

Trigemino-Cardiac Reflex During Orbital Floor Reconstruction: A Case Report and Review

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Abstract Trigemino-cardiac reflex is occurrence of hypotension and bradycardia upon surgical manipulation of areas supplied by the trigeminal nerve, and has been reported during craniofacial maxillofacial and ocular surgeries. Communication between the anaesthetic and surgical team is essential, and cessation of the precipitating stimulus is the first and most important therapeutic step. We report a case of immediate, reproducible, and reflexive response of Bradycardia and dysrhythmia upon manipulation of orbital fracture during orbital floor reconstruction in a 65-year-old man. Upon recognition of the reproducible relationship between falcine stimulation and increased vagal tone, the patient was given atropine in an effort to block cholinergic hyperactivity. After atropine administration, no further dysrhythmias occurred and surgery was carried uneventfully.

Keywords Occulo-cardiac reflex · Trigemino-cardiac reflex · Cardiac reflex · Vagal reflex · Bradycardia

Introduction

Trigemino-cardiac reflex is the sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, apnea or gastric hyper motility due to the stimulation of any of the sensory branches of the trigeminal nerve [1]. Stimulation of

the trigeminal receptors in its innervated regions provides an important stimulus for the initiation of the trigeminorespiratory reflex and cardiac arrhythmias which could arise with it [2].

It is more common with ophthalmic division of trigeminal nerve and it may also be elicited by stimulating the maxillary and mandibular divisions of the trigeminal nerve during craniomaxillofacial surgeries [3], temporomandibular joint arthroscopies [4], manipulation at the maxillary tuberosity [5], Le Fort I osteotomies [6], nasal fracture reconstructions [7], zygomatic arch fracture elevations [8], midface disimpactions [9] and neurosurgical procedures [10].

Here, we report the occurrence of trigemino-cardiac reflex during orbital floor reconstruction in a 65-year-old man with pan facial fracture under general anesthesia. The present paper reports a case, its intraoperative diagnosis and management update.

Case Report

A 65 year old male patient presented with loss of consciousness and bleeding from the nose following a head injury in a motorcycle accident. Patients' Glasgow Coma Scale (GCS) was 9 and immediate tracheotomy was done to restore the breathing. Pupils were reactive to light. There was bruise and swelling over the maxillary, frontal and temporal areas with subconjunctival ecchymosis and exophthalmos on the right side. Coronal CT scan (Fig. 1) examination revealed multiple fractures involving frontal, temporal, zygomatic and maxillary bones with fracture of orbital floor on the right side with soft tissue herniation into the maxillary antrum. The diagnosis of multiple bone fractures involving right frontal, temporal, zygomatic and Lefort I fracture along with blow out fracture-right orbit was established. Open reduction and internal

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fixation was planned along with reconstruction of orbital floor with silastic under general anesthesia.

Before the induction of general anesthesia, his heart rate was 84 bpm, blood pressure was 140/60 mm Hg, and arterial oxygen saturation (SaO₂) was 98%. 5 mg dexamethasone was given intravenously to reduce postoperative swelling. General anesthesia was induced with the intravenous administration of fentanyl (150 µg), and propofol (150 mg). Anesthesia was maintained with 2% enflurane in a mixture of 40% oxygen and 60% nitrous oxide. Tubocurarine (20 mg) was administered for muscle relaxation, and the patient's lungs were mechanically ventilated. The ventilation was adjusted to maintain an end-tidal carbon dioxide concentration (EtCO₂) of 30–35 torr. The ECG, pulse oximetry, and EtCO₂ were measured continuously, and the blood pressure was measured non invasively at 3-min intervals.

Orbital floor was approached through infraorbital incision. The infraorbital nerve was identified and protected. The herniated periorbital content including inferior rectus muscle and fat was disengaged from the bony defect. At this time, a sudden drop of heart rate, bradycardia with a flat ECG (Electrocardiogram) was detected followed by asystole. The surgeon was informed immediately, and the surgical stimulus was stopped. The inspiratory oxygen concentration was changed to 100%, and the enflurane vaporizer was switched off, atropine (0.6 mg) was given intravenously, after which the heart rate increased to 106 beats/min and the blood pressure increased to 110/70 mm Hg. Nitrous oxide and enflurane were restarted, and the operation (Fig. 2) was completed uneventfully. The pulse

remained stable at 80–96 beats/min. At the end of the procedure, muscle relaxation was reversed with 2.5 mg neostigmine and 0.6 mg atropine. The patient woke up 15 min after surgery had finished and recovered uneventfully.

To the best of our knowledge, there have been four cases reported about this reflex in the literature during the management of orbital blowout fracture by Chesley and Shapiro [11] in 1989, Ziccardi et al. [12] in 1991, Hirjak et al. [13] in 1993 and Sires [14] in 1999. This was the fifth report we encountered during orbital floor fracture reconstruction in our case.

Discussion

First step in the historic development of trigemino-cardiac reflex was considered with descriptions by Joseph Breuer [15] about self regulation of breathing through the vagus nerve in 1868. Kratschmer [16] showed sudden development of hypotension, asystole, cardiac dysrhythmia and gastric hypermotility in animals on manipulation of nasal mucosa. Further studies [17] confirmed that these reflexes were abolished when local anesthetics were applied to the trigeminal nerves branches. Later, the first description about trigemino cardiac reflex was given by Bernard Ascher [18] and Guisepe Dagnini [19] in 1908 as oculo-cardiac reflex because of its cardiac response (mainly bradycardia) associated with stimulation of the ophthalmic division of the trigeminal nerve during ocular surgeries. They described this response as pressure induced neural reflex which stimulate vagus nerve that causes cardiac depression.

However, several other reports [9, 20] showed occurrence of this bradycardia by stimulating non ocular sources.

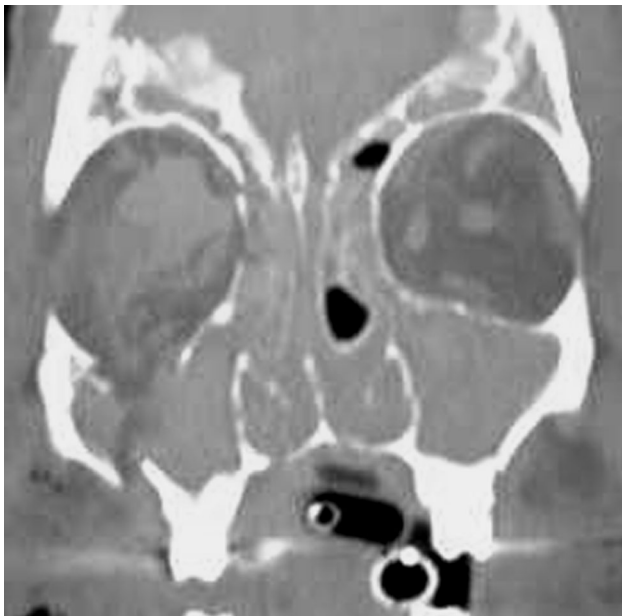


Fig. 1 CT scan-coronal view



Fig. 2 Orbital reconstruction with silastic

There after it is termed as trigemino-cardiac reflexes in 1988 as suggested by Shelly and Church [21].

Literature Review in Maxillofacial Surgery

Trigemino-cardiac reflex during maxillofacial surgical procedures has been described on several occasions [3–9]. The reason is due to stimulation of maxillofacial structures innervated by the trigeminal nerve. The trigeminal nerve provides sensory supply to the face, scalp, and mucosa of the nose and mouth [2, 16, 22]. Trigemino-cardiac reflex during maxillofacial procedures reported first in 1978 by Roubideaux [9] who documented a sudden decrease in heart rate with surgical disimpaction of a fractured maxilla in a 22-year-old healthy male. Overview of reports of TCR in maxillofacial procedures is given in Table 1. In 1987, Loewinger et al. [8] Shearer et al. [20] and Bainton [23] reported the episodes of bradycardia or asystole during elevations of zygomatic fractures. Gillespie [24] reported bradycardia during elevation of zygomatic fractures again in 1988. Reaume and MacNicol [25] also reported same phenomena during Lefort I osteotomy in the same year. Later Shelly and Church [22] reported bradycardia during facial surgery and suggested the term “trigemino-cardiac reflex”. In 1989 Ragno et al. [6] described several episodes of ventricular asystole in a single patient during the downfracturing of a maxilla as part of Le Fort I osteotomy. Stott [3] reported this reflex in facial surgery and described that traction or stretching of any structure innervated by trigeminal nerve might be associated with slowing down of pulse. In the same year the first case of bradycardia during treatment of an orbital blowout fracture was reported by Chesley and Shapiro [11]. In 1990 Precious et al. [26] reported first series of patients a 1.6% incidence (8/502 cases) of either asystole or bradycardia in patients undergoing maxillofacial orthognathic or temporomandibular surgery and Bainton et al. [27] reported sinus arrest complicating a bitemporal approach to the treatment of panfacial fractures. In 1991 Lang et al. [15] reported 3 cases of reflex bradycardia responses that occurred during orthognathic procedures and illustrated the existence of afferent pathways via the maxillary and/or mandibular divisions, in addition to the commonly reported pathway via the ophthalmic division of the trigeminal nerve. In 1996 Morey and Bjoraker [28] reported a case of asystole during temporomandibular joint arthrotomy. Schaller [2] demonstrated that this reflex occurs with stimulation of the intracranial portion of the trigeminal nerve and it occurs in 10–18% of the patients operated for tumors of the cerebello-pontine angle. Several others reported similar reflex during thyroid resection [29], endoscopic forehead lift surgery [30] etc.

The factors [31] known to increase the risk of this reflex include hypercapnia, hypoxemia, light general anesthesia, age (more pronounced in children), nature of the provoking stimulus (strength and duration) and drugs like narcotic agents [32], beta-blockers and calcium channel blockers [33]. Lübbers et al. [34] classified risk factors into low, medium and high risk depending on the surgeries involving trigeminal course and its prevention. These factors were ruled out in our case and other possibilities were considered. Hypovolaemia was ruled out, as the patient had received an infusion of one liter of fluids and intra-operative blood loss was also minimal. Electrolyte imbalance was excluded, as an arterial blood gas analysis prior to the event was within normal limits. The bradycardia that occurred was not preceded by either hypoxia or hypercarbia. Myocardial ischemia was ruled out, as the post-operative ECG was normal.

The most important factor involved in the occurrence of this reflex is the stimulation intensity during manipulation of the trigeminal pathway. In surgeries involving orbit, traction on the extra ocular muscles is particularly prone to cause reflex bradycardia [35] which is directly related to the tension applied to the extraocular muscles. However, the period of time to reach the minimal heart rate decreases as tension applied increases [12, 36]. Even intracranial manipulation of the trigeminal nerve is possible without inducing the reflex when this maneuver is performed gently [37]. Removing the triggering factor can cause cessation of the reflex, raising hemodynamic parameters to normal levels.

In our case, pressure applied over orbital floor during floor elevation, before placing silastic [38, 39] graft material must have triggered the trigemino-cardiac arc involving the neuronal transmission from the orbital structures to the ciliary ganglion, the ophthalmic division of the trigeminal nerve. When excessive pressure impulses are transmitted, the efferent pathway of the vagus nerve running alongside is stimulated, and the tonus in the parasympathetic nerve increases, resulting in Bradycardia. Previous studies [40, 41] reported development of trigemino cardiac reflex due to compression and contusion during ocular surgeries. Other possibility in our case could be the non pre-operative administration of a local anesthetic agent which might have prevented the reflex due to the blockade of the afferent pathways because similar reflex arises [23] without prerequisite manipulation of areas supplied by ophthalmic branch but during maxillary osteotomies [5–9]. Many authors [42–44] also reported intraoperative deaths possibly due to this reflex which demonstrate the (clinical) importance of the TCR which causes severe bradycardia that leads to death if not detected early. In addition, hypotension which occurs during reflex

Table 1 Review of trigemino cardiac reflex in maxillofacial cases

| Author | Year | Maxillofacial procedure |
|---------------------------------|------|-------------------------------------|
| Robideaux | 1978 | Midface disimpaction |
| Bainton R, Lizi E | 1987 | Management of zygomatic fracture |
| Loewinger J, Cohen M, Levi E | 1987 | Management of zygomatic fracture |
| Shearer et al. | 1987 | Management of zygomatic fracture |
| Reaume and Mac Nicol | 1988 | Lefort I osteotomy |
| Shelly and Church | 1988 | Facial surgery |
| Gillespie | 1988 | Zygomatic fracture |
| Ragano JR, Marcoot RM, Taylor | 1989 | Lefort I osteotomy |
| Baxandall et al. | 1989 | Nasoethmoidal fractures |
| Stott D.G | 1989 | Facial surgery |
| Chesley and Shapiro | 1989 | Orbital blowout fracture |
| Bainton, Barnard, Wiles | 1990 | Panfacial fracture |
| Precious, Skulsky | 1990 | Facial surgery |
| Gomez | 1991 | TMJ arthroscopy |
| S. Lang, Lanigan, M. vander Wal | 1991 | Orthognathic procedures |
| Ziccardi et al. | 1991 | Orbital fracture |
| Hirjak, Zajko, Satko | 1993 | Orbital fracture |
| Cambell | 1994 | Orthognathic procedure |
| Seo.K, Takayama, Araya | 1995 | Opening mouth under GA |
| Morey, Bjoraker | 1996 | TMJ arthrotomy |
| Roberts RS, Best JS, Shapiro | 1999 | TMJ arthroscopy |
| Locke MM et al. | 1999 | Nasal fracture |
| Sires BS | 1999 | Orbital trapdoor fracture |
| Kosaka, Asamura | 2000 | Management of zygomatic fracture |
| Webb, Unkel | 2007 | Mesiodense removal |
| Osborn et al. | 2008 | Periorbital laceration manipulation |
| Arekere et al. | 2010 | Extractions |

may lead to myocardial and cerebral infarction in those who are at risk for these conditions.

Management of patients with TCR can be directed in identifying risk factors especially areas supplied by trigeminal nerve. If any mechanical stimulation to the nerve is necessary, it should be as gentle as possible. If working in the vicinity of the nerve or its branches, the anesthesiologist should be notified by the surgeon. Prophylaxis of this reflex is better accomplished with local anesthetic infiltration or block of the nerve. Several [45, 46] studies showed that peribulbar blocks with local anesthetic agents significantly reduced the incidence of trigemino cardiac reflex. As soon as sudden bradycardia or asystole is detected, the surgery should be stopped immediately and any traction on soft tissues released. Usually cessation of the stimulus will enable the patient to regain a sinus rhythm. In severe cases of bradycardia and asystole, administration of vagolytic agents [47] such as atropine which blocks the peripheral muscarinic receptors at the heart or glycopyrrolate is considered. Anticholinergic drugs use is logical as the reflex is precipitated by parasympathetic stimulation, anticholinergic drugs [17, 47] may prevent further episodes of the reflex. When used, time should be allowed for

the drug to take effect. At least one recurrence was ascribed to the fact that the drug had not had time to act before a fresh stimulus was given. Treating the initial episode with anticholinergics does not guarantee protection from further episodes and the use of atropine is also questioned [48] because cholinergic blockage reduces but not totally prevent either bradycardia or hypotension and may cause serious cardiac arrhythmias itself.

In such cases that are refractory to atropine and other vagolytics may rather need to be managed with epinephrine [49]. Cardiac massage [11] should be reserved for cases where conservative measures have failed to reestablish cardiac output, as there is serious morbidity associated with it.

Conclusion

In summary, we described the occurrence of trigemino-cardiac reflex during the reconstruction of orbital floor in a panfacial fracture case and its management. Communication between the anaesthetic and the surgical team is essential, and

cessation of the precipitating stimulus is the first and most important step. Prevention of trigeminovagal reflexes requires awareness of the anatomical structures involved, cooperation between the surgeon and the anaesthetist, and knowledge of factors known to evoke these reflexes.

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