International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Anesthetic implications of Intraoperative Trigemino - Cardiac Reflex: A Systematic Literature Review

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Abstract: ***Trigemino cardiac reflex (TCR) is a sudden physiological response due to the pressure effect, stretching or any stimulus of the largest cranial nerve, the trigeminal nerve (cranial nerve v). TCR is actually an endogenous physiological protective mechanisms found in brain against ischemia. It is one of the oxygen conserving reflexes. Within seconds of initiation of such reflex, there is activation of sympathetic nerves which leads to cerebrovascular vasodilatation. These responses are sometimes exaggerated and put the patient at risk. During the initial period of vagal stimulation, the cardiac depression occurs leading to sinus arrest, asystole or VF Trigemino cardiac reflex (TCR) is a powerful brainstem reflex that manifests as sudden onset of hemodynamic instability with fall in blood pressure, heart rate, apnea and gastric hyper motility during stimulation of any branches of the Trigeminal nerve. This is one reflex that can catch the anesthesiologist off guard on a busy day leading to catastrophic situation intra operatively with bradycardia and asystole hence the need to revisit and review current literature to keep young Anesthesiologists aware and on guard for this reflex. Objectives: 1) Identify the anatomical structures involved 2) Describe the triggers risk factors 3) Review the management of a patient with TCR. 4) Outline strategies for improving early detection and management to improve outcomes.***

Keywords: Trigemino Cardiac Reflex, Intraoperative bradycardia, Asystole, Brainstem reflex

1. Introduction

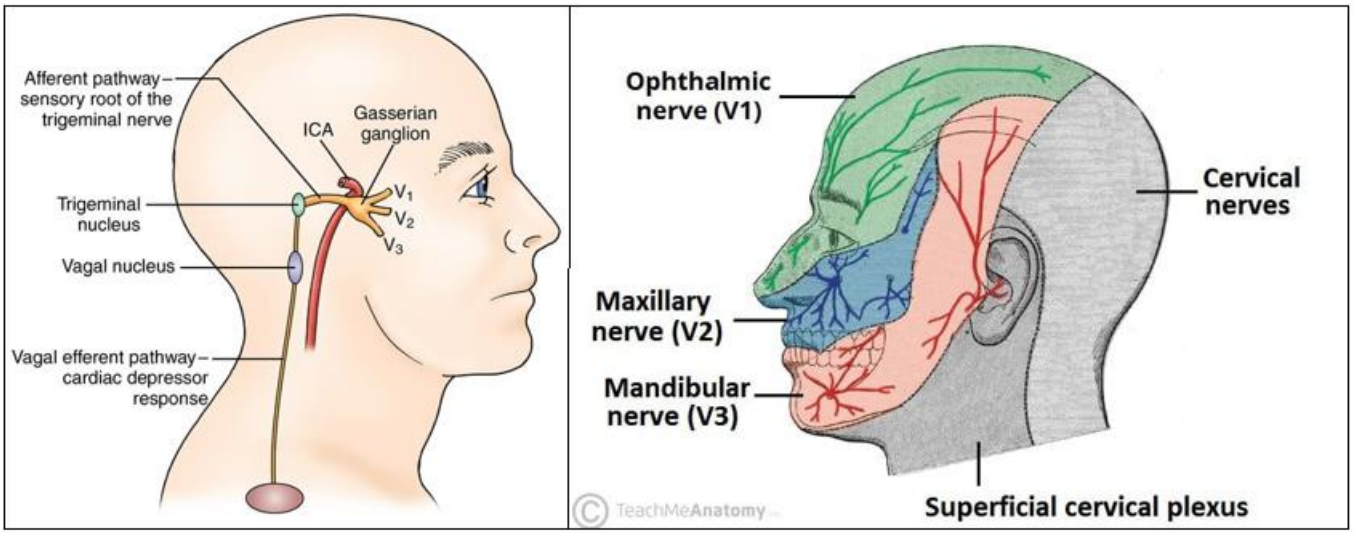
Trigeminocardiac reflex (TCR) is a well - established brainstem reflex that is commonly reported during different skull - base interventions and is a sudden onset of parasympathetic dysrhythmia including bradycardia and asystole, hypotension, apnea as well as gastric hypermotility during the stimulation of any of the sensory branches of the fifth cranial (trigeminal) nerve.

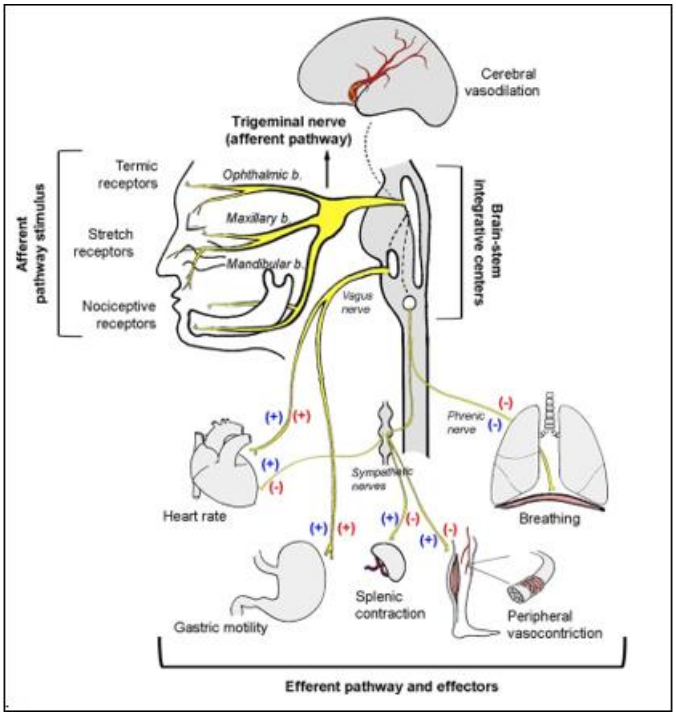
TCR episode can be defined as a sudden episode of *hypotension*, a drop in mean arterial blood pressure (MABP) of 20% or more and *bradycardia*, a drop of heart rate of 20% or more from the baseline, and/or asystole. The occurrence must be preceded with definitive stimuli including physical, chemical or electrical manipulation at or near the vicinity of the trigeminal nerve (peripheral or the central part).

Schaller and colleagues have differentiated TCR into peripheral, central, and ganglion Gasseri (GG) TCR subtypes based on where the afferent impulse originates.

Anatomy and Physiology

The TCR arc consists of an afferent and efferent limb. The trigeminal nerve, otherwise known as cranial nerve V, serves as the sensory afferent limb. The vagus nerve, known as cranial nerve X, comprises the efferent limb of the TCR. This pathway is initiated by the activation of stretch or pain receptors along the distribution of the trigeminal nerve. The three branches of the trigeminal nerve ophthalmic (V1), maxillary (V2) and mandibular (V3) nerves transmit the impulses to the Gasserian ganglion, followed by the trigeminal nucleus, where the afferent limb then terminates in the central nervous system (CNS). The CNS processes this sensory information and internuclear communication will occur between the trigeminal sensory nucleus and the visceral motor nucleus of the vagus nerve. This stimulates the efferent limb, causing impulses to exit the brainstem, transmit to the myocardium to synapse as the sinoatrial node, and activate the vagal motor response. The resultant effects include negative chronotropy, leading to bradycardia.





Types of TCR

There are 3 types of TCR

The central TCR is defined as an origin of the TCR cranial to Gasserian ganglion GG, the peripheral TCR, as distal to GG, and a TCR of the GG as a direct stimulus on the trigeminal ganglion.

Clinical subtype	Trigger point	Heart rate	Blood pressure	Respiration	Gastric motility
Peripheral	Stimulation of V1, V2, V3 innervated area	Bradycardia	Hypotension or normotension (hypertension in diving reflex)	Apnoea	Increased
Central	Stimulation of intracranial part of trigeminal nerve	Bradycardia	Hypotension	Apnoea	Increased
Ganglionic	Direct stimulation of gasserian ganglion	Bradycardia or tachycardia	Hypotension or hypertension	Apnoea	Increased

Common Triggers for TCR

Neurosurgery

TCR needs to be kept in mind for different neurosurgical and skull - base procedures, including cerebellopontine surgery, cranioplasty, transsphenoidal surgery, microvascular trigeminal decompression (the Jannetta procedure), and clipping of aneurysms and other skull - base tumors. Besides these classical surgical approaches, other neurosurgical procedures, such as subdural hematoma/ empyema evacuation

Maxillofacial Surgery

TCR can occur while surgeons deal with procedures on Maxilla, mandible, temporomandibular joint (TMJ) surgery and orthognathic surgery

ENT & Cosmetic Surgery

Nasal surgeries like rhinoplasty and palate surgery

Ophthalmology

The oculocardiac reflex (OCR), also known as the Aschner reflex or trigeminovagal reflex (TVR), was first described in 1908 as a reduction in heart rate secondary to direct pressure placed on the eyeball. It is defined by a decrease in heart rate by greater than 20% following globe pressure or traction of the extraocular muscles. This reflex has most notably been depicted during ophthalmologic procedures, more specifically during strabismus surgery.

Activation of the OCR (oculocardiac reflex) through the ophthalmic branch (V1) has also been associated with noncardiac consequences. Due to the activation of the vagal motor response, other reported vagal effects include hypotensive episodes, syncope, and gastrointestinal responses such as nausea and vomiting. The latter has been questioned to have an impact on the severity of postoperative nausea and vomiting (PONV). In the pediatric population, the incidence of PONV has been reported to be as high as 85% following strabismus surgery and is the most common reason for inpatient admission following an outpatient procedure. As a

fatigable reflex, the OCR's intensity will decrease with multiple, repeated stimuli

Anesthesia and Pain clinic: TCR may be additionally activated by mandibular extension, facial trauma surgery, regional anesthetic nerve blocks in trigeminal area, and mechanical stimulation**.**

Diving Reflex (DR) is similar to the TCR, the DR also acts as a protective oxygen - conserving reflex and aims to keep the body alive during cold water immersion. During diving into cold water the strong cold stimulus to face and breath holding the body goes into bradycardia, apnea and vasoconstriction to survive the hypoxia under water. This is an adaptation seen in mammals to survive while diving in cold water

Predisposing Factors

Some of the predisposing factors listed by Campbell et al are:

- Children due to high resting Vagal tone
- Males
- High sympathetic activity Hypoxemia
- Hypercarbia
- Light plane anesthesia.
- Drugs like Neuromuscular blockers, Opioids, beta blockers
- Strength and duration of stimulus

2. Prevention and Management

Incidence of TCR is bound to occur with any type of surgical procedure in the distribution of trigeminal nerve and hence its importance should not be under estimated. Arasho et al had summarized the management of TCR as the following

1) Identification of risk factors and their modification
2) Prophylactic treatment using vagolytic drugs and / or peripheral nerve blocks in procedures involving manipulation of structures in the trigeminal area
3) Cardiovascular monitoring during anesthesia

Controlled ventilation is absolutely essential in monitoring of arterial oxygen saturation and end tidal CO2 to prevent hypercarbia and hypoxemia.

Interaction between the anesthetist and surgeon before the surgery and during the surgical procedure especially when a traction or elevation is done in craniofacial region is important. In the event of an episode the stimulus has to be stopped immediately

Most importantly, preoperative infiltration of the possible afferent pathway to achieve local anesthesia should block the response and is highly recommended in craniomaxillofacial surgeries involving manipulation of the trigeminal nerve branches.

Prophylactic administration of glycopyrrolate is debatable.

The type of stimulus, strength and the duration of stimulus are to be considered. The depth of anesthesia is an important factor. In deeper anesthesia planes, the activation of TCR is minimal.

Pharmacological agents such as potent narcotics like sufentanil and alfentanil, beta - blockers, and calcium channel blockers may predispose to TCR.

The clinical importance of the TCR lies in the fact that its clinical features range from sudden onset of sinus bradycardia, bradycardia terminating asystole, asystole with no preceding bradycardia, arterial hypotension, apnea, and gastric hypermobility.

Recognition of bradycardia is the first step in treatment

Most cases are associated with only a 10% to 50% heart rate reduction and sinus rhythm usually returns to baseline upon stimuli cessation.

Most cases of TCR will therefore resolve spontaneously without any other therapeutic measures. If resolution does not happen during a reasonable amount of time after cessation of the surgical stimulus, atropine or glycopyrrolate should be administered intravenously.

Atropine would be given before epinephrine only if bradycardia was thought to be attributable to vagal stimulation and not due to some other cause such as hypoxia. Cardiac massage should be reserved for the cases in which routine treatment measures fail to reestablish the expected cardiac activity.

3. Conclusion

Maxillofacial, Neurosurgery, Ophthalmology, ENT surgeons and the Anesthesiologist should be familiar with TCR to combat with this sudden physiological response which may be even fatal at times.

The key points that should be kept in mind are:

- Abrupt and sustained traction of craniofacial structures should be avoided and stimulus stopped in the event of a TCR episode
- Administration of regional nerve block in the operating site especially if hypotensive anesthesia is planned
- Administration of glycopyrrolate (vagolytic agent) + lignocaine prophylactically
- Continuous cardiac monitoring, adequate oxygenation and etco2 monitoring
- If TCR found to be activated, removal of stimulus and administration of vagolytics atropine/glycopyrolate is to be done
- If refractory to vagolytic drugs, epinephrine is to be added.
- Cardiac massage is reserved for cases where normal cardiac activity is not established with above treatment

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