SIR,—I am sure many doctors in Britain will support Dr. Shee's plea for Birmingham University to revoke its decision on Salisbury Medical School (28 March, p. 817). Quite apart from the phariseeism of the Birmingham students, one wonders whether Government pressure had anything to do with the decision.

Your leading article on colour in Rhodesia (21 March, p. 705) oversimplifies the issue. It is difficult to see what effective protest can be made which will not backfire on the African doctors and patients, as has happened so often in South Africa.

Salisbury is an exciting medical school whose importance is vital for the future of medicine in Central Africa—not Rhodesia. It will be virtually impossible for African medical students there ever to leave the country, let alone find places in medical schools which can never fit them quite so well for work in their own environment.

The teaching staff at Salisbury are wide awake to the medical problems of developing Africa, and acutely aware of the personal, social, and political difficulties facing their African students. To embarrass them in this way is a great folly of which Birmingham should be deeply ashamed. Will a Salisbury graduate cease to be a doctor as soon as he steps over the Rhodesian border? Africa has few enough doctors. Nothing Birmingham can do will compensate for the loss of these graduates from Salisbury.—I am, etc.,

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Allergic Reaction to Quinestrol

SIR,—One-dose quinestrol tablets are being used with increasing frequency by maternity units.[1-5] Apart from nausea and vomiting few side effects have been reported. We wish to report a moderately severe allergic reaction.

A healthy 30-year-old patient with no history of hay-fever, asthma, or eczema was seen in her second pregnancy. Her first pregnancy, three years previously, had been uneventful. The present pregnancy was a twin one, and she had mild toxaemia which settled with bed rest and sodium amylobarbitone capsules. The patient was not on any therapy when she started in labour at 38 weeks. The sedation she received during labour was by the intramuscular route (10 mg./heroin, and later 100 mg./pethidine). The normal delivery of a 2·9 kg male and a 2·79 kg. female was followed by an intravenous injection of 0·5 mg. ergometrine and a normal third stage.

As the patient did not wish to breast feed lactation was suppressed by a single dose of quinestrol orally. Eight hours later the patient began to complain of an itching sensation over her arms and legs. By the next day this area was covered by an urticarial reaction, which became moderately severe. Slight dyspnoea was admitted to on questioning, though the patient was not shocked. Oedema of the hands and feet was noted on examination. The patient's condition rapidly improved following the injection of 10 mg. chlorpheniramine (Piriton) intramuscularly. This dose was repeated every eight hours over the next three days, by which time the rash had completely cleared. The oedema resolved in a few hours after the injection of the chlorpheniramine.

A rough patch test of quinestrol powder in alcohol, against an alcohol control, produced a local reaction.—We are, etc.,

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Serum and Vitamin B_{12}

SIR,—In his review of the literature on the correlation between serum and urinary vitamin B_{12} levels Dr. J. F. Adams (17 January, p. 138) seems to have overlooked our recent paper[1] in which we showed that urinary vitamin B_{12} levels rise sharply during acute hepatitis. In our single case of chronic myeloid leukaemia urinary vitamin B_{12} was not raised in spite of an elevated serum vitamin B_{12}. This suggests that the α-globulin vitamin B_{12} binder (TC I of Hall and Finkler[2]), responsible for serum vitamin B_{12} elevation in chronic myeloid leukaemia, might be less susceptible to urinary loss than the β-globulin binder (TC II). Our finding that relatively more β-globulin-bound vitamin B_{12} than α-globulin-bound vitamin B_{12} was lost in the urine during acute hepatitis supports this suggestion. Hom and Olesen[3] have furthermore shown that the β-globulin-binder has a much smaller molecule (approx. 40,000) than the α-globulin-binder (approx. 120,000); possibly the smaller molecule is more easily lost through the kidneys.—I am, etc.,

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Drugs for Gastric Ulcer

SIR,—In his letter (21 March, p. 757) Dr. M. J. S. Langman considers that the numbers of patients who had been treated with Cedona deglycyrrhizinated liquorice were insufficient to prove that this substance is as effective as carbenoxolone in treating patients with gastric ulcer, despite the fact that in each of the three studies cited the results were statistically significant.

I should like to point out that in addition to these studies there are many other published trials with Cedona deglycyrrhizinated liquorice which are mainly of continental origin. In all, some twenty trials have been published between 1958 and 1969 with a total of 911 patients, of whom some 130 suffered from gastric ulcer and the rest suffered from duodenal ulcer. The results from all these trials and those of carbenoxolone are in close agreement, and it can be concluded that deglycyrrhizinated liquorice (Cedona) is as effective in treating gastric ulcers as carbenoxolone but is almost entirely devoid of side-effects.

Other British trials not yet published again support these findings, as do the pharmacological and metabolic studies with Cedona deglycyrrhizinated liquorice.—I am, etc.,

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Diuretics in Hypertension

SIR,—I read with interest the paper by Dr. J. J. Healy and his colleagues on the body composition changes in hypertensive subjects on long-term oral diuretic therapy (21 March, p. 716).

The conclusions that K depletion is not severe and that it may not require treatment is borne out by prolonged clinical experience, so long as it is clearly understood that this relates only to uncomplicated hypertension in patients who take a normal mixed diet. It does not relate to patients with accelerated hypertension, when secondary hyperaldosteronism is practically constant, to patients with chronic pyelonephritis with renal potassium loss, and to patients in cardiac failure.

It is a pity that the diuretics chosen for study were chlorthalidone and frusemide. Chlorthalidone, like other long-acting diuretics, favours potassium loss, and frusemide is unnecessarily violent and short-acting and should be reserved for patients with associated severe cardiac failure. Both these preparations are expensive—an important consideration when treatment may be life-long. I strongly favour bendrofluazide, an effective, small, and cheap tablet.—I am, etc.,

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Fibrinolysis and Menstrual Bleeding

SIR,—Your leading article on fibrinolysis and menstrual bleeding (10 January, p. 61) set me thinking along some strange lines. It is well known that premenopausal women appear to be partially protected against coronary thrombosis and that this protection seems to be lost after the menopause. The effect is plausibly attributed to endocrine differences between the two sexes although the precise mechanism remains obscure.

The explanation of the protective effect may be superficially much simpler. Apart from endocrine function, another major difference between men and premenopausal women is that the latter regularly lose 50-100 ml. of blood. It is just conceivable that simple blood loss could be the protective factor.

Clearly this hypothesis is exceedingly unlikely to be true and the postulation of endocrine effects on the vascular system seems to be much more reasonable. However, it seems to me that the bleeding idea may be worth investigating for several reasons:

(1) It might prove easier to test than the