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Review Article

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An Insight into the Trigeminocardiac Reflex- Understanding the Phenomenon and its Management

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Abstract

Trigeminocardiac reflex is a brainstem neurogenic reflex incited by stimulation of trigeminal nerve anywhere along its course from central nucleus to its peripheral distribution. Surgeries involving the craniomaxillofacial region may stimulate this reflex and complicates the procedure by manifesting as bradycardia, asystole, hypotension, apnoea and gastric hypermotolity. Thorough understanding of the pathophysiology, manifestations and ways of prevention and management of the trigeminocardiac reflex are of utmost importance for both the maxillofacial surgeon and the anaesthetist.

Keywords: Trigeminocardiac Reflex, Oculocardiac Reflex, Bradycardia, Hypotension, Maxillofacial Surgery.

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Introduction

Trigeminal nerve is the largest cranial nerve providing sensory innervation to the region of scalp, face, oral and nasal mucosa. During the surgical intervention in the region supplied by the branches of trigeminal nerve as well as along its intra and extra cranial course, any stimulation of the nerve may sets off a cardiac depressor reflex via vagal stimulation. Trigeminocardiac reflex is a brainstem reflex which presents as parasympathetic and sympathetic imbalance.[1] Any sudden onset of parasympathetic cardiac dysrhythmia, sympathetic hypotension, apnea or gastric hypermotility during stimulation of sensory branches of trigeminal nerve is considered as trigeminocardiac reflex which is a powerful autonomic neurogenic reflex.[2] Any trigeminocardiac reflex episode classically shows decrease in heart rate and blood pressure of more than 20 % from the baseline values, where deceleration in heart rate is a constant observation while reduction in blood pressure is not fixed to a certain percentage of decrease.[3] Florian kratschmer first described the influences of reflexes of nasal mucosa on breathing and circulatory systems based on the studies on cats and rabbits in 1870.[4] Aschner and Dagnini in 1908 independently described the phenomenon as oculocardiac reflex which shows pressure induced cardiac depression through vagal stimulation as a physiological response to the physical stimulation of the eye and the adnexa.[5] Kumada et al in 1977 described it as trigeminal depressor response through their neurostimulation experiments on rabbits and also observed that not only peripheral but also the central stimulation of the trigeminal nerve can produce similar response.[6] Many cases of bradycardia or asystole were reported during cranio maxillofacial surgeries during the period between year 1978 to 1988.[7] Loewinger et al and Lang et al suggested that trigeminocardiac reflexes can also be observed on the stimulation of sensory branches of maxillary and mandibular divisions of trigeminal nerve other than the ophthalmic division.[8,9] Shelly and Church in 1988 proposed the term trigeminocardiac reflex and considered oculocardiac reflex as one of its manifestation when it involves only the ophthalmic division of the trigeminal nerve.[10] Schaller et al described and established the occurrence of central trigeminocardiac reflex manifestation in humans during cerebellopontine angle and brainstem surgeries.[11] Schaller et al classified trigeminocardiac reflex (TCR) into three subtypes based on the region of trigeminal pathway stimulated. These subtypes include central, ganglionic and peripheral TCR. Perpheral TCR can further be subdivided into oculocardiac reflex and maxillomandibulocardiac reflex. Any surgical intervention in the distribution of trigeminal nerve poses a risk of precipitating the reflex, so better understanding of the phenomenon is of paramount importance to both the anaesthesiologist and the surgeon.[12]

Discussion

Insight in to the maxillofacial literature

Roubideaux in 1978 reported a case of sudden bradycardia during surgical disimpaction of a fractured maxilla.[13] Loewinger et al and Shearer et al reported episodes of bradycardia during elevation of zygomatic arch independently in the year 1987.[8,14] Reume and MacNicol in 1988 noticed trigeminocardiac reflex phenonmenon during maxillofacial osteotomies.[15] Ragno et al were the first to report a case of asystole after inferior displacement of the Le Fort I osteotomy segment.[16] Matarasso in 1989 introduced this reflex as trigeminal-vagal mediated reflex arc and reported a case of oculocardiac reflex in blepharoplasty surgery. According to his observation the incidence of oculocardiac reflex in blepharoplasty was 25%.[17] Stott in 1989 presented case reports and suggested that stretching of any structure innervated by branches of trigeminal nerve can cause reflex bradycardia during craniofacial surgeries in addition to traction on ophthalmic division of trigeminal nerve, making the term oculocardiac reflex inappropriate in describing the phenomenon.[18]

Precious and Skulsky performed a retrospective review of 502 patients who underwent maxillofacial surgery and reported 8 cases of dysrhythmias (1.6% incidence). Six cases occurred during Le Fort I osteotomies (bradycardia when a maxillary segment was mobilized anteriorly) and 2 during temporomandibular joint reconstruction (asystole).[19] Bainton et al in 1990 presented two case reports of management of pan facial fractures via bi-temporal approach complicated by sinus arrest.[20] Barnard and Bainton further discussed the association of bradycardia and trigeminal nerve in the same year.[21] Lang et al reported three cases of reflex bradycardia during maxillofacial surgeries in response to manipulation of maxilla or mandible or traction of attached soft tissue suggesting alternative pathway via maxillary and mandibular divisions other than ophthalmic division of trigeminal nerve.[9] Campbell et al reported asystole during a Le Fort I osteotomy on cuttingthe maxillary tuberosity.[22]

Delap et al in 1995 presented a case of asystole during thyroid resection.[23] Morey and Bjoraker reported a case of asystole on irrigation of the temporomandibular joint during TM joint arthrotomy in the year 1996.[24] Roberts et al reported a case of reflex sinus bradycardia while doing instrumentation in superior TMJ space during temporomandibular joint arthroscopy in the year 1999.[25] Kosaka et al in 2000 presented a case of severe arrhythmia with A-V block in a patient with zygomatic fracture. Patient underwent cardiac pacing for arrhythmia. Once the zygomatic fracture was repaired, arrhythmia disappeared and cardiac pacemaker was removed on the first post operative day.[26] Cha et al in 2002 reported a case of recurrent asystole during bilateral trigeminal sensory root rhizotomy and pointed towards the central role of trigeminal nerve as afferent pathway of trigeminocardiac reflex.[27]

Yilmaz et al in the year 2006 reported a case of delayed TCR due to the presence of intra orbital foreign body. Bradycardia was corrected with the removal of the foreign body.[28] Schaller and Buchfelder in the same year reported a case of delayed trigeminocardiac reflex induced by an intra orbital foreign body.[29]

Webb and Unkel in 2007 reported a case of TCR following reflection of palatal flap for removing mesiodens in a five year child.[30] Bohliuli et al in 2009 presented a review on trigeminocardiac reflex in maxillofacial surgeries.[31] Arakeri and Arali presented a hypothesis in 2010 that TCR can also occur under local anaesthesia during extraction of maxillary molars and mediate syncope.[32]

Lubbers et al in 2010 presented a classification of potential risk factors for trigeminocardiac reflex in craniofacial surgery and also suggested that bradycardia during maxillofacial surgical procedures might happen much more frequently than what is reported in the literature.[33] Bohluli et al in 2010 conducted a cross over study in 25 patients undergoing Lefort I osteotomy and suggested that TCR is more prevalent than previously thought in lefort I osteotomy surgeries.[34] Arakeri and Brennan in 2010 suggested exodontia particularly extraction of upper molars as potential risk for TCR under local anaesthesia in addition to possibility of vasovagal syncope.[35]

Krishnan in 2011 suggested the use of regional nerve blocks to prevent the risk of TCR during maxillofacial surgery.[36] Bohluli et al in the same year conducted a randomized controlled trial on twenty patients undergoing bilateral sagittal split osteotomy procedures. Gow-gates nerve block was given on one side and was compared for TCR with control side. Prevalence of TCR was more on control side as compared to the side which received Gow-gates nerve block. It was concluded that the use of mandibular nerve block during surgery may minimize incidence of TCR.[37] Puri et al in 2011reported a case of TCR during injection of dimethyl sulfoxide prior to planned emboloization with onyx in a child with juvenile nasopharyngeal angiofibroma.[38] Potti et al in the same year described two cases of TCR following percutaneous injection of ethylene vinyl alcohol copolymer (onyx) into a juvenile nasopharyngeal angiofibroma.[39]

Yorgancilar et al in 2012 reported an incidence of 8.3% following lateral osteotomies and nasal pyramid infracture during rhinoplasty surgeries. It was concluded that manipulation of infra orbital nerve during rhinoplasty might precipitate TCR, even if local anaesthetic infiltration was used.[40] Wartak et al in 2012 reported a case of post traumatic bradycardia and recurrent asystole in the absence of surgical intervention in a patient with facial injury.[41]

Chowdhary and West in 2013 reported a case of intra operative asystole in a patient who underwent craniotomy when slight skin traction was applied during scalp closure.[42] Kamath et al in 2014 reported a case of oculocardiac reflex in a pediatric patient with orbital floor fracture.[43] Neils et al in the same year reported an incidence of 31.7% of oculocardiac reflex in a prospective study conducted on 104 patients undergoing surgery through orbitozygomatic approach.[44]

Shanab and Albargi in 2016 conducted a retrospective study in ASA class I patients who underwent maxillofacial surgery and found an overall TCR incidence of 20%. It was also concluded that higher incidence of TCR phenomenon was observed in patients with mid face fractures as compared to patients who had upper face fractures.[45] Kiani et al in the same year evaluated TCR and haemodynamic changes while performing lefort I osteotomy during orthognathic surgery. They concluded that gentle manipulation and transient cessation of the procedure is sufficient to stop TCR.[46]

Joshi et al conducted a study for TCR occurrence on 37 patients undergoing maxillofacial surgeries and found male preponderance and higher incidence in mid facial fractures.[47] Huang et al in 2017 reported a significant blood pressure drop during vital pulp extirpation under local anesthesia possibly related to occulocardiac reflex.[48] Dillon et al in the same year reported a case of TCR while resection of large osteoma over zygomatic arch.[49]

Gadicherla et al in 2018 conducted a retrospective study on patients who underwent management of zygomaticomaxillary complex fractures. Incidence of oculocardiac reflex was found to be 10.05% and it was also observed that co morbidities like hypertension, hyperlipidemia and use of beta – blockers increase the chances of occurrence of oculocardiac reflex.[50]

El-Habbash et al in 2018 reported a case of TCR while managing a case of temporomandibular joint synovial cyst.[51] Baronos et al in 2019 reported an episode of sudden bradycardia and asystole on placement of bite block while perfoming an orthgnathic surgery. Either the inferior alveolar nerve or the auricular branch of mandibular division might be stretched due to bite block placement.[52] Hammad et al conducted a retrospective cohort study on 94 patients who underwent nerve repair of maxillary and mandibular divisions of trigeminal nerve and found no incidence of TCR.[53] Sugiyama et al reported a case of trigeminocardiac reflex in a 31 year old female while performing bilateral saggital split osteotomy.[54] Hoshijima et al reported a case of asystole triggered by the mouth opening with a dental mouth gag under general anaesthesia during extraction of maxillary supernumerary tooth.[55] Recently Madanat et al reported a case of asystole during nasopharyngeal swab in a patient with Covid 19 infection.[56]

Incidence

Based on the nature and location of the surgical stimulus, incidence of trigeminocardiac reflex may vary from 5% to 90%.[52] Mataraso et al observed the incidence of oculocardiac reflex to be 25% in the patients who underwent blepharoplasty.[17] Patients who underwent strabismus surgery showed incidence ranging from 32% to 90%.[33] The incidence of oculocardiac reflex in the patients who underwent reduction of zygomaticomaxillary complex fracture was 10.05%.[50] The incidence of trigeminocardiac reflex in all craniofacial surgeries is 20% with the highest occurring in facial trauma (32%) and the lowest in lower face reconstruction (3.6%). Orthognathic surgery has a trigeminocardiac reflex incidence of 9.1%.[1,2,52] For skull base surgery an incidence of 8% to 18%, have been reported.[57] Bradycardia during maxillofacial surgical procedures might happen much more frequently than is published. Clinical features like nausea, vomiting, or bradycardia, particularly in children, can also be interpreted as commotio cerebri rather than being attributable to TCR.[33]

Classification

Schaller et al classified trigeminocardiac reflex into central, ganglionic and peripheral subtypes based on region of stimulation of the trigeminal nerve.[12]

TCR resulting from stimulation of the peripheral branches of the trigeminal nerve viz. ophthalmic, maxillary and mandibular divisions, is known as peripheral TCR. Oculocardiac reflex and maxilla-mandibular reflex are the parts of peripheral TCR. Diving reflex is also considered as modified TCR and a part of peripheral TCR. This reflex is provoked while submerging face into water or washing face with cold water, due to stimulation of peripheral branches of trigeminal nerve. This is an autonomic reflex inherited from diving birds and amphibians into humans. Diving reflex is a physiological reflex which help in conserving body oxygen in hypoxic or anoxic environmental conditions. Parasympathetically induced bradycardia reduce oxygen demand of the heart while sympathetically induced peripheral vasoconstriction redistribute blood supply from peripheral organs to vital organs like heart and brain, thereby reducing oxygen consumption during hypoxic state.[1,58]

During intracranial surgeries any stimulation of the trigeminal nerve along its course from gasserian ganglion to brainstem nuclei may result in central TCR.[11] Direct stimulation of the gasserian ganglion may result in ganglionic TCR. It is considered as separate sub type because its clinical manifestations are different from the other two subtypes.[1]

Another form known as chronic trigemino cardiac reflex has also been reported by some authors. Chowdhary et al defined chronic TCR as episodes of trigeminocardiac reflex persistent beyond 24 hours of primary insult or intervention. Vague and non –classical symptoms like nausea and dizziness may be seen in chronic TCR due to continuous or repetitive

stimulation.[59] Schaller et al,[29] in 2006 and Yang et al,[60] in 2011 reported cases of delayed trigeminocardiac reflex induced by an intra orbital foreign body. Chowdhary et al reported a case of chronic TCR in a patient with orbital floor fracture in 2014.[61] Involvement of ophthalmic branch of trigeminal nerve is more commonly reported in the literature.[1]

Pathway of the reflex

Stimulation of the trigeminal nerve anywhere along its course from brain to peripheral structures of head, neck and face by any stimuli (mechanical, chemical or electrical) sends neuronal signals to the sensory nucleus of the trigeminal nerve. This forms the afferent pathway of the reflex. The sensory nucleus of the trigeminal nerve is further linked to reticular formation through polysynaptic connections. The short internuncial fibres in the reticular formation connect the afferent pathway with the efferent pathway. Fibres of the neurons located primarily in the dorsal vagal nucleus and nucleus ambiguous innervate the heart through vagus nerve. This forms the efferent pathway of the reflex. Parabrachial nucleus, rostral ventrolateral medulla oblongata, dorsal medullary reticular field and paratrigeminal nucleus are the other areas of the brainstem involved in the TCR. There are variations in the afferent pathway of the reflex in the various clinical subtypes of the reflex resulting in variations in clinical manifestations.[1] Dutschmann and Herbert in their studies on rats suggested that in peripheral TCR impulses are relayed through the spinal nucleus of trigeminal nerve to Kolliker- fuse nucleus.[62] Schaller suggested that in central TCR, impulses are relayed through the nucleus of solitary tract to lateral parabrachial nucleus.[63] Activation of both sympathetic and parasympathetic autonomic responses occur in peripheral TCR while in the central TCR parasympathetic cardio inhibitory responses predominate.[57]

An episode of trigeminocardiac reflex usually manifests as sudden bradycardia or asystole, hypotension, apnoea and gastric hypermotility due to co-activation of sympathetic and parasympathetic nervous system. Hypertension instead of hypotension is seen in diving reflex due to strong sympathetic activity. In central TCR, bradycardia and hypotension are seen while in ganglionic TCR varied presentation ranging from bradycardia to tachycardia and hypotension to hypertension may be seen due to presence of mixed fibres of the three divisions of trigeminal nerve as well as sympathetic fibres from carotid plexus in the gasserian ganglion.[1]

Promptness with which the motor nucleus of the vagus tends to become refractory to a sustained stimulus is known as vagal escape while fatigue of oculocardiac reflex depends on the time period during which central nucleus of vagus remains relatively refractory to repetitive stimulation. Both vagal escape and fatigue are physiological mechanisms that defend against excessive vagal stimulation. During general anaesthesia these mechanisms become obtunded.[5]

Common procedures that can triggr trigeminocardiac reflex

Neurosurgical procedures (especially cerebellopontine angle surgeries), microvascular decompression or rhizolysis of the trigeminal nerve, transphenoidal pituitary resection, cavernous sinus procedures, ophthalmic surgeries, dental procedures like extraction of upper molars or dental implant placement, reduction of zygomaticomaxillary complex fractures or mandibular fractures etc may predispose to an episode of trigeminocardiac reflex.[1,2]

Risk factors for trigeminocardiac reflex

Patients of young age group are more susceptible to TCR particularly, it is more pronounced in children due to higher resting vagal tone. TCR is more commonly seen in male patients, incidence shown to be as high as 77%, this makes the male sex a risk factor. Hypoxemia, hypercapnia and higher resting parasympathetic tone may also predispose to TCR. Light plane of anaesthesia may increase the incidence of TCR. Mechanical stretch is the most powerful stimulus inciting TCR. Stronger and longer lasting provoking stimuli considerably increase the possibility of occurrence of TCR. Bilateral stimulation of trigeminal nerve produce more pronounced response than unilateral stimulation.[1,2,12,45]

Various drugs like opioids, beta –adrenergic blockers and calcium channel blockers inhibit sympathetic nervous system and cause peripheral vasodilation resulting in bradycardia and hypotension. This predispose patient to an episode of TCR.[64] Narcotics like sufentanil and alfentanil can also augment vagal tone by inhibiting sympathetic nervous system.[65,66]

Nothen et al in 2010 suggested that previous transient ishaemic attack within past 6 weeks duration may predispose to an event of intra operative TCR.[67]

Lubbers et al have classified the cranio-maxillofacial surgical procedures into low, medium and high risk for possibility of TCR event intensity. Insufflation of temporomandibular joint, Le fort osteotomy and elevation of zygomatic fractures were considered as low risk. Skull base surgeries were considered as posing medium risk while ophthalmic surgery, strabismus surgery, orbital exenteration and fractures in children with cardiac surgery were considered as procedures with high risk of causing episode of TCR. In low and medium risk procedures, TCR event could be prevented by informing the anaesthetist directly before the time of highest risk while in high risk procedures use of atropine or glycopyrrolate and the use of ketamine for anaesthesia induction can be considered.[33]

Prevention and Management

Management of TCR in patients involves three basic steps. First of all identification of the risk factors and its modifications, next step is the use of prophylactic measures and if the TCR episode still persist, then the administration of vagolytic agents or sympathomimetics. Operating surgeon and the anaesthesiologist should be aware of the possibility of an episode of TCR. Surgeon should use a delicate operative technique by minimizing the mechanical stimulation while exploring in the region supplied by trigeminal nerve and the anaesthesiologist should maintain a constant vision on the patient's blood pressure and heart rate. Controlled ventilation is preferred to spontaneous ventilation immediately before and during muscular traction.

In case of an episode of TCR, surgeon should immediately do the cessation of the manipulation in the region supplied by trigeminal nerve. This simple step will interrupt the episode of TCR and patient's blood pressure and heart rate comes to normal in most cases. Lighter plane of general anaesthesia should be avoided. Use of sevoflurane instead of halothane results in less incidence of TCR. In case, a controlled arterial hypotension is planned, prophylaxis of TCR can be achieved with either the local infiltration of the region with local anaesthetic or the use of nerve block. This will block the afferent limb of the reflex. Prophylactic use of atropine or glycopyrrolate intravenously may also partially prevent the episode of TCR.[1,5,12,64] Mirakhur et al concluded that intramuscular administration of glycopyrrolate is also efficacious in preventing TCR.[68]

The dose of atropine should be carefully selected as atropine itself can result in cardiac arrhythmias, premature ventricular contractions and bigeminy especially when halothane is used as primary anaesthetic agent.[5,69]

During the surgery, if on cessation of the stimulus by the surgeon, bradycardia and hypotension does not get corrected itself and TCR episode continues, use of intravenous anticholinergics like atropine or glycopyrrolate is recommended. TCR has both parasympathetically mediated increased vagal stimulation resulting in cardioinhibitory effects counteracted by vagolytics like atropine as well as sympathetically mediated peripheral vasodilation which can be corrected by using epinephrine.[1,64]

Epinephrine increase the peripheral resistance via alpha - 1 adrenoreceptor vasoconstriction and the blood is shunted to the vital organs of the body. Use of epinephrine is recommended when TCR is due to decreased sympathetic tone and is refractory to atropine. In situations where TCR do not responds to any of the above mentioned techniques, use of cardiopulmonary resuscitation is recommended. Good coordination between the operating surgeon and anaesthetist is needed and both must understand the importance of different factors causing trigeminocardiac reflex.[1,70]

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