monary embolus and no more bleeding at the operation site than would have been expected.

Of the 14 patients withdrawn because of failure to observe the protocol, one was in the heparin group (the preoperative dose was forgotten) and the remaining 13 in the stimulator group: in seven of these the surgeon omitted to use the machine and in six the machine failed to function.—We are, etc.,

I. LAWRENCE ROSENBERG
St. James's Hospital,
Leeds

MARY EVANS
A. V. POLLOCK
Scarborough Hospital,
Scarborough, N. Yorks

Survival after Postoperative Avascular Necrosis of Lesser Curve of Stomach

SIR,—I read with interest the paper by Dr. J. F. Halvorsen and others (14 June, p. 590) reporting the third death due to avascular necrosis of the lesser curve of the stomach following highly selective vagotomy (H.S.V.) for duodenal ulcer. I should like to describe a similar case in which the patient survived.

An otherwise healthy male patient aged 46 with a long history of chronic duodenal ulceration underwent H.S.V. on 28 November 1974. His condition remained satisfactory until the third postoperative day, when he developed left lower chest pain associated with fever, mild hypotension, and a pleural friction rub. It was assumed that he had developed a chest infection and he was treated accordingly with physiotherapy, systemic antibiotics, etc. Several hours later his condition deteriorated: his blood pressure fell, abdomen became distended, and bowel sounds were absent. An abdominal tap was performed and the fluid obtained was mistakenly thought to be blood. Intraperitoneal haemorrhage was therefore diagnosed and appropriate resuscitation was carried out. Once a suitable blood pressure was attained laparotomy was carried out, when a large defect was found in the lesser curve and about $2\frac{1}{2}$ l of blood-stained fluid, mostly gastric contents, in the peritoneal cavity. The defect was sutured and peritoneal toilet carried out. The patient's postoperative condition was satisfactory but he subsequently developed a left subphrenic abscess which required treatment with drainage and antibiotics. He was discharged on 10 January 1975 and when last seen at the clinic in May 1975 he was very well, had no recurrence of dyspepsia, and was prepared to go back to work.

This case also demonstrates the difficulty in diagnosis of this rare condition, which nonetheless should be thought of if deterioration occurs after H.S.V.

I should like to thank Mr. G. E. Schofield for permission to report this case which was under his care.

—I am, etc.,

STUART W. BRODIE
Law Hospital,
Carluke, Lanarkshire

G.M.C. and Indian Qualifications

SIR,—Mr. A. K. Varshneya (5 July, p. 43) writes that there is an increasing number of teaching schools in India with poorly equipped departments and that the standard of medical education has gone down in recent years. I spent nearly 13 years in the Indian Medical Service, so that I know the background, and I have been back twice, in 1969-70 and 1972, touring the Indian states on behalf of the World Health Organization. During these tours I visited a large number of primary health centres and district and general hospitals and some teaching hospitals. Though I saw in the primary health centres many examples of Indian doctors supplying a cheerful and unselfish service to their countrymen, the lack of even the simplest equipment and the elementary nature of the procedures in these centres was striking. In the district and general hospitals laboratory equipment was of the scantiest and in the teaching hospitals there was a conspicuous lack of the sort of modern equipment one would have expected to find there.

In the hospitals the few laboratory tests attempted were carried out usually in one room and there was a grave lack of adequately trained technical staff. I noticed that doctors generally had a knowledge of only the simplest techniques such as blood cell counting, examining malaria slides, or examining faecal specimens for helminth ova, and while one could argue that medicine could possibly be practised in rural India without a knowledge of laboratory procedures, such inadequately trained doctors would not be suitable for participating in a highly technical hospital service such as Britain's.

No doubt some graduates of the good Indian medical schools display a wider knowledge of medical practice, but those of us who know India well are aware that there are a great number of doctors coming to this country who are simply not well enough trained. It is absolutely imperative that there be a sorting-out examination, and no matter how disappointing its results may be for some candidates an exacting standard must be maintained.

It would be futile to run a three-month course (as has been suggested by an Indian doctor on the radio from Birmingham) to prepare doctors who have arrived in the U.K. to take the G.M.C. examination. How could a three-month habilitation course, especially for someone whose English is weak, make up for the deficiencies of several years in an inferior medical school?—I am, etc.,

R. J. HENDERSON
Public Health Laboratory,
Royal Infirmary,
Worcester

Sodium Nitroprusside in Anaesthesia

SIR,—We should like to amplify your brief statement (7 June, p. 524) on the metabolism of sodium nitroprusside (SNP).[1]

In patients infused with SNP we have found that there was an increase in blood cyanide (HCN) levels, even with small doses of the drug. Of this HCN, 98% was present in the red cells. Though β-mercaptopyruvate sulphur transferase occurs in the red cells of the rat,[2] it has not yet been shown to play any significant role in the direct detoxication of HCN in human erythrocytes. The blood HCN is slowly detoxicated to thiocyanate (SCN) by tissue rhodanese so that plasma SCN levels showed only a gradual rise. The ratio SCN:HCN in body fluids is normally around 1000:1 and so at the relatively low levels of SCN attained during hypotensive anaesthesia using SNP there would be insignificant formation of HCN from SCN by the ill-defined thiocyanate oxidase system.

You suggest a maximum SNP dose rate of 3 mg kg^{-1} h^{-1}, but this may be too high. Death due to cyanide poisoning is attributed to inhibition of cytochrome oxidase. In vitro 50% inhibition occurs at a concentration of 1·5 μmol HCN/l.[3] In vivo there will no doubt be a plasma/tissue concentration gradient and so plasma levels at which inhibition would occur would be somewhat higher. In dogs given a single intravenous dose of 1 mg SNP/kg, resulting in peak plasma levels of 3 μmol HCN/l, we have found evidence for histotoxic hypoxia.

Our studies in man have shown that plasma HCN levels immediately following short-term infusions (over a period of two hours or less) show a linear relationship to the *total dose of SNP*. A plasma level of 3 μmol HCN/l would be reached by the infusion of about 1·5 mg SNP/kg.

Deaths have been reported[4] following SNP infusions in which doses of approximately 4 mg/kg per two-hour period have been given. It therefore seems reasonable to suggest that plasma HCN levels should not be allowed to rise above 3 μmol/l and the maximum total dose of SNP be set at 1·5 mg/kg for hypotensive anaesthesia of relatively short duration. In the event of untoward symptoms attributable to SNP we would certainly encourage the use of hydroxocobalamin and sodium bicarbonate as you suggest, and in addition sodium thiosulphate.—We are, etc.,

CYRIL J. VESEY
PETER COLE
PETER SIMPSON
Department of Anaesthesia,
St. Bartholomew's Hospital,
London E.C.1

1 Vesey, C. J., *et al.*, *British Medical Journal*, 1974, **2**, 140.
2 Sorbo, B., in *Proceedings of the First International Pharmacological Meeting*, ed. B. B. Brodie, *et al.*, vol. 6, p. 21. London, Pergamon, 1962.
3 Schubert, J., and Brill, W. A., *Journal of Pharmacology and Experimental Therapeutics*, 1969, **162**, 352.
4 Merrifield, A. J., and Blundell, M. D., *British Journal of Anaesthesia*, 1974, **46**, 324.

Emigration of Doctors

SIR,—Dr. M. P. White (7 June, p. 561) writes from his experience as a district medical officer, but many of us who have served from the grass roots up to Ministry levels in many developing countries are unable to agree with his statements. There is no such thing as tropical medicine; it is the medical care of poverty, national and individual; and the paramedical and auxiliary cadres, urban and rural health centres, and a reconsideration of medical student training are all appearing in the so-called developed countries as they face up to the economic problems of medical care.

Dr. White specifically raises four points as some of the "certain facts . . . always ignored" by the protagonists of community health care.

I agree that in the developing countries there is a decline of morale, discipline, and output of the health services paralleling those of the U.K. But the root causes are very different. In the developing countries the indigenous are now taught by indigenous physicians and paramedicals who in turn were taught by expatriates. Thus the indigenous students continue to be taught an inappropriate medical care system. Indigenous training in many areas is still not orientated to a country's individual epidemiological and social needs and priorities. Doctors and paramedicals soon discover this