Grinding to a halt: Stimulation of the trigeminal cardiac reflex from severe bruxism

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Introduction

The trigeminal cardiac reflex (TCR) is a unique, powerful, and well-established neurocardiogenic reflex that is a result of stimulation along the path of the fifth cranial nerve (trigeminal nerve). It can produce adverse cardiorespiratory changes including hypotension, bradycardia, and asystole, as well as gastric consequences such as hypermotility. This reflex has been reported to occur in various surgical conditions, as well as in neurosurgical interventions.

Sleep bruxism, thought to be a more intense form of rhythmic masticatory muscle activity, has a prevalence of about 8%[1] and has been explicitly linked to the TCR.[2] We report a case of a young woman with severe bruxism who incited her TCR, which subsequently produced profound nocturnal pauses that ultimately required dual-chamber pacemaker implantation.

KEY TEACHING POINTS

- The autonomic nervous system has an intricate relationship with the heart; severe vagal stimulation can produce bradycardia and asystole.
- The trigeminal cardiac reflex is a powerful brain stem reflex that can be associated with a sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, or gastric hypermotility.
- Bruxism, which is reported in 8% of the population, can stimulate the trigeminal cardiac reflex and lead to profound vagal effects on the heart.

Case report

A 27-year-old woman presented with palpitations and syncope. Three years prior to presentation she developed nocturnal and early morning nausea and vomiting that would often wake her from sleep. She was noted to have a long-standing history of severe bruxism with physical signs on examination of significant attrition. This had persisted despite the use of a retainer and bite block. Evaluation with Holter monitoring revealed sinus bradycardia, intermittent second-degree type II atrioventricular (AV) block, and a pause of 8.6 seconds (Figure 1). Interestingly, the rhythm strips showed simultaneous effects on both the sinus and AV node, suggesting an autonomic etiology. Of note, these rhythm disturbances were principally nocturnal in nature. While she was wearing the Holter, the husband was awake and corroborated that she was having severe episodes of bruxism. Further cardiac evaluation was unrevealing, including a normal echocardiogram, cardiac magnetic resonance imaging, sleep study, and thorough autonomic testing. With her constellation of symptoms—severe bruxism, AV nodal block with cardiac pauses (that were predominantly

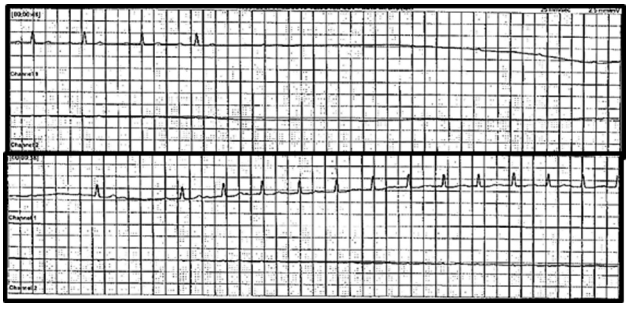

Figure 1 Holter monitoring revealed sinus bradycardia, intermittent second-degree type II atrioventricular block, and a pause of 8.6 seconds

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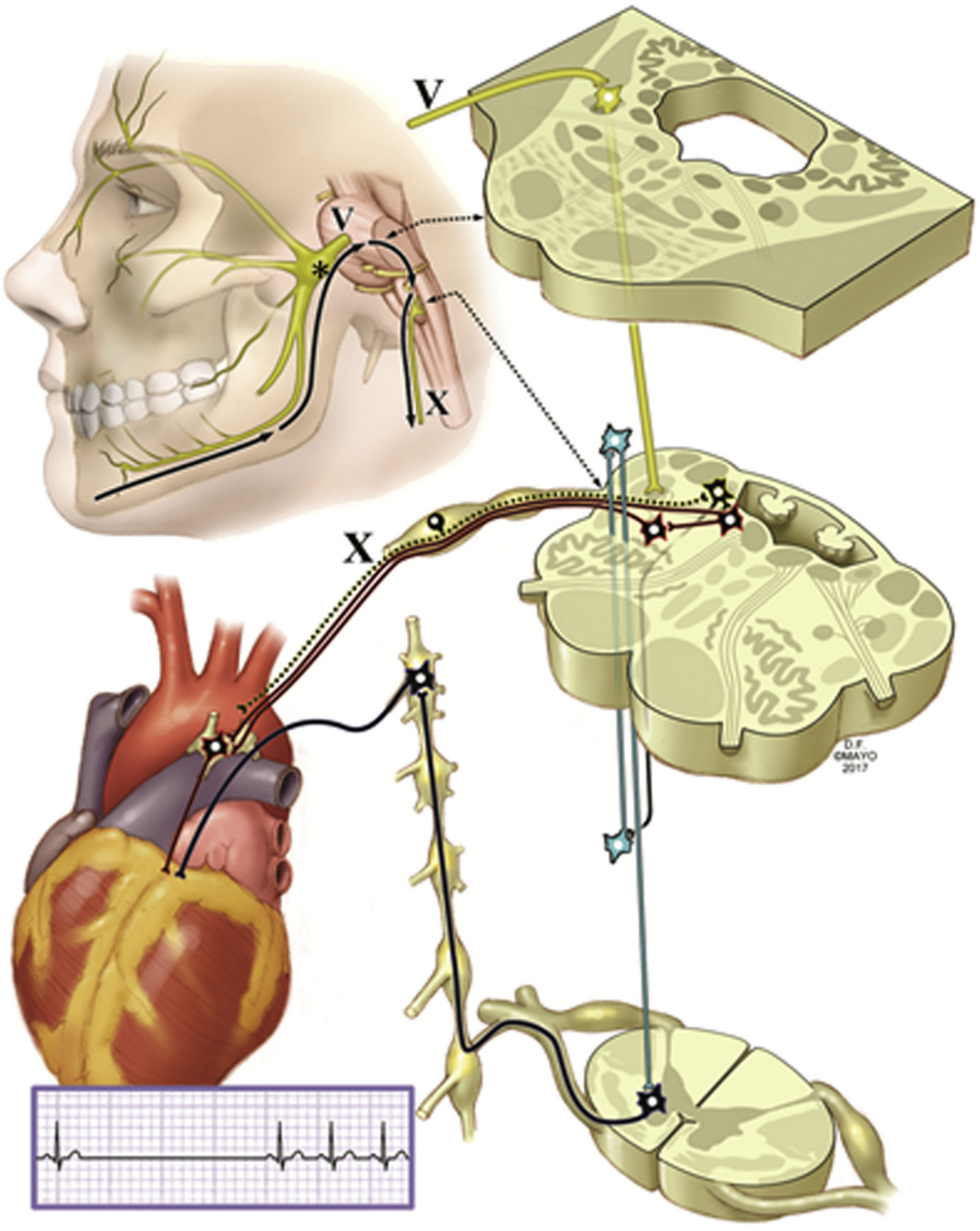

Figure 2 Illustration of the trigeminal cardiac reflex. X = motor nucleus of the vagus nerve; * = Gasserian ganglion; V = trigeminal nerve.

nocturnal), and gastrointestinal symptoms—we diagnosed her with hypervagotonia from stimulation of the powerful TCR from severe bruxism (Figure 2). Out of concern for risk of cardiac death from these pauses without a stable ventricular escape, we elected to place a dual-chamber pacemaker for bradycardic prevention.

Discussion

This case highlights the intricate and noteworthy relationship between the autonomic nervous system and the heart. Our patient developed high-grade AV block and syncope owing to significant and profound hypervagotonia. Based upon her evaluation and corroboration of these events by her husband, we deemed that her intense vagal stimulation was a consequence of her severe bruxism, which was eliciting the TCR.

The TCR (Figure 2) is a powerful brain stem reflex that can be associated with a sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, or gastric hypermotility. The proposed mechanism of this reflex is stimulation of the sensory nerve endings of the trigeminal nerve (Figure 2, cranial nerve V), which sends signals via the Gasserian ganglion (Figure 2, indicated by asterisk) to the sensory nucleus of the trigeminal nerve (Figure 2 inset). The afferent pathway then continues along the short internuncial nerve fibers in the reticular formation to connect with the

efferent pathway, the motor nucleus of the vagus nerve (Figure 2, cranial nerve X). The last part of the reflex is formed by cardioinhibitory efferent fibers, which connect the motor nucleus of the vagus nerve to the myocardium.[3]

Bruxism is a common occurrence in the population (8%) and has been associated with alterations in the autonomic nervous system and stimulation of the TCR.[2,4–6] The mechanism behind the TCR stimulation is felt to be 2-fold. Firstly, masticatory movements (rhythmic masticatory muscle activity) and secondly, teeth contact can stimulate mechanoreceptors in the periodontal tissue.[7] The link between bruxism, TCR, and alteration in the autonomic nervous system is important to highlight as it is well established that the autonomic nervous system plays a critical role in the pathogenesis of various cardiac arrhythmias, particularly atrial fibrillation. Although not specifically related to our patient, the fact that bruxism is so common raises the potential role it could be contributing to autonomic drivers of atrial fibrillation, and this is something that requires further research examination.

Conclusion

When evaluating patients who present with symptoms of significant hypervagotonia, it is important to consider sleep-related causes, in particular sleep bruxism and its role in the TCR.

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