



Original Article

Determinants of probable sleep bruxism in a pediatric mixed dentition population: a multivariate analysis of mouth vs. nasal breathing, tongue mobility, and tonsil size



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ABSTRACT

Objectives: This study aims to identify structural and functional craniofacial characteristics that correlate with higher incidence of 'probable' sleep bruxism in children.

Methods: From March 2018 until March 2019, a cross-sectional clinical study was performed with ninety-six healthy children ages 6–12 years who presented for routine dental examination at the UCLA pediatric dental clinic. Variables of interest included: (1) assessment of probable bruxism based on parental awareness on the frequency of tooth grinding during sleep and clinical signs of bruxism based on tooth wear; (2) parental reports of mouth breathing while awake and asleep, snoring during sleep, difficulty breathing and/or gasping for air during sleep; (3) parental reports of psychosocial distress; (4) assessment of tonsil hypertrophy, tongue mobility, and nasal obstruction. Three pediatric dental residents were calibrated to perform the clinical data collection. All dental residents were graduated dentists with licensure and at least one year of experience examining children. The methodology to take the specific measurements administered in the manuscript were calibrated between the data-collectors under the supervision of a board-certified pediatric dentist and orthodontist (AY).

Results: The mean age of individuals was 8.9 (SD = 1.9) years with a gender distribution of 46 males and 50 females. There were 23 out of the 96 (24%) individuals who met the diagnostic criteria for probable sleep bruxism (PSB). Sleep Disturbance Scale for Children (SDSC) scores were significantly elevated among children positive for PSB, indicating that they are at higher risk for sleep disturbances (PSB-positive: 45.1 ± 13.0 , PSB-negative: 34.8 ± 5.5 ; $p < 0.0001$). Impaired nasal breathing, parental reports of mouth breathing when awake or asleep, restricted tongue mobility, and tonsillar hypertrophy were found to be significant risk factors for PSB. Exploratory analysis further suggests a synergistic effect between tonsil hypertrophy, restricted tongue mobility, and nasal obstruction. The incidence of probable sleep bruxism among individuals without any of the exam findings of tonsillar hypertrophy, restricted tongue mobility, and nasal obstruction was 5/58 (8.6%), whereas the incidence of PSB among individuals with all three exam findings was 10/11 (90.9%), $p < 0.0001$. Among the 23 individuals with PSB, however, there were $n = 5$ (21.7%) who did not have any of the three exam findings, suggesting an additional role of psychosocial distress, postural maladaptation, malocclusion, or other factors in the etiology of sleep bruxism.

Conclusion: This study shows that tonsil hypertrophy, restricted tongue mobility, and nasal obstruction may have a synergistic association on the presentation of PSB. Dentists should evaluate for tonsillar hypertrophy, restricted tongue mobility, and nasal obstruction in the evaluation of PSB, as these exam findings are highly prevalent in the majority of cases.

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1. Introduction

Bruxism is defined as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible which may occur during sleep (sleep bruxism), during wakefulness (awake bruxism), or both. A diagnostic grading system of bruxism has been proposed for clinical and research purposes. ‘Possible’ bruxism is based on self-reported, ‘probable’ is based on self-reported plus the inspection part of clinical examination and ‘definite’ bruxism is based on self-reported, a clinical examination, and a polysomnographic recording preferably along with audio/video recording [1].

The prevalence of bruxism in children is highly variable with a range of 3.5–40.6% [2]. Identifying bruxism at an early age is crucial as it is a clinical risk factor for tooth wear, dental damage and fractures, jaw muscle fatigue, and pain [3].

Evolutionarily, keeping teeth sharp by bruxing has importance for food retrieval and defense. In humans, remnants of this mechanism is believed to may have remained in some individuals as a maladaptive, inherited behavior [4]. Whereas clenching and grinding of teeth may sometimes be associated with normal teething and play a fundamental role in dental health and maxillofacial development, bruxism is considered clinically maladaptive and pathologic when associated with excessive dental wear. Risk factors for pathologic bruxism include psychological or physical distress, behavioral abnormalities, and sleep disturbances [5]. Current treatment modalities in pediatric dentistry are often limited to focusing on the consequences of bruxism (as with occlusal splints) or dental restoration as opposed to understanding and addressing associated factors that may predispose to repetitive masticatory-muscle activity [6,7].

The primary aim of this study was to identify parent-reported measures and clinical findings associated with probable sleep bruxism (PSB) among children. The secondary objective of this study was to determine the association between the various factors through multivariate exploratory analysis.

2. Methods

2.1. Design

From March 2018 until March 2019, a prospective cohort study was conducted of ninety-six healthy children (mean age, 8.86 years; range, 6–12 years; 46 male, 50 female), who presented for routine dental examination at the University of California, Los Angeles (UCLA). Exclusion criteria were as follows: respiratory disease, known comorbidities, prescribed medications for chronic disease, premature birth, craniofacial defect, prior orthodontic therapy, prior tonsillectomy, prior oral or maxillofacial surgery. We assessed the impact of parent-reported measures based on parent report as well as clinical findings based on examination by three pediatric dental residents on the outcome measure of probable sleep bruxism. Clinical examiners were blinded to the subjective survey and SDSC tool administered. To test inter-examiner agreement, 10 individuals were selected at random and measured by all three residents. Intraclass correlation coefficient (ICC) in a mixed model was used to assess inter-rater agreement among the three residents for clinical item measurements. All showed excellent agreement (ICC>0.85) according to Cicchetti’s guideline.

2.2. Sleep Disturbance Scale for Children (SDSC)

SDSC, developed in 1996 by Bruni et al., is a screening tool for parent-reported symptoms of sleep disorders in children that has

been validated in previous studies. It comprises 26 items in a Likert-type scale with values 1–5, with higher numerical values reflecting a greater clinical severity of symptoms. The sum of the scores was calculated to be the individual’s sleep index score (range 26–130). Scores ranging from 26 to 35 are considered to be within normal range; 36–44, 45–51, >52 are considered to be at mildly, moderately, severely, increased risk for sleep disturbances, respectively.

2.3. Clinical assessment tool

In addition, the FAIREST-15 clinical examination screening tool also includes six questions regarding breathing route, posture, concentration, and anxiety measures. Parents were asked to rate on a scale of 1–4 the extent to which their child breathes through their mouth when awake and when asleep; difficulty with concentration; anxiety measures (see [appendix A](#)). The FAIREST-15 tool was adapted from FAIREST-21 tool which has been established in previous literature [10].

2.4. Clinical findings

The clinical findings included in this study were the assessment of tonsillar hypertrophy based on the Brodsky scale [Grade 1 (<25%); Grade 2 (26–50%); Grade 3 (51–75%); Grade 4 (>75%)] [8], tongue mobility based on the tongue range of motion ratio (TRMR) scale [Grade 1 (>80%); Grade 2 (50–80%); Grade 3 (<50%); Grade 4 (<25%)] [9], and nasal obstruction based on the nasal breathing test [<1 min; 1–2 min; 2–3 min; 3+ min] (see [appendix A](#)). Nasal breathing test was administered by sealing lips and mouth of the subject with gentle MicroPore paper tape. A timer was used to assess how long the subject could comfortably breathe through the nose for up to 180 s with the lips and mouth taped. Subjects were deemed to pass the test if they could successfully breathe through the nose for three minutes [10].

2.5. Assessment of bruxism

Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism).” A diagnostic grading system of ‘possible’, ‘probable’, and ‘definite’ sleep or awake bruxism has been developed for clinical and research purposes [1]. In this manuscript, assessment of bruxism was based on parent-report as well as clinical examination which meets the criteria for ‘probable’ sleep bruxism. Parent-report of sleep bruxism in this study was based on question #19 of the SDSC which asks the frequency to which “the child grinds teeth during sleep” rated on an ordinal scale: never, occasionally, sometimes, often, always. Clinical examination for the presence of dental wear was assessed on an ordinal scale: none, mild, moderate – severe. The assessment of probable bruxism was conferred if individuals met both criteria: affirmative parent reports (occasional-always teeth grinding during sleep) and presence of clinical dental wear (mild-severe) on exam findings.

2.6. Statistical analysis

Analysis of variance (ANOVA) was performed to assess for statistically significant differences in probable sleep bruxism outcomes among the parent-reported and clinical findings. Significance for all statistical tests was predetermined at $p < 0.05$. Associations of tonsil size, TRMR, and nasal breathing to probable sleep bruxism were assessed by Chi-square test. All analyses were

done using JMP-14 (SAS Institute, Inc.) and p-value <0.05 was considered to be statistically significant.

2.7. Ethics

This study was approved by the Institutional Review Board of the University of California, Los Angeles (Protocol ID: 18–000810).

3. Results

96 participants enrolled in this study (mean age, 8.86 years; range, 6–12 years; 46 male, 50 female) were evaluated for the items described in the clinical exam tool. There were n = 47 subjects with no dental wear, n = 37 subjects with mild dental wear, and n = 12 subjects with moderate to severe dental wear. 23 participants were found to have probable sleep bruxism based on clinical reports as well as exam findings. SDSC scores were significantly elevated among children positive for PSB for whom the diagnostic criteria were present (PSB-positive: 45.1 ± 13.0 vs. PSB-negative: 34.8 ± 5.5; p < 0.0001). There were no significant effects of gender or age differences on outcome measures.

Parental reports of mouth breathing while awake, mouth breathing during sleep, snoring during sleep, and difficulty breathing and/or gasping for air during sleep were all associated with increased odds of probable sleep bruxism. In addition, difficulty with concentration/focus was also associated with increased odds of probable sleep bruxism, and stress/anxiety demonstrated a trend to significance. See Table 1.

Increased tonsil size (according to the Brodsky scale) was associated with a greater odds of probable sleep bruxism. Pearson ChiSquare (n = 96, df 3) = 20.0, p < 0.0001. See Fig. 1. Functional ankyloglossia (according to the TRMR scale) was associated with a greater odds of probable sleep bruxism. Pearson ChiSquare (n = 96, df 3) = 31.4, p < 0.0001. See Fig. 2. Inability to breathe through the nose for at least 3 min was associated with a greater odds of probable sleep bruxism. Pearson ChiSquare (n = 96, df 1) = 16.2, p < 0.0001. See Fig. 3. Multivariate analysis suggests that restricted tongue mobility was an independent risk factor for bruxism when controlling for nasal breathing and tonsil size. See Table 2.

Of the 96 participants, 25 had tonsil hypertrophy (Brodsky grade 3–4), 18 had restricted tongue mobility (TRMR grade 3–4), and 30 had nasal obstruction (Nasal Breathing Test <3 min). There were 58 participants without any of the above factors, 10 participants with one of the above exam findings, 17 participants with two of the above findings, and 11 participants with all three of the factors above. Although tonsil hypertrophy, restricted tongue mobility, and nasal obstruction were each found to be statistically significant for an association with probable sleep bruxism, there was a synergistic effect in the study among individuals who were affected by all three factors. The incidence of probable sleep

bruxism among individuals without any of exam findings of tonsil hypertrophy, restricted tongue mobility, and nasal obstruction was 5/58 (8.6%), whereas the incidence of probable sleep bruxism among individuals with all three exam findings was 10/11 (90.9%), p < 0.0001. See Fig. 4.

4. Discussion

Bruxism is a clinical risk factor for tooth wear, dental damage and fractures, jaw muscle fatigue, and pain [3]. Identifying the clinical signs of bruxism at an early age is crucial in order to prevent the pathologic consequences that may occur. While the pathophysiology of bruxism remains poorly understood, the prevalence of bruxism appears to be significantly increased in participants with sleep and breathing disorders [2,11]. Bruxism has been associated with fragmented sleep [12,13], arousals during sleep leading to increase in parafunctional activity [14,15], and activation of upper airway muscles as a compensation for narrowed airway passages [16]. Prior studies have shown there is a positive correlation between the burden of sleep-disordered breathing due to adenotonsillar hypertrophy with bruxism in children and significant improvement of bruxism after adenotonsillectomy [13]. Among adult individuals with obstructive sleep apnea, successful treatment of breathing abnormalities with CPAP has been shown to eliminate bruxism during sleep [17]. Nevertheless, there are many individuals without sleep-disordered breathing who still present with reports of teeth grinding and signs of moderate to severe signs of dental wear. Other known risk factors for pathologic bruxism include psychological or physical distress, in addition to non-breathing related sources of sleep disturbances [5].

In this study, parental report of psychological distress, mouth breathing while awake, mouth breathing while asleep, snoring, gasping for air and/or difficulty breathing were all associated with increased odds of probable sleep bruxism. In addition, clinical findings of tonsil hypertrophy, nasal obstruction, and restricted tongue mobility were found to have a synergistic association with the incidence of probable sleep bruxism. Individuals without tonsil hypertrophy, nasal obstruction, or restricted tongue mobility were unlikely to present with probable sleep bruxism (8.6%), whereas individuals with all three above factors were very likely to present with probable sleep bruxism (90.9%). It appears that in this population, breathing and tongue mobility were the major determinants of probable sleep bruxism, however, it is important to note that there were n = 5 individuals without any of these findings that still persisted with probable sleep bruxism likely attributable to other psychological or physical distress. On the other hand, there were n = 1 individual with all three factors that did not present with PSB; it is important to note that this individual had symptoms of anxiety/depression, snoring, and

Table 1
Subjective parental reports related to spectrum of breathing dysfunction and psychosocial distress: Association with probable sleep bruxism.

Prognostic Factor	Probable Sleep Bruxism		Univariate Analysis		
	Yes	No	Odds Ratio	95% Confidence Interval	P-value (Pearson's Chi Square)
Spectrum of Breathing Dysfunction					
Mouth Breathing while awake	70.0% (16/23)	37.0% (27/73)	3.9	1.4–10.7	p = 0.04*
Mouth Breathing during sleep	73.9% (17/23)	42.5% (31/73)	3.8	1.36–10.86	p = 0.009*
Snoring during sleep	78.3% (18/23)	37.0% (27/73)	6.1	2.0–18.4	p < 0.0001*
Difficulty breathing and/or gasping for air during sleep	39.1% (9/23)	6.8% (5/73)	8.7	2.5–30.0	p < 0.0001*
Psychosocial Distress					
Difficulty with concentration/focus	65.2% (15/23)	17.8% (13/73)	8.7	3.0–24.7	p = 0.0002*
Almost always feels stressed/anxious	8.7% (2/23)	1.4% (1/73)	6.9	0.6–79.4	p = 0.078

Probable sleep bruxism was associated with impaired nasal breathing, habitual mouth breathing, restricted tongue mobility, and tonsillar hypertrophy. The * (asterisk) denotes statistical significance with p-value threshold <0.05.

Incidence of Probable Sleep Bruxism by Tonsil Size

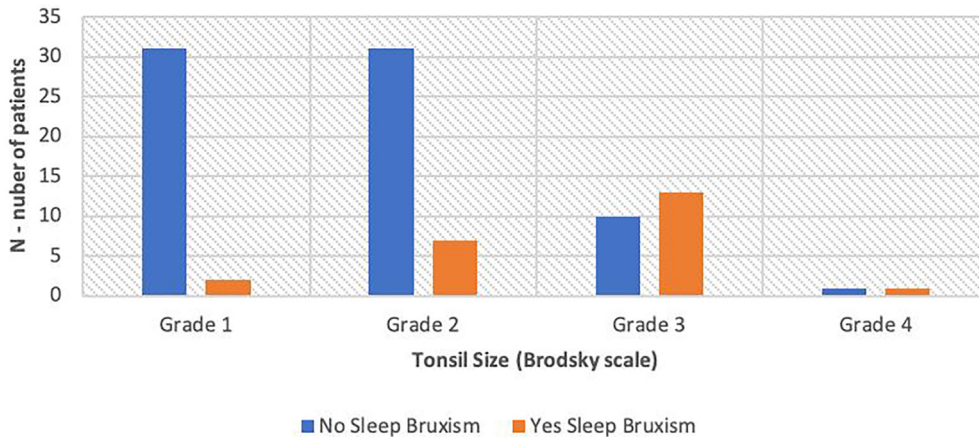


Fig. 1. Increased tonsil size (according to the Brodsky scale) is associated with a greater odds of probable sleep bruxism, Pearson Chi Square (n = 96, df 3) = 20.0, p < 0.0001.

Incidence of Probable Sleep Bruxism by TRMR

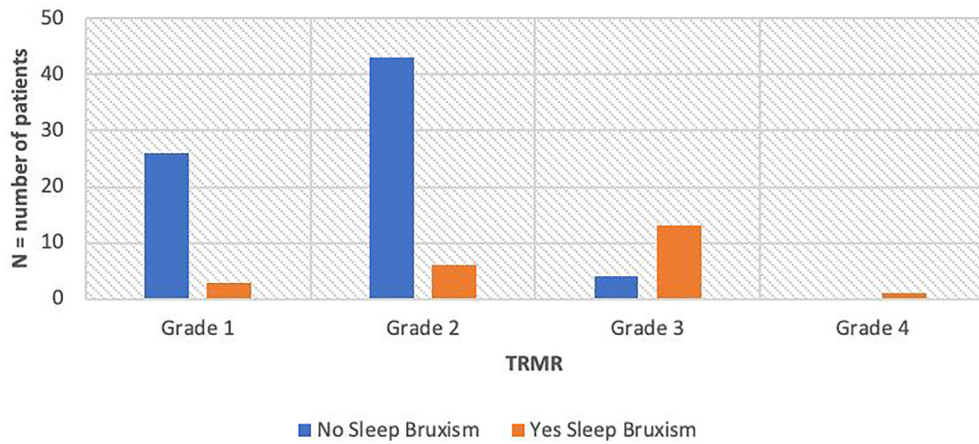


Fig. 2. Functional ankyloglossia (according to the TRMR scale) is associated with a greater odds of probable sleep bruxism, Pearson ChiSquare (n = 96, df 3) = 31.4, p < 0.0001.

Incidence of Probable Sleep Bruxism by Nasal Breathing Test

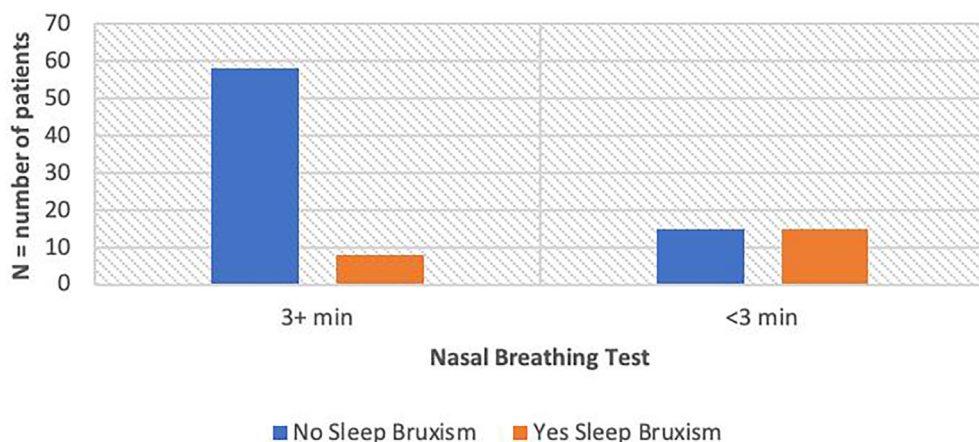


Fig. 3. Inability to breathe through the nose for at least 3 min is associated with a greater odds of probable sleep bruxism, Pearson ChiSquare (n = 96, df 1) = 16.2, p < 0.0001.

Table 2
Objective clinical factors related to PSB: Results of univariate and multivariate analysis.

Prognostic Factor	Probable Sleep Bruxism		Univariate Analysis			Multivariate Analysis		
	Yes	No	Odds Ratio	95% Confidence Interval	P-value (Pearson's Chi Square)	Odds Ratio	95% Confidence Interval	P-value (Pearson's Chi Square)
Ankyloglossia: TRMR Grade 3+	56.5% (13/23)	6.8% (5/73)	21.2	6.2–72.8	p < 0.0001 *	10.6	2.8–40.7	p = 0.0006 *
Nasal Breathing Test: Unable to breathe through nose for >3 min at time of exam	65.2% (15/23)	20.5% (15/73)	7.3	2.6–20.3	p < 0.0001 *	2.1	0.5–8.5	p = 0.2915
Tonsil Size: Brodsky Grade 3+	60.9% (14/23)	15.1% (11/73)	8.8	3.1–25.2	p < 0.0001 *	2.7	0.7–11.2	p = 0.1618

Probable sleep bruxism was associated with impaired nasal breathing, habitual mouth breathing, restricted tongue mobility, and tonsillar hypertrophy. The * (asterisk) denotes statistical significance with p-value threshold <0.05.

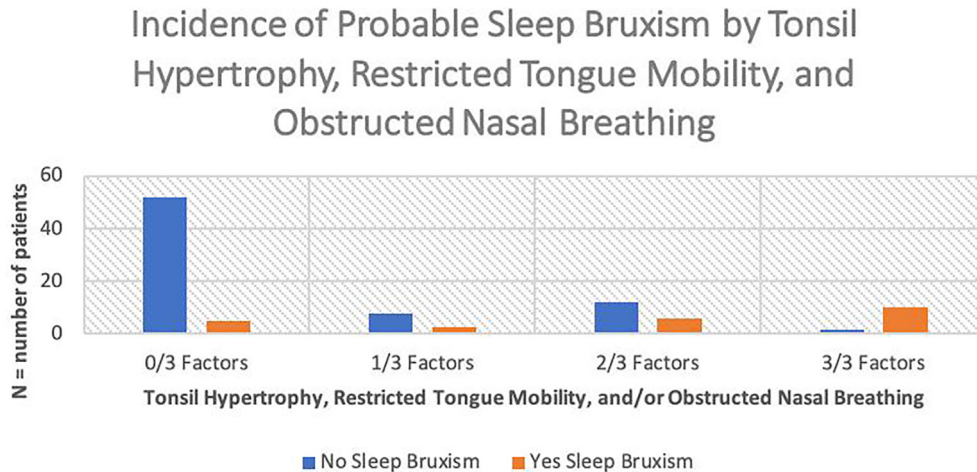


Fig. 4. There were 5/58 patients with 0/3 factors with PSB, as compared to 10/11 patients with 3/3 factors with PSB (p < 0.001) indicating a compound and synergistic effect between tonsil size, tongue mobility, and nasal breathing on PSB.

poor sleep despite the absence of parental recognition and physical exam findings of PSB.

4.1. Tonsillar hypertrophy

This study shows tonsillar hypertrophy with a Brodsky scale grade 3+ to have a statistically significant association to increased odds of moderate-severe signs of probable sleep bruxism (Table 2, Fig. 1). Previous studies have shown a significantly higher incidence of bruxism occurring in children with tonsil hypertrophy [18]. In a study comparing the incidence of bruxism before and after adenotonsillectomy, it was found that 45.6% of the individuals studied that presented with tonsillar hyperplasia and sleep disordered breathing from 2 to 12 years old presented with bruxism. Three months after adenotonsillectomy, just 11.8% of those individuals presented with bruxism [10]. Children with airway obstruction pull the jaw forward to mechanically maintain better airway patency. This may stimulate receptors in the upper airway to increased airway tone, leading to bruxism [16]. This draws researchers to conclude that the correction of the airway obstruction decreases the arousals during sleep, leading to a decrease in the parafunctional activity of bruxism.

4.2. TRMR

The results of this study showed a statistically significant association between ankyloglossia (TRMR: Grade 3+) and increased odds of PSB (Table 2, Fig. 2). We hypothesize that ankyloglossia may lead to altered resting oral posture (low tongue position) contributing to oral myofascial dysfunction [19].

4.3. Impaired nasal breathing

Mouth breathing while asleep as well as mouth breathing while awake were significantly associated with bruxism in our study. Bruxism is a common complaint of parents of mouth-breathing children [13]. Previous studies have explained the association of mouth breathing to bruxism by the theory that bruxism increases salivary excretion for oral lubrication and the esophagus, and excess of saliva may drain out of the mouth that would be open, based on mouth breathing [20,21]. Another explanation may be that mouth breathing interferes with the sleep cycle and affects cerebral oxygenation, bringing about somniloquy and involuntary muscle contractions of the facial muscles, triggering sleep bruxism [21,22]. Mouth breathing leading to oral dryness has also been associated with increased dental wear [23].

This study shows impaired nasal breathing statistically significantly associated with increased odds of PSB (Table 2, Fig. 3). In a previous study of children diagnosed with nasal obstruction from 2 to 13 years of age, 65.2% of those individuals presented with bruxism [24]. Intragroup analysis revealed a prevalence of allergic rhinitis associated with other airway diseases in the group with bruxism, confirming the fact that allergic children are more predisposed to bruxism than non-allergic children [25]. Hypotheses related to the occurrence of bruxism amongst individuals with nasal obstructions may include: altered saliva flow, changes to pressure in the eustachian tube, and altered resting oral posture (low tongue position, lips apart, mouth breathing) contributing to oral myofascial dysfunction [24].

4.4. Spectrum of breathing dysfunction

This study shows snoring, gasping for air and/or difficulty breathing are all associated with increased odds of PSB (Table 1). Individuals with sleep bruxism have reported a 2–3 times higher prevalence of obstructive sleep apnea [26]. A significant correlation between sleep bruxism and snoring has been supported by the previous studies in children [27,28]. Although studies have suggested the concomitant occurrence of tooth grinding in individuals with OSA, a limited upper airway, rather than upper airway obstruction, could be a factor contributing to the relationship between snoring and SB in children and adolescents [29]. OSA and its relationship to sleep bruxism is related to an arousal response that is often provoked by hypoxemia and breathing difficulty [26]. The termination of the apneic event is often accompanied with a variety of oral phenomena such as snoring, gasping, mumbling, and tooth grinding [26,30,31]. Sleep-related problems are commonly reported in 25–40% of preschoolers and school children (eg insomnia, obstructive sleep apnea (OSA), restless legs syndrome (RLS), somniloquy, enuresis, rhythmic movements, and disorders of arousal) [32,33]. It is imperative to recognize these pathologies early, as children with sleep bruxism can have a high likelihood of showing problematic daytime behavior which can also be frequently associated with sleep problems [27,32,34]. This can therefore impair children's learning ability throughout the day and effectiveness to perform well in school.

4.5. Psychological distress

This study shows parental reports of the child's psychological distress as significantly associated with bruxism (Table 1). Previous studies have shown that individuals with bruxism had elevated levels of catecholamines in their urine in comparison to non-bruxism participants; such findings support a link between emotional stress and bruxism [35–37]. Elevations of catecholamines may explain the body's response to stress, as the sympathetic nervous system is activated, therefore elevating catecholamine levels of the body [38]. In addition, studies show individuals report that they clench their teeth in periods of intense or frequent familial duties or increased workload [7]. Children may also learn to clench by watching a parent react to stress, anger or frustration, therefore mimicking their behaviors [7,19].

Recommendations in previous studies have identified bruxism as a clinical risk factor in recognizing psychological or psychiatric disorders, such as severe or pathological anxiety, mood, and personality disorders [7]. The expertise of a psychologist may be useful in those cases and dentists may be integral in providing referral services to facilitate patient treatment and education. In addition, sleep hygiene measures aim to reduce any influence of psychological stress on sleep bruxism [11]. This includes avoidance of caffeine close to bedtime, keeping the bedroom well-ventilated and quiet, relaxing close to bedtime, and relaxation techniques before sleep [39].

The results of this study suggest that altered resting oral posture (low tongue position due to mouth breathing and/or ankyloglossia) may predispose to bruxism in the pediatric dental population. Dentists are encouraged to look for signs of psychosocial stress, nasal obstruction, mouth breathing, tonsillar hypertrophy, and restricted tongue mobility among individuals with moderate to severe dental wear prior to restorative work as a potential underlying cause of the individual's recurrent poor dentition. Future studies from our group will investigate methods to reliably measure low tongue position: ranging from the whole tongue resting high in the roof of the mouth to the whole tongue lying low in the floor of mouth, and variations in between.

Limitations include the single institute study, practitioner experience, and limited sample size. Also, our assessment for sleep bruxism is limited by classification on parent-report and clinical inspection only, as 'definite' sleep bruxism requires audio/video or polysomnographic recordings. Future research designs would include a larger sample size, polysomnographic findings, and preferably combine ecological momentary assessment/experience sampling methodology (EMA/ESM).

5. Conclusion

In our study, probable sleep bruxism was found to be significantly associated with impaired nasal breathing, habitual mouth breathing, restricted tongue mobility, and/or tonsillar hypertrophy. Restricted tongue mobility was found to be an independent risk factor for bruxism among individuals with normal nasal breathing. This study further shows that tonsillar hypertrophy, restricted tongue mobility, and nasal obstruction may have a synergistic association on the presentation of PSB.

Dentists should evaluate for tonsillar hypertrophy, restricted tongue mobility, and nasal obstruction in the evaluation of PSB, as these exam findings are highly prevalent in the majority of cases.

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Conflict of interest

The authors declare they have no conflicts of interest.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <https://doi.org/10.1016/j.sleep.2020.11.007>.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.sleep.2020.11.007>.

References

- [1] Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: an international consensus. *J Oral Rehabil* 2013;40(1):2–4. <https://doi.org/10.1111/joor.12011>.
- [2] Manfredini D, Restrepo C, Diaz-Serrano K, et al. Prevalence of sleep bruxism in children: a systematic review of the literature. *J Oral Rehabil* 2013;40(8): 631–42.
- [3] Carra MC, Huynh N, Morton P, et al. Prevalence and risk factors of sleep bruxism and wake-time tooth clenching in a 7-to 17-yr-old population. *Eur J Oral Sci* 2011;119(5):386–94.
- [4] Kleinberg I. Bruxism: aetiology, clinical signs and symptoms. *Aust Prosthodont J* 1994;8:9.
- [5] Kuhn M, Türp JC. Risk factors for bruxism. *Swiss Dent J* 2018;128(2):118–24.
- [6] Restrepo C, Gómez S, Manrique R. Treatment of bruxism in children: a systematic review. *Quintessence Int (Berl)* 2009;40(10).
- [7] Lavigne GJ, Khoury S, Abe S, et al. Bruxism physiology and pathology: an overview for clinicians. *J Oral Rehabil* 2008;35(7):476–94.
- [8] Brodsky L. Modern assessment of tonsils and adenoids. *Pediatr Clin North Am* 1989;36(6):1551–69.
- [9] Yoon A, Zaghi S, Weitzman R, et al. Toward a functional definition of ankyloglossia: validating current grading scales for lingual frenulum length and tongue mobility in 1052 subjects. *Sleep Breath* 2017;21(3):767–75.

- [10] Zaghi S, Peterson C, Shamtoob S, et al. Assessment of nasal breathing using lip taping: a simple and effective screening tool. *Int J Otorhinolaryngol* 2020;6(1):10.
- [11] Beddis H, Pemberton M, Davies S. Sleep bruxism: an overview for clinicians. *Br Dent J* 2018;225(6):497–501.
- [12] Macaluso GM, Guerra P, Di Giovanni G, et al. Sleep bruxism is a disorder related to periodic arousals during sleep. *J Dent Res* 1998;77(4):565–73.
- [13] DiFrancesco RC, Junqueira PAS, Trezza PM, et al. Improvement of bruxism after T & A surgery. *Int J Pediatr Otorhinolaryngol* 2004;68(4):441–5.
- [14] Kato T, Rompre P, Montplaisir JY, et al. Sleep bruxism: an oromotor activity secondary to micro-arousal. *J Dent Res* 2001;80(10):1940–4.
- [15] Phillips BA, Okeson J, Paesani D, et al. Effect of sleep position on sleep apnea and parafunctional activity. *Chest* 1986;90(3):424–9.
- [16] Greenfeld M, Tauman R, DeRowe A, et al. Obstructive sleep apnea syndrome due to adenotonsillar hypertrophy in infants. *Int J Pediatr Otorhinolaryngol* 2003;67(10):1055–60.
- [17] Oksenberg A, Arons E. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. *Sleep Med* 2002;3(6):513–5.
- [18] Valera FCP, Travitzki LVV, Mattar SEM, et al. Muscular, functional and orthodontic changes in pre school children with enlarged adenoids and tonsils. *Int J Pediatr Otorhinolaryngol* 2003;67(7):761–70.
- [19] Yoon AJ, Zaghi S, Ha S, et al. Ankyloglossia as a risk factor for maxillary hypoplasia and soft palate elongation: a functional–morphological study. *Orthod Craniofac Res* 2017;20(4):237–44.
- [20] Thie NM, Lavigne GJ. Bruxism and orofacial movements during sleep. *Dent Clin North Am* 2001;45(4):657.
- [21] Serra-Negra JM, Ribeiro MB, Prado IM, et al. Association between possible sleep bruxism and sleep characteristics in children. *CRANIO®* 2017;35(5):315–20.
- [22] Camargo EP, Carvalho LBC, Prado LBF, et al. Is the population properly informed about sleep disorders? *Arq Neuropsiquiatr* 2013;71(2):92–9.
- [23] Wetselaar P, Manfredini D, Ahlberg J, et al. Associations between tooth wear and dental sleep disorders: a narrative overview. *J Oral Rehabil* 2019;46(8):765–75.
- [24] Grechi TH, Trawitzki LV V, de Felício CM, et al. Bruxism in children with nasal obstruction. *Int J Pediatr Otorhinolaryngol* 2008;72(3):391–6.
- [25] Seraj B, Shahrabi M, Ghadimi S, et al. The prevalence of Bruxism and correlated factors in children referred to dental schools of Tehran, based on parent's report. *Iran J Pediatr* 2010;20(2):174.
- [26] Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. *Chest* 2001;119(1):53–61.
- [27] Ng DKK, Kwok KL, Poon G, et al. Habitual snoring and sleep bruxism in a paediatric outpatient population in Hong Kong. *Singapore Med J* 2002;43(11):554–6.
- [28] Ersu R, Arman AR, Save D, et al. Prevalence of snoring and symptoms of sleep-disordered breathing in primary school children in Istanbul. *Chest* 2004;126(1):19–24.
- [29] Carra MC, Huynh N, Lavigne G. Sleep bruxism: a comprehensive overview for the dental clinician interested in sleep medicine. *Dent Clin* 2012;56(2):387–413.
- [30] Okeson JP, Phillips BA, Berry DTR, et al. Nocturnal bruxism events in subjects with sleep-disordered breathing and control subjects. *J Craniomandib Disord* 1991;5(4).
- [31] Saito M, Yamaguchi T, Mikami S, et al. Temporal association between sleep apnea–hypopnea and sleep bruxism events. *J Sleep Res* 2014;23(2):196–203.
- [32] Tachibana M, Kato T, Kato-Nishimura K, et al. Associations of sleep bruxism with age, sleep apnea, and daytime problematic behaviors in children. *Oral Dis* 2016;22(6):557–65.
- [33] Owens JA, Witmans M. Sleep problems. *Curr Probl Pediatr Adolesc Health Care* 2004;4(34):154–79.
- [34] Laberge L, Tremblay RE, Vitaro F, et al. Development of parasomnias from childhood to early adolescence. *Pediatrics* 2000;106(1):67–74.
- [35] Lavigne GJ, Kato T, Kolta A, et al. Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med* 2003;14(1):30–46.
- [36] Clark GT, Rugh JD, Handelman SL. Nocturnal masseter muscle activity and urinary catecholamine levels in bruxers. *J Dent Res* 1980;59(10):1571–6.
- [37] Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. *J Orofac Pain* 2009;23(2):153–66.
- [38] Vandas AP, Menenakou M, Kouimtzi TH, et al. Urinary catecholamine levels and bruxism in children. *J Oral Rehabil* 1999;26(2):103–10.
- [39] Valiente López M, van Selms MKA, Van Der Zaag J, et al. Do sleep hygiene measures and progressive muscle relaxation influence sleep bruxism? Report of a randomised controlled trial. *J Oral Rehabil* 2015;42(4):259–65.