is in the vicinity of 5,000 blood donations annually, apart from the associated equipment for the preparation of platelet concentrates and the salary of a technologist over and above the salaries of the blood collection staff necessary.

In this country we have available a "mini-exchange" set, comprising a needle and tubing connected to one limb of a Y-piece which has a free length of tubing from another limb, and the third limb fits the needle adaptor on the standard giving set. This enables the whole manoeuvre of mini-exchange and transfusion to be carried out with a single venepuncture.—I am, etc.,

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1 Shaw, A. E., Medical Journal of Australia, 1969, 2, 329.
2 Lancet, 1969, 2, 1176.
3 Obtained from Yuta Laboratories, Lane Cove, Sydney, N.S.W., Australia.

Tuberculous Meningitis in Children

Sir,—May I comment on one point in your recent leading article on this subject (2 January, p. 1) "An intradermal tuberculin test (Mantoux 1:1,000) should be carried out at once, but in advanced cases a negative result does not rule out tuberculous meningitis." Surely a negative result does not rule out an early case either? This warning is particularly needed in view of the higher incidence of the disease in underdeveloped countries where tuberculin reactions are often suppressed.2 3—I am, etc.,

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Low Molecular Weight Dextran in Systemic Sclerosis

Sir,—Dr. Peter Lane's paper (12 December, p. 657) calls for comments on at least two major points. He implies in his introduction that he attempted to repeat my findings by using identical methods and treatments but my paper, as its title indicates, dealt with the "effect of intermittent low molecular weight dextran infusions upon the digital circulation in systemic sclerosis." In fairness, Dr. Lane states at the end of his paper that final conclusions cannot be drawn from his own series of patients since each patient received only one infusion of low molecular weight dextran. I have more recently emphasized that patients who have not previously been treated with this regimen may require infusions of 2 litres of low molecular weight dextran, given over 48 hours, initially at intervals of three to four weeks or three to four occasions, before the peripheral bloodflow is improved. Subsequently intervals between infusions have been extended and my first 3 patients, treated since 1964, have not required dextran infusions for the past 14 months because of their maintained satisfactory digital bloodflow. Each has received 15 infusions so far. Others have confirmed the efficacy of this regimen.4 It is important to ensure that the kidneys are protected against unduly high concentrations of dextran. Our nursing staff has instructions to slow down the rate of infusions (normally 2 l over 48 hours) if the specific gravity of urine exceeds 1.045. During the past 6 months we have treated 35 patients suffering from systemic sclerosis and 98 affected by other disorders associated with obligatory disorders of small blood vessels with a total of 1,121 dextran infusions without adverse renal side effects in a single patient. Dr. Lane rightly states that systemic sclerosis is an intractable condition for which there is no specific treatment. Ischaemic digital changes, with or without intractable ulceration, can make the life of these patients utterly miserable. I hope that Dr. Lane's paper will not dissuade any physician from using a treatment which, in my experience, is beneficial as well as safe provided simple precautions are observed.—I am, etc.,

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Prostatic Acid Phosphatase

Sir,—I have read with interest the report of the detection of the antigen-antibody interaction in gel diffusion precipitation and enzymatic analysis.2 Results suggest that benign and malignant prostatic tissue are, in comparison with normal prostatic tissue, antigenically different. The possibility of a normal prostatic-tissue-specific antigen, particularly prostatic tissue-specific acid phosphatase antigens, in extract preparations of malignant prostatic tissue has been firmly supported by histochemical and histochemical reports of diminished quantities of acid phosphatase in patients with advanced carcinoma of the prostate.1 The possible prognostic and therapeutic value of the detection of the antigenic deficiency of specific prostatic tissue-specific acid phosphatase antigens looms as a possibility pending the demonstration of a definitive correlation between the presence or absence of specific tissue-specific acid phosphatase antigens and the histological configuration of the stage of prostatic carcinoma.—I am, etc.,

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