Sleep bruxism and myofascial temporomandibular disorders

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.