Females with Sleep Bruxism Show Lower Theta and Alpha Electroencephalographic Activity Irrespective of Transient Morning Masticatory Muscle Pain

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Aims: *To investigate the hypothesis that the presence of transient morning masticatory muscle pain in young, healthy sleep bruxers (SBr) is associated with sex-related differences in sleep electroencephalographic (EEG) activity.* ***Methods:*** *Data on morning masticatory muscle pain and sleep variables were obtained from visual analog scales and a second night of polysomnographic recordings. Nineteen normal control (CTRL) subjects were age- and sex-matched to 62 tooth-grinding SBr. Differences in sleep macrostructure (stage distribution and duration, number of sleep-stage shifts), number of rhythmic masticatory muscle activity (RMMA) events/hour, and EEG activity were analyzed blind to subject status. The influence of pain and gender in SBr and CTRL subjects was assessed with the Fisher's exact test, Mann-Whitney* U *test, two-sample* t *test, and analysis of variance (ANOVA).* ***Results:*** *Low-intensity morning transient orofacial pain was reported by 71% of SBr, with no sex difference. RMMA event frequency was higher in SB than CTRL subjects (4.5/hour vs 1.3/hour;* P < *.001). SBr had fewer sleep-stage shifts, irrespective of sex or pain status. Female SBr had significantly lower theta and alpha EEG activity compared to female CTRL subjects (*P = *.03), irrespective of pain.* ***Conclusion:*** *Female SBr had lower theta and alpha EEG activity irrespective of transient morning pain.* J OROFAC PAIN 2013;27:123–134. doi: 10.11607/jop.999

Key words: EEG power spectral analysis, pain, sex, sleep bruxism, theta wave activity

Sleep bruxism (SB) is a sleep-related movement disorder defined as the oral activity of tooth grinding and clenching during sleep.[1] SB is identified from electromyographic (EMG) recordings of the masseter and/or temporalis muscles, and is further defined as rhythmic masticatory muscle activity (RMMA).[2,3] Morning headache (MHA) is a condition that is empirically defined (ie, not yet defined by the International Headache Society) as a recurrent, bilateral, and pressing pain present at awakening ≥ 3 times/week and lasting from 30 minutes up to 4 hours, according to the latest evidence-based data.[4–8] MHA is usually a tension-type or migraine headache, both with reports of a sense of pressing (over 60% of cases) or throbbing (11% to 46%).

Young, healthy subjects suffering from SB often report low-intensity transient morning masticatory muscle pain, mostly localized in the masseter and temporalis muscles, in the first hours after awakening.[2] Notably, SB subjects (sleep bruxers, SBr) with the lowest RMMA frequency also have the highest risk of reporting transient morning orofacial pain and headache (odds ratio of 3.9 and 4.9, respectively).[2,9] Both MHA and morning masticatory muscle pain are transient, and it is possible that the two conditions overlap.

Transient morning masticatory muscle pain in SBr appears to differ from jaw-related myofascial pain.[10–13] SB-related transient morning pain is short-lasting, whereas myofascial pain tends to predominate in the late afternoon or evening.[13] It has been observed that 23% of masticatory myofascial pain patients report awakening from sleep due to pain. Conversely, none of the young SBr studied woke due to orofascial pain during the nights in the sleep laboratory.[2,14] In the presence of clinical pain complaints, the frequency of RMMA with grinding sounds tends to be lower. Similar findings were shown in experimental studies on jaw muscle pain.[15,16]

Electroencephalographic (EEG) activity can be analyzed using Fast Fourier Transform (FFT) to quantify the power at each EEG frequency, averaged across the entire sample, to obtain the power spectrum.[17] EEG spectral analysis allows quantifying sleep depth and intensity, and can be used to assess the influence of pain on sleep.[18] Estimated delta EEG power over the non–rapid eye movement (REM) to REM ultradian sleep cycle can be used to assess the sleep homeostatic process.[19] This method has been used to describe sleep EEG activity in chronic pain patients with fibromyalgia or chronic widespread pain (CWP).[20,21] These studies revealed that delta EEG power declines in fibromyalgia patients over consecutive sleep cycles across the night, and that female patients lose delta EEG power in the first non-REM to REM sleep cycle. In contrast, higher alpha EEG activity was frequently found in fibromyalgia patients. However, as alpha EEG activity is not pathognomonic of chronic pain, its power to explain pain persistence remains under debate.[20–24] It is still unknown whether the sex-specific EEG signature observed in CWP patients is present in SBr patients with morning masticatory muscle pain. Therefore, this study further investigates the hypothesis that the presence of transient morning masticatory muscle pain in young, healthy SBr is associated with sex-related differences in sleep EEG activity.

Materials and Methods

Study Population

A total of 106 subjects were invited to participate in a sleep laboratory study to compare patients with a positive diagnosis of SB to control (CTRL) subjects. Data were collected from 1999 to 2009. All subjects signed an informed consent form and the study was approved by the Sacré-Coeur Hospital Ethics Review Board. Both male and female subjects were selected to participate in a case-control study if they met the following criteria: *(1)* specific criteria for SB with and without report of transient morning masticatory muscle pain (SBr) and *(2)* absence of any clinical sign or symptom suggestive of SB or report of tooth-grinding noises during sleep (CTRL subjects).

Subject Selection Criteria

Subjects were recruited for the study through newspaper advertisements and notices posted at universities and colleges. An initial telephone interview was held to assess whether subjects met (or not) the inclusion and exclusion criteria. To be included, SBr had to report a history of frequent tooth grinding occurring at least 3 nights per week for the preceding 6 months, as confirmed by a sleep partner. In addition, they had to present one of the following: *(1)* abnormal tooth wear; *(2)* jaw muscle discomfort, pain, or jaw locking upon awakening; or *(3)* masseter muscle hypertrophy upon voluntary forceful clenching.[1,2,25,26] CTRL subjects had to report no awareness of tooth grinding, an indirect indication of SB. General exclusion criteria for both SBr and CTRL subjects were the presence of temporomandibular disorders (TMD), retrognathia or prognathia, a history of neurological or psychiatric disorders, bodily or orofacial pain (eg, musculoskeletal or neuropathic pain), or a known history of sleep disorders (eg, insomnia, periodic limb movement, sleep-disordered breathing such as apnea or hypopnea, REM sleep behavior disorder, somnambulism, nightmares, nocturnal terrors, or sleep enuresis). Participants with fewer than two posterior teeth (third molars excluded) or who wore a full or partial denture were excluded.

The initial selection and allocation to the CTRL or SBr group was confirmed by a polysomnographic (PSG) recording, in which RMMA was scored and grinding noises were monitored. Of the total sample of 106 subjects, 25 were excluded due to uncertain CTRL or SBr status (eg, low RMMA index, only one grinding sound, or missing EEG or EMG data). The final sample included 81 subjects (58% women and 42% men). Mean age (± SEM) was 25.8 ± 0.7 years (range 18 to 44 years). The total sample comprised 19 CTRL subjects (8 females and 11 males) and 62 SBr (39 females and 23 males).

Transient Morning Jaw Muscle Pain

SBr were further divided into two subgroups according to the regular presence or absence of transient morning masticatory muscle pain at awakening (once a week to a few times a week). No

CTRL subject reported regular morning jaw pain at awakening. Of the 62 SBr, 44 (71%) were classified into the SBr subgroup with morning masticatory muscle pain (SBrP) and 18 into the SBr subgroup without morning masticatory muscle pain (SBrN). Of the 44 SBrP subjects, 90.1% had frequent transient morning masticatory muscle pain and 9% had persistent pain during the day. Of these 44 SBrP subjects, 31% had transient masticatory muscle pain upon awakening on the morning of the second sleep laboratory night. The sex distribution for the 44 SBrP subjects was 30 females and 14 males. At the screening interview, subjects were also asked to report (using a yes or no response) if they had MHA.

Data on the intensity of morning jaw muscle pain and sleep quality were gathered from questionnaires. All subjects were asked to assess on a 0- to 100-mm visual analog scale (VAS) the intensity of their jaw muscle pain upon awakening in the sleep laboratory after the polysomnography (question 1), the level of sleep disruption related to tooth grinding-bruxism in the sleep laboratory night (question 2), and the overall orofacial pain intensity reported at the first visit (clinical screening examination) (question 3).

PSG Recording, Sleep Variable Scoring, and Analysis

PSG data were recorded in a sound-attenuated, temperature-controlled sleep laboratory room for two consecutive nights. No subjects were taking sleep medication or were under the influence of alcohol, nicotine, or caffeine at the time of recording. The first night allowed subjects to adapt to the sleep laboratory environment (habituation) and confirmed the absence of sleep disorders (eg, sleep breathing, periodic limb movements, or insomnia). Data recorded during the second night were used for the analysis presented in this paper.

Sleep data were collected as follows: brain activity with two surface EEG leads (C_3-A_2 and O_2-A_1), cardiac activity with electrocardiographic (ECG) recoding, eye movement for REM sleep recognition with bilateral electrooculographic (EOG) recordings, and muscle activity with EMG recordings of the chin/suprahyoid, bilateral masseter, temporalis, and tibialis muscles. Respiratory activity was monitored using an airflow sensor and a nasal cannula (from year 2004), chest movement sensors (chest and abdominal belts), and oxymetry for oxygen saturation levels (SaO_2). Audio-video recordings were made to distinguish SB from nonspecific movements such as snoring or breathing cessation (eg, apnea and/or hypopnea) and other orofacial movements.[27–29] All signals were sampled at an acquisition rate of 256 Hz and converted into 128 Hz for storage and off-line analysis using commercial software (formerly Harmonie, Stellate Systems Software, Montréal, QC, Canada; now Natus, USA). Sleep stages 1 to 4 were scored manually according to a modified method using 20-second sleep segments.[30]

For the SB variables, masseter and temporalis muscle activity was classified into three types: *(1)* phasic episode (three or more EMG bursts lasting 0.25 to 2.0 seconds each); *(2)* tonic episode (one EMG burst > 2.0 seconds); and *(3)* mixed episode (both burst types).[2,28] SB activity is presented as the RMMA index over total sleep time (hours).

All analyses were performed blind to subject status (ie, CTRL or SBr, without or with pain).

Quantitative Analysis of EEG Activity

EEG data were initially analyzed to exclude the presence of artifacts due to rhythmic masseter muscle activity, body movements, or electrode displacement. Artifacts were eliminated manually for each night by an investigator (SA) trained by an electrophysiology technician. The technician then confirmed the artifact exclusions for each subject. Inter-rater reliability between investigator and electrophysiology technician was good, and the intraclass correlation coefficient (ICC) using Cohen's Kappa test was 0.92.

Similar to a previous study, four artifact-free non-REM and four artifact-free REM sleep periods were selected over the total sleep period.[21] Continuous durations of non-REM and REM sleep cycles were at least 50 minutes and 5 minutes, respectively. To normalize the duration of the first four non-REM/REM sleep cycles, non-REM periods were divided into 20 intervals and REM periods into 5 intervals according to Achermann's method.[19,31] Thus, 80 non-REM and 20 REM intervals were selected. Each sleep cycle was then averaged into four non-REM sections and one REM section (each made by 5 intervals). Non-REM/REM sleep cycles from the first to the fourth sleep cycle were labeled C1 to C4. Individual sections in non-REM sleep cycles were labeled N1 to N4 from the first to the fourth section. The REM period section was labeled R. Labels were therefore assigned as C1N1 to C1R for each section over four consecutive sleep cycles.

The EEG power spectral analysis of the C_3-A_2 derivation was computed with Fast Fourier Transforms (FFT) using commercial software (formerly Harmonie, Stellate Systems Software; now Natus). EEG signals were quantified to estimate the power of delta band (0.50 to 4.00 Hz), theta band (4.00 to 8.00 Hz), alpha band (8.00 to 13.00 Hz), sigma band (12.75 to

Table 1 Self-Reports in Response to Questions (Q) About Morning Masticatory Muscle Pain Intensity (Q1), Perception of Sleep Disruption on Awakening in the Sleep Laboratory (Q2), and Orofacial Pain (OFP) Intensity at First Visit (Q3) Assessed on a 0- to 100-mm VAS: Comparisons Between Controls (CTRL) and Sleep Bruxers (SBr), CTRL and Subgroups SBr Without Pain (SBrN) and With Pain (SBrP), and CTRL and Males and Females

	CTRL Median (Min–Max)	SBr Median (Min–Max)	*P*
Q1: Morning pain	**0 (0–2)**	**0 (0–65)**	**.022**
Q2: Sleep disrupt	**0 (0–0)**	**0 (0–72)**	**.003**
Q3: OFP 1st visit	**0 (0–0)**	**0 (0–60)**	**.001**
	CTRL Median (Min–Max)	SBrN Median (Min–Max)	*P*
Q1: Morning pain	0 (0–2)	0 (0–0)	.317
Q2: Sleep disrupt	**0 (0–0)**	**0 (0–12)**	**.050**
Q3: OFP 1st visit	0 (0–0)	0 (0–10)	.240
	CTRL Median (Min–Max)	SBrP Median (Min–Max)	*P*
Q1: Morning pain	**0 (0–2)**	**0 (0–65)**	**.003**
Q2: Sleep disrupt	**0 (0–0)**	**2 (0–72)**	**.001**
Q3: OFP 1st visit	**0 (0–0)**	**4 (0–60)**	**< .001**
	Female CTRL Median (Min–Max)	Female SBrN Median (Min–Max)	*P*
Q1: Morning pain	0 (0–2)	0 (0–0)	.289
Q2: Sleep disrupt	0 (0–0)	0 (0–10)	.456
Q3: OFP 1st visit	0 (0–0)	0 (0–3)	.456
	Female CTRL Median (Min–Max)	Female SBrP Median (Min–Max)	*P*
Q1: Morning pain	0 (0–2)	0 (0–65)	.063
Q2: Sleep disrupt	**0 (0–0)**	**2 (0–72)**	**.017**
Q3: OFP 1st visit	**0 (0–0)**	**7 (0–60)**	**.005**
	Male CTRL Median (Min–Max)	Male SBrN Median (Min–Max)	*P*
Q1: Morning pain	0 (0–0)	0 (0–0)	1.000
Q2: Sleep disrupt	0 (0–0)	0 (0–12)	.051
Q3: OFP 1st visit	0 (0–0)	0 (0–10)	.378
	Male CTRL Median (Min–Max)	Male SBrP Median (Min–Max)	*P*
Q1: Morning pain	**0 (0–0)**	**0 (0–60)**	**.021**
Q2: Sleep disrupt	**0 (0–0)**	**0 (0–36)**	**.050**
Q3: OFP 1st visit	**0 (0–0)**	**3 (0–30)**	**.009**

Mann-Whitney *U* tests were used to compare groups. Significant differences are in **bold**.
Min, minimum; Max, maximum.

15.00 Hz), low beta band (13.00 to 22.00 Hz), and high beta band (22.00 to 32.00 Hz) activity.[21]

Statistical Analyses

Data on transient morning pain were analyzed using Fisher's exact test. Pain intensity and sleep disturbances reported by SBr were analyzed using Mann-Whitney *U* tests. The odds ratio (OR) for MHA was estimated using the chi-square test.

To assess differences in sleep architecture and SB variables, two-sample *t* tests were performed to compare SBr and CTRL subjects. The absence or presence of pain was compared between SBr and CTRL subjects by using one-way analysis of variance (ANOVA). The same test was used for sex comparisons. These tests were performed using SYSTAT 11 (Systat Software, Point Richmond, CA, USA).

The EEG power for all bands was analyzed using repeated measures ANOVA with one independent variable (subject group) as a between-subject variable, and sleep cycle and section in each sleep cycle as within-subject variables across frequency bands during non-REM sleep. The Huynh-Feldt correction was applied to *P* values. EEG power distribution for each band in each sleep cycle was graphically represented and assessed with the Shapiro-Wilk normality test. Because all EEG power distributions were stable, raw data were used to analyze EEG power for each band. All data were pooled and averaged

Table 2 Sleep and RMMA Parameters for Controls (CTRL) and Sleep Bruxers (SBr)

	CTLR (n = 19)	SBr (n = 62)	*P* value (*t* test)
Age	24.05 ± 1.26	26.37 ± 0.70	.13
Sex	8 F/11 M	39 F/23 M	
Sleep duration	431.05 ± 8.66	443.34 ± 4.69	.21
Sleep efficiency*	93.76 ± 1.24	95.17 ± 0.48	.17
Sleep latency*	11.46 ± 1.17	9.94 ± 1.19	.06
REM latency*	91.15 ± 7.24	80.60 ± 4.38	.15
Awakenings	28.29 ± 5.72	21.76 ± 2.22	.24
Stage 1 (%)	5.96 ± 0.51	6.25 ± 0.43	.74
Stage 2 (%)	55.21 ± 1.55	55.64 ± 1.18	.85
Stage 3 + 4 (%)	17.83 ± 1.55	14.90 ± 1.15	.21
Stage REM (%)	20.99 ± 0.84	23.18 ± 0.56	.06
Sleep-stage shift	**241.32 ± 15.35**	**198.55 ± 6.27**	**< .01**
Microarousals/hr*	9.33 ± 2.02	6.99 ± 0.52	.65
RMMA episodes			
Episodes/hr*	**1.34 ± 0.22**	**4.51 ± 0.39**	**< .001**
Phasic episodes/hr*	**1.00 ± 0.23**	**2.85 ± 0.31**	**< .001**
Tonic episodes/hr*	0.03 ± 0.02	0.12 ± 0.04	.25
Mixed episodes/hr*	**0.31 ± 0.09**	**1.50 ± 0.18**	**.01**
Episodes with noise*	**0.37 ± 0.17**	**8.89 ± 1.68**	**< .001**
RMMA bursts			
Bursts/hr*	**6.41 ± 1.27**	**31.28 ± 3.20**	**< .001**
Phasic bursts/hr*	**5.94 ± 1.25**	**28.55 ± 3.07**	**< .001**
Tonic bursts/hr*	**0.47 ± 0.15**	**2.14 ± 0.29**	**< .01**
Bursts/Episodes*	**4.93 ± 0.42**	**6.38 ± 0.28**	**< .01**

*Applied logarithm for normalization.
Data are presented as mean ± SEM. Significant differences are in **bold**.

for each subject (mean ± SEM). Conversely, abnormally distributed sleep and SB variables were normalized with a logarithm. A *P* value of .05 or less was considered statistically significant.

Results

Pain and Sleep Disruption Reports

Overall, the intensity of morning masticatory muscle pain reported by the 62 SBr was low, but significantly higher than that of CTRL subjects (Table 1). Of the 62 SBr, 44 (71%) reported low-intensity morning masticatory muscle pain. The comparison to CTRL subjects was statistically significant, with no sex difference. In the 62 SBr, the perceived disruption of sleep quality in the sleep laboratory due to tooth grinding was low, but significantly higher than in CTRL subjects. The overall daytime orofacial pain intensity at the first visit was also low, but it differed significantly when the 62 SBr were compared to CTRL subjects, an effect found irrespective of sex in SBrP only.

The proportion of SBr who reported MHA was 28.3% (based on the 53 of the 62 SBr who responded to this question), whereas only 1 CTRL subject (of the 13 who responded to this question) complained of this symptom. The OR of reporting MHA in SB was 4.7 (*P* = .16), and no further difference was found in terms of sex or overall pain status.

Sleep Architecture and Sleep Bruxism Variables

With the exception of sleep-stage shifts, which were significantly lower in SBr, the comparison of the other sleep variables between the 19 CTRL subjects and 62 SBr did not reach statistical significance (Table 2). Of the statistically significant RMMA-SB variables, SBr had about 3.5 times more RMMA episodes per hour of sleep, with a significant dominance of phasic and mixed episodes. Moreover, the number of RMMA bursts per hour was 4.5 times higher in SBr than in CTRL subjects.

The comparison of CTRL subjects (n = 19) to SBr with pain (SBrP; n = 44) and SBr without pain

Table 3 Sleep and RMMA Parameters for Controls (CTRL), Sleep Bruxers with Pain (SBrP), and Sleep Bruxers without Pain (SBrN)

	CTRL (n = 19) a	SBrP (n = 44) b	SBrN (n = 18) c	*P* value			
					Tukey test		
				ANOVA†	a vs b	a vs c	b vs c
Age	24.05 ± 1.26	26.27 ± 0.84	26.61 ± 1.31	.32			
Sex	8 F/11 M	30 F/14 M	9 F/9 M				
Sleep duration	431.05 ± 8.66	449.39 ± 5.44	428.54 ± 8.50	.06			
Sleep efficiency*	93.76 ± 1.24	95.32 ± 0.58	94.83 ± 0.90	.36			
Sleep latency*	11.46 ± 1.17	8.45 ± 1.39	13.58 ± 2.17	.06			
REM latency*	91.15 ± 7.24	82.19 ± 5.22	76.73 ± 8.16	.36			
Awakenings	28.29 ± 5.72	21.63 ± 2.66	22.09 ± 4.15	.49			
Stage 1 (%)	5.96 ± 0.51	6.07 ± 0.51	6.71 ± 0.80	.74			
Stage 2 (%)	55.21 ± 1.55	55.93 ± 1.41	54.95 ± 2.20	.91			
Stage 3 + 4 (%)	17.83 ± 1.55	14.61 ± 1.37	15.60 ± 2.14	.42			
Stage REM (%)	20.99 ± 0.84	23.38 ± 0.67	22.74 ± 1.04	.15			
Sleep stage shift	**241.32 ± 15.35**	**197.80 ± 7.49**	**200.39 ± 11.71**	**.01**	**< .01**	**.02**	.86
Microarousals/hr*	9.33 ± 2.02	6.82 ± 0.62	7.38 ± 0.96	.64			
RMMA episodes							
Episodes/hr*	**1.34 ± 0.22**	**4.25 ± 0.42**	**5.15 ± 0.83**	**< .001**	**< .001**	**< .001**	.72
Phasic episodes/hr*	**1.00 ± 0.23**	**2.79 ± 0.38**	**2.99 ± 0.56**	**< .01**	**< .01**	**< .01**	.81
Tonic episodes/hr*	0.03 ± 0.02	0.11 ± 0.03	0.15 ± 0.09	.38			
Mixed episodes/hr*	**0.31 ± 0.09**	**1.28 ± 0.18**	**2.01 ± 0.42**	**.01**	**.15**	**.01**	.20
Episodes with noise*	**0.37 ± 0.17**	**7.18 ± 1.48**	**13.06 ± 4.46**	**< .001**	**< .001**	**< .001**	.64
RMMA bursts							
Bursts/hr*	**6.41 ± 1.27**	**28.56 ± 3.77**	**37.92 ± 5.91**	**< .001**	**< .001**	**< .001**	.54
Phasic bursts/hr*	**5.94 ± 1.25**	**25.84 ± 3.66**	**34.87 ± 5.50**	**< .001**	**< .001**	**< .001**	.52
Tonic bursts/hr*	**0.47 ± 0.15**	**1.75 ± 0.25**	**3.05 ± 0.73**	**< .01**	**.03**	**< .01**	.33
Bursts/Episodes*	**4.93 ± 0.42**	**6.09 ± 0.30**	**7.07 ± 0.63**	**.01**	**.06**	**.01**	.48

*Applied logarithm for normalization.
†One-way ANOVA, followed by Tukey pairwise mean comparisons.
Data are presented as mean ± SEM. Significant differences are in **bold**.

(SBrN; n = 18) revealed no significant differences except for sleep-stage shifts, which again were lower in SBrP patients than in CTRL subjects and SBrN (Table 3). For the RMMA data, although RMMA indexes were significantly higher in SBr than in CTRL subjects, no statistical difference was noted between SBrP and SBrN patients (b vs c post-hoc comparisons in Table 3, right panel). Similar results were found for sex comparisons for females (Table 4a) and males (Table 4b).

Quantitative Analysis of EEG Power

SBr vs CTRL Subjects. A decline in the EEG power of delta and theta activity was observed for both SBr and CTRL subjects and over consecutive non-REM to REM ultradian cycles (linear polynomial contrast $P < .001$; Fig 1). Alpha activity was stable (ie, no decline in power) across non-REM to REM cycles (Fig 1). No significant interaction between sleep cycle and subject group was found. Moreover, no significant differences were observed for the EEG power of sigma, low beta, or high beta activity (data not shown).

SBr vs CTRL Subjects With or Without Pain. When SBrP patients were analyzed separately from SBrN patients, a similar decline in delta and theta power was found (linear polynomial contrast $P < .001$), with no difference in alpha power over consecutive non-REM to REM cycles (Fig 2). No significant interaction between sleep cycle and subject group was found. Moreover, no significant difference was observed for the EEG power of sigma, low beta, or high beta activity (data not shown).

Female SBr vs Female CTRL Subjects With or Without Pain. In female subjects, after separating for pain (SBrP and SBrN), a similar decline in delta and theta power was found (linear polynomial contrast $P < .001$), with no difference in alpha power over consecutive non-REM to REM cycles (Fig 3).

Table 4a Sleep and RMMA Parameters for Female Controls (Female-CTRL), Female Sleep Bruxers With Pain (Female-SBrP), and Female Sleep Bruxers Without Pain (Female-SBrN)

	Female-CTRL (n = 8) a	Female-SBrP (n = 30) b	Female-SBrN (n = 9) c	*P* value			
					Tukey test		
				ANOVA†	a vs b	a vs c	b vs c
Age	25.75 ± 2.01	26.07 ± 1.09	25.00 ± 1.19	.88			
Sleep duration	436.41 ± 12.27	456.36 ± 7.19	448.46 ± 11.71	.41			
Sleep efficiency*	93.45 ± 2.05	95.54 ± 0.58	96.60 ± 0.94	.21			
Sleep latency*	10.60 ± 1.88	8.60 ± 1.12	9.22 ± 2.31	.49			
REM latency*	80.49 ± 10.11	80.69 ± 7.50	77.59 ± 9.18	.92			
Awakenings	30.41 ± 9.46	20.73 ± 2.78	15.22 ± 4.21	.11			
Stage 1 (%)	5.68 ± 0.76	5.71 ± 0.48	4.19 ± 0.49	.24			
Stage 2 (%)	52.61 ± 2.00	56.52 ± 1.56	60.17 ± 2.90	.18			
Stage 3 + 4 (%)	20.53 ± 2.96	14.39 ± 1.47	13.84 ± 2.86	.16			
Stage REM (%)	21.20 ± 1.22	23.31 ± 0.86	21.79 ± 1.58	.42			
Sleep-stage shift	**285.63 ± 14.92**	**199.40 ± 8.25**	**181.67 ± 14.75**	**< .001**	**< .001**	**< .001**	.30
Microarousals/hr*	11.39 ± 3.50	7.20 ± 0.68	6.97 ± 2.30	.17			
RMMA episodes							
Episodes/hr*	**1.16 ± 0.31**	**4.02 ± 0.52**	**4.87 ± 0.83**	**< .01**	**< .001**	**< .001**	.72
Phasic episodes/hr*	0.97 ± 0.35	2.66 ± 0.45	3.22 ± 0.82	.07			
Tonic episodes/hr*	0.02 ± 0.02	0.10 ± 0.03	0.01 ± 0.01	.16			
Mixed episodes/hr*	0.17 ± 0.08	1.30 ± 0.21	1.64 ± 0.44	.19			
Episodes with noise*	**0.25 ± 0.25**	**8.27 ± 2.05**	**11.56 ± 3.92**	**.02**	**< .01**	**< .01**	.54
RMMA bursts							
Bursts/hr*	**4.31 ± 1.16**	**26.19 ± 4.20**	**40.08 ± 7.14**	**< .001**	**.04**	**.02**	.69
Phasic bursts/hr*	**4.07 ± 1.18**	**24.62 ± 4.17**	**37.55 ± 6.97**	**< .001**	**< .01**	**< .001**	.20
Tonic bursts/hr*	**0.23 ± 0.11**	**1.75 ± 0.29**	**2.54 ± 0.84**	**.02**	**.03**	**.03**	.85
Bursts/Episodes*	**4.39 ± 0.65**	**5.99 ± 0.37**	**8.15 ± 0.73**	**< .01**	**.05**	**< .01**	.08

*Applied logarithm for normalization.
†One-way ANOVA, followed by Tukey pairwise mean comparisons.
Data are presented as mean ± SEM. Significant differences are in **bold**.

A significant interaction between sleep cycle and subject group was found ($P = .02$) for theta EEG power, as the difference between groups varied across cycles. Although no difference was found for EEG delta power (data not shown), an overall statistical difference was observed for the theta EEG power, which was lower in SBrP and SBrN patients than in CTRL subjects, with further significant differences in the first and second non-REM cycle ($P = .03$). Similarly, alpha power was significantly lower in SBrP and SBrN patients in the overall sleep cycle ($P < .01$) and from the first non-REM to REM to the fourth sleep cycle ($P < .01$, $P < .01$, $P < .01$, $P = .04$, respectively). No difference was found between SBrP and SBrN patients for either theta or alpha EEG activity (data not shown).

Furthermore, no significant difference was observed for the EEG power of sigma, low beta, or high beta activity (data not shown). However, a significant difference in the interaction between sleep cycle and group for high beta activity in CTRL vs SBrN subjects was observed over four consecutive sleep cycles ($P = .02$; data not shown).

Male SBr vs Male CTRL Subjects With or Without Pain. In male subjects, after analyzing separately for pain (SBrP and SBrN), a similar decline in delta and theta power was found (linear polynomial contrast $P < .001$), with no difference in alpha power over consecutive non-REM to REM cycles (Fig 4). No significant interaction between sleep cycle and subject group was found. Moreover, no significant difference was observed in the EEG power of sigma, low beta, or high beta activity (data not shown).

Discussion

This study has shown that female SBr, irrespective of transient morning masticatory muscle pain, have lower EEG theta and alpha activity, with no notable

Table 4b Sleep and RMMA Parameters for Male Controls (Male-CTRL), Male Sleep Bruxers With Pain (Male-SBrP), and Male Sleep Bruxers Without Pain (Male-SBrN)

	Male-CTRL (n = 11) a	Male-SBrP (n = 4) b	Male-SBrN (n = 9) c	*P* value			
					Tukey test		
				ANOVA†	a vs b	a vs c	b vs c
Age	22.82 ± 1.60	26.71 ± 1.76	28.22 ± 2.27	.14			
Sleep duration	427.15 ± 12.36	434.47 ± 5.44	408.63 ± 9.23	.15			
Sleep efficiency*	93.98 ± 1.63	94.83 ± 0.96	93.06 ± 1.48	.66			
Sleep latency*	12.08 ± 1.54	8.14 ± 1.81	17.93 ± 6.66	.07			
REM latency*	98.91 ± 9.82	85.39 ± 8.58	75.87 ± 6.13	.23			
Awakenings	26.75 ± 7.45	23.55 ± 4.58	28.97 ± 6.51	.68			
Stage 1 (%)	6.17 ± 0.72	6.82 ± 1.08	9.23 ± 1.68	.21			
Stage 2 (%)	57.09 ± 2.15	54.65 ± 2.81	49.73 ± 3.27	.23			
Stage 3 + 4 (%)	15.86 ± 1.44	15.07 ± 3.25	17.37 ± 3.33	.86			
Stage REM (%)	20.85 ± 1.20	23.46 ± 1.35	23.69 ± 1.04	.24			
Sleep-stage shift	209.09 ± 19.30	194.36 ± 14.58	219.11 ± 17.69	.59			
Microarousals/hr*	7.83 ± 2.43	6.56 ± 0.95	7.80 ± 1.52	.78			
RMMA episodes							
Episodes/hr*	**1.46 ± 0.31**	**4.72 ± 0.76**	**5.43 ± 1.49**	**< .01**	**< .01**	**.01**	.95
Phasic episodes/hr*	**1.02 ± 0.32**	**3.06 ± 0.73**	**2.75 ± 0.80**	**.03**	**.03**	.07	.99
Tonic episodes/hr*	0.04 ± 0.03	0.12 ± 0.08	0.29 ± 0.17	.44			
Mixed episodes/hr *	0.40 ± 0.14	1.26 ± 0.33	2.38 ± 0.73	.06			
Episodes with noise*	**0.45 ± 0.25**	**4.86 ± 1.39**	**14.56 ± 8.28**	**.02**	**.05**	**.03**	.82
RMMA bursts							
Bursts/hr*	**7.94 ± 1.95**	**33.66 ± 7.77**	**35.76 ± 9.81**	**< .01**	**< .01**	**.03**	.88
Phasic bursts/hr*	**7.31 ± 1.92**	**28.56 ± 7.50**	**32.20 ± 8.86**	**< .01**	**.01**	**.05**	.91
Tonic bursts/hr*	0.64 ± 0.23	1.76 ± 0.47	3.57 ± 1.23	.10			
Bursts/Episodes*	5.32 ± 0.56	6.31 ± 0.52	6.00 ± 0.94	.48			

*Applied logarithm for normalization.
†One-way ANOVA, followed by Tukey pairwise mean comparisons.
Data are presented as mean ± SEM. Significant differences are in **bold**.

difference in perceptions of sleep disruption, an indirect indicator of sleep quality. Because they also reported low-intensity transient morning masticatory muscle pain, this group may differ from CWP and fibromyalgia patient populations.

Young SBr with transient morning masticatory muscle pain (SBrP) in this study reported much lower pain intensity than TMD patients of similar age.[13,32,33] It remains to be understood whether the low intensity of morning pain is due to the transient nature of jaw muscle pain (possibly postexercise muscle pain, which has also been reported at low intensity). This hypothesis remains to be confirmed in SBr.[34,35] In the present study, the OR for SBr to report MHA was 4.7 compared to CTRL subjects, which is similar to previous observations in both adults and pediatric populations.[9,36]

SBr with transient morning masticatory muscle pain also reported low disruption of sleep quality. Although a statistical difference was found between SBr and CTRL subjects, the clinical significance of this difference remains to be determined, ie, the risk of developing poor sleep or insomnia, as reported in TMD patients.[37,38] This study also showed that all SB subjects, irrespective of low-intensity transient pain in the morning, presented a high RMMA index per hour of sleep (4.25 for SBrP and 5.15 for SBrN, no statistical difference; Table 3). In a preliminary study, it was reported that six SBrP patients had higher pain intensity VAS scores in the evening and morning (36.7/100 mm and 44/100 mm, respectively) than SBrN patients.[15] Furthermore, the RMMA index per hour of sleep was significantly lower (40%). In the present study, with 44 SBrP and 18 SBrN patients, no statistical difference was observed for the RMMA index per hour of sleep, number of episodes with tooth grinding sounds, or bursts per hour. It is possible that some of the initial cohort of six SBrP patients observed more than 15 years ago could be better characterized as myofascial pain patients, without temporomandibular joint pain or locking or sounds, because they reported orofacial

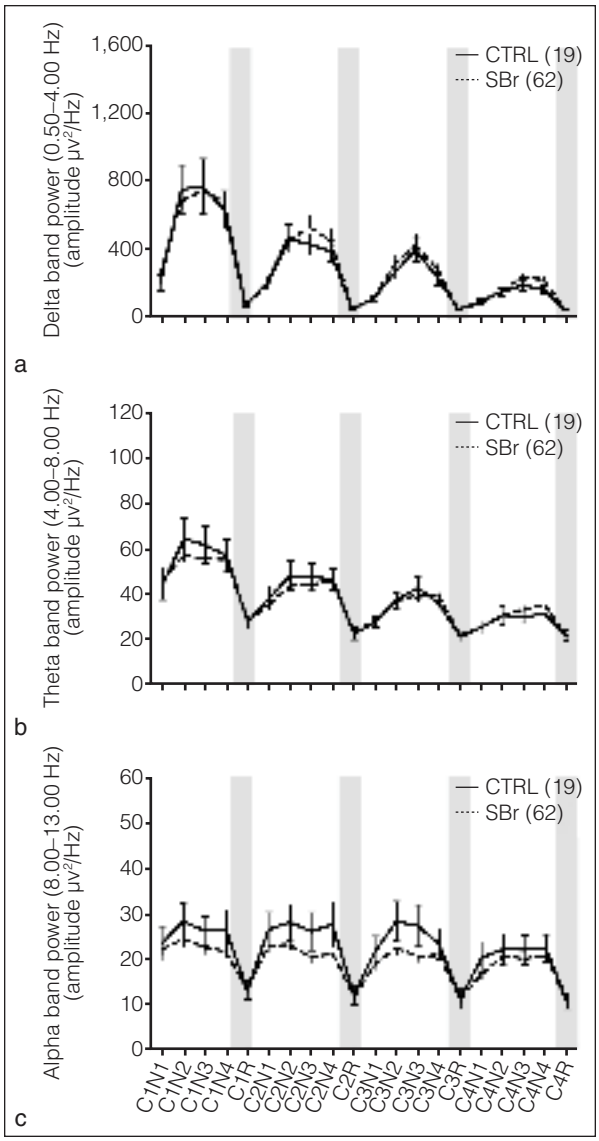

Fig 1 Comparison between control (CTRL, n = 19) subjects and sleep bruxers (SBr, n = 62) over four consecutive non-REM/REM sleep cycles. Solid lines with error bars indicate CTRL. Dashed lines without error bars indicate SBr. Shadowed boxes indicate REM sleep. *(a)* EEG delta power for CTRL and SBr; *(b)* EEG theta power for CTRL and SBr; *(c)* EEG alpha power for CTRL and SBr. C = non-REM and REM cycles. N = number of sections. R = REM sleep.

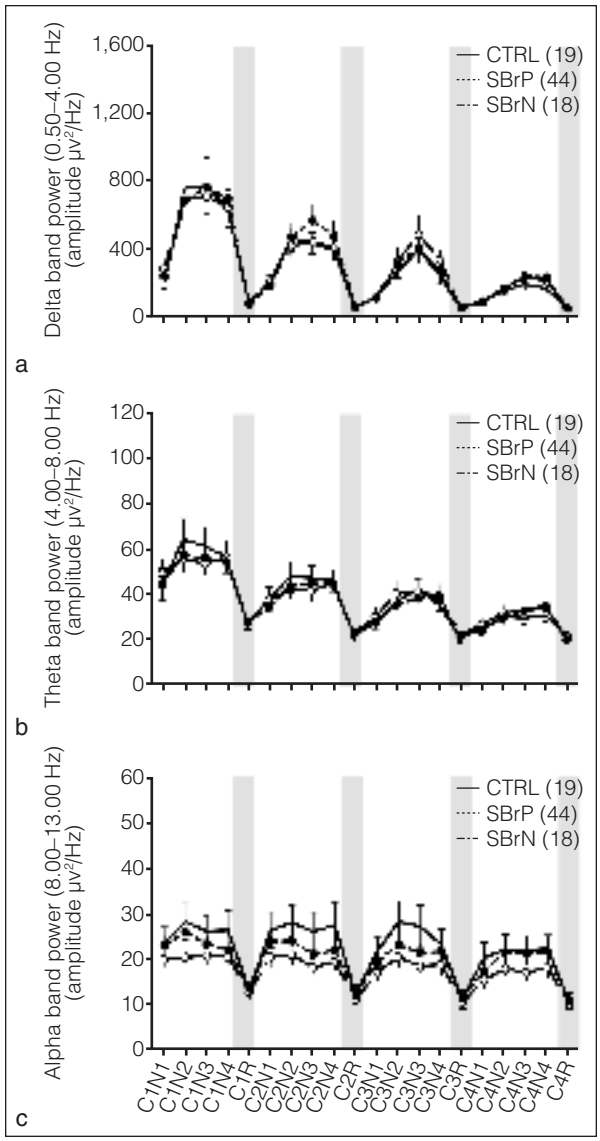

Fig 2 Comparison between control (CTRL, n = 19) subjects, sleep bruxers with pain (SBrP, n = 44), and sleep bruxers without pain (SBrN, n = 18) over four consecutive non-REM/REM sleep cycles. Solid lines with error bars indicate CTRL. Dashed lines with black circles indicate SBrP. Solid and dashed lines with white circles indicate SBrN. Shadowed boxes represent REM sleep. *(a)* EEG delta power for CTRL, SBrP, and SBrN; *(b)* EEG theta power for CTRL, SBrP, and SBrN; *(c)* EEG alpha power for CTRL, SBrP, and SBrN. C = non-REM and REM cycles. N = number of sections. R = REM sleep.

pain in the evening as well.[13,15] In a larger sample, these influences may have been normalized and/or the risk of type II (beta) error reduced. Furthermore, patients with craniofacial pain have significantly higher night-to-night variability in the number of EMG events compared with pain-free individuals.[10]

The present study demonstrated that the sleep power of EEG bands across sleep stages in young subjects with morning masticatory muscle pain differs from that in CWP and fibromyalgia patients. Lower delta power was recently reported in the first non-REM cycle in CWP patients, but also observed

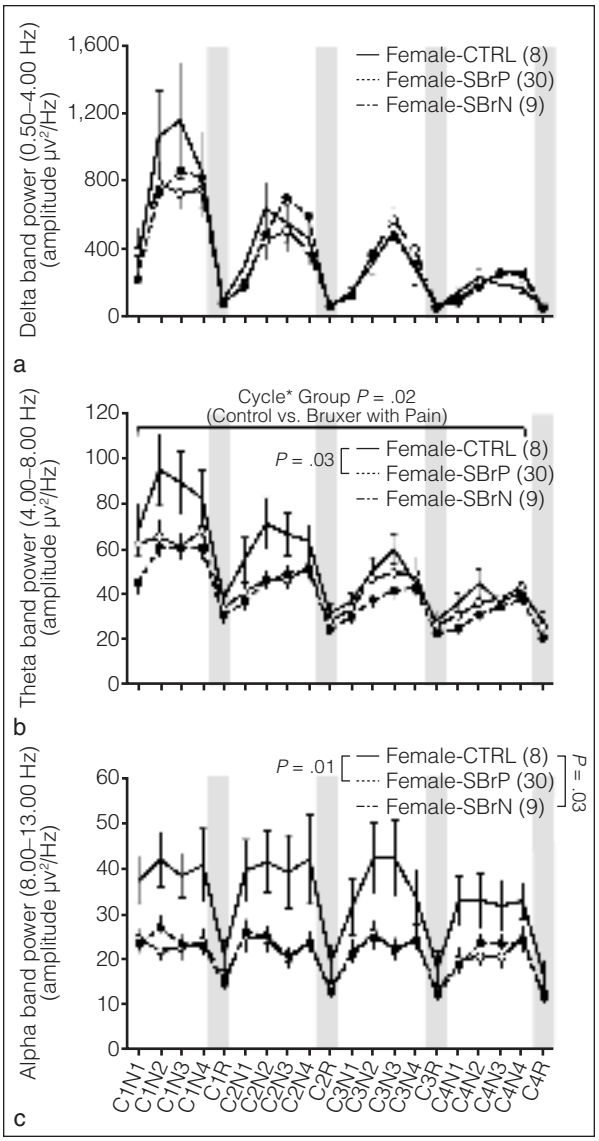

Fig 3 Comparison between female control subjects (Female-CTRL, n = 8), female sleep bruxers with pain (Female-SBrP, n = 30), and female sleep bruxers without pain (Female-SBrN, n = 9) over four consecutive non-REM/REM sleep cycles. Solid lines with error bars indicate Female-CTRL. Dashed lines with black circles indicate Female-SBrP. Solid and dashed lines with white circles indicate Female-SBrN. Shadowed boxes represent REM sleep. *(a)* EEG delta power for Female-CTRL, Female-SBrP, and Female-SBrN; *(b)* EEG theta power for Female-CTRL, Female-SBrP, and Female-SBrN; *(c)* EEG alpha power in Female-CTRL, Female-SBrP, and Female-SBrN. C = non-REM and REM cycles. N = number of sections. R = REM sleep.

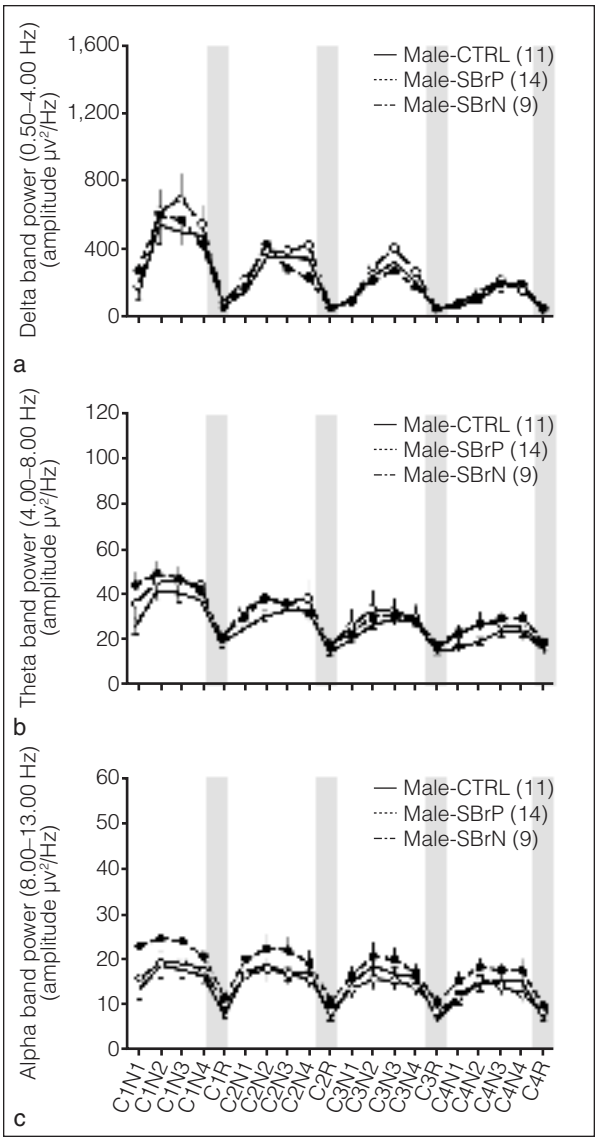

Fig 4 Comparison between male control subjects (Male-CTRL, n = 8), male sleep bruxers with pain (Male-SBrP, n = 30), and male sleep bruxers without pain (Male-SBrN, n = 9) over four consecutive non-REM/REM sleep cycles. Solid lines with error bars indicate Male-CTRL. Dashed lines with black circles indicate Male-SBrP. Solid and dashed lines with white circles indicate Male-SBrN. Shadowed boxes represent REM sleep. *(a)* EEG delta power for Male-CTRL, Male-SBrP, and Male-SBrN; *(b)* EEG theta power for Male-CTRL, Male-SBrP, and Male-SBrN; *(c)* EEG alpha power in Male-CTRL, Male-SBrP, and Male-SBrN. C = non-REM and REM cycles. N = number of sections. R = REM sleep.

in older patients after only one sleep laboratory night.[21] Another difference between the two studies is the nature of CWP and its circadian dominance in the late afternoon and evening. In the present study, jaw muscle pain was transient and reported in the morning. Furthermore, lower alpha activity was found in the present study irrespective of reported transient pain. The debate is ongoing as to the significance of alpha EEG activity as a marker of sleep instability or poor sleep.[21,22] In the previous

study, no difference was found in alpha EEG activity in CWP patients, who were older than the SB subjects in the present study.[21] Other researchers have found higher alpha EEG activity in fibromyalgia patients.[20,24]

A surprising finding of the present study is the lower power of theta EEG activity in female SB subjects irrespective of pain and in the absence of change in delta EEG activity (Fig 3). The delta and theta frequency bands are physiologically linked to brain activity maturation, and their power declines with age. Both are involved in the homeostatic process that preserves sleep quality and function.[19,39] Although the exact role of theta EEG activity is unknown, it has been associated with a variety of psychological processes involved in motor memory and sensory integration during both wake and REM sleep.[40–42] The relevance of the present findings on lower theta EEG activity concomitant with lower alpha EEG activity (lower EEG activity is usually associated with arousal) in SBr remains to be elucidated. It could reflect a pressure to re-establish equilibrium in order to preserve the homeostatic process and sleep continuity or quality following RMMA- or SB-related arousal.[19,43,44]

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