Topical lidocaine to suppress trigemino-cardiac reflex

Editor—A 68-yr-old woman had undergone microvascular decompression (MVD) 2 yr ago for trigeminal neuralgia but presented on this occasion for recurrence of symptoms. She was again scheduled for MVD by the same surgeon. Her medical history was unremarkable and all the laboratory investigations and ECG were within normal limits. Her medications included carbamazepine 200 mg and pregabalin 75 mg once daily.

Standard monitoring was established and anaesthesia was induced with fentanyl 100 μg, propofol 100 mg, and muscle relaxation achieved with vecuronium 5 mg after which the airway was secured with an oral tracheal tube. The lungs were ventilated with 50% O2 in N2O and sevoflurane to an E\text{\textsubscript{CO\textsubscript{2}}} 4.0–4.5 kPa. After induction, the left radial artery was cannulated for direct arterial pressure monitoring.

After craniotomy, the surgeon found an additional vascular loop at the root entry zone (REZ) and the graft placed at the previous surgery was in situ. During dissection, there was sudden and profound bradycardia (30 beats min\textsuperscript{-1}) and hypotension (60/40 mm Hg). As the surgeon was alerted to stop the stimulus and atropine 0.6 mg was being given i.v., the monitor showed asystole which lasted for about 10 s and then there was a gradual increase in the heart rate to 70 beats min\textsuperscript{-1} and arterial pressure increased to 120/70 mm Hg.

After waiting for 5 min, when the surgeon resumed the procedure, similar changes occurred including asystole which reverted to normal rhythm with the removal of the stimulus and without therapeutic intervention. The surgical and anaesthetic team were in a dilemma. Then, one of the authors (N.N.V.) advised the surgeon to place a gauze dipped in 2 ml of 2% lidocaine at the REZ. After 3 min of waiting with the gauze in place, the surgery was resumed and completed without any haemodynamic fluctuations. The surgeon could not recall any serious haemodynamic changes, nor did the previous anaesthetic chart show any such changes, during the previous surgery.

Residual neuromuscular block was reversed, the patient extubated, and the postoperative ECG was normal.

The afferent arc of the trigemino-cardiac reflex (TCR) is the trigeminal nerve and vagus nerve the efferent. The manifestations range from bradycardia, bradycardia terminating in asystole, hypotension, apnoea, and gastric hypermotility.

In our case, the fifth nerve was approached by the same surgeon using the same surgical approach and a similar anaesthetic technique. There was neither bradycardia nor asystole during the first surgery.

Postulated predisposing factors for TCR include hypotension, hypercapnia, hypoxaemia, light anaesthesia, opioids, calcium channel blockers, β-blockers, abrupt and sustained traction and surgical division of the nerve.

As there was no attempt at sectioning the nerve in this case, the only other factor was the surgery with the previously placed graft in situ, which could have been more reflexogenic.

Most reported cases of TCR reveal that the surgery was completed uneventfully after the administration of i.v. anticholinergic medication. But in our case the reflex returned despite anticholinergics. We thus resorted to topical application of local anaesthetic which proved to be effective. To our knowledge, there has been no case report of the use of topical lidocaine intraoperatively to blunt the afferent arc, although mention has been made of local anaesthetic infiltration or nerve block.

Declaration of interest

None declared.

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