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Polysomnographic analysis of bruxism

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The *American Academy of Sleep Medicine* (AASM) defines sleep bruxism as a stereotyped movement disorder characterized by clenching and grinding of the teeth during sleep. Bruxism is found in 14%-20% of children, 8% of adults <60 years old, and 3% of adults >60 years old. The mandibular movements of bruxism can be confused with rhythmic mandibular movements associated with other sleep disorders, such as arousals/microarousals, limb movement disorder, and obstructive sleep apnea/hypopnea syndrome. Polysomnography (PSG) is the study of sleep disorders based on the recording of physiological events throughout an entire night of sleep. This system involves electroencephalography, electrooculography, and electromyography of the submental/suprahyoid, tibialis anterior, mentalis, masseter, and temporal muscles, through which signs of sleep bruxism can be identified. The aim of the present study was to identify bruxism during a night of sleep in a laboratory. Thirty patients were analyzed clinically and underwent PSG. The descriptive analysis correlated apnea, arousals, and limb movements in the 12 patients who exhibited signs and symptoms of sleep bruxism. Of these patients, 4 were confirmed through PSG to have bruxism.

In a comparison between the 4 patients with confirmed bruxism (PSGB group) and the 8 patients confirmed not to have bruxism (NPSGB group), the respiratory event index was lower in the PSGB group (13.17 and 17.95, respectively). The mean leg movement index was higher in the PSGB group than the NPSGB group in total sleep time (21.36 and 8.42, respectively) and in rapid eye movement sleep time (34.54 and 10.30, respectively).

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According to Silber, sleep is a complex and highly organized physiological state.[1] In humans, the sleep-wakefulness cycle corresponds to 3 major phases: wakefulness, slow-wave sleep with no rapid eye movement (NREM), and sleep with rapid eye movement (REM). REM sleep accounts for approximately 20% to 25% of sleep. Stages 1 and 2 NREM account for over half of overall sleep time, whereas stage 3 NREM, which appears mainly in the first half of the sleep period, accounts for 15%.[1,2]

Repetitive movements with contact of the teeth beyond the normal functions of chewing and swallowing represents a parafunctional behavior known as bruxism or parasomnia movement. Motor activity in sleep bruxism was included in the AASM 2005 *International Classification of Sleep Disorders: Diagnostic and Coding Manual*.[2] Bruxism can be a conscious or unconscious habit, diurnal and/or nocturnal—all of which are seen as separate disorders with different etiologies.[3] The facial muscle contractions of sleep bruxism are associated with the sound of teeth grinding, which varies in frequency throughout the night. Sleep bruxism is found in 14%-20% of children, 8% of adults under age 60, and 3% of adults over 60. More in-depth investigations, addressing the fact that sleep bruxism occurs concomitantly with teeth clenching during waking hours in the presence of oral motor control alterations, are needed.[4,5] There is no consensus regarding a specific etiology for bruxism, thus multifactor etiology is the term that is most commonly employed.[6,7]

In the clinical evaluation for the diagnosis of sleep bruxism, a combination of at least 2 of the criteria suggested by Lavigne & Manzini in Table 1 should be considered.[8] The incidence of sleep bruxism is 1.9-fold higher among smokers and individuals who use caffeine, alcohol, drugs, or other substances that affect the central nervous system.[9-14]

Individuals with sleep bruxism may have other concomitant sleep disorders, such as obstructive sleep apnea/hypopnea (OSAH), restless leg syndrome, REM sleep behavior disorder, insomnia, and other parasomnia and dopamine disorders.[9,15,16] Sleep bruxism may be accompanied by secondary risk factors, such as tooth decay, tooth marks on the sides of the tongue, bilateral masseter hypertrophy, occlusal trauma, iatrogenic problems, chronic pain in the mandibular musculature or region of the temporomandibular joint, and limited mandibular movement.[5,17,18]

Polysomnography (PSG) is used for the diagnosis and study of different sleep disorders based on the recording of physiological events throughout an entire night of sleep using electrodes and sensors in a laboratory setting. One clinical routine is the determination of the frequency of rhythmic mandibular movements. This system involves electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG) tests to determine the electrical activity in the brain, eyes, and muscles, respectively. Audiovisual monitoring is also involved. The EMG measures the electrical activity of the submental/suprahyoid regions, thoracic-abdominal movements, oximetry, heart rate, nasal flow pressure, as well as the tibialis anterior, mentalis, masseter, and temporal muscles. With this system, signs of sleep disorders can been identified concomitantly, and episodes of sleep bruxism can be recognized.[19]

Table 1. Criteria used for the clinical diagnosis of sleep bruxism.[8]

- History of teeth grinding noises reported by bedroom partner or family member
- Presence of worn facets on the surface of teeth not compatible with age or function
- Headaches in temporal region
- Fatigued mandibular musculature at night or while awake
- Lockjaw, or difficulty opening the mouth in the morning
- Dental hypersensitivity
- Hypertrophy of the masseter muscles

Prior to initiating the exam, calibration of the muscles is performed based on the maximal voluntary clenching of the teeth for 15 seconds. A 10% increase in amplitude in relation to maximal voluntary clenching, whether or not accompanied by body movement, is considered to characterize a bruxism episode during sleep.[20] Conditions associated with aging and pain are important factors influencing sleep organization, and the prevalence of some sleep disorders (such as sleep apnea) is higher in an older population. Older subjects (>60 years) present fewer sleep bruxism episodes per hour of sleep than younger sleep bruxers.[21] Age may also influence the occurrence of rhythmic masticatory muscle activity in the elderly population.

Bruxism has been associated with poor sleep quality in patients with chronic pain or OSAH. The association between orofacial pain symptoms and sleep bruxism is probaby not independent of the interaction between pain and poor sleep. Sleep apnea, like insomnia, has been associated with increased pain sensivity (decreased pain threshold).[22,23]

Sleep bruxism predominantly occurs in NREM Stage 2 (60%-80% of cases), although it is important to note that some authors have suggested that grinding in the REM phase has a greater destructive potential for teeth.[20,24,25] To facilitate the diagnosis of sleep bruxism, variables such as total sleep time, sleep onset latency, distribution of sleep stages, periodic limb movements, OSAH, and frequency of arousals and microarousals are investigated.[24] Roehrs et al demonstrated that arousal is defined as an unconscious transition from 3 to 15 seconds of cerebral impulses of EEG activity, either alone or accompanied by tachycardia and an increase in EMG.[25] These events, together with teeth grinding, distinguish individuals with bruxism from those without this condition.[26] Studies have revealed that the occurrence of sleep bruxism is influenced by multiple factors, such as psychological/personality traits, genetics, neurochemical activity, and oropharyngeal function.[20,27]

Rhythmic mandibular movements (RMM) constitute the most frequent motor activity observed during sleep and are classified as bruxism in the presence of teeth grinding. The frequency of RMM during sleep is 3-fold greater in individuals with bruxism than individuals without this disorder.[28] The increase in motor activity in individuals with bruxism is understandable, as microarousals are associated with intrinsic differences in cortical autonomic recruitment and motor activation of the muscles of the body and mandible.[29]

The pathophysiology of bruxism is not yet fully understood. Oral somatosensory stimuli in the presence of occlusal abnormalities may trigger the synthesis of catecholamines, thereby enhancing sympathetic tonus (a partial constriction of blood vessels instigated by the sympathetic nervous system), thus increasing muscle reflex. Muscle symptoms in the face and head reported by individuals with bruxism should be distinguished from those indicative of other sleep disorders. Orofacial symptoms associated with temporomandibular disorder, such as limited mouth opening, joint noises, and facial muscle pain, may also be associated with sleep bruxism. Recent studies have strengthened the association between bruxism and apnea, but others have not found a significant number of respiratory disorders or oxygen desaturation cases among individuals with bruxism.[29] In some PSG studies, teeth clenching and RMM were found in 40%-60% of adult patients with OSAH.[22] However, these studies sought to demonstrate a temporal association between apnea events and electromyographic episodes of RMM in patients with OSAH, suggesting that post-apnea respiratory activation may be responsible for the respiratory activation that precedes RMM. Instead, masseter muscle activity is believed to occur at the end of apnea events in the form of a nonspecific oral motor activation causing apnea-induced arousal.[16] Further studies are needed to determine whether the concomitant occurrence of sleep bruxism in patients with OSAH is more associated with the degree of sleep fragmentation rather than an increase in post-apnea arousals.

The aim of the present study was to identify bruxism during a night in a sleep laboratory and determine possible correlations with other sleep disorders.

Materials and methods

The calibration of maximal voluntary teeth clenching was performed on 30 patients who sought treatment at the Sleep Unit of the Vigo General University Hospital in Spain. Of the 30 patients evaluated through PSG, a questionnaire specifically addressing bruxism, and the reports of bedroom partners, 12 exhibited signs and symptoms of bruxism; 4 had the diagnosis confirmed by PSG. All participants signed a statement of informed consent agreeing to participate in the study, in compliance with the norms established by the Vigo General University Hospital.

Besides the conventional PSG instruments, electrodes were also placed on the right and left masseter muscles. Calibration of the PSG apparatus was performed based on the contraction of the masseter muscles corresponding to 10% greater amplitude in relation to maximal voluntary teeth clenching for 15 seconds. The mean number and duration of total facial contraction movements were calculated for the respective episodes in different NREM and REM phases during a night of sleep in the PSGB group (n = 4). The index of total facial contraction movements in both duration and number was then calculated. The index and mean number of contractions of the mentalis muscle and the masseters were obtained by measuring the total number of events when these muscles contracted either in an isolated fashion or simultaneously in the 4 participants of the group. The percentages of the modes of contractions were calculated.

The number and duration of the times that the masseter muscles contracted were recorded, and the same was done for the mentalis muscle. These recordings were made to obtain the total index of bruxism events during all sleep phases in 1 night of sleep for the PSGB patients. Data from the PSG analysis were also used to determine the mean total number and index of the following events in both NREM and REM: apnea, leg movement (LM), and arousals. The same procedure was carried out for the NPSGB group. The total number of sleep disorder events (apnea, LM, and arousals) in the NREM and REM phases and the index data of the PSGB group were compared to those of the NPSGB group. In the descriptive analysis, bruxism events were correlated with apnea, LM, and arousal events in both groups.

Table 2. Mean isolated and combined mentalis and masseter contractions in PSGB group.

Index	Patient 1	Patient 2	Patient 3	Patient 4	Mean
Mentalis					
NREM	16.09	19.78	6.83	23.22	16.48
REM	30.73	7.84	24.26	8.42	17.81
TST	17.66	17.36	9.24	20.93	16.30
Masseter					
NREM	0.87	0.19	0.00	0.98	0.51
REM	2.93	0.00	0.00	2.11	1.26
TST	1.09	0.15	0.00	1.15	0.60
Masseter + Mentalis					
NREM	11.20	6.91	2.61	3.9	6.15
REM	16.10	7.84	24.26	8.42	14.15
TST	11.72	7.03	5.58	4.62	7.24
Total movements	195.00	164.00	85.00	162.00	151.50
Mentalis (%)	57.95	70.73	62.35	78.40	67.36
Masseter (%)	3.59	0.61	0.00	4.32	2.13
Mentalis + Masseter (%)	38.46	28.66	37.65	17.28	30.51

Table 3. Mean number and indices of arousal, apnea, and leg movement (LM) according to phases of sleep in PSGB group.

	Patient 1	Patient 2	Patient 3	Patient 4	Mean
Arousal number					
NREM	51.00	90.00	34.00	6.00	45.25
REM	24.00	35.00	23.00	10.00	23.00
Total	75.00	125.00	57.00	16.00	68.25
Arousal index					
NREM	10.17	21.06	6.83	1.17	9.81
REM	35.12	27.45	29.36	10.53	25.62
Total	12.98	23.19	9.94	2.64	12.19
Apnea number					
NREM	109.00	72.00	12.00	14.00	51.75
REM	43.00	64.00	6.00	19.00	33.00
Total	152.00	136.00	18.00	33.00	84.75
Apnea index					
NREM	19.07	13.44	2.41	2.73	9.41
REM	62.93	50.20	7.66	20.00	35.20
Total	23.75	20.35	3.14	5.44	13.17
LM number					
NREM	63.00	106.00	124.00	101.00	98.50
REM	12.00	50.00	25.00	47.00	33.50
Total	75.00	156.00	149.00	148.00	132.00
LM index					
NREM	11.02	19.78	24.92	19.71	18.86
REM	17.56	39.22	31.91	49.47	34.54
Total	11.72	23.34	25.99	24.40	21.36

Results

A mean total of 151.5 movements were counted, including movements in the mentalis muscle, the masseter muscles, and the mentalis and massester (MM) muscles simultaneously. The mean duration of the movements was similar in the 3 groups. The total masseter, mentalis, and MM movement index (events/period) in relation to total sleep time (TST) was 24.14 (33.22 in REM). The movement index was greater in the mentalis (16.30) than the masseters (0.60) and the MM (7.24) (Table 2). The mean arousal index (events/period) was 12.19 for TST (25.62 in REM). The TST respiratory event index (events/period), which is an indication for apnea, was 13.17 for TST (35.20 in REM). The mean LM index (events/period) was 21.36 for TST (34.54 in REM) (Table 3).

In the comparison of the PSGB and NPSGB groups, the TST respiratory event index was lower in the PSGB group (13.17 and 17.95, respectively). The mean LM index was higher in the PSGB group (21.36 TST and 34.54 REM) in comparison to the NPSGB group (8.42 TST and 10.30 REM) (Table 4).

Discussion

The data for the LM index in this study for both groups are in agreement with findings reported by Kato & Blanchet, who found that individuals with bruxism also exhibit restless leg syndrome and are affected by an increase in arousals and microarousals.[16] Analyses involving more than 1 night of sleep are needed regarding the clarification of whether or not LM is associated with a greater number of arousals in the REM phase. According to Lavigne et al, bruxism is a secondary motor activity of endogenous microarousals represented by the cardiac-autonomous EEG cycle and motor fluctuations.[23] Thus, an increase in activity of the brain and autonomous nervous system precedes teeth grinding in humans. Bader reports that a large number of bruxism episodes occur in conjunction with other bodily movements and these movements are interlinked with the occurrence of arousals.[29] Lavigne et al postulated that the occurrence of bruxism

Table 4. Mean number and indices of arousal, apnea, and leg movement (LM) according to phases of sleep in NPSGB vs PSGB groups.

	NPSGB	PSGB
Apnea number		
NREM	82.125	51.750
REM	25.125	33.000
Total	107.250	84.750
Apnea index		
NREM	16.490	9.410
REM	28.400	35.200
Total	17.950	13.170
LM number		
NREM	42.875	98.500
REM	9.875	33.500
Total	52.750	132.000
LM index		
NREM	8.150	18.860
REM	10.300	34.540
Total	8.420	21.360
Arousal		
Number	47.880	68.250
Index	7.590	12.190

episodes is more associated with the periodic fluctuations found in arousals than the influence of sudden changes in the autonomous nervous system during sleep.[5]

According to Bader, sleep onset latency, TST, and duration of sleep stages in individuals with bruxism generally do not differ from individuals without this condition.[29] Individuals with bruxism do tend to return to a light sleep, and there is an increase in the amount of time spent in this light sleep in comparison to deep sleep, thereby increasing the fragmentation of sleep, and consequently a greater number of arousals, increased intrasleep wakefulness, a greater number of transient changes in phases of sleep due to the increased responsiveness of oromotor activity to microarousals, and reduced sleep efficiency.[23,26,29] Ohayon et al reported an association between bruxism and OSAH resulting in nonrestorative sleep and daytime sleepiness.[9] The authors also proposed that OSAH was caused by hypoxia and breathing difficulty, resulting in multiple oral phenomena, including teeth clenching.[9] Ohayan et al suggested further investigations were needed in order to determine whether teeth grinding is actually linked more to OSAH than post-apnea arousal.[9]

The swallowing of saliva is reduced 10-fold during sleep, and the activation of this reflex is associated with arousals. According to Miyawaki et al, there is a greater need during the swallowing of saliva for the preservation of airway patency and esophageal lubrication, and sleep bruxism may occur as part of these physiological events.[20] Both Lavigne et al and Bader reported an association between sleep bruxism and poor sleep quality in elderly individuals with OSAH.[11,29] In the present study, however, no association was found between apnea and bruxism. Therefore, further investigations are needed, involving larger samples of patients and a longer study duration.

Conclusion

Patients with bruxism diagnosed based on a clinical examination and positive PSG findings had poorer sleep quality in relation to individuals for whom bruxism was not confirmed by PSG, as evidenced in the greater number of arousals, longer intrasleep wakefulness, lesser sleep efficiency, and a greater number of sleep phase changes. No association was found between episodes of bruxism and the number of apnea events, but there appears to be an association with LM. This finding suggests the need for further studies in order to clarify this possible association.

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