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Masticatory Muscle Sleep Background EMG Activity is Elevated in Myofascial TMD Patients

Karen G. Raphael[a], **Malvin N. Janal**[b], **David A. Sirois**[a], **Boris Dubrovsky**[c], **Pia E. Wigren**[d], **Jack J. Klausner**[a], **Ana C. Krieger**[e], and **Gilles J. Lavigne**[f]

[a]Dept. of Oral & Maxillofacial Pathology, Radiology & Medicine, New York University College of Dentistry, New York, NY, USA

[b]Dept. of Epidemiology and Health Promotion, New York University College of Dentistry, New York, NY, USA

[c]New York Methodist Hospital, Center for Sleep Disorders Medicine and Research, Brooklyn, NY, USA

[d]Private practice, Stockholm, Sweden

[d]Departments of Medicine, Neurology and Neuroscience, and Genetic Medicine, Weill Cornell Medical College, Cornell University, New York, NY, USA

[e]Faculté de Médecine Dentaire, Université de Montréal, Montréal, Quebec, Canada

Abstract

Despite theoretical speculation and strong clinical belief, recent research using laboratory polysomnographic (PSG) recording has provided new evidence that frequency of sleep bruxism (SB) masseter muscle events, including grinding or clenching of the teeth during sleep, is not increased for women with chronic myofascial temporomandibular disorder (TMD). The current case-control study compares a large sample of women suffering from chronic myofascial TMD (n=124) with a demographically matched control group without TMD (n=46) on sleep background electromyography (EMG) during a laboratory PSG study. Background EMG activity was measured as EMG root mean square (RMS) from the right masseter muscle after lights out. Sleep background EMG activity was defined as EMG RMS remaining after activity attributable to SB, other orofacial activity, other oromotor activity and movement artifacts were removed. Results indicated that median background EMG during these non SB-event periods was significantly higher ($p<.01$) for women with myofascial TMD (median=3.31 μV and mean=4.98 μV) than for control women (median=2.83 μV and mean=3.88 μV) with median activity in 72% of cases exceeding control activity. Moreover, for TMD cases, background EMG was positively associated and SB event-related EMG was negatively associated with pain intensity ratings (0–10 numerical scale) on post sleep waking. These data provide the foundation for a new focus on small, but persistent, elevations in sleep EMG activity over the course of the night as a mechanism of pain induction or maintenance.

Keywords

myofascial pain; temporomandibular disorders; TMD; sleep; bruxism; sleep bruxism; muscle tone; EMG; polysomnography

Corresponding author: Dr. Karen G. Raphael, New York University College of Dentistry, 380 Second Avenue, Suite 301, New York, NY 10010, kgr234@nyu.edu; tel: 212-992-7043, fax: 212-997-7130.

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Background

Myofascial temporomandibular disorder (TMD) is characterized by pain in the masticatory muscles. Since the 1960s, bruxism (1), involving tooth grinding and clenching, has been widely believed (2–4) to be an important risk factor. In 2012, a large, laboratory polysomnography (PSG) study of sleep bruxism (SB) (5) used state-of-the-art scoring of the electromyographic (EMG) signal to assess SB (6). It found similarly rare SB levels in both myofascial TMD cases and matched controls. Even when combining SB with other events causing marked elevations in masticatory muscle activity, such as yawning or sleep talking, elevations averaged approximately 5 minutes nightly in both groups. Thus, SB was rejected as a myofascial TMD maintenance factor.

Lower background masticatory muscle EMG activity during sleep, occurring outside of defined SB and other motor events, has not been examined in prior research on myofascial TMD. Low or isolated bursts of EMG activity not meeting SB scoring thresholds (6) may occur, as well as low increase in general muscle tone.

Studies of waking masticatory muscle activity have examined low-level elevations of EMG activity as contributory to myofascial TMD (7, 8). For example, Glaros et al. have documented (9, 10) that TMD patients engage in more frequent tooth-to-tooth contact than controls. Other research (11, 12) found TMD patients to have elevated awake resting EMG in some but not all masticatory muscle sites. Generally, results from daytime EMG studies vary. Studies show that purposeful low-force clenching in healthy individuals (13–16) can cause at least temporary pain and increases in masseter EMG. Some experimental stress induction studies show elevated EMG in TMD patients (17) during stress and rest (18) while others find mixed (19, 20) or negative results (21), depending on muscle group or stressor. Daytime stress studies are limited by the relatively brief time of observation and constraints or reactivity of experimental settings.

New analyses show that myofascial TMD patients have increased respiratory effort related arousals (RERAs) and sleep fragmentation (22). These arousals may be associated with an increase in nonspecific muscle tone (23). Thus, we may anticipate elevated sleep masticatory muscle EMG activity outside of periods when rare SB or other marked ‘events’ occur. Here, we define masticatory muscle EMG activity occurring outside of SB or other defined motor event periods as sleep “background” EMG.

This study aims to examine masseter muscle sleep background EMG in a large group of myofascial TMD patients and demographically equivalent controls participating in a laboratory PSG study. Specifically, we seek to determine whether sleep background EMG can be considered a candidate risk factor for myofascial TMD pain maintenance by (a) comparing sleep background EMG in myofascial TMD patients and controls, (b) determining whether case/control differences in sleep background EMG can be attributed to previously documented differences in sleep fragmentation or respiratory-effort related arousals, and (c) among myofascial TMD cases, examine and contrast the relationship of sleep background EMG and event-related activity with pain severity before and after sleep.

Material and Methods

This project was approved by the Institutional Review Board of the New York University School of Medicine. All participants completed a thorough informed consent process before enrolling.

Participants

All participants were women, given the strikingly higher prevalence of TMDs in women. (24–26). Participants were recruited, consented, examined and interviewed at the New York University College of Dentistry (NYUCD), New-York, USA. All had to be fluent in English. Laboratory-based PSG studies were conducted at a sleep center affiliated with the NYU School of Medicine.

Participants were enrolled solely on the basis of presence (cases) or absence (controls) of a myofascial TMD, and were enrolled explicitly independent of their belief or knowledge of their own SB, to ensure that SB prevalence and sleep EMG activity in general was not over- or under-represented in either sample.

Myofascial TMD Patients—Participating myofascial TMD patients met Research Diagnostic Criteria (RDC/TMD) for TMD Group II (27): pain of muscle origin, including a complaint of pain and pain associated with localized areas of tenderness to palpation in muscle. Criteria include: (1) Report of pain or ache in the jaw, temples, face, preauricular area, or inside the ear at rest or during function; plus: (2) Pain reported in response to palpation of 3 or more of the following 20 muscle sites: (left and right count as separate sites 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. At least one site must have been ipsilateral to the complaint of pain.

Non-TMD Controls—Non-TMD controls were recruited from other NYUCD dental clinics and acquaintances of participating patients, forming a sample that demographically matched cases on age, socio-economic status, self-identified race, and self-identified Hispanic ethnicity. Eligible Non-TMD controls could not have reported 1+ weeks of facial pain in the last two years or more than one painful site upon masticatory muscle palpation, using RDC/TMD examination procedures (27).

Exclusion Criteria—If potential cases or controls indicated that they had a motor vehicle accident or other major and identifiable physical trauma involving the face, they were excluded. Neither cases nor controls could have had dental treatment within 48 hours prior to the RDC/TMD eligibility examination.

Measures

Recognizing that sleep is not quiet for masticatory muscles, even after excluding specific functional and nonfunctional motor events (23), activity during this latter period was defined as sleep background EMG. Sleep background EMG activity represents EMG RMS remaining after activity attributable to SB, other orofacial activity, other oromotor activity and movement artifacts are removed.

Assessment of Sleep Masticatory Muscle Events—Since sleep monitoring, as well as sleeping in an unfamiliar laboratory environment can alter natural sleep (see (28)), participants spent two consecutive nights in the sleep laboratory. The first night was used for acclimation. Data from this night were not used in statistical analysis, except for three cases who failed to return for a second night and for six cases and one control when technical problems prevented second night scoring. Sleep recordings were made from approximately 10:30 pm to 7:00 am, adjusted to the participant's usual sleep time. The setting of the recordings has been described in detail elsewhere (6, 29). Technicians were blind to participants' case status.

Two Ph.D.s with expertise in sleep medicine were trained by Dr. Gilles Lavigne's Montreal-based staff to score "events" causing marked elevations in surface EMG signals in the masticatory muscles. The PSG record consisted of a 6-channel electroencephalogram (EEG), bilateral electrooculogram (EOG), bilateral submental (chin) and anterior tibialis EMG), right and left masseter and temporalis EMG, EKG, chest and abdominal motion (by belts with Piezoelectric sensors), body position, airflow by nasal pressure transducer and nasal/oral thermistor, and oximetry. Skin was abraded lightly with Nuprep® prior to electrode placement to reduce noise and skin impedance. Sleep data were recorded using SomnoStarPro acquisition system (San Diego, CA) using sampling rates at 200Hz (and bandpass filtering 15–70 Hz) for EMG channels. Audio and video signals were recorded in parallel.

All EMG records were carefully reviewed in 30 second epochs, and signal artifacts were manually edited out. Jaw muscle activity was scored after exporting data to Stellate Harmonie (formerly Montreal, Canada; currently Natus, USA). Overall right masseter RMS was calculated from lights out until lights on, excluding periods in which the sleep technician intervened for technical reasons such as a disengaged electrode or the subject explicitly woke (e.g., request to unhook and use the lavatory). Using clinical research diagnostic criteria for SB(RDC/SB) validated by Lavigne et al. (6, 30), audio-video signals were used to differentiate tooth-grinding sounds from other oral noise during sleep (e.g., snoring, sleep talking, TMJ clicking with yawning). The record was then scanned for periods in which the resulting signal was approximately twice the awake resting baseline, and also met a set of highly specific, predefined scoring rules for rhythmic masticatory muscle activity (RMMA), orofacial events, and oromotor events (see (5) for details). The beginning and end of such periods were marked, as were wake periods, during the period after lights out until lights on. After removing periods during which these specific functional and nonfunctional motor events occurred (23), remaining activity was defined as "sleep background EMG" and computed at the root mean square (RMS) (in μV) (29, 31) of these points. The number of RMMA events per hour, and whether subjects met criteria for increasing severity of SB (see (5) (scored 0/1 where 0=failed to meet criteria, 1=met criteria) were also noted.

Standard sleep parameters were also recorded. We focus here on sleep fragmentation as indicated by elevation in % stage N1 and sleep respiratory related event arousal (RERA), because prior analyses of this same set of sleep records indicated that myofascial TMD patients show elevations on these measures compared to demographically matched controls (Dubrovsky et al., in revision).

Pain—To ensure that sleep technicians were blind to case/control status of participants, a research associate prepared a packet of questionnaires to be distributed to the case or control participant, as appropriate, for each study night. TMD cases were asked to rate their current pain intensity and average pain in the day just ending, before lights out, on a 0–10 numerical scale, where 0=no pain and 10=worst pain imaginable. In the morning after being woken by the sleep technician, the packet for TMD patients included questions about their current pain on waking (labeled waking post sleep in tables) and about their average pain during the night just ending, again rated on a 0–10 scale.

Statistics—Due to skewness in the distribution of some EMG measures, these data were routinely rank-transformed. Thus, for example, a value of "87" indicates that the average subject in that group ranked 87th out of all 170 subjects (combined sample of cases and controls) in the analysis. To compare cases and controls, independent sample t-tests or ANOVAs were conducted on ranked sleep EMG values. Although not detailed here, we confirmed that conclusions from Mann-Whitney U independent sample tests were

equivalent to conclusions from independent sample t-tests on rank-transformed EMG data. Others (32) have shown that use of parametric statistics on rank-transformed data is equivalent to use of nonparametric statistics, when sample sizes are relatively large. Given the more familiar parametric approaches, we apply them here with ranked data. Most importantly, rank transformation produces homogeneous variances in the two groups, i.e., ratio of variances=1.09 and Levene statistics=0.26, p>.61.

To determine whether differences in sleep architecture explained differences in sleep background EMG, multiple linear regression was used. At the first step, sleep background EMG RMS was predicted from case status. At the second step, sleep background EMG was predicted from case status and measures of sleep fragmentation in a forward stepwise approach. Pearson product moment correlations tested the relationship of pain severity before and after sleep with measures of sleep bruxism and ranked EMG measures.

Results

As detailed in prior manuscripts based on this study (5, 22), case (n=124) and control (n=46) subjects did not differ on any measured demographic characteristic: most indicated that their race was white (62.6%), black (14.4%) or “other” (14.4%). Hispanic ethnicity was endorsed by 22.5%. Mean age was 39.2 yr (SD=14.6, range 19–78), and mean years of education was 15 (SD= 2.2, range 11–20). TMD patients reported moderate characteristic pain intensity on a 0–10 scale (Mean= 5.2, SD= 1.7) and relatively low levels of pain disability on a similar 0–10 scale (Mean=1.8, SD=2.2). Pain onset occurred more than 10 years before study entry (Mean= 126.1 months, SD= 127.1; Median=84).

The first aim is addressed in Table 1. It demonstrates that sleep background EMG was significantly higher in TMD cases than in controls. Because the RMS signal of EMG activity during scorable events (e.g., RMMA and various orofacial activities) did not differ between cases and controls, the overall difference in EMG RMS signal over the course of the entire sleep period (p=.005) can be attributed to case/control differences in sleep EMG during periods without events (i.e., background EMG). These data show that, while there appears to be higher sleep EMG activity in the masticatory muscles of cases than controls, this is not a result of either SB activity in particular, or defined sleep events in general.

The case/control difference in mean EMG RMS during sleep might result from a small subgroup with high background values, rather than a small increase in EMG activity among many cases. To rule out this possibility, the 11 subjects (8 cases and 3 controls) with the highest RMS vales were removed from the analysis. A one-way ANOVA continued to show significant case/control difference (p= .005); in fact, this difference was larger than the difference shown in the complete sample (p= .01). When one makes a similar comparison among the unranked scores, the complete sample failed to show a significant case/control difference (p= .20); selecting out the highest values, however, a case/control difference is found (p= .009). These results suggest that the outlying scores cannot explain case/control differences in sleep background EMG. Rather, they appear to be the result of small increases in EMG activity throughout the night among many case subjects.

To contextualize the magnitude of these effects, the median sleep background EMG during these non-event periods for controls was 2.83 μV (mean=3.88, 95% CI: 2.74 – 5.01), while that of the TMD cases was 3.31 μV (mean=4.98, 95% CI: 4.04 – 5.93). The median activity of controls was exceeded by 89 cases (71.8%). By contrast, the median EMG RMS during scorable SB-RMMA events was 13.81 μV (mean=13.55, 95% CI: 12.32 – 14.78) and 13.34 μV (mean=18.98, 95% CI: 14.81 – 23.14) in controls and cases respectively.

To address the next aim, we sought to determine whether case/control differences in sleep background EMG could be attributed to case/control differences in sleep fragmentation. When adding % total sleep time in stage N1 sleep and respiratory effort related arousal events per sleep hour (RERA index) to the model, only the % total sleep time in stage N1 sleep measure of sleep fragmentation remained. Furthermore, as shown in Table 2, case status continued to be a significant predictor of sleep background EMG RMS. Thus, case/control differences in sleep background EMG are independent of respiratory sleep fragmentation related events.

Table 3 addresses the final aim. It shows that pain ratings made by the cases before and after sleep were inversely proportional to measures of the number of RMMA episodes per hour of sleep, indicating that higher pain reported either before or after sleep was associated with fewer masticatory muscle events (but not lower RMS). In a similar way, cases who met criteria for the most stringent definition of SB (6) had lower levels of pre-sleep current pain than other cases (p=.017). On the other hand, higher levels of background masticatory muscle EMG RMS during sleep were associated with higher reports of pain before and after sleep. Thus, data indicate that more pain, assessed either right before sleep or upon awakening, is associated with fewer SB or other defined events but higher levels of residual, background activity.

Discussion

To our knowledge, this is the largest existing laboratory PSG study of myofascial TMD patients and matched control women. It is unique in that we have scored SB events and other orofacial and oromotor events using research criteria (6), permitting us to evaluate EMG activity during sleep periods when such events do not occur. Compared with ambulatory EMG studies, our audiovisual monitoring allowed us to identify and exclude artifacts associated with neither SB, other oromotor or orofacial events, and standard sleep software permitted differentiation of periods of wake from sleep after lights out. The data set has already been used to demonstrate that myofascial TMD patients do not have elevated frequencies of SB, using any of three varying levels of severity criteria (5). Additional analysis of this same data set now demonstrates that in sleep periods during which there are no masticatory EMG elevations defining various types of sleep bruxism-related events, background EMG levels are significantly elevated in myofascial TMD patients compared to controls.

These differences are unlikely to be attributed to demographic factors that can cause differences in EMG activity (33), since groups were well matched on age and were invariant on sex. Additional analyses not detailed here determined that cases and controls did not differ in BMI, so that differential attenuation of the EMG signal as it passed through subcutaneous fat (34) cannot account for case/control differences.

To contextualize our EMG values, median background EMG RMS values of 2.83 and 3.31 μV in controls and cases, respectively, are about 20% the size of event-related values of 13.81 and 13.34 μV, respectively. The masseter muscle EMG background values are comparable to those reported in one study of TMD cases and controls at waking rest (35) and in a small sample of healthy men and women during sleep (31). Event-related RMS values are less than 10% of values during maximal voluntary clenching (35).

While several types of SB-RMMA frequency measures were negatively associated with intensity of pre- and post-sleep pain in myofascial TMD patients, rank EMG RMS measures during sleep background periods were positively associated with severity of pain, both pre- and post-sleep. It is not possible to evaluate whether sleep background EMG has an

immediate or short-term effect on pain on waking, since pain ratings have a strong trait component, with pre-and post-sleep pain being highly correlated. Nevertheless, these data indicate that elevations in sleep background EMG are a potential risk factor for the maintenance of myofascial TMD pain. We suggest some caution in interpreting the different patterns between pain and SB-RMMA versus pain and sleep background EMG. Given multiple comparisons and possible inflation of Type I error, replication is recommended.

Other authors (e.g., (36–38)) have noted that the psychophysiological model of myofascial TMD, as developed in the 1960s (1), contradicts Lund's (39) pain adaptation model. The first postulates a vicious cycle in which stress leads to muscle hyperactivity leading to bruxism which leads to pain, which feeds back to a cycle of increased stress, bruxism and pain. The pain adaptation model, in striking contrast, is supported by data showing that acute pain (e.g., (40–43)), leads to a decrease in muscle activity in the painful area. An alternate path is suggested by the current data: mild but not marked elevations in EMG activity might avoid an immediate increase in pain that would invoke a motion-restricting pain adaptation response. Rather, the cumulative effect of long time periods of mild elevations in EMG activity may eventually cause persistent pain. This may produce a unique type of delayed-onset and persistent muscle soreness (44) that has been difficult to model in experimental pain studies. Our data support part of a modified theory proposed by Ohrbach and McCall (45) which focuses on chronic low-grade hyperactivity.

These cross-sectional data are not able to determine whether sleep background EMG is a risk factor for onset of myofascial TMD, but they support the hypothesis of elevated sleep background EMG as a candidate risk factor for ongoing masticatory muscle pain. Further tests of its role as a risk factor might involve treating myofascial TMD patients with interventions considered to explicitly manipulate sleep background EMG (controversially, oral splints (46–48), or Botulinum Toxin (49–52) or biofeedback (53)) and determine whether these treatments produce a robust reduction in pain related to reduction in sleep background EMG.

Sleep background EMG is undoubtedly not the single, simple explanation for persistent myofascial TMD: Although case/control differences were not caused by the action of a few outlying cases, there was overlap in the case/control distribution of sleep background EMG. Some controls with relatively high resting sleep EMG did not have pain, and some myofascial TMD cases with relatively low resting sleep EMG still had persistent pain. As others have noted, surface EMG, during sleep or wake, is neither sensitive nor specific enough to have utility as a diagnostic test for TMDs (11, 54). In part, limited diagnostic utility is due to error in measurement caused by factors that are difficult to perfectly standardize (55, 56), such as electrode placement (57). Although our technicians were carefully trained to standardize procedures, minor variation in procedural replication across subjects may have occurred. The fact that our technicians were blind to participants' case status suggests that any procedural variation was more likely to have added random measurement error than to have created systematic bias or confounding. Thus, the true magnitude of case-control differences in sleep background EMG may have been attenuated due to some measurement error.

Typically, pain on waking is considered to be a clinical indicator of SB. We find here that, rather than reflecting SB, pain on waking may reflect mild but prolonged elevations in sleep background EMG over the course of the night, leading to a pattern of muscle fatigue and pain. Thus, the utility of using self-reported pain on waking as an indicator of SB is negated by these data.

Several limitations of our investigation should be noted. Non-invasive surface electrodes are undoubtedly less accurate than intramuscular fine-wire electrodes for EMG recordings. Moreover, we did not separately score pre-sleep background EMG to determine whether similar case/control differences occurred during resting wakefulness. Additionally, we did not conduct frequency-spectral analysis of EMG's to decrease spillover of activity at one frequency into neighboring frequencies. Finally, we did not analyze data broken down by sleep stage, to isolate whether case/control differences were confined to certain sleep stages. Most critically, the current analysis does not identify the mechanism through which TMD patients demonstrate increased sleep background EMG, and further research is needed.

Combined with other research (7, 9) that has identified daytime tooth-to-tooth contact as much more common in TMD patients than controls and classic experimental work that has shown the fatiguing effect of purposive, low-intensity masticatory muscle activity (13–16), these data further encourage a shift in focus away from large and dramatic SB activity to a focus on more subtle but prolonged background EMG elevations in the masticatory muscles, as a path to further understanding the cause and persistence of myofascial TMD pain.

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Table 1

Case/control comparison of sleep bruxism and masticatory EMG activity

Measure	Cases (n=124)	Controls (n=46)	p-value
	Mean (se)	Mean (se)	
Bruxism Related Activity			
EMG activity during RMMA events (rank RMS)	87.31 (4.57)	80.63 (6.57)	0.406
EMG activity during all scorable events (RMMA, orofacial, oromotor) (rank RMS)	87.17 (4.53)	81.00 (6.80)	0.452
EMG activity during entire sleep period (rank RMS)	91.84 (4.40)	68.41 (6.79)	0.005
Sleep Background EMG			
Rank of RMS during non-event (sleep background) periods	91.34 (4.39)	69.76 (6.92)	0.01

Table 2

Linear regression analysis predicting sleep background EMG RMS from (a) case control status and (b) case control status plus sleep fragmentation measures.

Variables in model	B	S.E.	Beta	t	Sig.
Model a:					
Constant	69.76	7.14		9.75	.000
Case status	21.39	8.39	.194	2.55	.012
Model b:					
Constant	56.57	8.52		6.64	.000
Case status	17.11	8.38	.155	2.04	.043
% total sleep time in Stage 1	1.427	.523	.207	2.729	.007

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Table 3

Correlation of pre- and post-sleep immediate and daily average pain with sleep bruxism related activity and EMG RMS in Myofascial TMD Cases

		Pre-sleep current pain	Pre-sleep avg. daily pain	Waking (post sleep) current pain	Waking (post sleep) average nightly pain
Bruxism Related Activity					
RMMA episodes (per hr of sleep)	Pearson r	**−0.264**	**−0.217**	**−0.185**	**−0.239**
Meets criteria for mild bruxism (2+ episodes with noise)	Pearson r	−0.142	−0.088	−0.081	−0.141
Meets criteria for moderate bruxism (>2 and ≤4 episodes per hr)	Pearson r	−0.180	−0.138	−0.128	**−0.225**
Meets criteria for severe bruxism (4+ episodes or 25+bursts per hr)	Pearson r	**−0.221**	−0.148	−0.159	−0.157
EMG RMS (rank-transformed)					
EMG RMS during RMMA events	Pearson r	0.008	−0.063	−0.013	−0.099
EMG RMS during scorable events (include RMMAs, orofacial, oromotor)	Pearson r	−0.101	−0.130	−0.119	−0.123
EMG RMS during entire sleep period	Pearson r	0.076	0.081	0.136	0.117
EMG RMS during nonevent (background) periods	Pearson r	0.159	0.156	**0.205**	**0.239**

BF: p<.05