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Descriptive Physiological Data on a Sleep Bruxism Population

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Summary: We studied 24 bruxers (23–67 years old). They often complained of orofacial and bodily pain and presented autonomic symptoms (sweating 23%, palpitations at night 62%, decreased libido 50%); 19% had increased blood pressure requiring treatment, and 65% reported frequent headaches in the morning. Deep sleep and rapid eye movement (REM) were delayed. An average of 167 orofacial episodes developed during the night. The mean number of masseter bursts strictly defined as bruxism was 79, the mean delay for the first occurrence after sleep onset 18 minutes. The majority of bruxism occurred in stage 2 sleep and REM sleep. The mean number of shifts of sleep stages was 70, one-third occurring within the first minute following a bruxing episode, and 15% of bruxing episodes developed after a shift in sleep stage. Electroencephalogram showed alpha-delta pattern in 15% of the subjects. Short-lasting alpha activity was often encountered during the 10 seconds preceding the development of a bruxing episode. Tachycardia developed at its onset, persisting for 10 seconds. We suggest that, as a minor alarm response to endogenous/exogenous stimuli, arousal develops and is often followed by motor activation, such as a burst of bruxing, with, as in any situation when motor activity suddenly increases, a secondary increase of heart activity.
Key Words: Sleep bruxism—Microarousal—Tachycardia.

Little is known about the cause of bruxism. A relationship to stress and anxiety has been suggested (1–3), but the disorder can be chronic without apparent association with stress (4). Bruxism has been associated with other motor manifestations (3,5–8), such as periodic limb movement (PLM) disorders and sleep apnea. It has also been linked to autonomic activity and peripheral vasoconstriction (9). Increases of heart and respiratory rates as well as movement arousals have been reported to be associated with bruxing episodes (9–11). We initiated a multidisciplinary investigation to define the clinical and physiological characteristics of bruxism. The study was performed on carefully selected adult bruxing patients and included clinical examinations following established guidelines, self-reporting procedures, objective sleep recordings, and physiological evaluations using well-defined criteria. The purpose of this first report is to describe the sleep architecture of the patients, to see whether they suffered from other sleep-related disorders, and to identify the occurrence of arousals and autonomic responses associated with bruxing episodes.

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SUBJECTS

We investigated a total of 33 subjects, 11 men (mean age 33 years, range 23–63) and 22 women (mean age 38 years, range 18–67). Twenty-four subjects were selected from a group of professionally active persons who answered an advertisement published in a local newspaper in which we requested subjects complaining of clenching of the teeth or grinding sounds made during sleep. The remaining subjects were patients with known bruxism. All patients consented to the study, which was approved by the Ethical Committee of the Medical Faculty of the University of Gothenburg. The inclusion criteria were that the subjects, otherwise supposed to be healthy and active, would have been aware of their problem for at least 5 years. The parafunctional habit, present almost every night, had to be confirmed either by relatives or by the presence of one or more of the following: hypertrophied masseter muscles, excessive occlusal wear, or muscle and/or temporomandibular joint (TMJ) pain. The subject was included in the study only if the clinical examination confirmed the presence of the disorder. The present report is based on the analysis of 24 subjects, 9 men (mean age 30 years, range 23–42) and 15 women (mean age 38 years, range 23–63) selected from the total pool. The remaining subjects were excluded either because they did not comply with the complete experimental protocol, because the clinical

examination was not conclusive, or because they were later found not to be healthy (a tumor in one patient). The mean age of the subjects selected was 35 years (SD 12, range 23–63), the weight 66 kg (SD 11, range 52–90), and the height 169 cm (SD 9, range 153–190). Body mass index (BMI) was 23 (SD 2, range 18–27).

METHODS

All subjects answered questionnaires about their past dental history and medical and psychosocial situations. From the answers, an anamnestic dysfunction index (AI) was calculated according to Helkimo (12). Symptoms of craniomandibular dysfunction (CMD) and dental status were recorded according to the methods described by Krogh-Poulsen (13) and Carlsson and Helkimo (14). The subjects were examined by a dentist (T.K.), and the findings were summarized in a clinical dysfunction index (DI) according to Helkimo (12). Personality traits were studied by means of a personality inventory, the Karolinska scales of personality (KSP) (15). Sleep habits were reported using the sleep disorder questionnaire (SDQ), including 175 items developed by Douglass et al. (16) and translated into Swedish by one of the authors (G.B.). Daytime sleepiness was assessed using the Epworth sleepiness scale (17). The subjects selected for sleep recordings maintained a sleep diary for at least 14 continuous days. They were instructed not to drink alcohol at least 48 hours prior the recording and not to change sleep habits. Subjects came to the sleep laboratory around 2000 hours and went to bed at their usual time.

Two subjects were studied in separate rooms during the same night in the Sleep Unit at the Department of Clinical Neurophysiology, Sahlgren's University Hospital. Before going to bed, they were examined by a physician (G.B.). The subjects completed forms on which they reported their general condition, the events that had occurred the past day, and sleep quality, which is essential information to determine whether the recording night was representative. Two subjects reported unusual sleep and the recordings had to be repeated a few days later. Weather conditions and room temperature/humidity were also reported. Care was taken to offer comfortable sleeping milieu.

Recordings were performed by well-trained technicians. Three electroencephalography (EEG) channels were obtained using derivations C3-A2, C4-A1, and T3-O1. Two channels were used for electrooculography (EOG), one recording vertical, and the other horizontal, eye movements. Surface chest electrodes were used for recordings of electrocardiography (ECG) and electromyography (EMG) from intercostal muscles for qualitative measurement of respiratory efforts. Submental EMG was recorded by surface electrodes. Muscle activity from right and left masseter and temporal muscles was detected through bipolar electrodes applied to the skin over these muscles, whereas activity from frontal muscles could be detected through EOG and EEG derivations. Respiratory activity was measured using combined oral and nasal thermistors and an abdominal strain gauge. Two silver chloride electrodes were applied to the skin of the lower limb over the anterior tibial muscle to detect EMG activity mirroring limb movements. All 13 signals were recorded simultaneously on conventional paper polygraphs (Neurofax, Nihon-Kohden, Tokyo, Japan). One channel EEG, one channel EOG, submental EMG, and intercostal ECG were derived from the polygraph and simultaneously recorded on a paperless system, the SleepBox (Biosys AB, Goteborg, Sweden). A piezoelectric movement sensitive pad was placed under the patient's mattress and connected to this system. With the use of electronic filtering, respiratory and cardiac [ballistocardiography (BCG)] movements could be identified and separated from body movements. The oxygen saturation level was measured using a pulse oximeter probe placed on a finger (index).

Subjects were recorded in the dark using infrared video cameras and observed by the technicians on monitors outside the room. We used either small microphones, directly applied on the patient, or directionally sensitive microphones close to the patient for recording sounds on the videotapes and simultaneously on the paper polygraph. At the beginning of the recording, calibration was performed: the subjects were asked to clench their teeth at low and then maximum strength levels; to move their lower mandible to the right and left, to swallow, to move their feet and lower limbs, to hold breathing and to take deep breaths, and to move their eyes left and right, up and down.

Analysis

Sleep was analyzed visually and scored on a 20-second epoch base according to Rechtschaffen and Kales (18). All scorings were performed by the same trained sleep technician (M.B.) and supervised by a physician (G.B.). Event analysis was made only from the time of lights off to the time of lights on, hence not during the whole time in bed (TIB). If not otherwise specified, the latencies were calculated from the time of lights off. When used, the term "epoch" refers to a 20-second single page of recording. Using criteria from the literature (7,11,19,20), we defined bruxism as a sudden activity of the masseter muscles as observed on the paper chart, often as bursts, alone or together with activity in the temporal and frontal muscles. An epoch was marked as containing masseter activity if more than 0.5 second of such activity was observed

within the epoch. A bruxing "episode" was defined as any abrupt increase of the EMG signal (masseter) reaching an amplitude of either at least twice the amplitude of the signal preceding the episode or more than 25% of the amplitude of the maximum clenching strength as observed during calibration. When two such bursts were less than 5 seconds apart, they were considered as belonging to the same episode. We redefined the term of tonic activity as a slow progressive increase in EMG activity reaching a level equal to or above 25% of the maximum contraction level, or a continuous contraction with constant amplitude and a duration of more than 5 seconds. Phasic activity was defined as repetitive brief contractions lasting less than 1 second, with at least three bursts of EMG activity less than 1 second apart. Activity of the frontal muscles was considered when muscle activity lasting at least 0.5 second was recorded by the EOG or EEG electrodes simultaneously with the occurrence of a bruxing episode. Intraobserver reliability was very good (>95%, as controlled by randomly reanalyzing some of the records).

We calculated the following: 1) total number of episodes with increased orofacial muscle activity; indices were defined as the total number of episodes per hour of sleep; duration was measured with a ruler on the paper chart; 2) sleep structure (global description of sleep) and architecture (sleep stages), with the duration of each sleep stage expressed in both minutes and percent of total TIB; sleep period time [time of sleep onset to end of final sleep (SPT)]; total sleep time [time of sleep onset to end of final sleep minus time awake (TST)]; and the sleep efficiency (%) [(TST/SPT) × 100]; 3) distribution and frequency of orofacial bursts over sleep stages; 4) total number of body movements and duration in different sleep stages (computer and ruler); and 5) number and distribution of any observed events other than orofacial (apnea, PLM, etc.).

Alpha activity

Because the analysis of alpha activity was very time consuming, it was limited to 16 randomly selected subjects. For every burst of masseter activity lasting at least 0.5 seconds, EEG was visually scrutinized for alpha waves in the 10-second period preceding, and the two 10-second periods following the end of the episode, together with the 10-second period that included the bruxing episode (if the episode was longer, only the first 10 seconds were included). The period was identified as containing alpha activity if continuous alpha activity was present for at least 1 second. If, in the epoch preceding the bruxing episode, alpha activity was found to have started before the beginning of the epoch, the total duration of the alpha episode was calculated; thus, some alpha episodes could have a duration longer than 10 seconds. We did not separate in this study "no pain" from "pain" bruxers.

Heartbeat (R–R) interval analysis

This analysis was also limited to the same 16 subjects who were analyzed for alpha activity. The R–R intervals were computed from the paper chart using a digitizer. All bruxing episodes exceeding 0.5 second were selected unless the ECG was unreadable. The R–R intervals were calculated in four consecutive 10-second periods, starting at the epoch preceding the bruxism episode, and the mean value for each of these four periods was computed for each bruxing episode. Hence, the R–R interval was measured 10 seconds before the episode and during the occurrence of bruxism, as well as 10 and 20 seconds immediately after the end of the bruxing episode. Heart rhythm was also observed visually in these four episodes to trace any short-duration frequency change, mainly bradycardia, which was then marked as a special event.

Event analysis

Respiratory disorders such as apnea (complete cessation of air flow) and hypopnea [50% amplitude reduction of the air flow (thermistor) and respiratory movements (sensor pad/abdominal gauge) lasting at least 10 seconds] and PLMs (repetitive episodes of muscle contraction (0.5–5 seconds) separated by 20–40-second intervals) were recognized visually and counted. Periodic events, recognized mainly as the sudden amplitude change of a signal (more than 50% amplitude increase from baseline with at least three repetitive events with constant intervals), were counted. Episodic sounds detected by the microphones and printed on the polygraph were also counted. Indices (number of events per sleep hour) and distribution in different sleep stages were calculated.

Statistics

Data are presented as mean and standard deviation (SD). Student's *t* test was used, modified with Bonferroni's correction for multiple comparisons. Statistical analysis of the clinical symptoms (chi-square test for frequent clenchers, no clenchers) and correlations (Kendall's rank correlation coefficients) were previously reported (21,22).

RESULTS

Clinical symptoms and sleepiness

Results about personality traits are presented in detail in other articles (21,22); they were characterized

TABLE 1. *Sleep structure (24 subjects)*

	Mean	SD
Time of going to bed	10:58 p.m.	12:37 a.m.
Time of lights off	11:09 p.m.	12:37 a.m.
Interval "going to bed–lights off" (minutes)	11	23
Time of awakening	6:30 a.m.	12:45 a.m.
Time of leaving the bed	6:32 a.m.	12:45 a.m.
Interval "waking up–leaving the bed" (minutes)	2	4
% Sleep efficiency[a]	93	6
Time in bed (minutes)	440	47
SPT (minutes)	422	53
TST (minutes)	392	58
Awakenings	13	10
Wake time after sleep onset (minutes)	30	28
Stage shifts after sleep onset	70	23

SPT, sleep period time (time of sleep onset to end of final sleep); TST, total sleep time (time of sleep onset to end of final sleep minus time awake); SD, standard deviation.
[a] TST/SPT × 100.

by anxiety ($p < 0.05$), higher vulnerability for psychosomatic disorders and high muscular tension ($p < 0.01$), and decreased desire to socialize ($p < 0.05$). The subjects often complained of headaches (48%), pain in the neck, back, shoulders, or chest (69%), and stiffness and pain in the jaw in the morning (44%). Tenderness at palpation of the temporomandibular joint (TMJ) was found in 66%. There was also a significant but low correlation between frequent teeth clenching and these symptoms ($r = 0.53$, $p < 0.05$). According to the SDQ, headaches in the morning were reported by 65% of the subjects.

According to the SDQ, 80% of the subjects were often very sleepy during the daytime, and 68% sometimes struggled to stay awake; 52% had trouble doing their jobs because of sleepiness or fatigue, and 25% avoided driving because of sleepiness. Twenty-four percent (six subjects) felt the need to take short naps during an average work day. Sixty-six percent experienced daytime sleepiness during childhood, and 36% had bad grades at school because of sleepiness.

Thirty-eight percent of the subjects reported palpitations at night, four subjects (17%) very often and one subject always. Seventy-seven percent complained of sweating at night, 11 subjects (46%) rather often and 1 subject always. Muscle tension disturbing sleep was reported by 65% and abdominal pain by 77%. Libido was decreased in 50% (women and men), and 31% admitted having sexual problems, but only two men had difficulty maintaining erection. However, all men reported waking up during the night or in the morning with erection. Nineteen percent of the subjects reported being aware that their blood pressure was higher than normal.

Sleep quality and architecture

The sleep diary confirmed that the group had homogeneous sleep habits, the subjects going to bed and waking up around the same time. The majority of the subjects reported that their night sleep at the laboratory had not differed markedly from their normal sleep at home. Tables 1 and 2 present the sleep structure with a normal distribution of the different sleep stages (23).

Bruxing activity and sleep stage distribution

Bruxism, as defined in this study, was identified in all subjects. Table 3 presents the occurrence of the oro-

TABLE 2. *Sleep architecture, mean and standard deviation (24 subjects)*

	Latency to first occurrence (minutes)	Duration (minutes)	% TIB	% SPT	% TST
Total time awake (from time going to bed)	—	54(42)	13(10)	8(10)	—
Movement time[a]	50(73)	6(6)	1(2)	1(2)	2(2)
Stage 1 sleep	18(18)	41(60)	10(15)	10(16)	12(18)
Stage 2 sleep	33(44)	159(53)	36(11)	38(11)	42(12)
Stage 3 sleep	64(67)	42(21)	9(4)	10(5)	11(5)
Stage 4 sleep	78(74)	53(26)	12(6)	13(6)	14(6)
SWS (stages 3 and 4)	—	95(41)	21(9)	23(9)	25(9)
REM sleep	158(85)	81(42)	18(9)	19(9)	21(10)

TIB, time in bed; SPT, sleep period time; TST, total sleep time; SWS, slow-wave sleep; REM, rapid eye movement.
[a] Epochs with movements lasting at least 15 seconds, obscuring the recording.

TABLE 3. *Occurrence of orofacial events and sleep efficiency in 24 subjects*

Subject	% Sleep efficiency	Masseter number	Masseter latency (minutes)	Index	Tonic number	Frontal muscles number	Sound number
1	93	144	13	20	20	60	—
2	92	120	4	14	13	83	—
3	93	104	45	19	36	4	—
4	95	180	19	28	9	114	63
5	86	153	14	24	1	125	89
6	97	81	44	12	15	36	85
7	96	124	27	24	1	48	1
8	97	162	14	26	—	—	—
9	99	188	5	30	2	86	1
10	80	216	10	31	256	9	573
11	93	103	18	19	25	79	89
12	94	135	31	24	—	—	129
13	84	302	18	36	6	—	8
14	86	167	24	26	9	88	155
15	88	203	15	33	—	—	165
16	95	124	1	18	—	—	—
17	95	181	23	29	22	106	64
18	100	184	22	23	8	104	64
19	99	297	3	39	6	82	140
20	77	53	23	8	—	—	22
21	98	250	17	31	17	124	177
22	97	69	10	9	—	—	—
23	97	383	17	52	5	56	—
24	99	82	22	10	—	—	—
Mean	93	167	18	24	19	50	76
SD	6	80	11	10	51	47	122

SD, standard deviation.

"Masseter number" represents the number of epochs with activity of the masseter muscles, most often of phasic type. "Tonic number" is the number of epochs with tonic activity of the masseter groups. "Index" is the occurrence per sleep hour ("masseter number" divided by the sleep period time). "Frontal muscles number" is the number of epochs in which activity of the frontal muscles was recognized and occurring simultaneously with the masseter activity. "Sound number" represents the number of epochs with recorded sounds (unspecified).

facial events during the night among the 24 subjects. The number of epochs with masseter activity varied considerably, with a mean index of 24. Sleep efficiency did not correlate with the index of masseter activity, i.e. high efficiency and low index (subject 18 had high sleep efficiency and index, whereas subject 20 had low efficiency and index). During the night a progressive increase of the tonic activity of the masseter muscles could be observed in many subjects, not necessarily accompanied by an increase of the phasic activity. Although phasic activity usually dominated, one patient (subject 10) had more tonic than phasic activity. Sounds were not always correlated to the orofacial activities, and, as in subject 10, their frequency of occurrence even exceeded these activities. We did not attempt to differentiate between snoring or bruxing sounds. Table 4 presents the distribution and duration of the bruxing episodes among the different sleep stages. Bruxism occurred in all stages, with the majority of the episodes developed during stage 2 and REM. The duration of the bruxing episodes increased with the deepening of sleep ($p < 0.01$ in stage 2 compared with stage 1), and this was more pronounced in slow-wave sleep (SWS) ($p < 0.01$ in stage 3 compared with stage 2, and $p < 0.01$ in stage 4 compared with stage 3). During REM, the bruxings became short ($p < 0.01$ in comparison with stage 2) with a duration not significantly different from the one observed in the awakened state.

Shift of sleep stages

The number of shifts of sleep stages after sleep onset was 70 ± 24, with an average of more than 10 per hour. About one-third of all shifts (23 ± 14) occurred within 1 minute after a bruxing episode lasting at least 1 second. Conversely, 15% of all bruxing episodes, including some of very short duration (<5 seconds) were directly associated with a shift of the current sleep stage. A shift toward lighter sleep, meaning an arousal, was observed after 74% of the bruxings occurring during sleep (stages 1–4), and after 79% if REM was included (in which case any shift other than toward SWS was considered as an arousal). Of all shifts of sleep stage developed after bruxism, 21% occurred when the bruxing episodes were in the awakened state, 13% during stage 1, 17% during stage 2, 5% during stage 3, 8% during stage 4 and REM, and 27% when bruxism was observed in the readable part of the movement time (MT) epochs. The direction of the shifts of the sleep stages following bruxings was toward awakening in 28.4% of the cases (15% if the shifts following bruxings occurring during MT were not considered); 21.2% toward stage 1, 25.9% toward stage 2, 2 and 1% toward stages 3 and 4, respectively, 3.2% toward REM, and 18.2% toward MT. A shift of

TABLE 4. *Bruxing episodes (mean and SD) in different stages*

	During SPT	Awake	Stage 1 sleep	Stage 2 sleep	Stage 3 sleep	Stage 4 sleep	REM sleep
Mean number of episodes	79(45)	8(12)	9(7)	34(35)	4(4)	4(4)	21(22)
% occurrence in the different stages		10.2	11	43	4.5	5	26.4
Duration (seconds)		7.0(4.3)	6.3(3.2)	8(4)	11.1(7.5)	20.2(12.1)	6.5(2.9)

SPT, sleep period time; REM, rapid eye movement; SD, standard deviation.

TABLE 5. *Percentage of occurrence of alpha activity (at least 1 second) prior, during, and after a bruxing episode (1234 episodes in 16 subjects)*

Sleep stage	10 seconds prior to episode	During episode	10 seconds after end of episode	20 seconds after end of episode
Awake	100	93	89	84
Stage 1	61	42	27	21
Stage 2	48	23	7	4
Stage 3	26	8	9	2
Stage 4	27	13	8	8
REM	22	9	5	2
Stages 1 + 2	51	26	11	8
Stages 3 + 4	27	10	9	5

The "sleep stage" is the actual stage just prior to the occurrence of the bruxing episode. Duration of bruxing episode >0.5 second. No distinction between "tonic" and "phasic".

sleep stage after bruxings occurring in stage 1 was most often toward stage 2 (54%) and awakening (29%); when they occurred during stage 2, they were most often toward awakening (40%) and MT (32%); the few times bruxings developed during SWS, the shifts were never toward REM but toward stage 2 (about 36%), MT (about 36%), and awakenings (about 17%); when bruxings occurred during REM sleep, REM was often interrupted, the shifts were toward MT (32%), stage 2 (30%), and awakening in 28% of the cases.

Alpha activity

We looked for the occurrence of alpha activity of short duration in the EEG preceding, accompanying, or following 1,234 bruxing episodes. Alpha activity occurring in the 10 seconds before the bruxing episodes was especially prevalent during light sleep (stages 1 and 2, 61 and 48%, respectively) but even during SWS and REM (Table 5). Alpha activity of short duration could be observed within the epochs containing the bruxing episodes in 26% of bruxings occurring during light sleep, in 10% during SWS, and in 9% during REM. Twenty seconds after the end of the episodes, alpha was seen in less than 8% of the bruxings. The duration of the alpha activity occurring prior to the bruxing episode was variable, sometimes extending up to 12 seconds (thus starting before the 10-second measuring interval preceding the bruxing episode), but the mean duration was 2.8 seconds (Table 6). The longest duration was when bruxing occurred during stage 1. It decreased significantly in stage 2 ($p < 0.01$), remaining constant during SWS. When bruxings occurred during REM sleep, the duration was significantly shorter only in comparison with stage 1 ($P < 0.01$).

TABLE 6. *Mean duration (and SD) of alpha activity occurring during the 10 seconds preceding a bruxing episode (16 subjects)*

Sleep stage	Seconds
Stage 1	3.7(2.8)
Stage 2	2.7(1.7)
Stage 3	2.2(1.1)
Stage 4	2.9(2.2)
REM	2.5(1.5)
Mean value	2.8
SD	2.0

REM, rapid eye movement; SD, standard deviation.
Mean value (and SD) represent all the values measured independently of any sleep stage.

Other EEG features

The presence of alpha activity in the EEG occurring simultaneously with the slow delta pattern in deep sleep (alpha-delta activity) was occasionally encountered in many subjects, quite often in more than 15% of the patients. None of the subjects presented epileptic activity in the EEG.

R–R interval

R–R intervals are given in Table 7. During the bruxing episodes, tachycardia was present with a decrease of the R–R interval of almost 15% compared with the period immediately preceding the episode ($p < 0.01$), the R–R interval returning to its initial value 10–20 seconds after the end of the bruxing episode. The heart rate changes were similar in all sleep stages. Bradycardia secondary to the tachycardia was not observed. To confirm that tachycardia did not occur just prior to the development of the bruxing, we compared, within the 10-second period preceding the bruxing episode,

TABLE 7. *R–R intervals (seconds) prior, during, and after a bruxing episode (16 subjects)*

Sleep stage	Number of episodes analyzed	R–R 10 seconds prior to episode	During episode	10 seconds after end of episode	20 seconds after end of episode
Awake	66	0.90	0.79	0.83	0.90
Movement time	33	0.94	0.78	0.70	0.90
Stage 1	60	0.91	0.80	0.86	0.91
Stage 2	169	0.97	0.84	0.91	0.97
Stage 3	48	1.02	0.87	0.91	0.99
Stage 4	43	0.95	0.83	0.86	0.93
REM	135	0.97	0.84	0.93	0.99
Mean		0.95	0.82[a]	0.86[a]	0.94 (ns)
SD		0.04	0.03	0.08	0.04

R–R, rate and rhythm; REM, rapid eye movement; SD, standard deviation; ns, not significant.
[a] $p < 0.01$.
The "sleep stage" is the actual stage just prior to the occurrence of the bruxing episode.

the first two R–R intervals with the last two. The mean values of each pair were similar, as was the grand mean (mean of all first two and mean of all last two R–R intervals was 0.95 second).

Respiratory events and periodic movements

One subject reported in the SDQ that he snored every night (subject 15 in Table 3), and eight (36%) reported snoring intermittently, five of them loudly enough to disturb the bedroom partner (subjects 5, 10, 14, 16, and 20). Four patients had been observed by their roommate to have sleep apnea, two of them very seldom, one occasionally (subject 15), and one often (subject 5). Snoring or breathing problems worsened markedly after alcohol consumption in five patients. Sleeping on the back intensified snoring, intermittently for six subjects and constantly for two. Worsening in snoring and breathing occurred following allergic reactions or respiratory tract infections in 11 subjects. Five experienced nose blocking or "allergic-like" reactions in the nose while lying and trying to sleep, and nine reported frequent tonsillitis. The recordings showed respiratory irregularities (apnea or hypopnea exceeding 10 seconds) in only four patients (subjects 10 and 12–14 in Table 3). The apnea index and the oxygen desaturation index (episodes per sleep hour of decreased oxygen saturation >4% of the baseline level) were <5. No repeated or long lasting (>5 seconds) activation of the intercostal muscles (EMG), reflecting increased respiratory efforts, were seen.

Six patients reported in the SDQ that sleep onset was sometimes associated with discomfort in the legs or an urge to move the legs. For two patients such symptoms often disturbed their sleep. Periodic limb movements (PLM) were observed in the records in only one patient, who also reported discomfort in the SDQ and presented a few short apneas.

DISCUSSION

In this study we have reported the development of short-lasting alpha activity prior to the bruxing episode and tachycardia at the onset of the burst activity. Women dominated our group; this may be due to the selection procedure (advertisement), women being more interested in participating. There is no reason to believe that the results presented are influenced by this sex imbalance. The group was homogeneous as far as sleep habits were concerned. Sleep environment, according to the patients' reports, was satisfactory. The normal sleep structure and the relatively low amount of sleep stage shifts suggest that sleeping conditions in the laboratory were indeed good, although the tendency for prolonged sleep latency and delayed REM could be due to the unfamiliar sleeping surroundings.

A majority of the patients were anxious, reported muscular tension, and complained of both orofacial and diffuse body pain (21,22). Psychosocial factors such as anxiety and stress influence central nervous system regulatory systems and pain perception (24) and may induce muscle pain (25). Muscles exposed to unusual exercise develop delayed-onset pain, and some muscle fibers may show damage similar to that found in primary myopathies (26). Similarly, recurrent muscle contractions, such as in bruxism, can also result in muscular fatigue, pain, and stiffness. Secondary localized inflammations cannot be ruled out; they were observed in the masseter muscles of rats following mechanical lengthening of the contracted muscles (27). On the other hand, recent data suggest that pain exerts an inhibitory effect on agonist muscles as a protective mechanism (28), and sleep bruxism was implied to be reduced in association with pain (29).

The number of orofacial episodes recorded was higher than that reported in five subjects by Velly Miguel et al. (7), whereas the mean numbers of bruxing bursts and indices were within their ranges (22–71 and 12–86) but higher than the mean of up to 17 bursts per night reported by Clarke et al. (20). The mean duration during light sleep agrees with those observed by these authors. However, we found longer duration in deeper sleep; during REM, the bursts became short, similar to those observed during awakening.

Sjöholm (11) reported that the majority of bruxing episodes occurred during stages 1 and 2. In our study, bursts were found mainly in stage 2 and REM sleep [which agrees with the literature review made by Wruble et al. (10)]. Satoh and Harada (9) never observed bruxings during the REM bursts of REM sleep, and neither could we find them during these bursts. The interruption of REM following a bruxing episode that we observed parallels the suppression of the bursts of REM reported by Satoh and Harada (9). This may be due to the shift of sleep stage that often occurs after such a burst and differences in "scoring method".

Many bruxing episodes lead to a shift in sleep stage, usually toward awakening or lighter sleep, suggesting that bruxism may be part of an arousal phenomenon. This hypothesis has been proposed previously (5), but to our knowledge the present study is the first to support it. Most shifts developed when bruxings occurred during light sleep and REM, and very seldom during SWS; the "shift-triggering" factor may be either too weak or partly inhibited in SWS.

We found that prior to the bursts of the bruxing episodes, especially when occurring during light sleep, the subjects developed short-lasting alpha activity. These microawakenings were also observed, although

seldom, in deeper sleep. The periods of alpha activity preceding bruxings were too short to be scored as awakenings according to the sleep-staging criteria of Rechtschaffen and Kales (18) based on 20–30-second epochs. Such scoring resulted in a normal sleep structure, whereas in reality, sleep was fragmented by repeated short arousals/awakenings, which may cause the increased daytime sleepiness observed by us and others (30).

Alpha intrusion during SWS (alpha-delta) was observed in the EEGs of some patients. This is usually seen in disorders that disrupt nocturnal sleep and, although not specific to pain (25), is also a characteristic feature of the fibrositis syndrome (31), which presents many similarities with bruxism [chronic musculoskeletal pain, muscle tender points, morning stiffness, fatigue, and poor sleep, without specific laboratory findings (32)]. The presence of "alpha-delta sleep" (33), a "biological correlate" of nonrestorative sleep (34), further suggests a relationship between these two disorders, which may coincide. It is not actually possible to know whether the development of alpha activity observed in both disorders anticipates muscle pain or if the alpha intrusion follows the possible development of pain; it could also reflect poor sleep.

Although some authors have reported that bruxism is often associated with sleep apnea (6,35), we did not find marked breathing disorders or pathological levels of oxygen desaturations in our patients. This may be due to the female dominance in our group, with apnea predominant in men (36). According to the self-reports, 36% of our patients used to snore, but only four patients were observed by their roommates to have sleep apnea.

Between 75 and 85% of bruxing episodes have been reported to occur in association with EMG bursts in the anterior tibialis muscle (19). We did not find a simultaneous occurrence of these phenomena, having observed PLM in only one patient, although six complained about intermittent restless legs syndrome (RLS). Lavigne and Montplaisir (37), in a study based on a nationwide survey of Canada, reported RLS in about 10% of teeth grinders, whereas 15 to 17% who complained about RLS symptoms also had teeth grinding. They suggested that restless legs and bruxism are two concomitant, but not strongly associated, sleep movement disorders. We also think that restless legs and PLM are separate entities, sometimes coincidental (37) to bruxism, developed secondary to arousal known to facilitate motor activation (38,39) even expressed as apnea (40), PLM (5,11), and bruxism (9,10,35).

Reding et al. (35) reported that acceleration of the heart rhythm precedes the onset of a bruxing episode. We found increased heart rate, not prior to, but at the onset of the burst of bruxism.

The novelty of our study is that tachycardia develops together with bruxing episodes and not prior to them, and arousals/awakenings occur in advance of the development of bruxing episodes and tachycardia. Bruxings are thus not the primary disorder. We suggest that, following endogenous/exogenous stimuli, arousal develops easily in bruxers with fragile sleep; it is followed by a motor activation reflected by the development of a burst of bruxism with a secondary increase of heart activity, as in any situation when motor activity suddenly increases.

REFERENCES

1. Lowenthal U. Stress, anxiety and the dental patient: the missing specification. *Int Dent J* 1981;31:193–7.
2. Morse DR. Stress and bruxism: a critical review and report of cases. *J Hum Stress* 1982;8:43–52.
3. Faulkner KDB. Bruxism: a review of the literature. Part I. *Aust Dent J* 1990;35:266–76.
4. Pierce CJ, Chrisman K, Benett E, Close JM. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. *J Orofacial Pain* 1995;9:51–6.
5. Lavigne GJ, Montplaisir JV. Bruxism: epidemiology, diagnosis, pathophysiology and pharmacology. In: Fricton JR, Dubner R, eds. *Orofacial pain and temporomandibular disorders.* New York: Raven Press, 1995:387–404.
6. Okeson JP, Phillips BA, Berry DTR, Cook YR, Cabelka JF. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. *J Craniomandib Disord Facial Oral Pain* 1991;5:258–64.
7. Velly Miguel AM, Montplaisir J, Rompré PH, Lund JP, Lavigne GJ. Bruxism and other orofacial movements during sleep. *J Craniomandib Disord Facial Oral Pain* 1992;6:71–81.
8. Hartman E. Bruxism. In: Kryger MH, Roth T, Dement WC, eds. *Principles and practice of sleep medicine,* 2nd ed. Philadelphia: W.B. Saunders, 1994:598–601.
9. Satoh T, Harada Y. Electrophysiological study on tooth-grinding during sleep. *Electroencephalogr Clin Neurophysiol* 1973;35: 267–75.
10. Wruble MK, Lumley MA, McGlynn FD. Sleep-related bruxism and sleep variables: a critical review. *J Craniomandib Disord Facial Oral Pain* 1989;3:152–8.
11. Sjöholm T. Sleep bruxism. Thesis. *Pathophysiology, Diagnosis, and Treatment.* Turku, Annales Universitatis Turkuensis, Finland: Typopress OY, 1995.
12. Helkimo M. Studies on function and dysfunction of the masticatory system. II. Index for anamnestic and clinical dysfunction and occlusal state. *Swed Dent J* 1974;67:101–21.
13. Krogh-Poulsen W. Klinisk undersögelse. In: Krogh-Poulsen W, Carlsen O, eds. *Bidfunktion/bettfysiologi.* Copenhagen: Munksgaard, 1972:423–63.
14. Carlsson GE, Helkimo M. Funktionell undersökning av tuggapparaten. In: Holst JJ et al., eds. *Nordisk klinisk odontologi 8-I.* Copenhagen: A/S Forlaget for faglitteratur, 1972:5–6.
15. Schalling D, Åsberg M, Edman G, Oreland L. Markers of vulnerability to psychopathology: remperament traits associated with platelet MAO activity. *Acta Psychiatr Scand* 1987;76:172–82.
16. Douglass AB, Bornstein R, Nino-Murcia G, et al. The sleep disorders questionnaire I: creation and multivariate structure of SDQ. *Sleep* 1994;17:160–7.
17. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540–5.
18. Rechtschaffen A, Kales A, eds. A manual of standarized ter-

minology, techniques, and scoring system for sleep stages in human subjects. Los Angeles: U.C.L.A. Brain Information Service/Brain Research Institute, 1968.
19. Ware JC, Rugh JD. Destructive bruxism: sleep stage relationship. *Sleep* 1988;11:172–81.
20. Clarke NG, Townsend GC, Carey SE. Bruxing patterns in man during sleep. *J Oral Rehabil* 1984;11:123–7.
21. Kampe T, Edman G, Tagdae T, Bader G, Karlsson S. Personality traits in a group of subjects with longstanding bruxing behavior. In press: *J Oral Rehabil* 1996.
22. Kampe T, Edman G, Tagdae T, Bader G, Karlsson S. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. In press: *J Oral Rehabil* 1996.
23. Cooper R. Normal sleep. In: Cooper R, ed. *Sleep*. London: Chapman and Hall Medical, 1994:3–46.
24. Maixner W, Sigurdsson A, Fillingim RB, Lundeen T, Booker DK. Regulation of acute and chronic orofacial pain. In: Fricton JR, Dubner R, eds. Orofacial pain and temporomandibular disorders. New York: Raven Press, 1995:85–102.
25. Lavigne GJ, Velly-Miguel AM, Montplaisir J. Muscle pain, dyskinesia and sleep. *Can J Physiol Pharmacol* 1991;69:678–82.
26. Newham DJ. Fatigue, damage and muscle use—unresolved issues. Neural and neuromuscular aspects of muscle fatigue, Florida 1994. *Muscle Nerve* 1996;Suppl 4:S30 (abstract).
27. Hutchins MO, Skjonsby HS. Microtrauma to rat superficial masseter muscles following lengthening contractions. *J Dent Res* 1990;69:1580–5.
28. Lund J. Pain and the control of muscles. In: Fricton JR, Dubner R, eds. Orofacial pain and temporomandibular disorders. New York: Raven Press, 1995:103–15.
29. Lavigne GJ, Lobbezoo F, Rompre PH, Lund JP, Guitard F, Montplaisir JY. Modulation of sleep bruxism by jaw muscle pain: a pilot study. *Sleep Res* 1995;241,24A:398.
30. Stepanski E, Lamphere J, Badia P, et al. Sleep fragmentation and daytime sleepiness. *Sleep* 1984;7:18–26.
31. Thorpy MJ, Yager J. The encyclopedia of sleep and sleep disorders. New York: Facts on File, 1991:298.
32. Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology. 1990 criteria for the classification of fibromyalgia: report of the multicenter criteria committee. *Arthritis Rheum* 1990;33:160–72.
33. Hauri P, Hawkins DR. Alpha-delta sleep. *Electroencephalogr Clin Neurophysiol* 1973;34:233–7.
34. Moldofsky H. The contribution of sleep-wake physiology to fibromyalgia. *Adv Pain Res Ther* 1990;17:227–40.
35. Reding GR, Zepelin H, Robinson JE, Zimmerman SO, Smith VH. Nocturnal teeth-grinding: all night psychophysiologic studies. *J Dent Res* 1968;47:786–97.
36. Guilleminault C, Quera-Salva MA, Partinen M, Jamieson A. Women and the obstructive sleep apnea. *Chest* 1988;93:104–9.
37. Lavigne GJ, Montplaisir JV. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. *Sleep* 1994;17:739–43.
38. Coleman RM, Miles CE, Guilleminault C, Zarcone VP, van den Hoed J, Dement W. Sleep-wake disorders in the elderly: a polysomnographic analysis. *J Am Geriatr Soc* 1981;29:289–96.
39. Coccagna G. Restless legs syndrome/periodic leg movements in sleep. In: Thorpy MJ, ed. Handbook of sleep disorders. New York: Marcel Dekker, 1990:457–78.
40. Krieger J. Obstructive sleep apnea: clinical manifestations and pathophysiology. In: Thorpy MJ, ed. Handbook of sleep disorders. New York: Marcel Dekker, 1990:259–84.