their plantar fasciitis was secondary? It is well known that the peripheral manifestations of ankylosing spondylitis often respond well to deep x ray and, perusing the table of clinical details of the 17 patients, it appears that at least six of the responders did have an underlying seronegative spondylarthritis of some variety.

It would therefore be wrong to conclude from this report that when "simple" plantar fasciitis fails to improve with conventional treatment it is likely to respond to radiotherapy. A further study of "simple" plantar fasciitis in which seronegative spondylarthritis has been excluded is clearly required.

D N GOLDING

Department of Rheumatology,
Princess Alexandra Hospital,
Harlow, Essex

Spurious polycythaemia in a neonate?

SIR,—There have recently been published a short report by Dr L E Ramsay (13 May, p 1251) and a letter from Drs B W S Robinson and D Corless (22 July, p 280) referring to Gaisböck's syndrome (spurious or "stress" polycythaemia or pseudopolycythaemia). We have recently seen a neonate who may have been suffering from the same syndrome and I have as yet been unable to find any references to this syndrome in neonates.

A 5-day-old full-term child was transferred to our unit with a diagnosis of Gram-negative neonatal meningitis made on day 3. The initial haemoglobin concentration was reported as 18 g/dl; when repeated on day 5 by heel prick it was 21 g/dl. It was thought that the child might be dehydrated; however her serum electrolyte values were normal and her fluid intake by intravenous infusion was adequate. Alternatively the heel prick might have given a spurious result; the haemoglobin estimation was repeated by venepuncture and the result was 20.9 g/dl. There was no evidence of renal disease or cardiac disease throughout her illness and as her meningitis resolved her haemoglobin level returned to 17-18 g/dl over three weeks.

I wonder whether this syndrome has been described in neonates and whether any other readers have come across the situation.

S J ROSE

Royal Devon and Exeter Hospital
(Wonford),
Exeter

Antifibrinolytic agents for ruptured intracranial aneurysms

SIR,—In contrast to the study by Mr R S Maurice-Williams (15 April, p 945), which suggests a beneficial effect of tranexamic acid after rupture of an intracranial aneurysm, we found in a double-blind controlled clinical trial no difference in either mortality or rebleeding rates.¹

Mr Maurice-Williams's study lacked a double-blind procedure and the patients investigated were restricted to those in Botterell's grades I and II. The actual numbers of patients in a state of a clear or clouded consciousness in the two groups are not given, but theoretically this can have biased the results. Our own trial confirmed that the state of consciousness after a subarachnoid bleeding appeared to be the most important factor with regard to mortality.

The author's remark in the discussion that "the same striking improvement" with regard to the use of antifibrinolytic agents has been described in other controlled trials is hardly substantiated by the literature cited. The control group in Sengupta's series²—not double-blind—contained twice as many severely ill patients as the treatment group. The author's reference (No 10) to a personal communication cannot be regarded as additional evidence. Nibbelink's study³ had a follow-up period of only 14 days. Gibbs⁴ compared two groups of patients which were not comparable—namely, an operated group versus a group which apparently could not tolerate operation and subsequently was treated with tranexamic acid.

We do not think that this report adds valuable information to substantiate the assertion that tranexamic acid is of any use in the treatment of ruptured intracranial aneurysms.

J VAN ROSSUM
A R WINTZEN

Department of Neurology,
University Hospital,
Leiden, Netherlands

- ¹ van Rossum, J, *et al*, *Annals of Neurology*, 1977, 2, 238.
- ² Sengupta, R P, So, S C, and Villarejo-Ortega, F J, *Journal of Neurosurgery*, 1976, 44, 479.
- ³ Nibbelink, D W, Torner, J C, and Henderson, W G, *Stroke*, 1975, 6, 622.
- ⁴ Gibbs, J R, and Corkill, A G L, *Postgraduate Medical Journal*, 1971, 47, 199.

Autoimmunity in juvenile diabetics and their families

SIR,—Dr G F Bottazzo and his colleagues (15 July, p 165) re-emphasise the importance of both pancreatic islet cell and other antibodies in type I, "juvenile," diabetes. While these may be of importance in the aetiology of the disease, the evidence for their importance in the microangiopathic complications of diabetes is not proved by the evidence provided and their statements in the summary are misleading.

Diabetic microangiopathy, of which retinopathy is a manifestation, occurs in both type I and type II diabetes and there is no evidence to suggest that it is commoner in type I disease. It is a manifestation of the duration and also the degree of control of the metabolic abnormality.¹ After 15 years' duration up to 82% are expected to have retinopathy² and after 20 years up to 100%.³ If autoantibodies were of significance angiopathy would be expected to occur earlier. No patient in this series with disease duration under 10 years had such findings. The seven patients in table II (not 10 as suggested in the summary) with microangiopathy had all had diabetes for 12 years or more. The three who had both retinopathy and nephropathy had had it for over 20 years. In this small study of eight patients with diabetes for 12 years or more seven (88%) had retinopathy. This is not excessive and in view of the small numbers no conclusions can be drawn.

Besides duration of diabetes, diabetic control is of importance in the development of retinopathy, as shown in randomised controlled studies in man⁴ and animals⁵ and in the prospective studies by Pirart.⁶ Members of the same family are likely to have not only similar antibodies but similar eating habits, the same physician, and similar type of management. Control of the disease therefore is likely to be similar. By giving autoimmunity an unwarranted importance in the develop-

ment of the long-term sequelae of diabetes the authors suggest that, since these are "inherited," management of diabetes is of little importance. This is a view I wish to oppose strongly.

EVA M KOHNER

Department of Medicine,
Hammersmith Hospital,
London W12

- ¹ Colwell, A R, in *Small Blood Vessel Involvement in Diabetes Mellitus*, ed M D Siperstein, A R Colwell, and K Meyer, p 253. Washington, DC, American Institute of Biological Sciences, 1964.
- ² Larsson, Y, Sterky, G, and Christianson, G, *Acta Paediatrica*, 1962, 51, suppl 130, p 1.
- ³ Dolger, H, *Journal of the American Medical Association*, 1947, 134, 1289.
- ⁴ Job, D, *et al*, *Diabetes*, 1976, 25, 463.
- ⁵ Engerman, R, Bloodworth, J N B, jun, and Nelson, S, *Diabetes*, 1977, 26, 700.
- ⁶ Pirart, J, *Diabète et Métabolisme*, 1978, 3, 97.

Lymphatic fistula: a complication of arterial surgery

SIR,—Mr R J Croft (15 July, p 205) was using the wrong dye. The Patent Blue V which is distributed by May and Baker Ltd for Laboratoire André Guérin is a 2.5% solution of a calcium salt of sulpham blue with a molecular weight of 1159 and a colour index of 42051. It differs from Patent Blue V (Sigma), which is an 11% solution, and Disulphine Blue (Imperial Chemical Industries Ltd), which is a 6.2% solution of the sodium salt of sulpham blue which has a molecular weight of 567 and a colour index of 42045. These dyes, injected into the foot, will colour the groin lymphatics and nodes.

My colleagues and I have used this technique in 38 patients undergoing femoral profundaplasty.¹ Groin lymph structures showed up well and were either avoided, ligated, or sealed with surgical diathermy. No lymph cyst or fistula developed.

A V POLLOCK

Scarborough Hospital,
Scarborough, N Yorks

- ¹ Leaper, D J, Evans, M, and Pollock, A V, *British Journal of Surgery*. In press.

Surgery for intracranial aneurysm in the elderly

SIR,—I read with interest the paper by Mr R P Sengupta and others (22 July, p 246) on surgery for intracranial aneurysm in the elderly, but I am puzzled by the authors' conclusions that surgery is justifiable in this age group. Their data would appear to confirm the feelings of others¹ that the results of such surgery are unrewarding.

Eight patients out of 32 (or of 25 if only those over 60 are counted) died or were left seriously disabled after surgery and a further three were left partially disabled. Furthermore, we are told nothing of those patients who died from rebleeding before surgery would have been carried out and who must surely be added to the overall surgical picture. Nor are we told the interval from the haemorrhage until operation, except in the cases of those patients who fared badly. Judging from the figures given for the latter it would seem that in many cases operation was performed late, after the period of maximum rebleeding risk had passed. A person who survives three weeks in good condition without rebleeding has only a 5-10% chance of dying from rebleeding from then on² and it is against this