

variety of clinical performance tests. One finding of particular interest at that time was that levodopa caused an easily appreciable reduction in the severity of tremor—to an extent that was valued by the patients—in addition to its well-known effects on rigidity and akinesia. The mean Webster rating⁶ for tremor (\pm SD) during a control period was 1.434 ± 0.71 (30 patients), and during treatment with levodopa it fell to 1.030 ± 0.67 (29 patients). Thus a definite improvement in tremor was obtained which was statistically significant ($0.0125 < P < 0.025$) and was of great value to the patients in about one-third of the entire group. The use of levodopa with decarboxylase inhibitors appears to be of similar value in respect of tremor.

It is suggested that when confronted by patients complaining of tremor alone or suffering from the effects of a varying admixture of tremor, akinesia, and rigidity one should commence treatment with levodopa and decarboxylase inhibitor. The dosage should be increased cautiously until a maximum degree of improvement is obtained in all three pathophysiological features. There are some patients, unfortunately, in whom levodopa seems to aggravate the tremor even when at the same time rigidity and akinesia are being improved. The place of thalamotomy should now be restricted to those patients whose tremor fails to respond satisfactorily—or is exacerbated—in response to levodopa.

G M YUILL

Crumpsall Hospital,
Manchester

- ¹ Mawdsley, C, *British Medical Journal*, 1970, **1**, 331.
- ² Hughes, R C, *et al*, *British Medical Journal*, 1971, **1**, 7.
- ³ Calne, D B, *et al*, *Lancet*, 1969, **2**, 973.
- ⁴ Teychenne, P F, *et al*, *Lancet*, 1975, **2**, 473.
- ⁵ Cox, B, *et al*, *Journal of Neurology, Neurosurgery and Psychiatry*, 1973, **36**, 354.
- ⁶ Webster, D D, *Modern Treatment*, 1968, **5**, 257.

Trial by traction

SIR,—With regard to your leading article (3 January, p 2) I would support the need to mount bigger and better trials of lumbar traction but would make a plea for two conditions; firstly, to stop regarding traction as a single treatment, and secondly, for more accurate selection and diagnosis of cases.

Traction is not a single treatment. To prescribe traction is similar to prescribing aspirin. Traction is available in many different forms, different forces in relation to the body weight, different positions of the patient, lumbar flexion, lumbar extension, lateral flexion, etc. It is available in intermittent, continuous, or pulsed variety, and even the periodicity of the pulsing can be different. Future trials should more accurately discuss the reasons for the choice of the method of traction used, together with a detailed description of its procedure and control. Incidentally, in all traction treatments it is necessary to be sure that the harness is such that it applies the traction in the correct or chosen direction and does not slip.

In your article there was mention of a "suggestion that traction might help more when root signs were present,"¹ surely a significant statement when taken with the other report² that "no advantage for this form of treatment [was shown] in patients with lumbago and sciatica," both of which terms

are blanket and ill-defined description of symptoms and not diagnoses. It appears to me that rather more patients with "lumbago" and "sciatica" do not have their symptoms caused by discopathy than do from other causes. Indeed traction may help patients with a nipped synovial fringe in an apophysial joint (as does sometimes manipulation), but there are many causes of both these conditions, which, although mechanical, traumatic, or degenerative in origin, are unlikely to be affected in any way by traction save by the fact that traction is a time-consuming treatment which allows the passage of time. This time may enable nature to effect the healing or cure of symptoms.

BRYAN O SCOTT

Department of Rheumatology and
Rehabilitation,
Radcliffe Infirmary,
Oxford

- ¹ Christie, B G B, *Proceedings of the Royal Society of Medicine*, 1955, **48**, 811.
- ² Weber, H, *Journal of the Oslo City Hospital*, 1973, **23**, 167.

Thrombocytopenia, haemolytic anaemia, and sarcoidosis

SIR,—We read with interest Dr P d'A Semple's case report (22 November, p 440). His patient's clinical features overlap in part with our report¹ of a 63-year-old woman in whom sarcoidosis and idiopathic (immunological) thrombocytopenic purpura (ITP) coexisted. Dr Semple and Dickerman *et al*² seem to suggest that the occurrence of ITP in patients with sarcoidosis may reflect an underlying autoimmune diathesis in sarcoidosis. However, we are impressed by the paucity of reports of serological manifestations of autoimmunity and of clinical autoimmune diseases in patients with sarcoidosis.

We believe that the onus of proof is on those who claim an increased incidence of autoimmune diseases in sarcoidosis to demonstrate that such an association does not simply result from hospital patient selection bias.³

P J A HOLT

CHARLES A J WARDROP

ERIC WADDINGTON

Departments of Dermatology and
Haematology,
University Hospital of Wales,
Cardiff

- ¹ Holt, P J A, Wardrop, C A J, and Waddington, E, *British Journal of Dermatology*, 1973, **supp** 9, p 81.
- ² Dickerman, J D, Holbrook, P R, and Zinkham, W H, *Journal of Pediatrics*, 1972, **81**, 758.
- ³ Berkson, J, *Biometrics*, 1946, **2**, 47.

Low-protein diets in chronic renal failure

SIR,—In your excellent leading article (29 November, p 486) emphasis was given to nutritional deterioration of patients with chronic renal failure. We have found that all prolonged dietary regimens of less than 40 g protein/day, despite a high intake of calories, are subject to this hazard and should be avoided when dialysis and transplantation facilities are available.

Plasma albumin and body weight are unsatisfactory markers of nutritional status but have been used to support short-term nitrogen balance data in the formulation of many low-protein diets. Consequently the progressive malnutrition caused by these diets

may remain undetected until severe clinical symptoms occur. However, some indication of nutritional deterioration is given by the decrease in plasma essential amino-acids^{1,2} and in certain plasma proteins such as transferrin^{3,4} and complement C3.^{2,5} These indices also show increases in response to improvements in nutrition.

Composite diets based on essential amino-acids¹ and keto-acids⁷ are frequently nitrogen-deficient, unappetising, and not readily available. However, there are advantages in supplying essential amino-acids in a palatable form as a supplement to restricted protein diets where nitrogen may be inadequate or marginally adequate (that is, less than 40 g protein/day). This therapy may improve nitrogen balance,⁸ increase the synthesis of liver proteins,² and in the long term minimise uraemic neuropathy⁸ and other symptoms associated with nutritional deterioration.

Further investigations are necessary to prevent the nausea that occurs in some patients and also to evaluate certain semi-essential amino-acids. Studies with our own formulation of essential amino-acids and, more recently, with a commercial product taken as a drink (Nephranutrin, Geistlich and Sons Ltd, Chester) show that tyrosine should be given.² Histidine is also necessary when protein intake is less than 20 g.⁸

G A YOUNG

F M PARSONS

Renal Research Unit, Wellcome Wing,
General Infirmary,
Leeds

- ¹ Giordano, C, *et al*, *Proceedings 3rd International Congress of Nephrology*, 1966, **3**, 214.
- ² Young, G A, and Parsons, F M, *Clinica Chimica Acta*, 1975, **61**, 205.
- ³ Young, G A, and Parsons, F M, *Clinica Chimica Acta*, 1970, **27**, 491.
- ⁴ Ooi, B S, Daroccy, A F, and Pollak, V E, *Nephron*, 1972, **9**, 200.
- ⁵ Kult, J, *et al*, *Deutsche medizinische Wochenschrift*, 1974, **99**, 339.
- ⁶ Llach, F, Franklin, S S, and Maxwell, M H, *Nephron*, 1975, **14**, 401.
- ⁷ Giordano, C, *et al*, *Lancet*, 1972, **1**, 178.
- ⁸ Ahlberg, M, *et al*, *Infusionstherapie*, 1975, **2**, 151.

Incurable Patients Bill

SIR,—In your issue of 17 January (p 165) you publish the details of Lady Wootton's Incurable Patients Bill which is expected to receive a second reading in the House of Lords in February. The Bill sets out the right of a patient (a) to be relieved from the pain and distress of incurable disease, even if unconsciousness results from such relief, and (b) not to be kept alive if the brain becomes so damaged by accident or disease as to render him permanently incapable of giving directions. Undoubtedly this Bill is motivated by compassion for such patients, but I doubt if legislation is the right way in which to protect their rights, for the following reasons:

Firstly, the Bill seeks to regulate treatment by the law (the management of the dying is just as much a matter of treatment as the cure of the living). But however compassionate the reasons may be for doing this in this instance, if the Bill becomes law it will set a precedent which may well be extended into other areas of medical practice. It is fundamental to medical practice that treatment is based upon a doctor's competence and judgment.

Secondly, the relationship between doctor and patient is founded upon trust and responsibility. Most patients believe that