How Sleep Bruxism and Tension Headaches Affect the Masseter Inhibitory Reflex

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Received: March 14, 2017 Accepted: April 11, 2017 Published: April 18, 2017

Citation: Luco K (2017) How Sleep Bruxism and Tension Headaches Affect the Masseter Inhibitory Reflex. J Sleep Disor: Treat Care 6:3. doi: 10.4172/2325-9639.1000198

Abstract
Sleep bruxism (SB) is classified as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. In recent years, the inhibition of the masseter inhibitory reflex (MIR) has been shown to result in increased bite force during sleep bruxism events. Tension type headaches (TTH) are a hallmark symptom reported by sleep bruxism patients and affect the MIR very similarly to SB.

Keywords: Sleep Bruxism, Headaches, Masseter Inhibitory Reflex

Short Communication
Sleep bruxism (SB) is classified as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible [1]. In recent years, the inhibition of the masseter inhibitory reflex (MIR) has been shown to result in increased bite force during sleep bruxism events. Tension type headaches (TTH) are a hallmark symptom reported by sleep bruxism patients and affect the MIR very similarly to SB [1-5].

The masseter inhibitory reflex is a cranial reflex consisting of two distinct phases or silent periods termed exteroceptive suppressions (ES1 and ES2) [6]. This reflex is located in the trigeminal mesencephalic nucleus of the pons [7]. The first phase of the reflex, ES1, has its afferents located in the periodontal ligaments of the teeth as well as the mental nerve. The 1st inhibitory nucleus in the mesencephalic nucleus has only a single interneuron connecting it to the ipsilateral trigeminal motor nucleus (TMN), that controls the ipsilateral masseter and temporalis muscles. Normally, ES1 lasts 10-14ms (Figure 1). The 2nd phase, ES2, has its afferents located in the muscle spindle sensory organs located in the masseter and
temporalis muscles. These afferents descend down the lateral trigeminal tract to the second inhibitory interneuron. There are multiple interneurons for ES2 connecting it balaterally to both TMN. Normally, ES2 lasts 40-50 ms (Figure 2). The result of the activation of the MIR is suppression of the TMN and inhibition of contraction of the masseter and temporalis muscles. This protective reflex is normally inactive and is activated when chewing softer foods and encountering a hard object. The reflex activates, suppressing further contraction of the masseter and temporalis, preventing damage to the oral structures [8-12].

Figure 1: Exteroceptive suppression one.

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In sleep bruxism the ES2 phase and to a lesser extent, the ES1 phase are affected. The ES2 phase is shortened considerably or completely absent, whereas the ES1 phase is advanced slightly and shortened (Figure 2). There are reduced pain thresholds with TTH, relating to the decreased ES2 of the MIR. There is also excitability of the reticular nuclei which inhibit the medullary inhibitory internurons that mediate the ES2 phase of the MIR [2]. The EMG tracings in Figure 3 demonstrate the significant reduction or complete absence of ES2 in sleep bruxism [12].
The EMG tracing in Figure 4 compares the ES2 suppression in SB bruxism with the ES2 suppression in TTH. The resulting suppression of the MIR (Figure 4) in TTH is remarkably similar to the suppression of the MIR in SB, illuminating how SB and TTH are interrelated. This inhibition of the MIR in SB and TTH allows the masseter and temporolis muscles to contract with maximum uninhibited force resulting in the severe damage to teeth, muscles, TMJ and severe tension type headaches seen in SB and TTH [8,12-16].
Figure 4: EMG tracings of ES1 and ES2 with sleep bruxism and tension type headaches.

With oral appliance therapy for SB, the position of the bite is critical. If the bite on the device is placed in the molar region, the MIR will continue to be suppressed (with resulting excessive bite force) whereas if it is placed in the cuspid or incisal region, the MIR is reactivated and the bite force is limited to normal levels [17-20]. In the prototype stage of the Luco Hybrid OSA Appliance (Figure 5), these principles were implemented early on, achieving excellent results. The forward bite effectively reduced the symptoms of SB and TTH within 2 weeks in the majority of cases. This is attributed to the re-activation of the MIR with resulting control of bite force with the cuspid bite, confirming the previous research [10] and verified with EMG home sleep study recording.

This has significant implications in the treatment of obstructive sleep apnea (OSA) patients who also suffer from SB, as the two commonly occur together. The majority of OSA devices allow molar contact, which in SB maintains the suppression of the MIR. This often results in patient symptoms from SB and TTH. The FDA list the development of TMD as one of the known risks of mandibular advancement appliances in their guidance for these devices. The suppression of the MIR is likely the cause. The relationship between SB and TTH in suppression of the MIR is strong. Research has shown that, even in young patients [21-33], this suppression occurs, reinforcing the genetic cause [34].
Considering the MIR in the treatment of OSA and SB patients is critical. With a minimum of 8-10% of the population suffering from SB [1] and up to 95% suffering from primary headache [35] (which includes TTH) at some point in their life, this is a significant social problem. SB falls under dental treatment and the American Academy of Sleep Medicine classification of SB [1], states that a sleep study is not essential for a positive diagnosis of sleep bruxism. This puts this treatment in the realm of general dentistry. With the Luco Hybrid device (Figure 5) being the 1st treatment FDA cleared for the treatment of sleep bruxism/tension-migraine headaches (K160477), there is a safe and effective treatment available that any dentist can easily learn and implement in their practices.

References


