positioned. Failure is only likely when the saphena is duplicated. Making the probe elastic, as Murphy suggests, would help, especially when it is passed from below upwards, as the late Sir Henry Gray, who introduced the method into Britain, often did. The results of extraction were excellent in 20 soldiers traced five years after operation (*Brit. J. Surg.*, 1921, **8**, 486), but subsequent experience has shown that they are perhaps not so good in civilians.

The danger of deep thrombosis being caused by overflow into the deep veins, given as a possible reason for the newer method, must be rare in the absence of previous white leg, in which case any treatment other than Unna's semi-rigid method, used hydrostatically over a long period, seems to be contraindicated. The use of the larger salicylate and other injections, which often produced severe cellulitis, is largely in abeyance. The smaller quinine injection seems to carry no such risk, as the deep vein dilution is large and sudden, and the results are uniform and very good. A disadvantage is that the solution is not guidable by the pain it produces. It was shown (*British Medical Journal*, 1929, **2**, 848) that solutions which produced delayed pain after injection could be easily guided into any desired area affected with varicosity. Until someone with suitable material adjusts the percentage of urethane so as to produce only slight pain on injection we shall have to continue with the more haphazard method and hope that previous ligatures will not direct too large a quantity of the solution into the deep veins.—I am, etc.,

London, W 1. G. H. COLT.

Blood Changes and Diagnosis of Infectious Mononucleosis

SIR,—In their article on "Infectious Mononucleosis With Thrombocytopenic Purpura" (November 1, p. 977), Drs. C. M. Ogilvie and T. E. Parry suggest that patients presenting with purpura should have a Paul–Bunnell reaction carried out to exclude the possibility of infectious mononucleosis.

The only point with which we disagree is their obvious reliance on the Paul–Bunnell test for confirmation of the diagnosis. It has been our experience and that of others (Fuller, 1941[1]; Paul, 1941[2]; Vander Meer *et al.*, 1945[3]; Warren, 1941[4]; and Kaufman, 1944[5]) that cases commonly occur which are clinically and haematologically indistinguishable from infectious mononucleosis, but in which the Paul–Bunnell test is consistently negative. Moreover, in many cases the Paul–Bunnell test does not become positive until well after the clinical condition has subsided. Kaufman records many instances in which the heterophile antibodies did not appear until periods ranging from two to six months after recovery. However, in all cases of infectious mononucleosis the abnormal lymphocytes characteristic of the disease are invariably present, even in the absence of an absolute or relative lymphocytosis. We feel, therefore, that the blood changes are more important as an aid to diagnosis than the Paul–Bunnell test, although, of course, the finding of a positive Paul-Bunnell test would be further evidence in support of the diagnosis. We are at present investigating the association of mononucleosis with the Paul–Bunnell reaction, and this will be the subject of a future communication.—We are, etc.,

J. G. COLLEE.
S. SHUBERT.

Newcastle-upon-Tyne.

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1 *Lancet*, 1941, **1**, 69.
2 Ibid., 1941, **1**, 584
3 *Amer. J. med. Sci.*, 1945, **210**, 765.
4 Ibid., 1941, **201**, 483.
5 *Ann. intern. Med.*, 1944, **21**, 230.

Acquired Sensitivity to Streptomycin and P.A.S.

SIR,—Three cases have recently been described of acquired sensitivity to streptomycin and P.A.S.[1 2 3] One of these[1] was characterized by exfoliative dermatitis. The following case in which there was also exfoliative dermatitis seems worth recording.

The patient, a female aged 27 years, was receiving out-patient treatment with streptomycin (1 g. daily) and sodium P.A.S. (12 g. daily) for tuberculous endometritis. In the third week of treatment she began to suffer from sickness which persisted despite the fact that the P.A.S. was administered in different forms. On the evening of the 22nd day of treatment she had a severe rigor, and on the following day she became hot and flushed and developed on the trunk an erythematous eruption with large urticarial patches. There was intense pruritus. The rash spread to involve almost the entire body. There was well-marked oedema of the lower limbs and face, the latter resulting in closure of the eyes. The patient was extremely ill. There was no jaundice. P.A.S. and streptomycin were discontinued. Subcutaneous adrenaline produced only slight relief of symptoms and large doses of an antihistaminic drug, phenindamine hydrogen tartrate ("thephorin"), were given. Gradually the temperature returned to normal, and the oedema, urticaria, and pruritus disappeared. On the seventh day of illness the skin of the eyelids began to desquamate, and in the following two weeks the skin of the greater part of the body was shed, often in quite large plaques. The entire illness lasted for three weeks.

It was considered at that time that sensitivity to P.A.S. rather than to streptomycin was likely to have caused the illness, and this diagnosis appeared to be confirmed when, after the acute phase of the illness, a skin patch test with a 20% P.A.S. solution gave a strongly positive reaction. It was decided to continue treatment with streptomycin alone, but after one other injection (1 g.) a reaction developed similar to the first. The temperature rose, there was intense itching, and a widespread, though faint, erythematous eruption developed. Urticaria was less marked than during the first illness, but desquamation again followed, though not to such an alarming extent as on the first occasion. The most dramatic feature of the second reaction was the fact that the hair of the head began to fall out, depilation progressing to such an extent that the entire scalp and both eyebrows were denuded of hair. Hair was also shed from the extremities. Fortunately within two weeks the hair began to grow again and eventually returned to normal in quantity, texture, and colour.

I wish to thank Dr. A. M. Sutherland, Southern General Hospital, for access to the hospital case-record, and Dr. W. Blair for details of the illness while the patient was at home.—I am, etc.,

Glasgow. R. J. CUTHBERT.

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2 Julian, D. G. (1952). Ibid., **2**, 476.
3 Macpherson, P. (1952). Ibid., **2**, 723.

Arterial Spasm in Limb after Venepuncture

SIR,—Dr. M. Sutton's article on "Arterial Spasm due to an Intravenous Infusion" (October 18, p. 859) and the subsequent correspondence (November 15, p. 1097) make one wonder whether some of the effects of so-called "arterial injection" of thiopentone are not really caused by extravenous injection in the region of the brachial artery in patients whose vascular system is particularly susceptible to irritant solutions.

Recently I examined the forearms of 50 male and 50 female patients to ascertain the incidence of abnormalities of the brachial artery, and the relationship of the superficial veins in the antecubital fossa to the artery. In over 50% of patients the bicipital fascia was apparently poorly developed, and with the arm fully extended the artery was superficial for at least two-thirds of its course through the antecubital fossa. In 30% of males either the median cubital or the median basilic vein lay directly over the artery above the bicipital fascia. These figures suggested to me that in certain circumstances it would not be difficult to transfix the artery, which when the arm is extended is stretched over the supinator muscle, and relatively immobile.

Although on occasion I have inadvertently injected thiopentone subcutaneously in the antecubital fossa, I have never seen vasospasm. In view of what has already been published in these columns it would be interesting to know whether other anaesthetists have seen vasospasm of the brachial artery and its branches after thiopentone has been injected into or around the median cubital or basilic veins.—I am, etc.,

London, S.E.11. D. A. B. HOPKIN.