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Transvenous Pacing for the Anesthetic Management of Surgery for Glossopharyngeal Neuralgia

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Glossopharyngeal neuralgia is a rare clinical occurrence characterized by paroxysms of pain in the sensory distribution of the ninth cranial nerve. The typical pain is an excruciating repetitive series of electriclike stabs in the region of the tonsil, the base of the tongue, and the ipsilateral side of the face.¹ In many cases, pain radiates to the ear.² Swallowing, chewing, coughing, and talking can trigger the attacks.³ Of greater concern is the occurrence, though rare, of severe bradycardia and cardiac arrest,^{4,5} sometimes associated with syncope⁶ and seizure.⁷ We report a patient with severe glossopharyngeal neuralgia in whom repeated episodes of cardiac arrest occurred during a right retromastoid craniectomy.

CASE REPORT

A 48-yr-old woman was first diagnosed as having right glossopharyngeal neuralgia in 1986. Since that time she had been taking carbamazepine 200 mg three times per day with good relief of her symptoms. In June 1989, she experienced recurrence of the pain, which was partially relieved by increasing her medication to 1 g per day. She was readmitted in August 1989, complaining of very severe pharyngeal pain radiating to the ipsilateral ear and the mastoid area. The neuralgia was triggered by irritation of her right pharynx. During the last few days before her admission, the symptoms were so severe that the patient refused to eat and even to talk because of her fear of provoking an attack. The next day, severe pharyngeal pain occurred suddenly and was followed by bradycardia and loss of consciousness, which responded promptly to intravenous atropine. The same day, within 1 h, she experienced three other similar attacks, which were followed by tonic jerking of the four limbs. She was then transferred to the intensive care unit (ICU), where she had two other episodes, including one with a documented 6-s asystole. She was treated with atropine 0.4 mg iv every 6 h and with lidocaine spray on her right oropharynx every 2 h. The decision was made to proceed with surgery the next morning.

Prior to surgery the next morning, the physical examination was normal except for mild dehydration. The preoperative laboratory values, ECG, EEG, and chest x-ray all were within normal limits. The patient received atropine 0.4 mg iv and 2 mg sublingual lorazepam as preanesthetic medication. The ECG was continuously monitored as she was transferred from the ICU to the operating room. Monitoring

included two-lead ECG, intraarterial blood pressure, temperature probe, pulse oximetry, capnography, and gas analysis by mass spectrometry. A Swan Ganz Paceport[®] catheter was inserted under local anesthesia *via* the left subclavian vein, and the pacing probe was positioned into the right ventricle. The minimal stimulating threshold was 1 mA.

Before induction, the oropharynx, particularly on the right side, was anesthetized with topically applied lidocaine. Anesthesia was induced with sufentanil in divided doses up to 1 µg/kg and midazolam 0.35 mg/kg. Vecuronium 0.15 µg/kg was given to facilitate tracheal intubation. Anesthesia was maintained with increments of sufentanil and isoflurane (0.2–0.5% end-tidal) in nitrous oxide and oxygen (50:50).

After induction, the patient was placed in the semi-lateral position. When the surgical field was being prepared, she had an asystole that lasted about 10 s. The prepping was immediately stopped, and was resumed only after infiltration of the skin of the retromastoid area with lidocaine. At the craniectomy, the patient experienced another asystole of much longer duration, necessitating pacing for more than 2 min (fig. 1). At that time, blood pressure decreased from 120/80 to 80/50. It returned to baseline when normal sinus rhythm returned. The same phenomenon also occurred at the opening of the dura and during posterior fossa dissection, and each time lasted approximately 45 s. Before the final approach to the nerve, atropine 0.4 mg iv was given, increasing the heart rate from 82 to 95 beats per min. Despite this prophylaxis, the patient experienced another similar episode, which lasted 2.5 min. However, in this episode, blood pressure decreased to 60/30 despite pacing at 80 beats per min and two additional 0.4-mg boluses of iv atropine. A phenylephrine infusion was necessary to restore normal blood pressure. There was no recurrence of asystole or bradycardia after section of the ninth and partial section of the tenth nerve, as recommended.⁸ The patient made an uneventful recovery and was discharged from the hospital on the 14th postoperative day without any medication.

DISCUSSION

The incidence of glossopharyngeal neuralgia is about 1.5% that of trigeminal neuralgia.² Only 10% of glossopharyngeal neuralgia patients present with severe bradycardia or asystole, which sometimes produces syncope and seizures secondary to cerebral ischemia.⁹ Cardiac arrests have been explained by abnormal visceral afferent impulses originating from the trigger zones *via* the ninth nerve fibers. These impulses cause an abrupt discharge of the solitary tract neurons and spill-over to both the ambiguous and dorsal motor nuclei of the vagus.⁹ In our patient, asystole was triggered by preparation of the retromastoid area and by traction on the dura. We have not found any previous report of such an occurrence. The sensitivity of these areas is supplied by the somatic

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FIG. 1. ECG tracing showing asystole and pacemaker rhythm.

fibers of the ninth nerve. These fibers reach the spinal tract and spinal nucleus of the trigeminal nerve.³⁻¹⁰ Because these structures are in close relationship with the dorsal motor vagal nucleus, we speculate that the asystole was also mediated by the same spill-over mechanism.

Anesthetic management of neuralgic episodes should focus mainly on prevention of their onset and on preparation to treat them, should they occur. Prophylactic intravenous administration of atropine and local anesthesia of the pharynx before laryngoscopy have been recommended.¹¹ However, the asystole encountered during the preparation of the retromastoid area and at the opening of the dura indicates that these two structures should also be infiltrated directly with local anesthetic and that the whole medical team should be aware of their potential to trigger asystole.

This case is the first report of peroperative treatment of an asystole with a pacemaker during this procedure. It shows that despite careful prophylaxis, the risk of bradyarrhythmia is still present. Previous reports have emphasized the unreliability of isoproterenol to prevent and treat severe bradycardia and asystole.⁹ The last asystole in this patient occurred after prophylactic atropine and persisted despite two supplementary 0.4-mg boluses of atropine. Although a larger dose might have been more effective, this case suggests that atropine may not be totally reliable in the prevention or treatment of this complication. Furthermore, because the patient was in the semi-lateral position with her head in the Mayfield head-holder, it would have been very difficult to provide adequate CPR. Without the pacemaker, there certainly would have been a delay in reestablishing circulation, and the outcome probably would have been less satisfactory. During the final asystole, our patient was also hypotensive even as her heart was paced at 80 beats per min and even when three 0.4-mg doses of atropine were given. Hypotension despite adequate pacing has been reported in the neu-

rologic literature.¹² It is presumably caused by inhibition of the vasomotor center, leading to systemic vasodilation and a decrease of blood pressure. A phenylephrine infusion was useful in treating this occurrence.

In conclusion, we report anesthetic management of a patient with severe glossopharyngeal neuralgia. If clinical signs of syncope or documented bradyarrhythmia are present preoperatively, cardiac pacing should be considered. We also conclude that the retromastoid area and the dura are potential trigger zones and should be infiltrated before being manipulated.

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