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A laboratory–based polysomnographic investigation

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Bruxism is characterized by grinding or clenching of the teeth. For approximately 50 years, bruxism has been advanced as a predominant factor in the onset or continuance of myofascial or muscle-pain–predominant temporomandibular disorders (TMDs).[1,2] People who adhere to this view consider myofascial TMD to be a response to a barrage of afferent nociceptive signals secondary to bruxism that occurs during waking and sleep. Survey results have shown that most general dentists and TMD specialists believe that bruxism plays a significant role in the pathogenesis of TMDs.[3-5] Patients with TMD also believe that bruxism is a cause of their pain.[6]

Sleep bruxism (SB) involves grinding or clenching of the teeth during sleep and is considered a sleep-movement–related disorder, according to the most recent edition of the International Classification of Sleep Disorders.[7] SB is difficult to diagnose in nonlaboratory settings. As detailed in a comprehensive critical review of research studies published from 1998 through 2008, SB can be detected unequivocally only by means of polysomnographic

ABSTRACT

Background. Many dentists believe that sleep bruxism (SB) is a pathogenic factor in myofascial temporomandibular disorder (TMD), but almost all supportive data rely on patients' self-reports rather than on direct observation.

Methods. The authors administered a structured self-report interview to determine whether a large and well-characterized sample of patients with myofascial TMD (124 women) experienced SB more often than did matched control participants (46 women). The authors then used data from a two-night laboratory-based polysomnographic (PSG) study to determine whether the case participants exhibited more SB than the control participants.

Results. The results of independent sample t tests and χ^2 analyses showed that, although self-reported rates of SB were significantly higher in case participants (55.3 percent) than in control participants (15.2 percent), PSG-based measures showed much lower and statistically similar rates of SB in the two groups (9.7 percent and 10.9 percent, respectively). Grinding noises were common in both case participants (59.7 percent) and control participants (78.3 percent).

Conclusions. Most case participants did not exhibit SB, and the common belief that SB is a sufficient explanation for myofascial TMD should be abandoned.

Clinical Implications. Although other reasons to consider treating SB may exist, misplaced concern about SB's sustaining or exacerbating a chronic myofascial TMD condition should not be used to justify SB treatment.

Key Words. Temporomandibular dysfunction; facial pain; myofascial pain; bruxism.

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(PSG) recordings.[8] However, most of the research supporting the view that SB occurs at elevated rates in patients with TMDs relies on self-reports rather than on PSG recording data.

More than 20 years ago, Marbach and colleagues[9] expressed concerns about the reliability and potential bias of self-reports of SB, particularly because many people with TMD believe that they likely brux because their dentists have told them that they do on the basis of their TMD signs and symptoms. If dentists told patients that they likely brux because of existing pain symptoms, an impossible tautology occurs. Although consideration of factors such as tooth wear should improve clinical diagnosis, these evaluations are not reliable[10]; different dentists do not always agree about whether a patient bruxes, and they can change their evaluations across time. Moreover, tooth wear corresponds poorly to TMD symptoms.[8] Self-reports are a common and problematic method used to assess SB in both clinical practice and research. Because of limited technological access, cost and time investment, PSG studies are rare.

SB motor activity can be studied by means of ambulatory- or laboratory-based PSG recordings. Although ambulatory-based studies are easier to conduct, they do not allow for the combination of electromyographic (EMG), video and audio signal analyses that are available in laboratory-based studies. These types of signals are required to differentiate SB from other sleep motor activities (for example, chewing, sleep talking, yawning) that can confound its assessment.[11-13] The most typical EMG pattern related to SB is rhythmic masticatory muscle activity (RMMA) episodes. They are considered rhythmic because they repeat in a series of episodes over sleep periods. Within an RMMA, the following are scored: at least three EMG bursts for phasic episodes, a sustained long EMG burst for the more rare tonic (clenching) episodes or a mixture of both types. When episodes occur with tooth-grinding sounds during sleep, they are defined as tooth-grinding–type SB episodes. Lavigne and colleagues[14] provided preliminary validation of clinical research diagnostic criteria for SB (RDC/SB) involving more than four RMMA episodes per sleep hour or more than six bursts per episode combined with 25 bursts per sleep hour. These diagnostic criteria have been applied in PSG-based studies of SBs in TMDs.[15-17] Investigators of a study identified a lower-frequency subthreshold RDC/SB group that had more than two and less than four RMMA episodes per hour.[18] Patients in this group might be at greater risk of experiencing pain than are patients in a group that meets more stringent RDC/SB standards.

In the only published laboratory-based PSG SB study, investigators compared participants with myofascial TMD with matched asymptomatic control participants.[16] They found that 19 of 30 patients with myofascial TMD (63 percent) met the RDC/SB standard compared with 10 of 30 control participants (33 percent). Compared with prior estimates that 8 percent of the general population meets criteria for RDC/SB,[19] the rates in both groups of participants raise questions about the method used to assess RMMA, sample representativeness or both.

The primary aim of our investigation was to provide a definitive test to determine whether participants with myofascial TMD exhibited more SB than did demographically equivalent control participants who did not have TMD. In contrast to a previous study,[8] we defined SB by collecting laboratory-based PSG data in a large and well-defined sample and by using state-of-the-art methods for differentiating episodes of SB from other sleep motor activities. We also aimed to compare conclusions from PSG data with those from self-reports of SB.

METHODS

The Institutional Review Board at the New York University (NYU) School of Medicine (New York City) approved the study.

Participants. Given the markedly higher prevalence of TMDs in women,[20-22] we recruited only women to participate in the study. We recruited participants primarily from among patients attending clinics at the NYU College of Dentistry (NYUCD) or from among the acquaintances of patients at NYUCD. After they gave their informed consent, we examined and interviewed them.

We conducted laboratory-based PSG studies at a sleep laboratory affiliated with the NYU School of Medicine.

We enrolled participants on the basis of the presence (case participants) or absence (control participants) of a myofascial TMD and independent of their beliefs or knowledge regarding their own SB, to ensure that SB prevalence was

ABBREVIATION KEY. EMG: Electromyographic. **NYU:** New York University. **NYUCD:** New York University College of Dentistry. **PSG:** Polysomnographic. **RDC/SB:** Research diagnostic criteria for SB. **RDC/TMD:** Research Diagnostic Criteria for Temporomandibular Disorders. **RMMA:** Rhythmic masticatory muscle activity. **SB:** Sleep bruxism. **TMD:** Temporomandibular disorder.

not overrepresented or underrepresented in either sample.

Case participants. We recruited case participants from the orofacial pain clinic at NYUCD. Participants with myofascial TMD met Research Diagnostic Criteria (RDC/TMD) for Group I[23]—that is, pain of muscle origin, including a complaint of pain and pain associated with localized areas of tenderness to palpation in muscle. The complaint includes report of pain or ache in the jaw, temples, face, preauricular area or ears while at rest or during function, in addition to pain reported in response to palpation of three or more of the following 20 muscle sites (left and right for each muscle): posterior temporalis, middle temporalis, anterior temporalis, origin of masseter, body of masseter, insertion of masseter, posterior mandibular region, submandibular region, lateral pterygoid area and tendon of the temporalis. We required that at least one site be ipsilateral to the complaint of pain.

Control participants. We recruited control participants from other NYUCD dental clinics and from among acquaintances of case participants to constitute a control sample that was a demographic match to the case participants regarding age, socioeconomic status, self-identified race and self-identified Hispanic ethnicity. We did not include potential control participants if they had reported having one or more weeks of facial pain in the last two years or more than one painful site on masticatory muscle palpation, according to RDC/TMD examination procedures.[23]

Exclusion criteria. We excluded potential case participants or control participants if they indicated that they had been in a motor vehicle accident or experienced other major and identifiable physical trauma involving the face. Because extensive dental treatment might cause temporary masticatory muscle pain or temporarily change the participant's usual rates of bruxism, any acute dental problem for which treatment was sought needed to be resolved by the time of the RDC/TMD examination, and at least 48 hours must have passed between the latest dental treatment and the RDC/TMD examination.

Participation. Among the 169 potential case participants whom we approached in the orofacial pain clinic, 19 patients did not provide consent to undergo an RDC/TMD examination for study screening purposes. Of the 150 patients who did, 138 met RDC/TMD criteria. The most common reason for exclusion was having a history of trauma to the face (n = 6), followed by practical reasons such as regularly smoking during the night and sleeping less than four hours per night (n = 5). Among 63 potential control participants, the most common reason for exclusion was experiencing more than one tender point when undergoing the RDC/TMD examination (n = 6); eight of the 54 patients who met the eligibility criteria canceled study appointments or failed to appear for research procedures. In all, 124 women with a diagnosis of myofascial TMD and 46 control participants completed both the daytime interview and sleep laboratory studies.

Measures. ***Assessment of SB.*** *Self-report.* A clinical research coordinator (L.V.N.) conducted a structured interview with participants individually, asking whether they engaged in grinding their teeth at night during sleep. The investigator also asked about clenching and daytime behaviors, but these questions were extraneous to the current investigation. For each of the sleep-related questions to which participants answered "yes," the interviewer asked how they knew that they engaged in that behavior (Table 1). Questions referencing "ever" grinding and grinding in the "past two weeks" were asked separately.

PSG record. Since sleep monitoring, as well as sleeping in an unfamiliar laboratory environment, can alter natural sleep,[24] participants spent two consecutive nights in the sleep laboratory. The first night was to help the participant acclimate. Data from this night were not used in statistical analysis, except for data for three case participants who failed to return for the second night and for six case participants and one control participant for whom we were unable to obtain scores on the second night owing to technical problems. We made sleep recordings from approximately 10:30 p.m. to 7:00 a.m., closely adjusted to the participant's usual sleep time. The settings of the recordings have been described in detail elsewhere.[14,25]

Two people with doctorates and expertise in sleep medicine (P.E.W., B.D.) were trained to score all relevant bruxism parameters. The PSG record consisted of a six-channel electroencephalogram, a bilateral electrooculogram, a bilateral submental (chin) and an anterior tibialis EMG, a right and left masseter and temporalis EMG, an electrocardiogram, chest and abdominal motion (by means of belts with piezoelectric sensors), body position, airflow by nasal pressure transducer and nasal-oral thermistor, and oximetry. They recorded sleep data by using the SomnoStar Pro (Viasys Healthcare, San Diego) sleep diagnostics system with sampling

TABLE 1

Self-reported sleep bruxism traits among case and control participants.

SELF-REPORT INTERVIEW QUESTIONS	CONTROL PARTICIPANTS (n = 46)	CASE PARTICIPANTS (n = 124)	*P* VALUE (χ^2 OR FISHER EXACT)
	Agree,* % (No.)	Agree,* % (No.)	
Have you ever been told you grind your teeth at night during sleep?	15.2 (7)	55.3 (68)	< .001
Were you told this by a			
Dentist?	8.7 (4)	39.5 (49)	< .001
Other health care professional?	0.0 (0)	7.3 (9)	.016
Sleep partner?	6.5 (3)	25.8 (32)	.006
Have you ever noticed that you grind your teeth at night during sleep?	8.7 (4)	37.4 (46)	< .001
In the last two weeks, have you been told you grind your teeth at night during sleep?	0.0 (0)	15.3 (19)	.007
Were you told this by a			
Dentist?	0.0 (0)	8.9 (11)	.039
Other health care professional?	0.0 (0)	0.8 (1)	Value not calculable
Sleep partner?	0.0 (0)	7.3 (9)	.050
In the last two weeks, have you noticed that you grind your teeth at night during sleep?	0.0 (0)	23.6 (29)	< .001

* Excludes "don't know."

rates ranging from 50 to 200 hertz. They recorded audio and video signals in parallel.

The sleep scorers scored jaw muscle activity after exporting data to a sleep data recording and analysis software package (Stellate Harmonie, Natus, San Carlos, Calif.). Using RDC/SB validated by Lavigne and colleagues,[14] they used audio and video signals to differentiate tooth-grinding sounds from other oral noises made during sleep (for example, snoring, sleep talking, TMJ clicking with yawning). The experts performed a detailed analysis of SB muscle activity for the right masseter. They analyzed activity that exceeded twice the amplitude of the relaxed waking EMG level before sleep. Phasic episodes were defined by three or more brief (> 0.25 seconds and < 2.0 seconds) EMG bursts. They scored tonic episodes if the burst was longer than two seconds. When RMMAs were accompanied by grinding sounds, the experts considered them to be an episode of tooth grinding. EMG episodes had to be separated by at least three seconds to be scored as different episodes. For each jaw muscle episode that met this criterion, we calculated the frequency of RMMA episodes per hour of sleep, the number of RMMA bursts per hour, the number of RMMA episodes with sounds and the total episode duration.

We used three PSG-based measures of SB that were consistent with standards.[14] First, as our most stringent measure, we identified participants who met the RDC/SB criteria of having more than four episodes of SB per hour of sleep or more than 25 bursts per hour of sleep. The results of research by Lavigne and colleagues[26] has shown that the presence or absence of PSG-based diagnoses made by using RDC/SB criteria are likely to remain constant over time, and the results of reanalysis of these data have shown intraclass correlation coefficients ranging from 0.64 to 0.87, indicating good to excellent reliability in frequency of RMMA episodes between nights (J.E. Schwartz, Ph.D., professor, State University of New York at Stony Brook, unpublished data, 2006). Second, we identified a subthreshold RDC/SB group with moderate SB, defined as more than two but less than four episodes of SB per hour of sleep, which is consistent with the results of pain-related research on SB.[18] Finally, as the least stringent measure, we identified participants who had at least two episodes accompanied by grinding noises during the PSG study.

RMMA episodes were separated from sleep motor activities that can be differentiated from RMMA activity only with audiovisual signals.[12] We grouped together the frequency of "other orofacial activity" episodes involving muscular activity (for example, swallowing, snoring,

yawning) in the nose-mouth-jaw triangle that is not included in the definition of a SB episode. We also grouped together "other muscular activity" episodes, which consisted of activity not specific to the nose-mouth-jaw-triangle that nonetheless caused masticatory EMG elevation (for example, head or body movement or nose scratching). Across a sample of 846 episodes, the interrater reliability for identification of RMMA episodes by the two sleep scorers was excellent ($\kappa = 0.89$).[27]

Pain history and intensity. The clinical research coordinator asked RDC/TMD standardized pain history questions.[23] The interview included questions regarding pain onset, pain severity and pain-related disability. Characteristic pain intensity is the average of present or current, worst and average pain across the preceding six months, each rated on a scale of 0 to 10 (0 = no pain, 10 = pain as bad as could be). Pain interference is the extent to which pain interfered with daily activities, rated on a scale of 0 to 10 (0 = no interference, 10 = unable to carry on any activities).

Data analysis. Depending on the level of measurement, group differences were analyzed with either independent sample *t* tests or χ^2 tests. We used a significance level of 5 percent. When data were skewed, we reported medians, and we calculated *P* values on the basis of nonparametric Kruskal-Wallis tests. We designed the study to have more case participants than control participants (2:1 ratio) to power within-case–group analyses unrelated to our investigation, while maintaining power sufficient to detect moderate effect sizes between groups.

Our inclusion or removal of the 10 nights of first-night–scored data from the statistical analysis did not alter any conclusions. Thus, we treated these data as equivalent to second-night–scored data.

RESULTS

Case and control group participants did not differ regarding any measured demographic characteristic. Most of them indicated that their race was white (62.6 percent), black (14.4 percent) or "other" (14.4 percent). A total of 22.5 percent indicated that they were of Hispanic ethnicity. Mean age (standard deviation [SD]) was 39.2 (14.6) years (range, 19-78), and mean of years of education was 15 (2.2) years (range, 11-20). Case participants reported experiencing moderate intensities of characteristic pain (mean [SD] = 5.2 [1.7]) and having relatively low levels of pain disability (mean [SD] 1.8 [2.2]). Pain onset occurred more than 10 years before study entry (mean [SD] 126.1 [127.1] months, median = 84).

Self-reported SB in case participants and control participants. As shown in Table 1, case participants reported having SB (defined as grinding of the teeth at night during sleep) more often than did control participants. These differences were most marked when the source of this information was the dentist or participant, followed by the participant's sleep partner. We obtained similar results for reports of sleep grinding during the preceding two weeks, although differences were most marked for the participant's noticing ($P < .001$), followed by the dentist's or participant's sleep partner's noticing. When we combined sources of self-reported knowledge, we found that 19.6 percent of control participants and 64.5 percent of case participants reported that they ground their teeth at night, either because they noticed it themselves or someone told them they did ($P < .001$).

PSG-defined SB in case participants and control participants. If SB were a sufficient explanation for pain associated with TMD, one would see higher levels of SB in case participants than in control participants. However, no measure based on PSG recordings supported this hypothesis. Using recommended RDC/SB criteria,[14] we found that 10.9 percent of control participants and a statistically similar 9.7 percent of case participants had high levels of SB activity (Table 2). The rates of subthreshold RDC/SB activity also were similar in control participants (17.4 percent) and case participants (16.9 percent). Using the least stringent PSG measure, we found that control participants (78.3 percent) were more likely ($P < .05$) to have two or more episodes with grinding noises than were case participants (59.7 percent). The distribution of RMMA episodes per hour of sleep was positively skewed but similar in both groups, with only three control participants (6.5 percent) and three case participants (2.4 percent) having six or more RMMA episodes per hour. Seven control participants (15.2 percent) and 25 case participants (20.2 percent) did not have any RMMA episodes during sleep. When we examined the number of episodes per hour or their duration during sleep, measures were positively skewed in both groups, as shown by large SDs and by median values that were lower than the mean values (Table 2). When we used either parametric or nonparametric statistical tests, we found that the two groups did not significantly differ on any measure. For both groups, we found that

TABLE 2

Laboratory polysomnographic (PSG) comparison of sleep bruxism (SB) among case and control participants.

PSG	CONTROL PARTICIPANTS (n = 46)		CASE PARTICIPANTS (n = 124)		*P* VALUE (χ^2 OR FISHER EXACT TEST)
Criterion	**No. (%)**		**No. (%)**		
Met research diagnostic criteria (RDC)/SB criteria	5 (10.9)		12 (9.7)		.818
Met subthreshold RDC/SB criteria	8 (17.4)		21 (16.9)		.915
Had two or more episodes with grinding noise	36 (78.3)		74 (59.7)		.038
Measure	**Mean (SD*)**	**Median**	**Mean (SD)**	**Median**	***P* Value (Median Test)**
RMMA episodes					
Count per hour	1.7 (1.9)	1.0	1.5 (1.9)	0.8	.388
Duration, seconds	56.4 (69.5)	24.5	47.9 (69.7)	21	.989
RMMA episodes with grinding					
Count per hour	1.0 (1.1)	0.5	1.0 (1.5)	0.4	.615
Duration, seconds	36.2 (49.9)	15.5	31.3 (55.0)	11.0	.294
Other orofacial activities					
Count per hour	9.4 (7.2)	7.1	10.1 (8.5)	7.1	.863
Duration, seconds	122.5 (130.9)	87.0	127.8 (122.5)	87.0	.937
Other muscular activities					
Count per hour	6.1 (2.9)	5.8	6.5 (4.5)	5.1	.605
Duration, seconds	139.3 (70.3)	119.5	150.0 (104.7)	127.5	.937

* SD: Standard deviation.

RMMAs were associated with grinding sounds approximately one-half the time. Although both "other orofacial activities" and "other muscular activities" occurred more frequently and for longer durations during sleep than did RMMAs, case participants and control participants had similar levels. Participants in both groups spent an average of less than one minute per night in SB episodes and less than five minutes in episodes engaging the masticatory muscles. Thus, there was little difference between groups regarding PSG-defined SB. This finding failed to support the hypothesis that case participants engage in more SB than do control participants.

Characteristic pain in case participants who were positive and negative for SB. The bruxism hypothesis of TMD pain would predict that those who brux have higher levels of pain. Table 3 shows that pain duration is similar for patients with TMD with and without PSG evidence of SB. Those who met RDC/SB criteria had significantly lower levels of characteristic pain intensity than did those who did not meet the criteria. Participants with two or more episodes with grinding noise had significantly lower levels of pain interference than did those who did not. These data did not support the hypothesis.

DISCUSSION

If this study, like many others, relied solely on participants' self-reports of sleep grinding, we would have reached the common conclusion that rates of SB are highly elevated in patients with myofascial TMD. However, more compelling data were available. The reference standard[14] from laboratory-based PSG data failed to support the notion of an association between SB and myofascial TMD. When we examined multiple masticatory muscle EMG-related episode measures obtained at night, we found no suggestion of there being higher rates of SB in case participants than in control participants. The one comparison that did show a significant difference suggested that SB (grinding noise) was less likely in case participants than in control participants. Within the case group, participants with PSG evidence of SB reported having lower levels of characteristic pain intensity and less pain interference with daily activities. Thus, SB appeared to be more likely among those reporting less myofascial TMD pain, which is consistent with a "pain adaptation" model,[28] predictive of an inverse relationship between agonist muscle activity and pain intensity. The results of a previous study of patients with myofascial TMD and control participants, in which we used electron microscopy–based

TABLE 3

Pain characteristics among case participants with and without polysomnographic (PSG) evidence of sleep bruxism (SB).

PSG CRITERION	PAIN DURATION (NO. OF MONTHS SINCE ONSET OF FACIAL PAIN)		CHARACTERISTIC PAIN INTENSITY (0-10*)		INTERFERENCE OF PAIN WITH DAILY ACTIVITIES (0-10†)	
	Mean (SD‡)	Median	Mean (SD)	Median	Mean (SD)	Median
Met Research Diagnostic Criteria (RDC)/SB Criteria						
No (n = 112)	133.3 (131.6)	86.1	5.4 (1.7)	5.5	2.3 (2.6)	1.0
Yes (n = 12)	84.6 (102.8)	42.6	4.0 (1.7)§	4.3¶	0.9 (1.3)	0.0
Met Subthreshold RDC/SB Criteria						
No (n = 91)	132.2 (129.7)	80.1	5.3 (1.8)	5.3	2.4 (2.7)	1.0
Yes (n = 33)	118.5 (130.5)	78.7	4.9 (1.7)	5.3	1.6 (2.0)	0.5
Had Two or More Episodes With Grinding Noise						
No (n = 50)	141.5 (145.0)	94.6	5.6 (1.7)	5.7	3.1 (2.9)	2.5
Yes (n = 74)	119.9 (118.5)	79.5	5.0 (1.7)	5.0	1.5 (2.1)#	0.0§

* On a scale of 0 to 10, in which 0 = no pain, 10 = pain as bad as could be.
† On a scale of 0 to 10, in which 0 = no interference, 10 = unable to carry on any activities.
‡ SD: Standard deviation.
§ $P < .01$.
¶ $P < .05$.
# $P < .001$.

assessment of tooth wear as a measure of bruxism across time, also supported the pain adaptation model.[29]

These data support earlier findings that RMMA is a common but time-limited activity,[30] as some degree of RMMA was found in nearly 80 percent of the women in our study. Moreover, 59.7 percent of case participants and 78.3 percent of control participants engaged in some tooth grinding during sleep. On the other hand, our data also suggest that clinically significant SB is a relatively uncommon activity, occurring in 9.7 percent of case participants and 10.9 percent of control participants. These findings are in agreement with other estimates.[18] Thus, the data from our study helped us understand that although past observers may have been correct in noting that patients with TMD brux, this behavior is not unique and does not explain painful symptoms.

The results of a study suggested that people who have moderate frequencies of SB may be at the greatest risk of experiencing facial pain.[18] The results of our study did not support this finding, because moderate frequencies of SB were prevalent in both case and control participants. A likely explanation for the discrepancy between conclusions is that the investigators in the earlier study[18] associated intermediate frequency SB/RMMA activity with a complaint of jaw pain on waking or masticatory muscle fatigue and low levels of current pain, and our participants who reported having moderate levels of pain were seeking treatment and met RDC/TMD criteria[23] for myofascial pain. Thus, although some degree of SB activity might cause acute and mild masticatory muscle soreness, just as experimental grinding can produce short-lived pain,[31,32] neither moderate nor extreme levels of SB account for a chronic, clinically important myofascial TMD condition.

Several issues require clarification. The cross-sectional data from our study did not address the possibility that SB could be involved in the initial onset or triggering of a myofascial TMD. However, even if SB eventually were shown to play a role in the initiation of myofascial TMD pain but not in its course, as shown in our study, efforts aimed at reducing SB in patients with myofascial TMD still would be viewed as unwarranted.

We did not evaluate other relationships and measurements between SB and myofascial TMD. For example, we did not evaluate the amplitude of masticatory muscle EMG activity during sleep or temporal patterns of SB during the night. Nevertheless, the low rates of SB in both groups and the limited average time spent in oromotor activity during the night made it unlikely that such examinations would be fruitful.

We did not address the possible role daytime oromotor activities play in TMDs. Accurate assessment of waking oromotor activities continues to be a challenge, and innovative methods[33,34] are needed to quantify waking bruxism in natural settings accurately. It remains possible that daytime bruxism contributes to myofascial TMD.

For reasons unrelated to this substudy, the clinical research coordinator and one of the sleep scorers were not masked as to the participants' case-control status. In theory, failure to fully mask all study personnel could have led to bias in assessments and scorings. However, the results of a poststudy but predata-analysis debriefing showed that these personnel favored the belief that pain and bruxism would be associated with one another. This potential bias is contrary to the observed relationship between pain characteristics and PSG evidence of SB.

Another potential problem was that greater use of SB-altering medication could have attenuated rates of SB in the case participants. However, the results of exploratory analyses (not detailed in this article) that removed the 14 participants who were taking any medication that potentially exacerbated or attenuated SB[35] (for example, muscle relaxants, benzodiazepines, selective serotonin reuptake inhibitors) failed to change the pattern of results, suggesting that data were not confounded by medication use.

Finally, our self-reported rate of SB among control participants of 15.2 percent appeared to be unusually high compared with the 8 percent rate found in large surveys.[36-38] This observation might raise concern that we recruited an atypical sample of control participants. However, the 15.2 percent rate reflects reports of "ever" having engaged in SB. Time frames for questions in the large surveys[36-38] were more limited, and lifetime rates of any behavior should be higher than the rates we report. Nevertheless, it still is possible that those control participants who agreed to participate in the study were people who were atypically interested in learning about their own suspected SB, and that this factor elevated the rates in the control group. Although this possible factor related to selective participation may have made them less representative of the general population, it may have made them more comparable with the case participants, who believed that their pain was related to SB.

To avoid concerns being raised about sleep behaviors observed within a laboratory setting, we included an acclimation night in our study. Nevertheless, SB is associated with microarousal states[39] and sleep instability.[40] In theory, such instability might create a "permissive window" and appear as atypically high levels of SB in a laboratory setting. There is no theory to explain the similar low rates of SB we observed in the case and control groups in our study. Finally, although our conclusions cannot be generalized confidently to men or children with myofascial TMD, they are applicable to the population at greatest risk, adult women.

CONCLUSIONS

Our study results should lay to rest any remaining beliefs regarding a relationship between SB and the course of myofascial TMD. Although there may be other reasons for considering treating SB (for example, tooth wear[41,42]), the treatment decision should not be made based on a concern for maintaining or exacerbating a chronic, painful myofascial TMD condition. ■

Disclosure. Dr. Lavigne has given lectures to Pfizer (Kirkland, Quebec, Canada), UCB (Brussels) and ResMed (San Diego), and he is on the scientific board of Grindcare, Herlev, Denmark. None of the other authors reported any disclosures.

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