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Bruxism Relieved Under CPAP Treatment in a Patient With OSA Syndrome

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Bruxism is a heterogeneous condition related to various underlying mechanisms, including the presence of OSA. This case report illustrates that sleep mandibular movement monitoring and analysis could provide a useful opportunity for detection of both sleep bruxism and respiratory effort. The current case suggests that tracking of respiratory effort could enable evaluation of bruxism and its potential interactions. Successful treatment of sleep-related respiratory effort may lead to improved or resolution of bruxism in cases where such a causal relationship does exist.

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KEY WORDS: bruxism; mandibular movements; sleep apnea

Sleep bruxism is a common condition which consists of occasional and involuntary repetitive jaw muscle activity with clenching or grinding of the teeth and/or with bracing or thrusting of the mandible.[1] Notably, not all patients with bruxism have a present complaint of teeth grinding. The cause is frequently unidentified or sometimes only suspected to be related to a sleep breathing disorder or a neurologic disease. Prevalence decreases from 14% to 20% in childhood to 3% to 8% in adults.[1]

Typically, bruxism is ascertained in the presence of a rhythmic masticatory muscle activity (RMMA) captured by surface electromyography (EMG) of the masseter and/or temporalis muscles. This electromyographic activity consists of jaw muscle contractions that are phasic (at least three EMG bursts lasting 0.25-2.0 s each) and/or tonic (sustained for $>$ 2 s) and/or a combination of phasic and tonic activity.[2] RMMA is actually the bruxism diagnostic marker which could be captured using a one-channel recording home device. However, the surface electromyographic device cannot identify associated breathing disturbances, such as apnea or periods of increased respiratory effort, requiring referral to a sleep medicine specialist.[3] We present, for the first time to our knowledge, sleep stereotypical pictures of mandibular motions recorded in a patient during in-laboratory polysomnography and diagnosed with bruxism in the course of a sleep apnea syndrome. The effect of nasal CPAP treatment on bruxism is disclosed.

Case Report

A 61-year-old woman reported sleep disruption related to snoring and excessive teeth grinding which also disturbed her husband. The problem had increased over several months and was accompanied by daytime fatigue being reported by the couple. The patient took no

ABBREVIATIONS: EMG = electromyography; MM = mandibular movement; NREM = non-rapid eye movement; RMMA = rhythmic masticatory muscle activity

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medications, but indicated that she had recently gained 6 kg in weight.

The patient's BMI was 29.8 kg/m^2. Oral examination revealed signs of tooth attrition. There were no craniofacial abnormalities, and the general physical examination was normal.

Conventional laboratory audio-video polysomnography was performed. Midsagittal, vertical, and horizontal mandibular movements (MMs) were recorded with an inertial measurement unit (Sunrise, Sunrise, Namur, Belgium) and synchronized with the surface electromyographic activity of the masseters. All recordings were in the dorsal position.

The Sunrise device is composed of a coin-sized inertial measurement unit that was attached by the sleep technician on the chin of the patient between the inferior labial sulcus and the pogonion. Its embedded inertial measurement unit enables MM sensing and communicates with a smartphone application. The collected MM data were automatically transferred to a cloud-based infrastructure at the end of the night for automated analysis with a dedicated machine-learning algorithm. In this case, we proceeded after clock synchronization to the reincorporation of the signal into the polysomnography regarding other variables. MM signal was integrated over time to display the movement of the mandible in tenths of millimeters and manually analyzed.

Our previous work has identified normal and abnormal patterns of MMs. The normal pattern of MMs is oscillations with a peak-to-peak amplitude $<$ 0.3 mm driven by the respiratory cycle. During respiratory effort, this amplitude increases to $>$ 0.3 mm, a finding which was validated by measurement of pulse transit time[4] or electromyographic activity of the diaphragm.[5] Cortical arousals are marked by large abrupt MMs.

Over a total sleep time of 526 min, there were 132 episodes of obstructive hypopnea associated with 59 sustained periods of respiratory effort (mean duration, 258 s), terminating in cortical arousals (respiratory effort related arousal [RERA]). Polysomnographic scoring (sleep stages and respiratory events) was performed by two trained technicians who were blinded to the study aims and in strict accordance with the American Academy of Sleep Medicine rules.[6] Hypopnea was defined as a reduction in nasal pressure signal (flow) of $>$ 10 s ended by an arousal or a decrease in oxygen saturation percent of at least 3% relative to baseline. The respiratory disturbance hourly index was 22.1. The rapid eye movement/non-rapid eye movement respiratory disturbance hourly index ratio was 0.17. The associated oxygen desaturations were more pronounced during rapid eye movement sleep with an oxygen saturation percent nadir of 80%.

A total of 101 episodes of bruxism were identified, and most (95.5%) were preceded by periods of respiratory effort (in the form of obstructive hypopnea or RERAs, 62 during stage 1 or 2 NREM, and 13 during rapid eye movement sleep). All episodes of bruxism were terminated by arousals, and 26 led to full awakenings. No evidence of bruxism was observed during stage 3 NREM. Bruxism episodes lasted from 2 to 34.4 s and consisted of phasic or tonic-phasic electromyographic bursts of both masseters. An example of a polysomnographic sequence containing periods of obstructive hypopneas each followed by episodes of bruxism is depicted in Figure 1. The following three successive MM patterns were identified: (1) MM linked to respiratory movements (at a frequency of 0.35 Hz), (2) a sharp and large MM closing the mouth on arousal and accompanied by airflow restoration, and (3) MM at a higher frequency (0.8 Hz) and larger amplitude during the episode of bruxism. The former pattern of bruxism on MMs was accompanied by clear RMMA of both masseters; their power spectral density analysis (Fig 1, markers A and B) showed different distributions compared with EMG of the lower limb (Fig 1, marker C).

The patient was started on auto-CPAP via a nasal pillow mask. The dedicated software showed complete normalization of apnea hypopnea index after 1 week. The patient's husband confirmed the absence of any noise or grinding while the patient was receiving CPAP treatment over the week. His subjective report was verified and filmed during a split night in the sleep laboratory. No bruxism episodes were observed during 95 min of sleep under CPAP at the fixed target pressure of 7 cm H_2O (Fig 2). In contrast, during the next 227 min of sleep, when CPAP was removed, bruxism recurred at the end of 44 periods of respiratory effort.

The final diagnosis was a noisy bruxism triggered by obstructive sleep hypopneas and RERAs.

Discussion

This case illustrates that bruxism is closely associated with OSA. In addition, we report, for the first time, that the simple monitoring of MM is able to clearly identify not only RERA and OSA, but also bruxism.

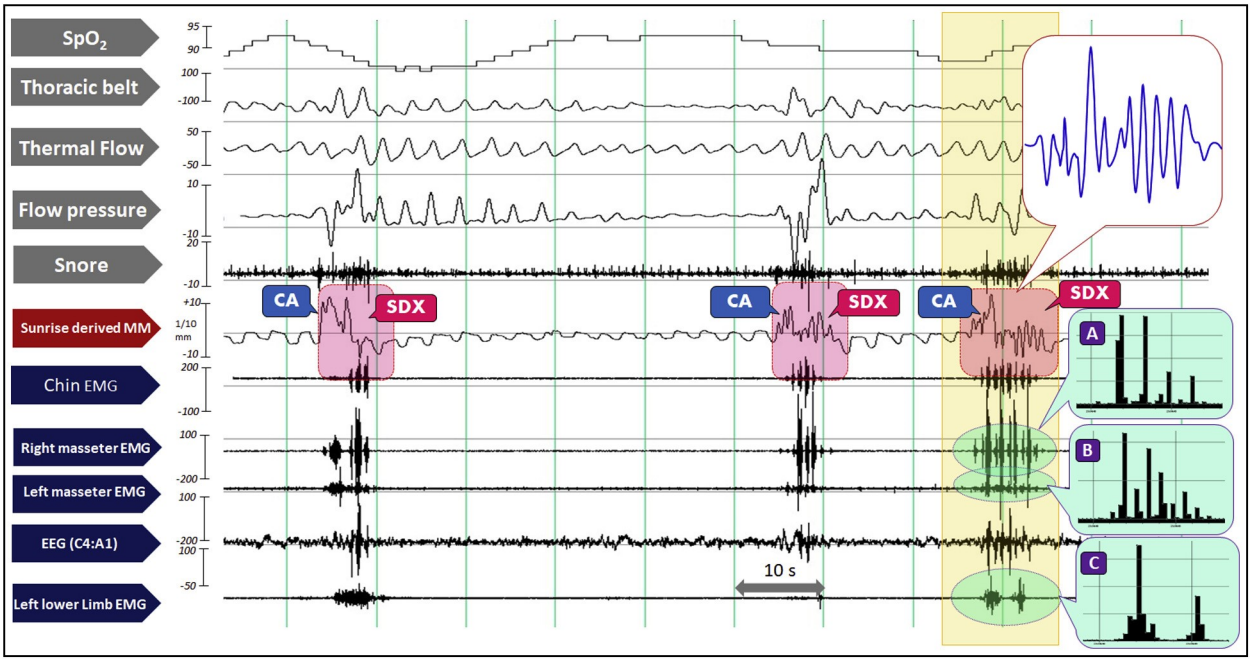

Figure 1 – *Episodes of bruxism preceded by obstructive hypopneas. The signal provided by the inertial unit is highlighted on the third episode of bruxism, regarding the spectrograms originated from the masseters (A, B) and the left lower limb surface EMG scans (C). CA = cortical arousal; EMG = electromyography; MM = mandibular movement; SDX = bruxism episode; Spo_2 = oxygen saturation percent.*

The potential causal relationships between sleep disordered breathing and bruxism are still being debated in the literature without any firm conclusions.[7-9] The temporal association between typical masseteric electromyographic activity and apnea or hypopnea is well known, sometimes leading to the conclusion that the respiratory event followed the episode of bruxism.[8] Until now, essential elements for the diagnosis of bruxism were reports of teeth grinding, and evidence of tooth wear, but definitive confirmation requires

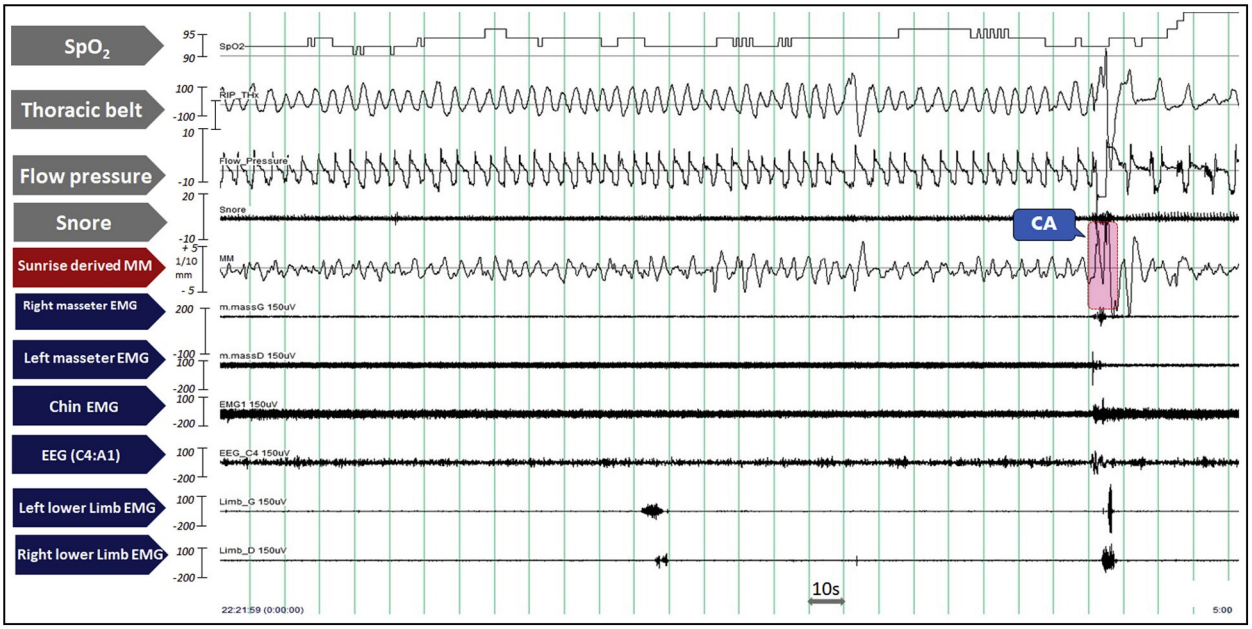

Figure 2 – *Effect of nasal CPAP on the risk of bruxism occurrence during a 5-min polysomnographic fragment. The mandible moved at the breathing frequency spanning peak-to-peak amplitudes with a mean two-tenths of a millimeter under the continuous nasal pressure of 7 mbar. No rhythmic masticatory muscle activity was recorded after the CA depicted. See Figure 1 legend for expansion of abbreviations.*

laboratory polysomnography including surface EMG of the masseter and temporalis muscles, and an audio-visual recording system and polysomnography that will also evaluate for concurrent sleep disorders. However, because most sleep studies do not include this specific montage, detection of bruxism is not necessarily straightforward during conventional polysomnography.

Bruxism in this observation was defined as a clinical situation where there were reports of recurrent annoying teeth grinding during sleep and related dental enamel damaging. Reports were confirmed by audio-video in-laboratory polysomnographic recording, and concomitant RMMA was demonstrated during bilateral masseteric electromyographic phasic activities. Therefore, the entire clinical picture was fully documented.

In stage 3 NREM, breathing disturbances were limited to periods of subtle increases in respiratory effort (RERAs) without episodes of apneas or hypopneas. Stage 3 NREM is associated with lower upper airway collapsibility explained by a protective shift in physiological traits with lower arousability and better upper airway muscle responsiveness.[10]

Remarkably, recording of sleep MMs and pattern analysis identified the temporal sequences evidenced by increases in respiratory efforts and followed by typical periods of bruxism activity. During the 2-min period of stage 2 NREM shown in Figure 1, three cortical arousals are depicted, each ending an obstructive hypopnea, and each accompanied by an episode of bruxism. Before cortical arousals, the mandible displaced at the breathing frequency with a peak-to-peak amplitude $>$ 0.3 mm. This is known to typically indicate an underlying exaggerated respiratory effort.[4,5] The spectral analysis comparison between masseteric and limb EMG during the episode of bruxism shows that contractions of jaw muscles are not nonspecific motor phenomena in response to arousals.[9] By closing the mouth and stretching the upper airways muscles, mandibular motion contributes to airflow restoration.[11] Furthermore, removing obstructive hypopneas and RERAs with nasal continuous positive pressure therapy was accompanied by the disappearance of bruxism as readily identified with MM signals; CPAP withdrawal led to recurrence of bruxism and of the hypopneas.

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