Prevalence of Sleep Bruxism and Its Association with Obstructive Sleep Apnea in Adult Patients: A Retrospective Polysomnographic Investigation

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Aims: To determine the prevalence of sleep bruxism (SB) in adult obstructive sleep apnea (OSA) patients, to assess the association between SB and OSA in terms of sleep macrostructure and respiratory parameters, and to determine possible OSA risk factors for SB. **Methods:** Type I polysomnographic data of 147 adult OSA patients (mean age 44.6 ± 12.8 years) were evaluated for SB. SB episodes were scored when masseter rhythmic masticatory muscle activity (RMMA) was twice the background electromyography amplitude, and SB was established when patients had more than four SB episodes per hour of sleep. Demographic characteristics, sleep macrostructure, and respiratory parameters, including respiratory-related arousal index (RAI), spontaneous arousal index (SAI), oxygen desaturation index (ODI), and Apnea-Hypopnea Index (AHI), were analyzed for differences between patients with and without SB using independent samples *t* test and Mann-Whitney *U* test. Multivariate logistic regression analysis was performed to determine the odds of OSA risk factors for SB. **Results:** Approximately one-third (33.3%) of the adult OSA patients had concomitant SB. Most of the RMMA observed in OSA-SB patients was phasic in nature. OSA patients with SB demonstrated significantly greater RAI (P = .001) and ODI (P = .005). RAI (odds ratio =1.05, 95% confidence interval = 1.00 to 1.10) and SAI (odds ratio = 0.89, 95% confidence interval = 0.80 to 0.96) demonstrated marginal effects on the odds of experiencing SB. **Conclusion:** About one-third of adult OSA patients had SB, and these patients demonstrated significantly more respiratory-related arousals and oxygen desaturations. These findings suggest that a phenotypic subtype of OSA patients with predominantly phasic SB exists and allude to a possible protective role of RMMA in respiratory-related arousals.
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Obstructive sleep apnea (OSA) and sleep bruxism (SB) are sleep-related disorders that may be concomitant in an individual. OSA is a sleep-related breathing disorder characterized by repeated partial or total airway obstructions that lead to airflow reduction.[1] This condition affects 9% to 38% of the general adult population,[2] and its prevalence rises with increasing age groups—from 30% at 50 years to as high as 84% above 70 years of age.[3] OSA has been associated with numerous medical comorbidities, such as hypertension,[4] diabetes mellitus,[5] neurocognitive impairment,[6] cardiovascular diseases,[7] and mortality.[8] In addition to increasing age, other major risk factors identified for OSA in the general population include obesity and male gender.[3,9,10] Craniofacial risk factors such as mandibular retrognathia, macroglossia, and high arch palate are thought to contribute to a narrower upper respiratory airway and are more notable in Asian populations compared to Caucasian populations.[11]

Bruxism is a repetitive jaw muscle activity that manifests as clenching or grinding of the teeth and/or bracing or thrusting of the mandible.[1] It is present in 5.5% to 8% of the adult population[12–14] and occurs in two distinct forms: awake bruxism (ie, occurs in the state of wakefulness) and sleep bruxism (SB).[15] SB may also be further classified as primary (idiopathic form) or secondary to medical comorbidities.[16,17] SB is

considered as a sleep movement disorder when detected over a sleep-recording threshold with concomitant neurologic or sleep breathing conditions. The effects of SB on the stomatognathic system may present as nonphysiologic tooth wear, temporomandibular disorders (TMD), and/or pain and headaches in the orofacial region.[18] Putative risk factors for primary SB include stress, anxiety, smoking, alcohol, and caffeine consumption, as well as genetic predisposition.[19–21] Certain medications, such as selective serotonin uptake inhibitors and dopamine antagonists, and certain sleep comorbidities, such as sleep apnea, insomnia, and periodic limb movements, have also been associated with secondary SB.[16,17] Among these associated factors, anxiety and OSA have been reported to be present in patients with higher risk for SB.[22] A recent large-scale Brazilian population study demonstrated a positive association between SB and insomnia, but not Apnea-Hypopnea Index (AHI) or OSA.[13]

Associations between OSA and SB have been reported,[23–25] but their exact relationship remains inconclusive.[26,27] The mechanisms for initiation of SB are thought to be associated with interactions between the autonomic nervous system and sleep-arousal axis. A series of biologic events that begins with cardiac sympathetic activation,[28] increases in cortical activity,[29] heart rate,[29] blood pressure,[30] and respiratory amplitude,[31] and culminating in the generation of rhythmic masticatory muscle activity (RMMA) in jaw elevator muscles suggests that SB is an oromotor activity (OMA) secondary to microarousals.[29] It was found that 88% of SB episodes were found to occur within sleep arousals of healthy individuals in a cyclic alternating pattern (CAP).[32] In OSA patients, masseter contractions have been proposed as a mechanism to protrude the mandible and increase airway patency.[33] It is unclear whether SB holds a physiologic-protective role in OSA. Hosoya et al demonstrated positive correlations between phasic-type SB events and obstructive apnea, desaturation, and microarousal event indices, suggesting that OSA might be a high-risk factor for SB.[23] In contrast, Saito et al reported that only 54.9% of SB episodes occurred after apnea-hypopnea events, while 25.5% preceded these events.[24] Sleep arousals were moderately correlated with the onset of RMMA in concomitant OSA-SB individuals,[25] but the sleep architecture of OSA-SB individuals also did not differ significantly from those without SB.[13,23]

The objectives of this retrospective study were thus to determine the prevalence of SB in an Asian sample of adult OSA patients and to examine the association between OSA and SB in terms of sleep and respiratory parameters. This study also aimed to identify possible OSA-related risk factors for SB in adult OSA patients.

Materials and Methods

Study Sample

Ethics approval was obtained from the National Healthcare Group Domain Specific Review Board (NHG DSRB) prior to commencement of the study (Reference no: 2016/00223). A waiver of informed consent was granted by the NHG DSRB. This retrospective database study involved analysis of sleep test data from 157 patients with suspected OSA who underwent a fully attended, audio-video (AV) controlled standard polysomnography (PSG) examination at the Ng Teng Fong General Hospital sleep clinic from July 2015 to February 2016. Patients diagnosed with mild, moderate, or severe OSA and aged 25 years and above were included, while those with a history of major neurologic, psychiatric, and/or other sleep disorders (eg, rapid eye movement [REM] behavior disorder, insomnia, periodic limb movement disorder) or psychoactive drug use were excluded. General demographic parameters such as age, body mass index[34] (BMI), and Epworth Sleepiness Scale[35] (ESS) scores were collected prior to the PSG study.

PSG Sleep Study

A single-night, AV-controlled Type I full PSG recording was performed on each patient. The minimum total sleep time was preset at 2 hours to allow for recording of all sleep stages and for the diagnosis of OSA.[36] Biocalibration was performed with maximal clenching or grinding movements prior to patients entering sleep. Sleep recordings were registered with surface electrodes at the following locations: 6 electroencephalogram (EEG) channels bilaterally at the F4-M1, C4-M1, O2-M1, F3-M2, C3-M2, and O1-M2 locations; 2 electrooculogram (EOG) channels at E1-M2 and E2-M2; and four Eelectromyography channels of the mentalis, anterior tibialis muscle, and the bodies of the right and left masseter muscles. A single modified 2-lead electrocardiograph (ECG) was used for cardiac monitoring. Respiratory variables measured included airflow, chest and abdominal respiratory effort signals, and arterial oxygen saturation. Body position was recorded by means of a position sensor. EEG, EOG, EMG, and ECG signals were amplified and recorded at a sampling rate of 200 Hz and stored offline with Embla RemLogic PSG software (Natus Neurology). AV footage was recorded simultaneously with each PSG. Audio recordings were used to assess audible and vibrational signals associated with snoring or other oromotor activities (eg, coughing and sleep talking), and video recordings were used to analyze movements in the leg or orofacial region. All technical and digital specifications were performed in accordance with the American Academy of Sleep Medicine

(AASM) manual for the scoring of sleep and associated events.[37]

OSA Scoring and Diagnosis

Preceding analyses of PSG data from each patient had been manually performed by a certified sleep technologist and validated by a sleep physician. Sleep, respiratory, cardiac, and movement parameters were scored in 30-second epochs according to the AASM manual for scoring of sleep and associated events.[37]

The following sleep parameters were obtained from the scored PSG data: percentage of time spent in sleep stages 1, 2, 3, and REM (time spent in each sleep stage / total sleep time * 100%); sleep efficiency (SE) (total sleep time / total time in bed * 100%); spontaneous arousal index (SAI) (total number of spontaneous arousals / total sleep time * 60); respiratory-related arousal index (RAI) (total number of respiratory-related arousals / total sleep time * 60); arousal index (AI) (total number of arousals / total sleep time * 60); minimum oxygen saturation (minOSAT) measured by pulse oximetry (ODI) (number of events of 3% drop in oxygen saturation per hour of sleep).

An apneic event was determined as a cessation in airflow of ≥ 90% for a minimum period of 10 seconds.[38] Hypopnea was identified when the airflow dropped by ≥ 30% for a period of ≥ 10 seconds, accompanied by an oxygen desaturation of ≥ 3%.[38] The AHI indicated the severity of OSA through the representation of number of apnea/hypopnea events per hour of sleep. Mild OSA was defined as having an AHI of 5 or more and less than 14 respiratory events per hour, moderate OSA between 15 or more and less than 30 events per hour, and severe OSA as 30 or more events per hour.[38]

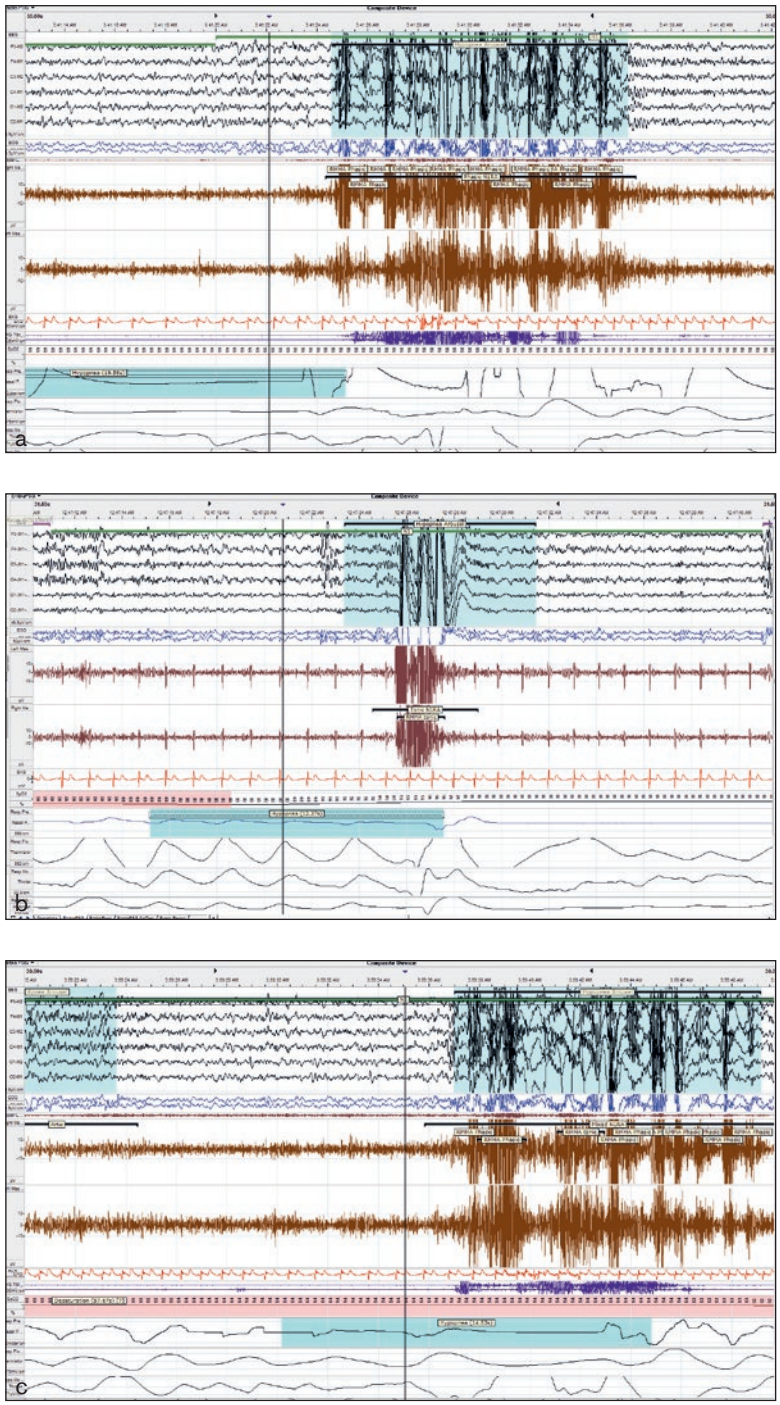

Fig 1 **(a)** Phasic, **(b)** tonic, and **(c)** mixed sleep bruxism episodes associated with hypopnea event.

SB Scoring and Diagnosis

A trained dentist and a certified sleep technologist independently and manually performed the scoring of SB. SB scoring rubrics were adapted from the AASM 2016 manual for scoring of sleep and associated events.[39] Patients were diagnosed with SB when they demonstrated more than four bruxism episodes per hour of sleep.[40]

SB episodes were established when masseter RMMA exhibited twice the background EMG amplitude and were preceded by a period of ≥ 3 seconds of stable background EMG.[39] SB episodes were scored as phasic when three or more RMMA contractions, each lasting for a duration of 0.25 to 2 seconds (Fig 1a), occurred

Table 1 Quantitative Differences in Sleep Bruxism Parameters in Sleep Bruxism (SB) and Non-SB Patients

	SB (n = 49)				Non-SB (n = 98)				
Type of RMMA/h	Mean	SD	Median	IQR	Mean	SD	Median	IQR	*P* value
RMMA/SB index (episodes/h)	8.47	5.50	7.28	4.79–9.95	2.16	1.15	2.19	1.27–3.00	< .001*
Phasic episode index (episodes/h)	4.04	4.88	2.48	1.36–4.13	0.53	0.55	0.38	0.10–0.88	< .001*
Tonic episode index (episodes/h)	3.73	2.08	3.37	2.37–4.92	1.48	0.90	1.43	0.79–2.14	< .001*
Mixed episode index (episodes/h)	0.70	0.75	0.47	0.17–0.97	0.14	0.27	0.00	0–0.19	< .001*

Mann-Whitney *U* test. RMMA = rhythmic masticatory muscle activity; SD = standard deviation; IQR = interquartile range. *Significant value ($P < .05$).

in a regular pattern; tonic when RMMA contractions lasted for ≥ 2 seconds (Fig 1b); or mixed (Fig 1c), which consisted of both phasic and tonic episodes.[39] OMA that occurred in sleep included swallowing, coughing, and sleep talking. Myoclonus contractions that lasted for less than 0.25 seconds were excluded from the analyses.[39] In instances where AV recordings were unclear, scoring of RMMA was based on EMG readings only.

Calibration was performed on a pilot sample of 11 PSG recordings to ensure evaluation reliability between scorers. In cases of SB scoring disagreement, the sleep physician was consulted.

Statistical Analyses

The prevalence of SB among OSA patients, with 95% confidence intervals (CI), was determined. Descriptive statistics were computed for the demographic characteristics of patients, including age, gender, BMI,[34] and ESS[35] scores. BMI was categorized as normal when 18.5 to 24.9 kg/m^2, overweight when 25 to 29.9 kg/m^2, or obese when ≥ 30 kg/m^2.[34] ESS was graded as normal for scores of 0 to 9 and excessive for scores of 10 to 24.[35] Categorical variables were summarized as counts and proportions, and associations among variables were determined using Pearson chi-square test. The Kolmogorov-Smirnov test was performed on all continuous variables to test for normality of distribution. They were summarized as means with standard deviations (SD) or, when the assumption of data normality was violated, medians with interquartile range (IQR). Parametric independent samples *t* test and nonparametric Mann-Whitney *U* test were performed for between-group comparisons when appropriate.

Further analyses of sleep and respiratory parameters, including RAI, SAI, AHI, and ODI, were performed using a logistic regression model, with having or not having SB as the dichotomous outcome variable. Confounding factors such as age, gender, and corresponding sleep and respiratory variables—including RAI, SAI, AHI, and ODI—were controlled for in the multivariate model. Adjusted odds ratios (OR) and 95% CI were estimated from the respective models. Statistical significance was determined at $P < .05$ except for comparisons of OSA patients with SB and without SB, where the significance was adjusted to $P < .005$ with Bonferroni correction to account for multiple comparisons. Analyses were performed using the statistical software SPSS Statistics version 21.0 (IBM).

Results

Study Sample

Data from 10 patients were omitted from the analysis (3 did not have OSA, 5 had insomnia, and 2 had incomplete data), providing a total sample of 147 patients. Among the 147 OSA patients (mean age 44.6 ± 12.8 years), 23 were diagnosed with mild OSA, 28 with moderate OSA, and 96 with severe OSA.

SB Diagnosis

Table 1 summarizes the total number of RMMA/SB phasic, tonic, and mixed episodes that occurred per hour in SB and non-SB patients. Of the 147 OSA patients, 49 (33.3%; 95% CI = 25.7% to 40.9%) were diagnosed with SB, while 98 (66.7%; 95% CI = 59.1% to 74.3%) had no SB. The occurrence of RMMA was significantly higher ($P < .001$) in SB patients (median = 7.28 episodes/hour, IQR = 4.79 to 9.95) compared to their non-SB counterparts (median = 2.19 episodes/hour, IQR = 1.27 to 3.00). Most masseteric activity occurred in the form of phasic contractions in SB patients and as tonic contractions in non-SB patients.

Comparison Between SB and non-SB Groups

Quantitative differences between the SB and non-SB groups are reflected in Tables 2 and 3. No significant differences in age, sex, BMI,[34] or ESS[35] were observed between the SB and non-SB groups. An association was found between AHI and SB ($\chi^2 = 11.64$, $P = .003$) (Table 2). Differences in sleep macrostructure and respiratory parameters between SB and non-SB patients are shown in Table 3. SB patients had significantly higher RAI (median = 44.42, IQR = 23.86 to 53.37, $P = .001$) and ODI

Table 2 Comparison of Categorical Variables in Sleep Bruxism (SB) and Non-SB Patients

	SB (n = 49)		Non-SB (n = 98)				
Categorical variables	n	%	n	%	Total no.	$\chi 2$	*P* value
Sex							
Male	37	75.5	63	64.3	100	1.89	.169
Female	12	24.5	35	35.7	47		
BMI							
Normal	9	18.4	31	31.6	40	2.92	.232
Overweight	19	38.8	31	31.6	50		
Obese	21	42.9	36	36.7	57		
ESS							
Normal	30	61.2	55	56.1	85	0.35	.555
Excessive	19	38.8	43	43.9	62		
AHI							
Mild	5	10.2	18	18.4	23	11.64	.003*
Moderate	3	6.1	25	25.5	28		
Severe	41	83.7	55	56.1	96		

Pearson chi-square test. BMI = body mass index (normal: 18.5 to 24.9; overweight: 25 to 29.9; obese: ≥ 30); ESS = Epworth Sleepiness Scale (normal: 0 to 9; excessive: 10 to 24); AHI = Apnea-Hypopnea Index (mild: 5 ≤ AHI < 15; moderate: 15 ≤ AHI < 30; severe: AHI ≥ 30).
*Significant value (*P* < .05).

Table 3 Comparison of Continuous Variables in Sleep Bruxism (SB) and Non-SB Patients

Continuous variables	SB (n = 49)				Non-SB (n = 98)				*P* value
	Mean	SD	Median	IQR	Mean	SD	Median	IQR	
Age[a]	44.02	12.47	42.00	34.00–52.50	44.95	12.97	47.00	36.00–54.00	.679
Stage 1[b]	20.09	9.38	17.82	13.54–24.80	19.63	12.55	16.76	11.84–22.15	.288
Stage 2[a]	53.22	8.53	53.64	47.61–59.04	50.03	9.50	49.61	43.50–56.02	.049
Stage 3[a]	12.35	7.69	11.68	6.34–18.55	15.08	9.17	15.34	7.58–21.42	.075
REM[a]	14.33	5.83	13.32	10.34–19.26	15.25	7.12	15.09	11.27–20.29	.434
SE[b]	85.20	12.40	90.00	80.50–93.50	85.49	13.01	90.00	82.75–94.00	.832
SAI[b]	6.63	4.37	5.89	3.41–8.90	11.83	11.95	9.15	5.22–15.53	.001*
RAI[b]	42.28	20.07	44.42	23.86–53.37	31.26	20.64	27.04	14.80–43.37	.001*
AI[b]	48.92	18.05	50.48	35.02–57.22	43.44	21.07	37.99	28.15–54.89	.033
minOSAT[b]	78.47	10.21	78.00	71.50–85.50	81.47	10.76	84.00	77.00–90.00	.043
ODI[b]	35.05	24.75	32.50	16.00–48.20	26.21	28.65	16.45	4.33–32.55	.005*

REM = rapid eye movement; SE = sleep efficiency; SAI = spontaneous arousal index; AI = arousal index; minOSAT = minimum oxygen saturation measured by pulse oximetry; ODI = oxygen desaturation index (number of events of 3% drop in oxygen saturation per hour of sleep); SD = standard deviation; IQR = interquartile range; RAI = respiratory-related arousal index.
*Significant value (*P* < .005).
[a]Independent samples *t* test.
[b]Mann-Whitney *U* test.

(median = 32.50, IQR = 16.00 to 48.20, *P* = .005), but lower SAI (median = 5.89, IQR = 3.41 to 8.90, *P* = .001) than non-SB patients.

Risk Factors Associated with SB

Sleep macrostructure and respiratory risk factors for SB were identified using logistic regression analysis (Table 4). After controlling for age, gender, AHI, SAI, and ODI, the odds of experiencing SB increased by 5% for an RAI increase of one event per hour (OR = 1.05; 95% CI = 1.00 to 1.10). Conversely, the odds of experiencing SB decreased by 11% for every SAI increase of one event per hour (OR = 0.89; 95%

Table 4 Adjusted Odds Ratios (OR) of Obstructive Sleep Apnea Risk Factors for Sleep Bruxism

Risk factors	Adjusted OR	95% CI	*P* value
AHI	0.99	0.94–1.04	.75
RAI	1.05	1.00–1.10	.07
SAI	0.89	0.80–0.96	.01*
ODI	0.98	0.95–1.01	.21

Multivariate logistic regression; ORs adjusted for age, sex, AHI, RAI, SAI, and ODI, excluding the modeled variable. AHI = Apnea-Hypopnea Index; RAI = respiratory-related arousal index; SAI = spontaneous arousal index; ODI = oxygen desaturation index (number of events of 3% drop in oxygen saturation per hour of sleep); CI = confidence interval.
*Significant value (*P* < .05).

CI = 0.80 to 0.96), after demographics and sleep factors were controlled for. There was, however, no evidence to suggest that AHI (OR = 0.99; 95% CI = 0.94 to 1.04) or ODI (OR = 0.98; 95% CI = 0.95 to 1.01) were associated with the odds of experiencing SB after controlling for demographic and sleep factors.

Discussion

This study aimed to determine the prevalence of SB in an Asian adult OSA sample and examined the association between OSA and SB in terms of sleep and respiratory parameters. Risk factors for SB in OSA patients were also identified. SB occurred in one-third of OSA patients, and significant differences in specific sleep and respiratory parameters existed between SB and non-SB OSA patients. In addition, RAI and SAI were associated with the presence of SB.

Prevalence of SB in OSA Patients

Earlier PSG studies investigating the association between OSA and SB employed sample sizes ranging from 21 to 67 patients. The sample size in the present study of 147 patients represents the largest sample of such a study to date. SB was found to be present in 33.3% of this sample. Concomitant OSA and SB was found in 48% of a group of 67 OSA Japanese patients[23] and in 51% in another recent study of 59 Japanese patients.[25] In a third study, SB was diagnosed in 54% of 11 mild OSA patients and in 40% of 10 moderate OSA patients.[41] The prevalence of SB in OSA patients was thus modestly lower in the current cohort, which may be because of the larger sample size in this study. Diagnosis of SB in this study was made through a single-night, objective sleep laboratory PSG scoring, which is currently considered the gold standard in SB diagnosis.[40] AV recordings were used to exclude other oromuscular activities and sleep movement disorders.[42,43] Although ambulatory EMG devices provide some objective identification of RMMA, they frequently overestimate SB events related to orofacial movements occurring in wake stages.[44] Other methods described for SB diagnosis in the literature include patient self-report and self-awareness, but the subjective nature of these methods often leads to overdiagnosis.[13,25] Clinical examination using wear in the dentition as a surrogate for SB is at best only a historical representation of the condition.

Current international consensus adopts a possible, probable, and definite classification system for the diagnosis of SB.[15] Definite diagnosis of SB is based on patient report and clinical examination supplemented by PSG findings.[15] A recent preliminary study, however, reported fair to moderate concordance between the International Classification of Sleep Disorders (ICSD) (3rd edition) SB criteria and PSG for SB diagnosis.[45] Successive recordings over 2 nights are generally recommended to exclude any first-night effect and sleep disorders.[46] Hasegawa et al suggested that a single-night recording with a full Type 1 PSG is also acceptable for the diagnosis of SB involving more than four SB episodes per hour.[47] The lower prevalence of SB in OSA patients reported in this study could also be due to the different SB diagnostic criteria prescribed across studies.[25,41] In this study, SB scoring was performed in accordance with the AASM 2016 guidelines,[39] and SB diagnosis was made based on the Research Diagnostic Criteria of four RMMA/SB episodes per hour.[40] The higher SB prevalence reported in the Japanese study[25] could be due to the lower cut-off criterion of 2 RMMA/SB episodes per hour employed[48]; this cut-off was 2.5 RMMA/SB episodes per hour in another Canadian sample.[41]

Relationship of SB Episodes to Respiratory and Spontaneous Arousals

This study found that SB patients had greater RAI, whereas non-SB patients had greater SAI. Neither group, however, showed a significant difference in total number of arousals, suggesting the possible presence of a protective compensatory mechanism in preserving the CAP of OSA-SB patients. Respiratory-related arousals involve apnea, hypopnea, respiratory-effort–related arousals, and snoring events that fragment sleep, while spontaneous arousals are perceived as physiologic cortical activations arranged within a period of 20 to 40 seconds, known as CAP.[49] Spontaneous arousals are normally distributed in healthy SB patients.[50] Higher RAI in OSA-SB patients suggests substitution of certain spontaneous arousals from the expected sleep distribution with respiratory-related arousals when sleep is not severely interrupted.[51]

In addition to larger RAI, it was observed that OSA patients with SB had greater oxygen desaturation each hour compared to non-SB patients. A preliminary study suggested transient reductions in oxygen saturations were associated with the initiation of RMMA episodes in certain idiopathic SB patients.[52] These hypoxic RMMA episodes were found to be independent of arousals and large body movements.[52] The frequency of RMMA was also increased by 7 times when experimental arousals were induced through vibrotactile and auditory stimuli in healthy SB patients.[53]

The relationship of RMMA/SB to oxygen desaturation and respiratory-related arousal may be assumed as cortical, autonomic, and somatomotor

activations resulting from respiratory disturbances. Respiratory control is modulated by the thalamocortical system via slow waves, which are replaced by faster EEG activities in greater respiratory obstructions to effect a larger autonomic response.[54] In severe OSA patients, the CAP rate also increases with a greater proportion of A3 subtype, in which most SB episodes are found to occur.[55] It may be possible that SB is an autonomic response to cortical arousals from a sufficiently large respiratory challenge to restore oxygen imbalances. Activation of masticatory muscles acts to stabilize the mandible and achieve airway patency during increased respiratory resistance.[33] This physiologic-protective role of SB in OSA has been previously discussed in the temporal associations between OSA and SB, where an apnea or hypopnea event precedes the initiation of an SB episode.[56] Nonetheless, it is not possible to establish a causal relationship between SB and respiratory-related arousals within the limitations of this study.

Association Between OSA and SB

This study demonstrated that RAI and SAI had marginal effects on the odds of experiencing SB after controlling for demographic and sleep variables. Although a significant difference in AHI was found between SB and non-SB OSA patients, this may be due to the inherently larger sample of severe OSA patients seeking treatment and being referred to the sleep clinic. The findings in this study thus underscore the weak association between OSA and SB. Consistent with these findings, Satio et al established a low correlation between RMMA/SB bursts and AI.[25] AHI was more related to other types of oromotor activities, such as swallowing, coughing, and face scratching, which suggests that the genesis of SB and OSA events may stem from different mechanisms.[25] In an earlier study, these authors had not been able to establish a causal relationship between OSA and SB.[24] It was reported that 54.9% of SB events occurred after apnea-hypopnea events in concomitant OSA-SB patients, while 25.5% of SB events occurred prior to the apnea-hypopnea event. The remaining 19.5% of SB events were unrelated to any apnea-hypopnea events.[24] The occurrence of SB and apnea-hypopnea events may be epiphenomena existing in the same time window with no relation between events.

This weak association also alludes to the possibility that SB may be manifested in specific OSA pathophysiologic phenotypes. It is plausible that OSA is a heterogenous disorder with multiple pathophysiologic causes. Upper airway anatomy plays a significant role in airway collapsibility and breathing stability, but other pathologic phenotypic traits such as low arousal threshold, oversensitive respiratory control, and poor upper airway neuromuscular responsiveness may coexist.[57] The smaller number of SB episodes in the remaining OSA patients with no SB could suggest a smaller anatomical cause but a greater contributory role in the other pathophysiologic factors. However, it is not possible to draw any associations between SB and the different OSA phenotypes from this study.

Other Study Limitations

Other limitations of this study include the use of a single-night PSG assessment, which may produce first-night masking of the true severity of SB in patients demonstrating low-frequency RMMA.[47] A clinical screening and examination for signs and symptoms of TMD was not conducted, which may have the potential to influence both OSA and SB. Another limitation of this study is the lack of a control group consisting of healthy SB patients for meaningful comparison of sleep and respiratory parameters. The use of PSG on a large scale is, however, expensive and tedious, which may limit the feasibility of a matched control group.

Conclusions

This study demonstrated that SB occurs in about one-third of OSA patients, with RMMA mainly in the phasic form. OSA-SB patients had greater respiratory-related events and arousals, but their sleep architecture did not differ greatly from OSA patients with no SB. This positive association suggests a phenotypic subtype of OSA patients may present with SB as a physiologic response to a respiratory-related event. Greater understanding and cross-disciplinary referrals between medical and dental clinicians will help in the appropriate prescription of therapeutic interventions for these patients.

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