mechanism of the condition. There is some agreement that the cartilage originates from the intervertebral disc and various possible mechanisms have been discussed.¹⁻³ A sudden increase in disc pressure, leading to injection of disc material into small vessels has been favoured, but the exact nature is not known.

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Accepted 11 May 1989

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Trigeminal neuralgia associated with contralateral intracranial tumour: a false localising sign caused by vascular compression? Report of two cases

Sir: Trigeminal disturbances clinically resembling "idiopathic" trigeminal neuralgia may rarely occur in association with contralateral intracranial tumours—both supratentorial and infratentorial. Only six cases have been reported previously.¹ We describe two further patients with details of the radiological investigation and operative findings. In one case, our study also provided some insight into the mechanism of contralateral trigeminal involvement.

Case 1: a 44 year old female with a history of headache from the age of 34 presented with a left trigeminal neuralgia (2nd and 3rd branches) of five months duration. Eating and speaking triggered paroxysms of pain and were only partially relieved by carbamazepine. On admission, neurological

Fig (a) Case 1—CT scan. Huge meningioma of the right posterior fossa attached to retromental portion of the petrous bone and to the tentorium. (b) Case 2—CT scan. Large meningioma of the left petro-clival area.

examination disclosed a mild cerebellar ataxia. A computed tomographic (CT) scan with contrast enhancement showed a large high-density mass on the right posterior fossa, displacing the brainstem contralaterally (fig (a)). Right vertebral angiography showed a very mild contralateral shift of the basilar artery. A right suboccipital craniectomy was carried out and a huge meningioma was excised which was attached to the tentorium and right petrous temporal bone. The postoperative course was uneventful. The trigeminal neuralgia progressively disappeared in the course of five months and treatment with carbamazepine was discontinued. No recurrence of trigeminal neuralgia occurred over a follow-up period of nearly two years.

Case 2: a 55 year old male presented with a right trigeminal neuralgia (3rd branch) of six months duration. He experienced recurrent, excruciating, stabbing pains, triggered by eating, speaking and light touch of the sensitive area. The patient obtained considerable relief in the first two months from carbamazepine; later, the pain became increasingly severe and frequent. Three months before admission, he complained of left facial twitching. Neurological examination revealed a left hemifacial spasm. A CT scan showed a large left petro-clival meningioma (fig (b)). A left vertebral angiography showed considerable contralateral displacement of the basilar artery. During surgery and after total removal of the tumour, it was evident that the basilar artery was displaced against the root entry zone of

the contralateral (right) fifth cranial nerve. Immediately after the operation, the patient had left abducent nerve palsy and moderate left facial paresis. These symptoms improved over a six month period but were still present three years later. His trigeminal neuralgia gradually attenuated and disappeared in the course of one year. Treatment with carbamazepine was discontinued. No recurrence of trigeminal neuralgia was reported over a three year follow up period.

The clinical syndrome of contralateral trigeminal nerve dysfunction as a false localising sign in intracranial tumours is known to occur, though rarely.^{1,2} In most cases the tumour responsible is a meningioma, usually infratentorial.¹ Patients with trigeminal false localising symptoms and signs can best be separated into two groups: trigeminal neuralgia (six reported cases) and those with other sensory function disturbances of the fifth cranial nerve (26 reported cases).^{1,2} Our two patients who experienced a typical trigeminal neuralgia contralateral to an infratentorial meningioma were included in the first group.

Some explanations have been put forward to account for the contralateral trigeminal neuralgia.^{1,2} Nevertheless, the mechanism of trigeminal involvement remains speculative since direct observation of the affected trigeminal nerve is rarely possible during surgery to remove the neoplasm. In case 2 of our series, however, the basilar artery was shown to be displaced by the tumour against the opposite trigeminal nerve.

Observations during surgery as well as at

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autopsy have provided extensive evidence, in the majority of cases, that "idiopathic" trigeminal neuralgia and hemifacial spasm are caused by vascular cross compression of the root entry zone of the fifth and seventh cranial nerves, respectively.³ Indeed, in case 2, direct surgical observation showed that the basilar artery impinged on the fifth nerve's root entry zone, in exactly the same way as that observed during the operation by one of us (FC) in cases of "idiopathic" trigeminal neuralgia.⁴ Recently, one patient with hemifacial spasm due to a contralateral infratentorial tumour has been reported:⁵ in this case the angiography suggested that vascular compression was responsible for facial nerve involvement.

Our data, though not entirely conclusive, suggest that trigeminal neuralgia occurring in patients with contralateral tumours may be related to displacement of one or more vessels, causing stretching and pulsatile com-

pression of the opposite fifth nerve's root entry zone in the posterior fossa.

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Accepted 17 April 1989