Effect of Bilateral Glossopharyngeal Nerve Section on Blood Pressure
A Case Report

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In 1927 Hering denervated the carotid sinus and discovered the baroceptive function of the glossopharyngeal nerve. He then combined denervation of the carotid sinus with section of the aortic nerves in the rabbit to produce neurogenic hypertension. Since then many attempts have been made to implicate altered activity of the pressor system in human hypertension. Thus far, there has been no case on record of complete denervation of all the pressor receptors in man, and only one case is reported in which the blood pressure has been observed following bilateral intracranial 9th nerve section.

Case Report

History. A 54-year-old white man was found to have a squamous cell carcinoma of the right palate in August, 1961. He was treated with radiation therapy, but in the intervening months the carcinoma extended into the right anterior tonsillar pillar, the glossopharyngeal fold, the left soft palate, and the left anterior tonsillar pillar. He was referred to Neurosurgery because of considerable pain bilaterally in the oropharynx and in the right mandibular division of the trigeminal nerve. This pain was only slightly relieved by the use of pontocaine sprays and narcotics. The tumor was of sufficient extent to require both a prophylactic tracheostomy and gastrostomy. Prior to surgery his blood pressure ranged from 110 to 136 systolic, and 70 to 94 diastolic.

Operation. On February 13, 1963, under general endotracheal anesthesia in the seated position a bilateral sub-occipital craniectomy was performed. The right 9th nerve was isolated. Electrical stimulation of this nerve produced a drop in systolic pressure from 120 to 110. The right 9th nerve was then sectioned between silver clips, and the pressure rose to 150 systolic. After the right 5th nerve was also sectioned, the left 9th nerve was isolated, but stimulation produced a drop of only a few millimeters in pressure. The left 9th nerve was then sectioned between silver clips and the pressure rose immediately to 180 systolic. During closure the pressure gradually drifted downward to a slightly hypotensive level and necessitated the use of a small amount of Neosynephrine intravenously.

Post-operative course. Upon arrival in the recovery room the patient's blood pressure began to increase and when it reached 230/120 Arfonad was administered intravenously. To avoid fatal acute hypertension an attempt was made to keep the systolic pressure below 220 by titration with intravenous Arfonad. This proved impractical as wide swings in the blood pressure occurred from minute to minute with a few drops of the Arfonad solution. The Arfonad was discontinued 4 hours after operation, and the patient was given 2.5 mg. of Reserpine intramuscularly. Eight hours post-operatively the blood pressure still ranged between 200 and 190 systolic; therefore, another 5 mg. of Reserpine was given intramuscularly. Sixteen hours post-operatively a final 5 mg. of Reserpine was given. From that time on, no further anti-hypertensive agents were administered. See Fig. 1.

The blood pressure fluctuated widely for several weeks, but never rose above 170. At first the blood pressure dropped while the patient was seated or standing, but these fluctuations gradually became less pronounced, and he stabilized at his pre-operative level. Ten months later his blood pressure still remained stable and averaged 110/70.

The pulse rate fluctuated in direct proportion to the blood pressure, and gradually stabilized at his pre-operative level 2 weeks after operation. The post-operative course was otherwise uneventful and he remained free of pain for 4 months. He then developed pain along the left trigeminal distribution. His condition at that time precluded any further surgical intervention, and he was treated with narcotics until his death in February, 1964.

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Fig. 1. Graph of blood pressure before and after bilateral ninth nerve section. Arfonad and Reserpine were administered during the time indicated by the shaded zone.
Discussion

Hering’s observations started a whole new train of investigation. Koch and Mies were among the first to describe the hypertensive effect of sectioning the moderator nerves (carotid sinus plus cardio-aortic nerves). The resulting chronic neurogenic hypertension was also confirmed by Heymans, Nowak, and Thomas. Extensive work with animals led Heymans to believe that altered resistance to stretch of the pressor receptor walls might be the primary mechanism of essential hypertension. Volhard also suggested that decreased elasticity of the arteries due to old age or wear and tear might cause a decrease in depressor tone and hypertension. This process would then further aggravate itself until it was joined by nephrosclerosis. Wakerlin helped substantiate this idea when he produced an elevation in blood pressure in dogs after giving external support to the carotid sinus by means of a plastic mold. Dammin then demonstrated the development and progression of nephrosclerosis in dogs 2 to 3½ years following bilateral section of the moderator nerve.

Transient elevation in blood pressure following unilateral intracranial section of the glossopharyngeal nerve was first reported in 1935, and has since been reported by other observers. Wyeis reported the first case of bilateral intracranial 9th nerve section in 1945. This was performed for pain secondary to malignancy, and was attended by an immediate rise in blood pressure which persisted for 4 weeks before it fell. Five days later the patient died, so the fall in pressure may well have been part of the terminal phenomenon. Nevertheless, this has been cited as an example of chronic neurogenic hypertension in man.

Although there have been no other reported cases of bilateral section of the 9th nerve, Ballantine did perform a staged, bilateral intracranial section of the 9th nerve which was attended by transient hypertension which subsided to normal levels within 9 hours. Following nerve section, the blood pressure rose from a resting level of 140/90 to 200/120, and then gradually fell to the resting level. There was no permanent change in the heart rate. The literature also contains numerous accounts of bilateral carotid sinus denervation for syncope, epilepsy, carotid sinus sensitivity, and myasthenia gravis. In these cases there is recorded either no serious after-effects, or only a mild transient elevation of the blood pressure. A case of transient hypertension secondary to polyneuritis in porphyria in which there were symptoms of partial glossopharyngeal and vagal nerve paralysis has also been reported.

These findings are in keeping with our observations, and seem to imply that the proof of a definite causal relationship between hypertension and activity of the pressor system may require complete denervation of all the pressor receptors in man.

Summary

A case of bilateral intracranial glossopharyngeal nerve section is reported. There was an immediate but transient elevation of the blood pressure with restoration of the pre-operative level and stability within 2 weeks.

References

2. BALLANTINE, H. T. Personal communication.


