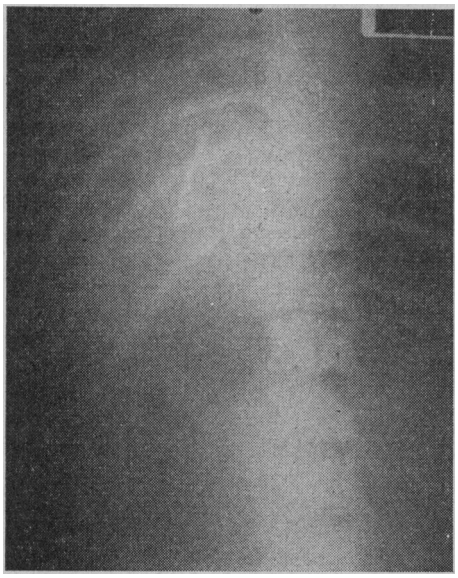
X-ray showed a foreign body of the shape of a long nail lying anterior to the spine in the upper abdomen just below the diaphragm (see Fig.). Anti-shock measures were instituted and blood was given intravenously. Later we were told he was working with a cartridge hammer and the hammer was fired by his colleague accidentally and the nail was shot into his thigh. The conclusion was drawn that the nail had travelled up his thigh, through the greater sciatic foramen, and had reached the upper abdomen.

Operation was performed after resuscitation under general anaesthesia. A right posterolateral incision excising the eleventh rib was made. There were signs of bruising retroperitoneally and on the anterior surface of the right kidney. The incision was extended into the peritoneal cavity, which contained much blood. There was no escape of gas and no faecal odour. The contents of the abdomen were systematically examined and there were four perforations in the



ileum, but no damage to other structures was found. The nail was felt embedded in the posterior part of the right lobe of the liver. The direction of the nail from the thigh right up to the upper abdomen might be expected to result in injury to adjacent viscera and structures such as the caecum and sciatic nerve. The fact that no other lesion resulted was fortunate and remarkable.

The perforations were repaired, then attention was turned to the nail, which was pulled out with little bleeding from the liver. The wound was closed in layers with Penrose drains from the peritoneal cavity. A Berman metal locator was available in the theatre. The patient made slow but good recovery and was discharged home on 15 August.

Two months later he was readmitted with a diagnosis of sudden rupture of an abdominal viscus. Laparotomy revealed a perforation in the terminal ileum. The perforation was repaired and the patient made a good recovery, but now he has signs and symptoms of subacute intestinal obstruction due to adhesions, for which a further laparotomy may be necessary.

It is believed that the manufacture of this cartridge hammer has stopped and that a modified form which is thought to be safer is now being made.

I wish to express my thanks to Mr. R. Derek Richards, F.R.C.S., consultant surgeon, Royal Gwent Hospital, Newport, Monmouthshire, for his encouragement and permission to publish this case, which was admitted under his care.

—I am, etc.,

MOHAMMAD ZAFRULLAH.

Royal Gwent Hospital,
Newport, Mon.

Epidemic Hysteria

SIR,—Dr. P. D. Moss and Dr. C. P. McEvedy in their article "An Epidemic of Overbreathing Among Schoolgirls" (26 November, p. 1295) refer to the theory of Professor H. J. Eysenck concerning the personality correlates of the neuroses, in particular of hysteria. While many other predictions from this general theory have been amply confirmed, there has been widespread failure to distinguish hysterics from normals along the extraversion dimension.

The results of seven investigations of this problem were summarized (together with the results of their own investigation) by Dr. J. G. Ingham and Dr. J. O. Robinson.[1] In none of these eight investigations was the hysteric group found to be more extraverted than the normal control group; in fact there was a tendency for the hysterics to be slightly *less* extraverted than the normals.

These facts would serve to explain the very small differences in extraversion found between affected ("hysterical") and unaffected ("normal") groups in the Blackburn epidemic reported by Moss and McEvedy, and the lack of any difference at all between affected and unaffected groups in the Portsmouth epidemic described by Drs. C. P. McEvedy, A. Griffith, and T. Hall (26 November, p. 1300), which might otherwise be thought to give some degree of uncertainty to the conclusion that the epidemics were, in fact, hysterical.—I am, etc.,

University College, Cardiff. H. L. WAGNER.

REFERENCE

[1] Ingham, J. G., and Robinson, J. O., *Brit. J. Psychol.*, 1964, **55**, 276.

Sex Chromatin

SIR,—The observations by Drs. K. T. Shetty, N. L. Sharma, and K. M. Wahal (9 July, p. 84) that prednisone caused a fall in the number of sex chromatin bodies seen in buccal smears prompted a similar investigation on a number of physically normal patients not receiving drug therapy. Counts were performed on two patients at the same time each day (12 noon) over a period of three months, using the method of Sanderson and Stewart.[1] One, a woman of 39 years, has shown a marked rise of from 30 to 40% to 60 to 70% regularly for a day or two after menstruation and also a similar rise 13 days later. The other, a 23-year-old woman who was receiving Sequens (mestranol, chlormadinone actetate) for dysmenorrhoea, showed a rise and fall of 30 to 60 to 70% every five to seven days.

Counts performed on four physically normal women between the ages of 25 and 40 at three-hourly intervals have shown great variation over the 24 hours, with as much as 30% difference in three hours. While the number of cases is very small it seems likely that there is a variation in count both over the 24 hours of the day and also over the 30 days of the month, and suggests that the usual practice of relying on one or two slides taken at a specific time of day may not be reliable if the percentage figure is very low.

Three other female patients in the age group 30 to 55 suffering from endogenous depression had counts which were high in the morning—6 a.m. to 12 noon—with a gradual fall in the afternoon and evening. Three schizophrenic patients in the same age group had counts which were low in the morning and rose in the afternoon and evening.—I am, etc.,

T. S. DAVIES.

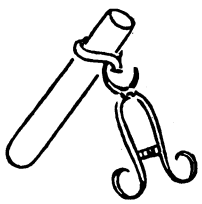
Llanfrechfa Grange Hospital,
Cwmbran, Mon.

REFERENCE

[1] Sanderson, A. R., and Stewart, J. S. S., *Brit. med. J.*, 1961, **2**, 1065.

The Diathermy Quiver

SIR,—A simple solution to the problem of a safe and satisfactory diathermy quiver might be the adoption in British hospitals of the technique which has been used here for many years (probably, if the truth were



known, because it is so inexpensive). The accompanying line-drawing shows how our scrub nurse attaches a large sterilized glass test tube to the drapes, using a towel clip and an elastic band.

To my knowledge this glass "quiver" has never been responsible for any of the trouble described in your columns.—I am, etc.,

Vancouver, B.C. T. F. WILKIE.

Venous Leg Ulcers

SIR,—Dr. S. T. Anning's article on "Venous Leg Ulcers" (12 November, p. 1183) contains valuable advice which if universally applied would greatly reduce the morbidity and loss of working time for which ulcers are responsible at present.

There is of course nothing new in the compression treatment of venous ulcers, and it is interesting that Benjamin Brodie[1] described in detail the same technique of bandaging as that used by Dr. Anning. Brodie also recognized that venous ulcers were healed by improving the flow of venous blood, and he advised against the use of ointments.

Why then have so many ulcers, and certainly the majority referred to this clinic, never had the benefit of compression bandages, but instead have been treated with a variety of local applications, none of which is of proved value?

The answer, we feel, is that many doctors may have tried bandaging without perhaps appreciating details of technique which make the difference between failure and success. We believe the following points to be important, and make no apology for reiterating some of what Dr. Anning has said.

(*a*) The bandage need not be elastic but must be strong; venous pressure reaches 100 mm. Hg. Elastic crêpe bandages are useless.

(*b*) The applied pressure must increase from above downwards, so that venous blood is squeezed upwards. This is easily achieved by applying the bandage with constant tension and by increasing the extent by which each turn overlaps the previous one from nearly nothing at the top to almost complete overlap at the foot.

(*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.