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Sleep bruxism can be a harbinger of sleep disordered breathing

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Abstract

Background & Objective: The mechanism of sleep bruxism (SB) remains an enigma to this day. The saga of SB was kept alive by dental surgeon's relentless research. Then the sleep physicians stepped in with their polysomnogram (PSG), having a hunch that the answer was in sleep disordered breathing (SDB). In the face of uncertainty and conflicting reports in the relationship between SDB and SDB that followed, we made an alternative approach to investigate the pathogenesis of SB, using CPAP as an experimental tool. The objective was to substantiate that SB can occur during the arousal phase of SDB. *Methods:* This is a PSG study of SB in 20 consecutive cases of SDB. First night PSG was diagnostic and followed by the second night PSG for Continuous Positive Airway Pressure (CPAP) titration for treatment. We studied every SDB wave and observed its relationship with arousal and SB. An experiment was conducted on the second night, where we used CPAP as a tool, to stent upper airway and toggle the pressure to cause apnea and await the aftermath. *Results:* Diagnostic PSG of all 20 patients showed a common sequence of events: SDB is followed by brain arousal which is followed by SB, there being an almost one to one relationship between SB and SDB. On the second night's PSG, toggling CPAP down or up causes SDB to come on or off, respectively. SB followed suit along with arousal.
Conclusion: SB commonly occurs during arousal from SDB. CPAP gets rid of SDB and SB.

Keywords: Sleep disordered breathing, sleep bruxism, continuous positive airway pressure

INTRODUCTION

Sleep bruxism (SB) is tooth clenching or grinding that can end up in tooth destruction. The masseter muscle electromyogram (EMG) activity is the hallmark of SB on polysomnogram (PSG). SB usually presents in dental clinics. The dentists are usually pre-occupied with the destruction of the teeth and do not see the concealed sleep disordered breathing (SDB) which is a public health burden. Ninety one percent SDB subjects remain undiagnosed and untreated in Singapore.[1]

A relationship between SB and SDB has received much attention after a population survey[2] in 2001 and a PSG study[3] in 2014. These studies showed that Obstructive Sleeping Apnea (OSA) is a risk factor to SB. However, studies that followed contradicted this. The investigators did not show a clear statistical correlation.[4] These studies used insufficient research methods and different published standard hypopnea definitions. These differences caused SDB counts to be underestimated.[5] However, SB prevalence reported remains consistent over time. Amidst the confusion, an evidence based review was published by experts on this subject.[6] They concluded: "Currently, there is no evidence to support the association or causality of SB and OSA". All these papers on this subject failed to show an association between OSA and SB.

When SB occurs in the presence of a neurological cause it is termed 'secondary'. It is termed 'primary' is if no clear cause was found. Diseases in the basal ganglia.[7,8] paroxysmal disorders like REM behaviour disorder (RBD) and epilepsy are uncommon secondary causes of SB.

During routine PSG services in the hospital, we have often observed that SB can occur during the arousal phase of SDB. This motivated us to conduct this study to substantiate the relationship between SB and SDB. We also hypothesized that continuous positive airway Pressure (CPAP) works to open up the upper airway in SDB, which then relieves SB.

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METHODS

We studied 20 consecutive cases of SDB. These clinically suspected SDB cases were studied with overnight PSG with audio-video recording in our Sleep Laboratory. A sleep technician and sleep physician were in attendance. We used state of the art recording and scoring techniques and inclusive hypopnea counting methods.

We paid special attention to observe intricates of all PSG wave forms of SDB, arousals and SB. SDB and SB were scored in accordance with the standards set by the American Academy of Sleep Medicine.[9] We detected respiratory effort by measuring airflow through nasal pressure signals. Muscle arousal and Electroencephalographic (EEG) arousals were routinely assessed. Pulse transit time (PTT) double checked arousal especially the autonomic arousals. SB was assessed by masseter muscle EMG. SB may have also been audible by the audio recording or by the technician. We did not differentiate between rhythmic activity and tonic activity in the masseter EMG, as the limit of the bite was not determined by the muscle, but by the teeth themselves.[10]

We repeated the PSG on the following night for CPAP titration as it was the treatment of choice. CPAP stented the upper airways of patients with SDB and then we toggled the pressure in increments up or down to take away or bring back the apnoea and looked for the SB events.

Thus CPAP titration was used as a double purpose tool. It gave the CPAP pressure as prescribed treatment to the patient. On the other hand, it provided data for the experiment. It charted SDB and SB events against the toggling air way pressure. Titration would start at a minimum of 4cm water pressure, and adjusted to eliminate all SDB. The technician set the minimum air pressure to effectively stop all SDB without compromising patient comfort.

RESULTS

The age range of the 20 study subjects were 16 to 70 years. There were 13 males and 7 females. All 20 patients have SDB on PSG. Sixteen patients had obstructive sleep apnoea-hypopnea, with AHI ranging from 9-74. Four patients had a diagnosis of upper airway resistance syndrome (UARS). Audible teeth grinding was heard in 15 patients. Amongst these 20 patients, 2 had additional sleep disorders, 1 had RBD and another had narcolepsy.

PSG of all 20 patients showed a common sequence of events: SDB was followed by EEG/ Autonomic arousal which then was followed by SB and leg muscle arousal. This is shown in Figure 1. In all patients, there was mostly one-to-one relationship between SDB and SB.

On the second night, all the SDB and SB disappeared with increased CPAP pressure; reducing the CPAP pressure would again bring back SB and arousal. As long as there was no SDB, there was then no SB. We see this in Figure 2 where all SDB and SB events have disappeared. CPAP pressure to open and close the air way are not necessarily the same. In each whole night hypnogram, about 3 SB events did not have an associated SDB event.

Figure 3 depicts a snapshot of PSG of 1 of 4 patients with UARS. On PSG, there are no obvious apnea preceding SB, only flow limitation. As such, there was no oxygen desaturation. With CPAP, we achieved normal sinusoidal air flow and all SB events were removed.

Another case is where SB is from RBD. Figure 4 shows the PSG of a 60-year-old male patient who had symptoms of enacting his dreams and daytime sleepiness. He had no audible SB. On PSG he had severe SDB (AHI 74/hr). Only after CPAP was administered and all SDB was eliminated did he show masseter activity of SB, along with movement of toes, hands, legs during REM sleep. He was enacting his dream at the time.

DISCUSSION

In this study of 20 patients with SDB, we have observed SB to be part of the arousal during SDB. This is reversed when CPAP was administered to relieve the SDB event. This shows that SB commonly occurs during arousals from SDB.

Previous researches were based on statistically significant association between indices of SDB and SB. However, the whole spectrum of SDB was not known during the studies. Therefore, there were missed scoring of SDB and brain arousal on PSG, thus underestimating SDB counts. Researchers were restricted by definitions and counting rules that were in a flux. This caused a situation where as much as 49% of hypopneas could have been lost between different counting rules set by American Academy of Sleep Medicine (AASM).[5,9] Not all SDB have recognisable EEG arousal[11]. Subcortical arousal detection would need added techniques to detect, such as Pulse transit time (PTT).[12] These were not available until more recently. The total of *missed cases of apnea* is shown by the pick-up rate of SDB in prevalence studies during these periods. It increased[13] from 2.7% in the early 1980s to 21% in 1999.[14] The current prevalence of SDB[15] has now gone well over 60%. Our sleep studies had

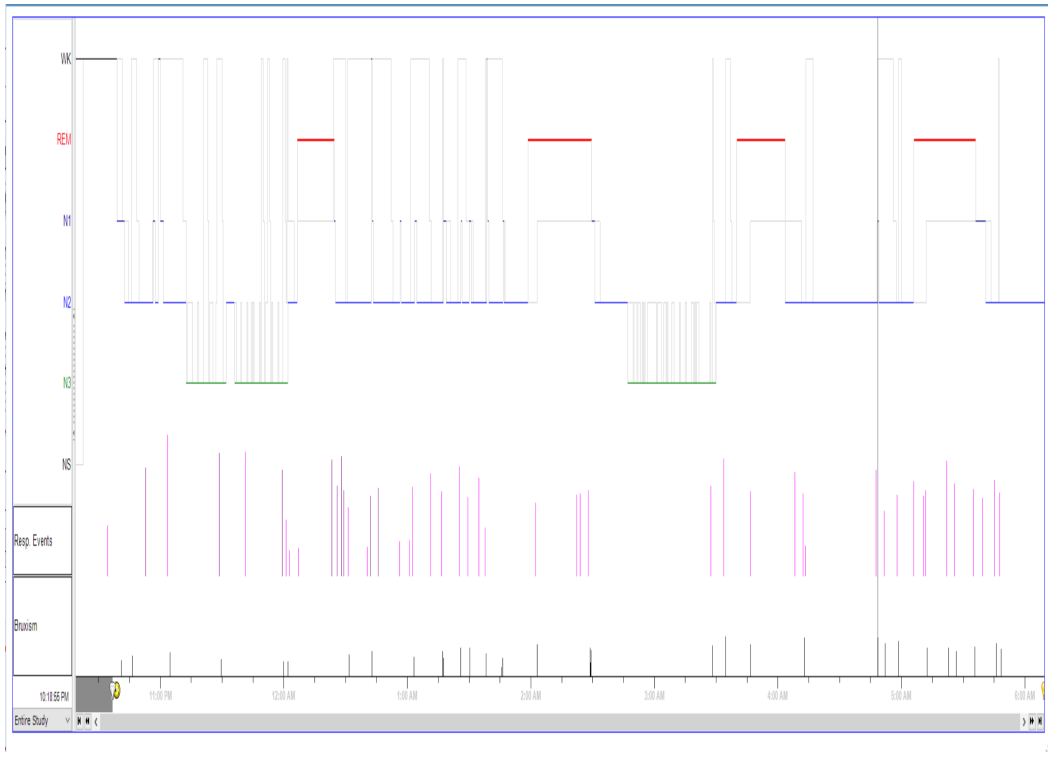

Figure 1. Whole night hypnogram of one of the patients. The vertical lines mark an event. Pink and red vertical lines are the same, depicting SDB events; Black vertical lines seen at the very bottom depicts SB events.

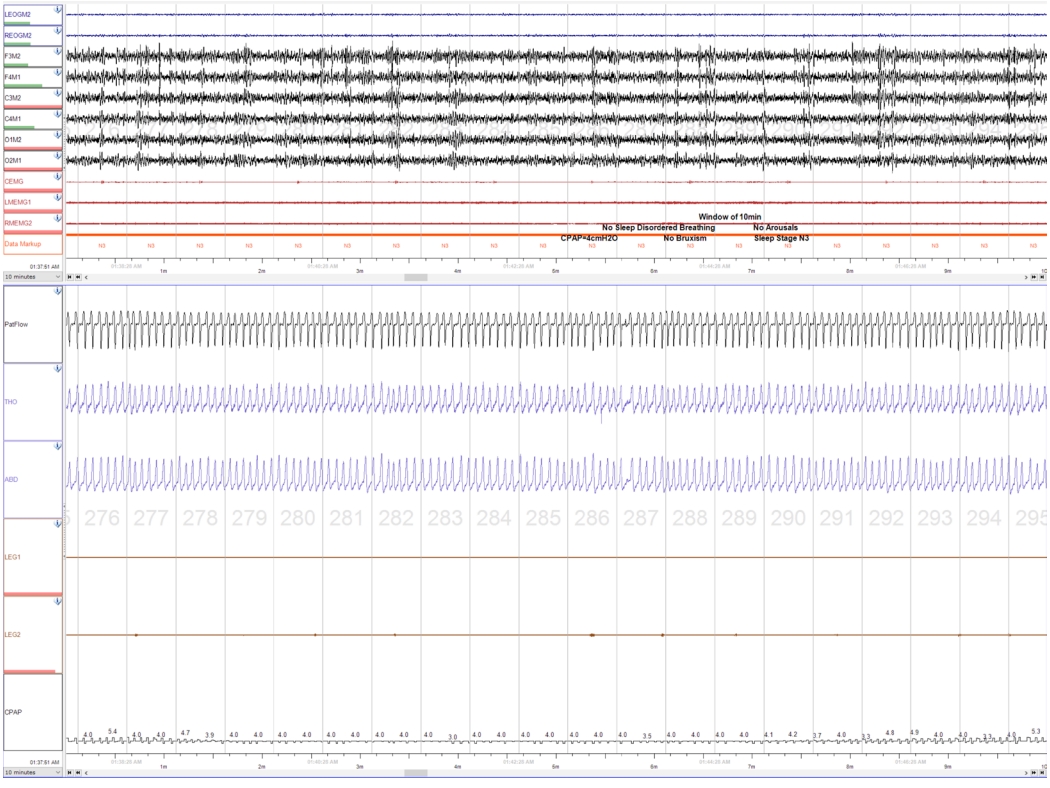

Figure 2. Same patient's CPAP titration hypnogram. Both SB and SDB disappeared at this pressure setting of 6cm water pressure.

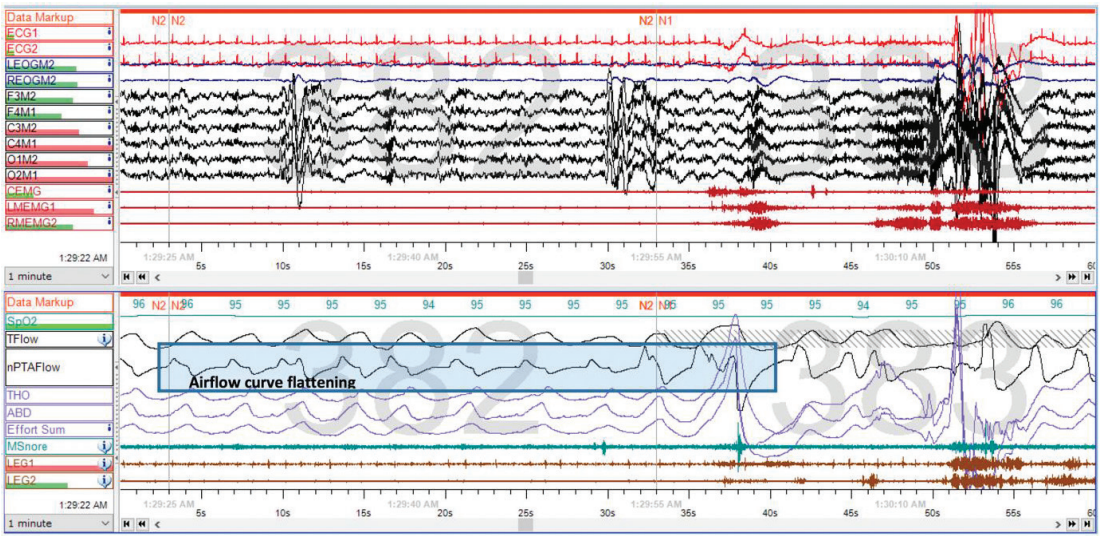

Figure 3. A 60 second PSG window from one of the patients showing UARS on PSG.
This PSG snapshot shows flow limitation (channel 12-13), followed by arousals in EEG, and in masseter muscle activity which is SB (channels 9-10). Channel 19-20 shows leg muscle EMG arousal at the same time. Respiratory effort related arousals (RERA) is seen in EEG and in all muscles monitored. There is no significant desaturation. (AHI<5/hour, RDI*>5/hour, Oxygen saturation>92%)
*Respiratory Disturbance Index

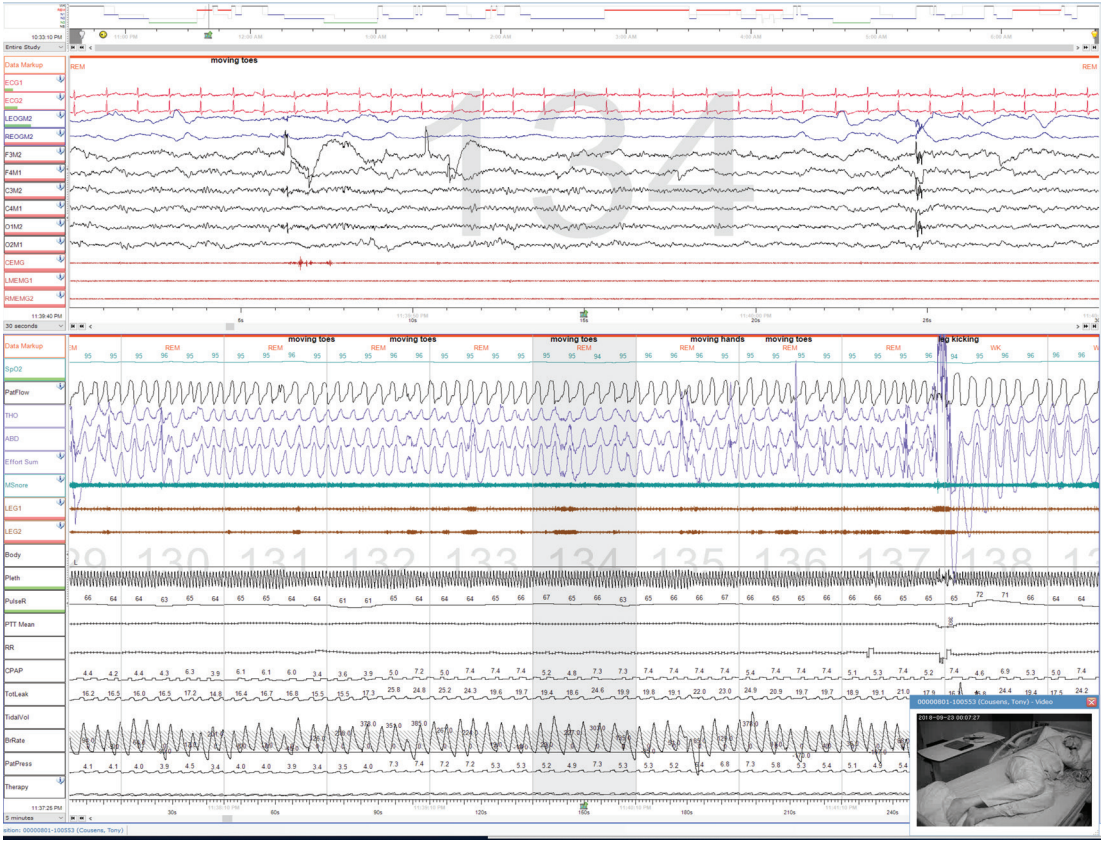

Figure 4. OSA with RBD. OSA removed completely with CPAP. Only now RBD is seen on video and hypnogram.
Note: This is a snapshot during REM stage of sleep. There are no apnoea seen, however there is muscle activity in masseters and leg muscles. Patient face has been edited out.

the advantage of more advanced technologies and more inclusive scoring of SDB. We were thus well-armed to identifying all range of SDB from apneas to UARS. When there was no EEG arousal, we picked up autonomic arousals with PTT and could see the association between SB and SDB.

In our paper, there are 2 scenarios where '*missed cases'* occurred in relationship to SB. Firstly, the cases of UARS where an apnea is not obvious as it is a flow limitation. Secondly, when SB occurs without SDB being the cause. This was in the RBD patient (Figure 4).

CPAP pressure to open and close the air way are not necessarily the same. This has bearing when setting the optimum CPAP pressure as treatment. "Down" titration is recommended. Pressure at which SDB reappears on down titration is lower due to the hysteresis phenomenon. This allows for a pressure that eliminates all SDB events while being a comfortable pressure for the patient. The phenomenon of hysteresis is the lagging of an effect behind its cause. It's effect extends from physics to medicine and many more.

In conclusion, our study has shown that SB occurs during arousal from SDB. It is not a disease. Rather it is the final common pathway to SDB. SB is not entirely due to SDB. Paroxysmal disorders like RBD can mimic masseter muscle activity on PSG. Clinicians should look out for SDB in patients complaining of SB because SB can be a harbinger of SDB.

DISCLOSURE

Conflict of interest: None.

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