Sleep Bruxism and Sleep-Disordered Breathing

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ABSTRACT
Sleep bruxism (SB) is a repetitive jaw muscle activity with clenching or grinding of the teeth during sleep. SB is characterized by what is known as rhythmic masticatory muscle activity (RMMA). RMMA is the laboratory polysomnographic finding that differentiates SB from other oromandibular movements seen during sleep. Most often RMMA episodes are associated with sleep arousal. Some patients will report similar complaints related to both SB and sleep disordered breathing (SDB). There are some reports that would suggest that SB is a result of SDB. It has has been postulated that SB is a compensatory mechanism to re establish muscle tone of the upper airway. While these disorders do in fact often present concomitantly, the relationship between the two is yet to be fully elucidated. This Critical Appraisal reviews 3 recent publications with the intent to better define what relationships may exists between SDB and SB. While the current evidence appears to support the notion that these are often concomitant disorders, it also makes clear that evidence to support the hypothesis that SDB is causative for SB is currently lacking. *(J Esthet Restor Dent 00:000–000, 2016)*

Sleep Bruxism in Patients with Sleep-disordered Breathing

T.T. SJÖHOLM, A.A. LOWE, K. MIYAMOTO, A. FLEETHAM, C.F. RYAN
Archives of Oral Biology 2000 (45:889–96)

ABSTRACT

Objective: The primary objective was to determine the occurrence of sleep bruxism (SB) in subjects with sleep-disordered breathing. A secondary objective was to test the diagnosis of SB based on three different data sets: questionnaires, clinical observations, and masseter muscle electromyography (EMG) activity.

Materials and Methods: A total of 21 subjects (19 males/two females, mean age 40 years) were randomly selected after receiving a diagnosis of obstructive sleep apnea (OSA) from a sleep physician. Subjects were included if they were 18 years of age or older, had healthy teeth without acute caries lesions and who had not previously used continuous positive airway pressure, oral appliance therapy or any surgical treatment for OSA. Also, subjects with a periodic limb movement-related arousal index of more than five per hour of sleep were excluded. Sleep breathing parameters were measured using polysomnagraphic (PSG) recordings. All studies were manually scored and obstructive apneas were defined as the cessation of air flow for at least 10 seconds and hypopneas were defined as a decrease of more than 50% in thoraco-abdominal amplitude (Respitrace sum) for at least 10 seconds. The diagnosis of SB was made from a combination of questionnaire, clinical observation, and all-night PSG recording which included masseter muscle EMG. If two of the three parameters were positive, the subject was considered to have SB. Based on the apnea hypopnea index (AHI), the patients were arbitrarily divided into two groups with mild (AHI < 15) and moderate (AHI > 15) OSA.

Results: The total sleep time and efficiency was the same for both the mild sleep apnea ($n = 11$) and moderate

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sleep apnea ($n = 10$) groups. The moderate sleep apnea patients demonstrated slightly less stage 2 and stage 4 sleep. Blood pressures and heart rates did not differ significantly between the groups. SB was diagnosed in 54% of patients with mild OSA and 40% of patients with moderate OSA. The percentage of masseter muscle contraction episodes which occurred at the end of the apnea event was only 3.5% in the mild and 14.4% in the moderate OSA group. A mild elevation in masseter muscle tone was frequently found at the termination of an apneic event. However, SB was not observed during snoring or apnea events in any subject.

Conclusions: There was no clear relationship between the termination of an apnea event and masseter muscle activity. Masseter muscle activity appeared more coincident with the general arousals resultant from the apneic events in both groups rather than the apneic event itself. As to the secondary objective, the diagnostic criteria of SB based on the combination of questionnaires, clinical observations and masseter muscle EMG activity may be valid but should first be studied in larger populations.

COMMENTARY

SB can be a significant factor in tooth wear or damage, failed dental restorations, and periodontal problems. It is also suggested to play a role in masticatory muscle pain, temporomandibular joint disorders and possibly headaches. The diagnosis of SB is difficult due to its night to night variability. It appears that SB occurs in response to disturbed sleep and is rarely directly associated with respiratory events during sleep.

Temporal Association between Sleep Apnea–Hypopnea and Sleep Bruxism Events

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Journal of Sleep Research 2014 (23:196–203)

ABSTRACT

Objective: To investigate the temporal association between OSA hypopnea events and SB events (SBEs) which may suggest a cause and effect relationship.

Materials and Methods: Ten male subjects with mean age of 46.7 years and a diagnosis of OSA hypopnea syndrome and SB were selected for the study based on sleep lab confirmation of both entities. Subjects were excluded for major neurological or psychiatric disorders, other sleep disorders, psychoactive medication use, or being edentulous. Their mean body mass index was 27.7 and the mean score on the Epworth Sleepiness Scale was 8/24. The study subjects slept one night in the sleep clinic. The PSG recording variables included electroencephalograms, right and left electrooculogram, EMGs of the submental and masseter muscles as well as the anterior tibialis muscles and an electrocardiogram. Sleep breathing was measured by airflow, chest, and abdominal effort, and SpO_2 measured by pulse oximetry. For SB identification, the PSG recordings included masseter EMG and audio–video recording. Apnea–hypopnea events (AHE) and sleep stage were scored according to American Academy of Sleep Medicine (AASM) criteria.

AHEs events were recorded within a 5-minute time interval before and after SBEs. The SBEs served as the reference point. Two temporal patterns were analyzed: the interval between AHE termination and SBE initiation, (T1); and secondly, the time interval between SBE termination and AHE beginning (T2). Events other than those identified as SBEs (swallowing, coughing, and face scratching) were determined by AV recording and were excluded.

Results: The mean AHI was 24.0 and the mean number of SBEs per hour was 13. The T1 interval had a mean length of 33.4 seconds while the T2 interval averaged 64.0 seconds. Significantly more SBEs were scored in association with the T1 than the T2 pattern.

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Conclusion: In the subjects with concomitant AHE and SBEs, most SBEs occurred within a brief time frame after AHEs. These results could suggest that SBEs occurring in close proximity to AHEs is a secondary form SB.

COMMENTARY

To date, no single etiology for SB has been identified. In fact, data would suggest that it is multifactorial and may present as a primary sleep disorder or concomitant with other sleep disorders. SBEs have been shown to occur following autonomic arousal in some instances. As AHEs may lead to autonomic activation, SB could be considered, in some instances, to be secondary to AHE. A significant limitation to this study is that the subjects were only monitored with PSG for one night. It is known that SB as well as OSA has a night-to-night variability. Also, there is the possibility of a first night effect in the sleep lab affecting sleep architecture. The authors make clear that more detailed analyses of arousal and sympathetic activation in subjects with OSA hypopnea syndrome are needed, as well as larger and more diverse samples. Future studies should also include subjects with central sleep apnea hypopnea syndrome.

Relationship between Sleep Bruxism and Sleep Respiratory Events in Patients with Obstructive Sleep Apnea

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Sleep Breath 2014 (18:837–844)

ABSTRACT

Objective: To determine the relationships between SB and sleep respiratory events in patients with OSA compared with healthy volunteers.

Materials and Methods: Sixty-seven patients (18 females and 49 males; mean age, 54 years; mean BMI, 28.1 kg/m^2) with OSA (five or more events per hour of sleep as determined by overnight polysomnography) were accepted for the study. Sixteen healthy volunteers (8 females and 8 males; mean age, 24 years; mean BMI, 19.7 kg/m2) were accepted as controls. None of the healthy volunteers had any sleep disorders or medical conditions. Data for the study were collected by polysomnography (PSG) during an overnight sleep tests in a dark, quiet room.

A cortical microarousal was defined as an abrupt electroencephalogram (EEG) frequency shift and simultaneous increase in the chin electromyogram (EMG) amplitude. Apnea was defined as a cessation of airflow lasting at least 10 seconds. Hypopneas were defined as a greater than 50% decrease in the thoracoabdominal amplitude associated with a greater than 3% decrease in the oxygen saturation.

EMG activity of the masseter muscles to determine SB was scored based on previously published diagnostic criteria. Events were scored as three different types of events: phasic, tonic, and mixed. A phasic type of SB event consists of at least three EMG bursts of 0.25–2.00 seconds duration separated by two intervals. A tonic type of SB event consists of an EMG burst lasting more than 2.00 seconds. The diagnosis of SB was confirmed when the subject had more than four events per hour of sleep.

SB events were categorized based on the events associated with them. The SB + microarousal + apnea/hypopnea category was defined as an SB event that occurred during a microarousal event that followed an apnea/hypopnea event. The SB + microarousal + no apnea/hypopnea category was defined as an SB event that occurred during a microarousal event that did not follow from an apnea/hypopnea event.

Results: As to the primary question; SB occurred more frequently in the OSA patient group than in the control

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group. Conversely, apnea/hypopnea and desaturation events occurred significantly more frequently in patients with SB than those without. The occurrence of the phasic type of SB appeared to be associated with the occurrence of OSA, microarousal, and oxygen desaturation events. The frequency of SB events associated with microarousal events that followed apnea/hypopnea events was significantly higher in the OSA group as compared to the controls. As to the secondary question; SB events did not affect the sleep time, sleep efficiency, sleep stages, microarousal, or snoring events. It should also be noted that the phasic type of SB tended to correlate more with OSA events as opposed to the tonic or mixed type of events.

Conclusion: Patients with OSA have a high risk of SB. Successful treatment of OSA may decrease SB events.

COMMENTARY

This study examined the relationship of OSA to SB. Based on the results; it would appear that SB events occur during the microarousals which follow OSA events. The findings would seem to support the notion that SB is more of an arousal response rather than a mechanism to physiologically stabilize the airway in response to OSA events. Previous studies have reported a positive correlation between the tonic (clenching) and mixed types of SB. However, this study did not find any correlation in these types of events with OSA. As with many studies assessing SB, a potential limitation of this study is that only one night of data was recorded. Previous reports have demonstrated night to night variability in SB as well as OSA. An interesting finding in this study was that the frequency of SB was higher in the older patients, correlating with the frequency of OSA being greater with increasing age. This finding would appear to contradict previous reports that indicate the frequency of SB decreases with age. It should be noted that most prevalence reports of SB are based on surveys and questionnaires and could be underreporting the true prevalence of this disorder. In this study, the control group age was less than that of the experimental group. The authors admit that it would be more ideal to have a more homogenous population to study.

SUGGESTED READING

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THE BOTTOM LINE

Sleep bruxism is a sleep-related movement disorder often associated with arousals.

Sleep-related breathing disorders may be considered a risk factor for sleep bruxism.

There does not appear to be significant evidence to support a cause/effect relationship between sleep-related breathing disorders and sleep bruxism.

Due to the night-to-night variability of sleep bruxism and sleep-related breathing disorders; more studies are needed to clearly define the relationship between these two disorders.

Further studies should include more homogenous patient populations.

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