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 1 error.

The only significant predictors of outcome were: (1) duration of PTA which predicted verbal learning ($r^2 = 0{\cdot}44$; $p = 0{\cdot}001$) and non-verbal problem solving ($r^2 = 0{\cdot}37$; $p = 0{\cdot}003$); and (2) GCS score on admission which predicted verbal learning ($r^2 = 0{\cdot}37$; $p = 0{\cdot}003$) and psychosocial quality of life ($r^2 = 0{\cdot}26$; $p = 0{\cdot}015$). Duration of coma was not significantly predictive of any of the outcome measures, the r^2 values were highest for verbal learning ($r^2 = 0{\cdot}20$; $p = 0{\cdot}035$) and non-verbal problem solving ($r^2 = 0{\cdot}18$; $p = 0{\cdot}046$). 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 administration 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 Artiola 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 $2{\cdot}35 \times 10^{12}$ 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 μmol/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 dimercaprol (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 μmol/l. The water lead level at the patient's house was harmless (0·095 μmol/l).

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

Table *Analythical markers of lead intoxication in the case reported*

Parameter	*Patient*	*Reference*	*Values*
Whole blood lead	4·34	0–1·93	μmol/l
Urine lead	2·17	0·53–5·79	μmol/day
Urine lead after EDTA chelation	328·2	0–24·13	μmol/day
Delta amino levulinic dehydrogenase activity	50	108–300 units/ml of erythrocytes	
Free erythrocyte protoporphyrin	3·52	0·28–0·64	μmol/l
Urine delta amino levulinic acid	637·8	7·6–53·4	μmol/day
Urine coproporphyrins	1,756	0–312	nmol/day
Urine uroporphyrins	1,014	0–31·2	nmol/day

nor structural cause was found. The presence of biological markers of lead intoxication and the favourable response to chelation therapy allowed the diagnosis of acute lead encephalopathy.[2] The search for recent occupational and environmental exposure was negative. The normal blood lead in the patient's wife rules out some other environmental source which might be unnoticed. Therefore, the only apparent lead exposure was his past work. The previously reported cases of lead encephalopathy occurred shortly after recent lead exposure[2] except for patients with retained bullets.[3-5] On the other hand, it is known that lead can be stored in bone for decades[6] and mobilised by an intercurrent stress with increased bone turnover.[1 3-5] The gastroenteritic process and bed rest in this patient could have been the conditions which caused lead mobilisation.

To our knowledge, this is the first case reported of acute encephalopathy as a likely delayed presentation of occupational lead exposure. This observation emphasises the possibility of severe late toxicity as the first manifestation of a remote lead exposure.

CARLOS GUIJARRO
JUAN DE D GARCÍA-DÍAZ
OLGA HERRERO
JOSÉ L ARANDA
Departamento de Medicina Interna, Hospital Primero de Octubre, Universidad Complutense, Carretera de Andalucía, km. 5,400, 28041 Madrid, Spain

Correspondence should be addressed to Carlos Guijarro.

References

1 Cullen MR, Robins JM, Eskenazi B. Adult inorganic lead intoxication: presentation of 31 new cases and a review of recent advances in the literature. *Medicine (Baltimore)* 1983; **62**:221–47.
2 Witfield CR, Ch'ien LT, Whitehead JD. Lead encephalopathy in adults. *Am J Med* 1972; **52**:281–98.
3 Cagin CR, Diloy-Puray M, Westerman MP. Bullets, lead poisoning and thyrotoxicosis. *Ann Intern Med* 1978;**89**:509–11.
4 Dillman RO, Crumb CK, Lidsky MJ. Lead poisoning from a gunshot wound. Report of a case and review of the literature. *Am J Med* 1979;**66**:509–14.
5 Linden MA, Manton WI, Stewart RM, Thal ER, Feit H. Lead poisoning from retained bullets. Pathogenesis, diagnosis and management. *Ann Surg* 1982;**195**:305–14.
6 Rabinowitz MB, Wetherill GW, Kopple JD. Lead metabolism in the normal human: stable isotope studies. *Science* 1973;**182**: 725–7.

Accepted 12 September 1988

Progressive aphasia with right-sided extrapyramidal signs: another manifestation of localised cerebral atrophy

Sir: Slowly progressive aphasia without generalised dementia has been recognised as a degenerative condition with non-Alzheimer pathology[1 2] and localised histological changes have been demonstrated in the dominant perisylvian regions.[3] We report a unique case in which progressive aphasia is combined with right-sided tremor and rigidity. Computed tomography and single photon emission tomography indicated a striking, predominantly left hemisphere disorder.

A right handed 64 year old man presented with an 18 month history of insidiously progressive deterioration in his language together with tremor of the right hand. He had been previously well and there was no history of vascular disease or head injury. His mother, who died aged 83, was said to have had Parkinson's disease and became "confused and wandering" late in life. General physical examination was normal. Visual fields were intact but eye movements were abnormal with reduced voluntary upgaze and impaired convergence. He had an expressionless facies and slight right-sided facial asymmetry. Tone was minimally increased on the right and there was a resting tremor of the right hand. Reflexes were symmetrically brisk and plantar responses flexor. Sensory testing, dexterity and gait were normal.

Neuropsychological assessment revealed a selective language disorder. His spontaneous speech was non-fluent, stuttering and effortful with word finding difficulty and the occasional intrusion of literal and verbal paraphasias. Comprehension and repetition were mildly impaired. He could read aloud and understand single words, but misread complete phrases and sentences. His writing, although conveying the sense adequately, contained spelling errors and was telegrammatic in style. In contrast to his impaired linguistic ability, praxis, visual perception, spatial localisation and navigational skills and non-verbal memory were preserved. His personality was unchanged and his social conduct was appropriate.

Computed tomography revealed widening of the left sylvian fissure and prominence of the left lateral ventricle. Otherwise, routine laboratory and neurological investigations including electroencephalography were normal. Carotid angiography demonstrated a tonsillar loop of the right internal carotid artery but was otherwise normal.

Two years later he had deteriorated both physically and in his powers of communication. Neurological examination revealed emotional lability, increase in tremor of his right hand, moderate right-sided hypertonia and bradykinesia. He dragged his right leg when walking. His spontaneous speech was limited to "yes", "no" and infrequent stereotyped phrases. Occasional neologisms occurred. Comprehension had deteriorated although he could follow elementary commands. He was severely anomic. He could no longer sign his name. He had difficulty communicating by gesture or pantomime. Perceptual and spatial abilities remained well preserved. Memory could not be formally tested, although his wife believed that he could recall day to day events.

Single photon emission tomography using the tracer ^{99m}Tc-HMPAO, demonstrated a striking reduction in uptake in the left frontal, temporal and parietal regions and in the left subcortex, whilst right hemisphere uptake was normal (fig (a)).

One year later a mild right-sided pyramidal weakness had developed in addition to the extrapyramidal signs. Tendon reflexes on the right were brisk and the right plantar response equivocal. He had a right-sided grasp reflex, with pout and sucking reflexes. Speech was limited to arbitrary production of "yes" and "no", and comprehension to following midline commands. Perception and spatial functions again appeared preserved in that he negotiated his environment without becoming lost, and recognised objects, oriented and used them appropriately. Performance was normal on a forced choice object recognition test suggesting some preservation of visual memory. His personality remained unchanged and he continued to demonstrate initiative in daily activities. A repeated electroencephalogram remained normal whilst computed tomography demonstrated increased atrophy and ventricular dilatation on the left, with the addition of mild right-sided involutional changes (fig (b)).

There are strong grounds for assuming that this patient has a degenerative condition due to localised cerebral atrophy. There has been a slow, progressive development of neurological signs in the complete absence of stroke-like episodes and without risk factors for vascular disease. Cerebral angiography has excluded large vessel disease and the possibility that emboli from the heart or small vessel disease would produce such a striking, predominant involvement of the left cerebral hemisphere is unlikely. Computed tomography showed no low density lesions to suggest cerebral infarction. The electroencephalogram was normal whereas in vascular dementia focal slowing is usual.[4]