Thrombotic complications in acute polyneuritis

SIR,—We are concerned at the high incidence of venous thrombosis and pulmonary embolism in our patients with acute polyneuritis. In 1973-4 three patients were treated. All received high doses of either prednisolone or corticotrophin and two required assisted ventilation. All had a regular physiotherapy programme. One patient, a 13-year-old girl, developed a left deep vein thrombosis in the sixth week of her illness while being weaned off the ventilator; one had several small pulmonary emboli without clinical evidence of venous thrombosis; and the third, a man of 21, died of a massive pulmonary embolus immediately after a physiotherapy session in the fifth week, again when beginning to improve. There was no clinical sign of venous thrombosis, but at necropsy the left femoral vein was found to be thrombosed.

In the light of this experience our last patient was given subcutaneous heparin 5000 U twice daily for four weeks until some spontaneous movement returned to the calf muscles. Five days after stopping this treatment he developed a pulmonary embolus, again after a physiotherapy session. He was treated with conventional anticoagulants.

It is interesting that in the series of 44 patients with polyneuritis reported by Newer *et al*¹ only three developed clinical evidence of deep venous thrombosis and in none was there definite evidence of pulmonary embolisation. However, using the ¹²⁵I-labelled fibrinogen technique, Warlow *et al*² found that 60% of patients with acute strokes had evidence of leg vein thrombosis in the paralysed leg compared with 7% in the non-paralysed leg. Fatal pulmonary embolism with or without previously detected venous thrombosis is a recognised problem in managing acute spinal injuries³ and the incidence has been reduced with prophylactic anticoagulant therapy.⁴ With the increasing evidence that low-dose subcutaneous heparin is both effective and safe in preventing venous thrombosis and pulmonary embolism^{5, 6} we would now recommend its routine use in patients with acute polyneuritis, continuing treatment until full muscle tone returns to the legs.

JANE LEESE

Communicable Diseases Unit,
St George's Hospital,
London SW17

- ¹ Hewer, R L, *et al*, *Quarterly Journal of Medicine*, 1968, **33**, 479.
- ² Warlow, C, Ogston, D, and Douglas, A S, *Lancet*, 1972, **1**, 1305.
- ³ Watson, N, *Paraplegia*, 1974, **12**, 197.
- ⁴ Hachen, H J, *Paraplegia*, 1974, **12**, 176.
- ⁵ Kakkar, V V, *Thrombosis et Diathesis Haemorrhagica*, 1975, **33**, 87.
- ⁶ International Multicentre Trial, *Lancet*, 1975, **2**, 45.

Guillain-Barré syndrome in acute hepatitis

SIR,—We were interested to read the report by Drs P Niermeijer and C H Gips on Guillain-Barré syndrome in acute hepatitis (27 December, p 732), particularly as they state that they were not aware of documented cases of the syndrome in patients with acute HBsAg-positive hepatitis. We have recently recorded a patient who developed this syndrome during the preicteric phase of type B hepatitis.¹ Leneman,² reviewing 1100 cases of acute idiopathic polyneuropathy, reported the association with viral hepatitis in 11 patients.

Plough and Ayerle³ also documented the association of the syndrome with acute hepatitis, while Balestra⁴ more recently reported the development of the syndrome during the preicteric phase of acute viral hepatitis.

As discussed by Drs Niermeijer and Gips, the mechanism for the association remains unexplained, although an immunological mechanism appears most likely, perhaps associated with the deposition of immune complexes.

P L NG
LAWRIE POWELL
BRYAN CAMPBELL

University of Queensland,
Department of Medicine,
Royal Brisbane Hospital,
Brisbane

- ¹ Ng, P L, Powell, L W, and Campbell, C B, *Australian and New Zealand Journal of Medicine*, 1975, **5**, 367.
- ² Leneman, F, *Archives of Internal Medicine*, 1966, **188**, 139.
- ³ Plough, I C, and Ayerle, R S, *New England Journal of Medicine*, 1953, **249**, 61.
- ⁴ Balestra, F, *Rassegna Internazionale di Clinica e Terapia*, 1969, **48**, 235.

Toxic effects on nerve conduction velocity

SIR,—Dr G M London and others (8 November, p 324) found that in patients with chronic end-stage renal disease the reduction in motor nerve conduction velocity (MNCV) was correlated with the free erythrocyte protoporphyrin (FEP) concentration but not with serum iron or transferrin saturation. They considered the possibility that the neurological dysfunction resulted from the direct toxic action of porphyrin precursors but dismissed this hypothesis because the protoporphyrin levels found in their patients were much lower than those observed in porphyrias. They rather favoured a secondary association between FEP and MNCV—that is, the toxicity of uraemia could depress both nerve conduction and haem-synthetase activity.

We would like to present an argument which reinforces their first hypothesis. It is well known that moderate exposure to inorganic lead increases FEP (before any biological sign of anaemia)^{1, 2} and also reduces MNCV.^{3, 4} An increased FEP level is a common biological disturbance for both conditions. Therefore the hypothesis that accumulation of porphyrin precursors could be directly responsible for the reduction in MNCV deserves further consideration.

R LAUWERYS
J P BUCHET
H ROELS
G HUBERMONT

Unité de Toxicologie Industrielle
et Médicale,
Université Catholique de Louvain,
Brussels, Belgium

- ¹ Stuk, E J, *Internationales Archiv für Arbeitsmedizin*, 1974, **33**, 83.
- ² Roels, H A, *et al*, *Archives of Environmental Health*. In press.
- ³ Seppäläinen, A M, and Hernberg, S, *British Journal of Industrial Medicine*, 1972, **29**, 443.
- ⁴ Seppäläinen, A M, in *Behavioral Toxicology, Early Detection of Occupational Hazards*, p 240. Washington, US Department of Health, Education, and Welfare, 1974.

Ocular toxicity due to rifampicin

SIR,—Drs F E Cayley and S K Majumdar (24 January, p 199) describe a patient in whom severe exudative conjunctivitis developed as

an adverse reaction attributable to daily rifampicin 450 mg and state that "rifampicin alone has not yet been reported to be oculo-toxic." In fact, conjunctival reactions to both daily and intermittent rifampicin are well documented and not uncommon, although the severity in Drs Cayley and Majumdar's patient was perhaps unusual. In a recent study involving 396 Chinese patients treated with rifampicin plus ethambutol in Hong Kong^{1, 2} cutaneous reactions consisting of "flushing and/or itching, with or without a rash, involving particularly the face and scalp, and often including redness and watering of the eyes" (my italics) were reported in 4% of the patients on daily, 8% of those on twice-weekly, 14% of those on once-weekly, and 5% of those on daily and then once-weekly rifampicin during the first 12 months of chemotherapy.² In almost all of these patients the reactions were attributable to the rifampicin.

D J GIRLING

Medical Research Council
Tuberculosis and Chest Diseases Unit,
Brompton Hospital,
London SW3

- ¹ Aquinas, M, *et al*, *British Medical Journal*, 1972, **1**, 765.
- ² Hong Kong Tuberculosis Treatment Services/Brompton Hospital/British Medical Research Council, *Tubercle*, 1974, **55**, 1.

United Kingdom in Figures, 1975 edition

SIR,—This little information card¹ issued free by the Government, contains a wealth of statistical information which is most useful for reference and teaching purposes. In the sections devoted to population and vital statistics and health are to be found, among other items, health indicators such as life expectancy at birth, crude death rate, and proportional mortality ratio,² but conspicuous by its absence is the infant mortality rate.

Britain's infant mortality rate has dropped behind those of many other developed countries in recent years³ and some statisticians have attempted, perhaps chauvinistically, to cast doubts on its validity as a health indicator. The rate has nevertheless accurately reflected slower national growth in many other aspects of the level of living. It is likely, however, that we shall see continuing improvements in infant mortality rates, especially if significant progress is made in preventing sudden deaths in infancy⁴ and if we experience the sharp fall in infant mortality that has been observed in other countries with low fertility and high abortion rates, possibly reflecting better care of fewer, healthier, wanted children.

The infant mortality rate remains the most useful single measure of the health of a nation and, whatever the reason for its omission, I hope we shall see it reinstated in the 1976 and subsequent issues of this useful publication, along with neonatal and postneonatal rates, if space permits.

M J BALL

Hertfordshire Area Health Authority,
Hemel Hempstead,
Herts

- ¹ Central Statistical Office and Central Office of Information, *United Kingdom in Figures, 1975 edition*. London, HMSO, 1975.
- ² United Nations Organisation, *International Definition and Measurement of Levels of Living: an Interim Guide*. New York, United Nations, 1961.
- ³ Weatherall, J A C, *Population Trends*, 1975, **1**, 9.
- ⁴ Fredrick, J, *British Journal of Preventive and Social Medicine*, 1973, **27**, 217.