Letters

Lumbar extradural arachnoid cyst presenting as a conus lesion in a man aged 77 years

Sir: Spinal arachnoid cysts are a well recognised but uncommon cause of spinal cord compression. They typically present as a slowly progressive thoracic cord lesion in a young adult.^{1,2} Extradural cysts in the thoracolumbar region are very rare.^{3,4} We report a case of extradural arachnoid cyst which presented as a very slowly progressive conus lesion in a man aged 77 years.

An active 77 year old man presented with a ten year history of intermittent pain in the right heel. Standing precipitated pain which radiated to the sole and ankle and eventually limited walking to about 50 yards. Similar but milder symptoms had been present in the left foot for about a year. There was a past history of acute lumbar backache 40 years ago and 20 years previously he had undergone a transurethral resection of a prostatic carcinoma and was maintained on stilboestrol. He had no new urinary symptoms. Neurological examination one year prior to admission was normal apart from an absent right ankle reflex.

On examination the cranial nerves and upper limbs were normal. There was no wasting in the legs but tone was increased at the right knee. The right knee jerk was pathologically brisk and the right ankle jerk was absent. The left ankle jerk was just present with reinforcement. Light touch and pain appreciation was reduced on the lateral aspect of the right foot and right lower leg. Proprioception was normal but vibration sense was absent at the right hallux. Straight leg raising was reduced to 60° on the right and 70° on the left.

Spinal radiographs showed widening of the interpedicular distance at T11-L2 with flattening of the lateral arch elements and posterior scalloping of the vertebral bodies. Myelography demonstrated forward displacement of the theca by a non opacified extradural lesion reducing the cephalad flow of contrast between T11-L2; computed tomography (CT), magnetic resonance imaging (MRI) (fig) and cyst puncture confirmed the presence of an extradural arachnoid cyst with a narrow neck at its upper margin. On 24 April 1987 he underwent thoraco-lumbar laminectomy for excision of the cyst which extended along the nerve roots between T10 and L1. His postoperative course was complicated by a brain stem stroke producing left sided facial weakness, a left sixth nerve palsy and ataxia. This

Fig MRI scan. TR 500 TE 40 spin echo sequence. Sagittal view of lower thoracic and lumbar regions demonstrating a low density expansive mass indenting the theca from behind between T11 and L2.

improved rapidly leaving him with mild facial asymmetry alone. There was considerable improvement in the right heel pain.

This case represents a very rare cause of a conus lesion and illustrates an unusual presentation of a spinal arachnoid cyst. The patient was older than patients previously described. Diagnosis was difficult because of the long history of pain, (initially suggesting a local lesion in the foot) and the lack of development of neurological signs. The severity of the pain in this case prompted further investigation, together with signs suggesting a lesion in an unusual location for degenerative disc disease. In retrospect the diagnosis of an arachnoid cyst was suggested by the intermittent nature of the symptoms⁵ and their relationship to posture.^{4,6} Pain is a more typical presentation of cysts in this area while thoracic lesions produce a slowly progressive spastic paraparesis.^{1,6}

The diagnosis in this case was established by myelography and CT. Although MRI was not performed as the primary diagnostic procedure it provided clear demonstration of the lesion in a non invasive manner.

JB WINER
AE HARDING
L SYMON
DPE KINGSLEY

The National Hospital for Nervous Diseases,
London, UK

Address for correspondence: Dr J B Winer, National Hospital for Nervous Diseases, Queen Square, London WC1N 3BG, UK

References

- Shaw CM, Alvord Jr EC. Congenital arachnoid cysts and their differential diagnosis. In: Vinken PJ and Bruyn GW, eds. *Handbook of Clinical Neurology*. Amsterdam: Elsevier 1977;31:75-136.
- Kendall BE, Valentine AR, Keis B. Spinal arachnoid cysts: clinical and radiological correlation with prognosis. *Neuroradiology* 1982;22:225-34.
- Gortvai P. Extradural cysts of the spinal canal. *J Neurol Neurosurg Psychiatry* 1963;26:223-30.
- Glasauer FE. Lumbar extradural cyst. *Neurosurg* 1966;25:567-70.
- Palmer JJ. Spinal arachnoid cysts. Report of six cases. *J Neurosurg* 1974;41:728-35.
- Nugent GR, Odom GL, Woodhall B. Spinal extradural cysts. *Neurology* 1959;9:397-406.

Accepted 31 January 1988

Comparison of the Westmead PTA Scale and Glasgow Coma Scale as predictors of neuro-psychological outcome following extremely severe blunt head injury

Sir: In 1974 Jennett and Teasdale reported on the Glasgow Coma Scale (GCS).¹ Since then this scale has gained widespread acceptance as a means of objectively assessing the depth and duration of coma in head injury and other conditions. Studies have demonstrated its usefulness in predicting outcome following head injury.^{2,3} However, Brooks *et al*⁴ have shown that a subjective and retrospective assessment of the duration of post-traumatic amnesia (PTA) is a better predictor of outcome than duration of coma in head injury patients. No study has compared the objective measurement of duration of coma with an objective measurement of the duration of PTA in predicting neuro-psychological outcome.

The Westmead PTA Scale, described in 1986,⁵ is an objective measurement of the duration of PTA following head injury. It has a high degree of inter-rater reliability, takes approximately only 3 minutes to administer and has been satisfactorily used with only a minimum of training by medical staff, nurses and occupational therapists. It is an extension of the Oxford Scale⁶ and different from the Galveston Orientation and Amnesia Test⁷ in that both orientation and the re-establishment of the daily ability to recall newly learned information are required for defining the end of the PTA period. The present report is of a 2 year follow-up study comparing GCS score of

admission to hospital, duration of coma as determined by the GCS and duration of PTA as determined by the Westmead PTA Scale, as predictors of neuropsychological outcome. The results demonstrate convincingly the predictive superiority of the duration of PTA over the duration of coma. Other factors investigated will be presented in a separate report.

Subjects for the study were selected from patients with blunt head injury referred for neuropsychological assessment in 1985. Selection was dependent upon the patients having been measured daily on the GCS and Westmead PTA Scale, until the respective criteria (a score of greater than 8 on the GCS and 3 successive days of a score of 12 on the Westmead PTA Scale) were met, to indicate the emergence from coma and PTA. Two years after the date of their injury, 22 who met these criteria were available for follow-up neuropsychological assessment. Mean duration of coma was 8.4 days (range < 1 hour to 40 days) and mean duration of PTA was 56 days (range 17 to 150 days). Five outcome measures were obtained: vocabulary score, non-verbal problem solving ability, verbal learning ability, speed of information processing capacity and psychosocial quality of life. The results were analysed using linear regression, with Bonferroni corrections to minimise the possibility of a Type I error.

The only significant predictors of outcome were: (1) duration of PTA which predicted verbal learning ($r^2 = 0.44$; $p = 0.001$) and non-verbal problem solving ($r^2 = 0.37$; $p = 0.003$); and (2) GCS score on admission which predicted verbal learning ($r^2 = 0.37$; $p = 0.003$) and psychosocial quality of life ($r^2 = 0.26$; $p = 0.015$). Duration of coma was not significantly predictive of any of the outcome measures, the r^2 values were highest for verbal learning ($r^2 = 0.20$; $p = 0.035$) and non-verbal problem solving ($r^2 = 0.18$; $p = 0.046$). Duration of PTA thus explained 24% more of the variance in terms of verbal learning outcome than did duration of coma and 19% more of the variance for non-verbal problem solving outcome.

For predictive purposes the Westmead PTA Scale is therefore recommended as an additional objective measure for use in centres managing patients with blunt head injury.

I thank Drs J E Marosszeky, J Sandanam, N W C Dorsch, Ms J Batchelor, Ms M Matotek, Mr P Fahey, and Asst Prof W L Walker for their support in this project. I also wish to acknowledge the help of the Westmead and Parramatta Hospitals' allied health staff who carried out the daily admin-

istration of the Westmead PTA Scale.

E ARTHUR SHORES
Neuropsychology Unit,
Dept of Clinical Psychology,
Westmead Hospital,
Westmead NSW 2145,
Australia

References

- 1 Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical Scale. *Lancet* 1974;ii:81-4.
- 2 Teasdale G, Jennett B. Assessment and prognosis of coma after head injury. *Acat Neurochir (Wein)* 1976;34:45-55.
- 3 Jennett B, Teasdale G, Braakman R, Minderhoud J, Heiden J, Kurze T. Prognosis of patients with severe head injury. *Neurosurgery* 1979;4:283-9.
- 4 Brooks DN, Aughton ME, Bond MR, Jones P, Rizvi S. Cognitive sequelae in relationship to early indices of severity of brain damage after severe blunt head injury. *J Neurol Neurosurg Psychiatry* 1980;43:529-34.
- 5 Shores EA, Marosszeky JE, Sandanam J, Batchelor J. Preliminary validation of a clinical scale for measuring the duration of post-traumatic amnesia. *The Med J Aust* 1986;144:569-72.
- 6 Artioli I, Fortuny L, Briggs M, Newcombe F, Ratcliff G, Thomas C. Measuring the duration of post traumatic amnesia. *J Neurol Neurosurg Psychiatry* 1980;43:377-9.
- 7 Levin HS, O'Donnell VM, Grossman RG. The Galveston orientation and amnesia test: A practical scale to assess cognition after head injury. *J Nerv Ment Dis* 1979;167:675-84.

Accepted 3 August 1988

Acute encephalopathy in adult as delayed presentation of occupational lead intoxication

Sir: Acute encephalopathy is an unusual feature of adult lead intoxication. Over 50 cases have been described in the literature,¹ usually related to recent ingestion of illicit alcohol.² We report an occupational lead intoxication in an adult presenting as acute encephalopathy 7 years after lead exposure.

A 72 year old man was admitted to hospital because of gait and behavioural disturbances. He had worked in a lead foundry until 7 years before. He drank little alcohol. He felt well until 3 weeks before admission, when he presented with diarrhoea and fever which resolved with symptomatic therapy. He remained asthenic and rested in bed. Five days before admission he developed irritability, insomnia and progressive ataxia. On examination, the patient appeared pale, confused and disoriented in time and place. He showed severe truncal ataxia without other neurological abnormalities. His vital signs, cardiopulmonary examination and abdomen were normal. Laboratory data disclosed 2.35×10^{12} red blood cells/l, haemoglobin 4.03 mmol/l, haematocrit 18.9%, and 10.2% reticulocytes. Red blood cell indices, leucocyte and platelet counts were normal. A biochemical battery (SMAC Technicon) was normal except for bilirubin (23.9 $\mu\text{mol/l}$) and lactic dehydrogenase (270 IU/l). A serological test for syphilis was negative. Serum folic acid and cyanocobalamin were normal and a therapeutic trial with thiamine was unsuccessful. An electroencephalographic study revealed diffuse slow waves. Computed tomography of brain was normal. Lumbar CSF showed no pleocytosis and a protein level of 2.6 g/l. A peripheral blood smear displayed intense basophilic stippling of erythrocytes and the analytical study of lead intoxication was positive (table). The patient received parenteral therapy with calcium disodium edetate (CaEDTA) and dimercapitol (BAL) for 5 days followed by oral penicillamine. After the second day of chelating therapy there was a dramatic clinical improvement with progressive disappearance of anaemia and EEG and laboratory abnormalities. An exhaustive questionnaire about recent activities at risk for lead exposure was negative. The patient lived at home with his wife. Her lead blood level was 1.06 $\mu\text{mol/l}$. The water lead level at the patient's house was harmless (0.095 $\mu\text{mol/l}$).

This patient presented a picture of diffuse encephalopathy. No metabolic, infectious

Table Analytical markers of lead intoxication in the case reported

Parameter	Patient	Reference	Values
Whole blood lead	4.34	0-1.93	$\mu\text{mol/l}$
Urine lead	2.17	0.53-5.79	$\mu\text{mol/day}$
Urine lead after EDTA chelation	328.2	0-24.13	$\mu\text{mol/day}$
Delta amino levulinic dehydrogenase activity	50	108-300 units/ml of erythrocytes	
Free erythrocyte protoporphyrin	3.52	0.28-0.64	$\mu\text{mol/l}$
Urine delta amino levulinic acid	637.8	7.6-53.4	$\mu\text{mol/day}$
Urine coproporphyrins	1,756	0-312	nmol/day
Urine uroporphyrins	1,014	0-31.2	nmol/day