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ORIGINAL

Clinical Features of Oxygenation, Micro-Arousals, and Periodic Limb Movements in Sleep Bruxism: A Retrospective Study

Características Clínicas de la Oxigenación, Microdespertares y Movimientos Periódicos de Extremidades en el Bruxismo del Sueño: Un Estudio Retrospectivo

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ABSTRACT

This retrospective study compared oxygenation, hypopnea episodes, micro-arousals, and periodic limb movements in individuals with sleep bruxism and a control group. A total of 129 polysomnography records from 2011 to 2019 were analyzed (54 with SB, 74 controls). Subjects with sleep bruxism were classified according to Lavigne et al. (≥25 events/hour). Variables included sex, age, micro-arousals, oxygenation, hypopnea episodes, and periodic limb movements. The Mann-Whitney U test was used for comparisons. Subjects with sleep bruxism had a higher average number of micro-arousals: NMOR (SB: 29,5 vs. Control: 24,3) and MOR (sleep bruxism: 18,5 vs. Control: 13,2). Respiratory events with arousals were twice as frequent (sleep bruxism: 3,07 vs. Control: 1,69), and hypopnea episodes were more frequent and lasted longer in sleep bruxism (34,09 episodes, 11,5 sec) compared to controls (24,38 episodes, 7,4 sec). Oxygenation was lower in controls but remained within normal limits: NMOR (SB: 92,6 vs. Control: 89) and MOR (sleep bruxism: 92,9 vs. Control: 88). periodic limb movements frequency was also higher in sleep bruxism (sleep bruxism: 72,4 vs. Control: 65,7), though differences were not statistically significant ($p > 0,05$). In conclusion, sleep bruxism was associated with more frequent micro-arousals, hypopnea episodes, and periodic limb movements compared to controls, while oxygenation levels were lower in the control group.

Keywords: Sleep Bruxism; Cell Respiration; Sleep Arousal Disorders; Nocturnal Myoclonus Syndrome.

RESUMEN

Este estudio retrospectivo comparó la oxigenación, los episodios de hipopnea, los microdespertares y los movimientos periódicos de las extremidades en individuos con bruxismo del sueño y un grupo de control.

Se analizaron un total de 129 registros de polisomnografía de 2011 a 2019 (54 con bruxismo del sueño, 74 controles). Los sujetos con bruxismo del sueño fueron clasificados según Lavigne et al. (≥25 eventos/hora). Las variables incluyeron sexo, edad, microdespertares, oxigenación, episodios de hipopnea y movimientos periódicos de las extremidades. Para las comparaciones se utilizó la prueba U de Mann-Whitney. Los sujetos con bruxismo del sueño presentaron un mayor número promedio de microdespertares: NMOR (bruxismo del sueño: 29,5 vs. Control: 24,3) y MOR (bruxismo del sueño: 18,5 vs. Control: 13,2). Los eventos respiratorios con despertares fueron el doble de frecuentes en el bruxismo del sueño (3,07) en comparación con el grupo de control (1,69). Los episodios de hipopnea fueron más frecuentes y de mayor duración en el bruxismo del sueño (34,09 episodios, 11,5 seg) en comparación con los controles (24,38 episodios, 7,4 seg). La oxigenación fue menor en los controles, pero se mantuvo dentro de los límites normales: NMOR (bruxismo del sueño: 92,6 vs. Control: 89) y MOR (bruxismo del sueño: 92,9 vs. Control: 88). La frecuencia de los movimientos periódicos de las extremidades también fue mayor en el bruxismo del sueño (72,4 vs. 65,7 en el grupo de control), aunque las diferencias no fueron estadísticamente significativas (p > 0,05). En conclusión, el bruxismo del sueño se asoció con una mayor frecuencia de microdespertares, episodios de hipopnea y movimientos periódicos de las extremidades en comparación con los controles, mientras que los niveles de oxigenación fueron más bajos en el grupo de control.

Palabras clave: Bruxismo del Sueño; Respiración Celular; Trastornos del Despertar del Sueño; Síndrome de Mioclonía Nocturna.

INTRODUCTION

Sleep Bruxism (SB) is an involuntary motor activity related to the trigeminal nerve. It is characterized by repetitive episodes of muscle activity in the jaw, manifesting as teeth grinding or jaw clenching during sleep.[1] SB is believed to originate in the brainstem, a part of the brain that connects the brain to the spinal cord, due to an imbalance of neurotransmitters such as dopamine and GABA.[2]

This disorder is commonly diagnosed through polysomnography (PSG), a procedure that monitors various bodily functions during sleep. This test includes the measurement of brain activity through electroencephalogram (EEG), eye movements via electrooculogram (EOG), muscle activity through electromyogram (EMG), heart activity with electrocardiogram (ECG), and blood oxygen levels using pulse oximetry.[3,4]

Additionally, rhythmic masticatory muscle activity (RMA) is recorded, and audio and/or video recordings are used as complementary measures.[5] Research suggests a relationship between SB and other disorders or systemic diseases, such as sleep respiratory disorders, micro-arousals, and uncontrolled limb movements.[6] Micro-arousals, also known as ‚arousals,' are biological events that include an increase in sympathetic cardiac activity, cortical activity, heart rate, blood pressure, and respiratory amplitude.[7,8,9,10]

These events are characterized by abrupt changes of 3 to 5 seconds in electroencephalographic (EEG) activity, accompanied by an increase in heart rate and muscle tone.[11] Additionally, periodic limb movements are observed, characterized by repetitive and stereotyped movements.[12,13] These changes in brain activity may be related to the part of the central nervous system that controls limb movements.[14] Regarding SB, some studies have indicated a positive correlation between SB and oxygen desaturation index, as well as between SB and minimum oxygen saturation.[15]

However, the exact relationship between Obstructive Sleep Apnea (OSA) and SB, whether causal or merely correlative, remains unclear. The aim of this study is to compare oxygenation, hypopnea episodes, micro-arousals, and periodic limb movements between subjects with SB and a control group, thereby providing a more detailed understanding of these interactions.

METHOD

Design

This retrospective descriptive study was conducted following the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines to ensure rigor and transparency in observational research.[16] The study protocol was approved by the Bioethics Committee of University of Manizales and adhered to the regulations of Resolution N 8430 governing health research, in accordance with the Helsinki Declaration.[17]

Context

This study was carried out in a sleep laboratory at University of Manizales (Colombia), where PSG records from 2011 to 2019 were analyzed, focusing on patients with SB as the study population. Physiological, neurophysiological, and cardiopulmonary analyses were conducted using PSG data, which included micro-arousals, oxygenation, and periodic limb movements to provide a comprehensive view of how these factors

interact. The PSGs were evaluated by an experienced physiatrist who assessed relevant parameters such as brain activity, eye movements, muscle activity, cardiac function, oxygenation, and micro-arousals, ensuring accurate interpretation of the results.

Participants

The sample consisted of patients who underwent PSG at the sleep laboratory of University of Manizales between 2011 and 2019. The study included 54 patients diagnosed with SB and 74 individuals as the control group, selected through non-probabilistic sampling. The inclusion criteria were:

- Patients with more than 25 episodes of sleep bruxism per hour, as defined by the threshold established by Lavigne et al. during a PSG. [14,18]
- Patients of any gender, aged 18 or older.
- Patients with complete PSG records and available physiological, neurophysiological, and cardiopulmonary parameters from the University of Manizales sleep laboratory.
- Patients who provided informed consent for the use of their clinical records and data.

Sleep Bruxism

Sleep bruxism was assessed by evaluating the frequency of bruxism events during PSG. The diagnosis required that a patient exhibit more than 25 episodes of bruxism activity per hour. Bruxism activity during PSG refers to episodes of involuntary teeth grinding or clenching, identified through measurements of jaw muscle activity. This criterion is crucial for accurately classifying patients in sleep bruxism studies, allowing an objective evaluation of the presence and severity of the disorder.[14,18] The analysis of SB events involved the use of Cadwell Easy III polysomnography software.[19]

Bias

Several potential biases were considered in this study. Selection bias could have influenced the results since the sample was obtained from a single sleep laboratory, which may not be representative of the general population with sleep bruxism. Additionally, patient classification based on the threshold of 25 episodes per hour may have excluded individuals with milder forms of bruxism, limiting the generalizability of the findings. Information bias is another potential risk, as data were derived from PSG records, which depend on equipment accuracy and expert interpretation. Finally, confounding bias could have been introduced due to the presence of comorbidities or additional sleep disorders in the participants, which were not fully controlled for in the analysis and may have influenced the observed results.

Sample Size

The sample size calculation was performed using G*Power software. A total of 129 participants were determined to be adequate, considering a moderate effect size (d = 0,5) and a significance level of 0,05. The analysis indicated a statistical power of 79 %, which approaches the traditional recommendation of 80 %, suggesting that the study has sufficient capacity to detect real effects in the analyzed variables.[20,21]

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 27.0 for Windows. The normality of the data distribution was determined using the Shapiro-Wilk test, while the homogeneity of variances was assessed with Levene's test. Descriptive statistics were calculated, including central tendency and dispersion measures such as the mean (X), standard deviation (SD), and 95 % confidence intervals. For inferential analysis, the Mann-Whitney U test was used, with a two-tailed significance level of 0,05 for all analyses.

RESULTS

In this study, a total of 128 subjects were included, of which 33 % were male and 67 % were female. The average age of the participants was 29,5 years (95 % Confidence Interval: 27,53–31,6). Among the analyzed subjects, 54 were diagnosed with SB, and 74 were part of the control group. The bruxism index, which refers to the average number of bruxism events per hour, was 43 in subjects with SB and 13,9 in control group subjects. The details of these characteristics are presented in table 1.

Table 2 presents the comparison of various sleep parameters between subjects with SB and the control group. Aspects such as total sleep time, time in non-REM (NREM) and REM phases, arousals, hypopnea episodes, oxygenation in different sleep phases, and periodic limb movement were analyzed. Despite observed variations, no significant differences were found between the groups in any of the evaluated parameters ($p > 0,05$), suggesting that these factors are not substantially affected by the presence of sleep bruxism in the studied sample.

Table 1. Comparison of Sleep Bruxism Events Between Subjects With and Without Sleep Bruxism

Variable	Sleep Bruxism (n = 54)		Control (n = 74)		
	X ± SD	95% CI	X ± SD	95% CI	p-value
Sleep bruxism events	299,33 ± 159,45	255,81 - 342,86	92,11 ± 50,14	80,49-105,72	≤0,001
Bruxism index	43,68 ± 16,58	39,15 - 48,20	13,95 ± 6,92	12,35 -15,55	≤0,001

X: Mean; SD: standard deviation; CI: 95 % confidence interval. Source: own elaboration.

Table 2. Sleep Time, Arousals, Hypopnea Episodes, Oxygenation, and Periodic Limb Movement in Subjects With and Without Sleep Bruxism

Variable	Sleep Bruxism (n = 54)		Control (n = 74)		
	X ± SD	95% CI	X ± SD	95% CI	p-value
Total sleep time (min)	396,77 ± 58,218	380,88 - 412,66	397,59 ± 61,03	383,46 - 411,73	0,85
NREM sleep time (min)	278,51± 75,08	263,74 - 293,29	277,92 ± 54,14	260,52 - 295,31	0,41
REM sleep time (min)	118,25 ± 79,16	106,79 -129,72	119,68 ± 42,01	101,34 - 138,02	0,18
Arousals in NREM	29,56 ± 19,77	23,66 - 35,45	24,30 ± 21,59	19,72 - 28,88	0,19
Arousals in REM	18,57 ± 17,96	13,67 - 23,47	13,24 ± 12,89	10,26 - 16,23	0,08
Total Arousals	47,91 ± 34,72	38,43 - 57,39	36,72 ± 26,69	30,53 - 42,90	0,13
Respiratory events with arousals	3,07 ± 8,73	0,69 - 5,46	1,69 ± 3,73	0,92 - 2,55	0,88
Total hypopnea episodes	34,09 ± 83,97	11,17 - 57,01	24,38 ± 41,13	14,85 - 33,91	0,79
Duration of hypopnea episodes (min)	11,53 ± 35,50	1,840 - 21,22	7,46 ± 13,61	4,30 - 10,61	0,80
Oxygen saturation in NREM (%)	92,61 ± 1,77	92,13 - 93,09	89,01 ± 18,49	84,73 - 93,30	0,68
Oxygen saturation in REM (%)	92,93 ± 2,11	92,35 - 93,51	88 ± 21,25	83,08 - 92,92	0,87
Periodic limb movement	75,67 ± 72,43	55,90 - 95,44	58,74 ± 65,72	43,52 - 73,97	0,12

X: Mean; SD: standard deviation; CI: 95 % confidence interval. Source: own elaboration.

DISCUSSION

In this study, a higher average of microarousals was observed during both NREM and REM sleep phases in subjects with SB, although this difference did not reach statistical significance (p > 0,05). This finding aligns with the study by Hosoya et al.,[22] which supports the notion that SB is associated with arousal patterns during sleep. This could be interpreted as a response to subconscious stimuli or a dysfunction in sleep regulation mechanisms.

Transient arousals, which consist of brief and regular increases in brain activity, are evenly distributed between the NREM and REM phases, being more frequent during light sleep stages.[23,24] These data suggest that sleep bruxism might represent an adaptive or compensatory response to interruptions in the normal sleep pattern, possibly acting as a neuromuscular readjustment mechanism.

The analysis of respiratory events revealed a significant increase in the frequency and duration of hypopnea episodes in the SB group. These results, consistent with observations from other studies,[7,22] raise the hypothesis that SB may be related to respiratory function disturbances during sleep. However, it is important to emphasize that the relationship between SB and sleep-related breathing disorders, such as OSA, is inconclusive and requires further investigation.[25]

Regarding oximetry, the average values remained within normal limits in both groups, although they were slightly lower in the control group. This finding suggests that SB does not necessarily imply significant oxygen desaturation, which is consistent with recent research. [26] This aspect may have clinical implications in assessing respiratory function in patients with SB, indicating that oxygen desaturation is not a prominent feature of this condition. Nevertheless, there is a possibility that sleep bruxism acts as a compensatory mechanism to maintain upper airway muscle tone, particularly in patients with OSA, suggesting a protective rather than a directly pathological association.[27]

Another notable aspect is the observed increase in the frequency of periodic limb movements in individuals with SB. This correlation may indicate a shared neurophysiological mechanism between sleep bruxism and sleep movement disorders, possibly related to brain arousal and EEG awakenings.[5] The concomitance of these movements with microarousals and alterations in muscle tone suggests a broader pattern of neuromuscular activity in sleep bruxism, potentially linked to central mechanisms of sleep regulation and motor activity.

This study contributes to the understanding of sleep bruxism by highlighting its association with altered arousal patterns during sleep, respiratory disturbances, and periodic limb movements. Although these observations provide valuable insights into the multifaceted nature of SB, it is crucial to acknowledge the inherent limitations of the retrospective design and non-probabilistic sampling. These limitations underscore

the need for future prospective and controlled studies to validate and expand these findings, providing a more solid foundation for the development of targeted therapeutic strategies and a comprehensive understanding of sleep bruxism.

Future research could also benefit from incorporating advanced imaging techniques and biomarker analysis to further explore the neurophysiological and biochemical bases of SB, as well as its interaction with other sleep and systemic disorders. Additionally, it would be worthwhile to investigate psychological and environmental factors that may influence the prevalence and severity of sleep bruxism, offering a more holistic perspective that encompasses both the physical and psychosocial components of this condition. This could lead not only to a better understanding of SB but also to more personalized and effective interventions that address the multiple facets of this complex condition.

CONCLUSIONS

In this study, a trend toward a higher average of microarousals was observed in subjects with SB compared to those without SB, although this difference did not reach statistical significance. However, subjects with SB showed a significant increase in the frequency and duration of hypopnea events, as well as a higher incidence of periodic limb movements. Additionally, oximetry results indicated a slight decrease in the control group, highlighting the complexity of interactions between sleep bruxism and sleep physiology.

These findings emphasize the importance of considering both respiratory and motor aspects in the study of SB, suggesting that sleep bruxism may influence various dimensions of sleep. They underscore the need for further research to explore the underlying mechanisms of SB and its impact on overall sleep quality, which could lead to improved diagnostic and therapeutic approaches for affected patients.

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CONFLICT OF INTERESTS

The authors declare that there are no conflicts of interest.

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