a rather simplistic way clinicians have assumed that neck flexion will cause mechanical stresses only to the cervical cord. Movement of the cord relative to the thoracic spine may also occur during neck flexion, and could be a mechanism of this woman's symptomatology. For this reason it is important that myelography include examination of the entire spinal cord in patients describing this symptom, in whom electric shock sensations radiate only to the legs.

We thank Mr RV Jeffreys and Dr P Buxton for surgical and pathological details respectively of this case.

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A tonic pupil with Horner's syndrome

Sir: The conjunction of a Horner's syndrome and features of a tonic pupil with an apical carcinoma in the superior pulmonary sulcus and paravertebral gutter produces unusual and misleading physical signs. We report such a case.

A 71-year-old female patient presented with a 2 year history of progressive tingling and numbness over the ulnar aspect of her left forearm and hand associated with shooting pains triggered by touch. Over a two month period she had noticed increasing pain, weakness and muscle wasting of her left hand with intermittent blurring of vision in her left eye. There was no past history of visual disturbance or neurological disorder.

On the left she had moderate ptosis, a pupil which was slightly larger than the right in daylight, and conjunctival injection. The left pupil was smaller than the right when examined in dim light. Infrared television pupillography[1] revealed an abnormally small resting darkness diameter of 3·7 mm (right eye: 5·2 mm) with almost no response to light (<0·2 mm constriction); accommodative effort to near vision resulted in a 0·9 mm constriction which was abnormally slow in both onset and offset, characteristic of a tonic pupil. Slit lamp examination revealed slight segmental movement in the upper part of the left iris in response to near accommodation. The right pupil was normal for age in all respects. Ocular movements were full and other cranial nerves were intact. In her left upper limb the skin was dry and there was wasting of forearm and hand muscles with weakness of triceps, finger extension, wrist and finger flexion and all small muscles of her hand. The triceps jerk was absent. Sensation was impaired over the C7, C8 and T1 dermatomes. No motor, sensory or reflex abnormality was found in other parts of the body. There was fullness in her left supraclavicular fossa although no mass was palpable.

Segmental electomyography demonstrated denervation of C7, C8 and T1-innervated muscles. Nerve conduction studies showed an absent left ulnar sensory action potential and a median sensory action potential of 15 μv. There was an absent flare response following intradermal injection of 0.016 ml histamine acid phosphate 1 mg/ml to the inner aspect of her left forearm; the flare was preserved on the right. Sweat testing with quinizarine powder was inconclusive. Radiographs of her cervical spine showed degenerative change but other radiological investigations, including chest radiography, AP tomography of the mediastinum and cervical spine, cervical myelogram and CT scan of neck, were all negative. Examination of the cerebrospinal fluid was normal and syphilis serology was negative.

Responses of both pupils to topical drug applications were tested on four occasions each separated by at least 3 days. The findings were:

Drug	*Diameter change (mm)*	
	Right pupil	*Left pupil*
Phenylephrine 2%	2·50	3·40
Hydroxyamphetamine 0·5%	2·00	1·82
Cocaine 4%	0·90	0·08
Pilocarpine 0·05%	0·75	−0·93

The affected pupil was moderately supersensitive to the direct-acting sympathomimetic phenylephrine, dilated normally to hydroxyamphetamine, which causes noradrenaline release from the sympathetic nerve teminal, and was unresponsive to cocaine, which blocks noradrenaline re-uptake. These findings are consistent with a pre-ganglionic sympathetic nerve lesion. The affected pupil was also supersensitive to the constrictor action of the direct-acting cholinomimetic pilocarpine, which is indicative of a parasympathetic nerve lesion.

Exploration (Mr K Burnand) revealed extensive tumour in the left para-vertebral gutter; the T1 root was oedematous and ran through the tumour mass. Biopsy of the tumour showed anaplastic carcinoma.

The left pre-ganglionic sympathetic lesion was caused by tumour infiltration in the region of the T1 root.[2 3] The ocular features were unusual for Horner's syndrome in a number of respects, namely that the pupil was slightly dilated when examined in normal room lighting, that there was a minimal light reflex and that accommodative effort produced a large but very slow response. In view of the segmental iris movement and supersensitivity to dilute pilocarpine, it seems likely that these atypical features were due to a concomitant pre-existing post-ganglionic parasympathetic lesion such as is seen in the Holmes-Adie pupil. There was no evidence of local infiltration by tumour of the ciliary ganglion, meninges or central nervous system. It was conceivable that the drug responses in this patient were influenced by a change in corneal permeability but we know of no evidence that this was the case.

Patients with Horner's syndrome associated either with ipsilateral accommodative paresis or with other disorders of accommodation have been described in the past.[4] However, this unusual conjunction of sympathetic and parasympathetic lesions has not, to our knowledge, been previously reported and it exemplifies the value of pupillography and pharmacological testing in the diagnosis of pupillary dysfunction.

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Iatrogenic internuclear ophthalmoplegia

Sir: Unilateral lesions of the medial longitudinal fasciculus, clinically manifest as internuclear ophthalmoplegia, are usually vascular in origin. Smith and Cogan[1] in a series of 29 patients, with unilateral internuclear ophthalmoplegia, attributed the condition to a vascular cause in 67% of cases. Despite the high incidence of a vascular aetiology, only one previous case of internuclear ophthalmoplegia following iatrogenic embolisation of the vertebrobasilar system has been reported.[2] We report a case of unilateral internuclear ophthalmoplegia following cardiac catheterisation.

A 15-year old male patient underwent cardiac catheterisation for the investigation of a suspected ventricular septal defect. The procedure was performed under local anaesthesia by percutaneous puncture of the right femoral vein. The foramen ovale was patent, facilitating the passage of the catheter into the left atrium. The catheter was then advanced into the left ventricle via the mitral valve. Left ventricular angiography was performed by the injection of 60 mls of "Hexabrix 320"* into the ventricle. *(Hexabrix 320 is an ionised, iodinated contrast agent, being a serile solution of meylamine ioxaglate 39·3% w/v and sodium ioxaglate 19·65% w/v containing 320 mg iodine in combined form per ml.) Ventricular septum profiles demonstrated a small perimembranous ventricular septal defect.

The patient reported no side effects during, or immediately after the investigation. However, the following day the patient complained of horizontal diplopia. This improved gradually after the next few days. He was examined in the ophthalmology department four days later, where he was found to have a horizontal diplopia, manifest on dextroversion. Further examination of his ocular movements revealed an underaction and updrift of the left eye on adduction, and nystagmus of the right eye on abduction. A pronounced slowing of the saccadic velocity in the left eye on dextroversion was also noted. The rest of the examination including visual acuity, pupil reactions and general neurological assessment was normal. The patient was examined one month later; he was now asymptomatic, the eye movements having returned to normal except for a minimal underaction of the left eye on adduction.

Disorders of ocular motility represent a rare complication of cardiac catheterisation. Indeed, Hildner *et al*[3] in a review of the complications in 600 adult patients who underwent transbrachial left heart catheterisation, failed to record any ocular motility problems. Thomas *et al*[4] have reported a case of a partial third nerve palsy in a 42-year-old male following retrograde cardiac catheterisation.

Unilateral internuclear ophthalmoplegia is most commonly associated with brainstem infarction.[1] Less common causes include demyelination, diabetes, systemic lupus erythematosis, Wernicke's syndrome, encephalitis, brainstem tumours,[5] trauma[6] and phenothiozine intoxication.[7] Only one previous case of iatrogenic embolisation as a cause of internuclear ophthalmoplegia has been reported. This report[2] described the sudden onset of unilateral ophthalmoplegia following carotid angiography for the investigation of a parasellar lesion in a 27-year-old woman. A persistent primative trigeminal artery connecting the carotid and basilar systems was present, the posterior communicating arteries being absent. In common with the patient reported here, a full recovery occurred within one month.

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The predictive value of 5 days CSF diversion for shunting in normal pressure hydrocephalus

Sir: The literature gives many different criteria for selection of patients with normal pressure hydrocephalus who might benefit from shunting. Apart from clinical criteria and the CT scan findings,[1 2] the most reliable predictive tests are: 24-hour intracranial pressure monitoring,[3 4] infusion test,[5] isotope cisternography,[6–8] and CSF outflow conductance measurement.[9] However, occasionally there are doubtful[9 10] or variant[11] cases, where the only test is to observe the postoperative neurological status. From the observation that any improvement of the clinical picture is evident during the first postoperative days,[1–12] we propose a simple, but invasive predictive test. CSF drawings for five consecutive days, with the opportunity to regulate the quantity of CSF removed every 24 hours, may predict with considerable precision whether the patient will improve with later permanent shunt. A similar test was introduced 3 years ago by Wikkels *et al*[12] who evaluated their patient's psycometric and motor capacities before and after lumbar puncture and removal of 40–50 ml of CSF. They found a close correlation between improvement after lumbar drainage, which started after 30–60 minutes and lasted several hours, and improvement after a permanent shunt.

Twenty four hours after lumbar pressure monitoring, we connected a lumbar catheter (Thuoy needle No 16) to a drainage bag hanging at the patient's side so that he