TRIGEMINO CARDIAC REFLEX IN ORAL & MAXILLOFACIAL SURGERY – A REVIEW

Clinical Research

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ABSTRACT

Trigemino cardiac reflex (TCR) which was originally called as OCCULOCARDIAC REFLEX is a physiological response due to the pressure effect on the largest cranial nerve, the trigeminal nerve. Oral and maxillofacial procedures can induce the development of this reflex. 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 Trigeminal nerve. TCR may be generated as a result of procedures or conditions that increase intraocular pressure, strabismus surgery, nasal packing after rhinoplasty, the reduction of zygoma and zygomatic arch fractures, elevation of bone flap or osteotomies, reflection of a palatal flap for removal of a mesiodens, during Le Fort I downfractures, sagittal split ramus retraction, midface disimpaction, cutting maxillary tuberosity, and temporomandibular joint arthroscopy.

The purpose of this paper is to discuss the pathophysiology and to review the main risk factors, treatment, prevention and management with emphasis on the role of maxillofacial surgeons and attending anesthetist. Maxillofacial surgeons should be familiar with presentations, preventive measures for the effective management of this complication.

KEYWORDS

Aschner Phenomenon, Bradycardia, Bradypnea, Oculocardiac reflex, Trigemino cardiac reflex, Trigemino vagal reflex,

INTRODUCTION:

Trigemino cardiac reflex, previously known as oculo cardiac reflex, may serve as a shocking event in the course of operation involving the head and neck region both for surgeons and anesthesiologists.¹ Trigeminal nerve is the largest of the cranial nerves, and it provides sensory supply to the face, scalp, and mucosa of the nose and mouth.² Any surgical intervention in the distribution of trigeminal nerve poses a risk of precipitating trigemino cardiac reflex.³ Trigemino cardiac reflex is also known as Aschner phenomenon, Oculo cardiac reflex or trigeminal vagal reflex.

BACKGROUND:

Joseph Breuer described the self-regulation of breathing through the vagus nerves in 1868, and Florian Kratschmer described the influences of reflexes of the nasal mucosa on breathing and circulatory in 1870.¹ Kratschmer described, a sudden development of cardiac dysrhythmia up to asystole, arterial hypotension, apnea, and gastric hypermotility was observed on manipulation of the nasal mucosa based on the studies on cats and rabbits.⁴ It is documented that these reflexes were fully abolished with the application of local anesthesia to the trigeminal nerve divisions.⁵ The TCR was first described as OCCULOCARDIAC reflex in 1908 by BERNARD ASCHER AND GUISEPPE.⁷ The term trigemino cardiac reflex was described by two anesthetists, Shelly and Church in the year 1988.⁸ Because the reflex does not appear to be limited to the ophthalmic branch and in order to be more inclusive and anatomically descriptive of the response, Shelly and Church suggested changing the name to Trigemino cardiac (TCR) or Trigemino vagal Reflex (TVR).⁸

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.⁹

BIOLOGIC RELEVANCE:

The TCR is seemingly an example of these physiological protective mechanisms detected in brain against ischemia.⁹ It may be part of a group of related responses generally defined as "oxygen-conserving reflexes."¹⁰ Within seconds after the initiation of such a reflex, there is a

powerful and differentiated activation of sympathetic nerves and consequently a primary cerebrovascular vasodilatation. However, these seemingly physiologic functions may become exaggerated and put the patients at risk. During the initial period of vagal stimulation, the resultant cardiac depression reaches a peak level, and it is at this critical time that sinus arrest, asystole, or ventricular fibrillation can occur.¹⁰

TRIGEMINOCARDIC REFLEX PATHWAY AND ARC :

Figure1: www.googleimages.com

Figure2: www.googleimages.com

PREDISPOSING FACTORS:

Hypercarbia, hypoxemia, and insufficient anesthesia are predisposing factors in the occurrence of TCR.⁹ Also, the nature of the provoking stimulus, meaning its strength and duration, contribute to the significance of the heart rate and blood pressure decrease.¹⁰ It occurs more pronouncedly in children due to the higher resting vagal tone.⁴ Pharmacological agents such as potent narcotics like sufentanil and alfentanil, beta-blockers, and calcium channel blockers may predispose to TCR.¹⁰ It has been claimed that OCR normally fatigues with repetitive stimuli.⁷

Lubbers et al¹¹ 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 al¹² tabulated the predisposing and risk factors for trigeminocardiac response.

Table 1: Predisposing And Risk Factors For Trigemino-cardiac Response

	PREDISPOSING AND RISK FACTORS
1	Children
2	Males
3	High Sympathetic Activity
4	Hypoxemia
5	Hypercarbia
6	Light Anesthesia
7	Neuromuscular blockers
8	Opioids
9	Beta Adrenergic Blockers
10	Strength and Duration of Stimulus

MAXILLOFACIAL LITERATURE REVIEW:

- I) TCR reported in fracture orbit and zygoma and its management. Lubbers et al¹¹ in the year 2010 reported TCR during the repositioning of zygoma and optic nerve manipulation. Schaller et al⁴ in the year 2006 reported delayed TCR induced by intraorbital foreign body.
- II) TCR reported in minor oral surgeries. Cha et al¹⁴ in the year 2002 reported asystole during bilateral peripheral rhizotomies to treat trigeminal neuralgia. Webb and Unkel¹³ in the year 2007, reported TCR during flap elevation for removal of mesiodens. Arakeri and Arali¹⁵ in the year 2010 observed the TCR during extraction of tooth.
- III) TCR reported in maxillary and mandibular osteotomies

Reaume and McNicoll¹⁶ in the year 1988 observed TCR during LeFort I osteotomy in a patient of mandibulofacial dysostosis.

Ragno et al¹⁷ in the year 1987 reported several episodes of ventricular asystole during downfracture of maxilla in LeFort I osteotomy.

Precious and Skulsky¹⁸ in the year 1990 reported asystole during advancement of maxilla and also during manipulation of temporalis muscle grafting in a case of total bony ankylosis.

Roberts et al¹⁹ in the year 1999 observed TCR during temporomandibular joint arthroscopy in a 29 year old woman.

PREVENTION AND MANAGEMENT:

Arasho et al² have 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 trigeminal nerve
3. Cardiovascular monitoring during anaesthesia particularly in cases with risk of reflex phenomenon.
4. In case of an event, cessation of the manipulation of the nerve with consideration of administration of vagolytic agents like glycopyrrolate or adrenaline in cases on asystole.

Controlled ventilation is the norm, along with monitoring of arterial oxygen saturation and end tidal carbondioxide to prevent both hypoxemia and hypercarbia.³ Depth of anesthesia is an important factor

as the activation of TCR is minimal in deeper anesthesia planes. Recognition of bradycardia is the first step in treatment.¹⁰ 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.¹³ Fortunately, most cases are associated with only a 10% to 50% heart rate reduction and sinus rhythm usually returns to baseline upon stimuli cessation.¹⁰ 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 maneuver (refractory bradycardia, asystole, or the development of bradycardia with hypotension), atropine or glycopyrrolate should be administered intravenously.⁷ Atropine would be given before epinephrine only if bradycardia was thought to be attributable to vagal stimulation and not some other cause such as hypoxia.¹³ Cardiac massage should be reserved for the cases in which routine treatment measures fail to reestablish the expected cardiac activity.¹⁰

CONCLUSION:

Maxillofacial surgeons should be aware of the potential for its occurrence in surgical procedures, provoking factors, prevention and management to combat with this sudden physiological response which may even fatal at times.

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