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Brief communication

Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure

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Abstract

Several studies have reported that sleep bruxism rarely occurs in isolation. Recently, in an epidemiological study of sleep bruxism and risk factors in the general population, it was found that among the associated sleep symptoms and disorders obstructive sleep apnea (OSA) was the highest risk factor for tooth grinding during sleep. The purpose of this report was to evaluate the effect of continuous positive airway pressure (CPAP) on sleep bruxism in a patient with both severe OSA and sleep tooth grinding. Two polysomnographic (PSG) recordings were carried out. The first showed 67 events of sounded tooth grinding, most of them appearing as an arousal response at the end of apnea/hypopnea events in both the supine and lateral postures. During the CPAP titration night most breathing abnormalities were eliminated and a complete eradication of the tooth grinding events was observed. The results of this study suggest that when sleep bruxism is related to apnea/hypopneas, the successful treatment of these breathing abnormalities may eliminate bruxism during sleep.

Keywords: Sleep bruxism; Obstructive sleep apnea; Continuous positive airway pressure

1. Introduction

Sleep bruxism has been considered for many years as an arousal response [1]. Macaluso et al. [2] analyzed polysomnographic (PSG) data of six subjects affected by sleep bruxism and six healthy age matched control volunteers and showed that bruxers had a higher number of transient arousals and that 88% of bruxism episodes were associated with cyclic alternating pattern and always occurred during transient arousals. Recently, a support to this notion was also provided by Kato et al. [3]. They showed an increase in cortical electroencephalogram activity 4 s before the onset of oromotor activity in 79% of sleep bruxism episodes.

Since sleep bruxism is strongly related to arousal episodes and obstructive sleep apnea (OSA) is also accompanied by a high amount of short arousals, a relation between these two entities may exist. Phillips et al. [4] found a positive relationship between the frequency of sleep disordered breathing and tooth clenching and also showed that sleeping in the supine posture increases the frequency of both abnormalities. However, other authors [5,6] have not found a clear relationship between these two entities.

Recently, in an epidemiological study of sleep bruxism and risk factors in the general population, it was found that among the associated sleep symptoms and disorders OSA was the highest risk factor for tooth grinding during sleep [7]. Continuous positive airway pressure (CPAP) is considered the best treatment for symptomatic OSA patients with a severity degree of moderate–severe [8]. The purpose of this report was to evaluate the effect of CPAP on sleep bruxism in a patient with severe OSA.

2. Methods

2.1. The patient

A 47-year-old taxi driver was referred by his family physician to our Sleep Disorders Unit due to sleep bruxism and snoring for many years. His bruxism appears soon after falling asleep and in all body positions. His wife noted that fatigue aggravates this condition. His Epworth Sleepiness Scale (ESS) was 9 out of a maximum of 24 (a score of $\geq$10 is considered as an indication of excessive daytime sleepiness [9]). His past medical history was unremarkable except for the existence of arterial hypertension during the past 3 years. His body mass index (BMI) is 38.4. A BMI value of $\geq$30 is considered as obesity [10].

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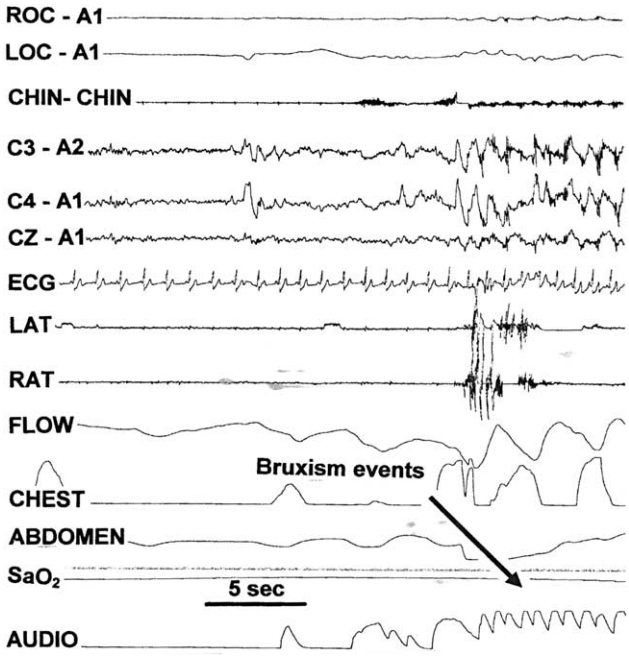

Fig. 1. Sounded bruxism events during sleep. ROC, right ocular cantus; LOC, left ocular cantus; CHIN-CHIN-submental EMG activity; ECG, Electrocardiogram; LAT-EMG of left anterior tibialis; RAT-EMG of right anterior tibialis; Audio-output from sound level meter in decibels.

2.2. Polysomnographic evaluation

The PSG recordings included the following parameters: electrooculogram (EOG) (two channels); electroencephalogram (EEG) (three channels); electromyogram (EMG) of sub-mental muscles (one channel); ECG (one channel); EMG of the anterior tibialis muscle of both legs (two channels) and airflow (with a nasal/oral thermistor). Chest and abdominal effort (two channels) were recorded using Resp-Ez breathing belts, SLP, Tel Aviv); SaO_2 was measured (one channel) by pulse oximetry (Ohmeda 3700e, Boulder, CO) using a finger probe. Snoring and sounded bruxism episodes were recorded (one channel) by a microphone located above the patient's head at a distance of 1 m and connected to a sound level meter (SLM) (Quest Electronics-model 2700, Oconomowoc,WI). The output from the SLM was recorded in parallel on a calibrated (40–80 dB) chart recorder at a paper speed of 10 cm/h. The recordings were carried out at a paper speed of 10 mm/s and sleep was scored according to the standard criteria of Rechtschaffen and Kales [11]. Obstructive apnoea was defined as an episode of complete cessation of breathing of 10 s or longer with continuing inspiratory effort. Hypopneas were considered as such if a partial breathing cessation (more than 20% reduction in oral/nasal airflow compared with the level of the previous five breaths) occurred accompanied either by a drop of SaO_2 of at least 3% or by an arousal event. Apnea index (AI) and respiratory disturbance index (RDI) were calculated as the number of apneas/sleep hour and the number of apneas + hypopneas/sleep hour, respectively. Arousals were scored according to accepted criteria [12].

Following an adaptation trial of about 15–20 min, manual CPAP titration was carried out during the second PSG recording. The scoring of sleep bruxism episodes was done manually according the criteria used by Ware and Rugh [13]: (A) The EMG artifact on the EEG or the EMG was $>75\ \mu V$ or twice the background activity; (B) The phasic events were repetitive with two or more discrete bursts; and (C) The phasic events lasted >0.25 s each.

Table 1
Comparison between polysomnographic (PSG) data without (PSG 1) and with CPAP (PSG 2)

Parameters	PSG 1	PSG 2 with CPAP
Maximum snoring loudness on back (dB)	71	Did not snore
Maximum snoring loudness on right side (dB)	66	Did not snore
Maximum snoring loudness on left side (dB)	72	Did not snore
Respiratory disturbance index	47.6	4.1
Supine sleep time (min/% of TST)	231.5/56%	147.5/35%
Lateral sleep time (min)	179.5	274.5
Supine respiratory disturbance index	55.7	2.8
Lateral respiratory disturbance index	37.1	4.8
PLM arousal index	4.1	7.3
Number of audible bruxism events	73	0
Number of arousals per sleep hour	62.7	17.5
REM sleep minimal SaO_2	86%	95%
Non-REM sleep minimal SaO_2	89%	95%

3. Results

The diagnostic PSG recording showed that the patient suffered from severe OSA (RDI = 47.6). Most of the breathing abnormalities were hypopneas (310 vs. 11 apneas). In this patient only sounded bruxism events were observed (see Fig. 1). Table 1 summarizes polysomnographic data of both PSG recordings.

3.1. Characteristics of bruxism events

Bruxism events appeared in the supine and lateral posture. The total bruxism time was 55 min. The bruxism events induced by apnea/hypopnea were 67 compared with only six spontaneous events. The average duration of bruxism events was 9.5 and 11 s for the supine and lateral postures. The bruxism indexes for both positions were 6.5 and 16.0, respectively. Most of the bruxism events had phasic characteristics (as observed in Fig. 1) ranging from 3 to 19 clenching/bruxism event and 92.5% were accompanied by a leg movement.

During the CPAP treatment, snoring was eliminated and only a few hypopneas were seen. A complete disappearance of all bruxism events occurred during the CPAP treatment night. The optimal CPAP level was 9.0 cm H_2O.

4. Discussion

The results of this study indicate that CPAP treatment eliminated sleep bruxism episodes in a patient who had both severe sleep bruxism and severe OSA. These results are not entirely surprising; the patient had most of his sleep bruxism episodes in parallel with the arousal response that characterizes the termination of most breathing abnormalities during sleep. Since CPAP is an effective therapy for obstructive apneas and hypopneas, the elimination of sleep bruxism episodes by CPAP is not an unexpected result. Thus, this report provides support to the notion that sleep abnormalities producing an important fragmentation of sleep (like OSA or PLM), and as a consequence an increase of arousals during sleep, may induce the appearance of an arousal phenomenon like sleep-related bruxism. Nevertheless, several authors have not found a clear association between the termination of apnea events and the occurrence of bruxism episodes [5,6]. Sjoholm et al. [5] found that sleep bruxism was diagnosed in six out of 11 (54%) mild OSA patients, but only in four out of 10 (40%) moderate OSA patients. According to their data, masseter activity was more related to general motor arousals than to the termination of apnea episodes. However, arousals of short duration, which are the most common type of arousal in OSA patients, were not analyzed in this study.

Two limitations of this study should be mentioned. During PSG recording, we had only one submental EMG channel, which is not considered sufficient for adequate scoring of sleep bruxism episodes [14]. However, since all bruxism episodes were clear sounded events, (see the figure) this limitation did not affect our results. And, although the results of this report provide clear-cut data, they relate only to one patient. More research with this type of patient is needed to corroborate these findings.

Recently, it was found that among the associated sleep symptoms and disorders OSA was the highest risk factor for tooth grinding during sleep [7]. Since the presence of sleep bruxism could represent a contraindication for the use of an oral appliance in the treatment of OSA [15], and CPAP being an effective therapy for OSA patients with or without bruxism, further emphasis should be placed on the need for dental professionals to be made aware of this relationship.

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