

The Relationship of the Trigemino-Cardiac Reflex to Sleep Bruxism



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Abstract

The relationship of the trigemino-cardiac reflex and sleep bruxism

The trigemino-cardiac reflex is well researched in medicine. A newly proposed classification based upon the region stimulated elucidates sleep bruxism's paradoxical effect on the trigemino-cardiac reflex, characterized by tachycardia, hyperpnea and hypertension. This article discusses this complex relationship and the resulting signs and symptoms reported in sleep bruxism, and how it meets these new criterion.

Keywords: Trigemino-cardiac reflex; Trigeminal cardiac reflex; Masseter inhibitory reflex; Sleep bruxism

Abbreviations: TCR: Trigemino Cardiac Reflex; MIR: Masseter Inhibitory Reflex; SB: Sleep Bruxism; GG: Gasserion (trigeminal) Ganglion; GERD: Gastro-Esophageal Reflux Disorder; OSA: Obstructive Sleep Apnea; RF: Reticular Formation; TMD: Temporomandibular Dysfunction; HR: Heart Rate (pulse); MABP Maximum Arterial Blood Pressure; 5-HT: Serotonin

Introduction

The trigemino-cardiac reflex (TCR) is a unique and powerful brainstem reflex that has received a great deal of research interest. Sleep bruxism (SB) is sleep disorder that affects the TCR as well as other brainstem reflexes via stimulation of the brainstem; at the level of the gasserion ganglion (GG). This paper will discuss the unusual relationship of the TCR and SB in addition to how well sleep bruxism meets a new proposed classification system for TCR activation.

Literary Research

Relevant literature was identified through searching PubMed; Research Gate; Google scholar database and Mendeley web library using the search terms "trigemino-cardiac reflex"; "sleep bruxism"; "GERD"; and "masseter inhibitory reflex".

Discussion

Sleep disorders are an increasing health problem in all countries that have a direct effect on quality of life and productivity/safety of workers [1-3]. Sleep bruxism (SB) is a movement type sleep disorder characterized by transient tachycardia; tachypnea and hypertension occurring slightly before; during or slightly after the bruxism event; resolving immediately after the event ceases [4].

This increase in sympathetic activity has been shown to result from stimulation of the TCR at the level of the gasserion ganglion (GGa) [5-7]. SB is seen to occur concurrently with sleep apnea; occurring immediately before or after apnea events; but may also be seen on EMG studies independently of OSA [8]. SB also results in micro-arousals from sleep; classifying it as a true sleep disorder. Epworth Sleepiness Scale scores of 4 to 9 are characteristic of SB whereas scores of 10 and higher are suggestive of OSA4. The daytime sleepiness (males) and tiredness (females) seen in SB have similar deleterious effects on alertness; productivity and quality of life to OSA. In addition; SB is associated with the inception of chronic myofascial pain [9] affecting the orofacial region; tension and migraine type headaches; and temporomandibular dysfunction syndrome (TMD) affecting the temporomandibular joints [9-10].

The TCR is a powerful brainstem reflex that manifests as a sudden onset of hemodynamic influences on heart rate (HR); blood pressure (MABP) and has been associated with cardiac arrhythmias; asystole; apnea and gastric mobility [11]. It is an oxygen-conserving reflex that was first discovered in 1999 [12]; with considerable research ensuing [13-17]. The reflex may be activated by mechanical or chemical stimulation of the trigeminal

nerve at any course along its distribution. Stimulation of the TCR results in neuronal signals being transmitted via the trigeminal nerve to the GGa; continuing to the sensory nucleus of the trigeminal nerve (V5) in the brain stem (mesencephalic nucleus). Signals are then transmitted polysynaptically through the reticular formation (RF); via short internuclear fibers; to the dorsal motor nucleus of the vagus nerve (X). This pathway is considered as an afferent to the TCR (Figures 1-3). Parasympathetic neurons comprise much

of reflex; arising in the motor nucleus of V5. Stimulation of V5 results in bradycardia; hypotension; as well as apnea and gastric hypermobility [17]. The reflex is of utmost importance during surgical procedures adjacent to the branches of the trigeminal nerve as the TCR can inadvertently be stimulated compromising the surgical procedure [18-21]. In procedures near or in the GGa; the opposite effect may be encountered: tachycardia; tachypnea and hypertension and gastric hypomobility [22,23].

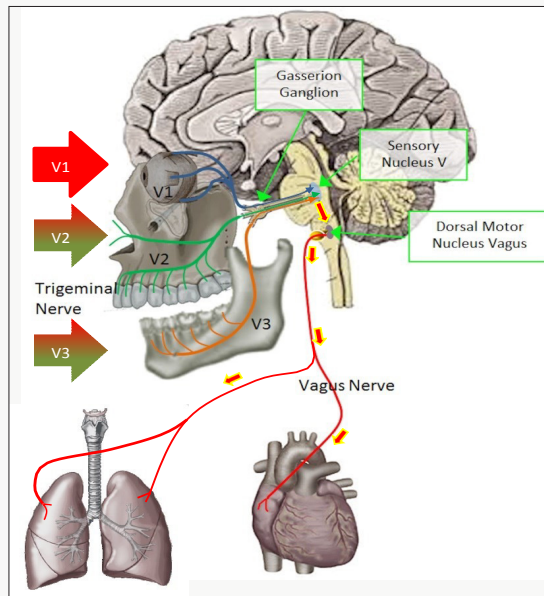


Figure 1: The Trigemino-Cardiac Reflex. Stimulation of the trigeminal nerve from branches V2 and V3 result in stimulation of the TCR very differently from V1, peripheral nerves and post- Gasserion ganglion regions. V2 and V3 can stimulate an increase in heart rate, respiration and blood pressure as opposed to a decrease as seen in the other regions. Only free nerve endings in the periodontal ligaments, the mental nerve and afferents from spindles in the masseter and temporalis muscles act to initiate the stimulation of the TCR at the level of the gasserion ganglion. External sensory nerves of V2 and V3 act to stimulate the TCR as expected. Short internuclear fibers in the reticulum formation connect the trigeminal sensory nucleus down to terminate at the dorsal motor nucleus and vagus nerve.

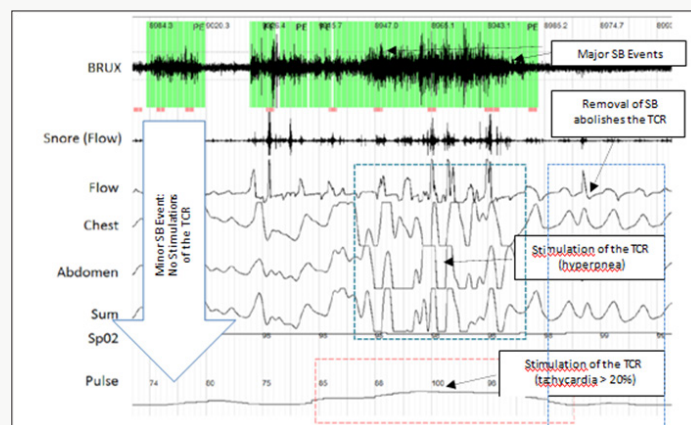
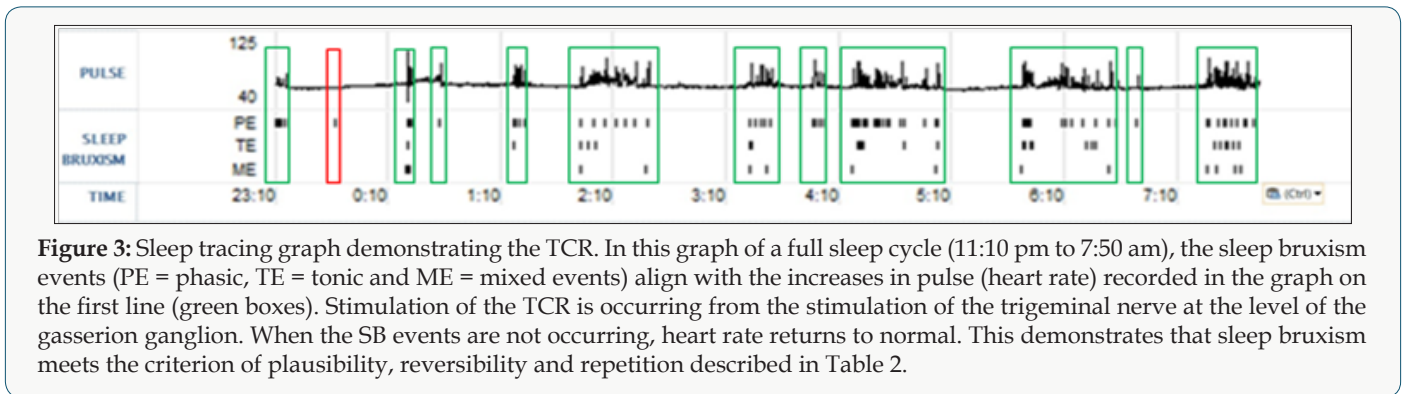


Figure 2: Sleep tracing demonstrating the TCR and Sleep Bruxism. This sleep study tracing demonstrate the effect of sleep bruxism on the TCR. The heart rate increases to 100bpm from 74bpm (a 26% increase) in response to the major SB event. There is also an increase in respiratory effort seen (blue dashed square). The effect disappears immediately after the SB stops. The stimulation of the TCR is obviously occurring at the level of the Gasserion ganglion. In this example, no respiratory events (central or obstructive apneas, hypopneas) occurred and the blood oxygen levels (SpO2) remained constant throughout (98-99%). The minor SB events (blue arrow) do not stimulate the TCR, only the major events do. This tracing demonstrates plausibility and reversibility of the TCR. It also demonstrates a lighter stimulation does not activate the TCR. This meets 4 of the cause and effect criterion proposed by Meuwly et al. (Table 2).



Meuwly; Galoanov et al have proposed a new classification for the TCR based upon where the trigeminal nerve is stimulated (Table 1) [17]. The classic “diver’s reflex” results from stimulation at the level of the first branch of the trigeminal nerve and the typical TCR response of bradycardia; hypotension and hypopnea. It is a cutaneous stimulation of the trigeminal nerve; on the ocular and nasal skin regions. The peripheral classification includes the actual orbit of the eye (by compressing the orbit); the maxillary branch (V2) and the mandibular branch (V3). Only the maxillary and mandibular stimulation regions result in the paradoxical sympathetic response of tachycardia; tachypnea and hypertension (Figures 1-3). In this classification; the only region of stimulation of the TCR occurs at the level of the GGa; all other regions react as expected. This stimulation at the level of the Gasserion ganglion can result in paroxysmal brady or tachycardia; hyper or hypotension and apnea or hyperpnea; depending upon the stimulus. A recent **Table 1:** The Extended Classification of the Trigemino-cardiac Reflex.

study described this phenomenon and concluded from the research that HR was the most significantly affected and the most useful measurement in the determination of TCR [24]. A diagnostic criterion that is generally accepted is of a heart rate decrease (or increase) of 20% or more from baseline; for a positive TCR diagnosis [17]. Table 2 lists other cause-effect relationship criterion proposed to diagnose and differentiate TCR proposed by Meuwly et al [17]. Sleep bruxism meets the first five of seven criteria of Table 2. GGa nerve blocks have an elevated risk profile and do not meet ethical standards for trials; and anticholinergic drugs fail to suppress the TGR effects of SB. By utilizing this additional criterion as well as the decrease (or increase in the case of SB) of HR by greater than 20% ; discrimination of other sympathetic and parasympathetic disorders that could affect HR and mimic the TCR is possible; rendering a more accurate diagnosis of this condition [24].

	Skin, nasal mucosa, the Diver’s reflex	Peripheral		Gasserion Ganglion	Central	Brainstem
		Oculocardiac	Maxillo-Mandibular			
Stimulus	V1	Pressure on Globe	V2 and V3	Direct Stimulation	Stimulation after Ganglion	Direct Stimulation on Brainstem
Heart Rate	Bradycardia	Bradycardia	Bradycardia	Bradycardia and Tachycardia	Bradycardia	Bradycardia
Blood Pressure	Hypertension	Hypotension, Normotension	Hypotension, Normotension	Hypotension or Hypertension	Hypotension	Hypotension
Respiration	Apnea	Apnea	Apnea	Apnea or Hyperpnea	Apnea	Apnea

(C. Meuwly, E. Galoanov, Trigeminal Cardiac Reflex: New Thinking Model About the Definition Based on a Literature Review, Medicine 94(5):e484)

Table 2: Additional Criterion based upon Cause and Effect (2 Positive Criterion = Positive TCR Stimulation).

Plausibility	δ Activation of the TCR must be explainable by stimulation of the trigeminal nerve δ The TCR must occur immediately following the stimulation
Reversibility	δ Removal of the stimulus abolishes the TCR and cardiopulmonary measurements return to normal
Repetition	δ Re-applying the same stimulus results in a recurrence of the TCR δ Lighter stimulus of the same type does not activate the TCR
Prevention	δ Trigeminal nerve block eliminates the TCR δ Application of anticholinergic drugs blocks the reflex

(C. Meuwly, E. Galoanov, Trigeminal Cardiac Reflex: New Thinking Model About the Definition Based on a Literature Review, Medicine 94(5): e484)

Research has also found a genetic commonality in SB sufferers: a polymorphism of the HTR2A gene on chromosome [13-25,26]. This gene codes for 5-HT (serotonin) receptors in the brain and gut. The masseter inhibitory reflex (MIR); another brainstem reflex; is located in the mesencephalic nucleus and has the MIR reflex nucleus affected by this polymorphism [27,28]. The resulting hypersensitivity to serotonin results in loss of inhibition by the MIR as well as activation of central pattern generators involved with chewing. In SB; extreme muscle contractions of the masseter; temporalis and suprahyoid muscle groups result [29]. The HTR2A polymorphism; seen in SB; was found to occur frequently in other conditions including obstructive sleep apnea; tension headaches; migraine headaches (without auras) and a myriad of psychiatric disorders [30-33]. Interestingly; OSA has headaches as a commonly reported symptom; often upon waking (assumed to be due to hypercapnia). In recent studies of the TCR it was demonstrated that; at the connection at the level of the reticular formation and nucleus ambiguus; there appeared to be an endogenous modulation. It was

also found that the 5-HTR1A and 5-HTR2A serotonin reception genes were involved in mediation (stimulation; depression) of the connections (antagonists altered the TCR) [17]. With the 5-HTR2A polymorphism known to exist in SB; it is reasonable to assume that this and other regions could also be similarly affected (potentially resulting in hypersensitivity of the TCR).

During SB bursts; there is an ensuing cascade of events that occurs (Table 3). There are a number of cranial reflexes involved including the TCR; the MIR; the sucking reflex 4 (which can result in trauma of the buccal mucosa and development of linea alba lesions); and the swallowing reflex [34-36]. Scalloped borders of the tongue are also a common finding in SB; resulting from activation of the genioglossus muscle pressing the tongue forcibly against the teeth [37,38]. In other research it has been shown that there is a commonality between SB and gastro-esophageal reflux disorder (GERD); supporting SB's influence on the vagus nerve [39,40]. This can manifest as tooth erosions on the inner or lingual surfaces of the teeth and increase susceptibility to dental caries [41].

Table 3: The Sleep Bruxism Cascade.

Stimulation of the TCR (Gasserion ganglion)		Tachycardia, tachypnea, hypertension
Activation of neural networks involved in sleep		Increase in brain activity, seen as a sleep arousal
Inhibition of the MIR, stimulation of sucking, swallowing reflexes	Stimulation of Central Pattern Generators	Contraction in the suprahyoid groups
		Intense contraction of the masseter and temporalis muscles
		Stimulation of the sucking reflex
		Often concludes with stimulation of the swallowing reflex
TCR trigemino-cardiac reflex, MIR masseter inhibitory reflex		

With the suppression of the MIR; the forces generated during SB events far exceed those of normal chewing [29]. Damage to the teeth is common including:

- a) Sensitive; loose or broken teeth [44]
- b) Abfraction lesions [44]
- c) Accelerated periodontal disease [45]
- d) Formation of mandibular tori [46]
- e) Eagle's syndrome (calcification of the stylohyoid ligaments) [47]
- f) Elongation of the coronoid processes [48]
- g) Excessive compression of the TMJ; often initiating or accelerating degenerative changes [49-51].

As expected; these forces can be a contraindication to some dental procedures including porcelain crowns and inlays [52,53] and dental implants [54,55].

Conclusion

When considering how SB affects the TCR and MIR; the range of signs and symptoms observed during SB events can be readily interpreted. Restless leg syndrome (RLS) is another movement

type sleep disorder that results in activation of the TCR similar to SB. Tachycardia; hyperpnea and hypertension (TCR stimulation); shown to also occur in RLS; has been shown to be a risk factor for heart disease in a number of studies [56,57]. To date SB has not been studied to the extent of RLS and it is not known at this time if SB is also a risk factor for heart disease. With the significant effect of SB on the TCR; Further research is certainly warranted.

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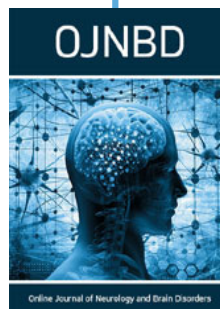
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