Sustained hypertension after section of the glossopharyngeal nerve

Sir,—The association of inflammatory lesions of the ninth cranial nerves with disorders in the vegetative nervous system is infrequent. The few publications on the subject agree in associating neuralgia of the glossopharyngeal with vagal irritative phenomena. Unilateral intracranial section of the glossopharyngeal nerve may produce the opposite clinical pattern with sinus tachycardia and transitory arterial hypertension. We have found only one case in which arterial hypertension was not transitory, but was maintained for a period longer than 10 days. We report a similar case, in which arterial hypertension was sustained for a period of four months.

The patient was a 69-year-old woman, with no history of arterial hypertension or cardiac disease. She had a history at the age of 67 years only of biliary dyspepsia and pneumonia, which resolved satisfactorily. Her blood pressure had always been approximately 120/60 mmHg. Six years ago, glossopharyngeal neuralgia occurred which improved with treatment with carbamazepine. Two years ago, pharyngeal biopsy proved normal. Six months ago there was considerable increase of painful attacks. Intracranial section of the ninth cranial nerve was performed. Computed tomography before operation was normal. Immediately after operation there was arterial hypertension (210/120 mmHg) with sinus tachycardia (120 min). Central venous pressure was normal. After first controlling arterial pressure with intravenous sodium nitroprusside, the patient was given oral propranolol in doses of 60 mg/day. Later it was found that when the drug dosage was reduced arterial pressure rose, with the result that it proved impossible to reduce the propranolol without hypertension appearing until four months after operation; arterial pressure after that time was 120/60 mmHg without medication.

Normally the contralateral glossopharyngeal nerve rapidly counteracts sympathetic vegetative effects, which may include arterial hypertension through increase of cardiac output or alteration of the angiotensin system. In our case, after unilateral intracranial section of the glossopharyngeal nerve, arterial hypertension lasted for four months, which leads us to suppose vegetative compensation was slow. Propranolol was effective, which supports the hypothesis that the arterial hypertension is caused by the action of the beta adrenergic system on cardiac output. We believe it will be of interest, therefore, to measure the cardiac output in other patients who develop arterial hypertension following intracranial section of the glossopharyngeal nerve.

References


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