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# Sleep Bruxism in Thai Obstructive Sleep Apnea Patients

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ABSTRACT

*Objectives:* To investigate the prevalence of sleep bruxism (SB) in Thai obstructive sleep apnea (OSA) patients and to identify demographic characteristics and specific sleep parameters associated with SB.

*Methods:* A total of 119 medical records, each containing full-night type I polysomnography from Thai patients with OSA, were included. SB was detected using surface electromyography of the masseter muscle. SB was diagnosed when the SB index reached at least two episodes per hour of sleep. The differences in demographic characteristics and sleep parameters between SB and non-SB groups were analysed. Multivariate logistic regression analysis was performed to determine the associated factors for SB.

*Results:* Among Thai patients diagnosed with OSA, 50.4% concurrently experienced SB, predominantly of the tonic type. The study revealed a higher prevalence of SB in males compared to females. The SB group demonstrated significantly higher values in the apnea-hypopnea index (AHI), Epworth Sleepiness Scale (ESS), Arousal Index (AI), and Respiratory Arousal Index (RAI) compared to the non-SB group. Multivariate logistic regression analysis indicated that a lower body mass index (BMI), higher ESS, and increased severity of AHI were significantly associated with SB.

*Conclusions:* The study revealed that half of Thai patients diagnosed with OSA also exhibited SB. Male, AHI, ESS, AI, and RAI appeared to be potential correlates for the presence of SB. Lower BMI, higher ESS, and elevated AHI can be factors associated with SB in Thai OSA patients.

*Clinical Relevance:* The prevalence of SB among Thai patients diagnosed with OSA and the factors associated with its occurrence were investigated.



## Introduction

Sleep bruxism (SB) refers to the activity of masticatory muscles during sleep, categorized as either non-rhythmic (tonic) or rhythmic (phasic). It is not considered as a movement or sleep disorder in otherwise healthy individuals.[1] Tonic contraction, identified by prolonged jaw clenching, and phasic contraction, a sequence of repetitive sleep-related muscle contractions, are labelled as rhythmic masticatory muscle activity (RMMA). Teeth grinding may occur when masticatory muscles contract forcefully.[2] The prevalence of SB is estimated to be 1%-15% in the general population[3] and decreases with age, exhibiting no significant gender difference.[4]

SB can be classified as either primary, with no identifiable cause, or secondary, linked to medical comorbidities such as OSA, Parkinson's disease, gastroesophageal reflux, periodic limb movements,[5] and hypertension.[6] However, there is insufficient evidence-based data to conclusively establish the impact of medications and addictive substances on the development or exacerbation of SB.[7] Moreover, research suggests a potential genetic contribution to the etiology of SB, which could influence the risk for SB development.[8] Nonetheless, the exact cause of SB remains unclear.

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SB is considered an oromotor activity (OMA) based on a series of biological events that begin with cardiac sympathetic activation, increased cortical activity, heart rate, blood pressure, and respiratory amplitude. These circumstances are followed by the development of RMMA in the jaw elevator muscles.[9] SB may result in muscle fatigue, orofacial discomfort, temporomandibular disorders, bilateral masseter hypertrophy, limited mandibular mobility, headaches, and/or significant impacts on oral and tooth structures.[10-12] Although SB is not classified as a specific pathology, several studies have considered it as a risk or protective factor for other disorders.[1] Thus, its treatment should focus on investigating the contributing factors rather than only focusing on SB.[13] Continuous positive airway pressure (CPAP) and mandibular advancement device (MAD) can potentially reduce the incidence of SB along with OSA upon their initial use.[14] However, early detection and diagnosis of SB are also essential for applying preventive management approaches, such as night guard appliance, to reduce the risk of tooth damage and orofacial problems.

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder identified by the complete (apnea) or partial (hypopnea) collapse of the upper airway, typically resulting in sleep arousal and decreased blood oxygen levels.[15] The etiology may involve both anatomical and physiological factors. Four phenotypes are typically associated with OSA: the collapsibility of upper airway anatomy, inadequate responsiveness of upper airway muscles, reduced threshold for arousal in response to respiratory stimuli, and high loop gain.[16] The prevalence of OSA in adults ranges from 9%-38%.[17] Its occurrence increases with age and is more frequently observed in Asians than Caucasians.[18] Obesity and males are considered to be risk factors for OSA.[19] Furthermore, OSA results in notable health concerns, such as excessive daytime sleepiness, hypertension, cardiovascular diseases, reflux esophagitis, diabetes mellitus, neurocognitive impairment, and elevated mortality.[20]

Many studies have attempted to determine the relationship between SB and OSA. A prior research suggested that recurrent hypoxic episodes may be an underlying link between SB, masticatory muscle pain, and sleep-related breathing disorders.[21] Lower blood serotonin levels have been observed in both OSA and SB.[22,23] Additionally, genetic variations in the serotonin receptor encoding gene have been related to the development of SB and may affect its association with OSA.[8] Current evidence is insufficient to confirm a definite relationship between SB and OSA. A prevalent theory posits that SB serves as a protective mechanism in response to OSA. In contrast, some theories suggest that SB may lead to the development of OSA. Additionally, other hypotheses argue that there is no link between SB and OSA, or that the two conditions may coexist independently.[24]

Multiple studies have indicated a higher prevalence of SB in patients with OSA compared to the general population.[9,25-28] Moreover, many studies have recognized a significant correlation between SB and OSA.[25,29-32] Despite the increasing recognition of SB and OSA as substantial public health issues, there is a notable deficiency in the literature concerning data specific to the Thai population. This study aimed to mitigate this deficiency by evaluating the prevalence of SB in Thai individuals diagnosed with OSA, and by elucidating the demographic characteristics and sleep parameters that correlated with SB within the group of Thais.

## Materials and methods

This retrospective cross-sectional study analysed 241 medical records of Thai patients who underwent polysomnography (PSG) at Golden Jubilee Medical Center, Faculty of Medicine Siriraj Hospital, Mahidol University, between May-December 2022. Thai OSA patients aged 20 years and above were included in this study. Attachment of electromyography electrodes on the left and right masseter muscles was required during the full-night type I PSG for SB diagnosis. Patients with a history of major neurologic, psychiatric, and/or sleep disorders (e.g., rapid eye movement (REM) behaviour disorder, insomnia, periodic limb movement disorder), psychoactive drug use, or sedative-hypnotic drug use were excluded.

PSG data, sleep parameters, and associated events were interpreted by two certified sleep specialists using the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events, version 2.6,[33] with the assistance of the DOMINO program, SOMNOmedics Germany. Hypopnea was monitored through a nasal pressure or an alternative hypopnea sensor for at least 10 seconds with a decrease in airflow of at least 30% from the pre-event baseline and a reduction of the oxygen saturation value of 3% or more from the pre-event baseline, or the event that was linked to arousal. Apnea was defined as a reduction in peak signal excursion by at least 90% from the pre-event baseline, lasting a minimum of 10 seconds, using either an oronasal thermal sensor or an alternative apnea sensor.[33] The severity of OSA was determined using the apnea-hypopnea index (AHI), which was classified into 4 categories as follows: normal (AHI $< 5$), mild ($5 \leq$ AHI $< 15$), moderate ($15 \leq$ AHI $< 30$), and severe (AHI $\geq 30$).[34]

The criteria for diagnosing and confirming SB required masseter assessment via EMG and necessitated at least twofold elevation from the background EMG amplitude. According to the sleep bruxism index (SBI), the diagnosis of SB was made in the presence of at least 2 episodes of SB per hour.[35] Three or more RMMA contractions of masseter EMG lasting 0.25 to 2 seconds were identified as phasic type, while RMMA contractions of masseter EMG lasting more than 2 seconds were identified as tonic type. The presence of both phasic and tonic types was considered a mixed type. A new episode of SB was identified, by leaving at least 3 seconds of stable background masseter EMG.[33]

The prevalence of SB in OSA patients was calculated, using SPSS version 28.0 for statistical analysis, and descriptive statistics were computed to present all demographic characteristics of the sample group. The Pearson chi-square test, independent samples t-test, or Mann-Whitney U test was used to compare the differences of each factor between non-SB and SB groups. Multivariate logistic regression was applied to identify the associated factors for SB in OSA patients.

## Results

Out of a total 241 medical records, 119 subjects were included in this study. Subjects who were under the age of 20 ($n = 4$), had AHI $< 5$ ($n = 4$), underwent split-night PSG ($n = 77$), lacked masseter EMG records ($n = 23$), had a history of major

**Table 1 – Quantitative differences in the types of SBI between non-SB and SB patients.**

| Type of SBI (episode/h) | Median (P25, P75) | | P-value |
|---|---|---|---|
| | Non-SB (SBI < 2) ($n$ = 59) | SB (SBI ≥ 2) ($n$ = 60) | |
| **Total SB index** | **0.8 (0.4, 1.1)** | **3.7 (2.8, 5.4)** | **< .001*** |
| Phasic episode index | 0.1 (0, 0.3) | 1.3 (0.6, 2.3) | < .001* |
| Tonic episode index | 0.4 (0.2, 0.8) | 1.7 (1.2, 2.7) | < .001* |
| Mixed episode index | 0 (0, 0.1) | 0.4 (0.1, 1.1) | < .001* |

Mann-Whitney U test.

* Significant difference (P-value < .05)

neurologic, psychiatric, and/or sleep disorders ($n$ = 6), or used psychoactive or sedative-hypnotic medication ($n$ = 8) were excluded from this study. There were 75 male and 44 female subjects, with an average age of 48.4 ± 15.0 years. Notably, 60 subjects were identified as having SB (50.4%), and 59 patients (49.6%) were classified as non-SB.

The comparison between the SB and non-SB groups is shown in Table 1. SB group exhibited a significantly higher episode index for all SB types. The tonic episode index was also slightly more pronounced than the other types in both the SB and non-SB groups.

Analysis of demographic characteristics between SB and non-SB groups revealed a significant difference in gender. SB group exhibited a significantly higher number of males than females (P = .049). According to sleep parameters, the SB group displayed a significantly higher Epworth Sleepiness Scale (ESS) score than the non-SB group (P = .019). OSA severity was significantly associated with a higher probability of SB (P = .034). The incidence of SB was 29.6% in the mild OSA group, 52.17% in the moderate OSA group, and 60.9% in the severe OSA group. Moreover, the SB group exhibited a significantly higher arousal index (AI) and respiratory arousal index (RAI) compared to the non-SB group (P = .047 and .005, respectively). Conversely, the spontaneous arousal index (SAI) did not significantly differ between the groups (Table 2).

An increase in BMI was associated with a reduced risk of SB (adjusted OR = 0.93, 95% confidence interval (CI) 0.87-0.99). Additionally, OSA patients with high ESS had an elevated risk of SB (adjusted OR = 1.09, 95% CI 1.00-1.19). According to the severity of OSA, moderate OSA (adjusted OR = 2.87, 95% CI 1.01-8.18) and severe OSA (adjusted OR = 3.84, 95% CI 1.30-11.35) had higher odds of having SB, compared to mild OSA (Table 3).

## Discussion

Our study revealed that half of Thai OSA patients were diagnosed with SB (50.4%). This finding aligned with previous studies that utilized similar diagnostic criteria for PSG-

**Table 2 – Baseline characteristics of the sample group and a comparison of factors between non-SB and SB patients.**

| Factors | $n$ (%) / Median (P25, P75) / Mean ± SD | | | P-value |
|---|---|---|---|---|
| | Total ($n$ = 119) | Non-SB ($n$ = 59) | SB ($n$ = 60) | |
| Sex | | | | |
| Female | **44** | 27 (61.4) | 17 (38.6) | .049*,† |
| Male | **75** | 32 (42.7) | 43 (57.3) | |
| Age | **48.4 ± 15.0** | 46.9 ± 14.3 | 50.4 ± 15.2 | .205§ |
| Neck circumferential (cm.) | **38.2 ± 5.0** | 38.6 ± 4.5 | 38.4 ± 3.4 | .748§ |
| BMI (kg/m$^2$) | **27.7 (25.0, 32.5)** | 27.7 (25.3, 35.1) | 27.2 (24.5, 30.9) | .131‡ |
| ESS | **6.0 (3.0, 10.0)** | 4.0 (2.0, 8.0) | 7.5 (4.0, 10.8) | .019*,‡ |
| AHI | **24.9 (15.5, 37.7)** | 23.0 (12.6, 35.9) | 28.7 (19.4, 38.0) | |
| Mild (5 ≤ AHI < 15) | **27** | 19 (70.4) | 8 (29.6) | .034*,† |
| Moderate (15 ≤ AHI < 30) | **46** | 22 (47.8) | 24 (52.2) | |
| Severe (AHI ≥ 30) | **46** | 18 (39.1) | 28 (60.9) | |
| AI | **20.7 (12.6, 27.7)** | 18.4 (10.7, 27.2) | 23.0 (15.5, 29.2) | .047*,*,† |
| SAI | **3.5 (1.5, 5.9)** | 3.8 (1.7, 6.1) | 2.8 (1.3, 5.8) | .335‡ |
| RAI | **13.0 (7.2, 20.0)** | 9.9 (5.7, 17.4) | 16.1 (10.0, 21.1) | .005*,‡ |
| Total sleep time (hrs) | **6.4 (5.3, 6.9)** | 6.3 (5.4, 6.8) | 6.5 (5.2, 7.1) | .425‡ |
| Sleep efficiency (%) | **80.5 (68.4, 88.7)** | 80.5 (68.5, 88.5) | 81.6 (68.5, 89.6) | .752‡ |
| Mean SpO2 (%) | **95.0 (94.0, 96.0)** | 94.0 (94.0, 96.0) | 95.0 (94.0, 96.0) | .329‡ |
| Min SpO2 (%) | **84.0 (79.0, 88.0)** | 85.0 (79.0, 89.0) | 84.0 (79.0, 87.0) | .432‡ |
| ODI | **14.50 (8.9, 26.8)** | 14.0 (5.4, 24.5) | 16.7 (9.9, 28.1) | .222‡ |
| Stage N1 (%) | **12.7 (8.3, 18.9)** | 12.2 (8.2, 18.3) | 14.5 (8.3, 20.6) | .513‡ |
| Stage N2 (%) | **53.33 ± 11.30** | 55.3 ± 10.1 | 52.1 ± 11.1 | .100§ |
| Stage N3 (%) | **13.36 ± 9.52** | 12.2 ± 8.5 | 14.5 ± 10.5 | .185§ |
| Stage R (%) | **17.05 ± 6.56** | 16.9 ± 7.3 | 17.4 ± 5.8 | .672§ |

BMI = body mass index; ESS = Epworth sleepiness scale; AHI = Apnea-Hypopnea index; AI = arousal index; SAI = spontaneous arousal index; RAI = respiratory-related arousal index; Mean SpO2 = the average oxygen saturation; Min SpO2 = the lowest oxygen desaturation; ODI = oxygen desaturation index (number of events of 3% drop in oxygen saturation per hour of sleep).

* Significant difference (P-value < .05).
† Pearson chi-square test.
‡ Mann-Whitney U test.
§ Independent samples t-test.

**Table 3 – Adjusted odds ratios for associated factors of SB in relation to OSA.**

| Associated factors | Adjusted OR | 95% CI | P-value |
|---|---|---|---|
| BMI | 0.93 | 0.87-0.99 | .022* |
| ESS | 1.09 | 1.00-1.19 | .047* |
| AHI | | | |
| Mild | 1 | Reference | |
| Moderate | 2.87 | 1.01-8.18 | .049* |
| Severe | 3.84 | 1.30-11.35 | .015* |

Multivariate logistic regression.

* Significant difference (P-value < .05).

assessed SB, reporting high SB prevalence rates in OSA patients (49%-50.8%).[26-28] However, variations in SB diagnosis criteria may lead to differences in SB prevalence. Tan et al and Hosoya et al, utilized criteria of SBI ≥ 4 episodes/h and observed a lower SB prevalence rate of 33.3%-47.8%.[9,25] Since there are several criteria for determining SB cutoff points, our study selected SBI ≥ 2 episodes/h. These criteria can enhance the sensitivity in identifying patients with mild SB, allowing prompt management to protect against tooth and oral structure damage.

Regarding the type of SB, our research observed a slightly higher occurrence of the tonic type compared to the phasic and mixed types in both the SB and non-SB groups. Previous studies showed a significant correlation between tonic EMG activity during SB episodes and sleep-related breathing disorders.[36] A study by Inoko et al, found that tonic contractions of the masseter muscles followed the termination of apnea and hypopnea events.[37]

In this study, the prevalence of SB was significantly higher in males. However, gender was not a significant factor during multivariate logistic regression analysis. This result may be affected by behaviours that elevate the risk of SB, such as smoking and alcohol consumption,[38] which are commonly found in males.[39,40] According to Li et al., the differences in the collapsibility of the upper airway, neurochemical control mechanisms, and sex hormones between males and females may influence the frequency and ventilatory response to sleep arousals, resulting in a higher prevalence of SB in males.[27]

In the general population, there is no correlation between excessive daytime sleepiness (EDS) and SB.[41] Conversely, our study showed elevated ESS scores in the SB group compared to the non-SB group. However, both groups exhibited ESS scores within the normal range, suggesting that SB may not be related to EDS. Multivariate logistic regression indicated a borderline significant association between ESS score and SB with a 95% CI of 1.00-1.19. Despite statistical significance, this difference may not be clinically relevant. Moreover, various additional factors, such as smoking, alcohol consumption, irregular sleep schedules, and an active or stressful lifestyle, may contribute to sleep arousal and heighten the risk of EDS, which can obscure the association between OSA and SB.[42]

Our study showed a positive association between the severity of OSA and the prevalence of SB. This finding suggests a potential compensatory mechanism, wherein SB may serve a physiological protective role in OSA. Advancement of the mandible and tongue with masseter muscle activation can stabilize the mandible and assist the genioglossus muscle to promote upper airway patency.[43-45] In response to OSA, the coactivation of jaw-opening and jaw-closing muscles during rhythmic masticatory muscle activity (RMMA) has been observed to reopen the upper airway.[46] In addition, the opening of the upper airway via CPAP has been shown to significantly reduce SB.[28,43] However, Martynowicz et al noted a positive correlation of SBI with only mild to moderate OSA, suggesting that SB may not be sufficient as a protective mechanism to restore airway in severe OSA. On the contrary, other mechanisms, such as excessive respiratory effort and/or increased respiratory rate, may be more effective.[47] Moreover, our results excluded patients who underwent split-night PSG (AHI ≥ 40), which may be one of the explanations for the difference in the outcomes.

In our findings, SB was significantly related to AI and RAI, except for SAI. It can be assumed that SB may be related only to AI involving respiratory events. Consistent with prior cross-sectional studies, an association was found between SB and arousal events in OSA patients.[48,49] SB is considered a central nervous system motor response to arousal induced by OSA.[50] This hypothesis supports our findings in which the SB group exhibited more severe OSA and a higher AI than the non-SB group. Similarly, a previous study by Lobbezoo et al, exhibited a link between masticatory muscle contractions after respiratory arousal episodes.[1]

Interestingly, our study noted an association of BMI with reduced risk of SB (adjusted OR = 0.93, 95% CI 0.87-0.99). In a prior study, a reverse correlation was observed between SB in OSA patients and BMI (OR = 0.951, $P < .001$).[27] This result may have been caused by the thickness of the masticatory muscle, which can affect EMG activity. Regalo et al found a reduction in EMG activity and an increase in the thickness of the masseter and temporal muscles with obesity. Excessive accumulation of intramuscular fat can disrupt the contractile and proper functioning of striated skeletal muscles, reducing EMG activity.[51]

Our principal findings provided information on SB prevalence, demographic characteristics, and sleep parameters associated with SB in Thai OSA patients. Implications for clinicians include adopting a multidisciplinary approach, whereby physicians can detect SB early by assessing PSG. Then, prompt referral of patients can be made to dentists for suitable interventions to prevent tooth damage and associated orofacial problems caused by SB. Meanwhile, it is important for dentists to consider underlying medical conditions or OSA of patients suffering from SB. Therefore, dentists should prioritize screening examinations for OSA in SB patients. In the presence of OSA related signs or symptoms, these patients should be referred to a sleep specialist for suitable treatment. Due to the positive association between SB and AHI, AI, and RAI in OSA patients, treating OSA with CPAP, MAD, upper airway surgery, or medications to diminish AHI and arousal events may effectively reduce SB.

A notable strength of our study was the utilization of full-night type I polysomnography (PSG), a gold standard for diagnosing SB.[52] The sleep parameters and associated events, including SB diagnosis, were scored and analysed by sleep specialists, ensuring the accuracy of the obtained information. However, our study had some limitations. Sleeping in an unfamiliar environment may lead to lower sleep quality due to the first-night effect, while night-to-night variation could potentially introduce inaccuracies in various sleep

parameters. Confirming a sleep bruxism (SB) diagnosis may require multiple nights of polysomnography (PSG).[53] Our study diagnosed SB based on a one-night PSG without considering the signs and symptoms of the patients.

Future research should investigate the correlation between electromyography (EMG) activity of the masseter muscle, and other clinical signs/symptoms of SB, such as attritions, abfractions, bony exostosis, tongue scalloping, and buccal mucosal ridging, to establish cut-point criteria for diagnosing SB from PSG data and to develop guidelines for the management of SB in OSA patients. Furthermore, a cohort study is needed to investigate the causal relationship between SB and OSA.

## Conclusions

Approximately half of Thai patients with OSA were diagnosed to have SB, with the tonic-type SB predominating in our sample. Correlates of SB in OSA patients were male, elevated AHI, ESS score, AI, and RAI. Moreover, lower BMI, higher ESS score, and increased AHI were identified as factors influencing SB in OSA patients.

## Conflict of 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.

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## Ethical approval

This study received ethical approval from Mahidol University Multi-Faculty Cooperative IRB Review, protocol number: MU-MOU 2022/DT141.

## Informed consent

Since the study was retrospective in nature, informed consent was not required.

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## Author contributions

Use this form to specify the contribution of each author of your manuscript. A distinction is made between five types of contributions: Conceived and designed the analysis; Collected the data; Contributed data or analysis tools; Performed the analysis; Wrote the paper.

For each author of your manuscript, please indicate the types of contributions the author has made. An author may have made more than one type of contribution. Optionally, for each contribution type, you may specify the contribution of an author in more detail by providing a one-sentence statement in which the contribution is summarized. In the case of an author who contributed to performing the analysis, the author's contribution for instance could be specified in more detail as "Performed the computer simulations," "Performed the statistical analysis," or "Performed the text mining analysis."

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