weakness in a poorly co-operative addict in the withdrawal stage. Tenderness and pain could well make bedside examination of sensory function impossible. Alcoholic peripheral neuropathy should also be considered as a possible cause of the sensory loss.

The case for peanut oil has certainly not been proved and it would be premature to add this substance to the long list of causes of rhabdomyolysis. In this context it is of interest that peanut butter in the melted form has been reported to be used intravenously by addicts in Los Angeles, though there is no known "high" effect.¹⁰

PETR SKRABANEK

Department of Neurology,
St Laurence's Hospital,
Dublin

- 1 Richter, R W, *et al*, *Journal of the American Medical Association*, 1971, **216**, 1172.
- 2 Penn, A S, Rowland, L P, and Fraser, D W, *Archives of Neurology*, 1972, **26**, 336.
- 3 Rowland, L P, and Penn, A S, *Medical Clinics of North America*, 1972, **56**, 1233.
- 4 Perkoff, G T, Hardy, P, and Velez-Garcia, E, *New England Journal of Medicine*, 1966, **274**, 1277.
- 5 Hed, R, Larsson, H, and Wahlgren, F, *Acta Medica Scandinavica*, 1955, **152**, 459.
- 6 Fahlgren, H, Hed, R, and Lundmark, C, *Acta Medica Scandinavica*, 1957, **158**, 405.
- 7 Hed, R, *et al*, *Acta Medica Scandinavica*, 1962, **171**, 585.
- 8 Schneider, R, *Southern Medical Journal*, 1970, **63**, 485.
- 9 Richter, R W, and Rosenberg, R N, *Journal of the American Medical Association*, 1968, **206**, 1255.
- 10 Landy, E E, *The Underground Dictionary*, p 147. London, MacGibbon and Kee, 1971.

Localised plasmacytoma in a patient with α -chain disease in remission

SIR,—We read with great interest the letter from Dr J Rogé and others (25 October, p 225) about their unique case of α -chain disease which remains in complete remission six years after treatment with antibiotics only. This case and our own case, to which they refer (25 May 1974, p 409) represent benign examples of α -chain disease that respond to treatment, remain in remission for long periods of time, and are possibly cured. At the other end of the scale is the fulminant type which shows poor response to treatment and fast deterioration and death from generalisation of the disease.¹⁻³

We should like to take this opportunity to report the further history of our case, which may be of interest. This patient had been symptom-free and in complete immunological and histological remission for 20 months after stopping all treatment when he developed a plasmacytoma involving the ileocaecal area. During this phase of his illness there was no evidence of reactivation of α -chain disease. The tumour was resected and the patient, nearly two years after the operation, remains symptom-free on a weekly maintenance dose of 200 mg of cyclophosphamide intravenously. It appears that the plasmacytic tumour of the ileocaecal area originated from a new abnormal clone and that the patient's α -chain disease is still in remission.

O N MANOUSOS
J C ECONOMIDOU

Second Department of Medicine,
University of Athens,
Hippokraton General Hospital,
Athens

- 1 Seligmann, M, *et al*, *Annals of the New York Academy of Sciences*, 1971, **190**, 487.

- 2 Economidou, J C, *et al*, *American Journal of Digestive Diseases*. In press.
- 3 Manousos, O N, in *Topics in Gastroenterology*: 3, ed C S Truelove and M J Goodman, p 259. Blackwell, Oxford, 1975.

Salt overdosage

SIR,—The report of a case of salt overdosage (15 November, p 386) and subsequent correspondence raise questions of practical importance in managing hyperosmolar states. Dr R C M McGouran, in discussing his report, recommends rapid lowering of the plasma sodium to limit the duration of damaging osmotic shrinkage of brain tissue. Dr Carol Fitzpatrick (29 November, p 517) feels that the standard practice of slow rehydration of hypernatraemic infants should be extended to adult cases to prevent brain damage due to a rebound cellular overhydration with cerebral oedema and convulsions.

In animal experiments Holliday *et al*¹ have shown that saline infusions producing constant hypernatraemia lead to shrinkage of brain cells over the first few hours followed by a gradual resumption of normal cell volume, which is complete within a few days and is accompanied by an increase in cell potassium. This increase falls short of the calculated requirement to restore normal cell volumes at the raised osmolality and they concluded that, in addition to the uptake of potassium ions, the cell substrate itself could generate, by molecular rearrangement, so called "ideogenic osmoles" to protect against a hyperosmolar extracellular milieu.

Consequently in treatment of a hyperosmolar patient, once normal cell volumes have been attained, if this augmented intracellular osmole quota (both potassium and ideogenic) cannot be jettisoned at a parallel rate with the fall in extracellular osmolality, further water may enter the cells and over-expansion of the intracellular space ensue. It could aid rational management if it were possible to detect clinically the point at which the initially contracted intracellular space had regained its normal size and was in imminent danger of over-expansion. Holliday's work suggests that at this point the avidity for potassium of the contracted cell should have declined to zero and potassium ions should begin to pass back into the extracellular fluid. This occurrence should be clinically recognisable. Dr McGouran's case demonstrates the initial phase of cellular potassium avidity, with a plasma potassium of 3.1 mmol/l in the absence of any stated reason for potassium depletion.

In a personal case of acute hypernatraemia without dehydration the initial plasma sodium level was 186 mmol/l and the patient deeply comatose. Treatment with one litre of 5% dextrose infused every eight hours and no diuretics produced a steady fall to normal sodium levels by the fifth day. No convulsions occurred, but consciousness took three weeks to return and recovery of the central nervous system was insufficient to allow the patient to return to an independent existence. The initial potassium level was 2.9 mmol/l and ECG changes of hypokalaemia were marked. As the plasma sodium declined to normal levels the potassium rose to 4.6 mmol/l and the ECG became normal. Once plasma potassium had risen within the normal range, on the third day, urinary potassium increased and over the subsequent four days there was a net body loss of over 400 mmol before a balance was reached.

This case illustrates how the considerable movements of potassium that occur in these gravely ill patients may be observed by simple estimations of plasma and urinary electrolytes supplemented by ECG traces.

With further experience I feel they will be increasingly useful in deciding the point at which the rapid initial lowering of osmolality, which some might deem advisable, must be abated to perhaps that which a functioning pair of kidneys and a normal fluid intake might attain.

R S MACDONALD

London W4

- 1 Holliday, M A, *et al*, *Journal of Clinical Investigation*, 1968, **47**, 1916.

Continuous positive airway pressure by facemask in newborn infants

SIR,—We should like to thank Mr L P Allen and his colleagues at University College Hospital for their timely article (18 October, p 137) on continuous positive airway pressure (CPAP) by Bennett facemask. Timely for us because on the day of publication we had an infant with severe respiratory distress syndrome who was rapidly deteriorating.

We had not previously attempted the use of CPAP because the many problems associated with head enclosure (Gregory box, Barrie's bag) and endotracheal tubes were too great, we felt, with insufficient and inexperienced staff. However, the use of the face mask seemed attractively simple. We did not have a Bennett mask and so we used a black rubber oxygen funnel (commonly used for administering oxygen during resuscitation following delivery and found on most resuscitation trolleys), and this produced an excellent seal.

When CPAP was first applied the infant was deteriorating and in 100% oxygen had a P_{aO_2} of 6.1 kPa (46 mm Hg). After two hours of CPAP at 8 cm H_2O the P_{aO_2} had risen to 10.2 kPa (77 mm Hg) and after three hours to 24.7 kPa (186 mm Hg).

The method proved to be simple, safe, and effective and could be easily managed by nursing staff unfamiliar with intensive neonatal care.

D G BANNISTER
C I HAINES

Bromsgrove General Hospital,
Bromsgrove, Worcs

Fluoride and bones

SIR,—Drs Jenifer Jowsey and B L Riggs (27 September, p 766) make a number of comments on our paper concerning prophylactic fluoride treatment and aged bones (12 July, p 73). Their letter includes a few points which we cannot fully understand.

With regard to calcium intake, as we mentioned in our discussion, the amount of milk our patients consumed daily was on average one litre. This contains 1200 mg of calcium, which according to all standards is more than needed. Drs Jowsey and Riggs consider "the lack of accompanying calcium" to have had a deleterious effect on our patients. We, however, think that it is the total amount of calcium consumed which counts and not whether this is taken in tablets or from natural sources. Of course it is important to examine the literature, but many studies done on test animals are in contradiction to the optimistic view of Drs Jowsey and Riggs as to the beneficial effect of fluoride on the strength of bones. And, again, no results from theoretical studies