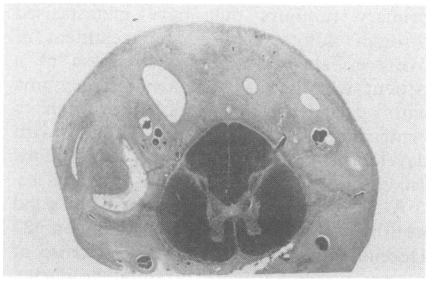

Fig A

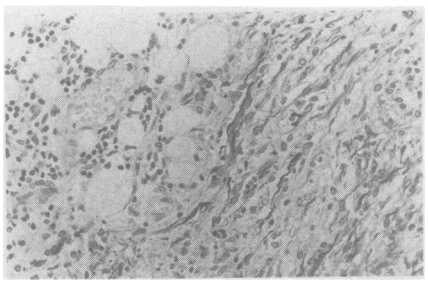

Fig B

Figure (A) Involvement of arachnoid of spinal cord sparing spinal cord tissue (Woelke). (B) Surface of peritoneum infiltrated by a tumour mass composed by spindle-shaped cells lying near the mesenteric adipose tissue. There is a fibrous reaction. The cells are positive for the PAP method for GFAP demonstrating their astrocytic origin.

Table Cases reported of extraneural metastases from CNS tumours through shunt systems. Histological and shunt system types

Primary tumour	*VP*	*VA*	*VPL*	*LP*	*VV*	*Total*
Medulloblastoma (1)	9	5		1	1	15
Germinoma	8	1				9
Glioblastoma (2)	4	1	2			6
Astrocytoma	1*	1	1			3
Astrocytoma-oligodendroglioma		1				1
Dysgerminoma	1					1
Optic nerve glioma	1					1
Gliomatosis cerebri				1		1
Ependymoma	1					1
Ependymoblastoma	1					1
Sarcoma	1					1
Pinealoblastoma	1					1
Chorinepithelioma		1				1
Atypical teratoma	1					1
Pineal unclassified tumour	1					1
Total	30	10	3	2	1	44

VP = ventriculoperitoneal; VA = ventriculoatrial; VPL = ventriculopleural; LP = lumboperitoneal; VV = ventriculo-venous.
(1) 1 patient had VP and VA shunts.
(2) 1 patient had VA and LP shunts.
*Case reported.

spinal cord and peritoneal metastases (fig B).

Extracranial metastases of CNS tumours are infrequent. In a recent review of the literature there was a total of 282 cases.[2] The placement of shunt systems to relieve hydrocephalus seems to be an important factor in metastasis, and some authors have proposed the systematic use of millipore filters in VPS to avoid tumour cells seeding through the shunt systems to the peritoneum.[3] The table shows the histological origin and shunt type of previously reported CNS primary tumours with extraneural metastases through shunt systems. Medulloblastoma is the most frequent histological type reported while astrocytoma was reported in three previous cases.[4-6] Nevertheless, our patient is the first to have metastases through VPS. Ascites as a complication of VPS is also infrequent. Our case is the tenth to be reported with metastatic ascites associated with VPS placement, and the first one due to astrocytoma. These features, together with the presence of spinal cord seeding, makes our case exceptional and to our knowledge not previously reported in the literature.

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Isolated hypoglossal nerve palsy and Horner's syndrome with benign course

The hypoglossal nerve may be affected by various diseases, sometimes together with other lower cranial nerves and sympathetic fibres as in Villaret's syndrome.[1] We describe a 54 year old woman with isolated hypoglossal nerve palsy and Horner's syndrome which disappeared within several months. We could find no similar case reports.

On 23 May 1988, our patient developed tinnitus and a narrowed palpebral fissure on the left side without any preceding episodes. Two hours later she had a severe throbbing headache in the left parieto-occipital region, and vomited twice. There was no dizziness, watering of the eyes, nasal stiffness or any sensory abnormalities. The next day physical examination was normal except for hypertension and left Horner's syndrome. Within two days the severe headaches subsided. On 26 May, she noticed difficulty in speaking and in tongue movements, and was referred to the Department of Neurosurgery. Left hypoglossal nerve palsy was revealed. Left vertebral angiograms, CT scan and MRI of the brain and upper cervical region were normal, as were the otological examinations. She was referred to the Department of Neurology of Tohoku University on 14 June. She had no personal or family history of recurrent headaches.

She had no lymphadenopathy or organomegaly. The diameter of the right pupil was 3·0 mm, and the left, 2·5 mm. Pupillary reactions were normal. The palpebral fissures were symmetric. The tongue curved to the right at rest in the mouth. Its left side was atonic with occasional fasciculation, and appeared larger than the right side. However, left-sided palsy was evident when the tongue was protruded (fig top). Other examinations were non-contributory. An hour after two drops of 1·25% epinephrine, the left pupil showed supersensitive dilatation up to 7·0 mm, while the right remained unchanged. Thermal sweating was absent on the left medial part of the forehead. This distribution of anhidrotic area indicated a sudomotor sympathetic lesion distal to the bifurcation of the common carotid artery.[2] Routine laboratory data and cerebrospinal fluid were normal. VDRL was negative. Serum antibody-titres against herpes simplex, varicella-zoster, Epstein-Barr (EB) and cytomegalovirus were examined on 14 and 28 June. IgG against cytomegalovirus was 1:11264 on both occasions. No significant increase or temporal changes in other antiviral titres were detected. Her symptoms regressed without specific treatment. On 11 July, the pupils were equal and no longer reacted to 1·25% epinephrine. The tongue was slightly atrophic on the left side, but its movements were normal (fig bottom). Carotid arteriography and jugular venography were not performed because of a spontaneous recovery. During the subsequent 19 months, she had no recurrence of headaches or any other neurological symptoms. Follow up CT and MRI were unremarkable. On the last examination in February 1990, the appearance and movements of the tongue were entirely normal.

Our patient had left hypoglossal nerve palsy and ipsilateral Horner's syndrome which was shown to be postganglionic by the supersensitivity to 1·25% epinephrine and anhidrosis over the left medial part of the forehead. The association suggests that the causative lesion must be in the left retroparotid space. Clinical and radiological examinations rule out mass lesions. Their acute onset with hemicrania, and subsequent regression suggest focal inflammation or vascular disorders. DeSimone and Snyder[3] reported hypoglossal nerve palsy with good

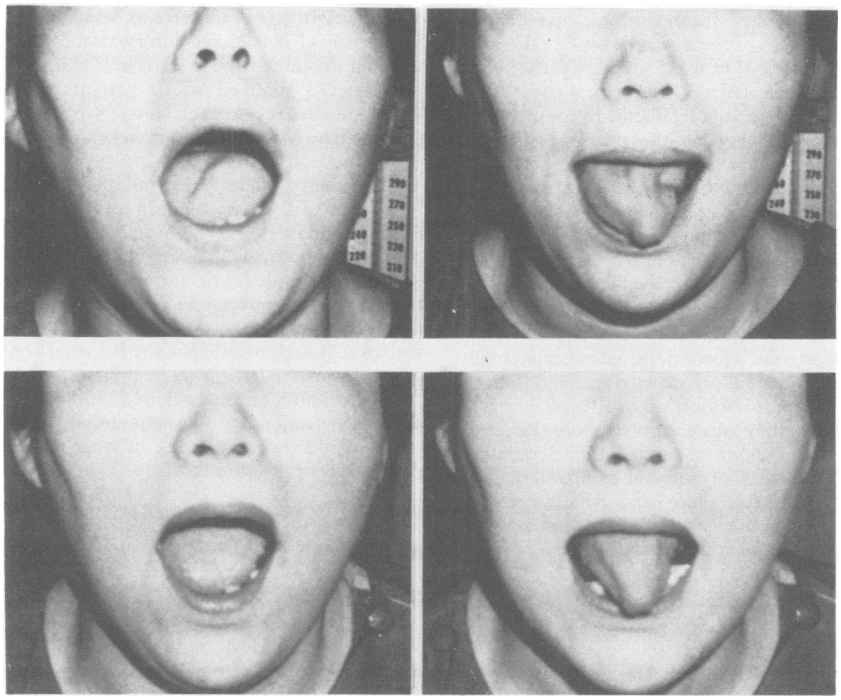

Figure Top: Nineteen days after the onset of the left hypoglossal nerve palsy, the tongue at rest curves to the right side, and its left half appears larger. But the left-sided palsy is evident on protrusion. Bottom: Four weeks later, the appearance and movements are almost normal.

recovery in a patient with infectious mononucleosis. But our patient did not have fever, lymphadenopathy nor other clinical features of general inflammation. Serum antibody titres against EB virus showed no significant alteration, though we did not examine them during the acute stage. In migraine and cluster headache, Horner's syndrome and cranial nerve palsy may be seen,[4] but hypoglossal nerve palsy has not been reported. Moreover, our patient had no severe headaches before and after the present episode.

The larger appearance of the paralysed left side at rest on initial observation deserves some comment. According to Gowers,[5] in paralysis of one hypoglossal nerve the tongue at rest is in its normal position in the mouth, but its root is higher on the paralysed than on the unparalysed side, in consequence of the loss of the tonic contraction of the posterior fibres of the hyoglossus. Adams and Victor[6] described how the tongue curves slightly to the healthy side as it lies in the mouth. This may be due to paralysis of the superior and inferior longitudinal muscles which normally shorten the tongue. In addition, paralysis of other intrinsic muscles, especially transverse and vertical lingual muscles, may cause enlargement of the tongue. These may be reasons why, in the early stage of unilateral hypoglossal nerve palsy, the paralysed side appears larger than the intact side.

Although we could not establish the cause of our patient's disease we consider that isolated hypoglossal nerve palsy with Horner's syndrome is worth noting.

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MATTERS ARISING

Whiplash injury

Several of the conclusions of Dr Pearce about whiplash injury deserve comment.[1] His first conclusion that there is no adequate published control group occurring outside the context of a compensation claim is incorrect.[2] Hohl, a private orthopaedist, found that 44 out of a total of 146 patients reviewed five years after whiplash injuries had no possible recourse to litigation, because either they were at fault or there was no collectable compensation: 50% of this group were still symptomatic at this time.

We disagree with Dr Pearce's conclusion that there is a striking resemblance to the natural history of whiplash and other "strains" elsewhere. Undoubtedly if one takes a consecutive group of patients with whiplash one will find that a large number will recover quickly. This suggests that many of these patients have had a very minor injury. Notwithstanding this there are a considerable number of whiplash patients who continue to have persistent symptoms, not only for longer periods than it should take for ligamentous strains to recover (approximately 12 weeks) but for periods long after compensation cases have been settled.

Gotten[3] found 12% of patients two years after settlement still had severe symptoms, McNab[4] found 45% of patients still had some neck symptoms two years after settlement, while Hohl[2] found 43% of patients had some neck pain five years after their injuries were sustained. These reports suggest that the pathology involved is different from a simple soft tissue injury. While the persistence of symptoms in some whiplash patients could be related to a slower capacity to regenerate damaged tissues, the return to normal painless function for associated ankle or wrist soft tissue damage within the expected time period suggests that different pathomechanisms are involved.[4]

Using brave volunteers, Severy has shown that in a 10 mph rear-end collision a force of 9 g is generated at the neck and that this is considerably greater over the frontal cortex (23 g).[5] Damage in these areas would not lead to isolated defects as paralysis of a muscle group but rather to changes in mood and cognitive ability.

Yarnell[6] has recently described poor performance in neurophysiological evaluations of cognitive function, in a group of patients with major debility after a minor head injury associated with a whiplash injury. It is likely that less severe forms of dysfunction are evident in some patients with persisting symptoms. The cervical lesion that causes local persistent symptoms may not be a soft tissue injury at all, notwithstanding the fact that soft tissue injuries occur at the same time. Rare cases of bilateral sympathetic dysfunction[7] post-whiplash suggest, as initially proposed by Barre and Lieou,[8] that disorganised functioning of the cervical sympathetic system is important in facilitating the persistence of neck pain in some patients.

A study in which acute whiplash cases are analysed by MRI may help resolve what type of neck soft tissue injuries occur and whether or not they are prognostically important. We have recently reported a case of post-whiplash dystonia well controlled by TENS[9] who despite marked limitation of neck movement three weeks after the injury had normal MRI of his neck and brain suggesting that the pathology may be very subtle.

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