mean age 44.6 ± 12.8 years) to determine the prevalence of SB, to assess the association between SB and OSA and to investigate OSA risk factors for SB. Of the 147 OSA patients, 49 were diagnosed with SB and these patients demonstrated a higher respiratory-related arousal index (median = 44.42, IQR 23.86 to 53.37, $p = 0.01$) and oxygen desaturation index (median = 32.50, IQR 16.00 to 48.20, $p = 0.005$) compared to the non-SB group. These results suggest that SB may be a protective response to respiratory-related events in a specific subtype of OSA patients. Besides, spontaneous arousal index SAI (OR = 0.89, 95% CI = 0.80 to 0.96) was associated with the odds of experiencing SB.

GRADE summary

The quality of evidence assessment (GRADE) was considered very low in all outcomes. Wide confidence intervals, high methodological heterogeneity, and study design (observational studies) were the explanations for very low quality of evidence (Table 3).

Discussion

This systematic review investigated the potential association between SB and OSA based on articles which suggest a causal relationship between these two sleep conditions [24, 25, 27–29]. Included in this review were studies whose main aim was to assess the association between OSA and SB via full-night polysomnography in the sleep laboratory. If this association was confirmed, SB would become a clinical predictor for OSA, this would lead the dentist to refer his patient for a general sleep quality investigation in an attempt to confirm the presence of secondary bruxism. In this context, the treatment of OSA would improve the clinical consequences of SB.

One of the possible scenarios for the temporal association between OSA and SB would be that the onset of OSA events precedes the onset of SB event. This hypothesis considers that events of apnea–hypopnea lead to respiratory efforts causing the arousal which induces SB events. Arousals trigger a

Table. 1 Risk of bias

Quality Assessment Tool for Experimental Bruxism Study (Qu-ATEBS) Scores *	Phillips et al. [35]	Okeson et al. [36]	Sjoholm et al. [37]	Hosoya et al. [28]	Saito et al. [38]	Saito et al. [39]	Tan et al. [40]
1-Quality of reporting: Were the studies' aims or hypothesis clearly described?	5	5	5	5	4	U5	44
Quality of design: Were the aims or hypothesis based on relevant theory?	4	4	5	5	5	55	5
2- Quality of reporting: Were the eligibility criteria, used to select participants, sufficiently described?	N/A	N/A	5	1	5	55	5
Quality of design: Were the eligibility criteria appropriate for the objectives of this study?	N/A	N/A	5	5	5	55	55
3- Quality of reporting: Was it clearly described whether a control group, control condition, or an experimental condition was used?	2	5	N/A	5	N/A	N/A	N/A
Quality of design: Were the control group, control condition, or experimental condition appropriate for this study?	2	5	N/A	2	N/A	N/A	N/A
4- Quality of reporting: Was the study design described in sufficient detail to permit replication?	4	4	3	5	5	54	55
Quality of design: Was the study design appropriately selected for the objectives of this study?	3	5	5	5	5	55	44
5- Quality of reporting: Was the experimental bruxism task described in such detail that replication is possible?	4	4	3	5	5	55	55
Quality of design: Was the experimental bruxism task appropriately selected for the objectives of this study?	4	5	4	5	4	55	55
6- Quality of reporting: Were statistical methods and data sufficiently described?	1	2	3	5	5	55	55
Quality of design: Were statistical methods and data appropriate for the objectives of this study?	1	2	4	5	5	45	55
7- Quality of reporting: Were the study's conclusions appropriately formulated?	3	44	4	5	4	44	44
Quality of design: Were aims and hypothesis clearly addressed in the conclusions and relevant to the objectives?	4	5	5	5	5	55	55
Total	37	50	51	60	57	58	57

cascade of physiological events, and at its end, there is an increase in the muscular activity of the mandibular depressors and the subsequent RMMA resulting in mandibular protrusion and opening of the airways [3, 41–43]. It is suggested, therefore, that SB plays a protective role against OSA. Some studies support the hypothesis described above in conclusion that, in patients with OSA, most bruxism events are secondary to apnea–hypopnea, with OSA being a risk factor for SB. In this case, bruxism would be a secondary manifestation [28]. However, this finding is not conclusive since not all episodes of SB have occurred in this sequence or even have a temporal association with respiratory events [38]. If the theory of bruxism secondary to OSA is confirmed, the improvement of respiratory quality and arousal index may reduce the occurrence of bruxism, a fact that should be the goal of future investigations.

A possible confirmation of the theory that SB is a potential OSA-protective mechanism comes from studies that show a decrease in SB events when using a mandibular protrusion device to reestablish compromised airways, a role that would be naturally played by the suprahyoid and masticatory muscles activity (RMMA). Landry-Schonbeck et al. [44], when considering that OSA may be observed concomitantly with SB, aimed to assess in an experimental study the efficacy of an adjustable mandibular device on SB motor activity. The results of the study revealed that the use of the mandibular advancement device for a short period is associated with a significant reduction of SB, ranging from 39 to 45%. Therefore, further studies should be developed in order to confirm if such devices may be an alternative treatment for patients with concomitant SB and OSA [49–52].

Furthermore, another interesting factor is the relationship between SB and the severity of OSA which could be assessed when comparing OSA individuals with concomitant SB to individuals with only OSA. The individuals with concomitant SB presented higher frequencies of both AH events and oxygen desaturation; hence, higher frequencies of SB are associated with more severe respiration disturbances, assuring the

Table 2 Study characteristics

Authors	Title	Year	Sample size	Inclusion criteria	SB assessment	Results	Conclusion
Phillips et al. [35]	Effect of sleep position on sleep apnea and parafunctional activity	1986	14 + 10 in control group	Patients with OSA	PSG (submental)	There is a correlation between clench index and apnea-hypopnea index ($R = 0.49$; $p < 0.05$)	There is an association between OSA and SB
Okeson et al. [36]	Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects	1991	12 + 12 in control group	Patients with OSA	PSG (submental)	An average of 16.1 bruxism events during the night for OSA group and 26.2 events for the control group	There were no statistically significant differences regarding the number of SB events between OSA and control group
Sjoholm et al. [37]	Sleep bruxism in patients with sleep-disordered breathing	2000	21	Patients with OSA	Reporting of grinding or clenching, clinical evaluation and PSG (masseter)	3.5% of SB events in moderate OSA group and 14.4% in mild OSA group are directly associated with the termination of AH events	SB events are rarely directly associated with the termination of AH events
Hosoya et al. [28]	Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea syndrome	2014	67 + 16 in control group	Patients with OSA	PSG (masseter and submental)	SB event index (events per hour) was higher in OSA group (7.02 ± 10.1) than in control group (2.88 ± 1.47) ($p < 0.05$).	Patients with OSA have a higher risk of SB when compared to healthy individuals
Saito et al. [38]	Temporal association between sleep apnea–hypopnea and sleep bruxism events	2013	10	Patients with OSA and SB	Subjective report, clinical evaluation, and PSG (masseter and submental)	55% of SB episodes occurred soon after (33.4 s; $p < 0.01$) AH events	In patients with concomitant SB and OSA, most SB events occurred after AH events
Saito et al. [39]	Weak association between sleep bruxism and obstructive sleep apnea. A sleep laboratory study.	2015	59	Patients with suspected OSA and SB	Subjective report, clinical evaluation and PSG (masseter and submental)	A low correlation was found between SB events and AH index (R = 0.09 and $p = 0.47$)	AH events did not statistically correlate with SB events
Tan et al. 2018 [40]		2018	147	Patients with OSA over 25 years old	PSG (mentalis and right-left masseter)	SB patients demonstrated a higher RAI (median = 44.42, $p = 0.01$) and ODI (median = 32.50, $p = 0.005$) than non-SB group	SB may be a physiologic response to a respiratory-related event in a subtype of OSA patients

Table 3 GRADE summary of findings

Outcomes	No. of participants (studies)	Certainty of the evidence (GRADE)	Relative effect (95% CI)
Association between OSA and SB (studies case-control)	131 (3 studies)	VERY LOW [a, b, c] ⨁○○○	− 2.94 (− 12.87 to 6.99)
Association between OSA and SB (studies cross-sectional)	235 (4 studies)	VERY LOW [a, b] ⨁○○○	7.33 (3.83 to 10.83)

CI, confidence interval

[a] High risk of bias

[b] High inconsistency (clinical and statistical heterogeneity between studies)

[c] High imprecision

OSA-protective role of SB [28. 30, 31, 53–58]. However, this hypothesis remains controversial, since Sjoholm et al. [37] noticed that SB was more prevalent in the group with mild OSA when compared to the moderate OSA, with bruxism occurring mostly in the transition from delta to lighter stages of sleep. As individuals with moderate OSA have significantly less deep sleep, these differences may be justified by the divergent pattern of sleep structure in patients with more severe OSA and those with milder OSA [49].

The other scenario poorly described in the literature supports the theory that the onset of the bruxism event precedes the onset of OSA event, assuming that SB has an OSA-inducing effect. It is suggested that during the REM sleep phase, there is lubrication of the upper airway mucosa leading to nasal congestion and, consequently, respiratory disturbance. The origin of this process is in the activation of the trigeminal cardiac reflex, through which bruxism events promote a decrease in heart rate, previously increased by arousal [50]. Thus, bruxism would be a phenomenon through which the heart rate is reestablished by the activation of the trigeminal cardiac reflex, which induces nasal congestion. This hypothesis is supported by the use of the antihypertensive clonidine and its effect on the reduction of bruxism episodes [51]. Saito et al. [38] stated that in their study, 25.5% of SB episodes occurred prior to the apnea–hypopnea events ($p < 0.05$), suggesting the possibility that SB events may trigger AH events.

The scenario in which SB and OSA are unrelated phenomena and do not have a temporal causality according to PSG recordings should also be considered since it is noticed in some studies that a significant percentage of bruxism episodes did not have a temporal association with AH events [35, 37, 38]. Besides, several studies confirmed a weak association between SB episodes and respiratory events of AH by correlation analyses or comparison of statistic data, concluding that there is no evidence to suggest that AHI was associated with the odds of experiencing SB. Furthermore, respiratory events were related to a higher occurrence of other sleep oromotor activities and not SB activity, suggesting that SB events appear to be more associated with body movements and isolated arousals in consequence of a disrupted sleep pattern [36, 39, 40].

Likewise, other factors may concurrently induce both disturbances, among which the arousal do not play a fundamental role [35, 36]. In this context, the SB mechanism would be different between apneic patients and healthy patients. It is also important to notice that SB may be associated with other sleep disorders such as restless legs syndrome since 10 to 20% of bruxism patients present such syndrome. This leads to an increase in the frequency of arousal and bruxism acts as a motor reflex in response to such arousals. In addition, both disorders share dopamine pathway dysfunction as its etiology [38, 52].

The criteria used to assess the association between SB and OSA were quite different between the included studies. Thus, it was not possible to statistically match the data from the included articles to reach more conclusive findings on the association. We aimed to assess the possible association of SB and OSA by a measure of association called odds ratio (OR), but many articles did not show enough information to calculate that measure. An attempt to reach that information from the authors was done but we did not succeed in contacting. Therefore, the findings of this study should be carefully analyzed since the discrepancy of the results leads to very low quality of evidence as shown in the GRADE summary of findings (Table 3).

SB and OSA are probably related to a common mechanism. In order to draw a definite conclusion on the strength of the association or on causality between the conditions, more studies should be developed. Future studies should involve a large sample of patients, previously diagnosed with OSA via PSG, organized in subgroups of patients with and without SB. Moreover, a control group of healthy patients should be adopted, preferably matched for age, sex, and body mass index. Studies which intend to assess the temporal association between SB and AH events must include a definition of the time windows in which the events would be considered associated, this specific interval is not clearly standardized in literature. Patients with respiratory resistance syndrome and snoring should also be evaluated in order to better understand the relationship between respiratory events and SB.

The association or causality between sleep bruxism and obstructive sleep apnea remains unclear and despite we cannot prove that sleep apnea is a trigger for sleep bruxism is important to consider them as overlapping comorbidities. The findings of the published studies are quite controversial and inconclusive and, thus, cannot objectively guide us in clinical practice. We strongly suggest the development of more studies that approach both pathologies, seeks mechanisms that can independently induce SB and OSA, and that are associated with the concomitant presence of both conditions.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

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