Case Report

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Anaesthesia Section

Intraoperative Bradycardia in a Child with Orbital Glioma: A Case of Trigeminocardiac Reflex

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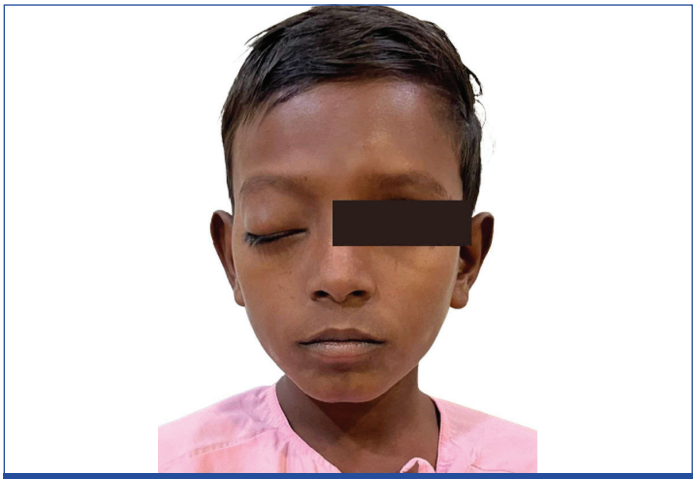
ABSTRACT

Trigeminocardiac Reflex (TCR) is a life-threatening but rare neurogenic reflex that can manifest during neurosurgical procedures. It presents as sudden bradycardia, hypotension, or even asystole triggered by stimulation of the trigeminal nerve. It is more frequent in children due to heightened vagal tone. Authors hereby, present the case of an 11-year-old male with a right-sided optic nerve glioma with intracranial extension who was electively operated on for tumour removal. The patient had a history of loss of vision in the right eye, ptosis, and seizures treated with levetiracetam. During intraoperative dissection near the superior orbital fissure and cavernous sinus, he suddenly developed bradycardia and hypotension, characteristic of TCR. The anaesthetic team promptly identified the reflex and implemented prompt management by alerting the surgical team to cease dissection, administering intravenous atropine, increasing FiO_2, and deepening the level of anaesthesia. The patient's vital signs returned to baseline within minutes, and the surgery proceeded uneventfully. He was successfully weaned on postoperative day 2 without neurological sequelae. The case underscores the necessity of anticipation and vigilance for TCR in paediatric neurosurgical procedures involving trigeminal nerve regions. Early recognition and timely, coordinated intervention by surgical and anaesthetic teams are crucial to ensure haemodynamic stability and optimal patient outcomes. Preventive anaesthetic management, anticipating reflex phenomena, is of utmost importance in the perioperative management of such high-risk procedures.

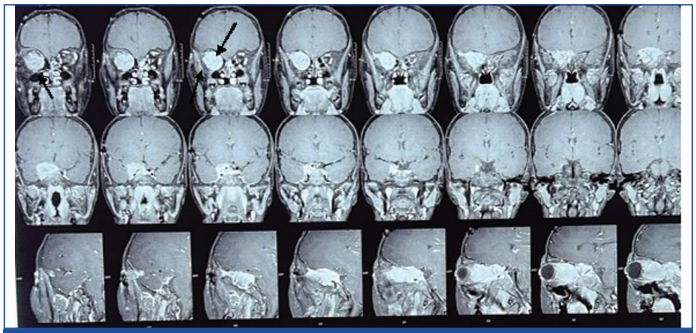
Keywords: Anaesthesia, Craniotomy, Hypotension, Paediatric, Trigeminal nerve

CASE REPORT

An 11-year-old male, American Society of Anaesthesiologists grade III (ASA III), weighing 24 kg and 135 cm in height, was posted for elective craniotomy and excision of a right-sided optic nerve glioma [Table/Fig-1]. He underwent surgery two years ago for the same optic nerve glioma, with gradual vision loss in the right eye and ptosis. One episode of generalised tonic-clonic seizures at diagnosis was present, which was well-controlled on oral levetiracetam (250 mg). The child was neurologically stable with no recent history of seizures or signs of increased intracranial pressure. Pre-anaesthetic evaluation was uneventful for airway anatomy and systemic disease. All vitals were stable. Tests such as haemoglobin (12.1 g/dL), total leukocyte count (9,200/mm^3), platelet count (3.8 lac/mm^3), coagulation profile, renal and liver function tests, and electrolytes were all within normal limits. Magnetic Resonance Imaging (MRI) of the brain and orbits revealed a right orbital glioma with intracranial extension, closely abutting the superior orbital fissure and cavernous sinus [Table/Fig-2].



[Table/Fig-1]: Patient showing signs of ptosis.

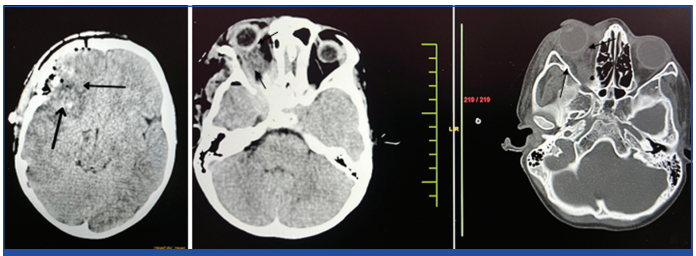


[Table/Fig-2]: MRI of the brain and orbits revealed a right orbital glioma with intrac ranial extension, closely abutting the superior orbital fissure and cavernous sinus(black arrow).

Preanaesthetic evaluation: After confirmation of fasting status and informed consent, the child was transferred to the operating room. ASA standard monitors were placed. Intravenous lignocaine (25 mg), fentanyl (50 mcg), thiopentone (120 mg), and rocuronium (24 mg) were administered to induce anaesthesia. Tracheal intubation was performed with a 6 mm cuffed endotracheal tube. A 5.5 Fr right internal jugular central venous catheter and a 22G left radial arterial line were inserted under ultrasound guidance. Intravenous mannitol (12 g) and dexamethasone (3 mg) were administered for prophylaxis against cerebral oedema. Maintenance of anaesthesia was achieved with sevoflurane to maintain a Minimum Alveolar Concentration (MAC) in oxygen/air, and rocuronium boluses were given as needed.

Approximately 90 minutes into the procedure, during microscopic dissection near the superior orbital fissure and cavernous sinus, the patient suddenly exhibited abrupt bradycardia (heart rate decreased from 98 to 42 beats per minute) and a concomitant drop in Mean Arterial Pressure (MAP) decreased by more than 30%). The patient maintained adequate oxygenation and stable end-tidal CO_2 levels, indicating that the cause was unlikely to

be related to ventilation or insufficient depth of anaesthesia, as an appropriate MAC of sevoflurane had been administered. Structures innervated by the ophthalmic (V1) division of the trigeminal nerve were part of the surgical field at that time, and there was a suspicion of a TCR. The anaesthesiology team immediately informed the surgical team, who stopped retraction and dissection. The anaesthetist deepened the anaesthesia and atropine 10 mcg/kg i.v. was administered. Warm saline was irrigated across the surgical field. Within minutes, the heart rate and blood pressure stabilised. Continuous arterial waveform and Electrocardiogram (ECG) monitoring were used to monitor for recurrence, and boluses of propofol were given prophylactically during subsequent high-risk manipulations. The rest of the surgery was uneventful, with careful dissection and excellent communication between team members. There were no further spells of reflexes. The child was electively ventilated overnight in the Intensive Care Unit (ICU) since the tumour was close to significant neurovascular structures. He was extubated safely on postoperative day 2 following full recovery. Postoperative imaging Computed Tomography (CT) scan confirmed sufficient debulking of the tumour without haemorrhage or infarct [Table/Fig-3].



[Table/Fig-3]: CT scan confirmed sufficient debulking of the tumour without haemorrhage or infarct.

DISCUSSION

The TCR is an established but potentially hazardous intraoperative event resulting from stimulation of the trigeminal nerve or its branches. It is characterised by an acute onset of bradycardia, hypotension, arrhythmias, or even asystole mediated by a brainstem reflex arc involving afferent input from the trigeminal nerve and efferent parasympathetic output via the vagus nerve. The reflex can be provoked by manipulation of the dura, orbit, skull base, or other areas innervated by the ophthalmic (V1), maxillary (V2), or mandibular (V3) divisions of the trigeminal nerve. Central presentation of TCR, wherein intracranial manipulation of the ganglion or nerve root is involved, such as in our patient, is accompanied by more significant haemodynamic alterations [1,2].

Trigeminocardiac Reflex is particularly pertinent in paediatric neurosurgery, where augmented resting vagal tone renders patients vulnerable to exaggerated reflex responses. The incidence of TCR in neurosurgery ranges from 8% to 18%, depending on the location of the lesion and the extent of surgical manipulation near trigeminal nerve pathways. Surgeries involving the cavernous sinus, superior orbital fissure, and skull base—such as optic glioma removals—are considered high-risk for triggering this reflex. In present case, dissection around the superior orbital fissure and cavernous sinus most likely caused direct stimulation of the ophthalmic division (V1) of the trigeminal nerve, leading to a sudden drop in heart rate and MAP. Prompt recognition and control of TCR are essential to prevent adverse outcomes. The reflex is generally self-limited if the offending stimulus is removed, but pharmacologic treatment may be required for significant bradycardia or persistent haemodynamic instability [1,3].

On this occasion, prompt recognition by the anaesthesia team, immediate notification of the surgical team to cease dissection, and intravenous administration of atropine were crucial in stabilising the patient. Concurrently, FiO_2 was increased to 100%, and anaesthetic depth was reassessed and deepened. Taken together, these maneuvers restored haemodynamic stability and allowed further surgery to proceed safely.

Several factors are recognised to aggravate TCR, such as light anaesthetic planes, hypercapnia, hypoxia, and the administration of potent opioids or beta-blockers. Prevention and diminution of the reflex depend centrally on avoiding the above conditions. In hazardous operations, some authors have recommended prophylaxis in the form of regional nerve block, anticholinergic administration, and maintaining adequate depth of anaesthesia [3,4]. More comprehensive prophylactic strategies may include preoperative administration of glycopyrrolate or atropine in high-risk surgeries, continuous invasive haemodynamic monitoring to detect early changes, and total intravenous anaesthesia to better modulate anaesthetic depth and autonomic tone [2,4]. Short-acting beta-agonists may also be considered if reflex bradycardia is profound and recurrent. Avoidance of hypoventilation, maintenance of normocapnia, and supplemental oxygenation are essential perioperatively. If unrecognised or untreated, severe TCR may lead to sustained bradycardia, hypotension-induced cerebral hypoperfusion, ventricular fibrillation, and cardiac arrest, particularly in paediatric patients or those with limited cardiac reserve. Thus, vigilance and readiness to intervene are critical in avoiding these life-threatening outcomes. Prophylactic anticholinergics should be administered, and clinicians should be aware of their mechanisms for reflex inhibition, which can be determinant in paediatric neurosurgical procedures [3,4]. The management of TCR in other cases is tabulated in [Table/Fig-4] [5-9].

Authors and Year of study	Cases	Management	Takeaways
Arasho B et al., 2009 [5]	60-year-old male undergoing retrosigmoid approach for vestibular schwannoma resection. Sudden hypotension (MABP 43.3 mmHg) and bradycardia (HR 40 bpm) occurred 2 hours into surgery.	Surgical pause, i.v. epinephrine, and fluids administered. Haemodynamic parameters normalised in 5 minutes, and surgery proceeded uneventfully.	Comprehensive review and clinical experience highlight that awareness, intraoperative communication, and risk factor modification (e.g., hypoxia, hypercapnia, depth of anaesthesia) are crucial. Management includes cessation of stimulus, vagolytics, and in some cases, epinephrine. Atropine may not always be effective. Prophylactic nerve blocks may help in peripheral TCR.
Amirjamshidi A et al., 2013 [6]	Case 1: 71-year-old male with pituitary adenoma developed bradycardia and hypotension during sella packing with autologous fat after trans-sphenoidal tumour resection. Case 2: 52-year-old female with clinoidal meningioma had severe bradycardia and hypotension when negative pressure was applied to a subgaleal drain post-craniotomy.	In both cases, immediate cessation of the triggering stimulus (fat packing and negative pressure suction) led to the return of haemodynamic stability. No additional drugs were administered. Suction was not reapplied in the second case; patient tolerated recovery well.	Demonstrates atypical triggers for TCR, including sella packing and subgaleal suction. Stresses that TCR can occur even without manipulation of the classic trigeminal regions. Highlights the importance of continuous vital sign monitoring even during closure and postoperative stages. Raises awareness about possible triggering mechanisms and need for gentle handling near trigeminal pathways.
Lakshminarasimhaiah G et al., 2018 [7]	Uncooperative adult with a large orbital tumour extending to upper lip and intracranially. Induction achieved with paediatric face mask; video laryngoscopy-assisted intubation. Repeated surgical manipulation of maxillary and mandibular nerves triggered severe TCR.	Vagal response managed with prompt vagolytic therapy during episodes of haemodynamic instability.	Highlights airway challenges in facial tumours with intracranial extension. Reinforces that TCR can occur in facial surgeries with deep nerve involvement. Emphasises the importance of preparedness for both airway and reflex management in neurofacial procedures.

Shakil H et al., 2019 [8]	60-year-old woman undergoing elective left orbitozygomatic craniotomy for recurrent skull base epidermoid cyst. Retraction of the temporalis muscle alone led to repeated bradycardia and asystole.	Surgical manipulation was stopped when asystole occurred; heart rate returned to normal. Normocapnia and normoxia were maintained. Anaesthesia included propofol, sufentanil, and continuous propofol infusion.	A rare neurosurgical TCR presentation triggered solely by temporalis muscle stretch. Highlights the need for vigilance in areas beyond classic TCR zones. First such reported case emphasises intraoperative communication and preparedness.
Morioka R et al., 2023 [9]	56-year-old female with a zygomatic maxillary complex fracture developed sudden asystole during repositioning of the zygoma under general anaesthesia.	Immediate chest compression by the surgical team; patient reverted to sinus rhythm and recovered without complications.	Rare case of asystole due to TCR during maxillofacial surgery. Emphasises that even non cranial surgeries involving facial bones can provoke severe TCR. Surgeons and anaesthesiologists must remain vigilant. Incidence of TCR in maxillofacial procedures is low but potentially life-threatening.
Present case	11-year-old boy with a recurrent right-sided optic glioma underwent elective craniotomy. During dissection near the superior orbital fissure and cavernous sinus patient developed sudden bradycardia and hypotension.	Immediate cessation of surgical procedure and deepening of anaesthesia using propofol boluses. Injection atropine 10 mcg/kg i.v. was administered to counteract the vagal response and warm saline irrigation in the surgical field was given to blunt the local nerve stimulation. Continuous intra-arterial blood pressure and ECG monitoring was done to ensure no such further events occur. Meticulous teamwork between the surgeon and the anaesthesiologist ensured safe continuation of the procedure with intermittent prophylactic propofol boluses were given during high-risk surgical manipulations. The child was electively ventilated postoperatively for 2 days and then extubated after meeting the extubation criteria.	Trigeminocardiac Reflex (TCR) is a possibly life-threatening and hazardous yet reversible event, which is seen intraoperatively especially in paediatric age group those undergoing neurosurgical procedures involving regions of the skull base or orbital area. Early recognition, immediate cessation of the surgical stimulus, adequate anaesthetic depth, and prompt administration of anticholinergics are key to successful management. Conscious vigilance can improve patient safety, and early measures can avert negative outcomes during high-risk neurosurgery procedures.

[Table/Fig-4]: Management of TCR in various cases [5-9].

CONCLUSION(S)

This case highlights the paramount importance of pre-emptive anticipation of reflex neurocardiogenic reactions in operations on anatomically sensitive areas. It illustrates the key role of anaesthesiologists in inclusive perioperative planning, proactive intraoperative monitoring, and immediate crisis management. Increased knowledge of TCR, especially in children, facilitates prompt detection and effective intervention, markedly enhancing patient safety and outcomes. In addition, this case underscores the importance of continuous, anticipatory communication between anaesthetic and surgical teams to navigate intricate neurosurgical hurdles successfully.

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