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CLINICAL REVIEW

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Intraoperative cardiac arrest etiologies in head and neck surgery: A comprehensive review

Constantine Tarabanis BS[1] | Nicholas B. Abt MD[2] | Heather A. Osborn MD[2]

[1] Harvard Medical School, Boston, Massachusetts

[2] Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, Massachusetts

Correspondence
Nicholas B. Abt, Massachusetts Eye and Ear Infirmary, Department of Otolaryngology, Harvard Medical School, 243 Charles Street, Boston, MA 02114.
Email: nicholas_abt@meei.harvard.edu

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Abstract

Background: The etiologies of intraoperative cardiac arrest within otolaryngology are not well understood as they are rare events.

Methods: A comprehensive review of the etiologies and corresponding pathophysiologic neural mechanisms of intraoperative cardiac arrest in otolaryngologic surgery are examined.

Results: The occurrence of this rare complication has been described in a range of head and neck procedures, including but not limited to suspension laryngoscopy and oncologic resections in the neck, maxilla and thyroid. Three anatomically distinct pathways leading to intraoperative cardiac arrest are described: direct vagal stimulation, the trigeminocardiac reflex and the baroreceptor reflex. All three share the final common pathway of parasympathetic signaling to the sinoatrial node via the cardiac fibers of the vagus nerve.

Conclusion: With a firm understanding of the mechanistic underpinning of this rare phenomenon, otolaryngologic surgeons can be better prepared for its occurrence.

KEYWORDS

arrest, baroreceptor, cardiac, CNXI, etiology, intraoperative, otolaryngology, spinal accessory nerve, surgery, trigeminocardiac reflex, vagal, vagus

1 | INTRODUCTION

Intraoperative cardiac arrest is a rare, yet potentially life-threatening event. Intraoperative cardiac arrest can be defined as the intraoperative complete absence of or presence of a significantly irregular cardiac rhythm, necessitating the initiation of any component of basic and/or advanced cardiac life support.[1] The reported incidence of intraoperative cardiac arrest varies across studies due to differences in sample selection and inconsistent case reporting. A comprehensive literature search did not reveal an incidence estimate for intraoperative cardiac arrest in otolaryngologic cases specifically. Instead, an analysis of 362 767 noncardiac operations (comprised of approximately 20% otolaryngologic cases) conducted between 2005 and 2007 concluded on an incidence equal to 7 per 10 000 noncardiac surgeries.[1] The most important risk factor for intraoperative cardiac arrest occurrence was the amount of intraoperative erythrocyte transfusion. With a 30-day mortality rate of 63%, authors concluded that primary prevention would be the best approach to reducing intraoperative cardiac arrest rates.[1]

The purpose of this comprehensive clinical review was to analyze the causes of intraoperative cardiac arrest after uneventful completion of anesthesia induction and endotracheal intubation in otolaryngologic surgery. In the review of the literature, cases of intraoperative cardiac arrest secondary to preoperative electrolyte imbalances or preexisting cardiac conditions were excluded. It is important for otolaryngologic surgeons to be prepared for this rare complication and be trained to respond appropriately. To this end, this clinical review serves as a guide to the otolaryngologic procedures

Constantine Tarabanis and Nicholas B. Abt contributed equally as first authors.

that could entail this complication, along with accompanying underlying pathophysiologic mechanisms.

1.1 | Trigeminocardiac reflex

The trigeminocardiac reflex (TCR) was first established as a phenomenon in the early 1990s and describes the onset of bradycardia and/or asystole with the surgical manipulation of structures innervated by branches of the trigeminal nerve (V).[2] The afferent pathway of the reflex arc is composed of sensory nerve endings of the trigeminal nerve traveling to the gasserian ganglion and then to the main trigeminal sensory nucleus in the pons. In the reticular formation, short internuncial nerve fibers connect with the efferent pathway, originating in the motor nucleus of the vagus nerve. The efferent pathway triggers parasympathetic output to the heart resulting in bradycardia, asystole, and/or arterial hypotension.[2,3]

It has been suggested that induction of the TCR depends on the nature of the surgical stimulus. More specifically, abrupt or sustained extracranial traction can be more reflexogenic as compared to gentle intracranial manipulation of the trigeminal nerve.[3] Other reported factors influencing the induction of the TCR include young age, light plane of anesthesia, and stimulus amplitude.[4] The physiologic relevance of this reflex is unclear, although it has been hypothesized to serve an oxygen conserving role in transiently anoxic (secondary to surgical manipulation, for example) sensory neurons.[3]

Cases of TCR have been described in literature of fields ranging from neurosurgery to maxillofacial surgery and ophthalmology.[3] To our knowledge, there are only 3 reports of TCR leading to intraoperative cardiac arrest in the otolaryngology literature, occurring during paranasal sinus surgery, parotidectomy, and infrastructure maxillectomy. Sudden onset of bradycardia/asystole occurred during ethmoid sinusotomy, dissection of the left internal maxillary artery, and osteotomy in the inferior maxilla, respectively.[3–5] Presumably, surgical manipulation of structures innervated by branches of the trigeminal nerve precipitated the TCR. For example, in the paranasal sinus surgery case, stimulation of the anterior or posterior ethmoidal nerves, branches of the nasociliary nerve coming from the ophthalmic nerve (CN V1), triggered asystole.[5] In all cases, sinus rhythm recurred with withdrawal of surgical instruments and/or administration of atropine. All operations were completed with no additional complications.[3–5]

1.2 | Direct vagal stimulation

The use of intraoperative neuromonitoring (IONM) to increase the detection rate of the recurrent laryngeal nerve and the external branch of the superior laryngeal nerve in thyroid and parathyroid surgery has been shown to be safe. After resection, a positive signal with vagal stimulation supports the postoperative integrity of the recurrent laryngeal nerve.[6]

In the otolaryngology literature, there have been 2 characteristic cases of cardiac arrest after IONM stimulation of the vagus nerve in 2 patients with no preexisting cardiac disease. One case involved the removal of a follicular thyroid tumor en bloc with the right internal jugular vein, and the other was a resection of a parathyroid adenoma.[7] In both cases, a 1 mA stimulation of the vagus nerve precipitated severe bradycardia and subsequent asystole. In both cases, sinus rhythm recurred after cardiopulmonary resuscitation with external cardiac massage and i.v. ephedrine and/or atropine. The underlying pathophysiologic mechanism was hypothesized to be increased by the parasympathetic output to the sinoatrial node via cardiac fibers of the vagus nerve due to IONM stimulation.[7] Correspondence after this publication underlined the importance of the current amplitude, duration, and frequency of the postresection stimulus in avoiding such occurrences.[8]

1.3 | Baroreceptor reflex

Mechanical compression of the carotid sinus at the common carotid artery bifurcation results in the baroreceptor/carotid sinus reflex.[9] More specifically, compression results in afferent baroreceptor signaling. The afferent limb of the baroreceptor reflex (BRR) is composed of two separate nerves. One is the nerve of Hering, a branch of the glossopharyngeal (IX) cranial nerve, carrying impulses from both the carotid sinus and the carotid body. The other is the aortic depressor nerve, a branch of the vagus, carrying impulses from stretch receptors in the aortic arch. These afferent fibers synapse with secondary neurons in the nucleus tractus solitarius of the dorsal medulla. The efferent limb of the reflex also has 2 components. First, parasympathetic signals to the sinoatrial node via primarily the right vagus nerve decrease the heart rate. Secondly, sympathetic signals to blood vessels result in hypotension.[10] This is the same reflex underlying the carotid massage technique used for the management of supraventricular tachycardia.[11] In certain patients, the carotid sinus can be overly sensitive to manipulation resulting in an exaggerated response with severe bradycardia and/or asystole, a condition known as carotid sinus hypersensitivity (CSH).[9]

Mechanical compression of the carotid sinus in otolaryngologic surgery could be due to direct surgical compression or due to neck hyperextension when positioning the patient. Two case reports of asystole secondary to neck hyperextension can be found in the literature. The first case involved a patient status post total thyroidectomy and radiotherapy now with recurrent medullary thyroid carcinoma undergoing cervical and mediastinal node dissection.[9] The second case

involved a patient undergoing a total thyroidectomy for multinodular goiter.[12] In both cases, the patients had an uneventful tracheal intubation and no preexisting cardiac disease. Asystole resulted immediately after neck hyperextension as the patient was being positioned. Normal sinus rhythm recurred after cessation of neck hyperextension, cardiac compressions, and i.v. ephedrine or atropine. Neither patient had reported a history of CSH.[9,12] However, the patient in the first case did have several risk factors for CSH, including neck malignancy and prior neck irradiation and surgery.[9]

Asystole secondary to direct surgical compression has also been reported in the literature. One case report involved a left maxillary carcinoma resection with a left radical neck dissection in a patient on a β-blocker preoperatively due to a history of poorly controlled hypertension.[13] Upon surgical compression of the left carotid artery during dissection, bradycardia quickly followed by asystole occurred. After chest compressions and administration of i.v. atropine, baseline heart rate and blood pressure were reestablished. The authors argued that asystole was precipitated by activation of the BRR, potentiated by recent β-blocker therapy.

Another otolaryngologic case serves to illuminate the anatomic distribution of the BRR. It involved a patient with no preexisting cardiac disease, status post right carotid body tumor resection with right internal jugular and right vagus nerve excision due to tumor involvement.[10] The patient presented with tumor recurrence involving the right carotid artery and underwent excision and replacement with an autologous vein graft. During this operation, surgical compression of the right carotid artery resulted in a sudden decrease in blood pressure to 0 mm Hg, absent heart sounds on auscultation, but normal sinus rhythm of 76 beats per minute on electrocardiogram. After chest compressions and administration of i.v. epinephrine, blood pressure returned to baseline and the remaining surgery was completed uneventfully.[10] The authors explained that this profound hypotension was likely secondary to BRR activation. The parasympathetic efferent leg of the reflex that would have resulted in a decreased heart rate was absent given before excision of the right vagus nerve. Hence, the efferent leg of the reflex consisting entirely of sympathetic signaling to the vasculature resulted in profound vasomotor collapse.[10]

1.4 | Baroreceptor reflex versus direct vagal stimulation

Suspension laryngoscopy is a common procedure used for the visualization of structures of the larynx allowing for a bimanual surgical technique. Common complications of this procedure include compressive paresthesias of the lingual nerve as well as trauma to structures, such as the lips, teeth, tongue, or oropharynx. A rare complication involves the induction of severe bradycardia or asystole during manipulation of the larynx.[14]

Two plausible pathophysiologic explanations have been offered to explain this phenomenon. The first entails activation of the BRR with compression of the carotid sinus by the laryngoscope.[14] The alternative explanation involves direct vagal stimulation. Glossopharyngeal sensory nerve fibers innervate the pharynx, whereas the internal branch of the superior laryngeal nerve (a sensory branch of the vagus nerve) innervates the laryngeal surface of the epiglottis.[14,15] The laryngoscope could stimulate the latter branch by mechanical irritation of the exposed laryngeal epiglottic surface during suspension. In turn, efferent parasympathetic vagal signaling could lead to downstream bradycardia/asystole.[14,15]

Three cases of bradycardia during suspension laryngoscopy have been reported in the otolaryngology literature. The first case was a resection of a true vocal fold lesion in a patient with well-controlled hypertension.[14] The second case was a laryngeal polypectomy in a patient with no preexisting cardiac disease.[15] The third case involved tracheal dilation for subglottic stenosis in a patient with a history of coronary artery disease and cardiac stent placement.[16] In all cases, bradycardia and/or asystole was induced on placement of the patient in suspension and the heart rate spontaneously recovered with removal of the suspension laryngoscope. Administration of i.v. atropine or glycopyrrolate before suspension prevented bradycardia allowing for the procedures to be completed.[14–16] Importantly, the authors of the first case noted that placement of the laryngoscope on the laryngeal surface of the epiglottis without significant tension did not trigger bradycardia. Instead, significant tension during suspension induced bradycardia regardless of epiglottic contact.[14] Hence, this report offers some support for the BRR-mediated pathophysiologic explanation. In all cases, hypoxia-induced bradyarrhythmia was ruled out, given the absence of significant desaturation on pulse oximetry.[14–16]

1.5 | Spinal accessory nerve stimulation

Preservation of the spinal accessory nerve (SAN), the motor innervation of the sternocleidomastoid and trapezius muscles, has been shown to decrease the postoperative incidence of shoulder morbidity[17] without increasing nodal recurrence rates in oncologic neck dissections.[18] An IONM of the SAN to assist in its detection and preservation in neck dissections has been previously demonstrated to be feasible and safe.[18] To our knowledge, there have been no reports in the otolaryngology literature of adverse cardiac events secondary to direct SAN stimulation. However, bradycardia and asystole after SAN and facial nerve stimulation with a Bard peripheral nerve stimulator while testing for neuromuscular blockade

TABLE 1 Summary of pathophysiologic neural mechanisms underlying intraoperative cardiac arrest in otolaryngologic surgeries and corresponding case reports

Pathophysiologic neural mechanism	Case report	Otolaryngologic procedure	Bradycardia/asystole-inducing maneuver	
TCR	Mohan et al[3] (2014)	Infrastructure maxillectomy	Manipulation of structures innervated by branches of the trigeminal nerve	Ethmoid sinusotomy
	Gupta et al[4] (2013)	Parotidectomy		Osteotomy in the inferior maxilla
	Schipke et al[5] (2013)	Paranasal sinus surgery		Dissection of left internal maxillary artery
Direct vagal stimulation	Almquist et al[7] (2016)	Follicular thyroid tumor resection en bloc with right internal jugular vein	1 mA stimulation of the vagus nerve during IONM	
		Parathyroid adenoma resection		
BRR	Truong et al[9] (2013)	Cervical node dissection for recurrent medullary thyroid carcinoma	Neck hyperextension	
	Lilitsis et al[12] (2016)	Total thyroidectomy of multinodular goiter		
	Higuchi et al[13] (2010)	Left radical neck dissection for left maxillary carcinoma	Carotid artery compression	
	Boyce and Peters[10] (2003)	Recurrent right carotid body tumor excision[a]		
BRR vs direct vagal stimulation	Latuska et al[14] (2016)	True vocal fold resection	Suspension laryngoscopy	
	Ko et al[15] (2010)	Laryngeal polypectomy		
	Redmann et al[16] (2016)	Tracheal dilation for subglottic stenosis		

Abbreviations: BRR, baroreceptor reflex; IONM, intraoperative nerve monitoring; TCR, trigeminocardiac reflex

[a]Baroreceptor reflex manifested as vasomotor collapse without bradycardia/asystole due to past right vagus nerve excision.

during an abdominal hysterectomy has been reported in anesthesia literature.[19]

We hypothesize that asystole secondary to SAN stimulation in oncologic neck dissections remains a possibility given the anatomic distribution of the SAN. The accessory nerve (XI) is formed by its cranial and spinal roots. The spinal accessory nucleus arises from cell bodies in the anterior horn of the upper 5 segments of the cervical cord. These motor neurons join as they exit the spinal cord and ascend into the cranial cavity through the foramen magnum. The cranial root of XI arises from the nucleus ambiguus in the caudal part of the medulla oblongata. Leaving the medulla, the cranial root briefly courses with the "spinal" root of the accessory nerve as they descend to the jugular foramen. At the jugular foramen, the cranial root fibers separate and join the vagus nerve (X) to be distributed within the recurrent laryngeal nerve and possibly the cardiac nerves. Hence, the cranial root of XI is considered both functionally and anatomically part of the vagus nerve.[19] It is hard to see how spread of the intraoperative stimulus to the spinal root of XI could lead to asystole, given its purely motor function. However, spread of the stimulus upstream to the cranial root of XI could result in bradycardia and asystole due to parasympathetic output to the heart via the vagus.[18] Given this anatomic distribution, asystole induced by stimulation of the SAN remains a possibility. All cases in the literature are summarized in Table 1.[3–5,7,9,10,12–16]

2 | CONCLUSION

In this review, we identified 3 anatomically distinct pathways whereby intraoperative cardiac arrest can occur during otolaryngologic surgery. More specifically, direct vagal stimulation and induction of the TCR or BRR have been shown to lead to asystole, with the final common pathway being parasympathetic signaling to the sinoatrial node via the vagus nerve. Understanding the anatomic and surgical considerations underlying intraoperative cardiac arrest in otolaryngologic surgery can aid in avoidance of this rare phenomenon.

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ORCID

Nicholas B. Abt MD http://orcid.org/0000-0002-2083-3523

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