**Case Report/Case Series**

**A Case of Trigeminocardiac Reflex During Infrastructure Maxillectomy**

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**IMPORTANCE** The trigeminocardiac reflex refers to the sudden development of bradycardia or even asystole with arterial hypotension from manipulation of any sensory branches of the trigeminal nerve. Although it has only rarely been associated with morbidity and tends to be self-limited with removal of the stimulus, it is an important phenomenon for head and neck surgeons to recognize and respond to.

**OBSERVATIONS** We present the case of a woman in her late 60s with maxillary alveolar ridge squamous cell carcinoma who developed episodes of asystole and bradycardia during posterior maxillary manipulation for an infrastructure maxillectomy at a tertiary academic medical center. Administration of atropine and removal of the inciting stimulus sufficed to extinguish the episodes and allow procedure completion.

**CONCLUSIONS AND RELEVANCE** The trigeminocardiac reflex can be provoked by a number of head and neck and skull base procedures including parotidectomy and posterior maxillectomy. Surgeons and anesthesiologists should be wary of inciting the reflex during manipulation of trigeminal branches. Careful dissection for prevention and early intervention with stimulus removal and anticholinergic use as needed are paramount to ensure good outcomes.

The trigeminocardiac reflex (TCR) describes the phenomenon of bradycardia or even asystole with arterial hypotension from surgical manipulation of the trigeminal nerve. Although its physiologic importance continues to be evaluated, it seems to play a neuroprotective role in oxygen conservation in response to brain injury. While rarely associated with morbidity and self-limited with removal of the stimulus, it is a clinically important phenomenon for head and neck surgeons to recognize, respond to, and prevent. We describe a woman in her late 60s with maxillary alveolar ridge squamous cell carcinoma who developed episodes of asystole and bradycardia during posterior maxillary manipulation for an infrastructure maxillectomy.

**Report of a Case**

The patient was a woman in her 60s with a T4N0M0 right maxillary alveolar ridge squamous cell carcinoma. She had a history of coronary artery disease, myocardial infarction with coronary artery bypass grafting 29 years previously, hypertension, and hypercholesterolemia. Her medications included atenolol, bupropion hydrochloride, simvastatin, omeprazole, sertraline hydrochloride, trazodone hydrochloride, aspirin, and gabapentin. A right infrastructure maxillectomy was recommended for management of her maxillary alveolar ridge squamous cell carcinoma. Her operation proceeded with an incision through the midline hard palate and gingivobuccal sulcus on the right. The palate and lateral aspect of the maxilla were exposed. A powered drill with a fissured burr was used to make osteotomies along the hard palate and inferior maxilla. The osteotome was used to extend the osteotomies posteriorly. As a posterior osteotomy was being made, the patient developed asystole. The drapes were pulled down, and chest compressions were initiated. After approximately 3 chest compressions were delivered, her heart rate returned to normal. She did not have any hypotension or oxygen desaturations during this episode. After her condition was stabilized, she was redraped and the operation was resumed. During surgical manipulation of the posterior maxillary tuberosity, she suddenly became bradycardic, with a heart rate between 20/min and 29/min. At this point, it was recognized that surgical manipulation was instigating her bradycardia. Atropine was given. Surgery was then completed without further dysrhythmia. The pterygoid muscles were divided with bipolar cautery, and the infrastructure maxillectomy was completed en bloc. The patient recovered in the postanesthesia care unit and had a normal postoperative course without complication.

**Discussion**

The oculocardiac reflex was the first well-recognized TCR phenomenon. It is a subtype of TCR elicited by traction or pressure on the globe or extraocular muscles, sending afferent signals via the trigeminal nerve to the autonomic centers in the brain stem. Additional reflexes are the oculovagal, the caroticotrigeminal, and the vagotrigeminal reflexes. The oculocardiac reflex is the result of stimulation of the oculomotor cranial nerve (CN III), and it is mediated primarily by vagal fibers. Since the TCR, other trigeminal reflexes have been identified. These include the trigeminocardiac reflex, aortic carotid and carotid sinus reflexes, and the prendural and vertebral arteries' reflexes. The oculocardiac reflex has been associated with intraocular surgery and eye movements, whereas the TCR is associated with surgical dissection of trigeminal branches.
the ophthalmic division of the trigeminal nerve.1,5 It was later recognized that surgical manipulation of other branches of the trigeminal nerve could trigger similar phenomena. Although cases of dysautonomia with injury or manipulation of the facial structures had been reported earlier, the TCR was first described and established as a phenomenon in the early 1990s. Since then, reports in the oral maxillofacial surgery literature have identified this phenomenon during orthognathic, eyelid, and facial trauma surgery.6-8 It has also been reported multiple times in the neurosurgery literature in operations on the skull base, cerebellopontine angle, and gasserian ganglion.2,4,9

To our knowledge, the only 2 recent reports of TCR in the otolaryngology literature were of cases that occurred during paranasal surgery and parotidectomy, respectively.10,11 Despite the frequency with which head and neck surgeons manipulate structures innervated by the trigeminal nerve, either during maxillectomies or craniofacial procedures, the TCR has not been commonly observed.

Whereas several procedures have been noted to trigger the TCR, there is poor understanding of the underlying mechanism and functional relevance of this reflex. A current explanation of the mechanism suggests that the afferent pathway of the reflex arc is made up of sensory nerve endings of the trigeminal nerve sending neuronal signals via the gasserian ganglion back to the sensory nucleus of the trigeminal nerve.1 This afferent pathway continues along short internuncial neurons in the reticular formation to connect with efferents from the motor nucleus of the vagus nerve.3,5,6,8 The efferent signals trigger parasympathetic output to cardiac muscle, resulting in a range of responses, including sudden onset of sinus bradycardia, bradycardia terminating in asystole, asystole with no preceding bradycardia, arterial hypotension, apnea, and gastric hypermotility.5,8

The functional relevance of this phenomenon is unclear. It has been suggested that the TCR is actually an endogenous neuroprotective strategy, ie, nerve manipulation causing transiently anoxic sensory neurons to trigger a systemic oxygen-conserving reflex.7 This is manifested as increased cholinergic tone to prevent potential brain injury, indicating that this may in fact be a physiologic reflex as opposed to a pathologic one.3 While an adequate explanation of the physiologic importance of this reflex continues to be delineated, a more clinically relevant endeavor for head and neck surgeons may be awareness of the treatment and potential prevention of the phenomenon.

Conclusions

No established protocol exists for treating effects of the TCR beyond anecdotal evidence. Many case reports indicate that simple removal of the stimulus at the onset of dysrhythmia is sufficient to reestablish sinus rhythm.6,12 Others advise that the nature of the stimulus is important; abrupt and sustained traction is more reflexogenic than smooth and gentle intracranial manipulation of the trigeminal nerve, which does not cause the noxious stimulation that provokes the TCR.5,9 Many authors suggest the use of anticholinergic medications to treat bradycardia, although there is varying success in preventing recurrence.4,6-9 In our case, in which additional reflexogenic manipulation was required to complete the operation, atropine therapy appeared useful to complete our posterior maxillotomy without eliciting bradycardia or asystole. In most cases, normalization is achieved with either removal of the stimulus or anticholinergic therapy and patients do not tend to develop additional complications. However, there are reports of recurrent bradycardia for a prolonged period refractory to anticholinergic therapy.1,8 A recent case report noted successful resolution of anticholinergic-resistant bradycardia with the use of topical lidocaine intraoperatively to blunt the afferent signal transmission.13 One intraoperative death has been reported in association with the TCR,12 but for most patients the prognosis is favorable with careful dissection, early recognition, removal of stimulus, and anticholinergic treatment as indicated.2,10

ARTICLE INFORMATION
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REFERENCES


