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Sleep bruxism: contemporary insights in diagnosis, etiology and management

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Chapter 4

Associations between sleep bruxism, periodic limb movements, and EEG arousals in sleep bruxers and healthy controls.

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Submitted for publication.

Summary

Background and purpose: Sleep bruxism (SB) and periodic limb movements during sleep (PLMS) may have a common underlying neurophysiologic mechanism, especially in relation to the occurrence of sleep-related EEG arousals. To test this hypothesis, three research questions were assessed: First, it was assessed whether PLMS events occur more frequently in SB patients than in healthy controls. Second, the question was put forward whether the combined presence of SB and PLMS events is more common than that of isolated SB or PLMS events in a group of SB patients. Third, as to further unravel the possible role of EEG arousals in the underlying neurophysiologic mechanism of SB and PLMS, it was assessed in a group of SB patients whether combined SB/PLMS events with associated EEG arousals are more common than those without associated EEG arousals. Positive answers to these questions could suggest a common neurophysiological basis for both movement disorders.

Patients and methods: Seventeen SB patients and eleven healthy controls were polysomnographically studied. SB, PLMS, and EEG arousals were scored. An association was noted when the occurrence was within a 3-sec association zone.

Results: The PLMS index was higher in SB patients than in healthy controls ($P = 0.000$). Within the group of SB patients, the combined SB/PLMS index was higher than the isolated SB index ($P = 0.000$) and the isolated PLMS index ($P = 0.010$). Similarly, the combined SB/PLMS index with EEG-arousal was higher than the combined SB/PLMS index without EEG-arousal in SB patients ($P = 0.001$).

Discussion: The results of this study indicate that SB, PLMS, and EEG arousals are highly associated and probably have a common underlying neurophysiological mechanism.

Keywords: sleep bruxism, PLMS, arousal, EMG, EEG, polysomnography

1. Introduction

The International Classification of Sleep Disorders (ICSD-2) distinguishes sleep-related movement disorders into simple, repetitive rhythmic movement disorders and parasomnias [1]. Examples of the first category are Sleep Bruxism (SB) and Periodic Limb Movements during Sleep (PLMS). SB is characterized by patient reports of tooth grinding sounds or tooth clenching during sleep, and one or more of the following conditions: abnormal wear of the teeth; jaw muscle discomfort, fatigue, or pain and/or jaw lock on awakening; and masseter muscle hypertrophy on forceful clenching [1]. SB has a prevalence of about 8% in the general adult population [2,3]. The disorder is periodic and has a cyclic pattern [4-6]. PLMS are periodic, repetitive, stereotyped limb movements occurring during sleep [7,8]. It is a common disorder with an estimated prevalence of 4-11% in the general adult population [7].

Although the etiology of SB is still not fully understood, it has been argued that SB is mainly caused by factors like psychological stress and /or disturbances in the central dopaminergic system and not, as frequently suggested in the past, by peripheral factors (e.g., deviations in dental occlusion and /or in the orofacial anatomy) [6, 9–10]. The etiology of PLMS is not yet well known either. Theories concerning the etiology strikingly resemble the ones for SB. Disturbances in the dopaminergic system [11], genetical factors [12], and (over-) use of drugs and stimulantia (e.g., caffeine, alcohol, antihistamines, selective serotonin re-uptake inhibitor antidepressants like paroxetine) [13] can cause both SB and PLMS.

According to several authors, an association exists between SB and PLMS [2,14,15]. Possibly, these motor events are different expressions of the same sleep disorder. The fact that the pharmacological management strategies of these movement disorders show similarities (viz., opioids, levodopa, D2- and D3-receptor agonists, and clonazepam) [13,16–20] corroborates with this possibility. In addition, both SB and PLMS have been associated with EEG arousals [4,21–26], which underlines the possibility that these conditions share a common background. To further assess this issue, three research questions were formulated. First, it was assessed whether PLMS events occur more frequently in SB patients than in healthy controls. Second, the question was put forward whether the combined presence of

SB and PLMS events is more common than that of isolated SB or PLMS events in a group of SB patients. Third, as to further unravel the possible role of EEG arousals in the underlying neurophysiologic mechanism of SB and PLMS, it was assessed in a group of SB patients whether combined SB/PLMS events with associated EEG arousals are more common than those without associated EEG arousals. Positive answers to these questions provide further evidence for a common neurophysiological background for SB and PLMS.

2. Patients and methods

2.1. Participants

Seventeen SB patients (12 women and 5 men; mean \pm SD age = 32.1 ± 6.5 years; range = 22 – 44 years) and 11 healthy subjects (7 women and 4 men; mean \pm SD age = 34.5 ± 12.8 years; range = 22 – 47 years), underwent one full-night polysomnographic recording (PSG) in the Amsterdam Center for Sleep and Wake Disorders of the Slotervaart Medical Center, Amsterdam, The Netherlands.

SB patients were recruited from among those who participated in former studies from our group [27,28]; by means of an announcement in a local newspaper; and from among the patients attending the clinic of the department of Oral Kinesiology of the Academic Centre for Dentistry Amsterdam (ACTA), Amsterdam, The Netherlands. The healthy subjects were recruited from among persons attending the department of Clinical Neurophysiology of the Slotervaart Medical Center for a routine medical examination. The inclusion criteria for the group of SB patients were: being at least 18 years of age; having a natural dentition with signs of tooth clenching (i.e., hyperkeratotic ridges in the cheeks, tongue scalloping, and/or incisal impressions in the lips; [29]; having a history of tooth grinding sounds for at least three nights per week during the last 6 months (adapted from Kato *et al.* 2001 [30]); and having occlusal tooth wear to at least the degree of exposed dentine (i.e., grade two; [31]). The healthy controls were included when they were at least 18 years of age; had a natural dentition; had no signs of clenching, a recent history of grinding sounds, or an occlusal tooth wear grade of two or more; and had no history of sleep disorders. Participants, SB patients and controls alike, were excluded when they fulfilled one or more of the following criteria: suffering from epilepsy or any sleep disorder other than SB or PLMS (e.g., obstructive sleep apnea); using any medication that has a known influence on sleep structure, SB, or PLMS (e.g., selective serotonin re-uptake inhibitors like paroxetine, or anti-Parkinson medication

like pramipexol; [32]; or being diagnosed with temporomandibular pain [33,34]. The scientific and ethical aspects of the protocol were reviewed and approved by the Medical Ethics Committee of the Slotervaart Medical Center (approval number 9909), and written informed consent was obtained from all participants.

2.2 Procedure

All PSG recordings took place according to the hospital's standard protocol for sleep recordings in a quiet, dark, single room. The recordings were performed with silver chloride surface electrodes. A Biosaca sleep-recording unit (Ortivus, Sweden) was used. The montage setup was as follows: electroencephalography (EEG: C₃A₂, O₂ A₁), electromyography (EMG: right and left masseter muscles, sub mental area, and anterior tibialis muscle), electro-oculography (EOG: right and left), oxygen saturation (SaO₂), heart rate (ECG), body position, and grinding sound (piezo-electric device). The EMG signals of the masseter muscles were recorded with a sampling frequency of 256 Hz per channel. Hardware filters were set at 50 Hz notch, 3 Hz high pass, and 100 Hz low pass. For more details about the procedure, see van der Zaag *et al.* (2005) [27].

2.3. Data Analysis

Sleep was analyzed by an experienced sleep scientist (DJW) in 30-sec epochs, according to Rechtschaffen and Kales (1968) [35]. Total sleep time (hours), sleep stages N1 till N4 (%), REM sleep (%), sleep efficiency (%), and arousal index (AI) were determined as sleep outcome variables.

SB was analyzed after Lavigne *et al.* (1996, 2001b) [36,37], using the number of episodes per hour of sleep for quantification (SB index; epi/hr). SB was scored at a threshold of 10% of the maximum voluntary contraction (MVC) level of the masseter EMG's, using an automatic bruxism analyzing tool incorporated in the REMBRANDT software (Medcare Automation BV, Amsterdam, The Netherlands) and following the procedures described in detail by van der Zaag *et al.* (2008) [28]. All automatically detected SB events were crosschecked visually for the possible unwanted scoring of artifacts. SB was only analyzed during sleeping periods, and the SB indices were averaged over left and right side masseter muscles.

PLMS were analyzed according to the revised criteria of the international classification of sleep disorders [38]. For a PLMS event to be present, there had to be at least 4 consecutive movements, each lasting 0.5-5 seconds, with movement onsets each 5-90 seconds apart. The number of events per hour of sleep was used for quantification (PLMS index; events/hr).

EEG arousals were also scored according to revised ICSD criteria [38], i.e., an abrupt shift in EEG frequency, which may include theta waves, alpha waves and/or frequencies greater than 16Hz, but not spindles for more than 3 seconds. In REM sleep, this increase in EEG frequency must be associated with a sub mental EMG increase. The EEG-arousal index was calculated by expressing the number of EEG arousals per hour of sleep.

An EEG arousal was considered associated with a SB and/or PLMS event if the arousal occurred within 3 seconds before the onset or after the termination of the motor event [38, 39]. In this study, a PLMS event was considered associated with a SB event if the PLMS event occurred within 3 seconds before the onset of the SB event, during the event, or within 3 seconds after the termination of the event.

2.4. Statistical analysis

To test whether or not group differences were present, a Chi-square test was used for gender; independent-samples t tests for equal variances (test statistic = T_E) were used for age, total sleep time, and sleep stages N1, N2, N3, N4, and REM; and independent-samples t tests for unequal variances (test statistic = T_U) were used for sleep efficiency, EEG-arousal index, and the total SB index (the total number of SB events per hour of sleep, i.e., regardless of its possible association with PLMS events).

To assess whether PLMS events occurred more frequently in SB patients than in healthy controls, the independent-samples t test for unequal variances was used to test for differences between both groups in the number of PLMS events per hour of sleep.

A repeated measures analysis of variance (ANOVA), followed by post-hoc contrasts, was used within the group of SB patients to assess possible differences between the number of isolated SB events per hour of sleep (i.e., SB events that are not associated with a PLMS event), the number of isolated PLMS events per hour of sleep (i.e., PLMS events that are not associated with a SB event), and the number of combined SB/PLMS

events per hour of sleep (i.e., SB events and PLMS events that are both present within the above-defined interval; see Data Analysis).

Finally, within the group of SB patients, paired-samples t tests were used to test for differences between the number of combined SB/PLMS events per hour of sleep without associated EEG arousals on the one hand and the number of combined SB/PLMS events with associated EEG-arousals on the other hand.

Statistical analysis was performed using SPSS software, version 18.0, and differences were considered significant when the P value was <0.05 .

3. Results

The distribution of gender did not differ significantly between the groups of healthy controls and SB patients, ($X^2 = 0.15$; $P = 0.701$). Likewise, the groups had the same mean age ($T_E = 0.86$; $P = 0.399$).

All PSG recordings of both the healthy controls and the SB patients were judged to have normal structures by an experienced sleep scientist who is specialized in sleep disorders (DJW). As shown in Table 1, the total sleep time did not differ significantly between both groups of participants and had a minimum value of 6 hours for both healthy controls and SB patients. Sleep stages and sleep efficiency did not differ significantly between both groups of participants either. The arousal index of the SB patients was significantly higher than that of the healthy controls. Finally, the total SB index differed significantly between the healthy controls and the SB patients. In line with the research diagnostic criteria as suggested by Lavigne *et al.* (1996) [36], the SB indices for the healthy controls did not exceed the value of 4 events per hour of sleep (maximum value = 2.70), while those of the SB patients were all above that threshold (minimum value = 5.70).

Table 1. Mean values, standard deviations, and ranges (between parentheses) of total sleep time, sleep stages (N1, N2, N3, N4, REM), sleep efficiency (SE), EEG-arousal index (AI), and total SB index for both healthy controls and SB patients. T_E = test statistic for independent-samples t test with equal variances; T_U = test statistic for independent-samples t test with unequal variances.

Outcome variable	Controls (n = 11)	SB patients (n = 17)	Two-samples t test T (P)
Total sleep time (hours)	7.6 ± 1.2 (6.0 – 10.2)	7.9 ± 0.8 (6.0 – 9.2)	$T_E = 0.77$ (P = 0.446)
N1 (%)	5.12 ± 3.05 (1.3 – 11.5)	4.74 ± 2.71 (0.9 – 9.2)	$T_E = -0.34$ (P = 0.735)
N2 (%)	56.16 ± 9.48 (39.4 – 69.3)	56.39 ± 8.74 (41.2 – 74.7)	$T_E = 0.07$ (P = 0.948)
N3 (%)	8.35 ± 4.59 (1.1 – 15.9)	8.66 ± 4.60 (2.5 – 23.3)	$T_E = 0.18$ (P = 0.446)
N4 (%)	8.15 ± 5.43 (0.0 – 17.4)	8.23 ± 6.33 (0.1 – 19.5)	$T_E = 0.04$ (P = 0.971)
REM (%)	22.29 ± 4.32 (13.8 – 28.8)	21.98 ± 3.55 (15.6 – 27.4)	$T_E = 0.20$ (P = 0.840)
SE (%)	83.32 ± 12.21 (61.1 – 95.5)	89.79 ± 5.41 (79.3 – 96.2)	$T_U = 1.66$ (P = 0.122)
AI (total per hr)	2.94 ± 2.37 (0.5 – 7.2)	35.11 ± 18.82 (17.0 – 93.7)	$T_U = 6.96$ (P = 0.000)
SB (total per hr)	1.05 ± 0.85 (0.20 – 2.70)	11.47 ± 5.99 (5.70 – 29.70)	$T_U = -7.06$ (P = 0.000)

From Fig. 1, it can be derived that the number of PLMS events per hour of sleep was significantly higher in the SB patients than in the healthy controls ($T_U = 6.41$; $P = 0.000$). Within the group of SB patients, the respective indices for isolated SB events,

isolated PLMS events, and combined SB/PLMS events differed significantly from each other ($F = 11.387$; $P = 0.000$) (Fig. 2). Contrasts revealed that the isolated SB index and the isolated PLMS index did significantly differ from each other, albeit marginally ($F = 4.610$; $P = 0.047$). In addition, both isolated indices were significantly smaller than the combined SB/PLMS index ($F = 20.891$; $P = 0.000$ and $F = 6.923$; $P = 0.018$, respectively). Finally, when the combined SB/PLMS index of the SB patients was split into combined SB/PLMS events without associated EEG arousals and combined SB/PLMS events with associated EEG arousals, the latter index was found to be significantly higher ($T = -7.47$; $P = 0.000$) (Fig. 3).

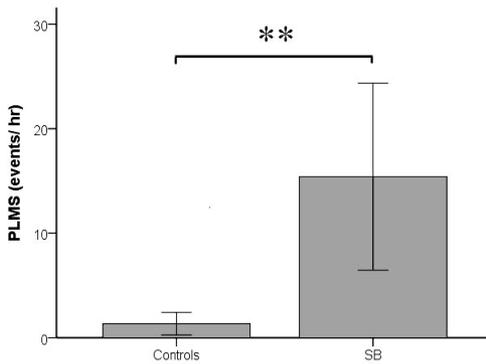


Fig. 1. Mean \pm SD of the number of PLMS events per hour of sleep for the healthy controls and the SB patients. **: $P < 0.01$.

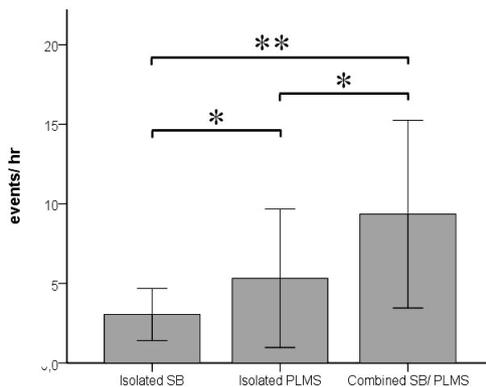


Fig. 2. Mean \pm SD of the number of isolated SB events per hour of sleep, the number of isolated PLMS events per hour of sleep, and the number of combined SB/PLMS events per hour of sleep within the group of SB patients. *: $P < 0.05$; **: $P < 0.01$.

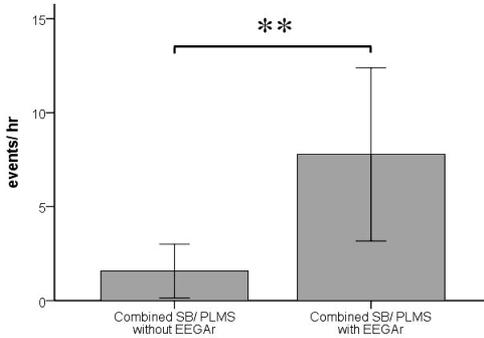


Fig. 3. Mean \pm SD of the number of combined SB/PLMS events without associated EEG arousals per hour of sleep and the number of combined SB/PLMS events with associated EEG-arousals per hour of sleep within the group of SB patients. **: $P < 0.01$.

4. Discussion

In this study, three research questions were assessed: First, it was assessed whether PLMS events occur more frequently in SB patients than in healthy controls. Second, the question was put forward whether the combined presence of SB and PLMS events is more common than that of isolated SB or PLMS events in a group of SB patients. Third, as to further unravel the possible role of EEG arousals in the underlying neurophysiologic mechanism of SB and PLMS, it was assessed in a group of SB patients whether combined SB/PLMS events with associated EEG arousals are more common than those without associated EEG arousals. Positive answers were found, which implies common neurophysiological grounds for both movement disorders.

Polysomnography (PSG) is recommended as the gold standard diagnostic tool for SB as well as for PLMS [15,36,40,41]. In the present study, single-night PSG recordings without audio/video control were used. Not using audio/video control to discriminate SB from other oromotor activities may be considered a disadvantage of the present study design. However, it is as yet unknown if audio/video-controlled PSG recordings actually differ from those not using such control in terms of the number of SB events detected. The influence of a so-called first night effect (FNE) was not taken into consideration in the present study, because its influence on the outcome was considered to be negligible as demonstrated in earlier studies by our group [27,28].

The indices for SB were significantly different between the controls and the SB patients, with the latter having the higher index values. In van der Zaag *et al.* (2008) [28], the time-variant nature of SB was discussed. Looking at the individual SB indices within our groups and applying the ‘cut-off band’ of 1.5 – 6.5 epi/hr (with a 90% probability level) as suggested by van der Zaag *et al.* (2008) [28], the SB status of only two SB patients could be disputed. These two patients showed SB indices of 5.7 and 5.9 (epi/hr), respectively, which are both below the upper limit of the afore-mentioned cut-off band. Therefore, there is a 10% chance that these two patients actually were healthy controls. In the healthy control group, three participants were slightly above the cut-off band’s lower limit of 1.5 epi/hr, viz., 1.9, 2.1, and 2.7 epi/hr, respectively. This indicates that there is a 10% chance that in consecutive PSG measurements, they would have been diagnosed as SB patients. To investigate the effect on the results of this possibility, the two SB patients and the three controls mentioned were crossed over to the other group to test for possible statistical consequences. Despite some small numerical changes, which were to be expected, the outcomes and thus the conclusions remained the same (data not shown).

In line with recommendations in the literature, a PLMS event was scored with and without accompanying EEG arousal [38,39,41]. An association zone of three seconds before the onset and after the termination of a motor event (SB and/or PLMS), in which an EEG arousal should occur, was chosen. For the association between SB and PLMS, no clear guidelines for an approach can be found in the literature. Therefore, as to follow the same approach as for the association between motor events and EEG arousals [41], a three-second association zone was also chosen for PLMS events occurring before or after a SB motor event.

In the present study, the scoring of the EEG arousals was done according to the revised ICSD criteria [38]. In 2005 and 2007, the AASM launched revised versions of the Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications [1,39]. The 2007 criteria for EEG arousal scoring are in essence identical to those published in 1997, with a small addition: an EEG arousal requires 10 seconds of stable sleep preceding the EEG change [39]. Application of this additional criterion would have had minimal or no influence on the outcome of the present study, because we scored motor events during sleep only and used the afore-mentioned three-second association zone between the motor event and the occurrence of the EEG arousal.

EEG arousals seem to play an important role, not only in the pathophysiology of sleep disorders, but also in the normal neurophysiologic regulation of the sleep process [25]. During sleep, the organism stays in contact with, and reacts to the outer world by making a relevant selection of the incoming information. This provides the sleeping individual with the ability to adapt, and react, if necessary. In this dynamic perspective, arousals can be considered a reaction on, or anticipation to this information [25]. Indeed, the occurrence of EEG arousals in relation to a PLMS event and/or a SB event is commonly seen in clinical PSG recordings.

So far, either SB motor events or PLMS events have been studied in relation to the occurrence of EEG arousals. In the present study, the combined occurrence of both types of motor events was studied in association with EEG arousals, along with the associations between both types of motor events themselves. It was shown that the number of PLMS events per hour of sleep was higher in SB patients than in healthy controls. This association has already been demonstrated in previous studies, and thus has a confirmatory nature [2,14,15]. A new finding in the present study is that the combined presence of SB and PLMS motor events is significantly more common than the isolated occurrence of these events. This is in line with the suggestion of a common neurophysiological background for both types of motor events. One of the possible potential indicators for this neurophysiological mechanism could be the observed close time relationship with EEG arousals, viz., the observation that the occurrence of the combined SB/PLMS events with associated EEG arousals was significantly more common than that of the combined SB/PLMS events without associated EEG arousals. As can be derived from the Introduction, also other mutual factors could play a role in both motor events. For example, in both SB and PLMS, genetic factors have been identified that make an individual more susceptible for these disorders [12,42], although a common gene variant has not yet been identified. Further, the fact that the pharmacological management strategies for both movement disorders show similarities (viz., opioids, levodopa, D2- and D3-receptor agonists, and clonazepam) [13,16,17–20,43], corroborates with a common neurophysiological background for SB and PLMS. Hence, the results of this study indicate that SB, PLMS, and EEG arousals are highly associated, thus corroborating the suggestion that SB and PLMS are possibly the results of the same underlying neurophysiological mechanism.

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