Effect of Jaw Muscle Pain and Soreness Evoked by Capsaicin Before Sleep on Orofacial Motor Activity During Sleep

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Aims: *Sleep bruxism, which is a form of orofacial motor activity (OMA), and jaw muscle pain and soreness have for a long time been thought to be mutually linked. The aim of this study was to investigate the effect of clinical and experimental jaw muscle pain and soreness on sleep OMA.* **Methods:** *Twelve healthy subjects aged 21 to 31 years old participated in this study. All of them were aware of signs or symptoms of sleep OMA and were subdivided into a group with clinical pain complaints (n = 5) and a group without pain (n = 7). All subjects slept in the laboratory for 3 consecutive nights, including a habituation night, a baseline night, and an experimental night. Electroencephalographic (EEG) activity and electromyographic (EMG) activity from the masseter muscles were recorded during sleep. On the experimental night, before sleep, all subjects received an injection of capsaicin (0.1 mL, 100 µg/mL) into the masseter muscle that had demonstrated the most EMG activity during the previous recordings. The OMA events and episodes were quantified and were compared between the baseline night and the experimental night. Every evening and morning during the study period, pain intensity, unpleasantness, and soreness were scored by the subjects on a visual analog scale (VAS), and pain detection thresholds (PDTs) in the masseter muscles and maximal voluntary occlusal force (MVOF) were also measured.* **Results:** *Pre-sleep injection of capsaicin did not cause significant differences between groups in peak pain intensity on the VAS. The PDT and MVOF did not show any significant differences between groups, injection and non-injection sides, or baseline and experimental nights and mornings. The number of EMG episodes/hour sleep, the number of bursts/hour sleep, and total area of all bursts and episodes during the baseline night were significantly higher in the subjects without pain than in the subjects with pain. However, the capsaicin injection did not cause any significant changes in these parameters.* **Conclusion:** *This study suggests that an acute pre-sleep painful stimulus does not have any effect on OMA during sleep, but the study extends previous findings that clinical jaw muscle pain and soreness are associated with less EMG activity in the masticatory muscles.*

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Key words: masticatory muscles, capsaicin, bruxism, pain detection thresholds, bite force, electromyography

Sleep bruxism is a form of orofacial motor activity (OMA) defined as a periodic, stereotyped jaw movement disorder with grinding or clenching of the teeth during sleep.[1] Patients with temporomandibular disorders (TMD) are frequently thought to clench or grind their teeth, which has led to the view that there

is a strong relationship between OMA and TMD.[2–5] Furthermore, Travell et al[6] have hypothesized that unaccustomed or abnormal contractions of muscles cause pain and once muscle pain has developed, it can in turn induce more muscle hyperactivity, setting up a vicious cycle. This hypothesis was also adapted to the masticatory muscles.[7] More recently, it was suggested that OMA might cause post-exercise muscle soreness (PEMS) in the masticatory muscles[8,9] that resembles the phenomenon in the muscles of the limbs and trunk.[10,11] Arima et al[12] examined jaw muscle pain and soreness levels evoked by standardized jaw movements at more than 50% of maximal voluntary occlusal force (MVOF) for 45 minutes, which may partly imitate the nature of sleep bruxism. This and other studies have shown that it is difficult to initiate the vicious cycle and to develop long-lasting jaw muscle pain that is similar to PEMS in healthy subjects.

Therefore, the question of how sleep OMA may lead to TMD is still not clear. Sleep OMA has been studied with polysomnographic methods, and the techniques to measure sleep OMA have been developed progressively. For example, Lavigne et al[13] suggested a method with definition of "phasic," "tonic," and "mixed" episodes of electromyographic (EMG) activity, and Ikeda et al,[14] using strict criteria for sleep bruxism, quantified sleep OMA at different EMG thresholds (3%, 10%, and 20%). Lavigne et al[15] later reported that about 56% of a study group had rhythmic OMA without any other signs or symptoms of sleep OMA, other sleep disorders, or jaw muscle pain. Moreover, there is evidence that bruxers with pain have 40% fewer EMG episodes per hour of sleep than bruxers without pain.[16] This suggests that jaw muscle pain may decrease the number of EMG episodes, which would contradict the second part of the "vicious cycle" theory, namely, that muscle pain causes more muscle hyperactivity.

The aim of this study was, therefore, to investigate the effect of pre-sleep acute jaw muscle pain and soreness evoked by a single injection of capsaicin on sleep OMA in a group of subjects with complaints of jaw pain and soreness versus a nonsymptomatic group. Previously published criteria were used to analyze sleep OMA,[13] but subjects with less activity than described by these criteria were also included to study a broader range of "bruxism."

Materials and Methods

Subjects

Nine men and 3 women participated in this study (mean age 24 years; range 21 to 31 years). All subjects were in good health but had reports of noise from the teeth during sleep by the sleep partner or family. Furthermore, they were aware of their dental wear and wear facets could be identified in all subjects by the investigator. All subjects were examined according to current TMD guidelines[17] and were subdivided into groups with pain and without pain. One group consisted of those subjects (mean age 23.2 years; range 22 to 28 years; n = 5) who answered "yes" to the question, "Do your jaws regularly feel stiff, tight, and/or tired in the mornings?"[18] All subjects in this group reported jaw muscle pain/soreness on a visual analog scale (VAS) the morning of a baseline night of sleep investigation (control) (13.0 ± 4.9 mm). In contrast, the other group of subjects (mean age 25.1 years; range 21 to 31 years; n = 7) had no problems in their temporomandibular joints and no pain or soreness in their masticatory muscles. Informed consent was obtained from all participants, and the study was approved by the local ethics committee.

Study Design

Each subject slept in the laboratory for 3 consecutive nights. The first night was used for adaptation to the laboratory environment and for training in the examinations (habituation), the second night was the baseline night (control), and the third night was the experimental night (capsaicin). On the experimental night, before going to sleep, the subjects received an injection of capsaicin into the masseter muscle that had showed the most EMG activity during sleep (see "Experimental Pain and Soreness"). Scores from the VAS for pain and soreness, values and descriptors on the McGill Pain Questionnaire (MPQ), pain distribution, pain detection threshold (PDT), and MVOF were used in this study as outcome parameters. These measurements were performed every evening (at 10:30 pm on the habituation and baseline nights, and at 10:00 pm on the experimental night before capsaicin injection) and every morning (7:45 am) during the study period. Subjects were allowed to go to bed at 11:00 pm, after the measurements were made, and they stayed in bed until 7:30 am the next morning. Then the measurements were repeated. A commercial electroencephalography

(EEG) system recorded the stages of sleep, and EMG recordings from the masseter muscles were obtained to recognize sleep OMA. All sleep recordings were performed with constant temperature in the sleep laboratory at Aalborg University.

Polysomnographic Recording

A Nightingale sleep analyzer (Judex A/S) was used for EEG recording and sleep analysis.[19,20] This system is based on a personal computer that implements an automated version of the standard rules for scoring of human sleep.[21] The system uses the frequency contents in the EEG signal to calculate the stage of sleep. The subjects were equipped with an EEG cap (Electro-Cap International) according to standard procedures, and the EEG signals from 6 surface recordings were recorded. The leads were Fp1-A2, Fp2-A1, C3-A2, C4-A1, O1-A2, and O2-A1. Eye movements (electro-oculography [EOG]) were recorded according to Rechtschaffen and Kales,[21] and EMG activity of the submental muscles was recorded bilaterally with surface electrodes (Neuroline, type 720-01-K, Medicotest). Signals were fed into a headbox, converted to digital information, sampled at 100 Hz, and stored in a computer, where display of the polysomnographic curves could be performed. Second-order filter settings for the EEG signals were 0.5 Hz high-pass and 35 Hz low-pass; the EMG activity from the submental muscles was filtered at 0.5 Hz high-pass and 50 Hz low-pass; and EOG signals were filtered at 0.5 Hz high-pass and 25 Hz low-pass.

EMG Recording and Analysis

The skin was cleaned with absolute alcohol, and bipolar disposable surface electrodes (Blue Sensor, type N-10-E, Medicotest) were placed with their long axis transverse to the main direction of the muscle fibers in the central part of the right and left masseter muscles. Electrode placement was based on palpation of the muscles during maximal clenching, as previously described by Møller.[22] The interelectrode distance was 10 mm. A ground electrode was placed on the neck. Because the Nightingale system can record only 100 Hz as maximal sampling frequency, the EMG signals were amplified (×250,000), filtered (1 Hz high-pass), rectified, and filtered again (5 Hz low-pass).

For analysis of muscle activity during sleep, the first epoch of non-rapid eye movement sleep stage 2 (NREM 2) and the last epoch of NREM 2 were used as the start and the end of sleep, respectively. Every EMG burst/episode was classified according to Lavigne et al.[13] The threshold for EMG activity of the masseter muscle was set to 3%, 10%, and 20% of the maximum activity measured in the awake state.[14] An episode was delineated by a quiescent interval of a minimum of 3 seconds between bursts. A phasic episode was recorded when at least 3 EMG bursts were separated by 2 interburst intervals (2 intervals being necessary to identify a rhythm), with each phasic burst lasting longer than 0.25 seconds and not exceeding 2 seconds. If a phasic episode had 1 or no interburst intervals, it was regarded as a burst. An episode with a burst lasting longer than 2 seconds was classified as a tonic episode, unless it was separated from the next or previous burst by less than 3 seconds, in which case it was identified as a mixed episode. Very rapid jerk-like contractions associated with EMG bursts of no longer than 0.25 seconds were classified as fragmentary myoclonus bursts. The total number of EMG episodes per hour of sleep, total number of EMG bursts per hour of sleep, total number of EMG bursts per episode, the total root mean square (RMS) of all EMG bursts and episodes, and the total area of all EMG bursts and episodes were determined in the rectified EMG signal and compared between the groups, 3 different threshold levels, and the baseline (control) and experimental (capsaicin) nights.

Experimental Pain and Soreness

Sterile capsaicin was prepared by the local pharmacy at the University Hospital in Aarhus in accordance with previous descriptions[23,24] and diluted with Tween 80 (University Hospital) dissolved in isotonic saline. Injections (0.1 mL, 100 µg/mL; pH 6) were used in this study to produce long-lasting muscle pain and soreness.[25] Intramuscular injection of capsaicin activates Aδ-mechanoheat fibers and polymodal C-fibers and evokes cramp-like muscle soreness in human subjects.[26] Furthermore, it has recently been observed that intramuscular injection of capsaicin causes longer-lasting pain (more than 30 minutes), with increasing sensitivity to pressure stimuli, compared to other substances (hypertonic saline).[24,27] The EMG recordings from the previous 2 nights (habituation and control) were used to define the predominant side of the masseter muscle for OMA during sleep in each subject. The side with the highest total number of EMG episodes per hour of sleep was determined to be the predominant side and was chosen as the injection side. A bolus of capsaicin was injected into the central part of the masseter muscle following the procedure from a

previous study,[25] and it was administered only on the experimental (capsaicin) night after PDT and MVOF measurements were made.

The subjects were asked to manually score their pain intensity, unpleasantness, and soreness on 100-mm VAS at rest every evening and morning and again 5 minutes and 15 minutes after the injection of capsaicin. The left end of the VAS was labeled by either "no pain," "no unpleasantness," or "no soreness" and the right end either "most pain," "most unpleasantness," or "most soreness." On the experimental day (capsaicin), the subjects scored their pain intensity continuously with the use of an electronic VAS for 15 minutes after the capsaicin injection, in addition to the manual VAS scores of unpleasantness and soreness after 5 and 15 minutes.

A Danish version of the MPQ was used to calculate the pain rating index (PRI) of the sensory, evaluative, affective, and miscellaneous components of pain.[28,29] Furthermore, the subjects were asked to draw the pain distribution on a figure showing the left and right profile of a face. The area of pain distribution was digitized (ACECAD, model D9000+ digitizer) and the area was calculated in arbitrary units (Sigma-Scan).

Pain Detection Thresholds

An electric pressure algometer (Somedic AB) was used with a probe diameter of 10 mm and a constant application rate of 30 kPa/second. After determination of the boundaries of the masseter muscles by palpation during voluntary contraction (ie, MVOF), the central part of each masseter muscle was marked.[25] A pair (right and left side of the face) of clear pliable plastic templates was indexed to the inferior surface of the earlobes, the lateral angle of the mouth, and the lateral angle of the eyes to reproduce the location of the measurement sites. Subjects were instructed to keep their teeth slightly apart (about 1 to 2 mm) to avoid contraction of the jaw-closing muscles during PDT measurements.[30] The PDT was defined as the pressure (in kPa) that the subjects first perceived to be painful. The subject pushed a small thumb switch when the threshold was reached, which froze the pressure on a digital display. The PDT was determined in triplicate. The interval between successive pressure stimuli was about 2 minutes.

Maximal Voluntary Occlusal Force

A U-shaped bite force transducer (7 mm high, 1.1×1.1 cm area, Aalborg University) was covered with plastic tubes to protect the teeth.[31] After the electrodes had been positioned on both sides of the masseter muscles and the EEG cap placed on the subject, the MVOF was measured on the right and left sides. The bite force transducer was placed between the first molars, and subjects were instructed to clench their teeth as hard as they could for 3 to 4 seconds. Verbal encouragement was given to obtain the maximum effort.

Statistics

Parametric statistics (mean ± SEM), *t* test analysis, and multivariate analyses of variance (MANOVA) were used to describe the data. The factors were: group (with and without pain), side (injection versus non-injection), time (baseline versus experimental night), and threshold (3%, 10%, and 20%). The levels of significance were adjusted for multiple comparisons with use of Student-Newman-Keuls (SNK) tests. Significance was accepted at $P < .05$.

Results

Experimental Pain

Immediately after the injection of capsaicin, electronic VAS scores of pain increased progressively and reached a peak (subjects with pain, 48 ± 8 mm; subjects without pain, 54 ± 7 mm) (Fig 1), with no significant difference between groups (Student-Newman-Keuls [SNK], $P = .560$). The area under the curve of the pain intensity profile (subjects with pain, 34,960 ± 6,725 seconds×mm; subjects without pain, 31,474 ± 6,214 seconds×mm; SNK, $P = .716$) and the duration of pain (subjects with pain, 1,163 ± 223 seconds; subjects without pain, 1,090 ± 143 seconds; SNK, $P = .778$) were not significantly different between groups.

Five minutes after capsaicin injection, the area of perceived pain was significantly increased as compared to the baseline (control) night (subjects with pain, 0.490 ± 0.135 units; subjects without pain, 1.179 ± 0.424 units; MANOVA, $F = 5.541$, $P = .001$), with significant differences between groups (SNK, $P = .043$). The VAS scores for pain intensity, unpleasantness, and soreness were significantly increased over baseline (control) values (SNK, $P \leq .001$) (Figs 2a to 2c) in both groups. Subjects without pain reported significantly greater soreness on the VAS as compared to subjects with pain (SNK, $P = .014$). However, all these VAS

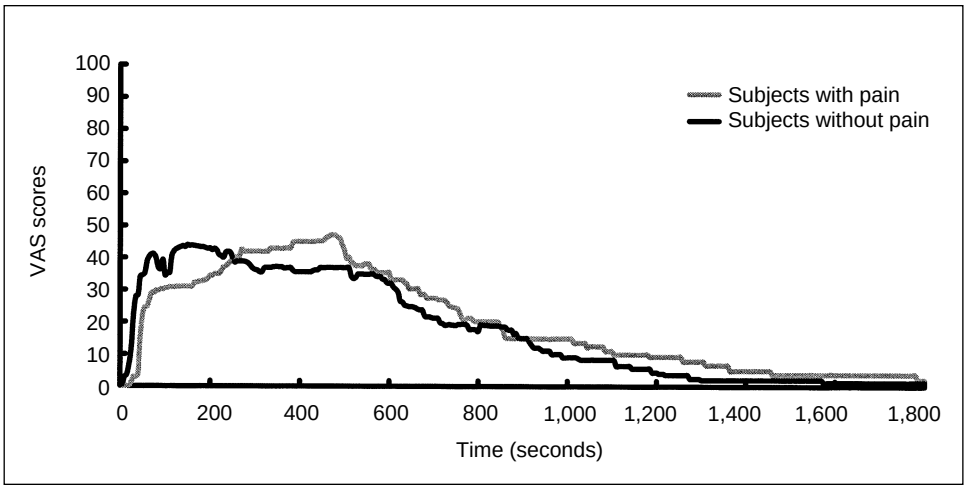


Fig 1 Mean profiles of pain recorded on an electronic VAS following injection of capsaicin into the right masseter muscle in subjects with pain (n = 5, gray line) and subjects without pain (n = 7, black line). Peak pain, pain duration, and the area under the curve did not show significant differences between groups ($P \geq .560$).

parameters (pain intensity, unpleasantness, and soreness) were not significantly different the morning after the capsaicin injection (SNK, $P \geq .713$). Analysis of the PRI from the MPQ demonstrated that the capsaicin injection had a significant effect on the sensory dimension of pain in both groups (SNK, $P = .001$), with no significant differences between groups (subjects with pain, 15.8 ± 5.0; subjects without pain, 13.9 ± 3.5; MANOVA, F = 3.238, $P = .079$). The most frequently chosen words (> 50%) from the MPQ were "boring" (50%), "intense" (50%), and "piercing" (50%) from subjects with pain, and "pinching" (50%), "tingling" (50%), "aching" (50%), "intense" (50%), and "squeezing" (50%) from subjects without pain.

Pain Detection Thresholds

The PDTs did not show any significant differences between groups, injection and non-injection sides, or baseline (control) and experimental (capsaicin) nights and mornings (MANOVA, $F \leq 2.478$, $P \geq .070$) (Table 1).

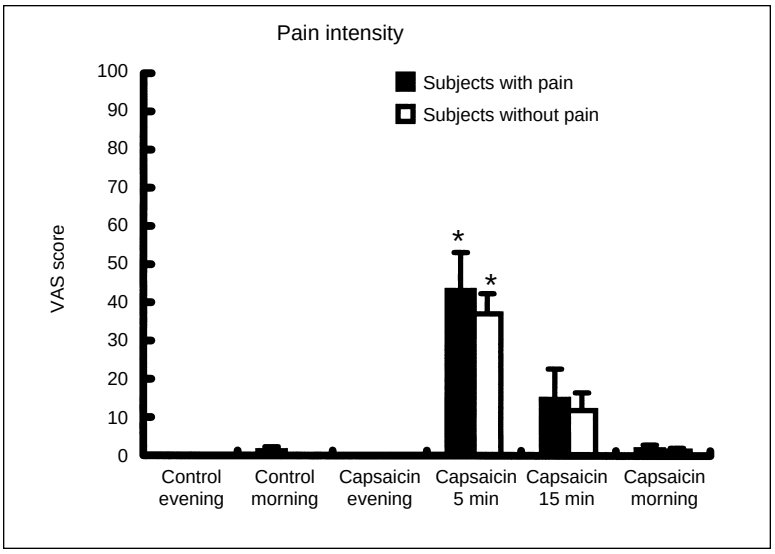
Maximal Voluntary Occlusal Force

Measurements of MVOF were performed in 7 subjects without pain and only 3 subjects with pain, since 2 subjects could not complete the MVOF measurements during the entire study period. There were no significant differences in MVOF between groups, injection and non-injection sides, or baseline (control) and experimental (capsaicin) nights and mornings (MANOVA, $F \leq 0.583$, $P \geq .629$) (Table 2).

EEG/EMG Analysis

Sleep Data. The mean hours of sleep did not show any significant differences between groups or between baseline (control) and experimental (capsaicin) nights (MANOVA, $F \leq 0.426$, $P \geq .529$) (Table 3). Furthermore, the analysis of the sleep proportion (%) is shown in Table 3. None of the parameters (REM, awakenings, NREM 1, NREM 2, NREM 3, or NREM 4) showed significant differences between groups or between baseline (control) and experimental (capsaicin) nights (MANOVA, $F \leq 2.546$, $P \geq .142$).

Number of EMG Episodes/Hour Sleep. The subjects without pain demonstrated a significantly higher number of EMG episodes/hour sleep as compared to the subjects with pain (MANOVA, F = 7.333, $P = .014$). There was a significant main effect of EMG thresholds on the number of EMG episodes/hour sleep (MANOVA, F = 41.831, $P = .001$), with more activity detected at the 3% level than at the 10% (SNK, $P = .001$) and the 20% levels (SNK, $P = .001$) (Figs 3a and 3b). The capsaicin injection did not cause any significant changes in the number of EMG episodes/hour sleep



Figs 2a to 2c Visual analog scale scores for pain intensity, unpleasantness, and soreness during the course of the study. The VAS scores (subjects with pain, n = 5; subjects without pain, n = 7) increased in the interval from 5 to 15 minutes after capsaicin injection, compared to the control night (*$P < .05$; SNK). Five minutes after capsaicin injection, soreness in subjects without pain was significantly higher than in subjects with pain ($^{\dagger}P < .05$; SNK).

Fig 2a

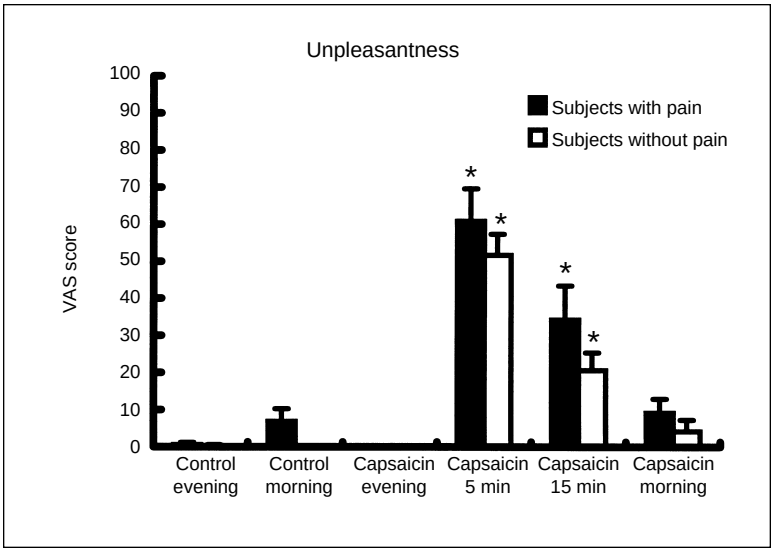


Fig 2b

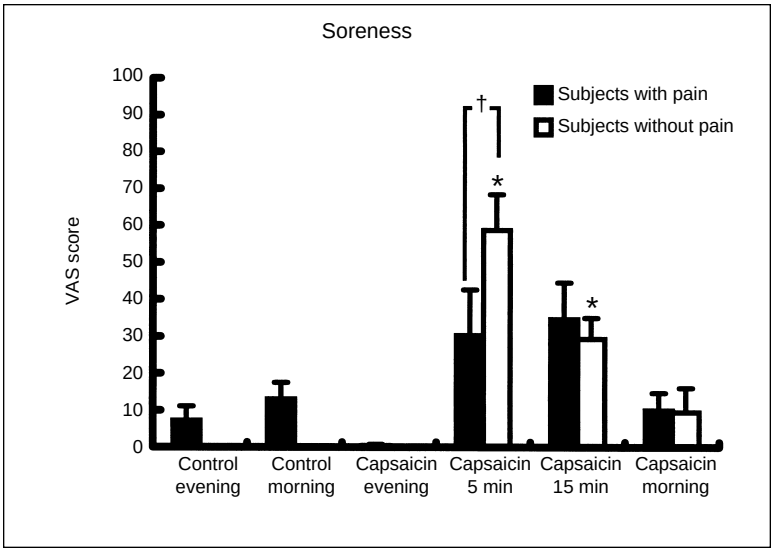


Fig 2c

Table 1 Mean Values and SEM of Pain Detection Thresholds in Non-injection and Injection Sides of the Masseter Muscle

	Pain detection thresholds (kPa)				
Side	Control evening	Control morning	Capsaicin evening	Capsaicin morning	*P* values*
Non-injection side					
Subjects with pain (n = 5)	189.5 ± 29.3	161.2 ± 18.6	184.0 ± 23.9	182.9 ± 22.7	> .580
Subjects without pain (n = 7)	212.3 ± 30.6	197.3 ± 23.2	192.6 ± 29.6	190.7 ± 25.4	> .771
Injection side					
Subjects with pain (n = 5)	199.8 ± 38.1	186.3 ± 29.3	190.8 ± 38.6	187.5 ± 35.3	> .944
Subjects without pain (n = 7)	204.3 ± 34.7	181.6 ± 14.1	191.2 ± 32.6	164.9 ± 16.7	> .328

*No significant differences were seen with respect to time (control, capsaicin, morning, evening); Student-Newman-Keuls tests.

Table 2 Mean Values and SEM of Maximal Voluntary Occlusal Force in Non-injection and Injection Sides of the Masseter Muscle

	Maximal voluntary occlusal force (kg)				
Side	Control evening	Control morning	Capsaicin evening	Capsaicin morning	*P* values*
Non-injection side					
Subjects with pain (n = 3)	51.6 ± 8.3	47.5 ± 1.7	51.2 ± 6.1	44.8 ± 2.8	> .957
Subjects without pain (n = 7)	51.5 ± 5.2	49.3 ± 5.6	47.3 ± 4.1	54.2 ± 4.8	> .980
Injection side					
Subjects with pain (n = 3)	48.7 ± 6.5	48.8 ± 2.4	50.4 ± 8.5	41.9 ± 7.9	> .774
Subjects without pain (n = 7)	52.2 ± 5.5	46.3 ± 4.5	49.9 ± 4.4	52.3 ± 3.2	> .990

*No significant differences were seen with respect to time (control, capsaicin, morning, evening); Student-Newman-Keuls tests.

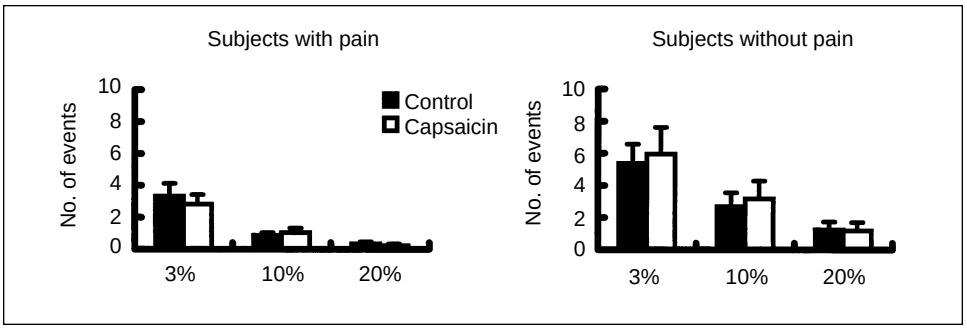
Table 3 Mean Values (%) and SEM of Sleep, and Mean Hours and SEM of Sleep

	Subjects with pain (n = 5)		Subjects without pain (n = 7)	
	Control	Capsaicin	Control	Capsaicin
Total sleep hours	7.9 ± 0.5	7.9 ± 0.4	8.0 ± 1.7	7.7 ± 0.4
Proportion of sleep (%)				
REM	9.8 ± 3.9	21.7 ± 10.8	15.1 ± 3.1	13.4 ± 4.1
Awakenings	1.5 ± 0.4	2.9 ± 1.3	3.3 ± 1.2	10.9 ± 7.0
NREM 1	0.9 ± 0.5	2.1 ± 1.3	4.9 ± 3.1	6.6 ± 2.0
NREM 2	68.6 ± 9.4	54.8 ± 12.5	52.1 ± 5.9	44.5 ± 8.3
NREM 3	9.0 ± 3.6	8.8 ± 2.8	13.9 ± 2.5	11.6 ± 2.5
NREM 4	10.1 ± 5.6	9.7 ± 3.9	10.8 ± 1.7	13.0 ± 3.0

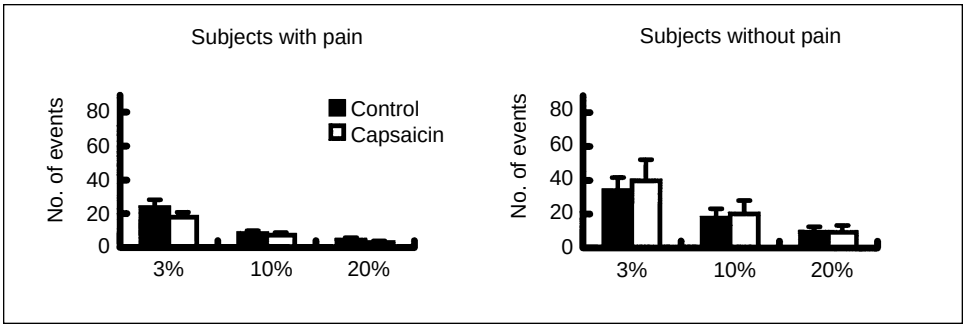
(MANOVA, F = 0.033, *P* = .857), and there was no significant interaction between group, threshold, and time (MANOVA, F = 0.485, *P* = .619). For the analysis of individual variation, the number of EMG episodes/hour sleep was normalized (baseline night = 100%) and compared. There was no significant interaction between group, threshold, and time (subjects with pain: mean 102% [range: 21% to 271%]; subjects without pain: mean 134% [range: 8% to 300%]; MANOVA, F = 0.306, *P* = .742).

Number of EMG Bursts/Hour Sleep. The subjects without pain demonstrated a significantly higher number of EMG bursts/hour sleep as compared to the subjects with pain (MANOVA, F = 5.182, *P* = .034). There was a significant main effect of EMG thresholds on the number of EMG bursts/hour sleep (MANOVA, F = 43.088, *P* = .001) with more activity detected at the 3% level as compared to the 10% level (SNK, *P* = .001) and the 20% level (SNK, *P* = .001) (Figs 3c and 3d). The MANOVA did not show any significant

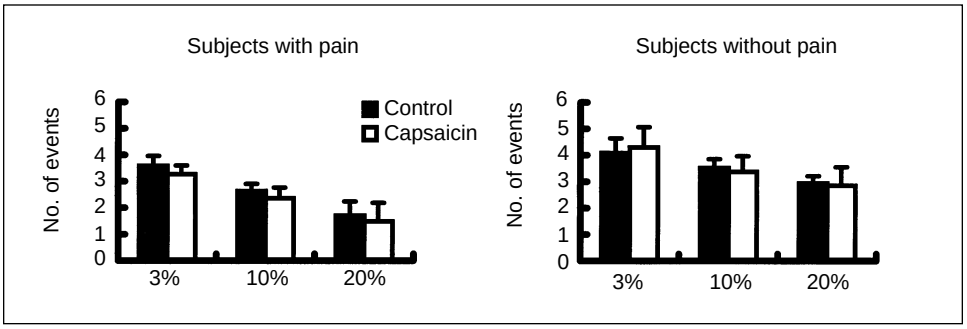
Figs 3a to 3j Analysis of sleep OMA with different EMG thresholds (3%, 10%, and 20%) in subjects with pain (n = 5) and without pain (n = 7). The EMG parameters are defined in the Materials and Methods section.



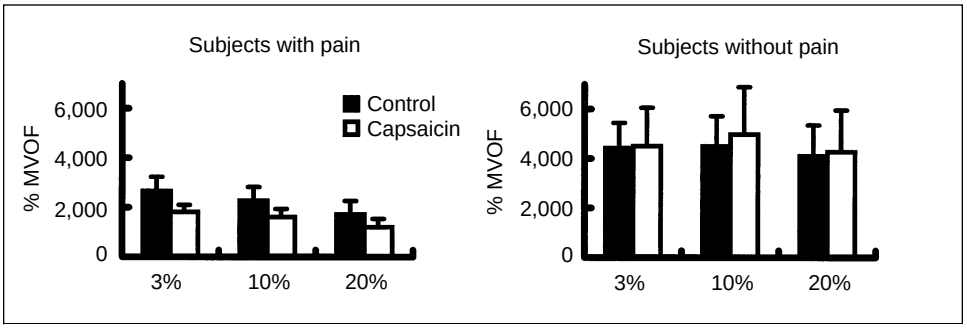
Figs 3a and 3b Number of EMG episodes/hour sleep.



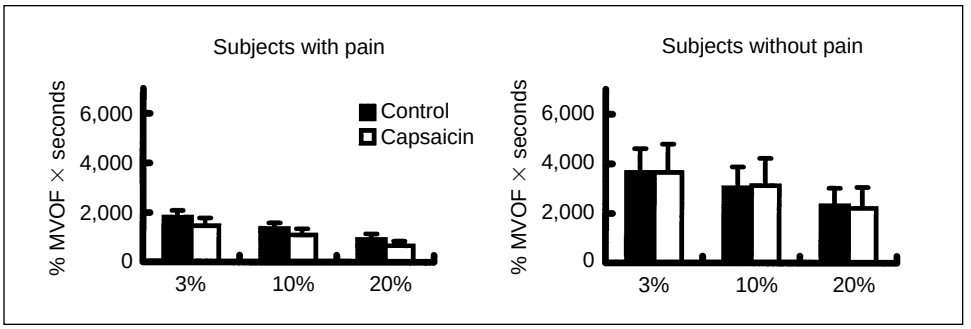
Figs 3c and 3d Number of EMG bursts/hour sleep.



Figs 3e and 3f Number of EMG bursts/episode.



Figs 3g and 3h Total RMS of all EMG bursts and episodes.



Figs 3i and 3j Total area of all EMG bursts and episodes.

differences between the baseline (control) and experimental (capsaicin) nights as an overall test (MANOVA, F = 0.001, *P* = .988). There was no significant interaction between group, threshold, and time (MANOVA, F = 0.761, *P* = .474), but the number of EMG bursts/hour sleep in the subjects with pain tended to be lower at the 3% threshold following the injection of capsaicin (SNK, *P* = .085). The normalized number of EMG bursts/hour sleep did not show any significant differences between group, threshold, and time (subjects with pain: mean 85% [range: 18% to 198%]; subjects without pain: mean 112% [range: 6% to 212%]; MANOVA, F = 0.440, *P* = .650).

Number of EMG Bursts/Episode. The subjects without pain demonstrated a significantly higher number of EMG bursts/episode as compared to the subjects with pain (MANOVA, F = 8.149, *P* = .010). There was a significant main effect of EMG thresholds on the number of EMG bursts/episode (MANOVA, F = 39.157, *P* = .001), with more activity detected at the 3% level as compared to the 10% level (SNK, *P* = .001) and the 20% level (SNK, *P* = .001) (Figs 3e and 3f). The capsaicin injection did not cause significant changes in the number of EMG bursts/episode (MANOVA, F = 0.073, *P* = .789), and there was no significant interaction between group, threshold, and time (MANOVA, F = 0.136, *P* = .874). The normalized number of EMG bursts/episode did not show any significant differences between group, threshold, and time (subjects with pain: mean 105% [range: 32% to 150%]; subjects without pain: mean 103% [range: 33% to 183%]; MANOVA, F = 0.754, *P* = .492).

Total RMS of All EMG Bursts and Episodes. The subjects without pain had significantly higher values of total RMS of all EMG bursts and episodes as compared to the subjects with pain (MANOVA, F = 6.802, *P* = .017). There was a significant main effect of EMG thresholds on the total RMS of all EMG bursts and episodes (MANOVA, F = 8.773, *P* = .001), with more activity detected at the 3% level than at the 20% level (SNK, *P* = .001), but not the 10% level (SNK, *P* = .695) (Figs 3g and 3h). The MANOVA did not show a significant effect of capsaicin injection (MANOVA, F = 0.046, *P* = .833). There was no significant interaction between group, threshold, and time (MANOVA, F = 0.550, *P* = .581), although the total RMS of all EMG bursts and episodes in the subjects with pain also tended to be lower at the 3% threshold following the injection of capsaicin (SNK, *P* = .086). The normalized total RMS of all EMG bursts and episodes did not show any significant differences between group, threshold, and time (subjects with pain: mean 101% [range: 14% to 403%]; subjects without pain: mean 98% [range: 17% to 202%]; MANOVA, F = 0.503, *P* = .612).

Total Area of All EMG Bursts and Episodes. The subjects without pain demonstrated a significantly greater area of EMG activity as compared to the subjects with pain (MANOVA, F = 7.304, *P* = .014). There was a significant main effect of EMG thresholds on the total area of all EMG bursts and episodes (MANOVA, F = 45.253, *P* = .001), with more activity detected at the 3% level as compared to the 10% level (SNK, *P* = .001) and the 20% level (SNK, *P* = .001) (Figs 3i and 3j). The MANOVA did not show a significant effect of capsaicin injection (MANOVA, F = 0.174, *P* = .681), and there was no interaction between group, threshold, and time (MANOVA, F = 0.363, *P* = .698), although the total area of all EMG bursts and episodes in the subjects with pain tended to be lower at the 3% threshold after the capsaicin injection (SNK, *P* = .083). The normalized total area of

all EMG bursts and episodes did not show any significant differences between group, threshold, and time (subjects with pain: mean 96% [range: 13% to 403%]; subjects without pain: mean 111% [range: 6% to 300%]; MANOVA, F = 0.006, *P* = .993).

Discussion

The main finding in this experimental pain study was that subjects with clinical pain and soreness in the masticatory muscles demonstrated significantly lower levels of OMA during sleep than subjects without subjective complaints. Furthermore, experimentally induced pre-sleep jaw muscle pain evoked by a single injection of capsaicin did not cause any carry-over effect on sleep OMA in the 2 groups of subjects.

Orofacial Motor Activity and Clinical Jaw Muscle Pain

Although all subjects in the present study reported a positive history of OMA during sleep, no subject actually fulfilled the criteria and cutoff values outlined by Lavigne et al.[13] As a consequence, the term "bruxers" was not used in the present study, but all subjects had signs of tooth wear and had reports of noises coming from the teeth during sleep. Furthermore, complaints about pain and soreness in the morning were reported by the subjects, but none of them were actively seeking treatment. This is a major difference between the present study and the study of Lavigne et al.[13]

Another point is that sleep bruxism or OMA does not seem to be consistent. Rugh and Harlan[8] reported that bruxism fluctuated over time. Furthermore, Lavigne et al[13] reported that some bruxers show an absence of grinding during an experimental period, although these subjects have a positive history of frequent grinding sounds, tooth wear, and muscle hypertrophy. This means that many baseline recordings should be performed in the sleep laboratory; unfortunately, this is a very time-consuming and expensive approach. Ambulatory EMG recordings might be an attractive option to study long-term patterns of OMA during both daytime and sleep.[32–37] With respect to the analysis of OMA, Ikeda et al[14] suggested that a 10% MVOF threshold is appropriate for analysis of the smoothed integrated EMG signal. It has been shown that a 20% EMG threshold eliminates about 49% of potential bruxism events, whereas a 10% EMG threshold eliminates only 22% of potential bruxism events. In accordance with this observation, we demonstrated a significant main effect of the 3 different EMG threshold levels, so that the lowest threshold was associated with the highest number of episodes/hour sleep, bursts/hour sleep, and bursts/episode and the highest levels of total RMS and EMG areas of all bursts and episodes (Figs 3a to 3i). Even when these potential confounding factors were taken into consideration, the present study showed a significantly lower number of EMG episodes/hour sleep, number of EMG bursts/hour sleep, number of EMG bursts/episode, total RMS of all EMG bursts and episodes, and total area of all EMG bursts and episodes in subjects with pain or soreness as compared to subjects without pain. These results support and extend the findings from Lavigne et al,[16] that bruxers with pain have about 40% fewer EMG episodes per hour of sleep than those without pain. Thus, even low levels of clinical pain and soreness seem to be associated with depression of jaw motor function in sleeping subjects. This observation is in agreement with the suggestion of the pain-adaptation model,[38] which also has gained support from experimental studies in decerebrate animals[39,40] and awake humans.[41–43]

Orofacial Motor Activity and Experimental Jaw Muscle Pain

A capsaicin injection was used in this study to evoke longer-lasting jaw muscle pain and soreness in subjects with and without clinical symptoms in the masticatory muscles.[24,25,27] The peak pain intensity on the electronic VAS, the area under the VAS curve, and the duration of pain were not significantly different between the 2 study groups, and the values were within the same range as that of a previous study.[12] Interestingly, 5 minutes after the injection of capsaicin, the subjects without pain showed significantly larger areas of pain on facial drawings, although they tended to report lower VAS scores for pain intensity compared to the subjects with pain (Figs 2a to 2c). Moreover, the subjects without pain perceived higher VAS levels of soreness 5 minutes after the capsaicin injection. The reason for the discrepancy between the pain drawings and VAS scores of pain and soreness is not known but could be related to individual differences in the definition and recognition of "pain" and "soreness." However, the number of subjects in the 2 groups is limited, and the results regarding perceived pain and soreness should be interpreted cautiously.

In contrast to the significant effect of clinical pain and soreness on OMA, there were no consistent changes in any of the EMG parameters between the baseline (control) and the experimental (capsaicin) nights. One reason may be the substantial within-subject variability in the measures of OMA. Furthermore, the low levels of experimental pain and soreness induced by the single capsaicin injection might not have been effective in changing OMA during sleep. The lack of changes in PDT and MVOF is in agreement with this observation. However, the experimentally induced pain and soreness clearly did not increase OMA, but rather tended to decrease sleep OMA, particularly in subjects with pain (number of EMG bursts/hour sleep, total RMS of all EMG bursts and episodes, and total area of all EMG bursts and episodes). It was not possible to determine whether the acute pain and soreness evoked by the single capsaicin injection persisted throughout the night, but it probably did only to a minor extent, because there were no significant changes in PDTs, MVOF, or subjective VAS scores the next morning. Recently, Drewes et al[44] and Lavigne et al[45] performed EEG recordings with several kinds of painful stimulation (thermal stimulation, saline injection, pressure/pain, and laser stimulation) applied during sleep, and they reported that acute pain during sleep is linked to arousal effects and shifts toward lighter sleep stages. However, another recent study showed no major sleep disruptions following experimentally induced PEMS.[46] Future studies might investigate the effect of intramuscular noxious stimuli administered during sleep on EEG responses and jaw muscle activity to obtain better insight into the relationship between jaw muscle pain and OMA during sleep.

The present study showed that subjects with pain and soreness in the masticatory muscles have significantly lower levels of OMA during sleep than subjects without complaints. Furthermore, the experimental painful stimulus applied to the masseter muscle before sleep did not cause any changes in sleep OMA. These findings suggest that pain per se may not be associated with an excitatory effect on human jaw-motor function.

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