Contact

Sleep-Related Bruxism for the Primary Care Physician

Dr. Ken Luco

Preamble

Sleep-related bruxism is a common and serious condition affecting 10-12% of the adult population[1]. It affects men and women equally and occurs at all ages. There is a genetic component that follows autosomal dominant inheritance, as we see this condition occurring in families. There is no predisposition for race, it affects all equally.

This document contains information that is directed for primary care physicians, to assist in identifying these patients and rendering appropriate therapies.

Dr. Ken Luco

What is sleep-related bruxism?

Sleep-related bruxism (SRB) occurs as the name suggests, during sleep and is characterized by episodes of clenching of the jaw and neck muscles and grinding of the teeth. Many patients suffering from this condition are completely unaware that they have it. This is very different from awake bruxism (AB), which occurs during waking hours and is stress and psychologically mediated.

Classification of Awake and Sleep-Related Bruxism:

Figure 1, below, is the ICD -11 classification of awake bruxism (AB), produced by the World health Organization. Of note, AB is classified under diseases of the digestive system subcategorized to dentofacial parafunctional disorders.

Figure 1 ICD-11 Classification of Awake Bruxism (AB)

DA0E.7 Dentofacial parafunctional disorders

All ancestors up to top

- 13 Diseases of the digestive system
  - Diseases or disorders of orofacial complex
    - DA0E Dentofacial anomalies
      - DA0E.7 Dentofacial parafunctional disorders

Hide ancestors

Description

Bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism)

Inclusions

- Teeth-clenching
- Teeth-grinding
- Bruxism
- Bruxomania

Exclusions

- Atypical facial pain (8B82.1)
- dyskinesia (MB47.4)
- trismus (DA0E.8)

Figure 1: Awake bruxism falls under diseases of the digestive system, specifically under dentofacial parafunctional disorders.

In contrast, SRB is classified much differently.

Figure 2 ICD-11 Classification of Sleep-Related Bruxism (SRB)

Figure 2: Sleep-related bruxism (SRB) is classified as a sleep-wake disorder, specifically under sleep-related movement disorders. In ICD-11, separate coding was added for severity (XS5W, XS0T, and XS25)

Unfortunately, both are usually referred to as one in the same and treated accordingly. Treatments such as physiotherapy, chiropractic therapy, hypnotherapy, psychotherapy and other similar modalities, although effective for AWB, have been shown to be totally ineffective in managing SRB (after all, the patient is sleeping when SRB occurs!).

AB has been shown in research to have a psychological component and phenotypes associated with it (high stress individuals, bullied children etc.). SRB however has no such psychological component, occurring in stages 1 and 2 of sleep, independent of consciousness.

Characteristics of Sleep-Related Bruxism:

There are three distinct forms of SRB seen on electromyography of the masseter and temporalis muscles during sleep.

Phasic SRB

This is the most common form of SRB seen on electromyography of SRB patients. It is characterized by rhythmic contractions of the masseter and temporalis

muscles. The force of contraction is significantly higher than normal chewing in part due the inhibition of a powerful protective reflex, the masseter inhibitory reflex or MIR. In SRB, the MIR is very significantly reduced allowing the masseter and temporalis muscles to contract with 100% of their bite force[3,4]. Figure 3 demonstrates and electromyography tracing of normal sleep.

Figure 3 Normal Jaw Movement during Sleep (EMG Recording)

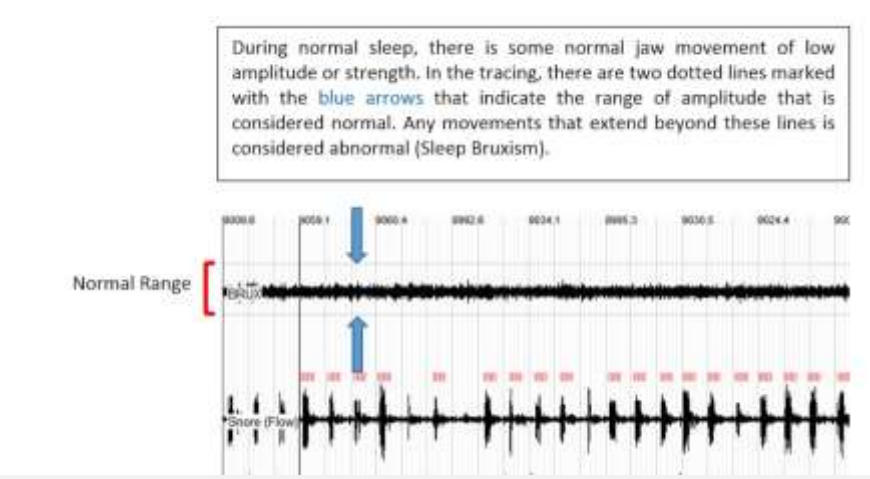


Figure 3 EMG of Normal Sleep. Of note, the normal jaw movements during sleep remain at all times between the dotted lines (normal range).

In contrast, Figure 4 is an EMG recording of SRB events. The areas marked in green demonstrate phasic type SRB. Of note, the amplitude of the SRB events fall well outside of the normal range, indicated in figures 3 and 4 with the dotted lines and red bracket.

Figure 4 EMG Recording of Sleep-Related Bruxism Events

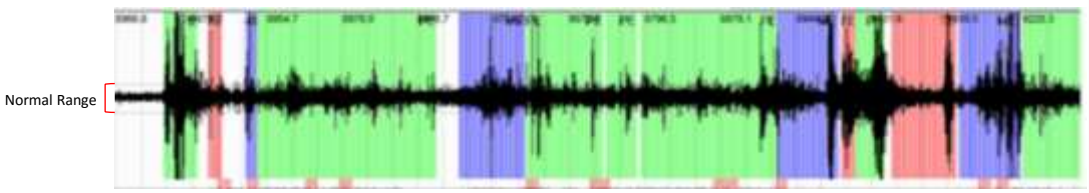


Figure 4 is a tracing of sleep-related bruxism. The areas marked in green are the phasic SRB and can be seen to increase and decrease in a rhythmic manner. The areas marked in red are the tonic events and blue are the mixed events. The events can be seen to be significantly greater than normal (4-6x greater than normal).

In fact, SRB events are often 4-6x greater in amplitude compared with normal movements and explains why structures such as tooth enamel wear considerably in SRB and very little in normal function. It also involves considerably greater muscle contractions (Figure 4) and explains the myriad of symptoms common to sufferers of this disorder including headaches and TMJ pain.

Phasic Sleep-Related Bruxism

In Figure 4, the areas marked in green are the phasic events. They are characterized by firing and relaxation of the muscles as is seen with grinding or chewing motions.

Tonic Sleep-Related Bruxism

In figure 4, the areas marked in red are the tonic SRB events. They appear very different and consist of a single strong contraction of the muscles. These contractions can last quite long. I have recording them lasting well over an hour in duration which is virtually impossible to accomplish while awake.

Mixed Sleep-Related Bruxism

In Figure 4, the areas marked in blue represent the mixed SRB events. As expected, they are a combination of clenching and grind episodes with characteristics of both phasic and tonic SRB.

It should be noted that all three presentations of SRB are far in excess of normal jaw movements. As well, phasic SRB is bar far the most common, tonic is next common and mixed the least common. The ratio varies considerably between patients and in some, tonic may predominate.

Interestingly, studies have shown that phasic SRB accounts for dental signs and symptoms and tonic is more related to headaches, TMJ and jaw pain. This makes perfect sense as the phasic grinding would be expected to damage the teeth whereas tonic sustained muscle contractions would be expected to generate jaw muscle pain, trigger point pain, crack teeth, and overload the TMJ. This is precisely what is seen.

What is the Effect of Untreated Sleep-Related Bruxism?

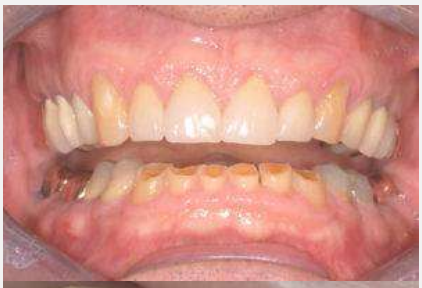
Already alluded to, SRB affects the teeth, TMJ, and muscles. It also affects the autonomic nervous system through activation of a powerful cranial reflex named the "Trigeminal Cardiac Reflex"[5]. As the name implies, this reflex involves the trigeminal nerve (cranial nerve 5) and the cardiovascular system. Stimulation of certain regions of the trigeminal nerve can activate the TCR through the Vagus nerve, affecting heart rate, blood pressure, respiration and gastric motility. SRB activates the sympathetic response resulting in rapid onset tachycardia, hypertension, hyperpnea, and gastric hypermobility (GERD). This stimulation occurs at the level of the Gasserion or trigeminal ganglion[6]. I will discuss this further later,

The Dentition:

SRB has been shown to cause the following dental conditions:

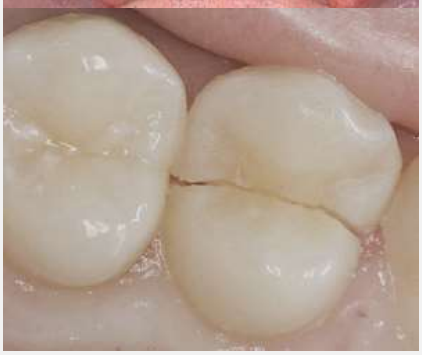
Figure 5 The effect on the dentition of SRB

Abnormal Tooth Wear
(Phasic SRB)



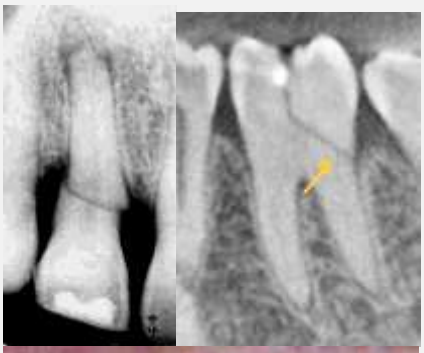
This wear can result in extreme tooth sensitivity, aseptic tooth abscess, loss of function, poor esthetics, etc.

Cracked Teeth
(Tonic and Phasic SRB)



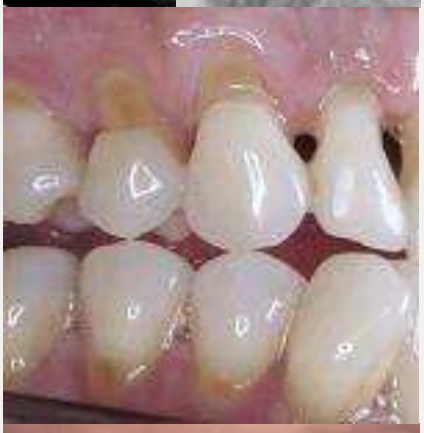
Cracked teeth are very painful. When small, the tooth only hurts when biting hard foods. When advanced, as in the image, they are extremely painful and must be removed.

Cracked Roots
(Phasic or Tonic SRB)



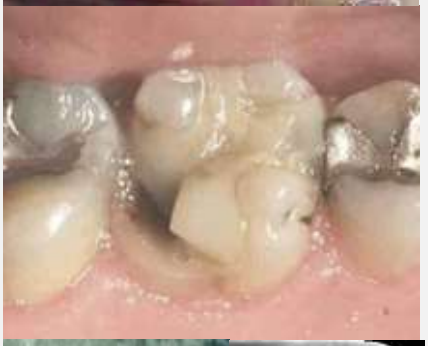
Cracked roots can occur, resulting in the crown of the tooth feeling “loose”. A radiograph is needed to locate the fracture. These teeth usually must be removed.

Abfraction Lesion
Formation (Phasic)



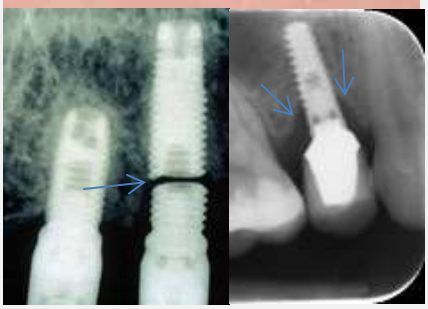
Abfraction lesions are enamel loss at the gum line due to rocking of teeth under pressure. These can be extremely sensitive to thermal stimulation and touch. These were previously thought to be caused by aggressive tooth brushing.

Premature failure of
dental restorations



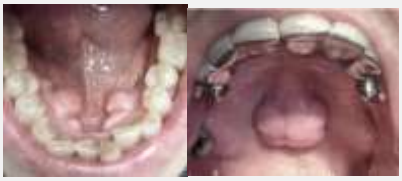
Restorative materials are designed with compressive and fracture strengths compatible with normal chewing forces. SRB exceeds these and the materials often fail prematurely.

Failure of dental
implants



SRB may cause fracturing of dental implants or bone loss around the implant (peri-implantitis) resulting in loosening and failure of the implant.

Formation of
mandibular and palatal
tori



Tori are considered exostoses that form in response to stress on the bone structures. Mandibular tori form in phasic SRB and palatal tori with tonic SRB.

Headaches:

SRB has as its two most common symptoms, tooth sensitivity and headaches. The headaches of SRB are generally muscular in origin, radiating from the masseter muscle, the temporalis muscle or the shoulder and cervical muscles (which are accessory chewing muscles). They may present as a tension headache on waking or as a tension or migraine type headache later in the day[7]. One important note is that migraine headaches associated with SRB are not preceded with an aura. Also important to note is that conventional pharmaceuticals used to treat tension and migraines do not necessarily work on SRB initiated headaches. They are usually reported as moderate to severe in intensity and tend to occur daily to weekly.

In some cases, patients will wake in the night with a severe headache. Headaches associated with SRB can affect a patient considerably and depression has also been associated with SRB.

Changes to the Maxilla and Mandible:

With extreme clenching forces seen in Tonic SRB, the mandible can be deformed. Antigonial notching and coronoid process elongation of the mandible are two common examples (Figures 5 and 6).

Figure 6 Antigonial Notching of the Mandible

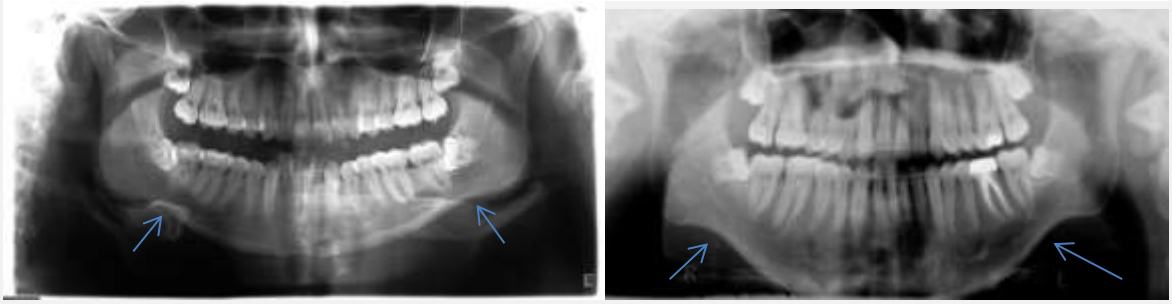


Figure 6 demonstrates a normal lower border of the mandible on the left and severe antigonial notching due to severe SRB on the right (blue arrows). This results in the masseter and medial pterygoid muscles being forced at a shortened working length which can lead to painful spasms in these muscles, a further deformation.

Figure 7 Elongation of the Coronoid Process

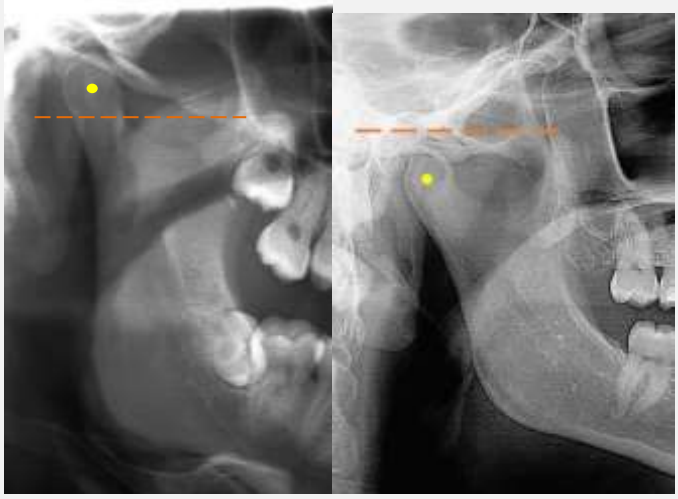


Figure 7 demonstrates a normal coronoid process on the left and a severely elongated one on the right (orange hashed line). This is due to excessive tension of the temporalis muscle on the coronoid process, the other masticatory muscle involved in SRB. If severe, it can be obstructed in lateral movements by the zygomatic arch resulting in loss of lateral movements of the mandible.

Other common bone changes seen in SRB include the formation of mandibular or palatal tori and bone exostoses. Tori are a result of torsion or bending of the mandible whereas exostoses are a result of severe side to side phasic SRB and rocking of the teeth side to side. The bone apposition helps to prevent breakage of the alveolar (tooth supporting) bone and mandible under the excessive loading.

Figure 8 Palatal and Mandibular Tori

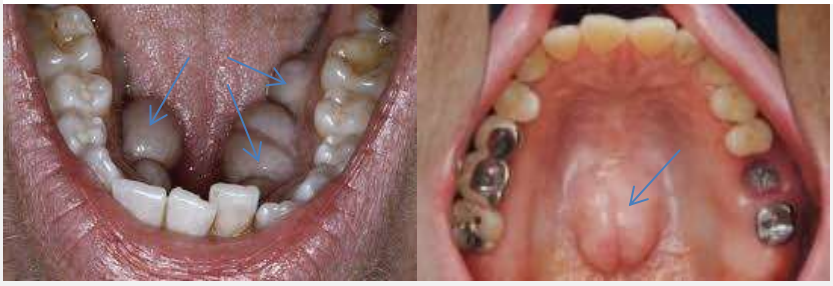


Figure 8 demonstrates typical mandibular and palatal tori. Palatal tori are felt to be intrusion of the nasal septum through the midpalatal suture due to extreme compressive forces of SRB and the resulting bone response. Over time they may pneumatize, becoming continuous with the nasal cavity or maxillary sinus. SRB has also been associated with deviate nasal septum. Mandibular tori result from twisting of the mandible during phasic SRB. This region is prone to fracturing and the apposition of bone acts to strengthen the mandible. The effect becomes exaggerated and the tori can get quite large. This also reduces tongue space in obstructive sleep disorders and may contribute to OSA.

Figure 9 Bone Exostoses

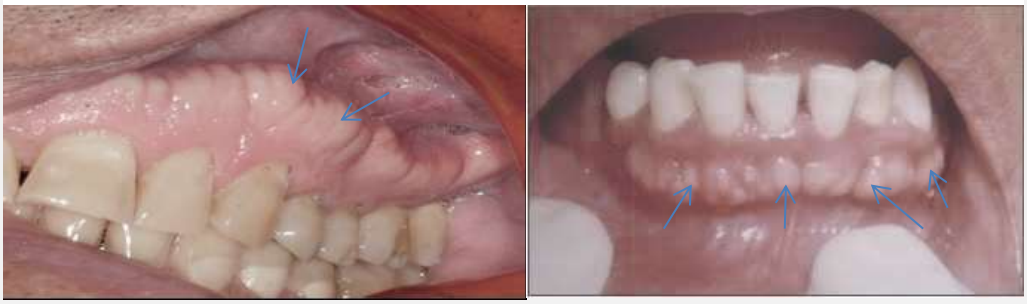


Figure 9 demonstrates maxillary and mandibular exostoses. These form in severe phasic type SRB and a firm to the touch. It is a physical response to rocking of the teeth and stress on the supporting alveolar bone. As with tori development, the bone apposition is often exaggerated as is seen in Figure 8 and are painless. Surprisingly, patients are often unaware of these and are shocked to learn that they are abnormal.

The Cardiovascular System:

As alluded to in the preamble, SRB has a profound effect on the autonomic nervous system, particularly the sympathetic nervous system. It has been known for some time that with every SRB event, there is a rapid increase in heart rate and blood pressure, dropping just as fast once the event ends. In the last five years, it was found that this was actually an activation of the *trigeminal cardiac reflex* or TCR. The TCR is actually made up of a group of powerful cranial reflexes that have one commonality: the stimulus is via the trigeminal nerve system[5,6]. The reflexes comprising the TCR are shown in Figure 9.

Figure 10 the Cranial Reflexes that Comprise the TCR

Reflex	Type	Effect
Occulo-cardiac reflex	Parasympathetic	Bradycardia, apnea, hypotension
Naso-cardiac reflex	Parasympathetic	Bradycardia, apnea, hypotension
Maxillo-mandibular reflex	Parasympathetic	Bradycardia, apnea, hypotension
Gasserion ganglion reflex	Sympathetic	Tachycardia, hyperpnea, hypertension
Brainstem reflex	Parasympathetic	Bradycardia, apnea, hypotension
Central reflex	Parasympathetic	Bradycardia, apnea, hypotension

Figure 10 lists the 6 reflexes that comprise the TCR. The occulo-cardiac is by the 1st branch, the naso-cardiac and maxillo-mandibular are via the 2nd and 3rd branches, the remainder are inside the brainstem. Of significance, only the Gasserion ganglion elicits a sympathetic response, all others elicit a parasympathetic response.

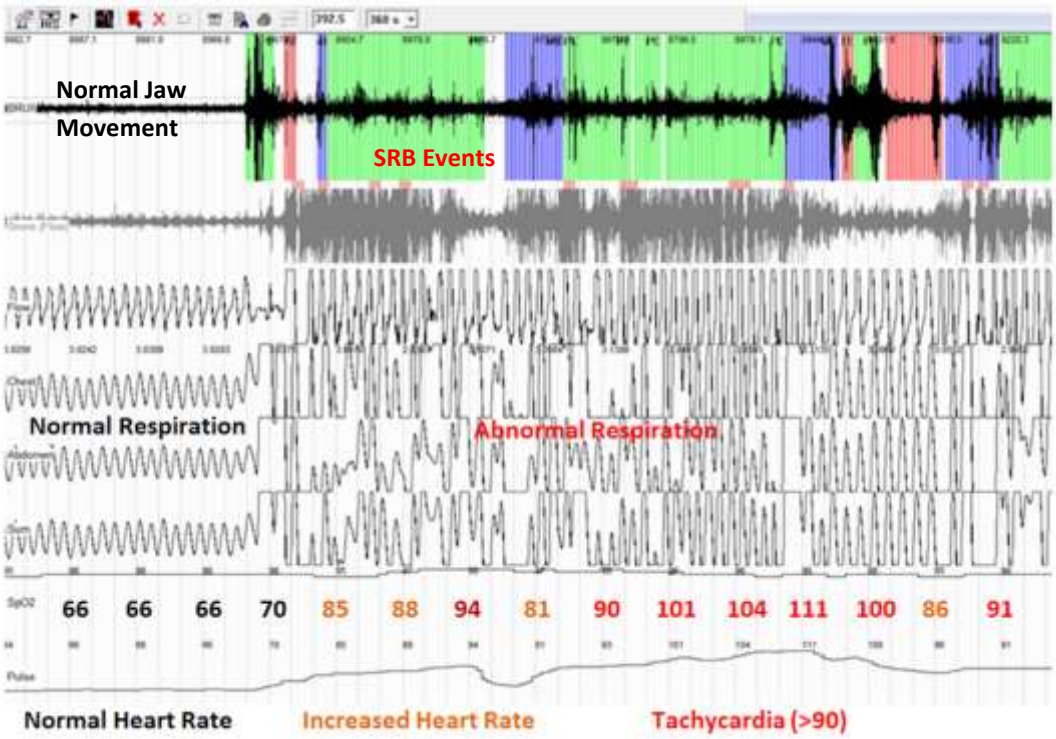
A recent article discussed the otic reflex, whereby one sneezes when looking at a bright light, as a variant of the occulo-cardiac reflex[8]. Another paper discussed when endodontic treatment was being conducted on cuspid and 1st bicuspid teeth, a strong parasympathetic TCR effect was observed. Endodontic treatment of all other teeth had no effect. This was attributed to the maxillo-mandibular reflex as a variant. There will likely be other variants found in the future which make the TCR not just the most powerful of the cranial reflexes, but the most interesting and far reaching as well.

But how does this relate to SRB?

It has been shown that the heart rate and blood pressure increase seen with each significant SRB event affects the TCR at the level of the Gasserion ganglion. With SRB, GERD is also common[9]. This is also a sympathetic stimulation of the gut associated with TCR activation (Vagus nerve).

Figure 11 demonstrates the profound hyper-stimulation of the TCR that SRB elicits.

Figure 5 SRB Activation of the TCR



It is medically accepted that there must be a change of greater than 20% in heart rate, with a rapid onset for a statement of TCR activation may be made. In Figure 10, the heart rate increases from 66 bpm to 111 bpm. This is an increase of 68%, indicating SRB hyper-stimulates the TCR. SRB has been implicated in the development of cardiovascular disease as a result of this effect.

Disturbed Sleep:

SRB is classified under sleep-wake disorders (figure 2) and affects sleep. Studies have shown that SRB results in a significantly shorter wake time after sleep onset, stage N1 sleep was shorter, stage N3 was longer[10,11]. SRB events often result in micro-arousals resulting in decreased REM sleep. Epworth sleepiness scores of 4-9 are common with SRB and daytime fatigue is often reported (mild OSA is 10+).

Figure 6 Sleep Stages where SRB Occur

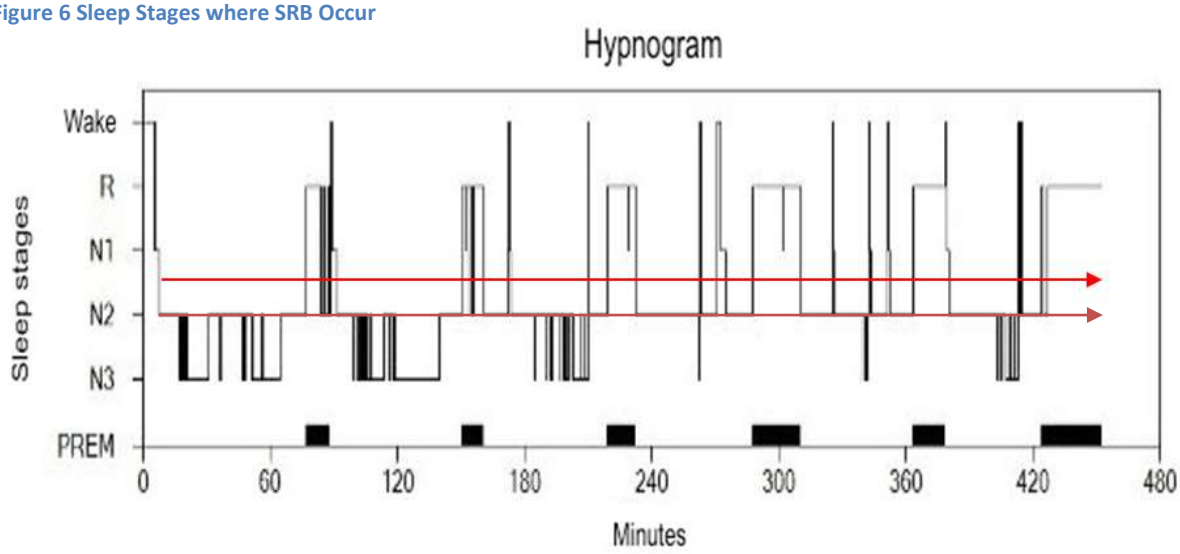


Figure 12 is a hypnogram of normal sleep architecture. The red arrows indicate the consensus of where SRB occurs between N1 and N2 sleep stages). Men and women are affected equally with daytime fatigue resulting from this altered sleep architecture. Each SRB event results in a sleep arousal, sometimes waking the patient, with an awareness of their teeth or jaws.

Primary vs Secondary SRB:

Some researchers claim that SRB is only a response to obstructive sleep apnea events. They claim that SRB is triggered by the hypoxia and is an attempt to partially wake the patient, to re-start normal breathing. This is incorrect as there are primary cases that do not demonstrate any correlation with obstructive apnea. There are also mediation induced SRB. The different presentations are better viewed as primary and secondary SRB.

Primary SRB is independent of sleep disordered breathing and is the most common form of SRB. Respiration is increased, often erratically, but never stops.

Figure 13 Primary SRB

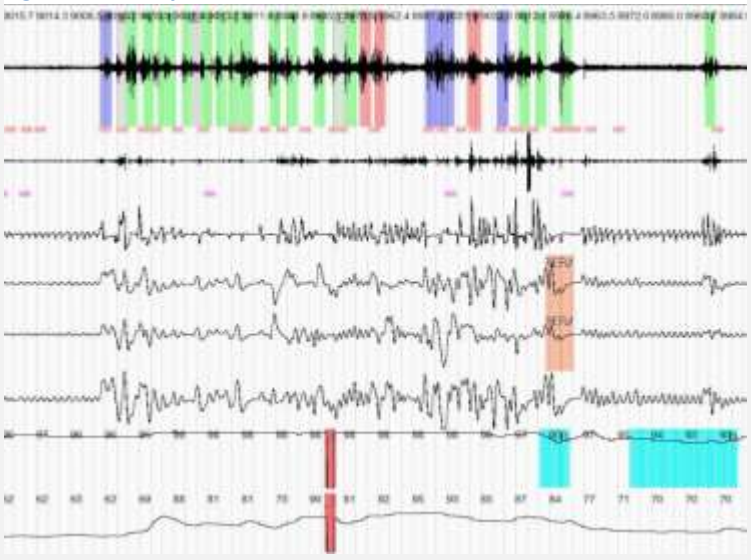


Figure 13 is a sleep tracing demonstrating primary SRB. Of note, there is activation of the TCR (53.2% increase in heart rate), and hyperpnea without cessation of respiration. This is true primary SRB. There is normal heart rate and respiration immediately before and after the SRB event.

Secondary SRB to Sleep Disordered Breathing / OSA

SRB has been shown to occur in conjunction with obstructive sleep apnea, often occurring just before or just after the respiratory event[12,13]. It is felt that this form of SRB works to stimulate the patient through micro-arousals, restoring normal respiration. Figure 14 is a good example of this, in a 28 year old male patient. The screenshot of his sleep study demonstrates a clear example of this. The SRB

events occur interspersed between the sleep disordered breathing events, synchronizing with them.

Figure 7 Secondary SRB occurring with OSA

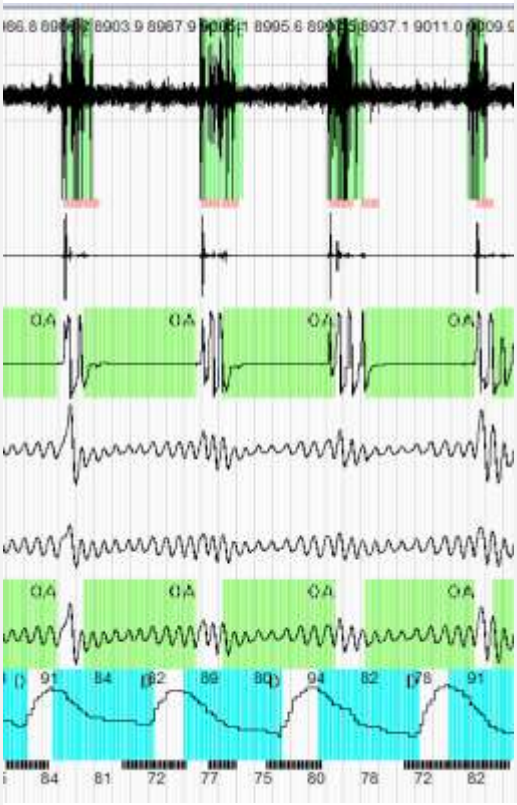


Figure 14 a sleep tracing demonstrating secondary SRB occurring with sleep disordered breathing events. The SRB synchronizes between each obstructive apnea event. It is felt that this presentation of SRB acts to wake the patient assisting in restoration of normal restoration.

Medication Induced SRB

In recent years it was found that certain classes of medications have bruxism, specifically SRB as a "side effect"[14-16]. Considering the TCR effect of SRB and potential for the development of cardiovascular disease, this is of great importance.

SRB patients are sensitive to serotonin levels in the CNS. These medications act by preventing reabsorption of serotonin resulting in stimulation of the SRB. Table 1 lists the medications known to initiate or worsen SRB.

Table 1 Medications Shown to Initiate or Increase SRB

Generic Name	***Drug Name***
Luvox	Fluvoxamine
Sarafem	Fluoxetine
Pexeva (Pro)	Paroxetine
Paxil CR(Pro)	Paroxetine
Lexapro (Pro)	Escitalopram
Celexa (Pro)	Citalopram
Prozac (Pro)	Sertraline
Zoloft	Fluvoxamine
Brisdelle (Pro)	Paroxetine
Prozac Weekly (Pro)	Fluoxetine
Rapiflux	Fluoxetine
Effexor XR	Venlafaxine
Pristiq	Desvenlafaxine
Cymbalta (Pro)	Duloxetine
Savella (Pro)	Milnacipran
Effexor (Pro)	Venlafaxine
Fetzima	Levomilnacipran
Irenka (Pro)	Duloxetine
Khedezla	Desvenlafaxine
Ritalin	Methylphenidate

Many of these medications are serotonin sparing, preventing the reabsorption of serotonin in the central nervous system.

Figure 85 sleep tracing of medication induced SRB-

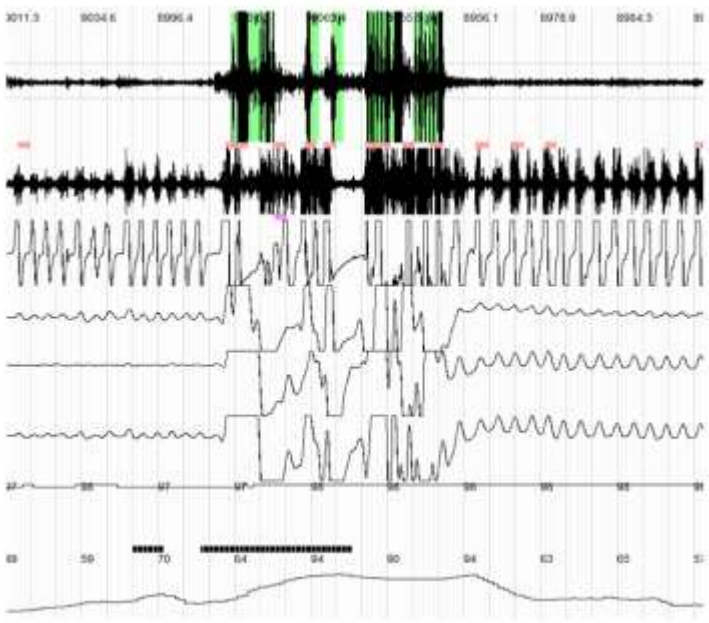


Figure 15 is a tracing of a patient who demonstrated SRB after taking Paxil for only a short time and developed severe headaches upon waking. It is similar in appearance to primary SRB, indistinguishably, on a sleep study necessitating a careful review of the medical history in all cases.

Figure 15 demonstrates SRB in a patient who was placed on Paxil for anxiety and who developed very rapidly severe headaches on waking. The sleep study was conducted to determine the cause and the SRB was found to be severe in this patient. The appearance is virtually the same as primary SRB. There are phasic SRB events far in excessive of the normal range resulting in tachycardia, hyperpnea, and activation of the trigeminal cardiac reflex (a rapid increase in heart rate from 49 to 94bpm, in excessive of 20% at 91%).

SRB's Effect on Cranial Reflexes

In SRB, studies have found a mutation of the gene HTR2A rs2770304[17-19] on chromosome 13 occurs in a majority of SRB patients. There is a polymorphism resulting in overexpression of the gene. This gene creates proteins that fold into suppressive serotonin receptors in the CNS. With an excess of receptors in these patients, there is overexpression of serotonin.

With the mediations in Table 1 sparing reabsorption of serotonin and the increased number of receptors in SRB patients, exacerbation is inevitable. IN SRB, there is suppression of a protective cranial reflex named the “masseter inhibitory reflex” or MIR (Figure 16)[4]. This reflex is activated if one bites unexpectedly into a hard food while chewing something soft. Immediately chewing stops, the object is removed and chewing resumes slowing. This is the MIR in action.

Figure 9 EMG tracing of the MIR

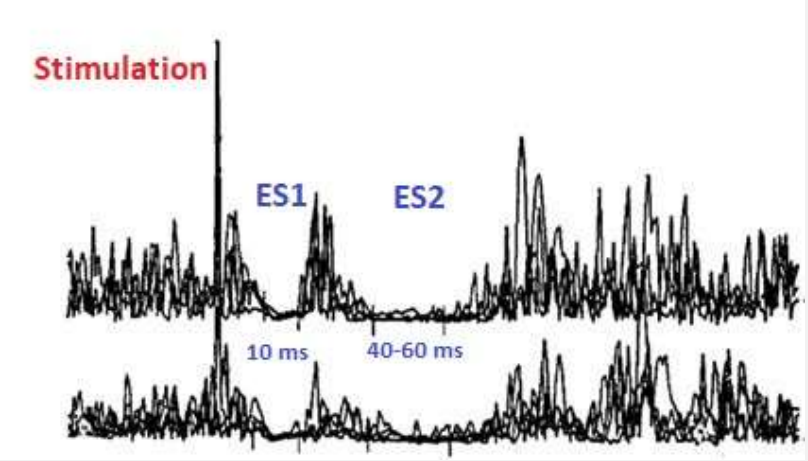


Figure 16 EMG tracing of a normal MIR. After stimulation, the ES1 phase occurs immediately, lasting 10ms. It is followed by the ES2 phase that lasts 40-60ms. The effect of this reflex is inhibition of the masseter and temporalis muscles from contracting.

There are two phases of the MIR, termed extroceptive suppression one (ES1), and extroceptive suppression two (ES2). In Figure 14, a stimulation of the reflex occurs and immediately ES1 activates, lasting 10ms. The decrease in muscle firing is evident. ES1 is innervated by sensory organs located in the periodontal ligament surrounding the teeth and in the mental nerve. When a ligament is excessively stretched (as in biting something hard), an impulse is sent via the trigeminal nerve to the sensory nucleus of the trigeminal nerve located in the pons. A unilateral impulse is sent to the contralateral trigeminal motor nucleus suppressing it from sending excitatory impulses to the masseter and temporalis muscles. This constitutes the ES1 phase of the MIR.

It is followed immediately after by the ES2 phase, which lasts 40-60ms and bilaterally suppresses the masseter and temporalis muscles. This phase is stimulated by Golgi tendon organs in the masseter and temporalis muscle bodies in response to contractions that exceed normal.

In SRB the MIR is affected and can be seen on EMG tracing in SRB patients.

Figure 10 SRB's Effect on the MIR

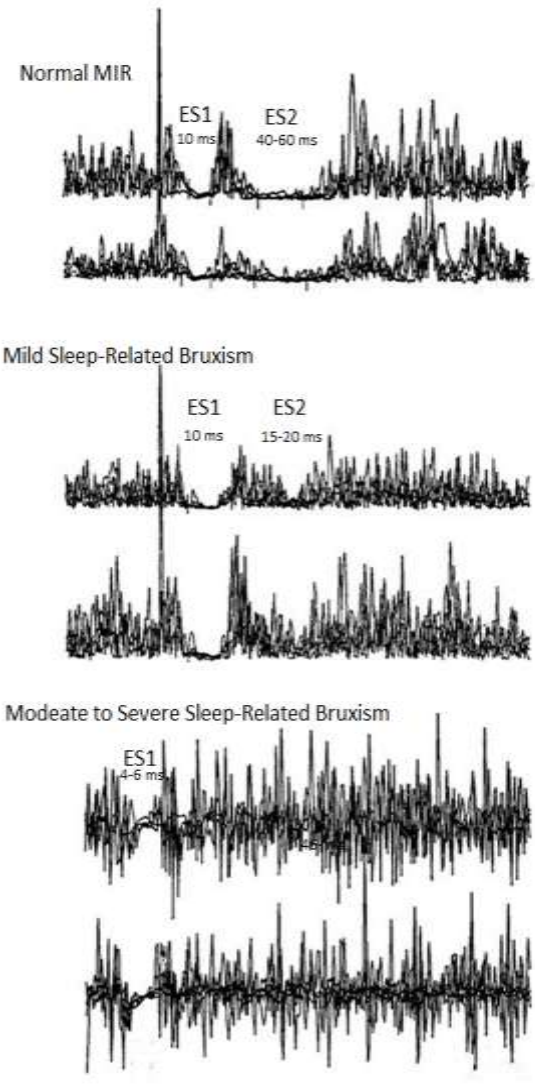


Figure 17 demonstrates three EMG tracings. The first is a normal MIR, with the ES1 phase lasting 10ms and the ES2 phase lasting 40-60ms. The second tracing, of mild SRB, shows a shortened ES2 phase to only 15-20 ms with a normal ES1. The third tracing is of moderate to severe SRB. The ES2 phase is completely absent and the ES1 phase is shortened to 4-6ms. SRB profoundly affects the MIR allowing the masseter and temporalis muscles to contract with full force as is seen on EMG tracings of SRB patients (Figures 16-17).

The ES2 phase is affected the greatest by SRB, with it being completely eliminated in moderate to severe SRB.

Tension Type Headaches and the MIR

Tension type headaches (TTH) have a great effect on the MIR[3]. This leads credence to the theory that TTH is related to the jaw musculature, particularly, the masseter and temporalis muscles.

Figure 118 EMG tracing of a patient with a tension headache

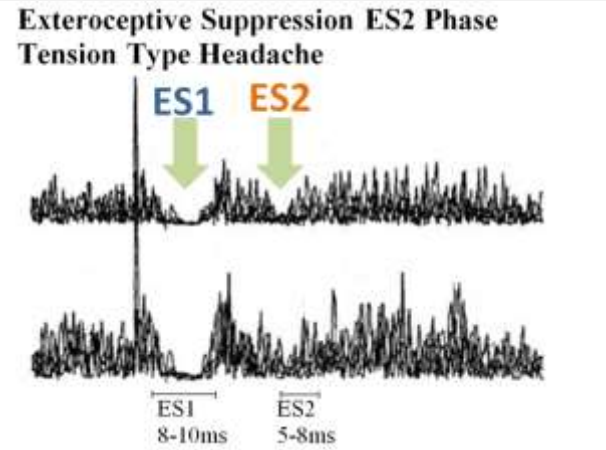
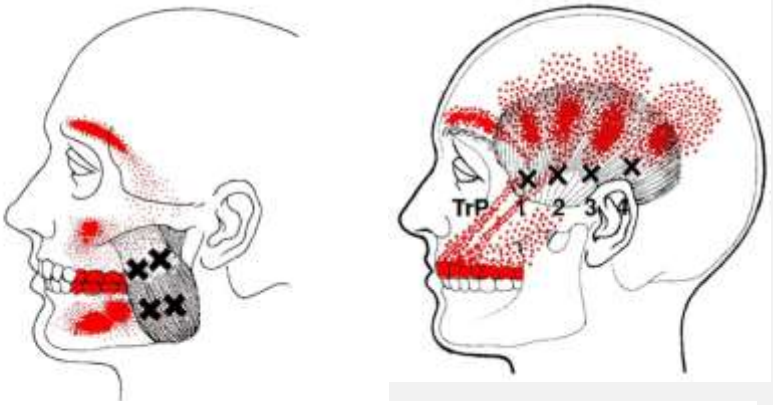


Figure 18 is an EMG tracing of the MIR during a tension headache. Of note, as in tension headache there is shortening of the ES2 phase, here to 5-8ms from 40-60ms. This would result in increased activity and intensity of contraction of the masseter and temporalis muscles, leading to fatigue in the muscles. Over time this can lead to the development of myofascial trigger points. These TP's explain the headaches seen in SRB and TPs can usually be detected.

In comparing Figure 17, mild SRB, to Figure 18, tension headaches, they are very similar in appearance, with the ES2 phase of the MIR affected similarly. The location of the headaches associated with SRB are usually the in the temple region.

Figure 12 Trigger Point Pain Referral Patterns for the Masseter and Temporalis Muscles



Masseter TP pain referral | Temporalis TP pain referral

Figure 19 outlines the classical myofascial trigger point pain referral patterns for the masseter and temporalis muscles. Of note, both muscles refer pain to the temple region, the most often reported region for tension headaches associated with SRB. Both muscles also refer pain to the teeth, and can account for some of the sensitivity of the teeth seen in SRB.

In Figure 19, the trigger point pain referral patterns for the masseter and temporalis muscles are shown. Theses account not only for the temporal tension type headaches but for some of the tooth sensitivity associated with SRB.

Migraine Headaches and SRB

SRB has been associated with migraine type headaches[20], but only those that present without an aura. The mechanism is not the same as tension type headaches and there are conflicting theories on the exact mechanism. All that is known is that there is a definite relationship between the two.

Clinical Implications

As can be seen, SRB presents with a myriad of signs and symptoms. Many will be more obvious to dentists however headaches fall under the care of the primary physician. When I practiced clinical dentistry, it was rare a patient would come in and say “I wake with terrible headaches. What can be done about this?” Normally they would present to their doctor asking the same question.

If the doctor is unaware of SRB’s connection to headaches, this could be completely overlooked. Adding SRB to the differential diagnosis can prevent inappropriate and potentially ineffective treatments for the SRB headaches. Keep in mind that temporal headaches and sensitive teeth are the two most commonly reported symptoms of SRB.

Testing for SRB

There is a very simple test that can be done clinically that offers a highly accurate result when screening for SRB.

Figure 20 the Mandibular Tremor of SRB

Figure 20 when an SRB patient protrudes their jaw forward until only the incisors are in contact, and hold this position lightly for 30-60 seconds, the mandible will develop a tremor and begin to shake and teeth chatter. This test is positive for most SRB patients.

Ask the patient to protrude their jaw until only the incisors are touching as in Figure 19. Instruct them to lightly contact the teeth for 45-60 seconds and to notify you if their mandible begins to tremor[21]. If it does, there is a very high

likelihood of SRB underlying the symptoms. A study in 2019 found that periodontal mechanoreceptors are responsible for this effect in SRB[22].

Diagnosis and Treatment of SRB

There are three levels of confidence associated with diagnosing SRB[23].

1. Lowest level: the patient reports they feel they are grinding their teeth while sleeping,
2. Moderate level: the patient reports grinding *and* there are signs of dental changes observed clinically (clinician verified), and
3. Highest Level: the patient reports grinding, there are signs of dental changes *and with* overnight polysomnography with positive EMG recordings of the masseter and temporalis muscles. Considered the "gold standard".

The American Academy of Sleep Medicine requires a moderate level of confidence to make a diagnosis of SRB[1]. Very few sleep labs are equipped with EMG recordings in real time and it has been found that the accuracy is very good using the moderate level. This may be done in a dental clinic setting. The FDA considers SRB to be a dental condition and, as such, is best treated by a dentist who is trained in this field.

Treatment of SRB

Medications such as muscle relaxants and analgesics may provide temporary relief in exacerbations, however cannot be used long term and only mask SRB.

Botox injections work in the short term by paralyzing the masseter and temporalis muscles in a relaxed state however this cannot be used long term either. There are some reports of sensitization with repeated use.

Physiotherapy, chiropractic therapy, hypnotherapy etc. have been shown to be ineffective, with most of the studies being anecdotal or case studies. No long term studies have demonstrated any benefit.

Clonidine has been shown to be effective in reducing SRB paradoxically in patients undergoing treatment for hypertension however its use in patients with normal blood pressure can result in unsafe drops and it is not considered or approved as a valid treatment of SRB[25].

The Luco Hybrid OSA Appliance

The Luco Hybrid OSA Appliance became the first FDA cleared treatment of SRB and to aid in the treatment of associated tension and migraine headaches in adults in 2016 (K160477)[26]. It is also FDA cleared for the treatment of obstructive sleep apnea and snoring in adults (K130797)[27] (Figure 21).

Figure 21 the Luco Hybrid OSA Appliance

It is classified as a mandibular advancement appliance and is quite different from other OSA appliances.

Figure 13 Side View and Forward Bite of the Luco Hybrid device

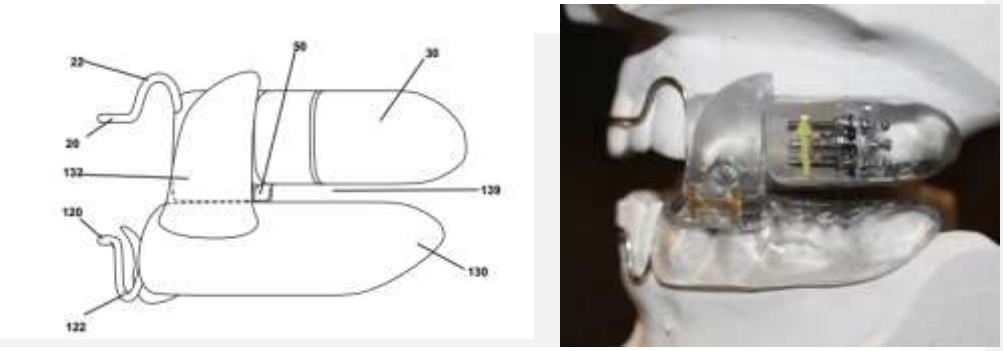


Figure 22 is a side view of the Luco Hybrid device. Of note, there is only contact in the region of the cuspid and 1st bicuspids; there is no contact in the molar regions. This is termed a "forward bite".

The forward bite (Figure 22) of the Luco Hybrid is a patented feature seen only in this device. There are three reasons for this bite.

1. Stress Distribution
2. Activation of the periodontal masseter reflex
3. Stabilization of the TCR effect

1. Stress Distribution

When we bite, stress is placed, through the teeth and alveolar supporting bone, on the skull and mandible. Understandably, the stress would be expected to be different when biting in different regions. This is precisely the case.

Figure 143 Stress Distribution when Biting in the Molar Regions

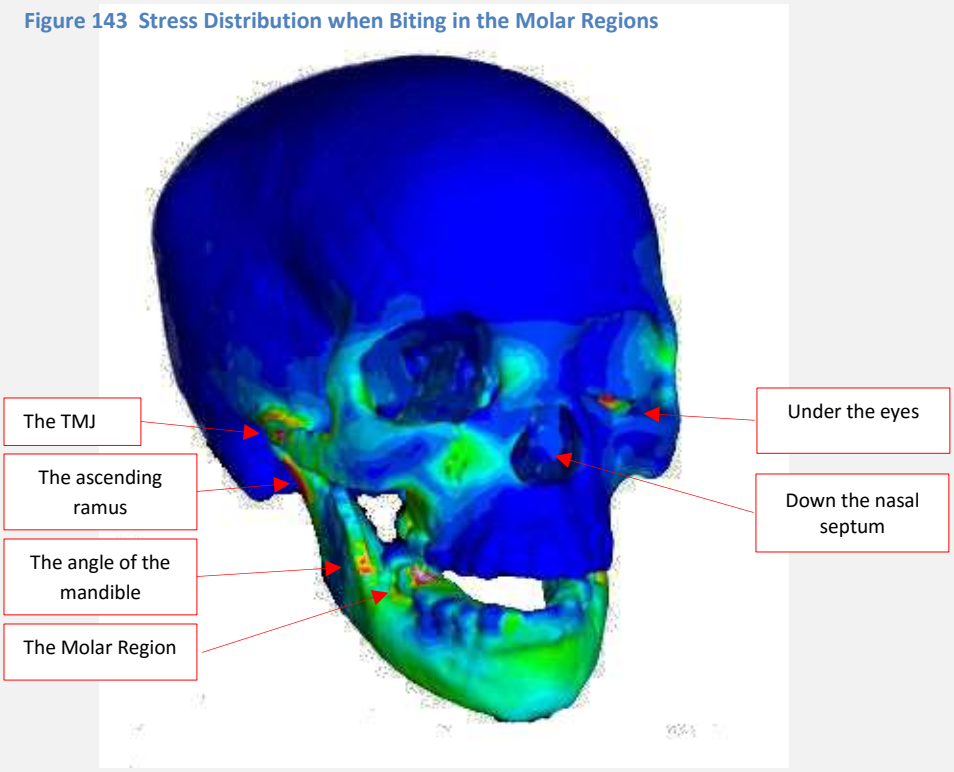


Figure 23 illustrates a stereolithic image of the stress on the skull when biting in the molar region. There are a number of regions that demonstrate high stress (indicated in red) including the molar region, the angle of the mandible, the ascending ramus, the condyle, the TMJ, under the eyes, and down the nasal septum. The increased loading of the TMJ explains why conventional OSA appliances can result in TMJ pain within a few weeks in some patients (those suffering from SRB!)[28].

The stress directed down the nasal septum is interesting as SRB has been associated with deviation of the nasal septum. The downward force of the stress distribution, accompanied by the upward force of the phasic and tonic SRB, place a considerable compressive force on the nasal septum. One of two things can happen: The nasal septum can buckle under the compression and deviate to one side or a palatal torus (Figure 5) will form. Both are associated with SRB and this is the suspected mechanism.

In complete contrast, when the bite is moved forward to the cuspid region, the stress distribution is much more favorable (Figure 23).

Figure 154 Stress Distribution when Biting in the Cuspid Region

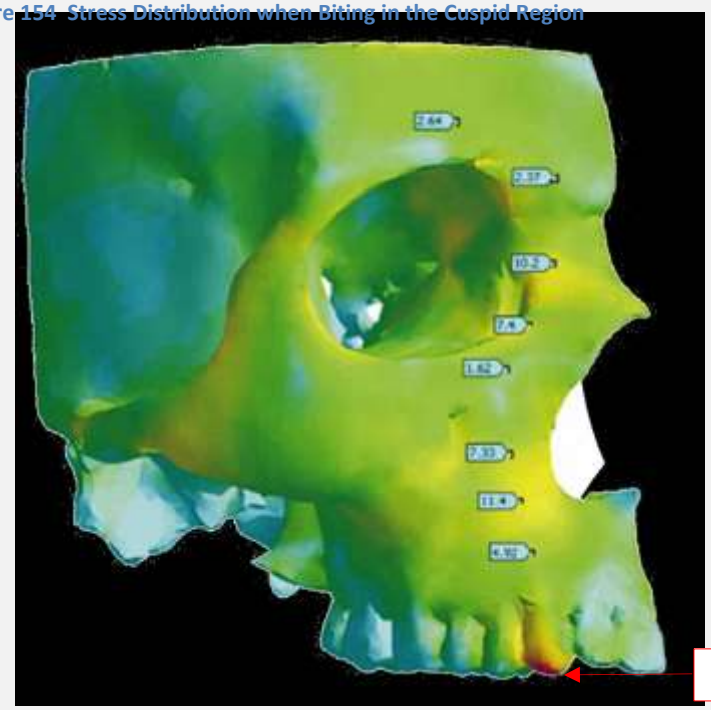


Figure 24 is the stress distribution of biting in the cuspid region on the skull. Of note, the only high stress area is the tip of the cuspid, one of our strongest teeth. All other stress is much reduced and evenly distributed.

Figure 24 illustrates a stereolithic image of the stress on the skull when biting in the cuspid region. As can be seen, the only high stress area (marked in red) is the tip of the cuspid; the stress is evenly distributed up and over the top of the skull.

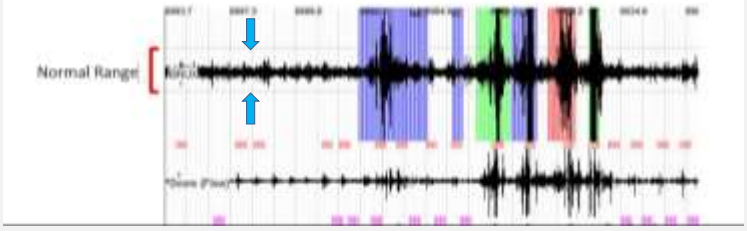
The Luco Hybrid device's forward bite places all force (stress) over the cuspid and first bicuspid (Figure 22) achieving the stress pattern as seen in Figure 24. All other OSA appliances (and for that matter, single arch TMJ and bruxism appliances) contact fully across the devices including the molar regions and have a molar stress distribution as is Figure 23. This is likely the reason these devices have no effect on the SRB and only provide protection from the extreme associated forces. Also this is likely the reason why TMJ pain is often reported with OSA appliance wear[28].

2. Activation of the Periodontal-Masseter Reflex

The periodontal-masseter reflex is another protective jaw reflex. It has been shown that sensory organs in the periodontal ligament of the cuspids and 1st

bicuspids activate this reflex. The incisors also can to a much lesser extent.

Figure 27 Before and After Tracings



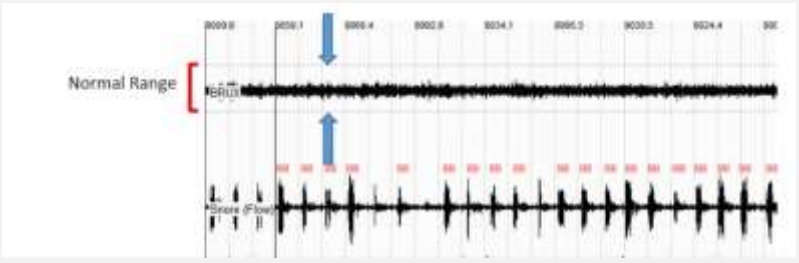


Figure 27 shows two sleep tracings. The first SRB events are apparent. In the second, they are not. This is the same patient while wearing the Luco Hybrid device after 4 weeks. The blue arrows indicate the normal range. The significant reduction in SRB events can be attributed to the Luco Hybrid device's activation of the PMR with the forward bite positioned over the cuspid and 1st bicuspid.

Figure 28 the Periodontal-Masseter Reflex

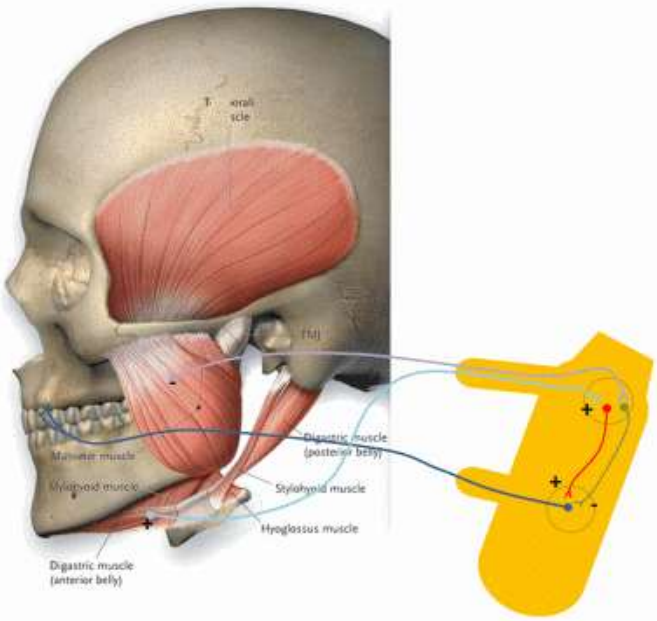


Figure 28 describes the periodontal-masseter reflex (PMR). The afferent for the reflex are sensory organs in the periodontal ligaments of the cuspids and 1st bicuspids. The reflex is unilateral on the ipsilateral side. Importantly, this reflex is not affected by the HTR2a polymorphism and functions normally (unlike the MIR which is suppressed).

Figure 28, the periodontal-masseter reflex, or PMR, is important as only the cuspid and 1st bicuspid teeth activate the PMR (an the a lesser extent, the incisors). The forward bite of the Luco Hybrid device (Figure 22) is positioned exactly in this location. When an SRB event begins, the PMR activates, suppressing contraction of the masseter and temporalis muscles and, as a result, the SRB events and TCR activation.

Figure 27 demonstrates that the Luco Hybrid device suppresses the SRB events, likely through activation of the PMR reflex. Within two weeks, SRB events numbering in the hundreds per night are reduced to less than 10 on average. This

translates into a dramatic reduction in headaches, tooth sensitivity, and restores deep restful sleep quickly and safely within weeks of use.

3. Stabilization of the Trigeminal Cardiac Reflex

The third effect of the Luco Hybrid device is the stabilization of TCR sympathetic stimulation seen in SRB (Figure 10). An endodontic study conducted in 2018 demonstrated when root canal therapy was performed on cuspids and 1st bicuspids, there was parasympathetic activation of the TCR with a rapid and pronounced drop in blood pressure and heart rate. With the forward bite of the Luco Hybrid device, the pressure on these two teeth by SRB results in a similar parasympathetic activation of the TCR. This sympathetic SRB stimulation of the TCR is negated by the inhibition by the PMR. Heart rate, blood pressure, breathing and GERD stabilize rapidly. Table 2 is a summary of 15 patients treated with the Luco Hybrid device in a clinical study by the author.

Table 2 Summary of 15 Patients Treated with the Luco Hybrid Device

Gender	*Age*	*Bruxism Index Before*	*Bruxism Index After*	*Max Heart Rate Before*	*Max Heart Rate After*	*% Increase Heart Rate Before*	*% Increase Heart Rate After*
Male	36	42	3	115	67	71%	9%
Female	29	23	2	129	71	81%	6%
Female	21	33	0	146	69	111%	7%
Female	47	29	5	112	66	68%	5%
Male	26	26	1	156	71	119%	6%
Female	31	20	2	114	74	54%	5%
Male	19	44	3	137	68	101%	3%
Male	28	32	1	128	66	93%	11%
Male	44	21	3	137	71	93%	4%
Female	22	38	1	157	68	131%	3%
Female	27	24	2	127	72	76%	11%
Female	39	35	5	133	66	102%	6%
Male	18	25	3	141	68	107%	7%
Male	51	31	4	160	71	125%	12%
Male	44	22	3	139	62	124%	4%

Mean	*32.1*	*29.7/hr*	*2.5/hr*	*135.4 bpm*	*68.7 bpm*	*97.1%*	*6.6%*

The average age was 32.1 years of age. Of interest are the last 4 columns (marked in blue) labelled maximum heart rate before and after treatment and % increase in heart rate before and after treatment.

You will note that the mean maximum heart rate was 135.4 bpm before treatment and 68.7 bpm after treatment. The % increase before treatment was 97.1% (clearly higher than the 20% threshold for TCR activation) was reduced to 6.6% after (normal range). This has been replicated in other studies and supports that the Luco Hybrid device not only reduces the bruxism index (number of bruxism events per hour of sleep), it also stabilizes the TCR, preventing the increase in heart rate (and blood pressure, etc.) associated with SRB.

The significance of this is that the repeated significant increase in heart rate and increase in blood pressure associated with the development of cardiovascular disease/renal damage is eliminated while wearing the Luco Hybrid device.

In Summary

I sincerely hope that the information contained in this document was helpful and informative for you. I have included a list of studies referenced in this document at the end.

If you wish to contact me for more information on sleep-related bruxism, the following contact information may be useful.

Tel 1-613-888-6019 email DrLuco@sympatico.ca

[redacted]

www.lucohybridosa.com www.sleepbruxism.ca

Please stay safe and healthy in these trying times with Covid 19.

RESEARCH ON SLEEP-RELATED BRUXISM

The following list studies referenced in this document.

References

1. American Academy of Sleep Medicine. Sleep related bruxism. In: International Classification of Sleep Disorders. 3rd ed. Westchester, Darien, Illinois: American Academy of Sleep Medicine; 2014.
2. World Health Organization. (2018). International classification of diseases for mortality and morbidity statistics (11th Revision). Retrieved from https://icd.who.int/browse11/l-m/en
3. 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
4. İnan R, Şenel GB, Yavlal F, Karadeniz D, Gündüz A, Kızıltan ME. Sleep bruxism is related to decreased inhibitory control of trigeminal motoneurons, but not with reticulobulbar system. Neurol Sci. 2017;38(1):75-81. doi:10.1007/s10072-016-2711-x
5. Meuwly C, Golanov E, Chowdhury T, Erne P, Schaller B. Trigeminal cardiac reflex: new thinking model about the definition based on a literature review. Medicine (Baltimore). 2015;94(5):e484. doi:10.1097/MD.0000000000000484
6. Luco, K. (2018). The Relationship of the Trigemino-Cardiac Reflex to Sleep Bruxism. Online Journal of Neurology and Brain Disorders, 1(2), Online Journal of Neurology and Brain Disorders, 2018-04-06, Vol.1 (2).
7. 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
8. Chowdhury, T., Sternberg, Z., Golanov, E., Gelpi, R., Rosemann, T., & Schaller, B. J. (2019). Photic sneeze reflex: Another variant of the trigeminocardiac reflex? Future Neurology. Future Medicine Ltd. https://doi.org/10.2217/fnl-2019-0007
9. Jokubauskas, L., & Baltrušaitytė, A. (2017, February 1). Relationship between obstructive sleep apnoea syndrome and sleep bruxism: a systematic review. Journal of Oral Rehabilitation. Blackwell Publishing Ltd. https://doi.org/10.1111/joor.12468
10. Carra, M. C., Huynh, N., Fleury, B., & Lavigne, G. (2015, September 1). Overview on Sleep Bruxism for Sleep Medicine Clinicians. Sleep Medicine Clinics. W.B. Saunders. https://doi.org/10.1016/j.jsmc.2015.05.005
11. Kim, H., & Han, H. J. (2015). Sleep quality in adult patients with sleep related bruxism. Sleep and Biological Rhythms, 13(1), 94–98. https://doi.org/10.1111/sbr.12090
12. Oksenberg A, Arons E. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. Sleep Med. 2002;3(6):513-515. doi:10.1016/s1389-9457(02)00130-2
13. Wieckiewicz, M., Bogunia-Kubik, K., Mazur, G. et al. Genetic basis of sleep bruxism and sleep apnea—response to a medical puzzle. Sci Rep 10, 7497 (2020). https://doi.org/10.1038/s41598-020-64615-y
14. Milanlioglu A. Paroxetine-induced severe sleep bruxism successfully treated with buspirone. Clinics (Sao Paulo). 2012;67(2):191-192. doi:10.6061/clinics/2012(02)17
15. Uvais NA, Sreeraj VS, Sathish Kumar SV. Sertraline induced mandibular dystonia and bruxism. J Family Med Prim Care. 2016;5(4):882-884. doi:10.4103/2249-4863.201168
16. Sabuncuoglu, O., Ekıncı, O. and Berkem, M. (2009), Fluoxetine-induced sleep bruxism in an adolescent treated with buspirone: a case report. Special Care in Dentistry, 29: 215-217. doi:10.1111/j.1754-4505.2009.00091.x
17. Halder, I., Muldoon, M. F., Ferrell, R. E., & Manuck, S. B. (2007). Serotonin receptor 2A (HTR2A) gene polymorphisms are associated with blood pressure, central adiposity, and the metabolic syndrome. Metabolic Syndrome and Related Disorders, 5(4), 323–330. https://doi.org/10.1089/met.2007.0008
18. Oporto, G. H., Bornhardt, T., Iturriaga, V., & Salazar, L. A. (2018). Single nucleotide polymorphisms in genes of dopaminergic pathways are associated with bruxism. Clinical Oral Investigations, 22(1), 331–337. https://doi.org/10.1007/s00784-017-2117-z
19. Butovskaya, M. L., Butovskaya, P. R., Vasilyev, V. A., Sukhodolskaya, J. M., Fekhredtinova, D. I., Karelin, D. V., … Lazebny, O. E. (2018). Serotonergic gene polymorphisms (5- HTTLPR, 5HTR1A, 5HTR2A), and population differences in aggression: Traditional (Hadza and Datoga) and industrial (Russians) populations compared. Journal of Physiological Anthropology, 37(1). https://doi.org/10.1186/s40101-018-0171-0
20. Graziela De Luca Canto, DDS, MSc, PhD; Vandana Singh, DDS, MSc; Marcelo E. Bigal, MD, PhD;Paul W. Major, DDS, MSc, FRCD(C); Carlos Flores-Mir, DDS, DSc, FRCD(C), 2014, Association Between Tension-

Type Headache and Migraine With Sleep Bruxism: A Systematic Review, Headache, doi: 10.1111/head.12446

21. Laine, C. M., Yavuz, U., D'Amico, J. M., Gorassini, M. A., Türker, K. S., & Farina, D. (2015). Jaw tremor as a physiological biomarker of bruxism. Clinical Neurophysiology, 126(9), 1746–1753. https://doi.org/10.1016/j.clinph.2014.11.022
22. Yilmaz G, Laine CM, Tinastepe N, Özyurt MG, Türker KS. Periodontal mechanoreceptors and bruxism at low bite forces. Arch Oral Biol. 2019;98:87-91. doi:10.1016/j.archoralbio.2018.11.011
23. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: Report of a work in progress. J Oral Rehabil. 2018;45(11):837-844. doi:10.1111/joor.12663
24. Amorim, C. S. M., Espirito Santo, A. S., Sommer, M., & Marques, A. P. (2018, June 1). Effect of Physical Therapy in Bruxism Treatment: A Systematic Review. Journal of Manipulative and Physiological Therapeutics. Mosby Inc. https://doi.org/10.1016/j.jmpt.2017.10.014
25. Takuro Sakai Takafumi Kato et al, 2017, Effect of clonazepam and clonidine on primary sleep bruxism: a double-blind, crossover, placebo-controlled trial, J Sleep Res, 26,73-83
26. https://www.accessdata.fda.gov/cdrh_docs/pdf16/k160477.pdf
27. https://www.accessdata.fda.gov/cdrh_docs/pdf13/k130797.pdf
28. Martins, O. de F. M., Chaves Junior, C. M., Rossi, R. R. P., Cunali, P. A., Dal-Fabbro, C., & Bittencourt, L. (2018). Side effects of mandibular advancement splints for the treatment of snoring and obstructive sleep apnea: A systematic review. Dental Press Journal of Orthodontics, 23(4). https://doi.org/10.1590/2177-6709.23.4.045-054.oar