that reported by McIntyre in industry.^{2,3} McIntyre suggested that about one half of cases were undisclosed, and our study supports this. According to Scambler and Hopkins, only one in 10 people with epilepsy always disclosed their disability.⁴ As Gloag pointed out, "people fear... that their job applications will not be considered on their merits if they admit a history of epilepsy and... their medical history will not be assessed by anyone knowledgeable."⁵

How might things be improved? One way is to establish guidelines that distinguish between how prospective new employees with epilepsy and employees who develop epilepsy are dealt with. In the first case the employer should state clearly if particular jobs are unsuitable for people with epilepsy. Espir suggested that people should be rejected on medical grounds only after a medical examination.⁵ In our view this examination should be undertaken by an occupational physician as it is on his advice that the employer relies. Guidelines would be beneficial to all those working in the NHS and ought to ensure a uniform procedure. There are undoubtedly some posts within the NHS that are unsuitable for people with epilepsy, particularly those that entail direct responsibility for the lives or safety of patients or other employees.

If onset of epilepsy occurs after the start of employment a full assessment must be made by the occupational physician in consultation with the employee's medical adviser. Recommendations should be made on the individual merits of each case. Every effort should be made to modify the demands of the post so that it is safe for the employee to continue in it or to find him suitable alternative work. Each employee with epilepsy should be regularly reviewed by the occupational physician so that he can give advice on matters related to work, monitor progress, and allay the anxiety of the employee.

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Accumulation of aluminium in chronic renal failure due to administration of albumin replacement solutions

In patients with renal failure aluminium may accumulate and cause osteomalacia resistant to vitamin D, dialysis encephalopathy, or microcytic anaemia.¹ The usual source of aluminium is the water supply used to prepare the dialysis fluids or aluminium compound ingested as phosphate binders. We report on a patient in whom high plasma aluminium concentrations resulted from another, little recognised source—that is, intravenous infusions of albumin replacement solutions.

Case report

A 62 year old woman receiving continuous ambulatory peritoneal dialysis for end stage renal failure of uncertain cause was admitted with peritonitis. She failed to respond to standard treatment with intraperitoneal and parenteral antibiotics and on day 25 of her admission was changed to haemodialysis. Thereafter she underwent dialysis for 12 hours each week in two sessions. Aluminium concentrations in the dialysis fluid varied from 0.1 to 0.4 $\mu\text{mol/l}$ (2.7 to 10.8 $\mu\text{g/l}$). On admission she was taking 2.85 g aluminium hydroxide daily (that is, six Alu-cap capsules daily), and this was continued until day 49, after which she did not receive any oral aluminium compounds.

Plasma aluminium concentrations (measured by atomic absorption spectrophotometry²) were low on admission and fell slightly until day 49 (figure). Plasma albumin concentration had fallen to 20 g/l on day 49, and she was given a total of 22 units (8800 ml) of 4.5% human albumin solution (Blood Products Laboratory, Elstree, Herts) during dialysis from day 48 to day 87. Coincident with the regular infusion of albumin replacement solutions the plasma aluminium concentrations increased to a potentially toxic value (10.9 $\mu\text{mol/l}$ (29.4 $\mu\text{g/100 ml}$)) and then fell

sharply when the treatment was stopped (figure). Analysis of 10 samples of the human albumin solution administered showed a mean aluminium concentration of 8.3 $\mu\text{mol/l}$ (22.4 $\mu\text{g/100 ml}$) (range 7.3-9.6 $\mu\text{mol/l}$ (19.7-25.9 $\mu\text{g/100 ml}$)).

Comment

Serum aluminium concentrations in excess of 3.7 $\mu\text{mol/l}$ (100 $\mu\text{g/l}$) are usually associated with deposition of aluminium in bone,³ which may lead to impaired bone formation and mineralisation. In our patient the raised aluminium concentration was clearly related to albumin replacement treatment and not to the usual sources of aluminium loading in patients receiving maintenance haemodialysis. Accumulation of aluminium in renal failure due to albumin replacement treatment has not previously been

Relation between plasma aluminium concentrations and albumin replacement.
Conversion: SI to traditional units—Aluminium: 1 $\mu\text{mol/l}$ $\approx$ 2.7 $\mu\text{g/100 ml}$.

described, although a transient increase in plasma aluminium concentration and urinary aluminium excretion after plasmapheresis was reported in a man with a normal serum creatinine concentration.⁴ Other intravenous infusions may contain appreciable amounts of aluminium—for example, parenteral feeding solutions⁵—but this source of aluminium is probably under-recognised.

Aluminium loading due to intravenous treatment is likely to be of particular importance in patients with renal impairment requiring prolonged parenteral nutrition, frequent albumin replacement treatment, or plasmapheresis.

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