Letters

any previous case where psychological symptoms caused by an unremoved cerebral tumour had been successfully treated by leucotomy but it is well-established that psychiatric symptoms caused by organic brain disease may at first respond well to other physical methods of treatment⁷ and this occurrence can be a pitfall for the unwary psychiatrist. We have experience of a further patient with a frontal meningioma whose initial depressive symptoms remitted for several months after electroconvulsive therapy.

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Accepted 23 February 1984

Kinesigenic foot dystonia as a presenting feature of Parkinson's disease

Sir: Both Charcot¹ and Gowers² make brief reference to the occurrence of dystonic foot postures in patients with established Parkinson's disease, but the symptom was disregarded until Duvoisin and colleagues reported that in-turning of the foot with associated dorsiflexion of the great toe could be aggravated by levodopa therapy.³ The occasional presentation of Parkinson's disease with writer's cramp is well known, but we would like to draw attention to a similar dystonic mode of onset in the foot

as an early symptom, first described by Purves-Stewart in 1898.⁴

At the age of 42 years a marathon runner of international repute began to experience cramp-like discomforts in his right foot after running about 10 miles. This gradually became more severe and he began to notice a curling in of his toes with a tendency for his right foot to twist inwards; this would bring him to a halt, but after a rest period of a few minutes to half an hour he would be able to continue albeit for a shorter distance. Pain and discomfort became more incapacitating in the right foot and he underwent myelography and an abortive exploration of his right common peroneal nerve. Within a year of the onset of these symptoms he was forced to give up competitive running, finding that after only 5 minutes stiffness in the right popliteal region and splaying of his right foot would cause him to lose control and trip. A year later he noticed increasing difficulty with writing, micrographia was confirmed and shortly after this a mild rest tremor of the right hand was noted. Within a few months he found it almost impossible to write and typing became inaccurate. Even walking short distances on the level would cause his toes to cramp up for several minutes. Examination now revealed facial hypomimia, cogwheel rigidity of the right arm and a static Parkinsonian tremor. There was also unequivocal right-sided bradykinesia and a tendency after walking for his right foot to claw.

A woman who developed mild right-sided bradykinesia with micrographia and a postural tremor of the right hand at the age of 39 years, two years later found that after jogging for about half a mile she had to rest because of severe cramps and twisting spasms of her right foot causing her lower limb to buckle with pain. A short period of rest would enable her to continue and she found that running over pebbles would also temporarily relieve the discomfort. This patient subsequently developed severe early morning foot dystonia while receiving levodopa therapy.

In a third case, a man's Parkinson's disease presented at the age of 45 years with tremor of the left hand and within four years of the onset he was experiencing curling in of the toes of the left foot after walking fifty yards on the level. Rest and massage for two to ten minutes would enable him to continue for a further distance.

This effort induced phenomenon resembles the intermediate form of familial paroxysmal dystonic choreoathetosis⁵ and the dystonic seizures occasionally seen in

multiple sclerosis.⁶ In preliminary studies on two additional Parkinsonian patients who were experiencing levodopa-induced early morning and end-of-dose dystonia,^{7,8} exercise on a bicycle has consistently induced shortlived dystonia of the affected foot even at times of peak dosage. Studies are in progress to determine the underlying pharmacological mechanism of this intriguing disability.

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Accepted 18 February 1984

Mild Reye's syndrome in an adult

Sir: Since the description of Reye's syndrome in 1963¹ several thousand cases have been notified in the USA alone.² The small numbers of adult patients all had severe encephalopathies, often with fatal outcome.³⁻⁷

A 16-year-old male developed typical varicella having been exposed to the illness by his sister. Initial complaints were solely of rash and mild headache. On the fourth day of the illness he suddenly developed profuse vomiting, confusion and aggressive behaviour. He received no medication

other than metoclopramide and, in particular, had not received aspirin. There was a past history of asthma controlled in recent years by inhaled salbutamol. On admission he was vomiting, confused and irritable. Except for typical varicella skin lesions and clinical evidence of dehydration, there were no abnormal signs. Intravenous normal saline was given. After a few hours combative behaviour necessitated sedation with intravenous diazepam and chlor-methiazole. At this time bilateral extensor plantar responses were found. CT head scan and CSF examinations were carried out. Intravenous fluids were continued but no other medication was given. By the following day he was alert, cooperative and orientated and without abnormal neurological signs. A prompt and complete recovery was made and he remained well two months later.

Initial hospital investigation gave the following results: haemoglobin 16.4 g/dl, white cell count $12,200 \times 10^9/l$ (62% polymorphs, 36% lymphocytes), platelet count $47,000 \times 10^9/l$, plasma glucose 6.1 mmol/l, urea 8.1 mmol/l, sodium 142 mmol/l, potassium 3.5 mmol/l; total bilirubin 13 $\mu\text{mol/l}$, aspartate aminotransferase activity 165 IU/l (normal 13–38), alkaline phosphate activity 396 IU/l (adult range 75–245) and plasma ammonia 248 $\mu\text{mol/l}$ (normal range 10–47). Clotting studies were normal. Two days later the platelet count was normal and the sole biochemical abnormality was aspartate aminotransferase activity 395 IU/l. Lumbar puncture on the second day of admission gave an opening pressure of 230 mm of water. The CSF contained no cells and protein content was 150 mg/l. CT head scan was normal. EEG performed on the third day of admission showed a mild bilateral excess of theta activity. Serum CFT to varicella zoster was positive at a titre of 1:320. Convalescent biochemical tests, including aspartate aminotransferase, were normal.

Perhaps due to wider recognition of the condition, mild cases of Reye's syndrome in children have been noted with increasing frequency.⁸ This case associated with an otherwise uncomplicated varicella infection indicates that mild cases also occur in adults. We suspect that Reye's syndrome may go unrecognised and emphasise the importance of measuring plasma ammonia levels in cases of acute encephalopathy with profuse vomiting. Prompt diagnosis is important because the mitochondrial changes and metabolic defects are normally self-limiting⁹ provided that the systemic biochemical disturbances particularly

dehydration, hypoglycaemia and cerebral oedema, can be controlled.

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Accepted 18 February 1984

Thiazides, compulsive water drinking and hyponatraemic encephalopathy

Sir, Compulsive water drinking frequently defies diagnosis until the patient develops hyponatraemic encephalopathy. This complication most often arises when normal homeostatic mechanisms are deranged.¹ When such mechanisms are preserved, additional factors must be sought, one of which is the use of a thiazide diuretic.^{1,2} To our knowledge, there have been 11 detailed case reports of hyponatraemic

encephalopathy in patients with compulsive water drinking taking a thiazide diuretic.^{1–7} The infrequency of this association was felt to merit a further addition to this literature.

A 48-year-old female was admitted to hospital in 1982 with a 7 day history of progressive confusion and restlessness. She had a 20 year history of alcohol abuse. In 1972 she underwent uncomplicated aneurysm surgery for subarachnoid haemorrhage. Four years later she had a single generalised seizure, since when she had taken phenobarbitone 90 mg per day. For one year she had taken Moduretic (hydrochlorothiazide 50 mg and amiloride hydrochloride 5 mg) 1 tablet per day for hypertension. On admission, she was restless, irritable and disorientated. There were no psychotic features. Physical examination was unremarkable. A provisional diagnosis of alcohol withdrawal was made and diazepam, 15 mg per day, and multivitamins were prescribed. The Moduretic and phenobarbitone were continued. Within 2 days her abnormal mental state had resolved, at which time blood biochemistry was normal.

Three weeks after admission, she complained of insatiable thirst and was noted to be drinking large volumes of water. Symptoms of headache and vomiting were followed by confusion, coma and a single seizure. At this time the serum sodium was 119 mmol/l; the potassium 3.3 mmol/l; the chloride 81 mmol/l; the creatinine 344 $\mu\text{mol/l}$; and the urea 4.9 mmol/l. The plasma osmolality was 237 mOsm/kg H_2O with coincident urine osmolality of 169 mOsm/kg H_2O , urine sodium of 24 mmol/l and urine potassium of 17 mmol/l. Other relevant investigations⁸ were normal. Moduretic was discontinued and phenytoin, 300 mg per day, was added. Phenobarbitone was withdrawn 4 days later. Restriction of water intake resulted in rapid clinical recovery and return of biochemical parameters to normal. Shortly thereafter, a further episode of excessive water drinking occurred. Intake was restricted and there were no sequelae. Five weeks later she was discharged on phenytoin, 300 mg per day, having displayed no further desire to drink excessively.

There are at least three mechanisms whereby thiazides could have contributed to the hyponatraemia in our patient and the 11 other reported cases.^{1–7} First, as a direct consequence of natriuresis. Second, as a result of reduced free water clearance. These mechanisms may have greatest relevance to the three patients who developed