References

- ¹ Lowenstein O, Loewenfeld IE. The Pupil. In: Dawson H, ed. *The Eye*. New York & London: Academic Press, 1969;vol 3:255-337.
- ² Pancoast HK. Superior pulmonary sulcus tumour: Tumour characterised by pain, Horner's syndrome, destruction of bone and atrophy of hand muscles. *JAMA* 1932; **99**:1391-6.
- ³ Paulson DL. Carcinomas in the superior pulmonary sulcus. *J Thorac Cardiovasc Surg* 1975;**70**:1095-104.
- ⁴ Weisfeld L, Streeten BW. Horner's syndrome with ipsilateral accommodative paresis. *Am J Ophthalmol* 1978;**86**:114-7.

Iatrogenic internuclear ophthalmoplegia

Sir: Unilateral lesions of the medial longitudinal fasciculus, clinically manifest as internuclear ophthalmoplegia, are usually vascular in origin. Smith and Cogan¹ 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 vertebro-basilar 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 60mls of "Hexabrix 320"* into the ventricle. *(Hexabrix 320 is an ionised, iodinated contrast agent, being a sterile 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 erythematosus, Wernicke's syndrome, encephalitis, brainstem tumours,⁵ trauma⁶ and phenothiazine intoxication.⁷ Only one previous case of iatrogenic embolisation as a cause of internuclear ophthalmoplegia has been reported. This report² 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 primitive 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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References

- ¹ Smith JL, Cogan DG. Internuclear ophthalmoplegia—a review of 58 cases. *Arch Ophthalmol* 1959;**61**:687-94.
- ² Cahill DW, Saleman M, Hirsch D, Rao CVGK. Unilateral internuclear ophthalmoplegia due to angiographic embolism through a prima-

tive trigeminal artery. *Neurology (NY)* 1981;**31**:751-3.

³ Hildner FJ, Drake EM, Gale HH, Ormond RS. Transbrachial retrograde left heart catheterisation. Evaluation of 600 consecutive cases in adults. *Am J Cardiology* 1966;**18**: 52-6.

⁴ Thomas C, Reny A, Raspillar A. Paralysis oculaires complications due catheterisms arteriel. *Bull Soc Ophthalmol Fr* 1969;**69**: 700-1.

⁵ Cogan DG, Wray SH. Internuclear ophthalmoplegia as an early sign of brainstem tumours. *Neurology (Minneapolis)* 1970;**20**(7):629-33.

⁶ Devereaux MW, Brust JC, Keane JR. Internuclear ophthalmoplegia caused by subdural haematoma. *Neurology (Minneapolis)* 1979;**29**: 251-5.

⁷ Cooke FF, Davis R, Russo LS. Internuclear ophthalmoplegia caused by phenothiazine intoxication. *Arch Neurol* 1981;**38**:465-6.

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 variant¹¹ 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 psychometric 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

could walk normally or remain sitting. A flow-control-extension-set (DIAL-A-FLOW, Abbott Laboratories, Chicago, USA), similar to that for intravenous infusion, enabled us to regulate the hourly amount of CSF removed with considerable precision. As the system used for intravenous infusion is set at 76 cm H₂O, we calculate the flow rate of DIAL-A-FLOW by inserting a No 16 Tuohy catheter into a bottle of Ringer lactate solution held at height of 40 cm H₂O; the correction values are as in the table.

The pressure of 40 cm H₂O was chosen as the average lumbar CSF pressure for patients that could walk or stand up. During the night and with bedridden patients, the Flow-Control set was held 20–30 cm below the level of the lumbar catheter. In this way, every 24 hours we could safely, slowly and continuously remove a given amount of CSF, which differed from one patient to another, according to the neurological status and ventricular sign, or even the depth of cerebral sulci, observed on the CT scan.

This method was used in 10 patients with CT scan findings of normal pressure hydrocephalus of various aetiologies. Clinically they had severe dementia and severe gait disorder; five of them were bedridden. The duration of symptoms varied from 3 to 7 months. The daily CSF drainage varied from 130 to 200 ml. Clinical evaluation was assessed at the fifth day; improvement was rated as poor, good or excellent taking into account impressions of the family and medical and nursing staff; psychometric tests were found to be useless. The first and most important sign of reversing of the dementia was reduction of hypokinesia with improvement of facial expression. No complication was seen (such as infection, headache, subdural collection). Two patients who did not improve with daily removal of 200 ml of CSF had a diagnosis of degenerative dementia and were not operated upon. The other patients, whose improvement was rated good or excellent, underwent a ventriculoperitoneal shunt operation with a medium-pressure valve. All the patients operated upon continued to do well in the follow-up.

The mechanism by which the shunt can reverse the syndrome of normal pressure hydrocephalus is poorly understood. Some patients responsive to shunting may have no change of ventricular size on CT scan, and patients unresponsive to shunting can show reduction of hydrocephalus.¹³ Borgesen and Gjerris⁹ state that there are two possible actions of the shunt: (a) diversion and removal from the ventricles of components in CSF which can play a role in the production of symptoms; (b) modification of intracranial pressure. In the pathogenesis of normal pressure hydrocephalus the following aspects must be considered: (1) Improvement after shunt is followed by an increase in cerebral blood flow as demonstrated by Tamaki, *et al.*¹⁴ the same authors gave evidence of deterioration of cerebral haemodynamics when the shunt procedure was performed in cases of degenerative dementia; (2) The pathophysiology of all cases of normal pressure hydrocephalus of various aetiologic is a deficit of CSF absorption as demonstrated by means of infusion tests⁵ or the calculation of conductance to CSF outflow.⁹

Once the chronic ventricular enlargement of normal pressure hydrocephalus, whatever the aetiology, has been established, CSF production, which is normal or near normal in normal pressure hydrocephalus¹⁶ must equal CSF absorption which is 500 ml/24 hours.¹⁵ As the CSF is normally renewed every 7 hours, the CSF in normal pressure hydrocephalus, which is several times larger in volume than normal, can only be renewed every 24 hours or more at a formation-absorption rate of 500 ml/24 hours. This very slow CSF flow may alter CSF excretory function¹⁷ to cause accumulate unknown metabolic substances which could play an important role in the production of symptoms in NPH. The increase in cerebral blood flow in the cases responsive to the shunt might be secondary to improvement in neuronal metabolism.

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References

- Fischer CA. The clinical picture in occult hydrocephalus. *Clin Neurosurg* 1977;24:270–84.
- Vassilouthis J. The syndrome of normal pressure hydrocephalus. *J Neurosurg* 1984;61:501–9.
- Gjerris F, Borgesen SE, Soresen SC. Predicting the results of ventricular shunting in normal pressure hydrocephalus, by lumboventricular perfusion test. *Intracranial Pressure IV*, Berlin: Springer-Verlag, 1980:594–97.
- Pickard J, Teasdale G, Lindsay K, *et al.* Intraventricular pressure waves. The best predictive test for shunting in normal pressure hydrocephalus. *Intracranial Pressure IV*, Berlin: Springer-Verlag 1980:498–500.
- Katzman R, Hussey F. A simple constant-infusion manometric test for measurement of CSF absorption. I Rationale and Method. *Neurology* 1970;20:534–44.
- Benson TF, Lemay M, Pattern DH, *et al.* Diagnosis of Normal Pressure Hydrocephalus. *N Engl J Med* 1970;283:609–15.
- Laws ER Jr, Mokri B. Occult Hydrocephalus: results of shunting correlated with diagnostic tests. *Clin Neurosurg* 1977;24:316–33.
- Symon L, Hinzpeter T. The enigma of normal pressure hydrocephalus: tests to select patients for surgery and to predict shunt function. *Clin Neurosurg* 1977;24:285–315.
- Borgesen SE, Gjerris F. The predictive value of conductance to outflow of CSF in Normal Pressure Hydrocephalus. *Brain* 1982;105:65–86.
- Ojemann RG. Normal pressure hydrocephalus. *Clin Neurosurg* 1971;18:337–70.
- Salibi NA, Lourieg GL, Lourie H. A variant of Normal Pressure Hydrocephalus simulating Pick's disease on Computerized Tomography. Report of two cases. *J Neurosurg* 1983;59:902–4.
- Wikkels C, Anderson H, Blomstrand C, *et al.* The clinical effect of lumbar puncture in Normal Pressure Hydrocephalus. *J Neurol Neurosurg Psychiatry* 1982;45:64–69.
- Shenkin HA, Greenberg JO, Grossman CB. Ventricular size after shunting for idiopathic Normal Pressure Hydrocephalus. *J Neurol Neurosurg Psychiatry* 1975;38:833–7.
- Tamaki N, Kusunoki T, Wakabayashi T, *et al.* Cerebral Hemodynamics in Normal Pressure Hydrocephalus. Evaluation by 133 Xe inhalation method and dynamic CT Scan study. *J Neurosurg* 1984;61:510–4.
- Lorenzo AV, Bresnan MJ, Barlow CF. Cerebrospinal fluid absorption deficit in Normal Pressure Hydrocephalus. *Arch Neurol* 1974;30:387–93.
- McComb JC. Recent research into the nature of cerebrospinal fluid formation and absorption. *N Neurosurg* 1983;59:369–83.
- Fisher RA. Cerebrospinal fluid in diseases of the Nervous System. WB Saunders Company, 1982:95–107.

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Flow rate at 76 cm H₂O
Standard calibration
125 ml/h
100 ml/h
50 ml/h
20 ml/h
10 ml/h

Flow rate at 40 cm H₂O
Calibration through a No 16 lumbar catheter
10 ml/h
7, 8 ml/h
5 ml/h
2 ml/h
1, 7 ml/h