Cardiovascular complications on upper vagal rootlet section for glossopharyngeal neuralgia

Case report

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Acute hypotension and right bundle-branch block occurred when the authors sectioned the uppermost rootlet of the vagus nerve in a case of glossopharyngeal neuralgia. Hypotension lasted for 20 minutes and arrhythmia for 4 days. A possible mechanism is discussed and cardiovascular disorders are reviewed in similar cases.

Key Words - glossopharyngeal nerve • vagus nerve • neuralgia • arrhythmia

Glossopharyngeal neuralgia is a rare disease and when it does occur often involves the vagus nerve as well. Its symptoms frequently recur unless the ninth nerve and the upper two rootlets of the vagus nerve are divided; therefore, combined sectioning has become the standard surgical treatment. Transient elevation in blood pressure after unilateral section of the ninth nerve was first reported by Bucy in 1936, and has since been reported by others. Bilateral intracranial ninth nerve section yielded immediate and pronounced rise in blood pressure.

The cardiovascular effects of unilateral sectioning of both the ninth nerve and the upper rootlets of the tenth nerve are not fully elucidated, although transient auricular flutter and ectopic ventricular contraction have been reported. We are reporting such a case in order to call attention to the importance of recognizing the cardiovascular problems of this combined sectioning for therapeutic reasons.

Case Report

A 74-year-old woman was first admitted to Saitama Medical School Hospital in 1971, complaining of severe pain in the left side of the throat and neck while eating. A trial of carbamazepine and diphenylhydantoin was effective at that time. The patient was discharged and continued on carbamazepine therapy. In 1974, severe stabbing, lancinating paroxysms of pain occurred 10 times a day in the left throat, and radiated into the pre- and retroauricular region. The pain was not alleviated by carbamazepine 1200 mg daily.
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FIG 1. Blood pressure and pulse recording. No change occurred on cutting the ninth nerve. Two episodes of acute hypotension were noted on manipulating the tenth nerve.

Examination. Typical pain could be elicited by talking, laughing, swallowing saliva, touching or pushing the left tragus (tragus sign), the external auditory meatus, or pulling the left ear. To confirm the diagnosis, a block of the left ninth nerve with 2 ml of 0.5% Xylocaine which produced complete relief of pain for 30 minutes to 1 hour. The electrocardiogram was normal with or without an attack of pain. Bilateral vertebral angiograms were negative. No elongated styloid process was noted in the skull films.

Operation. The patient was placed in the sitting position, with electrocardiographic (EKG) monitor and anesthetized with halothane, nitrous oxide and oxygen, supplemented by droperidol and pentazocine. The left ninth and tenth cranial nerves were exposed through a left suboccipital craniectomy. With microsurgical instrumentation, the left ninth nerve was divided first. Prior to the section, 0.2 mg of atropine sulphate was given intravenously: the pulse rate increased from 64 to 90/min (Fig. 1), and the blood pressure from 130/60 to 140/65 mm Hg. Ninth nerve section produced no change on the EKG, pulse rate and blood pressure (Fig. 2).

When the upper part of the vagus nerve was touched, an extrasystole suddenly occurred and was followed by acute hypotension (from 130/60 to 62 mm Hg) (Figs. 1 and 3). These responded to atropine sulphate and Carnigen (sprifen and adenosin); the blood pressure returned to 130/44 mm Hg, and the pulse rate increased. The surgeon (C.N.) felt that this
FIG 3. Electrocardiogram taken upon touching upper part of the tenth nerve. After the nerve was touched (arrow), the chain of events that occurred is continuously recorded. The extrasystole on touching the nerve is shown on the top, and another spontaneous extrasystole on the bottom.

represented a vagal depressor response secondary to hypersensitivity of the vagus nerve and therefore only the uppermost rootlet, rather than two rootlets, of the vagus nerve was cut to lessen the irritation. The blood pressure again dropped abruptly to 78/50 mm Hg (Fig. 1). The EKG showed a downward displacement of the S-T segment followed by a right bundle-branch block (Fig. 4). The hypotension and bundle-branch block gradually responded to atropine sulphate, Persantin (dipyridamole), and Carnigen during the next 20 minutes (Fig. 1), but apparently there was sufficient hypoxia to the brain to cause postoperative lethargy, disorientation, delusions and hallucinations. These symptoms completely disappeared on the fifth postoperative day.

Postoperative Course. The patient has been entirely free of pain during a follow-up period of 9 months, without neurological or mental deficit except for a diminished left gag reflex and taste on the left posterior third of the tongue. Attempts to reproduce the pain by stimulation of the tonsillar fossa, tragus, and the ear, have been unsuccessful. She does not have any decreased sensation in the external auditory meatus or tragus.

Discussion

Bucy's observation started a whole new train of investigation. He reported a marked rise in blood pressure after intracranial section of the ninth nerve in four cases of glossopharyngeal neuralgia, which lasted for several days. This was thought to be caused by interruption of the carotid sinus nerve, a branch of the glossopharyngeal nerve. Complete cardiac arrest, syncope, and a generalized convulsion associated with a paroxysm of glossopharyngeal neuralgia was first reported in 1942, and has since been reported by other investigators. Kjellin, et al., with simultaneous records of EKG, respiration, and blood pressure during a spontaneous attack of pain, demonstrated a severe fall in blood pressure and syncope, followed by
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irregular heart beats.Massage over the carotid sinus on both sides produced neither pain nor alteration of cardiac rate. A review of the literature also reveals that carotid sinus massage fails to produce alteration in cardiac rate, and it appears that the asystole is due to hypersensitivity of the dorsal motor nucleus of the vagus rather than to increased sensitivity of the carotid sinus.\textsuperscript{6,8,9}

In our patient, the EKG showed an extrasystole when the vagus was touched, followed by acute hypotension which gradually responded to atropine. This vagal depressor effect was reproduced by cutting the uppermost rootlet of the vagus. The right bundle-branch block which occurred during the second episode of hypotension is considered to be due to repeated attacks of coronary artery insufficiency that led to interference with the conducting system in the myocardium, with progressive depression of the S-T segment on EKG (Fig. 4).

Cardiovascular disorders produced when both the ninth nerve and a rootlet of the tenth nerve are sectioned, are summarized in Table 1. Acosta and Clark's patient, who had had cardiac arrests during a paroxysm, showed ectopic ventricular contractions for 2 hours after the nerve section.\textsuperscript{1} Transient auricular flutter is also noted in "Case 3" of the series of Chawla and Falconer.\textsuperscript{3}

The exact mechanism responsible for a vagal depressor response in our case is obscure. The most plausible explanation is a chronically induced hyperirritability of the dorsal motor nucleus of the vagus nerve as a result of intense bombardment of afferent impulses over a long period from the pharynx, tragus, and ear. These impulses could have been transmitted either directly to the vagal nucleus, or by way of secondary afferent collaterals\textsuperscript{6,8} from the nucleus of the tractus solitarius of the ninth nerve. The dorsal motor nucleus of the vagus is the principal autonomic nucleus supply to parasympathetic fibers to the heart, bronchi, and abdominal viscera. The degree of vagal inhibition to the heart appears to vary in in-

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**Fig. 4.** Electrocardiogram taken upon cutting the uppermost rootlet of the tenth nerve. Continuous record. After cutting, ST segment progressively lowered (top line); right bundle-branch block first appeared on the middle line, which became more marked and continuous (bottom line).
individuals. The carotid sinus nerve is another important afferent branch of the glossopharyngeal nerve that plays a role in the regulation of the heart rate. A hypersensitive carotid sinus nerve is unlikely, since the ninth nerve had been cut before manipulation of the tenth nerve. Since arrhythmia and blood pressure problems are not infrequently seen during operation for this disease, the cardiac status should be closely monitored throughout the surgery.

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References


C. Nagashima, et al.

TABLE 1
Operative cardiovascular disorders reported in the literature

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Age, Sex</th>
<th>Preoperative State*</th>
<th>Nerves Divided</th>
<th>Cardiovascular Changes</th>
<th>Duration of Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Svien, et al. (1957)</td>
<td>43 M</td>
<td>idiopathic GPN, syncope, cardiac arrest during attack</td>
<td>lt. 9th rostral quarter of vagus</td>
<td>tachycardia (80-100/min), no arrhythmia, no hyper- or hypotension</td>
<td>4 days</td>
</tr>
<tr>
<td>Chawla &amp; Falconer (1967)</td>
<td>70 M</td>
<td>idiopathic GPN cardiac arrest during attack</td>
<td>9th, upper two rootlets of vagus</td>
<td>auricular flutter</td>
<td>transient</td>
</tr>
<tr>
<td>Acosta &amp; Clark (1970)</td>
<td>46 F</td>
<td>idiopathic GPN cardiac arrest during attack</td>
<td>rt. 9th upper three layers of vagus</td>
<td>hypertension (120/80-200/100); ectopic ventricular contraction</td>
<td>hypertension for 1 hr; arrhythmia for 2 hrs</td>
</tr>
<tr>
<td>Nagashima, et al. (1976)</td>
<td>74 F</td>
<td>idiopathic GPN</td>
<td>lt. 9th uppermost rootlet of vagus</td>
<td>lt. 9th section, no change; touching vagus, hypotension; cutting rootlet, arrhythmia</td>
<td>hypotension for 20 min; arrhythmia for 4 days</td>
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</tbody>
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* GPN = glossopharyngeal neuralgia.

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