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Trigeminal cardiac reflex in faciomaxillary surgery

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Abstract: *Background:* Trigeminocardiac reflex is a physiologic response of the body to pressure effects in the region of distribution of the trigeminal nerve. Since oral and maxillofacial surgical procedures can induce the development of this reflex as most of them are in the distribution of trigeminal nerve, which leads to significant changes in the heart rate and sinus rhythms. This study intends to evaluate the effects of this reflex in patients with facial fractures and its subsequent management. *Methods:* A total of 40 patients with facial fractures who presented to department of Oral and Maxillofacial Surgery Govt Dental College Srinagar. *Results:* A male preponderance is observed with the most susceptible age group being 21–30 years. Fractures included 19 zmc fractures 9 leefort fractures and 12 mandibular fractures. A relative bradycardia was observed in the patients with midfacial trauma during the surgical reduction of midfacial fractures, which improved after completion of procedure in most of the patients. However, in one patient, the bradycardia progressed to a cardiac asystole during midface manipulation necessitating immediate halt of the procedure and intravenous administration of atropine. *Conclusion:* Trigeminocardiac reflex though physiologic, which usually tends to subside without complications is not to be neglected in the surgeries of the maxillofacial skeleton. A propensity for unforeseen complications due to this reflex has to be avoided by meticulous electrocardiography monitoring.
Keywords: Trigeminal Cardiasc Reflex, Asystole, Bradycardia.

Introduction

Trigeminal cardiac reflex (TCR) also called as occulocardiac reflex is a phenomenon which occurs secondary to the pressure effects on the distribution of trigeminal nerve. This effect is seen in the maxillofacial region while manipulating for orbital injuries or in the reduction of facial fracture in the region of trigeminal nerve and stimulation thereof .Considered as one of the most powerful autonomic reflexes in humans, it was first observed by Kratschmer in 1870 [1], the *peripheral* variant of the TCR was established as Aschner - Dagnini reflex (and later known as oculocardiac reflex) by Aschner and Dagnini in the year 1908 [2]. Kumada and colleagues described it as trigeminal-depression response[3]. The term trigeminal cardiac reflex was coined by Shelly and Chruch [4].

TCR was described as vagal nerve stimulated and induced neural reflex that causes cardiac depression. The change of ten percent or more in the heart rate or dysrhythmia as compared to the control was taken as positive TCR. Florian Kratschmer described the influences of reflexes produced by nasal mucosa on breathing and circulation[5]. The development of cardiac dysrhythmia upto asystole, arterial hypotension, apnoea and gastric hypermotility on manipulation of nasal mucosa on cats and rabbits was described by Schaller [2]. The reflex bradycardia through neural stimulation in rabbit was described by Kumada et al in 1977 [6].

The stimulation of nasal mucosa causes bradycardia, bradypnea and blood pressure changes and these changes are well described and these changes are abolished by applying local anaesthetics to the distribution of V5 nerve [7]. So TCR is defined as the sudden onset of sinus bradycardia (heart rate [HR] <60 beats/min and mean arterial pressure >20% lower than the baseline) [8]. Because this definition cannot be applicable to all cases including those with a <20% value and to avoid the underestimation caused by this

definition, Abdulazim *et al*., came with a more inclusive and simplified version as "any sudden onset of relative bradycardia upon the stimulation of any of the 3 branches of the trigeminal nerve [9].

Material and Methods

- A total of 40 patients who reported with for the treatment of facial fractures to the department of Oral and Maxillofacial surgery Govt Dental College Srinagar. The patients who were evaluated were with zygomatic, orbital, midface or orbital fractures who needed intervention amounting to manipulation. An informed consent was obtained from the patients and treatment outcome was explained to the patient. The study was conducted according to the Helnisky declaration. An ethical clearance was obtained for the study. The results were entered into master sheet and calcuted. 40 patients who needed treatment for different types of facial fractures.
- 32 males and 8 females were studied.
- Only ASA 1 patients enrolled.
- Fractures included 19 zmc fractures 9 lee fort fractures and 12 mandibular fractures.

Results

Out of 40 patients studied 4 patients with zygomatic fractures suffered TCR. The total patients with zygomatoic fracture were 19 (Fig 1 and FIG 2). Out of 9 patients with lee fort fracture I noticed TCR. Asystole was noticed in 1 patient who had multiple fractures. We didn't find any positive reflex in any mandibular fracture patients. A total of 6 patients observed positive reflex out of which one was asystole.

Fig-1: Reduction of zygomatic arch fracture

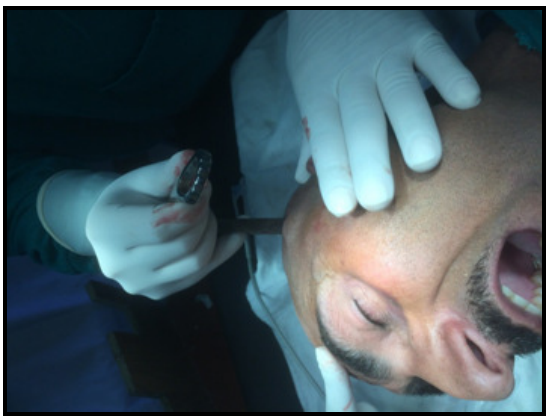
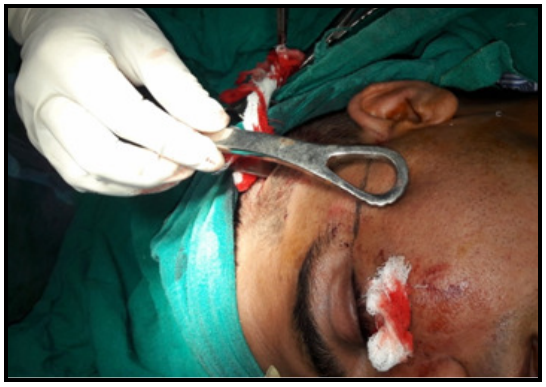


Fig-2: Zygoma fracture reduction



Mechanism of reflex: The sensory fibres of the trigeminal nerve serve as afferent limb 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 muscuranic receptors of heart where vagus mediated negative chronotropic and ionotropiic responses are effected. The efferents also travel to the stomach which increases gastric motility.

It is a physiological protective mechanisms against ischemia found in brain and one of the oxygen conserving reflexes. The initiation of such reflex, leads to activation of sympathetic nerves which leads to cerebro vascular vasodilatation. These responses are exaggerated and can put the patient at risk. The initial period of vagal stimulation, the cardiac depression can be at its peak leading to sinus arrest, asystole or ventricular fibrillation [10].

Discussion

Trigeminal cardiac reflex is mostly neglected phenomenon in maxillofacial surgery and it can have catastrophic bearing on the patient. Surgery in an area innervated by trigeminal nerve has a high risk of provoking TCR. Hypoxia hypercarbia, acidosis and light plane of anaesthesia are known to potentiate TCR and as such should be corrected beforehand to prevent development of this reflex. The injection of local anaesthetic or peripheral nerve block of the afferent nerve may be used as prophylaxis against peripheral TCR[11-12].

The surgeon should inform and alert the anaesthesiologist while manipulating zygoma fractures or midface fractures or approaching the trigeminal nerve or its branch. The risk of TCR can be reduced by gentle and smooth manipulation during surgery in the facial or orbital region. The heart rate and blood pressure monitoring allows early detection of the event and may warrant immediate interruption of stimulus by the surgeon, which leads to cessation of reflex and restores the heart rate and blood pressure to normal without use of drugs [13]. The anticholinergic medication (atropine or glycopyrrolate) should be administered intravenously if bradycardia or hypotension doesn't respond to the cessation of manipulation [14].

The TCR also has a sympathetic component and symptoms may occur due to decreased sympathetic tone rather than increased vagal stimulation, use of epinephrine rather than vagolytic agent will be efficacious in such cases [13, 15]. TCR effects may range from sudden onset of sinus bradycardia to bradycardia terminating asystole or asystole with no preceding bradycardia, arterial hypotension, apnea, and gastric hypermobility. Recognition of bradycardia is the first step in treatment. TCR refractory to all forms of treatment requiring cardiopulmonary resuscitation has been described [15]. The potent narcotics like sufentanil and alfentanil, beta-blockers, and calcium channel blockers may predispose to OCR. In our study the TCR was managed by cessation of procedure temporarily and blocking of stimuli by infiltration of local anesthesia into the area of nerve distribution contributing to reflex. One patuient with asystole was managed with iv atropine and resuscitation.

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 glycopyrolate (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 glycopyrolate 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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