# Trigeminocardiac reflex unresponsive to atropine during microvascular decompression of trigeminal nerve root: *A potentially lethal complication of a simple surgery*

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## INTRODUCTION

Microvascular decompression (MVD) of trigeminal nerve root entry zone is a well described technique in the treatment of trigeminal neuralgia. It is considered safe and is reported to have a high level of effectiveness.[1] Trigeminocardiac reflex (TCR), a reflex consisting of bradycardia, hypotension, apnoea and gastric hypermotility is reported to occur, during craniofacial surgery,[2,3] cerebellopontine angle surgery,[4] percutaneous micro decompression of trigeminal ganglion[5,6] and MVD for trigeminal neuralgia.[7] We describe a case of TCR during MVD in a patient on chronic amitryptyline and carbamazepine therapy presenting as bradycardia followed by cardiac arrest nonresponsive to atropine, adrenaline and dopamine. This was successfully treated with isoprenaline and noradrenaline.

## CASE REPORT

A 55 year old (height 168 cm, weight 70 kilogram) male, who was a chronic smoker, was scheduled for MVD of the trigeminal nerve for facial pain. The patient had been refractory to medical treatment with oral carbamazepine 400 mg twice daily and oral amitryptyline 25 mg once daily, for the previous two years. His past history was unremarkable except that he was diagnosed as being hypertensive during the pre-anaesthetic checkup. This was controlled on oral amlodipine 5 mg and oral atenolol 50 mg once daily, for 10 days. The patient did not have any cardiovascular symptoms. He had a complete cataract in the right eye. Pre-operative blood haematology and biochemistry were within normal limits. Electrocardiography (ECG) showed left axis deviation, a heart rate of 84/min, regular, with a PR interval of 144 ms, without any evidence of heart block. Chest X-ray demonstrated emphysematous changes, and echocardiography revealed an ejection fraction of 55%, no regional wall motion abnormality, and normal systolic and diastolic function. Magnetic resonance imaging of the brain was reported as normal.

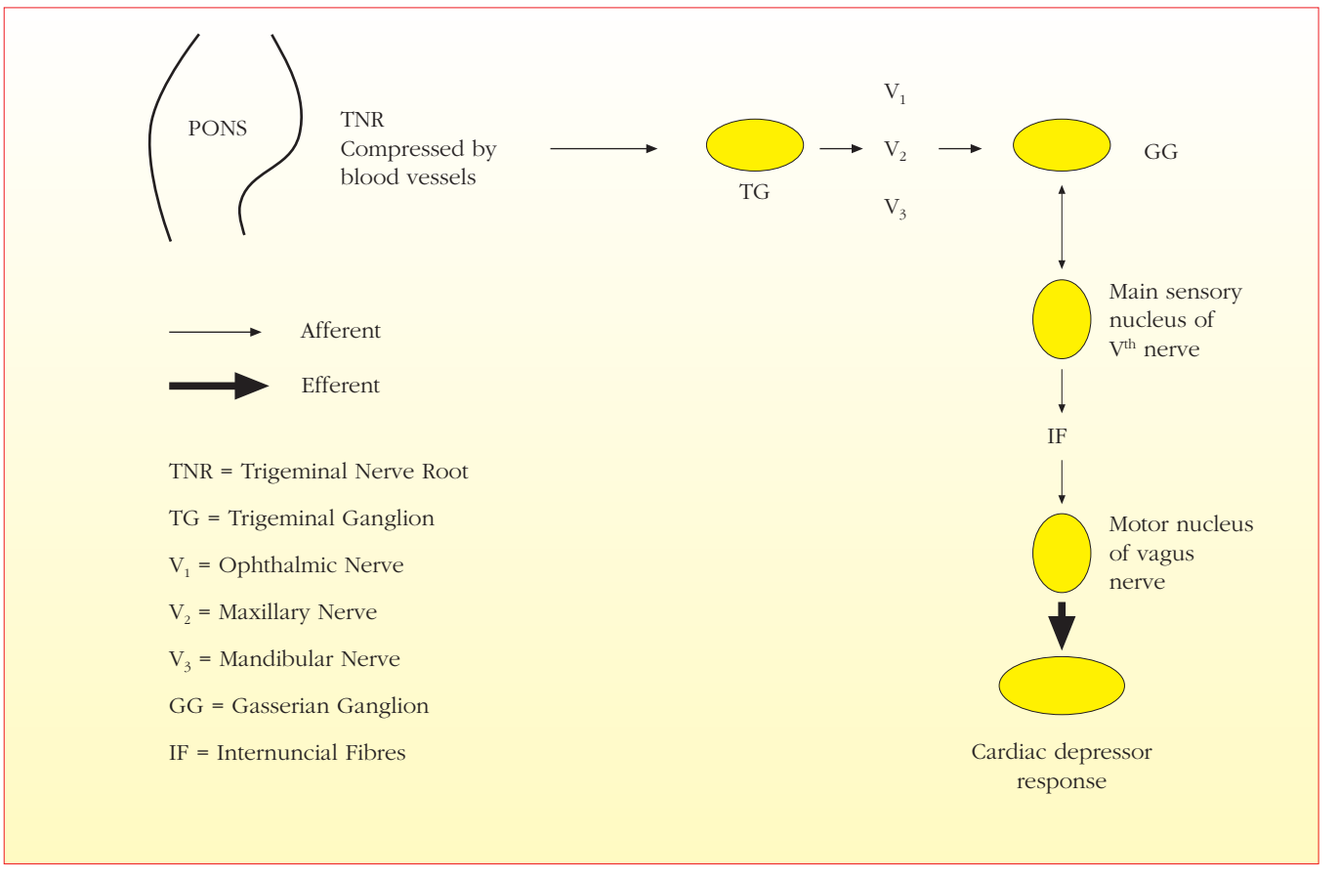
The patient was premeditated with diazepam 10 mg and ranitidine 150 mg orally at bedtime, and two hours prior to surgery. Amlodipine is a calcium channel blocker and is normally continued until the morning of surgery. It does not contribute to bradycardia, as one of the side-effects of calcium channel blockers is tachycardia. Anaesthesia was induced with propofol 1.5 mg/kg and morphine150 g/kg intravenously. Ecuronium 7 mg was administered intravenously to facilitate tracheal intubation. The trachea was intubated with an 8.5 mm internal diameter (ID) cuffed tube. The right radial artery was cannulated after induction. Monitoring included invasive arterial blood pressure (IABP), oxygen saturation ($SpO_2$), ECG, end tidal carbon dioxide ($EtCO_2$), and temperature. Anaesthesia was maintained with 67% $N_2O$ and 33% oxygen, propofol infusion and intermittent vecuronium. The patient was preloaded with one litre of normal saline. His position was changed from supine to right lateral decubitus. The intra-operative period of one hour was uneventful, and the arterial blood gas (ABG) that was done as part of the monitoring was within normal limits. Towards the end of surgery, when the surgeon was introducing the Teflon sponge between the trigeminal nerve root and the superior cerebellar artery, the patient developed sudden bradycardia. The heart rate (HR) decreased from 60 to 30/min (a 50% decrease) and the IABP decreased from 130/90 (103) to 60/40 (46) mmHg (57% decrease). The surgeon was requested to stop the surgery. Propofol and $N_2O$ were discontinued, and the patient was ventilated with 100% oxygen. Atropine 0.6 mg was administered and a further 1.2 mg repeated without any response. The patient was given adrenaline 10 mcg intravenously, cardiac massage was commenced, and dopamine was started at 10 g/kg/min. The patient simultaneously developed prolonged A-V conduction, followed by broad complexes and ventricular tachycardia. Defibrillation with 200J, 300J and 300J was attempted, followed by cardiopulmonary resuscitation. Intravenous adrenaline 1 mg was administered via the peripheral vein, and defibrillation was repeated. The patient developed A-V junctional bradycardia and hypotension which responded to the intravenous infusion of isoprenaline 4 g/min, and noradrenaline 4 g/min, and reverted to sinus rhythm. An ABG performed immediately after resuscitation showed a pH of 6.92, $PaO_2$ of 453 mmHg, $PaCO_2$ of 45 mmHg, $HCO_3$ = 9, BE = -22, $SpO_2$ = 99%. The serum potassium ($K^+$) was 4.5 meq/l. Sodium bicarbonate 230 meq was administered intravenously. There was no acidosis prior to the event. The duration of the cardiac arrest was 5 minutes, and the ABG sample was taken after sinus rhythm was restored. At that time hypotension was still present, and this could have led to the metabolic acidosis. Subsequent ABG showed the following: pH = 7.05, $PaO_2$ = 174 mmHg, $PaCO_2$ = 40 mmHg, $HCO_3$ = 16, BE = -14, $SapO_2$ = 98.4%,

$K^+$ 4.5 meq/l. Sodium bicarbonate 150 meq was repeated intravenously. The post resuscitation Glasgow Coma Scale (GCS) was $E_1V\_M_1$. The left pupil was dilated and was not reacting to light. The patient's temperature was 36 degree Celsius, IABP was 86/44(58) mmHg and HR was 130/min. The right internal jugular vein was cannulated and surgery was completed. Post operative CT scan of the head showed a normal study. The ECG showed sinus tachycardia and chest X-ray confirmed the correct placement of the central line. After four hours, the troponin T test was negative, GCS improved to $E_2V\_M_6$, the left pupil was normal in size and reacting to light. The IABP was 110/70(83) mmHg, HR 120/min, the ABG was normal and CVP was 12mmHg. Isoprenaline infusion was stopped. After twelve hours the GCS was $E_4V\_M_6$, IABP was 116/70(85) mmHg, HR 106/min, ABG was normal, CVP was 11 mmHg and urine output was 100 ml/hr. The infusion of noradrenaline was stopped and infusion dopamine was reduced to 5 g/kg/min. After twenty fours hours, the patient's GCS was $E_4V\_M_6$ with no neurological deficit, IABP was 130/86 (100) mmHg, HR was 94/min and hence his trachea was exubated. The patient had complete relief of his facial pain and did not require antihypertensive therapy for the control of blood pressure.

## DISCUSSION

TCR is defined as a decrease in mean arterial blood pressure and heart rate of more than 20% of the base line values before the stimulus, coinciding with the manipulation of the trigeminal nerve.[7] The trigeminal nerve and the vagus nerve constitute the afferent and efferent pathways in the reflex arc[8] (Figure1). Interruption of the surgical manoeuver immediately following TCR is considered sufficient for heart rate and blood pressure to return to normal without the necessity of adding cholinergic medication, in most cases.[3] This reflex has been discussed frequently in ophthalmology, as well as during maxillofacial surgery, but less attention has been paid to it, especially during the surgical manipulation of the trigeminal ganglion and root. The occurrence of significant autonomic changes during percutaneous compression of the trigeminal ganglion has been reported.[6,9] The incidence of TCR during MVD for trigeminal neuralgia under a standardised anaesthetic protocol is reported as 18% (5/28 patients) in only one retrospective study.[7] In this study, TCR reverted back to normal after cessation of the stimulus and administration of atropine, leading to the cessation of the reflex during the remainder of the procedure. Recurrent asystole due to TCR after the transaction of the sensory root of the trigeminal nerve for palliation of intractable trigeminal pain has been reported in a 72 year male. This responded to the intravenous administration of 150 g atropine and 1.0 mg of epinephrine.[8] Several factors are known to increase the risk of recurrence of TCR i.e. light plane of anaesthesia, hypercapnia, hypoxaemia and the nature of the stimulus (strong and long). These factors were ruled out in the case under review. Long term therapy

**Figure 1:** The trigeminocardiac reflex pathway



with drugs i.e. chronic calcium channel blocker, beta-blocker and carbamazepine may predispose the patient to this reflex.[7] The strongest association (80%) has been reported between chronic carbamazepine therapy and TCR.[7] In the case under review, chronic carbamazepine therapy probably predisposed the patient to TCR.

In the differential diagnosis of the intra-operative bradycardia and hypotension, several possibilities were considered. Hypovolaemia was ruled out, as the patient had received an infusion of one litre of fluid and intra-operative blood loss was minimal. Electrolyte imbalance was excluded, as an ABG done prior to the event was within normal limits. The bradycardia and hypotension that occurred were not preceded by either hypoxia or hypercarbia. Myocardial ischaemia was ruled out, as the post-operative ECG showed sinus tachycardia and the troponin T test was negative. A special note to exclude venous air embolism needs to be mentioned, as there was no tachycardia, right ventricular strain pattern, decrease in $EtCO_2$ or hypoxia at the time of the event.

Patients on chronic tricyclic antidepressant therapy (TCA) show decreased sensitivity to atropine.[10] Since their adrenergic receptors are either desensitised or their catecholamines are depleted, only direct acting sympathomimetics are effective in such patients.[11] Noradrenaline has been successfully used to manage circulatory shock, whilst dopamine was ineffective in a patient on chronic TCA therapy.[12] The short term infusion of 0.005 - 0.008 g/kg/min of isoprenaline is reported to improve atrial function after cardioversion.[13] In the case under review, bradycardia and hypotension towards the end of surgery most probably occurred due to TCR, which was precipitated by chronic carbamazepine therapy, and did not respond to atropine and dopamine, probably because of chronic amitryptyline therapy. Junctional bradycardia and hypotension after defibrillation successfully responded to the infusion of isoprenaline and norepinephrine in the case under review. The isoprenaline improved the atrial function and noradrenaline corrected the hypotension. However, the effect of adrenaline and dopamine in the successful resuscitation cannot be completely excluded for the following reasons: delivery via a peripheral vein during CPR may be inadequate; the ability of adrenaline to displace _ blockers from occupied receptors, increasing susceptibility to the subsequent administration of other _ stimulant drugs should not be overlooked. However, patients on chronic antidepressants respond to the blood pressure effects of direct-acting sympathomimetics, ranging from two to three times greater for phenylephrine, four to eight times greater for norepinephrine and two to four times greater for epinephrine.[12]

## CONCLUSIONS

Since patients with trigeminal neuralgia are frequently on chronic carbamazepine and antidepressant therapy, the predisposition of such patients to TCR, which is unresponsive to atropine and dopamine, should be kept in mind. TCR during manipulation of the trigeminal ganglion and root is usually self-limiting. However, at times, complications like cardiac arrest non-responsive to the usual resuscitation measures may occur, leading to significant morbidity. **SAJAA**

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