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Original Article

Companion of oral movements with limb movements in patients with sleep bruxism: preliminary findings

Yangping Zhang [a, b, 1], Junlan Lu [a, b, 1], Zilong Wang [c], Zhijun Zhong [b], Miao Xu [b], Xueliang Zou [a], Bin Yu [a, **], Dongyuan Yao [a, b, *]

[a] Jiangxi Mental Hospital, Nanchang University, Jiangxi, PR China
[b] School of Pharmaceutical Sciences, Nanchang University, Jiangxi, PR China
[c] Queen Mary College, Nanchang University, Jiangxi, PR China

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ABSTRACT

Background: Sleep bruxism (SB) patients show a higher incidence of leg movements than normal subjects.
Study objectives: The study aimed to characterize SB episodes and their relationships with limb movements (LMs).
Materials and methods: Polysomnographic (PSG) recordings were performed on eight SB patients. The intervals between the onsets of adjacent SB episodes and LMs were determined and linear correlation analyses were used to estimate the relationship between the SB index and SB episodes in clusters. The Pearson χ^2 and partitions of χ^2 tests were used to analyze the differences in incidence of SB episodes and clusters in different sleep stages.
Results: A majority of SB episodes (85.05%) were found to be accompanied by LMs and among them, 70.52% SB episodes occurred with movements of both upper and lower limbs and most of LMs (70.54%) occurred before the onset of SB episodes. Most of SB episodes especially those accompanied by LMs occurred with microarousals or awakenings. Linear correlation analysis showed a positive correlation between the SB index and SB episodes in clusters ($r^2 = 0.7027$, $P = 0.0093$). In addition, the percentage of SB episodes in clusters accompanied by LMs was significantly smaller than that of SB episodes not accompanied by LMs (χ^2 test, $P < 0.001$) and the percentage of SB episodes in clusters during REM sleep was significantly smaller than that during NREM sleep (χ^2 test, $P < 0.0001$).
Conclusions: Most SB episodes might not be isolated events, but rather a part of a series of movements second to changes in arousal level.

1. Introduction

Sleep bruxism (SB), a stereotyped movement disorder, is characterized by rhythmic masticatory muscle activity (RMMA) and associated with jaw clenching and tooth grinding [1]. It happens in about 14%–20% children [2] and 8% adults (younger than 60 years old). Polysomnographic studies have shown that SB episodes mainly occur during light non-rapid eye movement sleep [2–4]. SB may lead to tooth destruction, temporomandibular disorders, locked jaw, and temporal headache [5–9]. The pathophysiologic mechanisms of SB are not fully elaborated, but psychological (eg, stress, anxiety, and hypervigilance), familial, and environmental factors may be involved [10–12]. The neurotransmitters such as noradrenalin, dopamine, histamine, and 5-hydroxytryptamine might play a significant role in primary SB [10]. Although drugs such as clonazepam [13,14], clonidine [15] and botulinum toxin [16,17], oral appliance, cognitive–behavioral [18] therapy (CBT), biofeedback [19], and contingent electrical stimulation [20–22] are currently used for the treatment of SB, the results are not satisfactory. Therefore, more and better understandings of pathophysiologic mechanisms of SB are required.

SB episodes have been associated with changes in electroencephalographic (EEG) activities, heart rate (HR), and respiration

* Corresponding author. School of Pharmaceutical Sciences, Nanchang University, 461 Bayi Road, Nanchang, Jiangxi 330006, PR China.
** Corresponding author. Jiangxi Mental Hospital, Nanchang University, 43 Shangfang Road, Nanchang, Jiangxi 330029, PR China.
E-mail addresses: jxyb650321@126.com (B. Yu), yao9000@gmail.com (D. Yao).
[1] Co-first author.

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[4,24–26]. Relevant studies showed that sympathetic activity increased about 4–8 min prior to the onset of SB episodes, and then a change in cortical EEG activities was found about 4 s before the onset of SB episodes, followed by an increase in HR and suprahyoid muscle activity before the onset of SB episodes [4,23,24]. Similarly, prior to and during SB episodes, significant changes in respiration pattern and frequency were also observed [25]. These highlight increases in brain activities before and during SB episodes. It is possible that the increased brain activities before and during SB episodes may not be limited to the central nerve system (CNS) that controls oral movements (eg, cortical masticatory area) and may also include other parts of CNS that control LMs. Indeed, a previous study demonstrated that SB subjects had significantly more body movements during sleep, especially the movements lasting less than 5 s (ie, jerks, twitches, or any sudden brusque LMs) than normal controls [26]. In addition, it has also been reported that patients with SB showed a higher periodic leg movements (PLM) index than normal subjects, and within the tested SB patients, the combined SB/PLM index was significantly higher than the isolated SB index [27]. Moreover, a Canadian pioneer research reported that 14.5–17.3% of the subjects who were diagnosed with restless leg syndrome (RLS) also suffered from tooth grinding; conversely, 9.6–10.9% of the patients with SB also had RLS-related symptoms [28]. Moreover, it has been reported that about 60% of the bruxing events were associated with leg movements [29]. All of these elements/discoveries suggest that there is a close relationship between SB episodes and limb movements. Therefore, the aim of this study was to characterize SB episodes in relation to LMs.

2. Methods

2.1. Subjects

Eight SB subjects [four males and four females: mean (±SEM) age, 22.12 ± 0.61 years; age range: 19–24 years] were recruited for the study (Table 1). None had any psychological (eg, depression or anxiety) or medical (eg, neurological or sleep) disorders, nor were they taking any medication four weeks before and during their participation in the study [4]. All SB subjects were selected according to previously published criteria [1,30]. Briefly, the SB volunteers were chosen based on the following clinical presentations: 1) tooth grinding at least three days per week during the last six months, 2) complaints of facial muscle pain, weakness or other discomforts in the morning, or 3) clinical presence of protective oral appliance (ie, tooth wear), and 4) masseter and/or temporalis muscle hypertrophy upon clenching voluntarily [1,30]. SB was confirmed in the lab if one met the following evaluation criteria: 1) two or more episodes of grinding with noise per night, 2) more than four RMMA episodes per hour of sleep, and 3) more than 25 bursts of masseter EMG activity per hour of sleep [1–3,31]. When two or more of these three criteria for SB diagnosis were met, the subjects were diagnosed with moderate-to-high SB. If the subjects had a history of SB but only met one of the three criteria [2,30], the subjects were considered to have mild-to-low SB. The protocols and experimental procedures were reviewed and approved by the Jiangxi Mental Hospital Research Ethnic Committee. All subjects were provided with informed consent before experiments and they could withdraw from the study anytime.

Table 1
Demographic and sleep data for the study population.

	Mean ± SEM
Sex	
Male	4
Female	4
Age, yr	22.12 ± 0.61
Sleep variables	
Total sleep time, min	398.25 ± 29.74
Sleep efficacy, %	81.61 ± 6.41
Microarousal index, events/h	7.35 ± 1.63
Apnea–hypopnea index, events/h	2.03 ± 1.25
SB index, events/h	9.20 ± 1.80
PLMI	5.66 ± 2.86
Sleep stage (%)	
N1	7.93 ± 1.12
N2	38.49 ± 3.02
N3	33.91 ± 2.93
REM sleep	19.61 ± 2.96

2.2. Polysomnographic sleep recordings

Polysomnographic recordings were performed on the subjects for two consecutive nights in the sleep laboratory at the Jiangxi Mental Hospital. The first night was used for the subjects to habituate the sleep laboratory and to rule out other sleep disorders including sleep apnea (sleep apnea index > 5 per hour of sleep), periodic leg movements (PLMs, >5 per hour of sleep), and rapid eye movement (REM) sleep behavioral disorders [32,33]. Data from the second night were used for SB diagnosis according to the criteria described above [4,34] and for other analyses described in the study. Polysomnographic recordings of EEG (F3-A2, F4-A1, C3-A2, C4-A1, O1-A2, O2-A1), electrooculographic (EOG), electrocardiographic (ECG) activities, and electromyographic (EMG) activities from masseter and mylohyoid muscle, bilateral extensor and flexor carpi radialis [35] as well as bilateral tibialis anterior muscle and gastrocnemius were performed. Moreover, nasal air flow, thoracic and abdominal respiration, body posture, and peripheral capillary oxygen saturation in thumb or index finger (SpO_2) were also recorded [36,37]. All signals were amplified, recorded at a sampling rate of 256 Hz, and stored for off-line analyses by means of Compumedics Net Beacon Application (Pro Fusion PSG 3 Software, Compumedics Limited, Abbotsford, Australia) and Spike 2 (CED, Cambridge, UK). For the EMG amplification, the high cut-off filter was set at 0.3 Hz and the low-cut off filter at 100 Hz to remove frequency known in cardiac, respiratory, or brain electrical activities [38]. Audio and video recordings from the orofacial area and other parts of the body were analyzed simultaneously to exclude nonspecific oral activities and confirm the presence of tooth grinding and/or LMs [34,39,40].

2.3. Data scoring

All the sleep stages were scored and analyzed according to the criteria established by the American Association of Sleep Medicine (AASM) [1]. SB episodes were classified as phasic (briefly repeated contractions of masseter muscle with at least three consecutive EMG burst of 0.25–2.0 s duration), tonic (contractions of masseter muscle lasting > 2.0 s) and combined (variation of tonic and phasic episodes) [41]. SB episodes occurred either in isolation or in clusters. A SB episode was considered to occur in clusters when it was preceded within 40 s or followed within 60 s by another SB episode as described by Huynh et al. [4]. In contrast, a SB episode was considered to occur in isolation when no SB episode happened 60 s before and after the defined isolated SB episode.

All data were first analyzed with the Pro Fusion PSG 3 Software and then converted into the European data format (EDF) for further analysis with Spike 2 (Version 8.07). The onset of SB-related EMG activities of masseter muscle was determined when the EMG activities increased above two times of the background activities and the onset of LMs was defined when the amplitude of the LM associated EMG activities was 8 μV above the background and the