

Trigemino Cardiac Reflex and Its Importance in Maxillofacial Surgery- A Review

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Abstract: Trigemino cardiac reflex (TCR) is a sudden physiological response due to the pressure effect or stretching of the largest cranial nerve, the trigeminal nerve. TCR is a triad of bradycardia, bradypnea and gastric motility changes due to the efferent activation of the vagal nerve in response to the pressure distribution in V₅. TCR was originally called as oculo cardiac reflex (OCR) and the terminology was changed because the response is not only limited to the ophthalmic branch, but for the entire nerve V₅. Activation of TCR is seen in post traumatic patients and during surgical manipulation in cranio facial surgeries. TCR is more common in children with orbital fractures and in extra ocular surgeries. It is well documented in cases of ophthalmic injuries and ocular surgeries. It is imperative to know about this sudden physiological response in maxillofacial surgery which is bound to happen with any oral surgical procedures ranging from extractions, elevation of palatal flaps to maxillary disimpactions.

Knowledge of the TCR is essential as it may mimic a closed cranial injury or a cardiac dysarrhythmia in a post traumatic patient to avoid unwarranted surgical intervention. A detailed ophthalmic examination in maxillofacial injuries is essential. Whenever surgery is planned in maxillofacial region and if TCR is anticipated a discussion with the anaesthesiologist is mandatory. This paper reviews about the clinical papers and abstracts on TCR relevant to maxillofacial surgery.

Keywords: Achener Phenomenon, bradycardia, Oculocardiac reflex, Trigemino-cardiac reflex, Trigemino-vagal reflex.

I. Introduction

Trigemino cardiac reflex, previously called as oculo cardiac reflex is a cascade of physiological reactions secondary to the pressure effects in distribution of the largest cranial nerve - the trigeminal nerve. This unexpected phenomenon is usually seen in orbital injuries and during surgical manipulation of craniofacial structures in the distribution of trigeminal nerve. Maxillofacial surgeons should be aware of this phenomenon of TCR even though it is not common in day to day practise.

II. Discussion

Background

Florian Kratschmer described the influences of reflexes produced by nasal mucosa on breathing and circulation¹. Schaller described development of cardiac dysrhythmia upto asystole, arterial hypotension, apnoea and gastric hypermotility on manipulation of nasal mucosa on cats and rabbits². Kumada et al in 1977 described the reflex bradycardia through neural stimulation in rabbits³. It is well documented that stimulation of nasal mucosa causes bradycardia, bradypnea and blood pressure changes and these reflexes were abolished by applying local anaesthetics to the distribution of V₅ nerve⁴.

The TCR was first described as oculo cardiac reflex in 1908⁵. OCR was described as pressure induced neural reflex that causes cardiac depression through the stimulation of vagal nerve. Change of ten percent or more in the heart rate or dysrhythmia as compared to the control was taken as positive OCR⁶. This cascade of symptoms is not only limited to the ophthalmic branch of V₅, they are induced by pressure effects or stretching of entire division of V₅ and hence Shelly and Church coined the term trigemino cardiac reflex⁷.

Mechanism

The afferent limb is the sensory fibres of the trigeminal nerve which sends signals to trigeminal sensory nucleus via Gasserian ganglion. The afferent arm is connected to the efferent pathway via short internuncial fibres in the reticular formation and connecting the motor nucleus of the vagus nerve. The efferent travel and end in muscarinic receptors of heart causing vagus mediated negative chronotropic and ionotropic responses in the heart. The efferents also travel to the stomach which increases gastric motility.

TCR is actually 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 cerebro vascular vasodilatation. These responses are exaggerated and put the patient at risk. During initial period of vagal stimulation, the cardiac depression is peak leading to sinus arrest, asystole or ventricular fibrillation⁸.

Maxillofacial Literature Review

I) TCR reported in fracture orbit and zygoma and its management

AUTHOR	YEAR	TCR observed
Baiton and Lizi ⁹	1987	Cardiac asystole during surgery for zygomatic arch fracture
Loewinger et al ¹⁰	1987	Bradycardia - elevation of zygomatic arch
Shearer and Wensione ¹¹	1987	Bradycardia - elevation of zygomatic arch
Kosaka et al ¹²	2000	OCR induced by fracture zygoma. Initial diagnosis was total A-V block. Patient underwent cardiac pacing. After fracture repair, the dysrhythmia disappeared and pacemaker removed on first post operative day
Lynch and Parker ¹³	2000	Bilateral penetrating ocular injuries
Yilmaz ¹⁴ et al	2006	TCR developed after 48 hours in an orbital trauma patient with intra orbital metallic foreign body
Schaller ¹⁵ et al	2006	Delayed TCR induced by intra orbital foreign body
Lubbers ¹⁶ et al	2010	TCR observed during repositioning of zygoma and optic nerve manipulation

II) TCR reported in minor oral surgeries

Cha et al ¹⁷	2002	Asystole during bilateral peripheral rhizotomies to treat trigeminal neuralgia
Webb and Unkel ¹⁸	2007	TCR observed during flap elevation for removal of mesiodens
Arakeri and Arali ¹⁹	2010	TCR observed during extraction of tooth
Krishnan et al ²⁰	2011	Recommendation on use of suitable nerve blocks to prevent TCR in maxillofacial surgery

III) TCR reported in maxillary and mandibular osteotomies

Reaume and McNicol ²¹	1988	TCR observed during Lefort I osteotomy in a patient of mandibulofacial dysostosis
Ragno et al ²²	1987	Several episodes of ventricular asystole during downfracture of maxilla (Lefort I osteotomy)
Precious and Skulsky ²³	1990	Bradycardia or asystole during advancement of maxilla
Lang et al ²⁴	1991	Combination of bradycardia and asystole during maxillary and mandibular osteotomies

Bohuli et al ²⁵	2010	TCR observed in maxillary osteotomies
Bohuli et al ²⁶	2011	TCR in mandibular osteotomies
Robideaux ²⁷	1978	OCR during mid face disimpaction
Bainton et al ²⁸	1990	Sinus arrest during bitemporal approach for treatment of panfacial fracture
Precious and Skulsky ²³	1990	TCR during manipulation of temporalis muscle grafting in a case of total bony ankylosis
Roberts et al ²⁹	1999	TCR observed during temporomandibular joint arthroscopy in a 29 year old woman
Puri et al ³⁰	2011	TCR in juvenile nasopharyngeal angiofibroma embolisation
Potti et al ³¹	2011	TCR in percutaneous injection of ethylene vinyl alcohol copolymer in a juvenile nasopharyngeal angiofibroma
Yorgancilar et al ³²	2012	TCR observed in 8.3% of study patients following lateral osteotomies and nasal pyramid fracture procedures in rhinoplasty
Wartak et al ³³	2012	TCR observed in a facial injury in a 56 old male
Schames et al ³⁴	2012	Sleep bruxism as a cause for inducing TCR

Predisposing Factors

Hypercarbia, hypoxemia, insufficient anaesthesia and nature of stimulus are the pre disposing factors. It is more in children because of high resting vagal tone². It is observed that OCR normally fatigues with repetitive stimuli²⁴. Lubbers et al¹⁶ classified various facial surgery into low (TMJ surgeries, Le Fort I osteotomy, elevation of zygoma), medium (skull base surgeries), high risk surgeries (ophthalmic surgeries, orbital exenteration and fracture in children with cardiac disease) for the precipitation of TCR. Campbell R et al³⁵ tabulated the risk factor that precipitated TCR (table 1). So it is imperative for any craniofacial surgeon to know about TCR.

Predisposing and risk factors for trigeminocardiac response- Table 1

PREDISPOSING AND RISK FACTORS	
1	Children
2	Males
3	High sympathetic activity
4	Hypoxemia
5	Hypercarbia
6	Light anaesthesia
7	Neuromuscular blockers
8	Opioids
9	β Adrenergic blockers
10	Strength and duration of stimulus

Prevention and Management

Incidence of TCR is bound to occur with any type of oral surgical procedures and hence its importance should not be under estimated. Arasho et al³⁶ had summarised the management of TCR as 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 V₅
3. Cardiovascular monitoring during anaesthesia

Most importantly, preoperative infiltration of the possible afferent pathway to achieve local anaesthesia should block the response and is highly recommended in craniomaxillofacial surgeries involving manipulation of the trigeminal nerve branches. Prophylactic administration of glycopyrolate is debatable. The type of stimulus, strength and the duration of stimulus are to be considered. The depth of anaesthesia is an important factor. In deeper anaesthesia planes, the activation of TCR is minimal. Controlled ventilation is absolutely essential in

monitoring of arterial oxygen saturation and end tidal CO₂ to prevent hypercarbia and hypoxemia. Interaction between the anaesthetist and surgeon before the surgery and during the surgical procedure especially when a traction or elevation is done in craniofacial region is important

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

III. Conclusion

Maxillofacial surgeons 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:

1. Abrupt and sustained traction of craniofacial structures should be avoided
2. Administration of regional nerve block in the operating site especially if hypotensive anaesthesia is planned
3. Administration of glycopyrrolate (vagolytic agent) + lignocaine prophylactically
4. Continuous cardiac monitoring, adequate oxygenation and watching for additional CO₂ waves
5. If TCR found to be activated, removal of stimulus and administration of glycopyrrolate is to be done
6. If refractory to vagolytic drugs, epinephrine is to be added.
7. Cardiac massage is reserved for cases where normal cardiac activity is not established with above treatment

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