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]

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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, Larrson, 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.

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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.

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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 Pa_{O_2} of 6·1 kPa (46 mm Hg). After two hours of CPAP at 8 cm H_2O the Pa_{O_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.

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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

can be considered valid in the case of aged people, so that the results of our studies cannot be read from the literature but from our series alone.

We did not learn from Drs Jowsey and Riggs's letter whether their studies—which in general are based on a very small number of patients—included any control series. If there was one, were the studies done blind and did the possible control series also receive calcium and vitamin D, which are indeed very interesting and potent drugs even used separately? If not we cannot be very impressed with their results regarding the effect of fluoride but rather horrified because they observed three fractures in a series of eight patients during the first year and still continued the administration of fluoride. Drs Jowsey and Riggs claim that fluoride might have a beneficial effect on the bones beginning from the second year. It is only unfortunate that with each patient we cannot avoid the first year, during which according to their and our results the fluoride might just do harm. As an end result of treatment changes in bone biopsy and in quantitative radiology might be of a certain value but from the point of view of the patient and of the practising physician the most interesting thing to know is whether the bones break or not and that must also be the main guide for treatment.

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Nutritional protein needs

SIR,—I was astounded to read in the *BMJ* (30 August, p 547) what purported to be a review of a book by Francis Aylward and Mogens Jul on "Protein and Nutrition Policy in Low-income Countries" but which was in fact only a diatribe expounding the reviewer's one-sided approach to malnutrition.

In it Mr P R Payne dismisses the general need for protein by asserting that "there is now no general acceptance that protein deficiency even exists outside the laboratory." To substantiate this sweeping statement he cites only a Ministry of Overseas Development report which he coauthored. As an American scientist working in Britain I can attest that his conclusion is totally incorrect, as evidenced by the current US National Science Foundation study of protein resources, in which almost 100 of the country's food and nutrition experts are participating.

Mr Payne criticises Aylward and Jul for claiming that protein alone is the answer to world nutrition problems. This is a view they neither hold nor even imply in their book. He further goes on to say that malnutrition is merely "a symptom of poverty," a simplistic statement that offers little in the way of a solution to the extremely complex real problems. In fact, regarding nutrition there is only one acceptable position: all the elements which go to make up a nutritionally balanced diet for a population are equally important, and this must include protein.

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Measuring blood pressure

SIR,—Your leading article on measuring blood pressure (15 November, p 366) mentions preliminary assessment of the systolic blood pressure by palpation but fails to stress fully the importance of this procedure.

If blood pressure is taken by auscultation alone one may miss the "silent gap," which can occur during phase I or phase II Korotkoff sounds. The true systolic blood pressure will be that detected by palpation and may be as much as 30 mm Hg greater than that measured by auscultation.

Furthermore, at any level of diastolic blood pressure cardiovascular mortality increases in proportion to the associated systolic blood pressure.[1] Isolated systolic hypertension is associated with increased morbidity and mortality from cardiovascular disease.[2] Thus accurate measurement of the systolic blood pressure is as important as measuring diastolic blood pressure. The silent gap can be reduced by inflating the sphygmomanometer cuff quickly and by not having the arm hanging down.[3]

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1 Gubner, R S, *American Journal of Cardiology*, 1962, **9**, 773.
2 Colandrea, M A, *et al*, *Circulation*, 1970, **41**, 239.
3 Wood, P, *Diseases of the Heart and Circulation*, 3rd edn. London, Eyre and Spottiswoode, 1968.

Serum α-fetoprotein in cystic fibrosis

SIR,—In a recent article Professor R K Chandra and his colleagues (29 March, p 714) reported abnormally high levels of α_1-fetoprotein (AFP) in the serum of 18 Canadian patients suffering from cystic fibrosis (range 56-8825 μg/l); furthermore, they claimed that all 16 heterozygotes tested also had elevated serum AFP levels (mean 178 μg/l). Their results were contradicted by three British studies (Dr J C Wallwork and others and Dr D J H Brock and others, 17 May, p 392, and Dr J S Fitzsimmons and others, 30 August, p 544) but confirmed by another North American series (Dr J A Smith, 17 May, p 392). In view of this geographical discrepancy and because of the potential importance of the reported finding, we attempted to clarify this controversy.

Sera from 40 well-confirmed cystic fibrosis patients and 60 of their parents were obtained through two Canadian (Chicoutimi and Quebec) and one United States (San Diego) cystic fibrosis clinics. AFP was measured by two methods: a double antibody enzyme immunoassay[1] and a sodium sulphate precipitation radioimmunoassay.[2] Both assays had a sensitivity limit of 3 μg/l when referred to the World Health Organisation AFP standard (kindly supplied by Dr P Sizaret, International Agency for Research on Cancer, Lyon, France). The reagents involved in each technique had been independently prepared by LB's (enzyme assay) and SS's (radioimmunoassay) groups.

All sera yielded AFP values below 20 μg/l (upper normal limit). In order to eliminate the possibility of false-negative results the sera were reassayed by the enzyme method after the addition of 100 μg/l of purified AFP. The analytical recovery ranged between 85 and 117 μg/l. Sera also failed to react by counterimmunoelectrophoresis (sensitivity limit 150 μg/l) against anti-AFP serum.

Our results, obtained from subjects from three North American geographical areas and determined by two different, well-characterised methods, thus confirm that serum AFP levels are normal in cystic fibrosis. The results obtained by Professor Chandra and his colleagues and by Dr Smith might be explained by the reactivity of their antisera towards another antigen; the preparation of their antisera is not documented well enough to rule out this possibility. Therefore sera from cystic fibrosis patients may actually contain elevated concentrations of an antigen other than AFP which constitutes a marker of the disease.

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2 Sell, S, and Gord, D, *Immunochemistry*, 1973, **10**, 439.

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Penicillamine therapy, antistriational antibody, and myasthenia gravis

SIR,—In view of recent reports of myasthenia gravis apparently induced by D-penicillamine[1-3] and our own experience of a patient with Wilson's disease complicated by myasthenia gravis, thymic hyperplasia, and anti-acetylcholine receptor antibody while on penicillamine we have reviewed 34 patients treated with penicillamine for rheumatoid arthritis. In particular, we have examined sera for the antistriational antibody found in association with thymomata and myasthenia gravis.[4] Six of these patients (18%) developed antistriational antibody at some stage during the course of therapy. One further patient was found to have antistriational antibody before and during therapy. The titre of the antibody was found to vary somewhat from time to time and in this respect the results are similar to those that we have found in the case of antinuclear factor apparently induced by penicillamine. In the six patients who developed antistriational antibody the latent period after starting penicillamine was 6-24 months. The period of follow-up after the development of the antibody ranges only from one to four months, but none of these patients has manifested overt myasthenia gravis.