Sleep Medicine 114 (2024) 1–7

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Is there an association between sleep bruxism and obstructive sleep apnea? A case-control polysomnographic investigation

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ARTICLE INFO

Keywords:
Sleep bruxism
Sleep apnea
Polysomnography
Sleep breathing disorder

ABSTRACT

Objectives: To estimate the statistical and epidemiological association between Sleep bruxism (SB) and Obstructive sleep apnea (OSA) based on OSA severity, and to describe sleep data findings within the analyzed population.

Methods: A case-control study (N = 37) was conducted on subjects with and without OSA. All subjects underwent a full-night polysomnographic recording at the Sleep Unit (Clinical Neurophysiology Department) of San Carlos University Hospital. The diagnosis and severity of OSA were determined using ICSD-3 and AASM-2.6 scoring. The definitive SB diagnosis was obtained through a self-report test, physical examination, and PSG recordings. Variables used to study the association between both conditions included the apnea and hypopnea episodes, the Apnea-hypopnea index (AHI), the number of SB episodes per night, and the bruxism index. Chi^2, correlations, and ANOVA were calculated. The epidemiological association was calculated using the OR.

Results: SB showed an epidemiological association with OSA, with an OR of 0.15 (0.036-0.68), suggesting it could be considered a protective factor ($p < 0.05$). OSA patients presented fewer average SB episodes (6.8 ± 12.31) than non-OSA patients (25.08 ± 31.68). SB episodes correlated negatively ($p < 0.05$) with the AHI and the number of hypopneas ($p < 0.05$). The average number of SB episodes was significantly higher in patients with mild OSA compared to those with severe OSA.

Conclusions: In this sample of patients with subclinical and mild OSA, SB may act as a protective factor. However, confirmation of these results with a larger sample size is necessary.

1. Introduction

The most recent consensus about sleep bruxism (SB) definition describes it as a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals [1]. The basic pattern of SB consists of rhythmic activity of the masticatory muscles (RMMA) and is attributed to changes in the autonomic nervous system, increasing heart rate [2,3]. RMMA is normal in sleep and is present in 60% of the adult population [4,5]. Nonetheless, other motor events such as masticatory muscular activity (MMA) are also associated with SB but are not fully explained by these mechanisms. SB can also occur alongside other sleep disorders like obstructive sleep apnea (OSA) [6–11].

OSA is a sleep disorder that causes upper airway blockage, snoring [12], and other complications like hypertension, arrhythmias, and cardiovascular disease [13,14]. OSA affects 17% of females and 34% of males and is more common in males over 50 and those who are obese [14–16]. Authors suggest SB often occurs with OSA near apnea-hypopnea (AH) events [17–20]. Moreover, the causal relationship between SB and OSA is unclear, with different possible theories, such as OSA-inducing SB, SB-inducing OSA, or other overlapping factors causing both conditions [21–23].

Several epidemiological studies have explored the relationship between SB and OSA in different populations. In Singapore, a retrospective study that used PSG (polysomnography) found a 33.3% association

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https://doi.org/10.1016/j.sleep.2023.12.006
Received 29 May 2023; Received in revised form 30 November 2023; Accepted 10 December 2023
Available online 16 December 2023

between SB and OSA [24]. Another study conducted in Brazil found that 7.6% of the general population had SB [25]. In Europe, an epidemiological study used telephone questionnaires to establish a relationship between SB and OSA, although the data are inconclusive [26]. Ohayon's study showed that snoring-SB had an odds ratio (OR) of 1.2 and OSA-SB had an OR of 1.8, but this epidemiological association was calculated based on data obtained through self-referred tests and roommate reports [27].

Studies show that SB episodes are linked with short-term awakenings (EEG arousals) and an increase in RMMA [28,29]. SB patients exhibit an increase in sympathetic activity and a decrease in parasympathetic activity 3 min prior to the onset of RMMA [30], which occurs more frequently and prominently during the transition from deep non-rapid eye movement (NREM) to rapid eye movement (REM) sleep [31,32]. This may be influenced by sympathetic-parasympathetic fluctuation during sleep [23].

Manfredini et al. suggested that SB activity may protect against OSA by temporarily advancing the airway through the mandible [33]. Altogether, the connection between SB and OSA is still not entirely clear. This study aims to explore the possible relationship between SB and OSA, excluding increased muscle tone after an apnea episode, part of the American Academy of Sleep Medicine's (AASM) definition of arousal [34].

2. Material and methods

2.1. Sample selection

The participants of the study are adult patients attended by the Sleep Unit (Clinical Neurophysiology Department) of San Carlos University Hospital, all of them snorers and with clinical suspicion of OSA, who underwent an earlier screening according to the suspicion of SB and OSA, using self-referred tests and physical examination. In parallel, screening for OSA was conducted through anamnesis and clinical examination.

Other sleep disorders were assessed in the initial anamnesis by a sleep expert, using specific scales (Insomnia Severity Index, ISI; International Restless Legs Scale, IRLS; Pittsburgh Sleep Quality Index, PSQI) when necessary, according to the standard protocol of the Sleep Unit. All patients also completed the Epworth Sleepiness Scale and the Zung anxiety/depression test [35,36].

Exclusion criteria were major neurological disorders, psychiatric disorders, other sleep disorders, psychoactive medication use, or being edentulous. Bruxism was assessed by an expert in orofacial pain using the self-referred modified Paseani Test [37]. The clinical examination (tooth wear, masticatory muscle myalgia, TMJ arthralgia, hard tissue, soft tissue, and masseter and/or temporal hypertrophy) was performed according to Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) and American Academy of Orofacial Pain (AAOP) criteria and conducted by a dentist with ability in orofacial pain [5,38].

Finally, all PSG recordings were manually reviewed and scored by a sleep expert (clinical neurophysiologist) with extensive expertise in sleep disorders and SB. As a result, a sample of 37 subjects was achieved, including 24 men (65%) and 13 women (35%). A case-control design was used, where 43,2% (n = 16) of the patients presented SB and 52% (n = 21) of the total sample were OSA patients. Controls were classified as non-SB (n = 21) and non-OSA (n = 16). The sample of OSA patients was segmented by degree of severity in two groups (no OSA or mild OSA, and moderate or severe OSA). The study was approved by the local ethics committee.

2.2. PSG recordings

The full-night monitoring recordings in the Sleep Laboratory (minimum of 8 h in bed) were performed using a Deltamed Coherence 5.0 system. PSG recordings were made according to the AASM recommendations [34], including six electroencephalographic (EEG) derivations; right and left electrooculogram (EOG); submental, masseter, and leg electromyography (EMG); nasal cannula/pressure and oronasal thermal flow; thoracic and abdominal respiratory effort bands; snoring; body position sensor; pulse oximetry; and audio and video recordings.

In addition, a flex disposable sensor was placed in the suprasternal fossa to measure upper airway resistance (UARS) and detect respiratory effort-related arousals (RERAS). Impedance values were checked and adjusted (< 5 Ω), and standard calibrations were performed. All PSG recordings were manually reviewed according to international criteria [34]. In the SB group, the diagnosis was confirmed by PSG performed by a sleep expert, following blinding masking concerning the clinical examination.

2.2.1. PSG sleep bruxism analysis

SB events are estimated through rhythmic (RMMA), and non-rhythmic (AMM) masticatory muscle activity recorded with EMG on the masseter muscles (surface electrodes). Published criteria for SB episodes in PSG were followed [34]. For the calculation of dichotomous variables, the presence of >2 RMMA-MMA/SB episodes/h was considered. The type of SB event is determined as phasic event (three or more EMG bursts, at least 0.25 s and up to 2.0 s), tonic event (at least one EMG burst >2.0 s) as shown in Fig. 1, and mixed event (both types) for the calculation of quantitative variables [39–41].

EMG events immediately following a respiratory event were excluded as depicted in Fig. 2, as well as Sleep-related oromotor activity (OMA), corresponding with snoring, lip sucking, and swallowing, different from RMMA-MMA/SB to avoid possible confounding bias as shown in Fig. 3. RERAS preceding any EMG activity were discarded in all patients by using the UARS sensor and the thoracic abdominal respiratory effort bands.

Due to the difficulty of distinguishing EMG activity immediately after a respiratory event from SB and the lack of standard parameters of such activity, the following criteria were applied: 1) If the EMG event immediately after an AH event was accompanied by arousal, it was excluded; 2) If the EMG event immediately after an AH event was not accompanied by arousal, it was accepted; 3) If the EMG event was not immediately preceded by AH event (window >5 s) it was accepted. All isolated SB events, independent of respiratory events, were accepted according to EMG criteria, regardless of whether accompanied by arousals (Fig. 4).

2.3. Statistical analysis

Continuous variables as means ± SD, ANOVA, and corresponding non-parametric tests were applied when necessary. Pearson's Correlation test was used for comparison of the variables: Apnea Hypopnea Index (AHI), SB Index, number of apnea events, number of hypopnea events, and number of SB events. In addition, the sample was segmented according to the degree of severity of OSA and according to the types of SB episodes. Finally, the epidemiological association was calculated using the Odds Ratio (OR). All calculations were performed with the SPSS v24.0 statistical package (SPSS Inc., Chicago, IL), and values equal to or less than 0.05 were considered statistically significant. Epidat ver.3.1 epidemiological software was used to calculate the OR.

3. Results

The average age of the participants was 49.63 ± 11.59 years Table 1. The patients were on average overweight, had an acceptable average sleep efficiency, and the percentage of time spent in the rates of the different sleep stages was within normal standards.

The number of SB episodes correlates negatively and significantly ($p < 0.05$) with the AHI and with the number of hypopnea events, as illustrated in Supplementary Figs. 1 and 2.

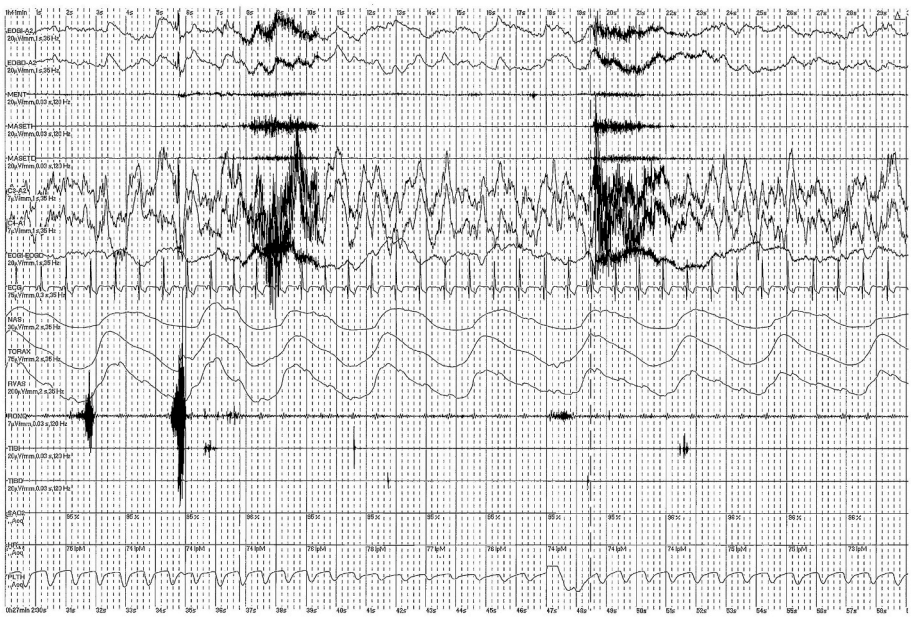

Fig. 1. Epoch (30 s), showed a sleep Bruxism (SB) tonic episode in a Bruxism patient, recorder by Polysomnography (PSG).

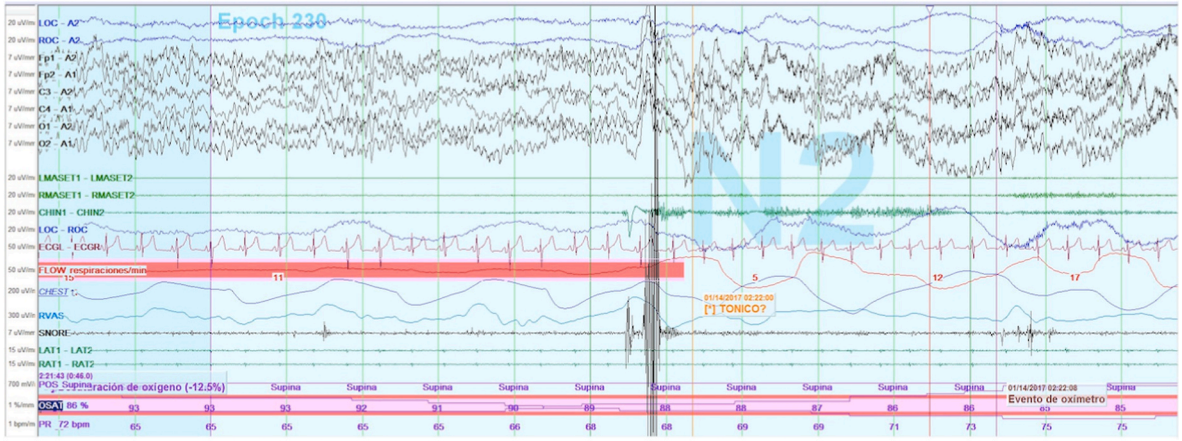

Fig. 2. Epoch (30 s). OSA episode preceding a tonic EMG event accompanied by arousal, which is considered excluded as SB episode.

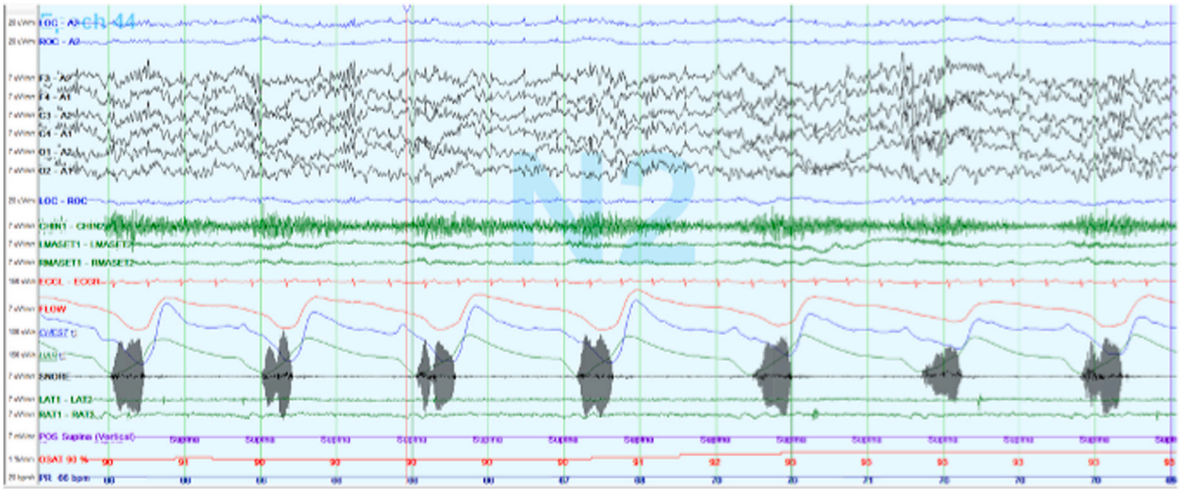

Fig. 3. Epoch (30 s). Tonic EMG episode, corresponding to OMA, following snoring. It is excluded as SB episode.

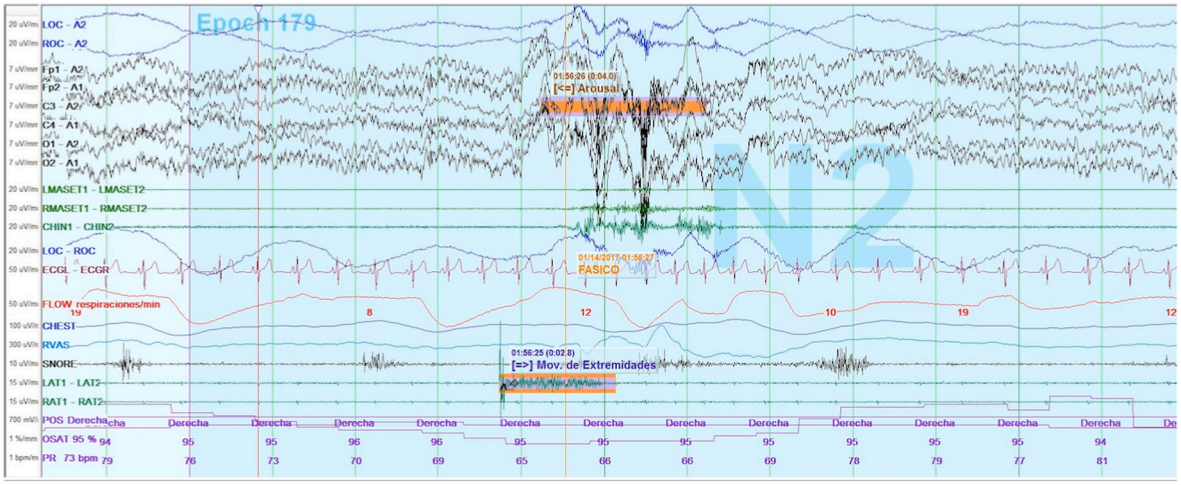

Fig. 4. Epoch (30 s). Phasic EMG episode, accompanied by arousal and myoclonus in the tibial muscles: periodic leg movement, which is considered as SB episode.

Table 1
Descriptive sleep data of the sample
SPT sleep period time, TST total sleep time, SLT sleep latency time, WASO wake time after sleep onset, CT90 total time lower 90% O2Sat.

	(n = 37)
	Mean ± SD
Physical data	
Age	49.63 ± 11.59
BMI	28.67 ± 5.13
Sleep data	
SPT (min)	422.11 ± 33.75
TST (min)	338.27 ± 56.99
SLT (min)	13.76 ± 21.44
Sleep Efficiency (%)	80.48 ± 13.02
WASO (min)	57.80 ± 13.02
Awakes (number)	45.05 ± 22.89
Sleep stage distribution	
N1/SPT (%)	23.53 ± 1 4.84
N2/SPT (%)	41.87 ± 9.50
N3/SPT (%)	17.72 ± 12.21
R/SPT (%)	16.69 ± 6.20
Pulsioximetry data	
Mean (%)	93.37 ± 2.63
Max (%)	98.37 ± 0.89
Min (%)	80.94 ± 10.09
CT90 (%)	11.18 ± 19.62

Table 2
Sleep bruxism data with the segmented sample.

	OSA ± SD (N = 25)	Non-OSA ± SD (N = 12)	Non OSA-mild OSA ± SD (N = 23)	Moderate-Severe OSA ± SD (N = 14)
Sleep Bruxism episodes				
Total episodes	6.8 ± 12.31 *	25.08 ± 31.68 *	18.13 ± 25.83 *	3.86 ± 8.46 *
Phasic episodes	1 ± 2.10 *	6.3 ± 8.3 *	4 ± 6.64 *	0.64 ± 1.49 *
Tonic episodes	4.88 ± 8.71 *	14.50 ± 17.53 *	11.26 ± 14.9 *	2.64 ± 5.41 *
Mixed episodes	0.92 ± 2.01	4.42 ± 10.32	2.96 ± 7.64	0.57 ± 1.65
Episodes/ hour	1.28 ± 2.58	2.12 ± 3.66	2.91 ± 4.18 *	0.82 ± 2.13 *

Total SB events along the TST total sleep time, excluded the Sleep-related Oromotor Activity (OMA) with sample segmented (OSA and non-OSA, and non-OSA-mild OSA, Moderare-Severe OSA). The significance level was set as *p < 0.05.

The mean number of SB episodes was higher (25.08 ± 31.68) in non-OSA patients compared to those with OSA (6.8 ± 12.31). When segmenting the sample by OSA severity into two groups (no OSA or mild OSA, and moderate or severe OSA), the mean number of SB episodes was higher (18.13 ± 25.83) in the no OSA-mild OSA group compared to the moderate-severe OSA group (3.86 ± 8.46), being significant (< 0.05) in all the cases above-described (Table 2).

In the categorization of SB episodes by type (Tonic, Phasic, Mixed), the mean number of tonic episodes predominates (14.50 ± 17.53), being higher in the no OSA-mild OSA group ($p < 0.05$). Statistical significance was observed in all cases, except for mixed episodes in both the unsegmented and segmented samples; and for the SB index, when the sample is not segmented.

When calculating the OR (OSA dependent variable), using two different cut-off points to determine SB: one more lax (>2 ep/h) and the other more severe (>4 ep/h), as reported in the literature [41–43]; SB shows an epidemiological association and it could even be considered as a protective factor against OSA ($p < 0.05$). This association was more pronounced (OR 0.15; 95% CI 0.036–0.68), when the cut-off point for SB index was set at >2 ep/h (Fig. 5).

4. Discussion

There is currently insufficient evidence to confirm an association between SB and OSA [44]. While studies in this field have yielded inconsistent results [11,42,45–50], our findings suggest a possible association between SB and OSA. It should be noted that research on this relationship is limited and has different methodological designs and objectives. The decision to exclude or include the EMG event following the respiratory event, which our study has discarded, could explain the observed heterogeneity in results and potentially lead to an overestimation of SB when such EMG activity is counted.

Sleep architecture is a topic of interest when it comes to patients with SB. Some authors, such as Palinkas et al. and Kim et al., have reported sleep alterations in these patients [51,52]. On the other hand, Kato et al. found that in OSA patients, the highest number of SB events occurred after the AH event, but only a third of these events were associated with arousal [53]. In our sample, the sleep parameters are within normal ranges. This may be because EMG and SB events secondary to apnea were excluded, and fragmentation was not counted as a cause of SB. Arousals, in this case, were attributable to the respiratory event.

Our study found a significant negative correlation between SB episodes and both AHI and hypopnea events. This differs from earlier research by Hosoya et al., which found a positive correlation between SB

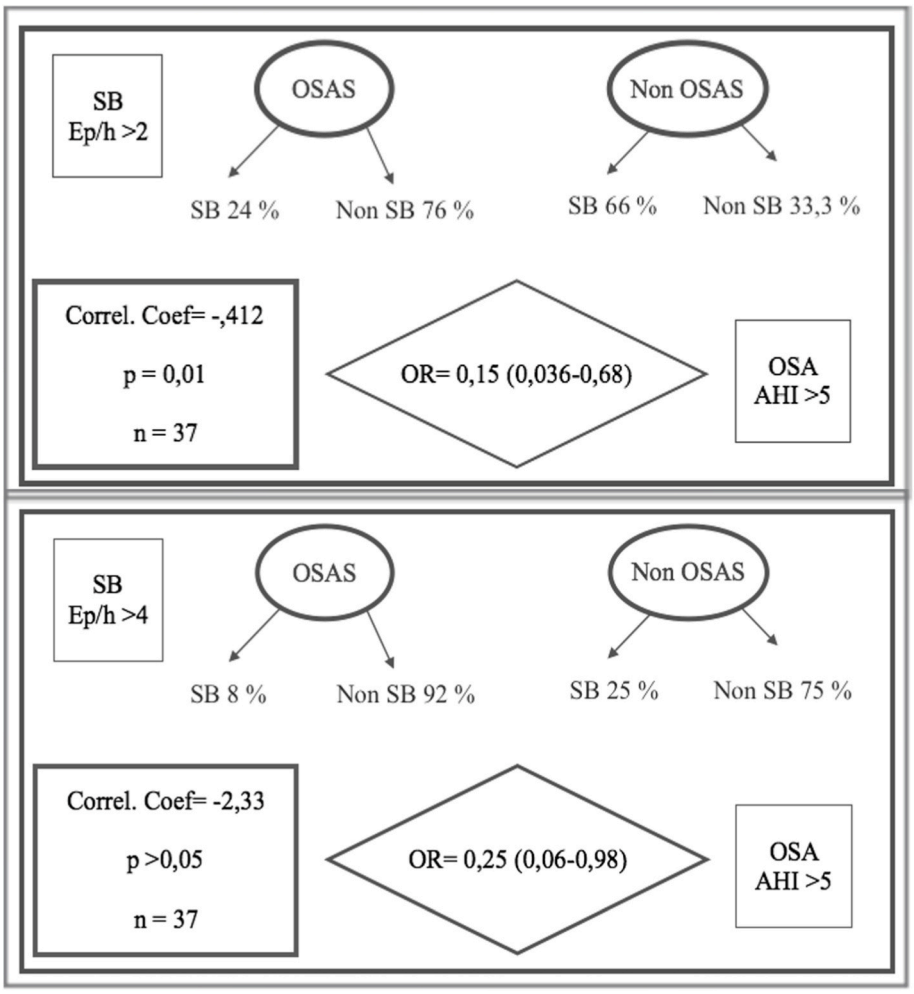

Fig. 5. A Diagram representation of participants distribution based on SB and OSA sleep laboratory diagnosis and OR Odds Ratio Value, with an Ep/h > 2 for the SB criteria and Ep/h > 4 for the SB criteria.

index and AHI [48]. However, when EMG activity following a respiratory event was excluded, both the control and OSA groups showed a higher frequency of SB index. When included, the control group had a lower frequency compared to the OSA group. Our results support this and suggest that SB mechanisms may differ between OSA and non-OSA patients.

In a study conducted by Saito et al., they analyzed the relationship between SB and other sleep-related events [42]. With a small sample and no control group, they found that SB events following AH events were more common and mostly accompanied by arousal. Another study by the same authors found a positive correlation between SB and OMA, RMMA, and OSA, but only significant for OMA versus AHI [49]. In our study, we found that the number of SB episodes correlates negatively with AHI and the number of hypopnea events. This suggests that OMA activity can bias neurophysiological analyses if not excluded. Our study design cannot determine a cause-effect relationship between SB and OSA. Future studies should compare the excluded post-AH EMG events with the included ones to further explore this relationship.

Several authors suggested that the SB following a respiratory event can be an ending of the AH episode, allowing a decrease in SB once OSA is treated [54–58]. Nevertheless, authors such as Okeson et al. have not found differences in terms of SB in OSA versus non-OSA patients [45,46], and Sjöholm et al. points low percentages of SB episodes associated with the end of AH events [47]. Therefore, perhaps, SB events occurring after a respiratory event in OSA patients may simply be a physiological response to restore airflow after an AH event, rather than SB secondary to AH. This approach can help avoid biases in diagnosis and establish a more uniform criterion, considering that we may be overestimating SB in OSA patients based on PSG analysis criteria. It is important to note that individual differences and the type and level of upper airway obstruction should be considered, making it difficult to establish a standard criterion.

In a study conducted by Hosoya et al., it was found that patients with sleep-disordered breathing have a higher risk of SB, and phasic SB episodes suggest that intervening in OSA could prevent the exacerbation of SB [48]. However, in our sample, tonic episodes were more frequent in non-OSA patients, indicating that both phasic and tonic jaw movements could be a protective mechanism in patients with mild apnea, but may not be enough in severe cases. Differences in study results may be due to variations in design and diagnostic methods. Age is a crucial factor in studying the SB-OSA association as SB decreases with age and OSA increases [42,59].

Our findings suggest a negative correlation between SB and OSA. Patients with non-OSA or mild OSA exhibited the highest number of SB episodes. This result is consistent with one of the conclusions from the studies by Lavigne et al. and Macaluso et al., which suggests that primary SB patients do not usually experience abnormal respiratory events during sleep [39,60]. Based on these observations, it is recommended to conduct a screening test to detect possible sleep disorders before treating bruxism.

The clinical implications of our findings suggest that stabilization splints, commonly used to treat SB, can worsen the effects of gravity and

increase the risk of developing OSA in patients with mild or undiagnosed OSA. Patients with these symptoms may benefit from other therapeutic approaches such as a mandibular advancement device (MAD). In a study conducted by Gagnon et al., 10 patients with SB and mild-moderate OSA were fitted with stabilization splints, resulting in a >50% increase in AHI in five of the patients and a >50% increase in snoring in four [61].

Miyawaki et al. found a higher incidence of SB in the supine position so controlling the sleep position can help prevent sleep bruxism [62]. Using a MAD without pushing the jaw forward can be an alternative treatment to the stabilization splint for patients with SB and mild or subclinical OSA, we must consider the device design and the side effects derived from its installation [63].

Authors such as Tan et al. and Tsujisaka et al. describe the possibility that there may be a subtype of young patients with subclinical or mild OSA and phasic SB and that such EMG activity may have a protective role against OSA [50,59]. In cases where medication is needed to treat SB and concurrent OSA, it is important to consider the potential effects of certain drugs (e.g., anxiolytics, benzodiazepines) on OSA [64].

SB and OSA have similar structures that protect during sleep. However, individual differences make it hard to establish a cause-effect relationship. Therefore, we must establish PSG diagnosis basics for SB, conduct high-quality studies, and avoid biases in diagnosis.

The PSG criteria for SB are being questioned [65], despite it being the Gold Standard. Bruxism activity is a continuum, and portable measurement instruments should be improved to record multiple nights in a cost-effective manner, enabling the determination of new correlations and updated cut-off points [66].

Although it is not the aim of this study, collecting data on clinical diagnosis within the design of this type of study (as in our case), would allow correlating neurophysiological findings with clinical signs and symptoms. Using portable respiratory polygraphs that include masseter EMG, as used by Winck et al. [67], and include masseter EMG in sleep units as routine would be useful to study the SB-OSA relationship.

4.1. Limitations

PSG recording in the Sleep Laboratory is limited to one or two nights due to time and economic constraints, which introduces a bias when studying SB. However, PSG is the reference tool for studying the neurophysiological relationship between SB and OSA. Though first-night effects may occur, previous studies report no overall impact on the severity of RMMA frequency in patients with SB [68]. A multicenter study involving another hospital is underway to improve sample size and balance the predominantly OSA population. Our results are inconclusive as to the protective role of SB for OSA, due to the low sample size.

CRediT authorship contribution statement

Rosana Cid-Verdejo: Conceptualization, Formal analysis, Investigation, Methodology, Writing – original draft, Writing – review & editing. **Adelaida A. Domínguez Gordillo:** Conceptualization, Formal analysis, Methodology, Validation. **Fadi Hallal-Peche:** Visualization, Writing – review & editing. **Ignacio Ardizone García:** Conceptualization, Resources, Supervision, Writing – review & editing. **Francisco J. Martínez Orozco:** Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Resources, Supervision, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

We would like to thank the staff of the Clinical Neurophysiology Unit-Sleep Unit of the San Carlos University Hospital (Madrid), as well as Ángela, Paloma, and María, nurses of the sleep unit, for their competent technical assistance.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2023.12.006.

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