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ORIGINAL ARTICLE

Time-linked concurrence of sleep bruxism, periodic limb movements, and EEG arousals in sleep bruxers and healthy controls

Jacques van der Zaag · Machiel Naeije · Darrel J. Wicks · Hans L. Hamburger · Frank Lobbezoo

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Abstract
Objective 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 electroencephalographic (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 individuals without SB. 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.
Materials and methods Seventeen SB patients and 11 healthy controls were polysomnographically studied. SB, PLMS, and EEG arousals were scored. An association was noted when the occurrence was within a 3-s association zone.
Results The PLMS index was higher in SB patients than in healthy controls ($P<0.001$). Within the group of SB patients, the combined SB/PLMS index was higher than the isolated SB index ($P<0.001$) and the isolated PLMS index ($P=0.018$). Similarly, the combined SB/PLMS index with EEG arousal was higher than the combined SB/PLMS index without EEG arousal in SB patients ($P<0.001$).
Conclusion The results of this study indicate that SB, PLMS, and EEG arousals commonly concur during sleep in a time-linked manner.
Clinical relevance SB and PLMS probably have a common underlying neurophysiological mechanism.

Keywords Sleep bruxism · PLMS · Arousal · EMG · EEG · Polysomnography

J. van der Zaag · M. Naeije · F. Lobbezoo (✉)
Department of Oral Kinesiology, Academic Centre for Dentistry Amsterdam (ACTA), University of Amsterdam and VU University Amsterdam, MOVE Research Institute Amsterdam, Gustav Mahlerlaan 3004,
1081 LA Amsterdam, The Netherlands
e-mail: f.lobbezoo@acta.nl

D. J. Wicks · H. L. Hamburger
Amsterdam Center for Sleep and Wake Disorders and Department of Clinical Neurophysiology, Slotervaart Medical Center, Amsterdam, The Netherlands

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, both for men and for women [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) [9–11]. 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 [12], genetic factors [13], and overuse of drugs and stimulantia (e.g., caffeine, alcohol, antihistamines, selective serotonin reuptake inhibitor antidepressants like paroxetine) [14] can cause both SB and PLMS.

According to several authors, an association exists between SB and PLMS [2, 15, 16]. 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 [11, 14, 17–21] corroborates with this possibility. In addition, both SB and PLMS have been associated with electroencephalographic (EEG) arousals [4, 9, 22–26], which underlines the possibility that these conditions share a common background. To further assess this issue, as a first step, the question should be answered whether PLMS events occur more frequently in SB patients than in individuals without SB. When this question is answered positively, the second question should be 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. Finally, as to further unravel the possible role of EEG arousals in the underlying neurophysiologic mechanism of SB and PLMS, the third question that needs to be answered in a group of SB patients is whether combined SB/PLMS events with associated EEG arousals are more common than those without associated EEG arousals. Positive answers to all three questions provide further evidence for a common neurophysiological background for SB and PLMS.

Patients and methods

Participants

Seventeen SB patients (12 women and 5 men; mean ± SD age=32.1±6.5 years; range=22–44 years) and 11 healthy subjects (7 women and 4 men; mean ± SD age=34.5± 12.8 years; range=22–47 years), underwent one full-night polysomnographic (PSG) recording 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, Amsterdam, The Netherlands. The healthy subjects were recruited from among the persons attending 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. [30]), and having occlusal tooth wear to at least the degree of exposed dentine (i.e., grade 2 [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 2 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 reuptake inhibitors like paroxetine or anti-Parkinson medication like pramipexole) [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 a written informed consent was obtained from all participants.

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: C_3A_2, O_2A_1), electromyography (EMG: right and left masseter muscles, submental area, and anterior tibialis muscle), electrooculography (right and left), oxygen saturation, heart rate (ECG), body position, and grinding sound (piezoelectric 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. [27].

Data analysis

Sleep was analyzed by an experienced sleep scientist (DJW) in 30-s epochs, according to Rechtschaffen and Kales [35]. Total sleep time (hours), sleep stages N1 until N4 (percent), REM sleep (percent), sleep

efficiency (percent), and arousal index (AI) were determined as sleep outcome variables.

SB was analyzed after Lavigne et al. [36, 37], using an automatic bruxism analyzing tool incorporated in the REMbrandt software (Medcare Automation B.V., Amsterdam, The Netherlands) and following the procedures described in detail by van der Zaag et al. [28]. In short, the first step of the tool consisted of down-sampling the EMG signal to approximately 100 Hz. Thereafter, the EMG signals were rectified and low-pass filtered (time constant, 0.1 s) as to locate areas of increased amplitude. Subsequently, periods of elevated EMG activity were detected, using an EMG threshold of 10 % of the maximum voluntary contraction level of the left and right masseter muscles. Finally, the number of bruxism episodes per hour of sleep (SB index; episodes per hour) was derived for the quantification of SB. 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]. Individual movements were scored as a PLMS if they lasted 0.5–5 s. To be considered periodic, at least four consecutive movements were needed to occur, with movement onsets each 5–90 s apart. The number of individual movements (events) per hour of sleep was used for quantification (PLMS index; events per hour).

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 16 Hz, but not spindles for more than 3 s. In REM sleep, this increase in EEG frequency must be associated with a submental 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 an SB and/or PLMS event if the arousal occurred within 3 s before the onset or after the termination of the motor event [38, 39]. In this study, a PLMS event was considered associated with an SB event if the PLMS event occurred within 3 s before the onset of the SB event, during the event, or within 3 s after the termination of the event.

Statistical analysis

To screen whether or not the outcome measures were normally distributed, Shapiro–Wilk tests and an assessment of normal Q–Q plots were performed.

To test whether or not group differences were present, Fisher's exact test was used for gender; independent-sample *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-sample *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). The Bonferroni method was used to correct for multiple comparisons.

To assess whether PLMS events occurred more frequently in SB patients than in healthy controls, the independent-sample *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, 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 an 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-sample *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.

Results

The distribution of gender did not differ significantly between the groups of healthy controls and SB patients (P=1.000). Likewise, there were no significant age differences between both groups (T_E=0.86; P=0.399). All data followed a normal distribution.

All PSG recordings of both the healthy controls and the SB patients were judged to have normal structures by an experienced sleep scientist who 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 h 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. In addition, 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. [36], the SB indices for the healthy controls did not exceed the value of four events per hour of sleep (maximum value=2.70), while

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

Outcome variable	Controls (n=11)	SB patients (n=17)	Two-sample 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 hour)	2.94±2.37 (0.5–7.2)	35.11±18.82 (17.0–93.7)	T_U=6.96 (P<0.001)[a]
SB (total per hour)	1.05±0.85 (0.2–2.7)	11.47±5.99 (5.7–29.7)	T_U=−7.06 (P<0.001)[a]
PLMS (total per hour)	1.35±1.08 (0.1–2.9)	15.41±8.95 (5.6–30.4)	T_U=6.41 (P<0.001)[a]

T_E = test statistic for independent-sample t test with equal variances, T_U = test statistic for independent-sample t test with unequal variances

[a]Statistically significant after Bonferroni correction

those of the SB patients were all above that threshold (minimum value=5.70). Finally, the number of PLMS events per hour of sleep was significantly higher in the SB patients than in the healthy controls.

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<0.001) (Fig. 1). 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<0.001 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<0.001) (Fig. 2).

Discussion

In this study, three research questions were assessed. First, it was assessed whether PLMS events occur more frequently in SB patients than in individuals without SB. 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 all questions 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 [16,

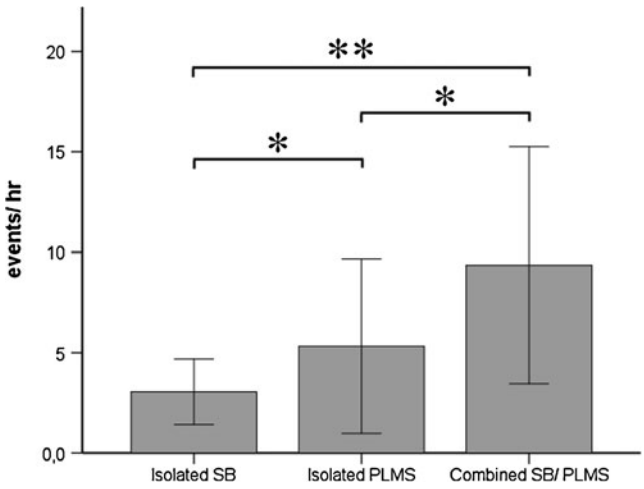

Fig. 1 Mean ± SD of the number of isolated SB events per hour of sleep, the number of isolated PLMS events per hour of sleep, and the number of combined SB/PLMS events per hour of sleep within the group of SB patients. *P<0.05; **P<0.001

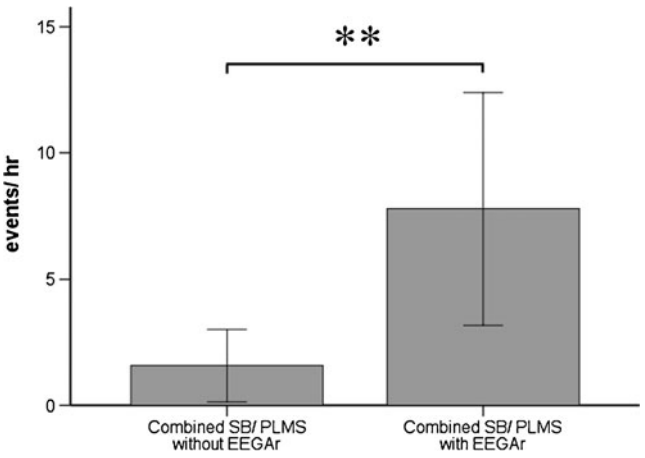

Fig. 2 Mean ± SD of the number of combined SB/PLMS events without associated EEG arousals per hour of sleep and the number of combined SB/PLMS events with associated EEG arousals per hour of sleep within the group of SB patients. **P<0.001

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 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. [28], the time-variant nature of SB was discussed. Looking at the individual SB indices within our groups and applying the "cutoff band" of 1.5–6.5 epi/h (with a 90 % probability level) as suggested by van der Zaag et al. [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/h, respectively, which are both below the upper limit of the aforementioned cutoff 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 cutoff band's lower limit of 1.5 epi/h, viz., 1.9, 2.1, and 2.7 epi/h, 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 3 s 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 time-linked concurrence of 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 3-s association zone was also chosen for PLMS events occurring before or after an 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 American Academy of Sleep Medicine 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 s 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 aforementioned 3-s 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 an 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 relation to EEG arousals, along with the time-linked concurrence of 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 has already been demonstrated in previous studies and thus has a confirmatory nature [2, 15, 16]. 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," other mutual factors could also 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 to these disorders [13, 42], although a common gene variant has not yet been identified. Further, even though some studies demonstrate contradictory results using clonidine [43], the pharmacological management strategies for both movement disorders show similarities (viz., opioids, levodopa, D2 and D3 receptor agonists, and clonazepam) [11, 14, 17–21]. This corroborates with a common neurophysiological background for SB and PLMS. Hence, the results of this study indicate that SB, PLMS, and EEG arousals commonly concur in a time-linked manner, thus

corroborating the suggestion that SB and PLMS are possibly the results of the same underlying neurophysiological mechanism.

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Conflicts of interest The authors declare that they have no conflict of interest.

References

1. American Academy of Sleep Medicine (2005) International classification of sleep disorders, 2nd edn. American Academy of Sleep Medicine, Westchester
2. Lavigne GJ, Montplaisir JY (1994) Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep 17:739–743
3. Ohayon MM, Li KK, Guilleminault C (2001) Risk factors for sleep bruxism in the general population. Chest 119:53–61
4. Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, Terzano MG (1998) Sleep bruxism is a disorder related to periodic arousals during sleep. J Dent Res 77:565–573
5. Lavigne GJ, Rompré PH, Poirier G, Huard H, Kato T, Montplaisir JY (2001) Rhythmic masticatory muscle activity during sleep in humans. J Dent Res 80:443–448
6. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K (2008) Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil 35:476–494
7. Hornyak M, Feige B, Riemann D, Voderholzer U (2006) Periodic leg movements in sleep and periodic limb movement disorder: prevalence, clinical significance and treatment. Sleep Med Rev 10:169–177
8. Karatas M (2009) Restless legs syndrome and periodic limb movements during sleep: diagnosis and treatment. Neurologist 13:294–301
9. Lavigne GJ, Kato T, Kolta A, Sessle BJ (2003) Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med 14:30–46
10. Lobbezoo F, Naeije M (2001) Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 28:1085–1091
11. Lobbezoo F, van der Zaag J, van Selms MKA, Hamburger HL, Naeije M (2008) Principles for the management of bruxism. J Oral Rehabil 35:509–523
12. Vetrugno R, Provini F, Montagna P (2006) Restless legs syndrome and periodic limb movements. Rev Neurol Dis 3:61–70
13. Stefansson H, Rye DB, Hicks A, Petursson H, Ingason A, Thorgeirsson TE (2007) A genetic risk factor for periodic limb movements in sleep. N Engl J Med 357:639–647
14. Chaudhuri KR, Appiah-Kubi LS, Trenkwalder C (2001) Restless legs syndrome. J Neurol Neurosurg Psychiatry 71:143–146
15. Bader G, Lavigne G (2000) Sleep bruxism: overview of an oromandibular sleep movement disorder. Sleep Med Rev 4:27–43
16. Walters AS (2007) Clinical identification of the simple sleep-related movement disorders. Chest 131:1260–1266
17. Montplaisir J, Nicolas A, Denesle R, Gomez-Mancilla B (1999) Restless legs syndrome improved by pramipexole: a double-blind randomized trial. Neurology 52:938–943
18. Ruottinen HM, Partinen M, Hublin C et al (2000) An FDOPA PET study in patients with periodic limb movement disorder and restless legs syndrome. Neurology 54:502–504
19. Saletu A, Parapatics S, Saletu B et al (2005) On the pharmacotherapy of sleep bruxism: placebo-controlled polysomnographic and psychometric studies with clonazepam. Neuropsychobiology 51:214–225
20. Saletu A, Parapatics S, Anderer P, Matejka M, Saletu B (2010) Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. Eur Arch Psychiatry Clin Neurosci 260:163–174
21. Scholz H, Trenkwalder C, Kohnen R, Kriston L, Riemann D, Hornyak M (2011) Dopamine agonists for the treatment of restless legs syndrome. Cochrane Database Syst Rev (3):CD006009. doi:10.1002/14651858.CD006009.pub2
22. Kato T, Rompré P, Montplaisir JY, Sessle BJ, Lavigne GJ (2001) Sleep bruxism: an oromotor activity secondary to micro-arousal. J Dent Res 80:1940–1944
23. Kato T, Montplaisir JY, Guitard F, Sessle BJ, Lund JP, Lavigne GJ (2003) Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res 82:284–288
24. Sforza E, Juony C, Ibanez V (2002) Time-dependent variation in cerebral and autonomic activity during periodic leg movements in sleep: implications for arousal mechanisms. Clin Neurophysiol 113:883–891
25. Halász P, Terzano M, Parrino L, Bódizs R (2004) The nature of arousal in sleep. J Sleep Res 13:1–23
26. Allena M, Campus C, Morrone E et al (2009) Periodic limb movements both in non-REM and REM sleep: relationships between cerebral and autonomic activities. Clin Neurophysiol 120:1282–1290
27. van der Zaag J, Lobbezoo F, Wicks DJ, Visscher CM, Hamburger HL, Naeije M (2005) Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. J Orofac Pain 19:151–158
28. van der Zaag J, Lobbezoo F, Visscher CM, Hamburger HL, Naeije M (2008) Time-variant nature of sleep bruxism outcome variables using ambulatory polysomnography: implications for recognition and therapy evaluation. J Oral Rehabil 35:577–584
29. Carlsson GE, Magnusson T (1999) Management of Temporomandibular Disorders in the General Dental Practice. Quintessence Publishing, Chicago, pp 36–37
30. Kato T, Thie NM, Montplaisir JY, Lavigne GJ (2001) Bruxism and orofacial movements during sleep. In: Attanasio A, Bailey DR (eds) Dental clinics of North America. W.B. Saunders, Philadelphia, pp 657–684
31. Lobbezoo F, Naeije M (2001) A reliability study of clinical tooth wear measurements. J Prosthet Dent 86:597–602
32. Winocur E, Gavish A, Voikovitch M, Emodi-Perlman A, Eli I (2003) Drugs and bruxism: a critical review. J Orofac Pain 17:99–111
33. Dworkin SF, LeResche L (1992) Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord 6:301–355
34. Visscher CM, Naeije M, De Laat A et al (2009) Diagnostic accuracy of TMD pain tests: a multicentre study. J Orofac Pain 23:108–114
35. Rechtschaffen A, Kales A (1968) A manual of standardized terminology: techniques and scoring system for sleep stages of human subjects. UCLA Brain Information Service/Brain Research Institute, Los Angeles
36. Lavigne GJ, Rompré PH, Montplaisir JY (1996) Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 75:546–552
37. Lavigne GJ, Guitard F, Rompré PH, Montplaisir JY (2001) Variability in sleep bruxism activity over time. J Sleep Res 10:237–244

38. American Academy of Sleep Medicine (1997) The international classification of sleep disorders, revised. American Sleep Disorders Association, Rochester
39. American Academy of Sleep Medicine (2007) The AASM manual 2007 for the scoring of sleep and associated events. Rules, terminology and technical specifications. American Academy of Sleep Medicine, Westchester
40. Gallo LM, Lavigne GJ, Rompré P, Palla S (1997) Reliability of scoring EMG orofacial events: polysomnography compared with ambulatory recordings. J Sleep Res 6:259–263
41. Walters AS, Lavigne G, Hening W et al (2007) The scoring of movements in sleep. A review by the movement task force of the American Academy of Sleep Medicine. J Clin Sleep Med 3:155–167
42. Hublin C, Kaprio J, Partinen M, Heikkilä K, Koskenvuo M (1998) Sleep bruxism based on self report in a nationwide twin cohort. J Sleep Res 7:61–68
43. Carra MC, Macaluso GM, Rompré PH et al (2010) Clonidine has a paradoxical effect on cyclic arousal and sleep bruxism during NREM sleep. Sleep 33:1711–1716