(*c*) No ointment or dressing should be applied to the ulcer.

(*d*) Profuse discharge in the early stages may necessitate frequent changes of bandage. This can be prevented by cutting a hole in the bandage over the ulcer, so that the discharge can escape into a dressing which can be changed by the patient.

(*e*) A satisfactory bandage abolishes all swelling and quickly relieves all discomfort. A bandage which does not achieve these two aims is unsatisfactory and should be changed.

When an ulcer is healed the underlying veins should be treated. We agree that the removal of superficial veins is insufficient, for the disorder lies in the muscular pump. The part which damaged deep veins play in venous ulceration is yet to be defined, but ulcers only occur in the presence of incompetent perforating veins, and occlusion of these is always beneficial. The value of compressive bandages in venous stasis extends beyond the dermatological complications. For thrombophlebitis rest in bed is still advised by some despite the risk of deep vein thrombosis, while with compression bandages the patient can walk, and return to work in a few days. When the calf has been explored surgically, such as by Cockett's operation, the wound is bandaged as though it were an ulcer. Primary healing is then the rule, and an early return to work is possible.—We are, etc.,

Vein Clinic,
Royal Infirmary,
Cardiff.

HUGH JONES.
JULIAN TOWNSEND.

REFERENCE

Brodie, B. C., *The Works of Sir Benjamin Collins Brodie*, 1865, Vol. 3, p. 248. London.

SIR,—I read with interest the splendid article on venous leg ulcers by Dr. S. T. Anning (12 November, p. 1183) and the subsequent letter by Dr. W. A. Dewar (26 November, p. 1323), and would like to endorse their views in condemning the local application of steroids and antibiotics to these ulcers. This treatment is ineffective and often harmful to the patient, and reveals an ignorance of the underlying pathology.

Dr. Dewar describes a rapid deterioration of leg ulcers when treated with local application of corticosteroids, with deep penetration down to tendon and even to bone. While agreeing that deterioration is frequently observed in these cases, I have rarely seen deep penetration without coexistent arterial insufficiency.—I am, etc.,

Liverpool. DAVID SYKES.

SIR,—Mr. W. G. Fegan and Dr. J. M. Pegum (3 December, p. 1391) do not agree with my statement that deep-vein thrombosis, and the recanalization of the veins which follows, is the commonest reason for failure of the leg-muscle pump (12 November, p. 1183).

My statement was based on findings in an unselected series of 1,933 patients with venous leg ulcers. In 68% there was a clear history of such thrombosis, and in 14% the history was doubtful but suspicious. Details of the clinical conditions associated with venous thrombosis in a smaller part of the same series (963 patients) are given elsewhere.[1] Of these patients 74% had a history of deep-vein thrombosis. The incidence of post-thrombotic ulcers was found by Bauer[2] to be 80 to 90%, and by Boyd *et al.*[3] to be 70%. Many authors, I admit—for example, Dodd and Cockett[4]—believe the incidence of post-thrombotic ulcers to be lower, but the figures quoted are from quite large series and from varying types of clinic.

In view of this evidence I see no reason to change the opinion I expressed, based as it is on my findings and on those of others. However, I entirely agree with Mr. Fegan and Dr. Pegum on the importance of incompetence of the valves of the perforating veins. In my view this is usually secondary to hypertension in the deep veins, the result of previous thrombosis with damage to the valves of those veins.—I am, etc.,

Leeds. S. T. ANNING.

REFERENCES

1 Anning, S. T., *Leg Ulcers: Their Causes and Treatment*, 1954, p. 43. Churchill, London.
2 Bauer, G., *Lancet*, 1946, **1**, 447.
3 Boyd, A. M., Jepson, R. P., Ratcliffe, A. H., and Rose, S. S., *Angiology*, 1952, **3**, 207.
4 Dodd H., and Cockett, F. B., *The Pathology and Surgery of the Veins of the Lower Limb*, 1956, p. 345. Livingstone, Edinburgh and London.

Mercurial Poisoning and Aplastic Anaemia

SIR,—The following report is of a patient with aplastic anaemia who had ingested metallic mercury continuously over a period of 33 years. He had a high urinary excretion of mercury, but no other symptoms of mercurialism.

The patient, a 77-year-old man, was admitted to hospital with blood loss per rectum. He had been transfused with 14 pints (8 litres) of blood previously. His haemoglobin was 46% (6.8 g. per 100 ml.), W.B.C. 2,400 per cu. mm. (17% polymorphs, 83% lymphocytes), platelet count 10,000 per cu. mm., blood group O Rh D-negative. Bone-marrow examination showed marked decrease in cellularity, and megakaryocytes were virtually absent. The appearance was typical of aplastic anaemia. In spite of blood transfusion and steroid treatment, which maintained the haemoglobin level for a short time, the patient had a severe haemorrhage 18 days after admission and died.

When the patient's history was taken the usual marrow toxins were considered, but the only relevant information was that the patient took tablets of hydrarg. cum creta, because he had been told 33 years previously that he might have a "syphilitic lesion," and that he should never be without the tablets. Each tablet contained one grain (65 mg.) of metallic mercury. As he had taken one on every day of every other month for 33 years it can be calculated that he had taken 360 g. of mercury during that time.

There were no other symptoms of chronic mercurial poisoning, except that the patient was known to have been temperamentally difficult for some time. Giese has described this in chronic poisoning.[1] The patient's urinary excretion of mercury was estimated and found to be at the high level of 674 μg. in 24 hours. Buckell[2] considered that a figure of 0.1 to 1.0 μg. of mercury excreted in the urine per 24 hours to be a normal value; in subjects suffering from chronic mercurialism he found that about 1,000 μg. of mercury would be excreted in 24 hours.

It is interesting to note that aplastic anaemia was described by Ehrlich in 1888[3] and at that time mercurials in various forms were being used in abundance to treat syphilis. However, it seems that aplastic anaemia was never described in association with this treatment, neither was it described in workers exposed to mercury in industry.[4] Whether there is any aetiological relationship between the ingestion of mercury and depression of bone marrow without any other symptoms of mercurialism, as commonly recognized, remains speculative.

I am grateful to Dr. R. A. Bruce, consultant physician, Wharfedale Group of Hospitals, for permission to publish this letter and for help in its preparation, and to Dr. K. M. Jones, of the Department of Chemical Pathology, University of Leeds, for the mercury estimations.

—I am, etc.,

Wakefield, Yorkshire. D. R. WILSON.

REFERENCES

1 Giese, A. C., *Science*, 1940, **91**, 476.
2 Buckell, M., Hunter, D., Milton, R., and Perry, K., *Brit. J. industr. Med.*, 1946, **3**, 55.
3 Ehrlich, P., *Charite-Ann.*, 1888, **13**, 300.
4 Hunter, D., *The Diseases of Occupation*, 2nd ed., 1957. English University Press, London.

Fluphenazine Enanthate in the Maintenance Treatment of Schizophrenia

SIR,—Although fully appreciating the important therapeutic potential of a drug which may be administered to schizophrenics who fail to take phenothiazine tablets after they have been discharged from hospital, my personal experiences with fluphenazine enanthate have hitherto proved somewhat disappointing. A typical case I have been treating illustrates this.

A schizophrenic farm labourer, aged 27, has had three admissions to this hospital in the past two years. After admission, delusions and hallucinations have always been rapidly controlled with chlorpromazine 100 mg. t.d.s., trifluoperazine 5 mg. t.d.s., and Cogentin (benztropine methanesulphonate) 1 tablet t.d.s. After discharge he soon ceases to take either tablets or syrup, fails to attend outpatient appointments, and invariably relapses. Fluphenazine enanthate injections were commenced following his last admission in August—12.5 mg. or 0.5 ml. initially followed by 25 mg. fortnightly, with excellent symptomatic improvement. After four injections had been given he complained of tremors and severe rigors following each injection, which lasted for over a week and completely prevented him from working. As he had persistently failed to take all tablets in the past for more than a few days when at home, it was considered unlikely that he would persevere with oral anti-Parkinsonian agents. Attempts are therefore being made to continue treatment by adjusting dosage.

I entirely agree with Dr. J. Lowther (19 November, p. 1262) that there is a real need for a similar long-acting anti-Parkinsonian preparation which could be combined or administered with the injections of fluphenazine enanthate. To me the object of a long-acting injection seems to be largely negated if one then has to resort to tablets to control side-effects.—I am, etc.,

Shelton Hospital, Shrewsbury, Salop. J. C. BARKER.