Letters

Neuropsychological functions in the follow-up of transient global amnesia

Sir: Since the description by Fisher and Adams,¹ transient global amnesia has been defined as loss of short-term memory characterised by the inability to retain new information, with repetitive queries and retrograde amnesia, but with no other neurological signs and symptoms. Although patients of transient global amnesia seem to recover completely after an episode, Mazzuchi *et al* have demonstrated that a persistent decrease in verbal IQ can occur.² We have conducted a prospective analysis of 29 patients with transient global amnesia. The mean age of the patients at the time of their episode was 60.9 years (± 8.0). All criteria for the diagnosis of transient global amnesia were present at the time of the episode: (1) transient amnesic attack with no direct relation to cranial trauma, (2) evidence given by a witness of the inability to form new memories, (3) repetitive queries, (4) apparently normal behaviour and orientation, (5) evidence given by a witness of normal long-term memory and (6) presence of retrograde amnesia at least during the episode. These criteria, more restrictive than those of other authors,³ rule out other possible causes. The patients were submitted to the Wechsler Bellevue Intelligence Scale six months after admission. A group of 29 control subjects with a mean age of 61.8 years (± 6.9) were submitted to the same neuropsychological test. Student's *t* test was used for statistical evaluation.

The following values were obtained: transient global amnesia-verbal IQ 92.8 ± 15.4 , Control-verbal IQ 90.2 ± 14.6 ($p = 0.25$); transient global amnesia-performance IQ 100.4 ± 12.0 , Control-performance IQ 99.6 ± 15.9 ($p = 0.42$); transient global amnesia-Full IQ 95.0 ± 14.8 , Control-Full IQ 94.1 ± 16.4 ($p = 0.41$). The most striking feature of the study was that no difference could be demonstrated between patients with transient global amnesia and control subjects. No difference was evident either in relation to the episode's duration, or in relation to the duration of the retrograde amnesia.

The sixteen patients described by Mazzuchi *et al*² had a verbal IQ and performance IQ greater than a control group and we think that the decrease in verbal IQ in relation to performance IQ could be due to a poor selection of the control group.

These authors interpreted their results as indicative of left hemisphere involvement in transient global amnesia, but our own results, the report of a patient with a mass in the non-dominant hemisphere,⁴ and a patient described by Ladurner *et al*⁵ with a hypodense lesion in the right hemisphere demonstrated by CT examination seem to discount this hypothesis on the role of the left hemisphere in the production of episodes of transient global amnesia.

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Neuropsychiatric symptoms in the course of Wegener's granulomatosis

Sir: Wegener's granulomatosis is an uncommon connective tissue disorder resembling polyarteritis nodosa; it causes granulomata and vasculitic lesions throughout the body, predominantly in upper respiratory tract, lungs and kidney.¹ Treatment with cyclophosphamide (usually combined with steroids) produces survival rates of about 85%. In a review of 104 patients, Drachman² reported that 54% had neurological involvement, predominantly peripheral lesions. Reports of central nervous system involvement are rare,²⁻⁴ and include a patient with immunosuppressant reversible dysphasia, thought to be secondary to cerebral vasculitis. The case reported here suggests that both neurological and neuropsychiatric disturbance may

occur in the course of the disease as the result of central nervous system involvement. This has not been documented previously.

A 55-year-old woman presented with a three-week history of right-sided bloody nasal discharge. Biopsy of granular tissue from the right nostril suggested Wegener's granulomatosis, confirmed by biopsy of a right lower lobe bronchus lesion. Treatment was started with cyclophosphamide and prednisone. Despite resolution of her nasal symptoms, her relatives noticed that she was becoming drowsy and unsteady on her feet. She complained of blurred vision and polydipsia although blood glucose estimations were normal. Following development of a herpes zoster infection over her right hemithorax, the immunosuppressants were stopped. She rapidly became morose, apathetic, and prone to spells of tearfulness. This apparent depression was thought to be due to her having been widowed shortly before the onset of her illness. There was no family or past personal history of psychiatric disorder.

Examination revealed a pale, distressed woman, mildly clouded and slow to respond, with a marked expressive dysphasia. Her mood was fearful and depressed with secondary delusions of persecution by family and nursing staff. Cognitive testing showed impaired concentration with disorientation for time and a tendency to perseverate. She scored 17/37 on a standardised test of global cognitive function, with severe impairment (1/5) on the short-term memory sub-test. Apart from her dysphasia and an unsteady gait, there were no abnormal neurological signs. An EEG showed fluctuating delta components on both sides, superimposed on a slow dominant rhythm posteriorly indicative of a widespread or multi-focal organic disturbance. A diagnosis of sub-acute organic brain syndrome (ICD 293.1) with paranoid and depressive symptoms was made. Cultures of blood, sputum, urine and CSF failed to detect an infective cause. The CSF was normal.

Cyclophosphamide 150 mg daily and prednisone 60 mg daily were re-started because of her progressive deterioration. Within two weeks her mental state was much improved with significant resolution of her dysphasia and a measured increase in cognitive ability to 32/37. Her short-term memory impairment was unchanged. Her paranoid and depressive symptoms became less evident. An EEG recorded eight weeks after the first showed a return