Glossopharyngeal Neuralgia—Implications for the Anesthesiologist

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Glossopharyngeal neuralgia, first described by Weisenburg in 1910, has been said [1] to occur only one-hundredth as frequently as its relatively rare counterpart, trigeminal neuralgia. Although there are few distinguishing clinical or laboratory findings with this disease, the primary complaint is periodic lancinating pain which begins in the posterior pharynx or tongue and radiates to the angle of the jaw or ear. The pain is usually spontaneous in onset but can often be precipitated by swallowing, coughing, or talking. Treatment with carbamazepine (Tegretol) and/or diphenylhydantoin (Dilantin) is often effective in relieving symptoms, although resistant cases, and those which involve drug toxicity, can be treated surgically. The anesthesiologist may make a significant contribution in the diagnosis and management of this disease, as the following case history illustrates.

REPORT OF A CASE

A 50-year-old Caucasian woman was first seen at The Mason Clinic because of complaints of episodic pain in the right neck of three weeks' duration. Approximately 10 to 25 times per day severe knife-like paroxysms of pain occurred behind the right mandibular angle and radiated into the ear. Typical pain could be elicited by direct stimulation of the right posterior pharynx. Anesthetizing the posterior tongue and pharynx by the topical application of viscous 2 per cent lidocaine blocked this response. To confirm the diagnosis of glossopharyngeal neuralgia, block of the right ninth cranial nerve was accomplished with 4 ml of 0.25 per cent tetracaine with 1:200,000 epinephrine. This block produced complete relief of pain, and the pain could not be elicited even by direct posterior pharyngeal stimulation. A trial of carbamazepine and diphenylhydantoin was moderately effective in this patient, but she developed leukopenia. It was elected, therefore, to discontinue drug therapy and perform intracranial section of the ninth cranial nerve. The patient was anesthetized with halothane/nitrous oxide in the sitting position and the right ninth cranial nerve was exposed through a posterior occipital craniectomy. At the time of manipulation and avulsion of the nerve, the patient developed acute hypertension and an irregular pulse. The blood pressure rose acutely from 90/60 torr to 150/100 torr, and the oscilloscope electrocardiogram showed a chaotic rhythm of multifocal premature contractions and short runs of tachycardia. This arrhythmia did not respond to a total of 300 mg lidocaine, iv. Only after the nerve section had been accomplished and the retractors removed from the area of the brainstem did normal sinus rhythm reappear. Blood–gas values of arterial blood drawn during the period of cardiac irregularity were the same as those of an earlier sample, pH 7.37, P_{CO_2} 41 torr, P_{O_2} 140 torr. Blood pressure gradually returned to normal and the operation was completed without further incident. The patient had an uncomplicated postoperative course and has had no recurrence of the pain.

DISCUSSION

In the long delay between the onset of this patient's symptoms and definitive treatment numerous efforts at diagnosis and treatment were made. Important factors in the diagnosis were the marked relief of pain in response to viscous lidocaine given orally and the complete relief following glossopharyngeal nerve block. Although it is not commonly performed, block of the ninth cranial nerve is not difficult. Labat first described a technique in which a local anesthetic was injected in a fan-wise fashion just above the hyoid bone. An easier and more acceptable approach was described by Rovenstine and Papper in 1948.[2] Injection is made midway on a line connecting the tip of the mastoid process with the angle of the mandible. At a depth of 2 to 4 cm, contact is made with the styloid process. Reinserting the needle posterior to the styloid and 0.5 to 1 cm deeper will bring the point near the glossopharyngeal nerve. A volume of 3 to 5 ml of local anesthetic solution is injected. Paresthesias are not sought. There may be slight difficulty in locating the styloid process, as it is a very thin spicule of bone. However, careful search at the proper depth

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should reveal it without undue discomfort to the patient. Although good anesthesia of the glossopharyngeal nerve can be obtained for diagnostic purposes, it is not tenable to consider injection of neurolytic drugs such as alcohol into the peripheral nerve. The tenth and eleventh cranial nerves leave the skull with the ninth cranial nerve through the jugular foramen and are in close proximity to the internal carotid artery and internal jugular vein. Since damage to these adjacent structures could occur with injection of neurolytic agents, glossopharyngeal nerve block is best used as a diagnostic measure only.

Carbamazepine was effective for producing relief of pain in this patient. However, toxic effects of this drug are not uncommon, and periodic checks of blood count and liver function should be performed. Although carbamazepine was first used to treat cranial-nerve neuralgias in 1962, the total clinical experience to this time does not provide conclusive data regarding the incidence of failure or the duration that the drug's effect will persist.[3] There does appear to be a significant number of patients who will "break through" or develop decreasing responsiveness despite increasing doses of the drug.[4]

If medical treatment fails, one of a variety of surgical procedures should be considered. One approach is to perform a cervical laminectomy using local anesthesia. By mechanical stimulation of fibers within the descending trigeminal tract, the responsible pain fibers can be localized precisely and selective tractotomy performed.[5]

The surgical procedure used in this patient was intracranial section of the rootlets of the ninth cranial nerve. Various cardiovascular disturbances, such as hypertension, premature contractions, and tachycardia, occurring during the course of this operation, have been reported.[6] Some of these responses might have been initiated by the sudden loss of sensory input from baroreceptors on the ipsilateral side of the nerve section. Likewise, direct pressure on cardioregulatory centers of the brainstem can initiate cardiac or vasomotor changes. Attacks of pain in the awake patient may produce an "overflow" of impulses from the ninth cranial nerve into the vagal motor nucleus, resulting in bradycardia, cardiac arrest, and syncope. Such responses may be blocked with atropine.[7]

In conclusion, a knowledge of the anatomy of the fifth, seventh, ninth, and tenth cranial nerves is important in evaluating pain problems in the region of the throat and ear. The afferent pain fibers in these four nerves unite in a common bundle which becomes the descending trigeminal tract of the brainstem, and this fact should be considered in understanding the etiology, treatment, and complications in cases of cranial nerve neuralgia. Nerve blocks are helpful in achieving a proper diagnosis of glossopharyngeal neuralgia. Arrhythmias and blood pressure problems are frequently seen during operations for this disease, and the cardiac status should be closely monitored. Intravenous administration of lidocaine and atropine may be indicated. A remarkable correlation of anatomy, physiology, and pharmacology can be found in the medical management of this rare condition.

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