

# Association Between Rhythmic Masticatory Muscle Activity During Sleep and Masticatory Myofascial Pain: A Polysomnographic Study

Leylha Maria Nunes Rossetti, DDS,  
MSc, PhD

Private Practice

Carlos Dos Reis Pereira de Araujo,  
DDS, MSc, PhD

Assistant Professor

Paulo Henrique Orlato Rossetti, DDS,  
MSc, PhD

Assistant Professor

Paulo César Rodrigues Conti, DDS, PhD

Associate Professor

Department of Prosthodontics  
Bauru School of Dentistry  
University of São Paulo  
Bauru, Brazil

and

Department of Prosthodontics  
School of Dentistry  
Universidade do Sagrado Coração  
Bauru, Brazil

## Correspondence to:

Dr Leylha Maria Nunes Rossetti  
Rua Dr. Sérgio Túlio Carrijo Coube,  
3-33 apto 83A  
Fax: 55 14 3223 4678  
E-mail: leylharossetti@uol.com.br

***Aims:** To test for an association between rhythmic masticatory muscle activity during sleep, as assessed according to polysomnographic criteria for sleep bruxism (RMMA-SB), and myofascial pain (MFP), as well as the chance of occurrence of MFP in patients with RMMA-SB. **Methods:** Thirty MFP patients (diagnosed according to the Research Diagnostic Criteria for Temporomandibular Disorders) and 30 age- and gender-matched asymptomatic controls underwent a polysomnographic examination. Also, any self-reporting of daytime clenching (DC) was registered in 58 of these subjects. **Results:** Most MFP patients reported mild or moderate pain (46.67% and 43.33%, respectively), and only 3 (10%) reported severe pain. Pain duration ranged from 2 to 120 months (mean  $34.67 \pm 36.96$  months). Significant associations were observed between RMMA-SB and MFP as well as between DC and MFP. **Conclusions:** (1) RMMA-SB is significantly associated with MFP; (2) although RMMA-SB represents a risk factor for MFP, this risk is low; and (3) DC probably constitutes a stronger risk factor for MFP than RMMA-SB. J OROFAC PAIN 2008; 22:190–200*

**Key words:** myofascial pain syndromes, polysomnography, risk, sleep bruxism

**M**yofascial pain (MFP) is one of the most common findings in temporomandibular disorders (TMD) patients.<sup>1</sup> Although the precise sequence of events that causes MFP remains unclear, key features, such as muscle tenderness on palpation, limitation of mouth opening, altered dental occlusal perception, and mood alterations, are often present and considered consequences of pain.<sup>2</sup> The myofascial TMD should not be understood as a single, discrete disease entity,<sup>2–5</sup> because usually, a variety of overlapping conditions accompanies the painful symptomatology.<sup>2</sup>

Sleep bruxism (SB) is considered a sleep-related movement disorder<sup>6</sup> comprising parafunctional clenching and grinding activities during sleep. Discrimination between SB and bruxism during wakefulness (daytime bruxism) is dependent on there being different etiologies for these phenomena.<sup>7</sup> In epidemiologic studies, the prevalence of self-report varies widely according to the type of bruxism and the phrasing used in the questionnaire used. One report found prevalences of 13.4% for exclusively daytime brux-

ism, 12.0% for past daytime bruxism, 3.3% for exclusively SB, and 6.6% for past SB.<sup>8</sup> Moreover, prevalences of daytime bruxism, SB reported by another person, and awareness of SB were reported as 34.2%, 16.1%, and 20.8%, respectively.<sup>9</sup> The prevalences of tooth clenching and nocturnal grinding have also been estimated as 20% and 6%, respectively.<sup>10</sup>

It is unclear whether bruxism and muscle hyperactivity can cause pain and vice versa. If such a relationship existed, it would suggest the existence of a vicious cycle between these variables.<sup>11-19</sup> Some authors,<sup>20-35</sup> however, have not found any strong relationship between these factors. Different confusing factors are involved in this scenario: One is the heterogeneity of the diagnostic method. Muscle or articular pain can be considered separately; on the other hand, TMD are considered diseases. Plus, there is bias inherent in assessment of SB (eg, self-report, dental wear).<sup>36</sup> In addition, 2 types of bruxers (with and without MFP) have been detected and described.<sup>23</sup>

The aim of this study was to test for an association between rhythmic masticatory muscle activity (RMMA) diagnosed according to polysomnographic (PSG) criteria for SB (RMMA-SB)<sup>37</sup> and MFP, as well as the chance of occurrence of MFP in patients with RMMA-SB. Because the PSG diagnostic criteria for SB are based on RMMA and episodes with grinding noises, the term RMMA-SB is used to distinguish this entity from SB recognized by means of clinical criteria (including self-report, report from sleep partner of tooth grinding, fatigue or soreness of masticatory muscles on awakening, dental wear,<sup>38</sup> or masseter hypertrophy). For reasons of convenience, subjects with RMMA above the cutoff values of PSG criteria<sup>37</sup> of SB are referred to in the present study as sleep bruxers. The hypothesis tested was that a statistical association between RMMA-SB and MFP does exist.

## Materials and Methods

### Population and Selection Criteria

The study protocol was approved by the Local Ethics Committee, Bauru School of Dentistry, University of São Paulo, Brazil. First, informed consent was obtained from all subjects. Patients were examined at the orofacial pain clinic of this university.

The exclusion criteria for the whole sample were: more than 2 missing posterior teeth (exclud-

ing third molars) or the presence of a removable dental prosthesis; presence of gross malocclusion (overjet or overbite greater than 6 mm, unilateral or anterior crossbite, centric relation/maximal intercuspal position discrepancy greater than 5 mm); use of medications with possible effects on sleep or motor behavior (eg, benzodiazepine, L-dopa, neuroleptics, antidepressants, and/or alcohol or drug abuse); and presence of major neurologic or psychiatric disorders, as assessed through a specific questionnaire. Subjects presenting with sleep disorders, such as orofacial or cervical myoclonus, narcolepsy, insomnia, periodic leg movements (PLM) during sleep (with an index of more than 10 events per hour of sleep), electroencephalographic (EEG) evidence of epileptiform activity, and sleep apnea (with an index above 5 events per hour of sleep), confirmed during the PSG examination,<sup>39,40</sup> were also excluded. The examiner was blinded to the possible presence of parafunctional habits. Thus, the SB diagnosis was established only after PSG recording. For all subjects, the examination included a questionnaire about sleep disorders, medication intake, drug or alcohol abuse, motor or neurologic disorders, and general health. An occlusal examination was also performed.

To classify the subjects into the MFP or control groups, a specific questionnaire was used to screen for pain complaint and pain location as well as pain frequency, intensity, and history. Subjects were asked to record their pain intensity at rest by means of a visual analog scale (VAS). The VAS consisted of a 100-mm line oriented horizontally, with the left endpoint of the scale indicating "no pain at all" and the right endpoint corresponding to "the worst pain I can now imagine." Questions about the presence of articular pain and sounds, restriction of mandibular movement or locking, mandibular luxation, and headache were also asked. Also, an anamnestic questionnaire<sup>41</sup> composed of 10 questions was used to assess the intensity of TMD.

The physical TMD examination included functional evaluation of the temporomandibular joint (TMJ), mouth opening pattern, vertical range of mandibular motion (unassisted opening without pain), TMJ sound inspection, and palpation. The following muscles (right side and left side count as separate sites for each muscle) were examined: temporalis (anterior, middle, and posterior); masseter (origin, body, insertion, and deep portion); submandibular region (medial pterygoid, suprahyoid, anterior digastric region); and posterior mandibular region (stylohyoid/posterior digastric region). Digital pressure exerted during palpation

by the examiner was previously calibrated with the aid of an algometer (Kratos). Recommended values of 1.0 kg-ft and 1.5 kg-ft were used for the TMJ and muscles, respectively.<sup>42</sup> All examinations were carried out by the same examiner. Muscle and TMJ palpation was performed following the instructions of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).<sup>42</sup> Subjects were asked to determine whether the palpation hurt (painful) or whether merely a sensation of pressure was felt (scored as 0). If it hurt, the subjects were asked to indicate whether the pain was mild (1), moderate (2), or severe (3). These scores were employed in the calculation of score of sensitivity.

The criteria for MFP diagnosis were: report of pain or ache in the jaw, temples, face, preauricular area, or inside the ear at rest or during function for at least 2 months, accompanied by tenderness to palpation in 3 or more of the 18 palpated muscle sites (RDC/TMD).<sup>42</sup> For the control group, the inclusion criterion was the total absence of TMD signs and symptoms, except for asymptomatic clicking because of its high prevalence in the general population.<sup>9,43</sup>

Initially, 142 patients were screened. Eighty-two individuals were excluded for the following reasons: pain or articular dysfunction (42), age (6), odontogenic pain (3), unwillingness to complete the study (4), chief complaint of tinnitus (2), sleep disorders (5), sleep disorders diagnosed after PSG (4), malocclusion or tooth loss (10), TMD signs in symptom-free individuals (5), and extraoral orthodontic appliance (1). The final sample was composed of 30 MFP subjects (24 women and 6 men) with a mean ( $\pm$  standard deviation [SD]) age of  $26.6 \pm 5.0$  years (range, 19 to 39 years) and 30 healthy subjects ("controls"; 24 women and 6 men) with a mean age of  $26.0 \pm 4.5$  years (range, 20 to 42 years), fulfilling the requirements of this study.

### Polysomnography

PSG recordings of all selected subjects were carried out in the sleep laboratory for 2 consecutive nights, as suggested by the American Academy of Sleep Medicine.<sup>6</sup> The first night was used for adaptation to the environment and to rule out other sleep disorders, and the second night was used to collect experimental data. PSG recordings were performed in a dark, sound-attenuated, and temperature-controlled room. The mean time of the start of sleep recording was 11:30 PM, and the finishing time was 6:00 AM or upon the subject's spontaneous awak-

ening. Sleep was recorded and scored by a standard method.<sup>44</sup> This method specifies electrode placement and scoring criteria for sleep stages based on 3 parameters: EEG, electro-oculographic (EOG) activities, and chin-electromyographic (EMG) activities. Video cameras were focused on the head and neck area, and audio recordings were made throughout the night. Audio and visual recordings of the orofacial area were analyzed simultaneously to rule out SB-nonspecific activity as well as to confirm the presence of tooth grinding over other oral sounds. In addition to chin/submental EMG activity (essential for sleep stage scoring), EMG activities were recorded also from the right and left masseter and anterior tibialis muscles. Prior to sleep recording, each patient performed a series of 10 tasks of 2 seconds' duration to allow for signal recognition and calibration of EMG amplification. The 10 tasks were 3 voluntary clenches (maximal intercuspal occlusion), including a maximum voluntary contraction; 1 voluntary clenching with moderate contraction; 1 voluntary clenching with light contraction; lateral right and left mandibular movements and contraction of masticatory muscles at the end of each movement; protrusive mandibular movement and contraction of masticatory muscles at the end of movement; mouth opening and closing; and swallowing.

### PSG Analysis

PSG analysis was done by a researcher who was blinded to subject status (MFP or control).

**Sleep.** The following sleep parameters were calculated (Table 1): total sleep time, sleep efficiency (% of actual time asleep), sleep latency (time before first sleep stage 1), first rapid eye movement (REM) sleep stage latency, number of microarousals per hour, and percentage of time spent in each sleep stage per 30-second epoch.

**Leg Motor Activity.** EMG activities from both anterior tibialis muscles were analyzed visually, and periodic leg movements during sleep (PLMS) were scored according to the method of Coleman (see Montplaisir et al<sup>45</sup>). The PLM diagnosis was positive if at least 10 events per hour of sleep were scored. Two subjects met this criterion. They were excluded from the study and replaced by other volunteers screened with the same method.

**Apnea/Hypopnea.** The number of apneic/hypopneic events was scored according to American Academy of Sleep Medicine criteria.<sup>46</sup> One subject presented more than 5 events of apnea/hypopnea events per hour and was also excluded from the study and replaced by another volunteer.

**Table 1** Sleep Variables (Means  $\pm$  SDs or Medians) in MFP and Control Groups

Sleep variable	Control group	MFP group	<i>P</i>
Sleep latency (min)	13.74	10.11	.214 <sup>†</sup>
Recording time (h)	6.431 $\pm$ 0.584	6.539 $\pm$ 0.715	.524*
Sleep efficiency (%)	92.400	92.350	.762 <sup>†</sup>
Total sleep time (h)	5.824 $\pm$ 0.568	5.969 $\pm$ 0.609	.345*
Microarousals (no./h)	5.750	5.500	.337 <sup>†</sup>
Sleep stage 1 (%) <sup>‡</sup>	9.150	8.950	.877 <sup>†</sup>
Sleep stage 2 (%) <sup>‡</sup>	50.897 $\pm$ 7.169	51.980 $\pm$ 8.783	.603*
Sleep stage 3 or 4 (%) <sup>‡</sup>	19.183 $\pm$ 5.315 <sup>a</sup>	19.230 $\pm$ 7.394 <sup>a</sup>	.978*
Sleep stage REM (%) <sup>‡</sup>	19.883 $\pm$ 4.624 <sup>a</sup>	18.437 $\pm$ 6.632 <sup>a</sup>	.331*
Phasic episodes (%) <sup>§</sup>	70.38 $\pm$ 16.67 <sup>a</sup>	65.55 $\pm$ 25.32 <sup>a</sup>	.391*
Tonic episodes (%) <sup>§</sup>	12.08 $\pm$ 6.25 <sup>b</sup>	17.44 $\pm$ 1.11 <sup>b</sup>	.104 <sup>†</sup>
Mixed episodes (%) <sup>§</sup>	16.67 $\pm$ 10.09 <sup>b</sup>	12.65 $\pm$ 14.48 <sup>b</sup>	.217*

*P* values are for intergroup analysis (<sup>†</sup>t test; <sup>‡</sup>Mann-Whitney test).

\*Intragroup analysis for time of sleep stages: Kruskal-Wallis multiple-comparison Z value test. Equal letters represent statistically similar values.

<sup>§</sup>Intragroup analysis (t test or Mann-Whitney test).

**Jaw Muscle Activity.** All masticatory EMG activities with an amplitude of at least 20% of the maximum voluntary contraction not associated with another SB-nonspecific activity (eg, coughing, sleep talking, grimacing)<sup>47</sup> were retained for analysis. According to Lavigne et al,<sup>37</sup> this threshold corresponded to the root mean square EMG signal that was the most frequently associated, when controlled with audiovisual signals, to the beginning of a bruxism episode. Events were defined and scored as 3 different types of episodes<sup>37</sup>: phasic (rhythmic), tonic (sustained), or mixed (both phasic and tonic). A phasic episode corresponds to at least 3 EMG bursts of 0.25 to 2.0 seconds' duration, separated by 2 interburst intervals. A tonic episode corresponds to an EMG burst lasting more than 2.0 seconds. A mixed episode corresponds to phasic and tonic episodes separated by an interval lasting less than 2.0 seconds. The intrarater reliability of the bruxism scorer was assessed on 2 occasions from a sample of 87 bruxism episodes selected blindly across the study population. The kappa coefficient demonstrated fair to high agreement<sup>48</sup> to phasic ( $\kappa = 0.73$ ), mixed ( $\kappa = 0.71$ ), and tonic ( $\kappa = 0.59$ ) episodes.

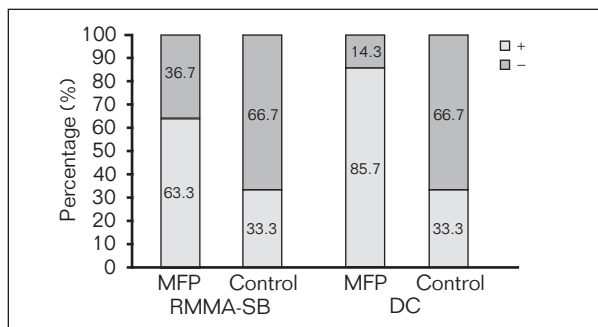
After the end of the experimental phase of this study (about 1 week), 58 of the same subjects (28 MFP patients and 30 controls) were contacted again for the assessment of daytime clenching, since this question was not present on the original questionnaire. They were asked, "Do you clench your teeth during the day?" Two patients could not be contacted because they had moved out of the city. The answers were registered dichotomously (yes or no).

## Statistical Analyses

Statistical analyses were performed on sleep and jaw muscle activity parameters. Significance was determined with independent-sample *t* tests for normally distributed variables and with the Mann-Whitney test when the data were not normally distributed (SigmaStat 2.0, Jandel Scientific). When the distribution was normal, parametric tests were performed, and descriptive values were represented as means  $\pm$  SDs. When the distribution was non-normal, nonparametric tests were performed, and descriptive values were expressed as medians followed by maximum and minimum values. The chi-square test was used to verify possible associations between RMMA-SB and MFP, daytime clenching and MFP, report of period of worst pain and RMMA-SB, and RMMA-SB and daytime clenching. Odds ratios (ORs) and confidence intervals (CIs) were also calculated. A 5% level of significance was adopted.

## Results

The mean VAS pain intensity of MFP patients was 39  $\pm$  27 mm. Of the MFP patients, 46.7% (14) reported mild pain, 43.3% (13) reported moderate pain, and only 10% (3) reported severe pain. No subjects in the control group reported pain, in accordance with the selection criteria. According to the TMD index,<sup>49</sup> 1 MFP patient (3.3%) was classified as having no TMD, 14 (46.7%) as having mild TMD, 11 (36.7%) as having moderate TMD, and 4 (13.3%) as having severe TMD.



Twenty-six controls (86.7%) were classified as having no TMD, and 4 (13.3%) were determined to have mild TMD. There was a significant difference in the TMD index<sup>49</sup> between the 2 groups:  $8.8 \pm 4.2$  for MFP patients and  $1.87 \pm 1.69$  for controls ( $P < .001$ ).

A statistically (but not clinically) significant decrease in mouth opening was found for the MFP patients ( $46.97 \pm 6.53$  mm) when compared to controls ( $50.43 \pm 6.47$  mm;  $P = .043$ ). Seventeen MFP patients (56.7%) reported that the morning was the time of the worst pain, 6 (20%) reported afternoon, and 4 (13.3%) reported night. Three (10.0%) could not define the worst time of day. Nine (30%) controls and 7 (23.3%) MFP patients reported articular clicking ( $P = .662$ ). No significant association was found between sleep bruxers and the report of worst pain in the morning (Fisher exact test,  $P = .706$ ). Eighty-six percent of MFP patients reported frequent headaches. The MFP patients presented significantly more sites of tenderness ( $7.8 \pm 4.01$ ) than controls ( $0.73 \pm 0.94$ ,  $t$  test;  $P < .01$ ) and a significantly higher score of sensitivity among tenderness sites ( $0.65 \pm 0.42$ ) than controls ( $0.06 \pm 0.07$ ,  $t$  test;  $P < .001$ ).

## RMMA

No significant differences were found between control and MFP patients when sleep variables were considered (Table 1). According to the previously established PSG criteria,<sup>37</sup> the MFP group ( $n = 30$ ) had 19 (63.3%) sleep bruxers and 11 (36.7%) non-sleep bruxers, whereas the control group comprised 10 (33.3%) sleep bruxers and 20 (66.7%) non-sleep bruxers ( $n = 30$ ; Fig 1). Among bruxers ( $n = 29$ ), there were 19 (65.5%) MFP patients and 10 (34.5%) controls. Among non-bruxers ( $n = 31$ ), there were 11 (35.5%) MFP patients and 20 (64.5%) controls. Significant associations were found between RMMA-SB and MFP (chi-square 4.27;  $P = .04$ ; OR 3.45; 95% CI, 1.07 to 11.19).

**Fig 1** Percentages of MFP patients and control subjects, classified follows: RMMA-SB (+) = subjects with RMMA-SB; RMMA-SB (-) = subjects whose RMMA did not reach the polysomnographic criteria for sleep bruxism; DC (+) = subjects who reported daytime clenching; and DC (-) = subjects who did not report daytime clenching.

## Daytime Clenching

Fifty-eight (58) subjects (28 MFP patients and 30 controls) were asked to report possible existence of daytime clenching. Twenty-four (85.7%) MFP patients and 10 (33.3%) controls reported daytime clenching, and a significant association was found (chi-square 14.29;  $P < .001$ ; OR 12.0; 95% CI, 3.26 to 44.15) between daytime clenching and MFP (Fig 1). Among the entire sample, those with daytime clenching had a mean age of  $27.12 \pm 2.27$  years; 28 were women and 6 were men. Those without daytime clenching had a mean age of  $25.5 \pm 3.71$  years (19 women and 5 men;  $t$  test between groups,  $P = .201$ ). To assess whether daytime clenching would have contributed to the severity of pain among MFP patients, some clinical variables were compared between MFP patients with and without daytime clenching (Table 2). The multiple logistic regression model revealed a significant influence of daytime clenching ( $P < .001$ ) and RMMA-SB ( $P = .006$ ) as independent variables on the presence of MFP (group as dependent variable). Despite its major prevalence in the MFP group (70.6%), daytime clenching did not exert a significant influence on mean VAS of MFP patients (linear regression,  $P = .885$ ). No significant association between self-report of daytime clenching and the report of worst pain on evening was found (Fisher exact test,  $P = .613$ ). Eleven subjects (45.8%) in the daytime clenching group and 1 (25%) who did not report daytime clenching reported that the afternoon/evening was the period of worst pain. No significant difference on evening VAS was found between MFP patients who reported daytime clenching and those who did not ( $t$  test,  $P = .846$ ).

## Oromotor Activities

There was no difference between MFP patients and controls in the percentages of phasic, tonic, and mixed episodes (Table 1). Asymptomatic sleep brux-



**Table 2 Clinical Comparison of MFP Patients With and Without Daytime Clenching (DC)**

Clinical variable	With DC (n = 24)	Without DC (n = 4)	P
Sex distribution	19 women, 5 men	4 women, 0 men	.313*
Age (mean ± SD, in y)	27.58 ± 5.05	23.25 ± 2.50	.108 <sup>†</sup>
Mean VAS (mean ± SD, in mm)	36.33 ± 23.66	52.93 ± 28.00	.215 <sup>†</sup>
Evening VAS (mean ± SD, in mm)	13.38 ± 16.74	20.25 ± 15.17	.449 <sup>†</sup>
Pain intensity report	11 with mild pain, 11 with moderate pain, 2 with severe pain	1 with mild pain, 2 with moderate pain, 1 with severe pain	.536*
TMD index (mean ± SD)	9.00 ± 3.55	10.00 ± 7.26	.659 <sup>†</sup>
Mouth opening (mean ± SD, in mm)	47.54 ± 6.76	47.50 ± 6.56	.579 <sup>†</sup>
Pain duration (median, in mo)	24.00	12.00	.450 <sup>†</sup>
Joint noise	7 (29.17%)	0 (0%)	.212*

\*Chi-square test.

<sup>†</sup>t test.

\*Mann-Whitney test.

**Table 3a Analysis of Sleep Bruxers (MFP Versus Controls) for Subtypes of Episodes (Phasic, Tonic, or Mixed)**

Episode type	MFP (n = 19) (mean ± SD or median)	Control (n = 10) (mean ± SD or median)	P
Phasic episodes (%)	72.00 <sup>a</sup>	62.59 <sup>a</sup>	.281 <sup>†</sup>
Tonic episodes (%)	13.09 ± 7.34 <sup>b</sup>	20.90 ± 9.72 <sup>b</sup>	.022*
Mixed episodes (%)	17.09 ± 10.91 <sup>b</sup>	15.53 ± 7.96 <sup>b</sup>	.692*

\*t test; <sup>†</sup>Mann-Whitney test.

P values are for intergroup analysis. Same superscript letters represent lack of statistically significant difference for intragroup analysis.

**Table 3b Analysis of Sleep Bruxers (With DC Versus Without DC) for Subtypes of Episodes (Phasic, Tonic, or Mixed)**

Episode type	With DC (n = 17) (mean ± SD)	Without DC (n = 12) (mean ± SD)	P
Phasic episodes (%)	70.68 ± 14.80 <sup>a</sup>	63.72 ± 12.64 <sup>a</sup>	.197*
Tonic episodes (%)	13.14 ± 8.11 <sup>b</sup>	19.52 ± 8.11 <sup>b</sup>	.056*
Mixed episodes (%)	16.41 ± 11.32 <sup>b</sup>	16.75 ± 7.85 <sup>b</sup>	.929*

P values are for intergroup analysis. Same superscript letters represent lack of statistically significant difference for intragroup analysis.

\*t test.

ers, however, presented significantly higher percentages of tonic episodes than sleep bruxers with MFP (Tables 3a and 3b). Both sleep bruxers and non-sleep bruxers had significantly more phasic RMMA episodes than tonic or mixed episodes (Table 4).

None of the sleep variables showed any significant difference between sleep bruxers and non-sleep bruxers (Table 4). Table 5 shows the RMMA indices (numbers of episodes/night, episodes/hour, bursts/hour, bursts/episode, and episodes with grinding noise). All these indices

were statistically greater in sleep bruxers than in non-sleep bruxers. The diagnostic values were reported as sensitivity of 72% for the number of episodes/hour, 78% for the number of bursts/hour, and 78% for the number of episodes with grinding noise and specificity of 94% for the number of episodes/hour, 100% for the number of bursts/hour, and 94% for the number of episodes of grinding sounds during the night.<sup>37</sup>

The percentages of episodes per sleep stage were not statistically different (Table 5), reflecting the

**Table 4 Comparison of Sleep Variables (Means ± SDs or Medians) Between Sleep Bruxers and Non-Sleep Bruxers**

Sleep variable	Sleep bruxers (n = 29)	Non-sleep bruxers (n = 31)	P
Sleep latency (min)	10.50	12.00	.383*
Recorder time (h)	6.426 ± 0.669	6.539 ± 0.638	.505
Sleep efficiency (%)	93.700	91.800	.107*
Total sleep time (h)	5.903 ± 0.591	5.891 ± 0.596	.936 <sup>†</sup>
Microarousals (no./h)	5.900	4.800	.280*
Sleep stage 1 (%)	9.600	8.600	.178*
Sleep stage 2(%)	51.755 ± 7.248	51.142 ± 8.695	.769 <sup>†</sup>
Sleep stage 3 or 4 (%)	17.893 ± 6.799	20.177 ± 5.871	.172 <sup>†</sup>
Sleep stage REM (%)	19.035 ± 5.457	19.277 ± 6.035	.870 <sup>†</sup>
Phasic episodes (%)	67.80 ± 14.15 <sup>a</sup>	68.12 ± 26.93 <sup>a</sup>	.955 <sup>†</sup>
Tonic episodes (%)	15.38 <sup>b</sup>	13.04 <sup>b</sup>	.739*
Mixed episodes (%)	16.55 ± 9.87 <sup>b</sup>	12.88 ± 14.54 <sup>b</sup>	.260 <sup>†</sup>

\**t*-test.<sup>†</sup>Mann-Whitney test.

P values are for intergroup analysis. Same superscript letters represent lack of statistically significant difference for intragroup analysis.

**Table 5 RMMA Indices in Sleep Bruxers and Non-Sleep Bruxers**

Variable	Sleep bruxers (n = 29)		Non-sleep bruxers (n = 31)		P
	Mean ± SD	Median (max, min)	Mean ± SD	Median (max, min)	
No. of episodes/night	37.72 ± 11.54	36.00 (78.0, 25.0)	12.90 ± 6.48	11.00 (24.0, 3.0)	< .001*
No. of episodes/hour	6.40 ± 1.91	7.78 (13.7, 4.2)	2.14 ± 0.99	2.08 (3.8, 0.5)	< .001*
No. of bursts/hour	28.39 ± 7.28	28.12 (49.3, 12.2)	8.91 ± 5.33	8.56 (19.4, 1.2)	< .001*
No. of bursts/episode	4.55 ± 0.92	4.6 (7.0, 2.6)	3.97 ± 1.24	4.00 (7.3, 1.3)	.044*
No. of episodes with grinding noise	16.14 ± 9.61	17 (32.0, 2.0)	0.0 ± 0.0	0.0 (0.0, 0.0)	< .001 <sup>†</sup>
Episodes in stage 1 (%)	16.36 ± 3.57	16.00 (27.00, 11.00) <sup>a§</sup>	14.35 ± 8.69	12.50 (30.00, 0.00) <sup>a†</sup>	.153 <sup>†</sup>
Episodes in stage 2 (%)	64.52 ± 9.28	66.00 (78, 40) <sup>§</sup>	67.48 ± 8.60	68.00 (80.00, 55.00) <sup>†</sup>	.399 <sup>†</sup>
Episodes in stage 3 or 4 (%)	6.76 ± 3.57	7.00 (13.04, 0.00) <sup>b§</sup>	5.96 ± 6.07	5.88 (50.00, 0.00) <sup>†</sup>	.544 <sup>†</sup>
Episodes in stage 5 (%)	12.75 ± 8.08	11.00 (37.93, 2.5) <sup>ab§</sup>	12.48 ± 7.81	12.00 (46.67, 0.00) <sup>a†</sup>	.745 <sup>†</sup>

Same letters represent statistically similar values in intra-group analysis of percentage number of episodes per sleep stage.

\**t* test (intergroup analysis); <sup>†</sup>Mann-Whitney test (intergroup analysis); <sup>‡</sup>one-way analysis of variance followed by Tukey test (intragroup analysis); <sup>§</sup>Kruskal-Wallis 1-way analysis of variance on ranks followed by Dunn test (intragroup analysis).

similar sleep macrostructure in these groups. Based on the PSG criteria, the MFP sleep bruxer subgroup had 16 women and 3 men (mean age 27.26 ± 5.07 years), while the MFP non-sleep bruxers included 8 women and 3 men (mean age 25.54 ± 4.82 years) (*t* test for age, *P* = .149). No differences were detected between MFP sleep bruxers and non-sleep bruxers in TMD indices (*t* test, *P* = .519); vertical range of motion of mandible (*t* test, *P* = .139); time of pain onset in months (Mann-Whitney test, *P* = .863); presence of articular

sounds (Fisher exact test, *P* = .371); number of tender muscle sites (*t* test, *P* = .733); and sensitivity score (*t* test, *P* = .722).

Eleven (57.9%) MFP sleep bruxers and 5 (45.5%) MFP non-sleep bruxers reported the morning as the period of worst pain. No association was detected between the period of worst pain and RMMA-SB (*P* = .706). No statistical differences in RMMA indices were found when the period of the worst pain (morning or other period) was considered, regardless of the presence

**Table 6** Comparison of RMMA Indices of MFP Sleep Bruxers (n = 19) Who Reported the Worst Pain at Morning and Those Who Reported Worst Pain at Other Periods

Variable	Morning (n = 11)		Other periods (n = 8)		P*
	Mean ± SD	Max, min	Mean ± SD	Max, min	
No. of episodes/night	40.27 ± 14.88	78.0, 25.0	36.13 ± 10.90	52.0, 25.0	.514
No. of episodes/h	6.67 ± 2.59	13.7, 4.4	6.26 ± 1.67	8.9, 4.2	.703
No. of bursts/h	30.39 ± 7.98	49.3, 20.6	29.47 ± 6.35	36.8, 21.6	.790
No. of bursts/episode	4.81 ± 0.96	6.3, 2.6	4.81 ± 0.99	7.04, 4.09	.988

of RMMA-SB (Table 6). In addition, no significant differences were found for evening (*t* test, *P* = .487) and morning (Mann-Whitney test, *P* = .813) VAS pain intensity between MFP sleep bruxers and non-sleep bruxers.

## Discussion

The prevalence of TMD is much higher in women, regardless of general population or clinical settings.<sup>40,49</sup> This was very well observed in the present study, where the MFP group was 80% female. Some characteristics of the MFP sample used in this study are very similar to those previously described.<sup>42,50–52</sup> The mean pain intensity (VAS, 39 mm) was slightly lower than that reported by Lobbezoo-Scholte et al<sup>52</sup> (52 mm). The low frequency of articular clicking (23.3%) and the high levels of frequent headache (86.2%) are also in agreement with Lobbezoo-Scholte et al,<sup>51</sup> who found that 57% of myogenic TMD patients reported recurrent headache. The MFP group was composed predominantly of patients with mild and moderate pain (90.0%); this was probably a result of the exclusion of patients with more than 1 diagnosis (based on the RDC/TMD) and those with depression and sleep disturbances. The coexistence of other TMD problems and/or a major contributory condition certainly would cause more severe pain sensation. Stohler<sup>2</sup> reported that only a small percentage of subjects continued to be diagnosed only with a muscular condition at follow-up examinations after 1 year (23%), 3 years (13.3%), and 5 years (6.7%). Associations between sleep disorders and pain,<sup>53–55</sup> particularly chronic myogenic disorders,<sup>56,57</sup> and between chronic myofascial pain and psychologic disorders, particularly anxiety and depression, as well as obsessive-compulsive behavior and hostility, have been reported.<sup>56</sup>

The possible causal relationship between SB and MFP is still a matter of controversy. An inherent bias in the SB diagnosis based on self-report or

dental wear,<sup>5,58,59</sup> partial sample studies,<sup>13,58,60</sup> TMD heterogeneity, and SB variability is a possible reason. In addition, the lack of evidence of a causal relationship has been reported by several authors.<sup>20,21,23–35</sup> The results of the present study show a significant association between RMMA-SB and MFP. This parameter provides information only about the role of RMMA-SB as a risk factor for MFP, and it is not possible to assume any cause-and-effect relationship. In agreement with our findings, a significant association between bruxism and TMD has been reported in the literature.<sup>51,60–67</sup> The prevalence of asymptomatic subjects in the RMMA-SB group (34.5%) agrees with the findings of Camparis et al<sup>68</sup> (30%), and the proportion of sleep bruxers in the control group (33.3%) also agrees with Manfredini et al<sup>69</sup> (36.2%). The present study found approximately 3 times more episodes per night and episodes per hour and 1.5 times more bursts/episode in sleep bruxers than in non-sleep bruxers; this is in agreement with figures reported by Lavigne et al.<sup>37</sup> Other authors have found episodes of RMMA predominantly in the non-REM sleep stage 2,<sup>37,70</sup> which agrees with the present data (64.5% and 67.5% of RMMA for sleep bruxers and non-sleep bruxers, respectively). The predominance of phasic episodes agrees with Lavigne et al,<sup>37</sup> although a mild discrepancy in the percentages exists between the studies. This could be attributed to interindividual variations in the screening of episodes (about 15%). A novel finding was that asymptomatic sleep bruxers presented a significantly greater percentage of tonic episodes than MFP sleep bruxers.

The OR represents the ratio of the probability of MFP occurring to the probability that it will not occur, after exposure to a risk factor—in this case, SB. The OR value obtained here is similar to those reported elsewhere for self-report of bruxism and other signs/symptoms of TMD: difficulty in closing the mouth (OR 2.84),<sup>63</sup> craniofacial pain (OR 1.84),<sup>63</sup> orofacial pain (relative risk 1.6),<sup>65</sup> articular



sounds (ORs of 1.64<sup>63</sup> and 3.3<sup>67</sup>), and stiffness/locking (relative risk 2.7).<sup>65</sup> According to a scale of magnitudes for effect statistics,<sup>71</sup> OR values below 3.5 represent a small effect of a risk factor (in this case, RMMA-SB) in the occurrence of a disease (in this case, MFP).

Although most sleep bruxers had indicated the morning as the period of worst pain, there was not a significant association between these variables. Moreover, the lack of difference in RMMA indices between sleep bruxers who reported the morning as the period of worst pain and those who did not supports the lack of association between these variables. Van Selms et al<sup>72</sup> reported no influence of nocturnal masticatory muscle activity on the reported morning jaw muscle pain in a single case study of a 53-year-old woman with clinical evidence of SB during a period of 13 weeks. Some PSG-controlled studies on the relationship between bruxism and masticatory muscle pain suggest that masticatory muscle pain is associated with a decrease rather than an increase in RMMA.<sup>22,73</sup> However, the present study only examined motor activity in relation to pain in MFP TMD patients. This may be different from those studies<sup>22,37</sup> that assessed pain in clinically recognized sleep bruxers. Yet the diagnostic criteria of pain were not the same as used in the present study. The sample of one study<sup>22</sup> was composed of subjects who were aware of signs or symptoms of sleep oromotor activity and who were divided into a group with clinical pain complaints (report of jaw stiffness, tightness, and muscle pain or soreness in the mornings) and a group without pain. The other study<sup>73</sup> assessed a sample of patients with a chief complaint of SB and reports of concomitant nonmyofascial jaw muscle pain. These methodologic distinctions make comparison with the present findings difficult.

The higher values of chi-square and ORs found between reports of daytime clenching and MFP than between RMMA-SB and MFP are in accordance with the findings of van Selms et al,<sup>72</sup> who observed that evening jaw muscle pain could be explained by variations in daytime clenching but that variations in morning jaw muscle pain could not be explained by fluctuations in SB as established by EMG recordings. Hence, as assumed by these authors,<sup>72</sup> the causal relationship between nocturnal bruxism and jaw muscle pain in the morning is less obvious than previously assumed. In accordance with such observations, the ORs for daytime clenching obtained in the present study (12.0) are considered large, according to the magnitude scale of Cohen.<sup>74</sup> In addition, the chi-square value (14.29,  $P < .001$ ) and the multiple logistic

regression model ( $P < .001$ ) point to daytime clenching as a more representative risk factor for MFP than RMMA-SB ( $= .006$ ). Perhaps because daytime clenching was dichotomously assessed in our sample, while van Selms et al carried out their assessment with the aid of a 5-point scale, and because of the small sample size (MFP sleep bruxers and non-sleep bruxers), differences in evening VAS values between those who reported daytime clenching and those who did not were not found. Yet daytime clenching was not assessed objectively and is susceptible to bias inherent in self-reports. Because the evaluation of daytime clenching activity was not the major aim of this study, the sample size, sample distribution, and the method of assessment of this parafunctional activity constitute limitations in the interpretation of the results. These aspects should be considered in further analyses.

The present cross-sectional study supports the model that RMMA during sleep is associated with MFP and constitutes a risk factor (although small) for MFP. Daytime clenching may represent a significant risk factor for MFP.

## Acknowledgments

This work was supported by the Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP - 02/03469-5) and by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), Brazil.

## References

1. Mense S. Nociception from skeletal muscle in relation to clinical muscle pain. *Pain* 1993;54:241–289.
2. Stohler CS. Muscle-related temporomandibular disorders. *J Orofac Pain* 1999;13:273–284.
3. Raphael KG, Marbach JJ. Widespread pain and the effectiveness of oral splints in myofascial face pain. *J Am Dent Assoc* 2001;132:305–316.
4. Raphael KG, Marbach JJ, Klausner J. Myofascial face pain. Clinical characteristics of those with regional vs. widespread pain. *J Am Dent Assoc* 2000;131:161–171.
5. Raphael KG, Marbach JJ, Klausner JJ, Teaford MF, Fischhoff DK. Is bruxism severity a predictor of oral splint efficacy in patients with myofascial face pain? *J Oral Rehabil* 2003;30:17–29.
6. American Academy of Sleep Medicine. *The International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual*, ed 2. Westchester, IL: American Academy of Sleep Medicine, 2005.
7. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil* 2001;28:1085–1091.
8. Glaros A. Incidence of diurnal and nocturnal bruxism. *J Prosthet Dent* 1981;45:545–549.

9. Rieder CE, Martinoff JT, Wilcox SA. The prevalence of mandibular dysfunction. Part I: Sex and age distribution of related signs and symptoms. *J Prosthet Dent* 1983; 50:81–88.
10. Goulet JP, Lund JP, Montplaisir J, Lavigne G. Daily clenching, nocturnal bruxism, and stress and their association with TMD symptoms [abstract]. *J Orofac Pain* 1993; 7:120.
11. Clark GT, Beemstermoer PL, Rugh JD. Nocturnal masseter muscle activity and the symptoms of masticatory dysfunction. *J Oral Rehabil* 1981;8:279–286.
12. Laskin DM. Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 1969;79:147–153.
13. Molina OF, Santos J Jr, Nelson SJ, Nowlin T. A clinical study of specific signs and symptoms of CMD in bruxers classified by the degree of severity. *J Craniomandib Pract* 1999;17:268–279.
14. Molina OF, Santos J Jr, Mazzetto M, Nelson S, Nowlin T, Manieri ET. Oral jaw behaviors in TMD and bruxism: A comparison study by severity of bruxism. *J Craniomandib Pract* 2001;19:114–122.
15. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. *Adv Neurol* 1988;49:329–341.
16. Schwartz L. *Disorders of the Temporomandibular Joint*. Philadelphia: Saunders, 1959.
17. Travell J, Rinzler S, Herman M. Pain and disability of the shoulder and arm: Treatment by intramuscular infiltration with procaine hydrochloride. *J Am Dent Assoc* 1942;120: 417–422.
18. Ware JC, Rugh JD. Destructive bruxism: Sleep stage relationship. *Sleep* 1988;11:172–181.
19. Yemm R. A neurophysiological approach to the pathology and etiology of temporomandibular dysfunction. *J Oral Rehabil* 1985;12:343–353.
20. Arima T, Svensson P, Arendt-Nielsen L. Experimental grinding in healthy subjects: A model for postexercise jaw muscle soreness. *J Orofac Pain* 1999;13:104–114.
21. Arima T, Svensson P, Arendt-Nielsen L. Capsaicin-induced muscle hyperalgesia in the exercised and non-exercised human masseter muscle. *J Orofac Pain* 2000;14: 213–223.
22. Arima T, Arendt-Nielsen L, Svensson P. Effect of jaw muscle pain and soreness evoked by capsaicin before sleep on orofacial motor activity during sleep. *J Orofac Pain* 2001;15:245–256.
23. Dao TTT, Lund JP, Lavigne GJ. Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. *J Orofac Pain* 1994;8: 350–356.
24. Dao TTT, Lund JP, Lavigne GJ. Pain responses of experimental chewing in myofascial pain patients. *J Dent Res* 1994;73:1163–1167.
25. Graven-Nielsen T, Lund H, Arendt-Nielsen L, Danneskiold-Samsøe B, Bliddal H. Inhibitory of maximal voluntary contraction force by experimental muscle pain: A centrally mediated mechanism. *Muscle Nerve* 2002; 25:576–584.
26. Junge D, Clark GT. Electromyographic turns analysis of sustained contraction in human masseter muscles at various isometric force levels. *Arch Oral Biol* 1993;38: 583–588.
27. Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: A discussion of the relationship between chronic musculoskeletal pain and motor activity. *Can J Physiol Pharmacol* 1991;69:683–694.
28. Mense S, Simons DG. *Muscle Pain: Understanding Its Nature, Diagnosis and Treatment*. Baltimore: Lippincott Williams & Wilkins, 2001.
29. Matre DA, Sinkjaer T, Svensson P, Arendt-Nielsen L. Experimental muscle pain increases the human stretch reflex. *Pain* 1998;75:331–339.
30. Stohler CS, Ashton-Miller JA, Carlson DS. The effects of pain from the mandibular joint and muscles on masticatory motor behavior in men. *Arch Oral Biol* 1988;33: 175–182.
31. Stohler CS, Zhang X, Lund JP. The effect of experimental jaw muscle pain on postural muscle activity. *Pain* 1996; 66:215–221.
32. Svensson P, Arendt-Nielsen L. Effects of 5 days of repeated submaximal clenching on masticatory muscle pain and tenderness: An experimental study. *J Orofac Pain* 1996;10:330–338.
33. Svensson P, Arendt-Nielsen L, Houe L. Sensory-motor interactions of human experimental unilateral jaw muscle pain: A quantitative analysis. *Pain* 1995;64:241–249.
34. Svensson P, Arendt-Nielsen L, Houe L. Muscle pain modulates mastication: An experimental study in humans. *J Orofac Pain* 1998;12:343–353.
35. Svensson P, Graven-Nielsen T. Craniofacial muscle pain: Review of mechanism and clinical manifestations. *J Orofac Pain* 2001;15:117–145.
36. Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? *J Orofac Pain* 1997;11:15–23.
37. Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res* 1996;75:546–552.
38. American Sleep Disorders Association. *The International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual*. Rochester, MN: American Sleep Disorders Association, 1997.
39. Thorpy MJ. Parasomnia. In: American Sleep Disorders Association. *International Classification of Sleep Disorders: Diagnostic and Coding Manual*. Rochester, MN: American Sleep Disorders Association, 1990:142–185.
40. Kato T, Rompré P, Montplaisir JY, Sessle BJ, Lavigne GJ. Sleep bruxism: An oromotor activity secondary to microarousal. *J Dent Res* 2001;80:1940–1944.
41. Conti PC, Ferreira PM, Pegoraro LF, Conti JV, Salvador MC. A cross-sectional study of prevalence and etiology of signs and symptoms of temporomandibular disorders in high school and university students. *J Orofac Pain* 1996;10: 254–262.
42. Dworkin SF, LeResche L (eds). *Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Examinations and Specifications, Critique*. *J Craniomandib Disord* 1992;6:301–355.
43. Goulet JP, Lavigne GJ, Lund JP. Jaw pain prevalence among French-speaking Canadians in Quebec and related symptoms of temporomandibular disorders. *J Dent Res* 1995;74:1738–1744.
44. Rechtschaffen A, Kales A. *A Manual of Standardized Terminology: Techniques and Scoring System for Sleep Stages of Human Subjects*. Los Angeles: Brain Information Service/Brain Research Institute, 1968.
45. Montplaisir J, Godbout R, Pelletier G, Warnes H. Restless legs syndrome and periodic limb movements during sleep. In: Kryger MJ, Roth T, Demet WC (eds). *Principles and Practice of Sleep Medicine*. Philadelphia: Saunders, 1994:589–597.

46. American Academy of Sleep Medicine. Sleep-related breathing disorders in adults: Recommendation for syndrome definitions and measurements techniques in clinical research. *Sleep* 1999;22:667–689.
47. Velly-Miguel AX, Montplaisir J, Rompré PH, Lund JP, Lavigne GJ. Bruxism and other orofacial movements during sleep. *J Craniomandib Disord* 1992;6:71–81.
48. Brunette DM. Reliability, Sensitivity, and Specificity of Diagnostic Tests and Measurements: Critical Thinking, Understanding, and Evaluating Dental Research. Chicago: Quintessence, 1996:99–112.
49. Conti A, Freitas M, Conti P, Henriques J, Janson G. Relationship between signs and symptoms of temporomandibular disorders and orthodontic treatment: A cross-sectional study. *Angle Orthod* 2003;73:411–417.
50. Curran SL, Carlson CR, Okeson JP. Emotional and physiologic responses to laboratory challenges: Patients with temporomandibular disorders versus matched control subjects. *J Orofac Pain* 1996;10:141–150.
51. Lobbezoo-Scholte AM, De Leeuw JRJ, Steenks MH, Bosman F, Buchner R, Olthoff LW. Diagnostic subgroups of craniomandibular disorders. Part I: Self-report data and clinical findings. *J Orofac Pain* 1995;9:24–36.
52. Lobbezoo-Scholte AM, Lobbezoo F, Steenks MH, De Leeuw JR, Bosman F. Diagnostic subgroups of craniomandibular disorders. Part II: Symptom profiles. *J Orofac Pain* 1995;9:37–43.
53. Brousseau M, Manzini C, Thie N, Lavigne G. Understanding and managing the interaction between sleep and pain: An update for the dentist. *J Can Dent Assoc* 2003;69:437–442.
54. Moldofsky H. Sleep and pain. *Sleep Med Rev* 2001;5:385–396.
55. Yatani H, Studts J, Cordova M, Carlson CR, Okeson J. Comparison of sleep quality and clinical and psychologic characteristics in patients with temporomandibular disorders. *J Orofac Pain* 2002;16:221–228.
56. Lindroth JE, Schmidt JE, Carlson CR. A comparison between masticatory muscle pain patients and intracapsular pain patients on behavioral and psychosocial domains. *J Orofac Pain* 2002;16:277–283.
57. Vazquez-Delgado E, Schmidt JE, Carlson CR, DeLeeuw R, Okeson JP. Psychological and sleep quality differences between chronic daily headache and temporomandibular disorders patients. *Cephalalgia* 2004;24:446–454.
58. Kieser JA, Groeneveld HT. Relationship between juvenile bruxism and craniomandibular dysfunction. *J Oral Rehabil* 1998;25:662–665.
59. Pergamalian A, Rudy TE, Zaki HS, Greco CM. The association between wear facets, bruxism, and severity of facial pain in patients with temporomandibular disorders. *J Prosthet Dent* 2003;90:194–200.
60. Kampe T, Tagdae T, Bader G, Edman G, Karlsson S. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. *J Oral Rehabil* 1997;24:581–587.
61. Ahlberg K, Ahlberg J, Kononen M, Alakuijala A, Partinen M, Savolainen A. Perceived orofacial pain and its associations with reported bruxism and insomnia symptoms in media personnel with or without irregular shift work. *Acta Odontol Scand* 2005;63:213–217.
62. Carlsson GE, Egermark I, Magnusson T. Predictors of signs and symptoms of temporomandibular disorders: A 20-year follow-up study from childhood to adulthood. *Acta Odontol Scand* 2002;60:180–185.
63. Ciancaglini R, Gherlone E, Radaelli G. The relationship of bruxism with craniofacial pain and symptoms from the masticatory system in the adult population. *J Oral Rehabil* 2001;28:842–848.
64. Guler N, Yatmaz PI, Ataoglu H, Emlik D, Uckan S. Temporomandibular internal derangement: Correlation of MRI findings with clinical symptoms of pain and joint sounds in patients with bruxing behaviour. *Dentomaxillofac Radiol* 2003;32:304–310.
65. Macfarlane TV, Blinkhorn AS, Davies RM, Worthington HV. Association between local mechanical factors and orofacial pain: Survey in the community. *J Dent* 2003;31:535–542.
66. Magnusson T, Egermark I, Carlsson GE. A longitudinal epidemiologic study of signs and symptoms of temporomandibular disorders from 15 to 35 years of age. *J Orofac Pain* 2000;14:310–319.
67. Magnusson T, Egermark I, Carlsson GE. A prospective investigation over two decades on signs and symptoms of temporomandibular disorders and associated variables. A final summary. *Acta Odontol Scand* 2005;63:99–109.
68. Camparis CM, Formigoni G, Teixeira MJ, Bittencourt LR, Tufik S, Siqueira JT. Sleep bruxism and temporomandibular disorders: Clinical and polysomnographic evaluation. *Arch Oral Biol* 2006;51:721–728.
69. Manfredini D, Ciapparelli A, Dell’Osso L, Bosco M. Mood disorders in subjects with bruxing behavior. *J Dent* 2005;33:485–490.
70. Lavigne GJ, Rompré PH, Poirier G, Huard H, Kato T, Montplaisir JY. Rhythmic masticatory muscle activity during sleep in humans. *J Dent Res* 2001;80:443–448.
71. Hopkins WG. A new view of statistics. Internet Society for Sport Science. Available at: <http://www.sportsci.org/resource/stats/>.
72. van Selms MK, Lobbezoo F, Wicks DJ, Hamburger HL, Naeije M. Craniomandibular pain, oral parafunctions, and psychological stress in a longitudinal case study. *J Oral Rehabil* 2004;31:738–745.
73. Lavigne GJ, Rompre PH, Montplaisir JY, Lobbezoo F. Motor activity in sleep bruxism with concomitant jaw muscle pain. A retrospective pilot study. *Eur J Oral Sci* 1997;105:92–95.
74. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*, ed 2. Mahwah, NJ: Lawrence Erlbaum, 1988.