https://doi.org/10.5664/jcsm.9078

JCSM | Journal of Clinical Sleep Medicine

SCIENTIFIC INVESTIGATIONS

Identifying predictive factors for sleep bruxism severity using clinical and polysomnographic parameters: a principal component analysis

João Vicente Rosar, DDS, PhD[1]; Maria Carolina Salomé Marquezin, DDS, PhD[2]; Aianne Souto Pizzolato, DDS[2]; Fernanda Yukie Kobayashi, DDS, PhD[3]; Sandra Kalil Bussadori, DDS, PhD[3]; Luciano José Pereira, DDS, PhD[4]; Paula Midori Castelo, DDS, PhD[2]

[1]Department of Orthodontics, Universidade de Chapecó, Chapecó, Brazil; [2]Department of Pharmaceutical Sciences, Universidade Federal de São Paulo, Diadema, Brazil; [3]Department of Rehabilitation Sciences, Universidade Nove de Julho, São Paulo, Brazil; [4]Department of Health Sciences, Universidade Federal de Lavras, Lavras, Brazil

Study Objectives: The aim was to identify predictive factors for sleep bruxism (SB) severity among polysomnographic parameters, salivary cortisol levels, temporomandibular disorders, age, and sex.

Methods: Young adults (19–30 years) were screened for self-/roommate reports of teeth grinding/clenching during sleep associated with clinical signs of tooth wear. Individuals positive for both conditions were administered a polysomnographic exam to provide a definite diagnosis of SB (n = 28). Healthy participants without SB signs/symptoms were also included (n = 15). The Research Diagnostic Criteria for Temporomandibular Disorders was applied to determine functional, muscular, and articular domains of the Temporomandibular Index. Cortisol awakening levels were measured in saliva. Principal component analysis was used to extract the latent components emerging from polysomnographic results, and 2 regression models were adjusted to predict the number and duration of bruxism episodes.

Results: Principal component analysis resulted in 4 components—C1: %N1, total sleep time, sleep efficiency, arousals/microarousals; C2: %N2, %N3; C3: periodic limb movements and apneas; C4: %REM and REM latency. The number of SB episodes/h was predicted by increasing muscular scores and C2 (decrease in %N2 and increase in %N3) (adjusted R^2 = 45%; P =.001). The total time of SB episodes was predicted by decreased articular and increased functional scores, age, and female sex (adjusted R^2 = 36%; P = 0.010). Salivary cortisol levels were not associated with SB severity and did not differ between groups.

Conclusions: The findings showed that SB severity was predicted by muscular and functional scores, female sex, and distinct polysomnographic patterns, contributing to the deeper knowledge of the underlying pathophysiology of SB severity; additionally, the findings can help to formulate health approaches that are specific to the patient and will better assist in treating this condition.

Keywords: bruxism, sleep disturbance, temporomandibular disorders

Citation: Rosar JV, Marquezin MCS, Pizzolato AS, et al. Identifying predictive factors for sleep bruxism severity using clinical and polysomnographic parameters: a principal component analysis. *J Clin Sleep Med*. 2021;17(5):949–956.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Sleep bruxism diagnosis and therapeutic approach (if any) is challenging. Although polysomnography is the gold standard for definite diagnosis, there is a lack of information on how sleep parameters are associated with sleep bruxism severity. Principal component analysis is a useful statistical method to analyze a large number of sleep characteristics which may predict sleep bruxism severity.

Study Impact: Increased muscular symptomatology, reduced N2, and increased %N3 sleep stages were considered factors predictive of the number of sleep bruxism episodes/h. In addition, the duration of sleep bruxism episodes was associated with lower articular and higher functional scores, also increasing with age and female sex. These results are clinically important to the identification of the demographic and polysomnographic characteristics associated with sleep bruxism severity and may help improve treatment of this condition.

INTRODUCTION

Bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding the teeth and/or by bracing or thrusting of the mandible,[1,2] which can occur during sleep (sleep bruxism, SB) or wakefulness (awake bruxism).[1,3] Sleep bruxism involves rhythmic (phasic) or nonrhythmic (tonic) masticatory muscle activity during sleep. According to the latest international consensus, it should not be considered a sleep disorder in healthy individuals.[4] However, depending on its severity, sleep bruxism may be associated with detrimental consequences to orofacial structures, such as teeth resorption, fractured cusps/restorations, and pulpitis, in addition to headaches and poor sleep quality.[5,6] As a multifactorial condition, a large number of oral and systemic aspects may be involved; thus, a comprehensive and multivariate approach is essential to better understand its characteristics and to design a treatment plan according to its severity.

For diagnostic purposes, sleep bruxism is best assessed by polysomnography (PSG), which is considered the gold-standard protocol. However, as an expensive and complex diagnostic exam, it may not be accessible (and most often not justified in the general population).[7] For this reason, information on how sleep parameters are associated with the severity of SB is scarce. Most events of SB are observed in N1 and N2 stages of sleep in polysomnograms,[8] and these episodes are associated with light body movements and microarousals. Due to the large number of

variables derived from PSG, principal component analysis may be a useful multivariate approach to summarize PSG data to further test the association between PSG findings and SB severity.

The mandibular movements during SB are centrally mediated by several neurotransmitters, especially dopamine, serotonin, γ-aminobutyric acid, and noradrenaline.[8,9] Risk factors may include disturbances in neurotransmitters, neurological diseases, trauma, drug use, smoking, alcohol, and psychological factors, including stress.[10–12] The behavioral factors, such as depression and stress, induce hormonal responses with a corresponding increase in cortisol levels by the adrenal cortex, due to stimulation of the hypothalamus-pituitary-adrenal axis.[13]

Electromyographic (EMG) patterns comprise repetitive and recurrent episodes of rhythmic masticatory muscle activity (RMMA) of the masseter and temporalis muscles. PSG recording allows RMMA episodes to be distinguished as phasic, tonic, or mixed and SB to be diagnosed as mild (when RMMA episodes are greater than or equal to 2 episodes/h) or severe (when greater than or equal to 4 episodes/h).[14] The excessive masticatory muscle activity in SB supports its role as a risk factor for temporomandibular disorders (TMD).[3,15] The imbalance between muscle activity and recovery might lead to overloading and muscle pain,[16,17] and for this reason SB has been associated with chronic local muscle contracture, inflammation, and localized muscle hypertrophy that can become a cause of myofascial pain.[18,19] Thus, the knowledge of the underlying pathophysiological mechanisms of SB severity is of clinical importance.

Therefore, the objective of our study was to evaluate whether the number and duration of bruxism episodes (SB severity) can be predicted by PSG distinct sleep latent factors, as well as with salivary cortisol levels and TMD signs and symptoms.

METHODS

Study design and participants

This cross-sectional study was approved by the Ethics Committee of Piracicaba Dental School, University of Campinas, Protocol No. 750.187. Verbal and written consent were obtained from each participant, who was informed about the procedures, possible discomforts, and risks and benefits of the study, following the recommendations of the Declaration of Helsinki and with Ministry of Health Resolution 466/12.

Sample selection

Initially, 250 young adults, among students and employees from the University of Chapecó (Santa Catarina, Brazil), were invited to participate. The eligible participants were first interviewed regarding personal data, sociodemographic characteristics, medical and dental histories, in addition to self- or roommate report of any sound of grinding or clenching the teeth during sleep (and other sleep complaints). From them, 190 individuals were considered "healthy" (absent of chronic diseases or did not use chronic medications, tobacco, alcohol, among others) and answered the specific questionnaires. Eighty individuals reported no symptom of TMD or bruxism. Further, a comprehensive clinical evaluation was performed to select a group of healthy individuals without any sign/symptom of awake or sleep bruxism and no diagnosis of TMD based on Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) criteria, and, on the other hand, a group of individuals with evident signs and symptoms of SB.

The inclusion criteria were healthy adults, aged between 18 and 30 years, of both sexes, and with complete permanent dentition. The exclusion criteria were as follows: individuals with systemic disorders and/or current/chronic use of medicines that could interfere with the central nervous system, muscle activity, or saliva secretion (eg, neurological disorders, hypertension, cancer, rheumatoid arthritis, diabetes, asthma, dyslipidemia, xerostomia, antihistamines, benzodiazepines, antidepressants, anxiolytics, syrups, anti-inflammatory agents, corticoids, tobacco, and alcohol consumption, among others). Dental health conditions were also considered (after clinical examination described below), and individuals were excluded who showed any tooth loss (except for third molars), soft tissue abnormalities, toothache report, presence of periodontal pockets and/or involvement of the supporting tissues, caries lesions, current orthodontic or speech language therapies, fixed or removable dental prosthesis, or severe/extreme malocclusion.[20]

Based on the inclusion/exclusion criteria, and on the clinical and PSG findings described below, 2 groups of participants were included in the study. The bruxism group comprised 28 participants with definite sleep bruxism; 3 of them performed the PSG exam twice to confirm the SB diagnosis. The healthy group of individuals included 15 participants who did not report any teeth clenching/grinding or orofacial pain symptoms, or presented dental wear facets or other clinical sign of bruxism, neither have been diagnosed as having TMD based on RDC/TMD. As the group of healthy individuals included only young adults without any sign or symptom of oral or systemic disease (neither respiratory disorders nor complaints), this group did not take part in the PSG exams. The recruitment period was 4 months (period between the invitation and the last PSG exam).

Clinical examination

Dental, temporomandibular joint, and masticatory muscle examinations were performed by one trained/experienced researcher (JVR). The examination was carried out in a dental office, under adequate lighting for wear facets diagnosis. The presence of wear facets on enamel or dentine or polished and shiny facets between opposing teeth were detected during mandibular excursions.

Temporomandibular dysfunction clinical exam was conducted using the RDC/TMD criteria (axis I and status and perception of pain from axis II)[21–23] by the same calibrated researcher (JVR; κ coefficient > 0.9). RDC/TMD procedures were applied to calculate the temporomandibular index (TMI), in which an algorithm designates the severity of TMD in 3 domains: functional, muscular, and articular. For each domain, scores ranging from 0 (no clinical sign) to 1 (presence of clinical sign) were determined.[24] Briefly, the functional domain consists of 12 items related to mandibular movements: unassisted and assisted opening with or without pain, right-left range of motion, protrusion, and mandibular opening pattern. The muscular domain refers to pain associated with bilateral digital palpation

of selected intraoral and extraoral masticatory muscles (20 sites). The articular domain is composed of 8 items evaluating evoked-pain after digital palpation of 2 sites for each TMJ and the occurrence of noise (click and/or crepitus). The overall TMI index is then calculated by the arithmetic mean of these 3 domains; the closer the score is to 1, the greater the severity of signs and symptoms of TMD.[25]

Sleep bruxism diagnosis and polysomnographic exams

During the interview, information on the nocturnal tooth ranging, at least 3 days a week, mainly reported by the room partner, associated or not with facial and cervical muscles pain/fatigue, joint and/or dental discomfort upon awakening, and fractured restorations or cusps were gathered. In this way, those participants with self- or roommate report of sound of grinding/clenching the teeth during sleep associated with evident clinical signs of tooth wear were referred to PSG exam, in order to provide a definite diagnosis of SB.[4]

PSG exams were carried out at the Center of Sleep Disturbances (Meditron-Sonolab 620, Meditron Eletromedicina Ltda., São Paulo, Brazil), Unimed Hospital, Chapecó (Santa Catarina, Brazil). All participants started the procedure at 9 PM and remained monitored for 1 night, as described previously.[24] The PSG equipment included a 6-channel electroencephalogram, bilateral electrooculogram, submental electromyogram (chin EMG), bilateral anterior tibialis EMG, electrocardiogram using a single modified lead II, rib cage and abdominal movement belts with piezoelectric sensors, nasal airflow pressure transducer and nasal/oral thermistor, oximetry, a snoring sensor, and a body position sensor. In addition, bilateral masseter EMG and bilateral temporalis EMG channels were used to detect bruxism events. A SomnoStarPro acquisition system (Viasys Healthcare, San Diego, CA) with sampling rates ranging from 50 Hz to 200 Hz was used. Audio and video signals were recorded continuously. The onset and offset times of nocturnal PSG recordings were determined from each participant's habitual bedtime and uptime.[24]

The following variables were considered in this study: rapid eye movement (REM) sleep latency (min); non-REM sleep latency (min); total sleep time (min); sleep efficiency (%); percentages of sleep stages N1, N2, and N3; REM sleep (%); periodic leg movements (n/h); obstructive sleep apnea (n/h); arousals (n/h); and microarousals (n/h). The electromyographic analysis of SB episodes was performed based on the American Academy of Sleep Medicine criteria.[7] The number of rhythmic masticatory muscle activity per sleep bruxism episode (RMMA/SB) per hour and the type of episode (phasic, tonic, or mixed) in the different stages of sleep were assessed. The diagnosis was considered positive when the RMMA index was ≥ 2 episodes/h of sleep.[14]

Salivary cortisol levels

Two samples of stimulated saliva from each participant were collected at home, on a scheduled day in the same week of the polysomnography exam, using Salivettes (Sarstedt, Nümbrecht, Germany). The first saliva sample was collected early in the morning, immediately after waking up, and the second sample was collected after 30 min (still fasting). The individuals were asked to keep the polyester roll inside the mouth sufficiently to enable soaking it with saliva. The roll was returned to the tube and stored in the refrigerator until delivery to the researcher (on the same day). At the laboratory facility, the samples were centrifuged (at 3500 rpm, 10 min, 4°C) and stored at −80°C until analysis. Patients were instructed to wake up between 6 and 8 AM, not to brush their teeth before collection, and not to drink beverages with caffeine/alcohol or practice physical exercise on the day before.

The salivary cortisol levels were determined according to the manufacturer's instructions (product no. 1-1102, Salimetrics, State College, PA) and ELISA plate-based assay, as previously described.[24] Further, the area under the curve against time was estimated by the trapezoid formula respective to the ground level to estimate the cortisol awakening response.[26]

Data analysis

Statistical analysis was performed by one of the authors (PMC, Applied Statistics Spec, São Paulo, Brazil) using SPSS 26.0 software (IBM, Armonk, NY) and considering an alpha level of 5%. Descriptive analysis consisted of means, standard deviation, medians, and quartiles. Salivary cortisol levels and TMI scores were compared between groups using a 1-way analysis of variance test, also considering the effect size and power of the test.

Principal component analysis was used to estimate the number of latent variables emerging from the PSG findings, to derive optimal noncorrelated components among the parameters: total sleep time; REM sleep latency; non-REM sleep latency; sleep efficiency; N1, N2, N3, and REM sleep as percentage of total sleep time; periodic limb movements; apneas; arousals; and microarousals. First, the correlation matrix was examined and a decision on the number of components to be retained was based on eigenvalues, total of explained variance, and scree plot examination. The overall Kaiser-Meyer-Olkin measure and Bartlett's test of sphericity were examined, as required for a good principal component analysis.

With the objective of explaining the variation in the number and duration of bruxism episodes (rhythmic masticatory muscle activity), components extracted from the analysis were used as explanatory variables in the adjustment of regression models, in addition to the variables age, sex, salivary cortisol (area under the curve), and functional, muscular, and articular TMI domains. This analysis included participants with SB only. The backward procedure was used to obtain the final model, after examination of the changes in the adjusted R^2 and F-values for each new independent variable that was added. The assumptions of the test: normality, collinearity (variance inflation factor and tolerance), independence of errors (Durbin-Watson), and homoscedasticity (residual analysis) were also considered to reach the best fit.

RESULTS

Considering the clinical and PSG findings, sleep bruxism diagnosis was considered definite for the 28 participants who composed the bruxism group. The group of healthy individuals comprised 15 participants who did not report teeth clenching/grinding or

Table 1—Description of the clinical groups according to salivary cortisol levels and temporomandibular index domains.

	Age (y)	Cortisol (μg/dL)		Cortisol AUC	Functional Index	Muscular Index	Articular Index	TMI Index
		Upon Waking	After 30 Min					
Bruxism group[a]								
Mean (SD)	22.57 (2.74)	0.19 (0.21)	0.24 (0.28)	6.71 (5.65)	0.44* (0.15)	0.29* (0.18)	0.25 (0.22)	0.33* (0.12)
Median (25–75%)	—	0.2 (0.0–0.3)	0.2 (0.1–0.2)	5.9 (2.8–7.5)	0.4 (0.3–0.6)	0.3 (0.2–0.4)	0.3 (0.0–0.4)	0.3 (0.2–0.4)
Healthy individuals[b]								
Mean (SD)	21.60 (1.72)	0.16 (0.13)	0.16 (0.09)	4.98 (2.07)	0.28* (0.11)	0.13* (0.12)	0.18 (0.15)	0.20* (0.10)
Median (25–75%)	—	0.1 (0.1–0.2)	0.2 (0.1–0.2)	4.9 (3.6–5.6)	0.3 (0.2–0.3)	0.1 (0.0–0.2)	0.3 (0.0–0.3)	0.1 (0.1–0.2)

[a]n = 28; 6 M, 22 F. [b]n = 15; 4 M, 11 F. *P <05; one-way analysis of variance test. AUC = area under the curve, SD = standard deviation, TMI = temporomandibular index.

Table 2—Characteristics of the polysomnographic findings of the bruxism group.

Polysomnographic Findings	Mean (SD)	Median (25–75%)
NREM sleep latency (min)	19.54 (11.78)	16.0 (10.7–22.0)
REM sleep latency (min)	129.09 (44.07)	129.0 (105.5–157.2)
Total sleep time (min)	343.37 (46.16)	347.0 (320.2–364.7)
Sleep efficiency (%)	86.82 (6.72)	88.5 (84.7–91.0)
Stage 1 (%)	7.70 (3.49)	6.8 (4.8–9.8)
Stage 2 (%)	47.73 (7.52)	47.2 (42.0–52.5)
Stage 3 (%)	24.79 (8.44)	23.9 (19.6–31.3)
REM sleep (%)	19.41 (3.99)	19.3 (17.1–21.5)
Periodic leg movements (events/h)	5.12 (3.40)	4.9 (2.5–6.6)
Obstructive sleep apnea (events/h)	0.58 (0.64)	0.3 (0.2–0.8)
Arousals (events/h)	2.95 (1.17)	2.8 (2.0–3.6)
Microarousals (events/h)	10.34 (3.81)	9.7 (7.4–13.6)

NREM = nonrapid eye movement, REM = rapid-eye movement, SD = standard deviation.

Table 3—Characteristics of the number and duration of bruxism episodes of the bruxism group.

	Phasic Bruxism			Tonic Bruxism			Mixed Bruxism			Bruxism All Types
	Phasic Bruxism (s)	Longer Duration (s)	n/h	Tonic Bruxism (s)	Longer Duration (s)	n/h	Mixed Bruxism (s)	Longer Duration (s)	n/h	n/h
Mean (SD)	6.82 (1.96)	10.21 (3.85)	1.48 (1.47)	3.62 (0.91)	5.80 (2.37)	1.57 (1.36)	6.33 (4.48)	7.91 (5.70)	0.59 (0.99)	3.81 (2.73)
Median (25%–75%)	6.4 (5.3–8.1)	9.7 (7.6–12.0)	0.9 (0.6–1.5)	3.6 (3.0–3.9)	5.9 (4.3–7.1)	1.2 (0.7–2.0)	7.7 (1.0–8.7)	8.5 (1.0–12.6)	0.2 (0.0–0.7)	3.0 (2.0–4.3)

n/h = number per hour, s = seconds, SD = standard deviation.

pain symptoms, nor presented dental wear facets. These 2 groups showed moderate to large differences regarding TMI muscular (P = .003; eta partial squared = 0.19; power of the test = 86%) and functional (P < .001; eta partial squared = 0.24; power of the test = 94%) domains scores and TMI total score (P < .001; eta partial squared = 0.22; power of the test = 92%), with higher scores for SB group (**Table 1**). On the other hand, salivary cortisol levels were similar between groups on awakening, after 30 minutes, and in the area under the curve (P >.05).

Descriptive analysis of the bruxism group according to PSG findings are expressed in **Table 2**. Severe sleep bruxism was found in 9 participants (32% of the sample).[14] Total sleep time was around 6 hours, and sleep efficiency above 85%, with most of the time in N2 stage. Obstructive sleep apnea was rare. The mean number of SB episodes/h was 3.8, with a higher number of tonic episodes (**Table 3**).

A principal component analysis was run to extract components emerging from the PSG sleep parameters. The suitability was assessed prior to analysis, and the inspection of the correlation matrix indicated those variables that had at least 1 correlation coefficient greater than 0.30. Bartlett's test of sphericity was statistically significant (P < .0001). Principal component analysis retained 4 components that had eigenvalues greater than 1 and which explained 78.4% of the total variance, as confirmed by visual inspection of the scree plot (**Figure 1**). As such, 4 components met the interpretability criterion and no rotation was needed.

Figure 1—Scree plot derived from principal component analysis used to observe the inflection point.

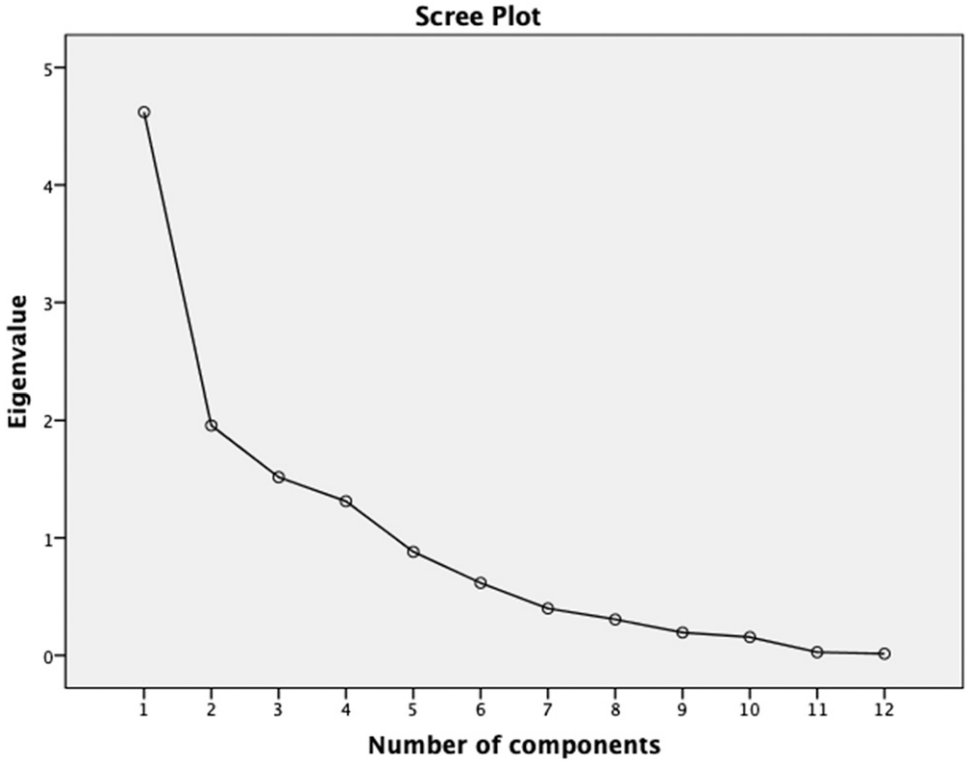

Table 4—Component loadings obtained by principal component analysis of the polysomnographic findings.

	Components			
	1	2	3	4
NREM sleep latency	0.720	—	–0.491	—
REM sleep latency	—	0.526	—	–0.650
Total sleep time	–0.759	—	0.418	—
Sleep efficiency	–0.816	—	0.459	—
Stage 1	0.892	—	—	—
Stage 2	—	–0.831	—	–0.441
Stage 3	–0.401	0.865	—	—
REM sleep	–0.432	—	—	0.693
Periodic leg movements	0.562	—	0.564	0.358
Apnea	—	0.324	0.623	—
Arousals	0.770	—	—	—
Microarousals	0.830	—	—	—

NREM = nonrapid eye movement, REM = rapid eye movement.

As shown in **Table 4**, the interpretation of the data was consistent with the attributes of the involved aspects: %N1, total sleep time, sleep efficiency, arousals, microarousals loaded highly onto Component 1 (C1); Component 2 (C2): %N2, %N3; Component 3 (C3): the number of periodic limb movements and apneas; and Component 4 (C4): %REM sleep and REM sleep latency. Thus, the higher the C1, the lower the time and sleep efficiency and the higher the %N1, number of arousals, and microarousals. The higher the C2, the higher %N3 and lower % N2; the higher the C3, the higher the number of periodic limb movements and apneas; and the higher the C4, the higher the % REM sleep and lower the REM sleep latency.

Linear regression models were adjusted with the aim of explaining the variation in the number and duration of sleep bruxism episodes (**Table 5**). Considering the number of bruxism episodes/h, the significant independent variables retained in the final model were muscular TMI score and C2 (lower %N2 and higher %N3) (adjR^2 = 45%; P = .001). The total time of bruxism

Table 5—Final predictive models for estimation of the number and duration of bruxism episodes.

Dependent Variable	Independent Variables	B	95% CI	*t*	Sig	*F*	Adjusted R^2	Durbin– Watson
Number of bruxism episodes/h	Constant	2.1	0.5 to 3.6	2.7	0.012	8.3 *P* = .001	0.45	2.2
	Muscular index	5.9	1.3 to 10.5	2.6	0.014			
	Component 2	1.4	0.6 to 2.3	3.5	0.002			
	Component 4	–0.7	–1.6 to 0.1	–1.8	0.088			
Duration of bruxism episodes (h)	Constant	–389.0	–714.6 to –63.5	–2.5	0.021	4.0 *P* = .010	0.36	2.0
	Articular index	–211.7	–389.5 to –33.8	–2.5	0.022			
	Functional index	287.9	29.6 to 546.2	2.3	0.031			
	Age	15.5	1.7 to 29.3	2.3	0.029			
	Female sex	103.8	4.1 to 203.6	2.1	0.042			
	Component 1	–35.9	–73.8 to 1.9	–1.9	0.062			

B = regression coefficient, CI = confidence interval, *F* = *F*-test statistic, Sig = significance, *t* = *t* statistic.

episodes (duration) was predicted by lower articular and higher functional TMI scores, age, and female sex (adjR^2 = 36%; *P* = .010). Both regression models showed good fit, as observed by the parameters of tolerance, variance inflation factor, residual analysis, and independence of errors (Durbin-Watson). Cortisol area under the curve was not a significant predictive variable.

DISCUSSION

The main results of the present study indicated that SB severity, eg, "the number of bruxism episodes per hour" and "the total time of bruxism episodes (duration)," can be predicted by muscular symptomatology, lower %N2, and higher % N3 sleep stages, in addition to lower articular and higher functional scores, age, and female sex, respectively. SB has a complex diagnosis and the self-report may not necessarily indicate the presence and severity of repetitive jaw activity during sleep. Identifying the relationship between SB severity and PSG and clinical predictive factors contributes to the identification of factors related to the degree of this condition.[27]

The association of the number of SB episodes and muscular scores emphasizes that the higher the repetitive jaw activity during sleep, the worse the muscle symptomatology and pain.[28] Sleep bruxism has been shown to negatively affect masticatory muscle function[29] and, indeed, the present results also showed that the SB group presented scores in the muscular index domain higher than those in the group of healthy individuals. However, a systematic review of this topic concluded that the evidence of the relation between muscle symptomatology and SB is conflicting and seems to be dependent on many factors, such as age, the quality of the diagnostic methodology regarding SB, and the type of musculoskeletal signs and symptoms.[30] In this way, the relationship between the repetitive jaw activity related to SB and higher muscle scores might not be directly caused by muscle pain but also by the feeling of unpleasantness, tiredness, and soreness which characterizes other unspecific muscle symptoms.[28] Masticatory muscle pain might be a form of delayed onset muscle soreness as a result of repetitive muscle activities.[31]

Regarding the relationship between SB and TMD signs and symptoms, a recent study using polysomnography indicated that the distribution of TMD among patients with SB and those without SB was similar, and the authors concluded that SB would not be a risk factor for TMD occurrence.[32] However, consideration of the subcategories of TMD based on joint pain, muscle pain, or a mix seems to be advisable. In a recent study, patients with muscle and joint pain did not show elevated background EMG or SB. In contrast, patients with isolated muscle pain showed significantly higher background EMG and a trend toward elevated prevalence of SB.[33] These results may explain the association between lower articular and higher muscle TMI scores and SB in the present study. Additionally, this finding may explain previous results that did not find a consistent positive association between SB and TMD (since samples are usually made up of heterogeneous joint and muscle TMD groups).[32] As reported, a positive association between SB and TMD may be limited to the muscular subgroup without joint pain,[33] and that seemed to happen in the present study.

Female sex was a predictive factor for SB episode duration, and one may hypothesize that as females may have greater severity of SB, the signs/symptoms can be more evident, thus facilitating the diagnosis. While a previous study with children between 8 and 10 years of age reported that boys were 79% more likely to have SB,[34] the literature suggests that female sex tends to be associated with SB from adolescence[31] to adulthood, with women reporting both awake bruxism and SB more often than men.[35,36] The association between female sex and SB might be linked to myofascial symptoms, which are also more frequent in women. There is a statistically significant association between self-reported SB and women who have painful symptoms of TMD.[37] Female TMD patients show greater pain and muscle tenderness on palpation compared to males with TMD.[38] Men

are reported to have greater bite force and greater masseter cross-sectional area of type II fibers than women,[39] and it is hypothesized that the smaller cross-sectional muscle area and higher frequency of type I fibers may lead to the higher prevalence of muscle tenderness in women.[38]

In addition, age was also associated to SB severity (duration). Although SB tends to decrease with age,[35] the present sample comprised only young adults aged between 19 and 30 years, which is reported to be the peak of the trend along the life span.[35,40] SB bruxism prevalence and severity may fluctuate with time, and accurate estimation of the prevalence of SB is difficult and varies significantly due to diagnostic strategies and populations characteristics.[41]

SB did not affect the quality and duration of sleep in comparison to the reference values (with mean values of sleep efficiency above 85%), corroborating other studies,[42,43] and according to the present findings, the number of SB episodes was associated with lower %N2 and higher %N3 sleep stages. Stage N2 follows stage N1, in which breathing and heart rate begin to slow (usually corresponding to half of sleep time). Next comes stage N3, called slow wave sleep, which is the deep sleep and regenerative period.[44] Previous findings showed a positive correlation between phasic bruxism episode index and N3 sleep for nonobstructive sleep apnea participants[43] and significantly less continuous N2% in SB patients than in controls.[42,43] The results agree with a previous report that in patients with SB, RMMA occurs following longer runs of stage N3, suggesting the association with underlying processes related to dissipation of homeostatic sleep pressure.[42] Indeed, it is important to consider, when scoring sleep and events in a polysomnogram (*International Classification of Sleep Disorders*),[45] that increases in N1 and N3 follow compensatory decreases in N2.[46]

No difference was found in the levels of salivary cortisol between groups and cortisol levels were not associated with either the number or duration of sleep bruxism episodes, corroborating previous research from our group in children between 7 and 14 years old.[47] However, other studies found higher salivary cortisol levels in women with self-reported SB and general anxiety.[12] Although SB has been related to stress or anxiety, it only represents a sleep-related-motor disorder over a certain threshold.[48] Since the occurrence of RMMA during the night is related to circadian rhythm, the interindividual variability of the circadian phase may be a confounding factor when investigating the association between the neuroendocrine system and RMMA.[42]

Indeed, it is worth mentioning the limitations of the study. Due to ethical reasons and to the strict inclusion and exclusion criteria applied with the aim of assembling a homogeneous sample, a small convenience sample of individuals with SB was included and may have skewed the data and lowered the statistical power. Thus, the multivariate analysis proposed in this study should be further validated in a larger sample of individuals with definite SB and paired controls.

To our knowledge, this is the first study using a multivariate analysis to identify factors predictive of SB severity among PSG and clinical parameters. This analysis may provide insights into the underlying pathophysiological mechanisms of SB and can be useful for determining SB severity and formulating health approaches specific to patients and more effective in improving their condition.

ABBREVIATIONS

C1, Component 1
C2, Component 2
C3, Component 3
C4, Component 4
EMG, electromyography
PSG, polysomnographic
RDC/TMD, Research Diagnostic Criteria for Temporomandibular Disorders
REM, rapid eye movement
RMMA, rhythmic masticatory muscle activity
SB, sleep bruxism
TMD, temporomandibular disorder
TMI, temporomandibular index

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.
2. Muzalev K, Visscher CM, Koutris M, Lobbezoo F. Effect of experimental temporomandibular disorder pain on sleep bruxism: a pilot study in males. *Clin Oral Investig*. 2020;24(1):103–111.
3. Restrepo C, Lobbezoo F, Castrillon E, et al. Agreement between jaw-muscle activity measurement with portable single-channel electromyography and polysomnography in children. *Int J Paediatr Dent*. 2018;28(1):33–42.
4. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil*. 2018;45(11):837–844.
5. Yoshida Y, Suganuma T, Takaba M, et al. Association between patterns of jaw motor activity during sleep and clinical signs and symptoms of sleep bruxism. *J Sleep Res*. 2017;26(4):415–421.
6. Vieira KRM, Folchini CM, Heyde MDVD, et al. Wake-up headache is associated with sleep bruxism. *Headache*. 2020;60(5):974–980.
7. Sateia MJ. International classification of sleep disorders-third edition: highlights and modifications. *Chest*. 2014;146(5):1387–1394.
8. Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res*. 1996;75(1):546–552.
9. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med*. 2003;14(1):30–46.
10. Winocur E, Uziel N, Lisha T, Goldsmith C, Eli I. Self-reported bruxism—associations with perceived stress, motivation for control, dental anxiety and gagging. *J Oral Rehabil*. 2011;38(1):3–11.
11. Gungormus Z, Erciyas K. Evaluation of the relationship between anxiety and depression and bruxism. *J Int Med Res*. 2009;37(2):547–550.
12. Fluerasu MI, Bocsan IC, Buduru S, et al. The correlation between sleep bruxism, salivary cortisol, and psychological status in young, Caucasian healthy adults [published online ahead of print, 2019 May 27]. *Cranio*.
13. Refulio Z, Rocafuerte M, de la Rosa M, Mendoza G, Chambrone L. Association among stress, salivary cortisol levels, and chronic periodontitis. *J Periodontal Implant Sci*. 2013;43(2):96–100.
14. Carra MC, Huynh N, Lavigne G. Sleep bruxism: a comprehensive overview for the dental clinician interested in sleep medicine. *Dent Clin North Am*. 2012;56(2):387–413.

15. Raphael KG, Janal MN, Sirois DA, et al. Validity of self-reported sleep bruxism among myofascial temporomandibular disorder patients and controls. *J Oral Rehabil*. 2015;42(10):751–758.
16. Meeusen R, Duclos M, Foster C, et al. Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Med Sci Sports Exerc*. 2013;45(1):186–205.
17. Muzalev K, Lobbezoo F, Janal MN, Raphael KG. Interepisode sleep bruxism intervals and myofascial face pain. *Sleep*. 2017;40(8):zsx078.
18. Jiménez–Silva A, Peña–Durán C, Tobar–Reyes J, Frugone–Zambra R. Sleep and awake bruxism in adults and its relationship with temporomandibular disorders: a systematic review from 2003 to 2014. *Acta Odontol Scand*. 2017; 75(1):36–58.
19. Yalçın Yeler D, Yılmaz N, Koraltan M, Aydın E. A survey on the potential relationships between TMD, possible sleep bruxism, unilateral chewing, and occlusal factors in Turkish university students. *Cranio*. 2017;35(5):308–314.
20. Üçüncü N, Ertugay E. The use of the Index of Orthodontic Treatment need (IOTN) in a school population and referred population. *J Orthod*. 2001;28(1):45–52.
21. Dworkin SF. Research diagnostic criteria for temporomandibular disorders: current status and future relevance. *J Oral Rehabil*. 2010;37(10):734–743.
22. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord*. 1992;6(4):301–355.
23. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic criteria for temporomandibular disorders (dc/tmd) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. *J Oral Facial Pain Headache*. 2014; 28(1):6–27.
24. Rosar JV, Barbosa TS, Dias IOV, et al. Effect of interocclusal appliance on bite force, sleep quality, salivary cortisol levels and signs and symptoms of temporomandibular dysfunction in adults with sleep bruxism. *Arch Oral Biol*. 2017;82:62–70.
25. Pehling J, Schiffman E, Look J, Shaefer J, Lenton P, Fricton J. Interexaminer reliability and clinical validity of the temporomandibular index: a new outcome measure for temporomandibular disorders. *J Orofac Pain*. 2002;16(4):296–304.
26. Castelo PM, Barbosa TS, Pereira LJ, Fonseca FLA, Gavião MBD. Awakening salivary cortisol levels of children with sleep bruxism. *Clin Biochem*. 2012;45(9): 651–654.
27. Castrillon EE, Ou KL, Wang K, Zhang J, Zhou X, Svensson P. Sleep bruxism: an updated review of an old problem. *Acta Odontol Scand*. 2016;74(5): 328–334.
28. Shimada A, Castrillon EE, Svensson P. Revisited relationships between probable sleep bruxism and clinical muscle symptoms. *J Dent*. 2019;82: 85–90.
29. Garip H, Tufekcioglu S, Kaya E. Changes in the temporomandibular joint disc and temporal and masseter muscles secondary to bruxism in Turkish patients. *Saudi Med J*. 2018;39(1):81–85.
30. Baad–Hansen L, Thymi M, Lobbezoo F, Svensson P. To what extent is bruxism associated with musculoskeletal signs and symptoms? A systematic review. *J Oral Rehabi.l* 2019;46(9):845–861.
31. van Selms MKA, Visscher CM, Naeije M, Lobbezoo F. Bruxism and associated factors among Dutch adolescents. *Community Dent Oral Epidemiol*. 2013;41(4): 353–363.
32. Wieckiewicz M, Smardz J, Martynowicz H, Wojakowska A, Mazur G, Winocur E. Distribution of temporomandibular disorders among sleep bruxers and non-bruxers—a polysomnographic study. *J Oral Rehabil*. 2020;47(7): 820–826.
33. Santiago V, Raphael K. Absence of joint pain identifies high levels of sleep masticatory muscle activity in myofascial temporomandibular disorder. *J Oral Rehabil*. 2019;46(12):1161–1169.
34. Soares JP, Giacomin A, Cardoso M, Serra–Negra JM, Bolan M. Association of gender, oral habits, and poor sleep quality with possible sleep bruxism in schoolchildren. *Braz Oral Res*. 2020;34:e019.
35. Wetselaar P, Vermaire EJH, Lobbezoo F, Schuller AA. The prevalence of awake bruxism and sleep bruxism in the Dutch adult population. *J Oral Rehabil*. 2019; 46(7):617–623.
36. Nogueira Coutinho E, Pereira Rodrigues dos Santos K, Henrique Barros Ferreira E, Grailea Silva Pinto R, de Oliveira Sanchez M. Association between self-reported sleep bruxism and temporomandibular disorder in undergraduate students from Brazil. *Cranio*. 2020;38(2):91–98.
37. Blanco Aguilera A, Gonzalez Lopez L, Blanco Aguilera E, et al. Relationship between self-reported sleep bruxism and pain in patients with temporomandibular disorders. *J Oral Rehabil*. 2014;41(8):564–572.
38. Schmid–Schwap M, Bristela M, Kundi M, Piehslinger E. Sex-specific differences in patients with temporomandibular disorders. *J Orofac Pain*. 2013; 27(1):42–50.
39. Tuxen A, Bakke M, Pinholt EM. Comparative data from young men and women on masseter muscle fibres, function and facial morphology. *Arch Oral Biol*. 1999; 44(6):509–517.
40. Pontes L da S, Prietsch SOM. Sleep bruxism: population-based study in people with 18 years or more in the city of Rio Grande, Brazil. *Rev Bras Epidemiol*. 2019; 22:e190038.
41. Huhtela OS, Näpänkangas R, Joensuu T, Raustia A, Kunttu K, Sipilä K. Self-reported bruxism and symptoms of temporomandibular disorders in Finnish university students. *J Oral Facial Pain Headache*. 2016;30(4):311–317.
42. Kishi A, Haraki S, Toyota R, et al. Sleep stage dynamics in young patients with sleep bruxism. *Sleep*. 2020;43(1):zsz202.
43. Wieczorek T, Wieckiewicz M, Smardz J, et al. Sleep structure in sleep bruxism: a polysomnographic study including bruxism activity phenotypes across sleep stages. *J Sleep Res*. 2020;29(6):e13028.
44. Patel AK, Araujo JF. *Physiology, Sleep Stages*. Tampa/St. Petersburg, FL: StatPearls Publishing; 2020.
45. American Academy of Sleep Medicine. *International Classification of Sleep Disorders*. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
46. Grigg–Damberger MM. The AASM scoring manual: a critical appraisal. *Curr Opin Pulm Med*. 2009;15(6):540–549.
47. Kobayashi FY, Gavião MBD, Marquezin MCS, et al. Salivary stress biomarkers and anxiety symptoms in children with and without temporomandibular disorders. *Braz Oral Res*. 2017;31:e78.
48. Raphael KG, Santiago V, Lobbezoo F. Is bruxism a disorder or a behaviour? Rethinking the international consensus on defining and grading of bruxism. *J Oral Rehabil*. 2016;43(10):791–798.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication August 6, 2020
Submitted in final revised form December 20, 2020
Accepted for publication December 22, 2020
Address correspondence to: Paula Midori Castelo, DDS, PhD, Department of Pharmaceutical Sciences, Universidade Federal de São Paulo, R. São Nicolau, 210, 1°. andar, Diadema, SP, Brasil, CEP 09913-030;
Email: pcastelo@yahoo.com or paula.castelo@unifesp.br

DISCLOSURE STATEMENT

All authors read and approved the manuscript. Work for this study was performed at the Department of Orthodontics of Universidade Comunitária da Região de Chapecó and the Department of Pharmaceutical Sciences of Universidade Federal de São Paulo. This study was funded by the agreement between Universidade Comunitária da Região de Chapecó and Faculdade de Odontologia de Piracicaba (Doutorado Interinstitucional/ Dinter, process no. 7626-0). Maria Carolina Salomé Marquezin (postdoctoral fellow) received a scholarship from Fundação de Amparo à Pesquisa do Estado de São Paulo (grant numbers 16/10940-9 and 17/26400-6). The remaining authors report no conflicts of interest.