Nocturnal Bruxism as a Protective Mechanism Against Obstructive Breathing During Sleep

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Introduction : Bruxism is defined as consisting of either a grinding movement of the teeth and / or clenching of the jaw. It occurs to some degree in almost everyone, but typically not to a degree that would cause symptoms of pain or destruction of the teeth. Many people with Bruxism tend to brux only while asleep or primarily while asleep. For the purposes of our study we defined patients with Nocturnal Bruxism as complaining of TMJ discomfort on awakening or demonstrating grinding or clenching during sleep as witnessed by a bed partner. It has been previously recognized that Bruxism can occur in many patient with OSA. In many of these OSA patients the Bruxism can occur to the degree that results in chronic pain and referred to as TMJ (or TMD for Temporal Mandibular Dysfunction). Our clinical experience has demonstrated improvement in Bruxism following treatment with CPAP. We sought to identify the frequency of the relationship between Bruxism and Sleep Related Breathing disorders and the response in Bruxism by treating with CPAP.

Methods: We retrospectively reviewed 729 consecutive charts of patients diagnosed with clinically significant obstructive breathing during sleep (OSA or Upper Airway Resistance Syndrome (UARS)) and prescribed CPAP therapy at the Sadler Clinic Sleep Disorders Center at least six months prior to initiating our assessment. The population consisted of 296 females and 433 males, average age 51 yrs old (+/- 13.8). All patients were evaluated clinically at the sleep center. A patient was placed in the Bruxism group by either positive responses on a sleep questionnaire that included several questions assessing for nocturnal Bruxism (such as witnessed clenching or awakening with aching jaw pain), or from information obtained from the clinical history and exam. The main objective was to identify patients who presented with aspects of their history that suggested increased masseter muscle activity during sleep to a degree that would be considered abnormal. NPSG testing with performed on all patients who's charts were reviewed and the results of the NPSG demonstrated OSA (which included those with UARS). UARS was established by esophageal pressure monitoring using a water catheter technique. A follow up phone interview was performed and a questionnaire was utilized to assess outcome in patients with Bruxism placed on CPAP. On the follow-up questioning assessment was made of the patients Bruxism Symptoms as rates from No Improvement, Partial Improvement and Complete Improvement. The patients were also asked to estimate the frequency in which they use their CPAP mask as a nights per week with values ranging from 0 to 7. Other measures included Epworth Sleepiness Scale and tabulation of other therapies for treatment of OSA such as Mandibular Advancing Dental Appliances or Surgery.

Results :

Of the 729 patient charts reviewed:

183 demonstrated Bruxism by history (25.1% of the OSA population) 95 females and 88 males.
119 patients were successfully contacted for follow up questioning by phone. 17 of these patients were using a bite guard as part of their treatment.

Statistical assessment was performed on the remaining 102 patients. 75 stated using CPAP to some degree of which 60 reported nightly use (100% compliant or 7 nights per week) and 27 patients were not using CPAP at all.

Of the 60 patients using CPAP nightly 33 demonstrated improvement (55%) 11 sated partial and 16 stated complete improvement.

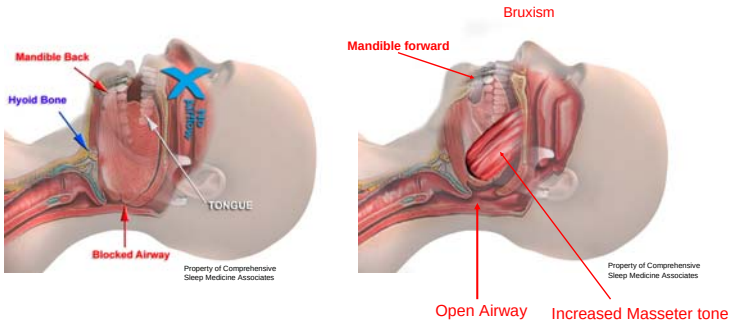
A multivariate analysis was performed to determine if there was a relationship between CPAP use and degree of improvement in Bruxism.

	No Improvement	Partial Improvement	Complete Improvement	F and P Values
Age	46.8±13.7	48.8±10.6	49.6±12.3	F(2,66)=0.43, p=0.67
AHI	23.6±23.5	21.5±15.6	22.4±31.3	F(2,66)=0.13, p=0.88
RDI	31.9±25.1	31.9±25.1	36.2±27.1	F(2,66)=0.16, p=0.85
CPAP (nights/wk)	4.4±3.1	6.4±1.2	6.0±2.1	F(2,66)=5.65, p=0.0054

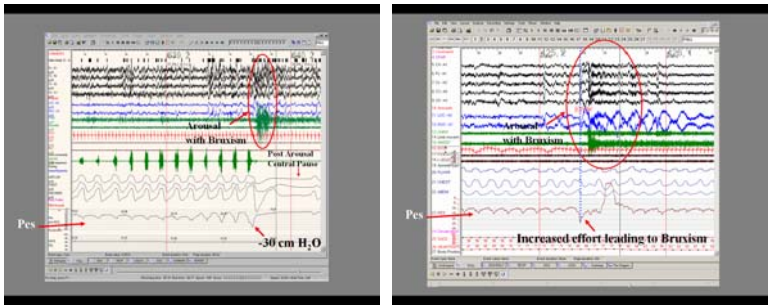
No differences were found between the groups with respect to Age, AHI, or RDI. There were a significant differences with respect to CPAP use as measured by Nights per week with a p=.0054:

Additional assessment of patients who were found to have UARS:

Of the 102 patients 25 had AHI < 6 but found to have the Upper Airway Resistance Syndrome with Effort related arousals identified by the Pes (pressure within the esophagus). 14 of the 25 used CPAP nightly of which 7 stated complete resolution of Bruxism and 5 stated partial improvement. Combined this represented 85.7% of the Bruxism group. Therefore, Bruxism patients with The Upper Airway Resistance Syndrome who used CPAP nightly demonstrated improvement in Bruxism 85.7 of the time by treatment with CPAP alone.



When most patients exhibit obstructive respirations during sleep the mandible falls back bringing the back of the tongue with it. This triggers a series of events that in some people results in a reflexive attempt to open up the airway by increasing masseter tone. This brings the mandible forward and in many patients improves respirations. Unfortunately over time this can lead to symptoms of pain in the TMJ and other problems such as morning headaches.



These are two examples of effort arousals associated with increased masseter muscle activity. The first example on the left clearly demonstrated snoring and a crescendo increase in effort leading to the arousal. The second example on the left is much more subtle. Without the Pes demonstrating the increased effort preceding the increase in masseter muscle activity, this event would not have been recognized as being associated with obstructive respirations.

Conclusion : We postulate that nocturnal Bruxism is a compensatory mechanism of the upper airway to help overcome upper airway obstruction by activation of the clenching muscles which results in bringing the mandible, and therefore the tongue, forward. We recognize that this process may reduce the obstruction and therefore increasing the need for adding Pes (Pressure in the esophagus) monitoring to NPSG testing. This is why we included patients with the UARS in our OSA group. After treating the airway with CPAP this protective mechanism is no longer needed and over time the Bruxism resolves. This study suggests such a compensatory mechanism is the etiological force behind nocturnal Bruxism in many patients. Additional assessment of this relationship is in progress and may result in earlier identification of those patients with sleep related obstructive breathing.

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