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Original Article

Prevalence and risks of sleep bruxism in children and adolescents presenting for orthodontic treatment

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Abstract

Objectives: This study determined the prevalence and risks of definite sleep bruxism (SB) among children and adolescents presenting for orthodontic treatment.

Methods: This was a cross-sectional study of 7–16-year-old subjects pursuing orthodontic treatment for the first time. The presence or absence of SB was determined using an overnight mandibular movement monitoring inertial measurement sensor, worn by each participant for two consecutive nights. Data from the sensor were extrapolated, then processed and analyzed to automatically identify rhythmic masticatory muscle activity for SB assessment. SB risks were evaluated from previously validated questionnaires, clinical examinations, lateral cephalometric radiographs, and digital study models.

Results: A total of 87 subjects with a mean age of 12.82 years ± 2.24 and body mass index of 21.45 ± 5.49 participated in the study. The prevalence of SB was 60.7%. Multiple linear regression analysis revealed that SB had statistically significant association with microarousals (events/h) (β=0.31, 95% Confidence Interval [CI] 0.25–0.36, $P < .001$) and maxillary 6–6 dimension (mm) (β = 0.08, 95% CI 0.02–0.13, $P = .008$). A second model excluding microarousals showed that SB had a statistically significant association with sleep efficiency (SE) percentage (β = −0.15, 95% CI −0.28 to −0.01, $P = .026$) and obstructive respiratory disturbance index (ORDI) (events/h) (β = 0.33, 95% CI 0.15–0.51, $P < .001$).

Conclusions and implications: In a growing orthodontic population, definite SB is very common. SB is related to microarousals, maxillary intermolar width, SE percentage, and ORDI.

Keywords: sleep bruxism; mandibular movement; adolescent

Introduction

The 2017 International Consensus on the Assessment of Bruxism defined sleep bruxism (SB) as a repetitive masticatory muscle activity (MMA) that occurs during sleep and is characterized as rhythmic (phasic) or non-rhythmic (tonic) [1]. Rhythmic MMA (RMMA) is a 1 Hz slow chewing like movement with tooth grinding, occurring in 60% of healthy individuals [2]. In subjects with SB, RMMA is three times more frequent and 30% more intense compared to normal subjects [2]. Non-rhythmic MMA (tonic) refers to SB episodes that are sustained for a period of time (more than 2 s) such as those seen in clenching [3]. Non-rhythmic MMA represents 4.8% of all oromandibular events in normal subjects and 7.2% of the same events in sleep bruxers [2].

The prevalence of SB in the pediatric population varies widely, ranging from 9.1% to 67.3% [4, 5]. No significant gender differences have been observed, and the prevalence shows a general decreasing trend with age [6]. Factors that contribute to the wide prevalence range include the multiple definitions of SB, diagnostic criteria [6] and, to a lesser extent, the difficulty of distinguishing clinical signs of sleep and awake bruxism [7].

Pediatric SB, classified as a muscle activity behavior, is often self-limiting and may not always have negative consequences. However, intervention becomes essential when excessive forces during bruxism episodes lead to significant intraoral health problems. These forces can contribute to headaches, muscle pain, temporomandibular joint (TMJ) dysfunction, and premature tooth loss [8]. Beyond oral health, SB can also impact overall health by disrupting sleep patterns and adversely affecting overall quality of life [8, 9].

Multiple factors have been implicated in the occurrence of SB. These include genetic polymorphisms [10, 11], stress and feelings of anxiousness [12, 13], craniofacial morphology [14], variations in head posture [15], prolonged screen time, and excessive sugar consumption [16].

Type I sleep laboratory polysomnography (PSG), complemented by audio-video recordings and surface electromyographic channels to monitor MMA, is widely regarded as the gold standard for diagnosing SB [17]. However, this tool has

significant limitations regarding accessibility, affordability, and convenience, relying heavily on the quality of audio-video equipment [18]. These limitations are especially applicable to a pediatric population.

A novel, noninvasive alternative to PSG for detecting SB utilizes mandibular movement (MM) monitoring during sleep through a lightweight sensor placed on the chin [19]. This approach allows data collection in a home setting, with MM signals transmitted via a smartphone application to a cloud-based platform. The data are then processed and analyzed using advanced machine learning algorithms to automatically detect stereotypical (normal) MM and identify RMMA, enabling accurate SB assessment [20].

MM represents muscular activity of the jaw antagonists which are innervated by the trigeminal nerve and are driven by the respiratory centers in the brain stem [21]. MM monitoring with sensors has been validated against PSG in pediatric populations [22].

Evidence on SB in pediatric populations remains limited, relying predominantly on self-reported data. Early diagnosis of pediatric SB is critical, as it may prevent progression into adulthood and mitigate its associated negative consequences. To gain a deeper understanding of the prevalence and associated factors for this condition, further studies employing precise diagnostic tools are essential. The goal of this cross-sectional study was to determine the prevalence and risks of definite SB among children and adolescents presenting for orthodontic treatment through monitoring MM during sleep.

Materials and methods

Study design

This cross-sectional study evaluated the prevalence and risks of definite SB in adolescents and children presenting for orthodontic treatment at the University at Buffalo (UB) Post-Graduate Orthodontic Clinic. This study was conducted between June 2021 and April 2023 after obtaining approval by the UB Institutional Review Board (#00005013).

Study population

The sample inclusion criteria were males and females with an age range of 7–16 years, good general health, any skeletal and dental relationship, ability to read and write in English, and compliance with sensor use. Subjects were excluded if they had a history of previous orthodontic treatment or orthognathic surgery, history of trauma to the face and/or jaws, history of adenotonsillectomy, craniofacial syndromes/anomalies, or medical and behavioral conditions that require special treatment considerations such as cognitive disorders.

Sample size

Based on an SB prevalence of 32.3% [23], margin of error of 8%, and 90% confidence level, a minimum sample of 93 subjects was estimated.

Study procedures

Subjects who met the eligibility criteria were invited to participate in the study during their orthodontic screening appointment and written informed consent/assent were obtained from each subject and parent/guardian.

Data on the presence of SB was collected by one investigator using a wireless sensor and data related to the risks of SB were obtained using self-administered questionnaires as well as clinical and radiographic examinations.

Sleep bruxism assessment

SB was determined by overnight automated MM monitoring with the use of a disposable self-adhesive inertial measurement sensor (Sunrise®, Namur, Belgium) [19, 22]. Detailed instructions on sensor use were given to each participant and parent/guardian.

Before going to sleep at night, each participant placed the sensor on the chin in the mentolabial sulcus area. The sensor utilized a mobile application and transferred all recorded information via Bluetooth to a cloud-based platform. Data were extrapolated from the sensor, processed, and analyzed using a trained and validated machine learning algorithm (Python programming) [19] which automatically identifies RMMA for SB assessment. To identify RMMA, the algorithm looks for and identifies cyclical movements at a specific frequency and amplitude that are characteristic of SB [24].

The sensor exclusively identifies RMMA and is unable to detect non-rhythmic MMA, as the sensor requires MM to classify the activity. To minimize the night-to-night variability of MMA events, participants were instructed to repeat MM monitoring for two consecutive nights. Then the night with the largest RMMA index (events/h) was selected for the prevalence of SB. SB was recorded as mean number of events during a sleep night.

Additionally, the following objective sleep variables were assessed: total sleep time (TST; h); wake duration after sleep onset (WASO; min); sleep efficiency (SE; %); rapid eye movement sleep (REM% of TST); light sleep referring to stages 1 and 2 of non-REM sleep (% of TST); deep sleep referring to stage 3 of non-REM sleep (% of TST); microarousal index (events/h); obstructive respiratory disturbance index (ORDI; events/h); apnea–hypopnea index (AHI; events/h); respiratory effort (RE; % of TST); respiratory effort-related arousals index (RERA; events/h); ORDI in REM sleep (events/h); ORDI in NREM sleep (events/h); ORDI in supine position (events/h); ORDI in non-supine position (events/h); TST in supine position (% of TST); and TST in non-supine position (% of TST).

Sleep bruxism risks

SB risks were examined using questionnaires, drawing upon previous literature on the etiology and pathophysiology of SB. The questionnaires were administered using Research Electronic Data Capture for each subject and parent during their initial records appointment.

Parents/guardians completed a modified abbreviated Children's Sleep Habit Questionnaire (CSHQ-A) [25]. The 22-item CSHQ-A assesses sleep behavior, daytime sleepiness, and bedtime habits over a 'typical' recent week. Questions were answered by selecting one of the following: always (occurs every night for your child), usually (occurs 5–6 times a week), sometimes (occurs 2–4 times a week), rarely (occurs once a week), or never (occurs less than once a week). Responses were given a numerical value (always = 5, usually = 4, sometimes = 3, rarely = 2, and never = 1). Questions #1, 2, 3, 10, and 19 were reversed scored. A cumulative score was then calculated with a relative cutoff point of 41. Higher scores indicated the subject had sleep problems.

The validated Revised Children's Anxiety and Depression Scale for subjects (RCADS) and their parents (RCADS-P) [26] was then administered. The 25-item questionnaire assesses the subjects' generalized and separation anxiety disorders, panic disorder, mood disorder, obsessive-compulsive disorder, and social phobias. To complete the RCADS questionnaire, the subject indicated the frequency of events by selecting one of the following options: never, sometimes, often, or always. Similarly, for the RCADS-P questionnaire, the parent provided responses based on the frequency of events using the same options [26]. To analyze the responses from both questionnaires, scores were assigned on a scale from 0 (never) to 3 (always). These numerical values were subsequently entered into the University of California, Los Angeles's RCADS-25 Child Version Scoring Program and RCADS-25 Parent Version Scoring Program [27] to calculate the total depression and anxiety scores based on the child and parent responses. Cumulative scores < 65 were considered below clinic threshold, scores > 65 were borderline, and scores > 70 were above clinical threshold [27]. Overall, higher scores implied symptoms of substantial anxiety and depression.

Demographic variables were also collected from the subjects including sex, age in years, height in inches, weight in pounds, and academic performance over the past year (very good [A student], good [B student], okay [C student], poor [D student], and very poor [E or F student]). In addition, subjects were asked if they grind their teeth, if they feel like their teeth are worn down, if they have pain or soreness in the muscles of their upper and lower jaws, if they have pain in the temples of their head when they wake up, if they have difficulty opening their mouth when they wake up, and lastly, if they ever wake up grasping for air. Additionally, parents/guardians answered a series of questions regarding their child's race/ethnicity following the National Institute of Health guidelines [28], household income (a yearly income below $60,070 is considered as being in poverty) [29], parental education level, marital status, child's medical and dental history, medications, and child's acidic food and drink consumption [30].

One investigator who was blinded to the participants' questionnaire responses conducted clinical examinations to evaluate the Friedman tongue position classification [31] as well as the TMJs and masticatory muscles, following the guidelines of Pertes and Gross [32]. Tooth wear was measured using the Basic Erosive Wear Examination Index [33], which assesses six sextants in the maxillary and mandibular arches. Each sextant was scored on a four-level scale (0, 1, 2, 3, and 4) based on the most severely eroded or worn surface [33]. A cumulative score was then assigned: a score of less than 2 indicated 'no risk', 3–8 indicated 'low risk', 9–13 indicated 'medium risk', and above 14 indicated 'high risk' [33]. In addition, clinical evaluations were made for Angle molar classification, the presence of anterior and posterior cross-bites, facial asymmetry, and any functional shifts.

Lateral cephalometric radiographs were taken to assess the craniofacial morphology and dentoalveolar tooth positions. The cephalograms were traced and analyzed for one soft tissue (nasolabial angle), nine skeletal (SNA, SNB, ANB, maxillary length, mandibular length, gonial angle, lower facial height, Y-axis, and SN-GoGn), and six dentoalveolar variables (interincisal angle, IMPA, U1-SN, L1-Apo, overbite, and overjet). Digital study models were also utilized to measure the maxillary and mandibular transverse inter-first molar dimensions (6–6 width in mm) and tooth size-arch length discrepancy.

Intra-examiner reliability

Around 18% of subjects and their parents repeated the questionnaires more than 2 weeks after initially completing them. Rater reliability was examined with Cohen's kappa.

Statistical analysis

Analyses were performed using RStudio 2022.02.1+461 with R version 4.2.2 and all testing was assessed at the 5% level for significance. All categorical and continuous variables were individually analyzed using simple linear regression with SB as the dependent variable. To control the false discovery rate, multiple testing adjustments were performed using the Benjamini-Hochberg method. Additionally, the RCADS completed by the subjects and their parents/guardians had the scores separately linearly regressed against the subjects' SB values. Two multiple linear regression models were constructed with SB as the response variable: one that included microarousal as a candidate variable, and another that excluded it. Variable selection for the final models was performed using forward selection based on *P*-value, with a focus on interpretability. The maximum number of covariates was restricted due to sample size. In addition, an alternative modeling approach was applied for the response variable to further explore the data. In this approach, SB was converted into a three-level categorical variable: 'no SB' (RMMA < 2), 'mild SB' (2 ≤ RMMA < 6), and 'severe SB' (RMMA ≥ 6). This three-level factor was modeled using ordinal regression analysis. Again, two models were constructed: one that included microarousal and another that did not, following the same procedure used in the linear regression analysis.

Results

Reliability

Cohen's kappa revealed that the parent responses on the questionnaires had strong reliability (kappa = 0.65). Subject responses also showed strong reliability (kappa = 0.66).

Descriptive statistics

A total of 176 patients were screened for inclusion in this study and 87 agreed to participate. The other 89 subjects either failed to meet the inclusion criteria or refused to participate. Overall, the subjects' mean age was 12.82 ± 2.24 years, and mean body mass index (BMI) was 21.45 ± 5.50 indicating normal weight (Table 1).

Parent-reported medical history revealed that 34 subjects (39%) had allergies and 14 (16%) took medications, 19 (22%) had a family history of obstructive sleep apnea (OSA), 11 (13%) had asthma, 2 (2%) had gastroesophageal reflux disease (GERD), 5 (6%) had middle ear infections, 2 (2%) had psychiatric illness, and 7 (8%) had psychological illness.

Subject dietary consumption showed that 58 (67%) consumed citrus fruits, 25 (29%) consumed coffee or tea, 20 (23%) consumed mineral water, 13 (15%) consumed other acidic foods/drinks, 55 (63%) consumed soft drinks, and 51 (59%) consumed sports drinks.

Overall, 17 (19.5%) subjects reported clenching/grinding of teeth during sleep, 18 (20.7%) felt that their teeth were more worn than they should be, 14 (16.1%) experienced soreness or pain in the muscles of their upper and lower jaws, 7

(8%) reported pain in the temples upon waking up, 4 (4.6%) reported difficulty opening their mouth when waking up, and 3 (3.4%) reported waking up grasping for air.

Out of the 87 subjects, only 56 wore the sensors for two nights and had a TST greater than 4 h. The prevalence of SB as determined by the MM sensor was 60.7% with a mean number of events per hour of 4.59 and mean number of events per TST of 31.22.

Univariate analysis of SB risks

A statistically significant association was initially found between subjects' academic performance and SB ($P = .030$) (Table 2). After adjusting for multiple comparisons, this association was no longer significant ($P = .330$).

Table 3 illustrates self-reported SB symptoms among the subjects, revealing a statistically significant relationship between self-reported clenching and grinding during sleep and

Table 1. Mean age, height, weight, and BMI for subjects ($n = 87$).

Variable	Min	Max	Median	IQR	Mean	SD
Chronological age (years)	7.67	16	13.33	2.83	12.82	2.24
Stature (in)	48	75	61	6.50	60.93	5.71
Weight (lbs)	35	229	115	40	114.19	34.08
BMI	10.46	43.26	20.48	5.99	21.45	5.49
SB index per hour*	0.94	11.39	4.29	1.67	4.59	2.13
SB per TST*	4.98	85.97	29.97	16.77	31.22	16.36

*Based on 56 observations; BMI= body mass index; SB = sleep bruxism; TST = total sleep time.

Table 2. Demographic characteristics of subjects and parents.

Variable	Total	Beta	95% CI	*P*-value *	Adj. *P*-value**
	N (%)				
All	$n = 56$				
Subject					
Sex					
Male	23 (41)	(ref)	–		
Female	33 (59)	−0.05	(−1.22, 1.12)	.937	.937
Race/ethnicity					
White	35 (62)	(ref)	–		
Non-White	19 (34)	1.08	(−0.12, 2.29)	.076	.383
Prefer not to answer	2 (4)	0.26	(−2.81, 3.33)	.866	.937
Skeletal age					
Postpubertal	18 (32)	(ref)	–		
Circumpubertal	21 (38)	−1.10	(−2.48, 0.28)	.117	.383
Prepubertal	17 (30)	−0.92	(−2.35, 0.51)	.204	.383
Academic performance					
Poor (C or below student)	13 (24)	(ref)	–		
Average (B student)	12 (21)	1.29	(−0.51, 3.09)	.156	.383
Good (A student)	31 (55)	1.96	(0.20, 3.72)	.030	.330
Parent					
Education level					
College or higher	35 (62)	(ref)	–		
High school or less	21 (38)	0.08	(−1.11, 1.26)	.898	.937
Marital status					
Married or common law partner	39 (70)	(ref)	–		
Never married, divorced, widow, or separated	17 (30)	0.78	(−0.45, 2.01)	.209	.383
Yearly household income					
$82 621 or above	19 (34)	(ref)	-		
$57 424 to <$86 621	19 (34)	0.60	(−0.79, 1.98)	.392	.616
$23 828 to <$57 424	18 (33)	−0.35	(−1.75, 1.06)	.624	.858

*Simple linear regression *P*-values; Significance was set at $P < .05$.
**Multiple testing adjusted *P*-value by Benjamini–Hochberg method to control false discovery rate.

SB (β = 2.27, 95% Confidence Interval [CI] 1.01–3.53, adjusted $P = .003$).

Table 4 presents the responses of the RCADS. For both parent/guardian and child's responses, there were no significant associations between SB and anxiety, depression, and combined anxiety and depression.

Clinical and cephalometric variables can be seen in Table 5. No statistically significant associations were observed for any variable.

Table 6 illustrates the mean sleep variables and their correlation with SB. Among all the sleep variables examined, the ones demonstrating a significant association with SB were SE%, microarousal index, ORDI based on all events, ORDI related to non-REM sleep, and ORDI related to non-supine position (adjusted $P < .05$).

Multiple regression analysis of SB risk

Two linear regression models were constructed to identify the risks of SB where the response variable was the continuous RMMA values (Table 7). The first model showed that microarousals index (events/h) (β = 0.31, 95% CI 0.25–0.36, $P < .001$) and maxillary 6–6 dimension (mm) (β = 0.08, 95% CI 0.02–0.13, $P = .008$) were significantly associated with SB. The second model excluded microarousals, and revealed that SE% (β = –0.15, 95% CI –0.28 to –0.01, $P = .026$) and ORDI based on all events (events/h) (β = 0.33, 95% CI 0.15–0.51, $P < .001$) are independent risks of SB.

The variables identified as significant in the linear regression models also demonstrated significance in the ordinal regression models (Table 8). In the first model, microarousal index (β = 1.73, 95% CI 1.37–2.37, $P < .001$) and maxillary 6–6 dimension (β = 1.31, 95% CI 1.04–1.69, $P = .025$) were

Table 3. Self-reported SB for subjects.

Variable	Total	Beta	95% CI	*P*-value *	Adj. *P*-value**
	N (%)				
All	*n* = 56				
Do you clench/grind your teeth when you sleep?					
No	44 (79)	(ref)	–		
Yes	12 (21)	2.27	(1.01, 3.53)	<0.001	0.003
Do you feel as though your teeth are more worn than they should be?					
No	45 (80)	(ref)	–		
Yes	11 (20)	–0.33	(–1.78, 1.11)	0.647	0.647
Do you feel tiredness/soreness or pain in the muscles of your upper and lower jaws when you wake up?					
No	47 (84)	(ref)	–		
Yes	9 (16)	0.72	(–0.84, 2.27)	0.359	0.647
Do you feel pain in the temples (sides of the head, above the ears) when you wake up?					
No	50 (89)	(ref)	–		
Yes	6 (11)	0.61	(–1.24, 2.47)	0.509	0.647
Do you have difficulty opening your mouth when you wake up?					
No	53 (95)	(ref)	–		
Yes	3 (5)	–0.68	(–3.23, 1.87)	0.594	0.647
Do you ever wake up gasping for air?					
No	55 (98)	(ref)	–		
Yes	1 (2)	NA	NA	NA	NA

*Simple linear regression *P*-values; Significance was set at $P < .05$.
**Multiple testing adjusted *P*-value by Benjamini–Hochberg method to control false discovery rate.

Table 4. Revised children's anxiety and depression scale completed by subjects (RCADS-C) and parents/guardians (RCADS-P), *n* = 87.

	Subject		Parent/Guardian	
Level	Beta	*P*-value*	Beta	*P*-value*
RCADS child/parent anxiety level	0.01	.773	0.04	.158
RCADS child/parent depression level	0.02	.632	0.05	.161
RCADS child/parent anxiety and depression level	0.01	.655	0.05	.111

*Simple linear regression of child's RMMA to RCADS score; Significance was set at $P < .05$.

Table 5. Clinical and cephalometric findings of subjects with SB.

Variable	Count (%) or Mean ± SD	Beta	95% CI	*P*-value *	Adj. *P*-value**
Clinical variables					
Angle classification, *N* (%)					
Class I	22 (39.3)	1 (ref)	–	–	–
Class II Division I	13 (23.2)	−0.83	−2.33–0.68	.275	.489
Class II Division II	13 (23.2)	−0.92	−2.42–0.58	.224	.489
Class III	8 (14.3)	−1.01	−2.78–0.76	.259	.489
Friedman tongue score, *N* (%)					
1	8 (14.3)	1 (ref)	–	–	–
2a	8 (14.3)	1.68	−0.40–3.77	.112	.411
2b	27 (48.2)	0.50	−1.18–2.18	.553	.743
3	10 (17.9)	1.78	−0.20–3.76	.077	.411
4	3 (5.3)	2.22	−0.61–5.04	.122	.411
Signs of asymmetry, *N* (%)	14 (25.0)	−0.94	−2.24–0.36	.154	.411
Functional shift, *N* (%)	9 (16.1)	−0.06	−1.62–1.51	.942	.962
Anterior crossbite, N (%)	10 (17.9)	0.15	−1.35–1.65	.843	.962
Posterior crossbite, *N* (%)	10 (17.9)	−0.24	−1.74–1.26	.748	.921
Maxillary TSALD (mm)	2.37 ± 9.92	0.02	−0.04–0.08	.460	.736
Mandibular TSALD (mm)	0.56 ± 9.48	0.00	−0.06–0.06	.962	.962
Maxillary 6–6 dimension (mm)	41.26 ± 5.56	0.08	−0.02–0.19	.107	.411
Mandibular 6–6 dimension (mm)	41.79 ± 4.90	0.09	−0.03–0.20	.149	.411
BEWEI cumulative score	0.29 ± 0.87	0.20	−0.47–0.86	.557	.743
Cephalometric variables					
SNA (°)	82.73 ± 4.85	0.15	0.04–0.27	.007	.056
SNB (°)	79.63 ± 4.62	0.12	−0.00–0.234	.057	.304
ANB (°)	3.11 ± 3.06	0.12	−0.07–0.31	.199	.602
Maxillary length (mm)	80.19 ± 12.99	−0.00	−0.05–0.04	.942	.944
Mandibular length (mm)	105.43 ± 10.28	-0.02	−0.07–0.04	.503	.894
Gonial angle (°)	122.57 ± 15.46	−0.03	−0.06–0.01	.139	.556
LFH (%)	55.87 ± 3.10	0.24	0.07–0.42	.007	.056
Y-axis (°)	56.82 ± 3.26	0.10	−0.08–0.27	.280	.602
SN-GoGn (°)	28.12 ± 6.76	−0.04	−0.13–0.04	.301	.602
Interincisal angle (°)	125.39 ± 43.59	0.00	−0.01–0.01	.940	.944
Nasolabial angle (°)	109.47 ± 14.54	−0.02	−0.06–0.02	.278	.602
IMPA (°)	92.67 ± 9.37	−0.01	−0.07–0.05	.803	.944
U1-SN (°)	104.38 ± 11.12	0.01	−0.04–0.07	.594	.944
L1-Apo (mm)	3.33 ± 13.96	0.00	−0.04–0.04	.920	.944
Overbite (mm)	3.39 ± 2.67	0.04	−0.18–0.25	.734	.944
Overjet (mm)	3.67 ± 3.06	−0.01	−0.20–0.18	.944	.944

TSALD = tooth size-arch length discrepancy; BEWEI = Basic erosive wear examination index; 6–6 dimension = inter-first molar width.
*Simple linear regression *P*-values.
**Multiple testing adjusted *P*-value by Benjamini–Hochberg method to control false discovery rate within the clinical group and cephalometric group separately; Significance was set at $P < .05$.

statistically significant. In the second model, SE% (β = 0.75, 95% CI 0.58–0.93, $P = .019$) and ORDI (β = 1.44, 95% CI 1.08–2.05, $P = .025$) were significant.

Discussion

The goal of this cross-sectional study was to evaluate the prevalence and risk factors of SB in adolescents and children presenting for orthodontic treatment at one orthodontic clinic. This is the first study that used MM monitoring to determine the prevalence of SB in an orthodontic population and determined it to be 60.7%. In the literature, the reported prevalence of SB in adolescents ranged from 9.1% to 67.3% [4, 5]. Prior studies however utilized unreliable methodologies including parental reports and/or self-reports, clinical wear of the teeth, and reported pain of the muscles of mastication.

Huynh *et al.* [34] investigated the prevalence of SB in children undergoing orthodontic treatment using an ambulatory type II PSG device, intraoral examinations, and parental reports. Their findings revealed no significant correlation between PSG-diagnosed SB, parental reports, or clinically observed tooth wear [34]. Additionally, in their study, 73.7% of

Table 6. Sleep variables for subjects with SB.

Variable	Mean ± SD	Beta*	95% CI	*P*-value *	Adj. *P*-value**
Subjective sleep variables (*n* = 87)					
Children's sleep habit questionnaire- cumulative score	41.64 ± 7.51	0.06	−0.02–0.13	.129	.256
Objective sleep variables (*n* = 56)					
TST (h)	6.74 ± 1.54	0.11	−0.27–0.49	.556	.741
Time to fall asleep (min)	28.05 ± 30.70	0.01	−0.00–0.03	.124	.194
Wake duration after sleep onset (min)	69.20 ± 28.20	0.02	0.00–0.04	.018	.061
SE (%)	81.91 ± 3.91	−0.22	(−0.36)–(−0.09)	.002	.006
REM sleep (%)	9.80 ± 5.96	−0.01	−0.10–0.09	.889	.889
Light sleep (%)	76.21 ± 8.94	−0.01	−0.07–0.06	.840	.889
Deep sleep (%)	13.98 ± 7.65	0.013	−0.06–0.09	.731	.889
REM latency (min)	159.93 ± 136.87	−0.01	−0.00–0.00	.053	.117
Microarousal index (events/h)	19.98 ± 5.73	0.31	0.25–0.37	<.001	<.001
ORDI all events (events/h)	4.86 ± 2.80	0.40	0.22–0.57	<.001	<.001
Estimated AHI (events/h)	2.47 ± 0.73	−0.07	−0.87–0.73	.862	.889
RE (% of TST)	22.25 ± 10.53	−0.01	−0.06–0.05	.800	.889
RERA index (events/h)	1.65 ± 1.37	0.41	0.00–0.82	.048	.117
ORDI in REM sleep (events/h)	5.34 ± 5.47	0.08	−0.02–0.19	.118	.194
ORDI in non-REM sleep (events/h)	4.13 ± 2.39	0.39	0.18–0.61	.001	.003
ORDI in supine position (events/h)	5.41 ± 5.26	0.17	0.02–0.31	.025	.072
ORDI in non-supine position (events/h)	4.81 ± 2.80	0.38	0.20–0.56	<.001	.001
TST in supine position (%)	12.77 ± 13.73	0.03	−0.01–0.07	.126	0.194
TST in other positions (%)	87.23 ± 13.74	−0.03	−0.07–0.01	.126	.194

TST = total sleep time; SE = sleep efficiency; ORDI = obstructive respiratory disturbance index; AHI = apnea hypopnea index; RE = Respiratory effort; RERA = respiratory effort-related arousals; REM = rapid eye movement.
*Simple linear regression *P*-values.
**Multiple testing adjusted *P*-value by Benjamini–Hochberg method to control false discovery rate; Significance was set at $P < .05$.

Table 7. Linear regression analysis for SB.

Model 1				Model 2			
Variable	Beta	95% CI	*P*-value*	Variable	Beta	95% CI	*P*-value*
Microarousal index (events/h)	0.31	0.25–0.36	<.001	SE (%)	−0.15	(−0.28)–(−0.01)	.026
Maxillary 6–6 dimension (mm)	0.08	0.02–0.13	.008	ORDI (events/hr)	0.33	0.15–0.51	<.001

*Significance was set at $P < .05$.

Table 8. Ordinal regression analysis for SB.

Model 1				Model 2			
Variable	OR	95% CI	*P*-value*	Variable	OR	95% CI	*P*-value*
Microarousal index (events/h)	1.73	1.37–2.37	<.001	SE (%)	0.75	0.58–0.93	.019
Maxillary 6–6 dimension (mm)	1.31	1.04–1.69	.025	ORDI (events/hr)	1.44	1.08–2.05	.025

*Significance was set at $P < .05$.

the parents were unaware of their child's SB [34], suggesting that it maybe underreported. In contrast, MM monitoring, a home sleep apnea testing method, has demonstrated a high level of accuracy in assessing SB, with 91% accuracy compared to PSG [20].

In the current study, parents/guardians were aware that their children grind their teeth, as indicated by responses to the abbreviated CSHQ. Specifically, question 1 asked: 'Does your child grind their teeth during sleep (your dentist may have told you this)?'. A significant association was found between parental reports and MM-diagnosed SB. This suggests greater parental awareness of their children's SB compared to Huynh *et al*'.s findings [34]. This heightened awareness could be attributed to parents' firsthand observation of their child's

grinding or clenching behaviors or to reports from dentists, as dental clearance is a prerequisite for initiating orthodontic treatment in this clinic.

This study assessed various factors from multiple sources, such as clinical examination, radiographic evaluation, questionnaires, and sleep variables, to determine factors that are implicated in the development of SB in children and adolescents. Research on SB and microarousals has shown that SB is the final event or secondary event to a microarousal [3]. Supporting this, evidence has shown that mean heart rate increases about 10 s before the RMMA episodes as well as brain activity determined by EEG [35]. Prior to the RMMA event, there is a significant breathing effort which is proceeded by the activation of the jaw closing muscles therefore, highlighting that the SB episode is the final event of a microarousal [2, 12]. Martinot *et al*. [36], stated that the analysis of MM delivers precise assessment of the arousal hourly index and that the intensity of the arousal correlates well with the amplitude of mandibular jaw movement waveform. They concluded that this is likely due to the cortico-bulbar reflex [36]. Based on these results, the dependent nature of SB and microarousals is confirmed, hence the construction of two linear regression models, one that included microarousals and another that did not. The study also constructed an ordinal model assessing SB according to severity despite the lack of validated cut off points. This study hopes that future studies involve determining the severity score that may aid in diagnosis and managing SB.

The relationship between SE% and SB has been emphasized in previous studies [37] and this relationship was corroborated in the present study. In the multiple linear regression analysis excluding microarousals, SE% had an inverse statistical association with SB (β = −0.15, 95% CI −0.28 to −0.01, P = .026) confirming that lower SE% (poor sleep quality) is associated with increased SB risk. Additionally, the ORDI was a significant factor in subjects with SB (β = 0.33, 95% CI 0.15–0.51, P < .001). Each unit increase in ORDI, increased RMMA by 0.33 confirming a significant association between SB and obstructive events during sleep indicative of OSA.

The relationship between SB and sleep breathing disorders (SBD) has long been a subject of interest. Ferreira et al. conducted a cross-sectional study of 496 preschool children, assessing SB and OSA risk through clinical examinations and parental questionnaires [38]. Their findings demonstrated a significant association between SB and elevated OSA risk. Orradre-Burusco *et al*., in a systematic review of 29 studies examining SB-SDB associations in children, reported mixed findings [39]. Among these studies, 16 focused on snoring, 11 analyzed SBDs more broadly, and three specifically addressed OSA. Except for four studies, most identified a comorbid relationship between SB and SBD. However, the overall quality of evidence was low, with a heavy reliance on parental reporting.

Repeated episodes of moderate-to-severe intermittent hypoxia are hypothesized to contribute to the connection between SB, masticatory muscle pain, and SBD [40]. Additional hypotheses propose that SB functions as a reactive mechanism to counteract upper airway obstruction, safeguarding individuals during obstructive events characteristic of OSA [41]. It is further theorized that SB episodes are initiated by activation of the suprahyoid muscles, followed by engagement of the jaw-opening muscles. This sequence of muscle activity increases airway patency, effectively enhancing airway opening [42]. Consequently, SB may serve as a protective reflex to help maintain airway integrity in patients with OSA.

The mean estimated AHI among subjects in the current study was 2.47 ± 0.73 events/h. However, this finding was not significantly associated with SB. Although this may suggest the presence of mild OSA, it is important to consider the inherent limitations of AHI as a measure, given its susceptibility to physiological variations across different sleep stages and body positions, as highlighted in prior studies [43, 44]. Instead, this study utilized the ORDI to evaluate the presence or absence of OSA.

In the other linear regression model that considered all variables, it was found that the microarousal index had a significant correlation with SB. This finding is similar to Bonacina *et al*. who examined SB in children aged 4 to 9 years diagnosed with mild OSA and compared them to a control period (4 min prior to the SB event) [45]. They reported that SB is linked to various physiological events such as microarousals, tachycardia, and leg movement.

This study evaluated maxillary and mandibular arch widths using digital models and found that the maxillary 6–6 dimension (mm) was significantly associated with SB in the multiple linear regression analysis (β = 0.08, 95% CI 0.02–0.13, P = .008). On average, each unit increase in maxillary 6–6 dimension resulted in a 0.08 increase in SB, while holding all other variables constant. The finding may reflect the impact of SB force and frequency on dental arch growth or could simply be attributed to the maxillary growth process involving both the maxillary basal bone and dentoalveolar region during normal craniofacial growth. Only a few studies have evaluated the relationship between SB and orthodontic variables. DiFrancesco *et al*. found that 60.71% of children with SB had malocclusion [46]. Carra *et al*. determined that a dental Class II relationship was present in over 60% of adolescents with SB [47]. Along with this, 28.1% of these adolescents with SB were of the brachyfacial pattern. A recent systematic review and meta-analysis provided limited evidence supporting a link between SB and orthodontic factors, suggesting a potential increase in SB in cases of dental crowding and the absence of maxillary posterior crossbite [48].

Contrary to these findings, Bodrumlu *et al*. recently reported no significant association between maxillary arch length and width in children with SB, based on parental reports, compared to non-bruxing children [49]. This discrepancy may stem from differences in methodology, as this is the first study to apply MM monitoring to objectively assess SB prevalence. Future research utilizing objective measures of SB is essential to validate these results.

This study had several limitations. First, there was an issue with compliance with the two consecutive nights of sensor wear (T1 and T2). Although 87 participants were included in the study only 56 wore the sensors as instructed. The other 29 subjects either did not wear the sensors or did not have a TST greater than 4 h and therefore were excluded from the bivariate and multivariate analyses.

Secondly, it is possible that the reported prevalence of SB may not fully capture the variable nature of the condition. SB exhibits night-to-night variability, meaning that the absence of SB on one or two nights does not necessarily indicate its absence on subsequent nights [50]. Finally, the sample in this study consisted of individuals seeking orthodontic treatment, limiting the generalizability of the results to the broader pediatric population. Future studies should include a larger

sample size for better representation of the entire population regarding the prevalence of SB.

Conclusion

In a growing orthodontic population, definite SB is very common. SB is related to microarousals, SE percentage, ORDI, and maxillary intermolar width. However, no other dental or orthodontic variables showed a significant association with SB. MM monitoring is a user-friendly tool for identifying SB in adolescents.

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Author contributions

Ryan Koch (Conceptualization [equal], Data curation [equal], Investigation [equal], Methodology [equal], Writing—original draft [equal]), Alberto Monegro (Investigation [equal], Writing—review & editing [equal]), Stephen Warunek (Investigation [equal], Writing—review & editing [equal]), William Tanberg (Formal analysis [equal], Writing—review & editing [equal]), and Thikriat Al-Jewair (Conceptualization [equal], Data curation [equal], Methodology [equal], Project administration [equal], Supervision [equal], Writing—original draft [equal], Writing—review & editing [equal])

Conflict of interest

The authors declare that there is no conflict of interest.

Ethics approval and consent to participate

The study was approved by the University at Buffalo Health Sciences Institutional Review Board (STUDY00005013).

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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